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MORTALITY, MORBIDITY, AND LEAD POISONING OF EAGLES IN WESTERN CANADA, 1986–98

MARK WAYLAND[1]
Environment Canada, P&NR, Prairie and Northern Wildlife Research Centre, Saskatoon, SK S7N 0X4 Canada

LAURIE K. WILSON AND JOHN E. ELLIOTT
Canadian Wildlife Service, P&YR, Pacific Wildlife Research Centre, Delta, BC V4K 3N2 Canada

MICHAEL J.R. MILLER
Iolaire Ecological Consulting, 210-112th St., Saskatoon, SK S7N 1V2 Canada

TRENT BOLLINGER
Canadian Cooperative Wildlife Health Centre, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK S7N 5B4 Canada

MALCOLM MCADIE
British Columbia Ministry of the Environment, 2080A Labieux Rd., Nanaimo, BC V9T 6J9 Canada

KEN LANGELIER
Island Veterinary Hospital, 1800 Bowen Rd., Nanaimo, BC V9S 5W4 Canada

JONATHAN KEATING AND JENNIFER M.W. FROESE
Environment Canada, P&NR, Prairie and Northern Wildlife Research Centre, Saskatoon, SK S7N 0X4 Canada

ABSTRACT.—Between 1986 and 1998, we investigated causes of mortality and morbidity, and assessed lead (Pb) contamination in Bald (*Haliaeetus leucocephalus*) and Golden eagles (*Aquila chrysaetos*) in western Canada. The most common diagnoses were trauma (13.0%), electrocution (11.5%), pesticide poisoning (7.6%), gunshot wounds (7.3%), and Pb poisoning (6.4%). It was not possible to determine the cause of mortality or morbidity in 29% of the 546 eagles. Excluding undetermined cases and those with an unknown collection date, toxicoses accounted for 40% of the diagnoses in the prairie provinces, but only 19% of those in British Columbia ($P < 0.001$). Ten percent of eagles ($N = 372$) had tissue Pb levels consistent with Pb poisoning and 5% had elevated, but non-lethal Pb levels. Age class and species differed significantly among eagles with either background or high (Pb-poisoned and elevated Pb) Pb levels ($P \leq 0.01$). When data from both species were combined, high Pb levels were detected in 19.5% of adult and subadult eagles ($N = 220$) but only 7% of immature eagles ($N = 128$). Twenty-eight percent of Golden Eagles ($N = 39$) but only 13% of Bald Eagles ($N = 333$) had high Pb levels. Proportions of eagles with high and background Pb levels were not different based on the level of waterfowl hunting near the locations of collection ($P \geq 0.48$). Golden Eagles with high tissue Pb concentrations tended to be found more often during or soon after the fall hunting season than eagles with background Pb concentrations ($P = 0.08$). The greater incidence of high Pb in Golden Eagles compared to Bald Eagles and the lack of relationship with waterfowl hunting intensity suggest that waterfowl is not the primary source of Pb for eagles in western Canada.

KEY WORDS: *Bald Eagle*; Haliaeetus leucocephalus; *Golden Eagle*; Aquila chrysaetos; *lead*; *lead poisoning*; *pesticide poisoning*; *toxicity*.

[1] E-mail address: mark.wayland@ec.gc.ca

MORTALIDAD, MORBILIDAD, E INTOXICACIÓN POR PLOMO DE ÁGUILAS EN EL OESTE DE CANADÁ, 1986–98

RESUMEN.—Entre 1986 y 1998, investigamos las causas de mortalidad y morbilidad, y evaluamos la contaminación por plomo (Pb) en águilas calvas (*Haliaeetus leucocephalus*) y águilas doradas (*Aquila chrysaetos*) en el oeste de Canadá. El diagnostico mas común fue trauma (13.0%), electrocución (11.5%), envenenamiento por pesticidas (7.6%), heridas por arma de fuego (7.3%), y envenenamiento con plomo Pb (6.4%). No fue posible determinar la causa de mortalidad o morbilidad en 29% de las 546 águilas. Excluyendo los casos indeterminados, la toxicosis dio cuenta de 40% de los diagnósticos en las provincias de la pradera, pero únicamente en 19% de los mismos para British Columbia ($P < 0.001$). Diez por ciento de las águilas (N = 372) tuvo niveles de Pb en los tejidos consistentes con el envenenamiento por Pb y 5% tuvieron niveles elevados pero no letales de plomo. Las clases de edad y las especies difirieron significativamente tanto entre águilas con antecedentes como en aquellas con altos niveles de plomo (envenenadas o con Pb elevado) ($P \leq 0.01$). Cuando los datos de ambas especies se combinaron, los altos niveles de plomo se detectaron en 19.5% de las águilas adultas y subadultas ($N = 220$) y únicamente en 7% de las águilas inmaduras ($N = 128$). Veintiocho por ciento de las águilas doradas ($N = 39$) y únicamente 13% de las águilas calvas ($N = 333$) tuvieron altos niveles de plomo. Las proporciones de águilas con antecedentes y altos niveles de plomo no difirieron con base en el nivel de aves acuáticas cazadas cerca de las localidades de colección ($P \geq 0.48$). Las águilas doradas con alta concentración de plomo en los tejidos se hallaron más frecuentemente durante o un poco después de la estación de caza de otoño que las águilas con antecedentes de concentraciones de plomo ($P = 0.08$). La mayor incidencia de altos niveles de plomo en águilas doradas que en águilas calvas y la carencia de interrelaciones con la intensidad de caza de aves acuáticas, sugiere que las aves acuáticas no son la fuente primaria de plomo para las águilas del occidente de Canadá.

[Traducción de César Márquez]

Lead (Pb) poisoning in waterfowl has long been recognized as an environmental problem in North America (Bellrose 1959). More recently, it has been documented in other avian species (Locke and Friend 1992). Since the 1960s, Pb poisoning has accounted for ca. 1–15% of recorded mortality in Bald Eagles (*Haliaeetus leucocephalus*) and other raptors (Mulhern et al. 1970, Pattee and Hennes 1983, U.S. Fish and Wildlife Service 1986, Pain and Amiard-Triquet 1993, Wayland and Bollinger 1999). The primary source of Pb in poisoned raptors is assumed to be shot or bullets used by hunters (Pattee and Hennes 1983, Scheuhammer and Norris 1995, Kendall et al. 1996). Raptors are exposed to Pb by consuming prey containing Pb shot or bullet fragments (Pattee and Hennes 1983, Pattee et al. 1990, Pain et al. 1993, Harmata and Restani 1995, Pain et al. 1997, Mateo et al. 1999).

While there is a strong association between the ingestion of Pb ammunition from prey tissues and Pb poisoning in raptors, the importance of Pb shot in waterfowl as the main contributing factor is less certain. Pattee and Hennes (1983) hypothesized that Pb shot in waterfowl was the major source of Pb exposure in Bald Eagles; however, Pb ammunition embedded in the tissues of other prey animals may also be important (Kramer and Redig 1997). In the case of Golden Eagles (*Aquila chrysaetos*), upland game animals are believed to be a more important source of Pb shot than are waterfowl (Pattee et al. 1990). It is important to distinguish between waterfowl and other prey as the main source of Pb exposure, because Pb ammunition has been banned in Canada since 1999 for hunting waterfowl, but not for hunting other types of game.

We examined causes of death, illness, and injury in Bald and Golden eagles from western Canada to evaluate Pb poisoning as a cause of mortality and morbidity. In addition, we assessed whether sublethal concentrations of Pb were associated with toxicosis, physical injury, or disease, as sublethal exposure to Pb can predispose birds to other causes of death (Burger 1995). We compared the prevalence of high Pb levels among different age classes of eagles because population levels of some raptors are linked to the survival of adult, reproducing birds (Grier 1980, Newton 1988). To assess whether Pb ammunition is the probable, primary source of Pb exposure in eagles in western Canada, we determined whether the prevalence of elevated Pb levels was highest during and soon after the fall hunting season. Moreover, we examined elevated Pb levels in the context of waterfowl hunting intensity and interspecific dietary differences to test the hypothesis that consumption of waterfowl is a major source of Pb exposure in eagles.

METHODS

Sample Collection. From 1986–98, provincial wildlife agencies and raptor rehabilitation organizations in Manitoba, Saskatchewan, Alberta, and British Columbia (BC) received dead, sick, and injured Bald and Golden eagles. The sex of birds in rehabilitation centers was determined based on morphometric measurements (Bortolotti 1984a, 1984b), and all dead birds were sexed during necropsy. Plumage characteristics were used to estimate age (Bortolotti 1984b, McCollough 1989), and eagles were designated as immature, subadult, or adult. Birds in rehabilitation centers were diagnosed by the staff of these facilities, not necessarily a veterinarian. Dead eagles were frozen until necropsies could be performed by veterinary pathologists.

Blood samples were drawn from live eagles in rehabilitation centers, placed in vials prewashed with nitric acid, and frozen at −20°C until subsequent Pb analysis. In addition, plasma samples from 96 birds in rehabilitation centers in BC were analyzed for cholinesterase activity. Kidneys and livers were removed during necropsy and refrozen at −20°C in glassware prewashed with nitric acid or acetone/hexane. These tissue samples were sent to the National Wildlife Research Centre in Hull, Québec, for Pb analysis.

Brain tissue was collected and frozen for analysis of cholinesterase activity when organophosphate (OP) or carbamate pesticide poisoning was suspected based on evidence found in the field or at the time of necropsy. Such evidence included the presence of insecticide-laced bait or other dead animals at the field site, and copious quantities of meat in the gastrointestinal tract of an eagle carcass in good condition. Diagnoses of pesticide poisoning were made based on this evidence, residue analysis, and cholinesterase activity ≤60% of normal levels for that species (Greig-Smith 1991). Stomach and crop contents of 23 eagles that were suspected to have been poisoned were analyzed for residues of OP and carbamate pesticides known to be used widely in the study area (Elliott et al. 1996, 1997, T. Bollinger unpubl. data). Brain cholinesterase activity was determined in 19% of eagles collected from BC and in 22% of those from the prairies, using methods similar to those described by Martin et al. (1981) and Hill and Fleming (1982).

Lead Analysis. Blood samples (100 µl) were pipetted into 1500 µl Eppendorf micro-centrifuge tubes containing 400 µl of a 0.2% $NH_4H_2PO_4$ plus 0.5% Triton X-100 solution. Each tube was capped and shaken for 10 sec. Pb was determined by graphite furnace atomic absorption spectrometry (GFAAS) using a Perkin-Elmer 3030b spectrophotometer equipped with a HGA-300 graphite furnace and an AS-40 autosampler, according to methods described by Fernandez and Hilligoss (1981). Blood Pb values are expressed as µg Pb/ml blood. Blank solutions were analyzed to verify that Pb was below detection limits. Sample detection limits ranged from 0.005–0.025 µg/ml. Recoveries of spiked samples ranged from 87–110% and averaged 97%. Coefficients of variation of duplicate and triplicate analyses ranged from 7–11% and averaged 9%.

Aliquots of liver and kidney samples were weighed to determine wet weights; placed in plastic, nitric acid-washed test tubes; freeze-dried; and their dry weights recorded. Samples were digested in a solution of deionized water and 70% nitric acid, at a concentration of 0.5 ml solution per 0.1 g dry weight of sample. Pb levels in liver and kidney tissues were expressed on a dry weight basis. Pb was analyzed by flame-atomic absorption spectrometry (AAS) using an atom concentrator tube (ACT-80). The detection limit ranged from 1.0–2.0 µg/g dry weight. Samples in which Pb was not detected using flame-AAS were analyzed by GFAAS as described above. Detection limits using the GFAAS ranged from 0.3–1.0 µg/g dry weight. Recovery of Pb from spiked samples and standard reference materials ranged from 68–118% and averaged 93%. Coefficients of variation ranged from 0–19% and averaged 6%.

Pb concentrations greater than 1.0 µg/ml blood, 30 µg/g dry weight liver, or 20 µg/g dry weight kidney were considered to be diagnostic of Pb poisoning, while concentrations greater than 0.2 µg/ml blood or 6 µg/g dry weight liver or kidney indicated elevated Pb exposure (Pattee et al. 1981, Redig et al. 1984, Reichel et al. 1984, Pain et al. 1994, Franson 1996). When liver and kidney Pb levels were reported in the literature on a wet-weight basis, they were converted to dry weight values using mean tissue moisture values determined in this study to be 76.5% for eagle kidney and 71% for eagle liver. For certain analyses, the Pb-poisoned and elevated Pb categories were combined into a single high Pb group. Pb levels lower than those values stated above were considered to be due to background exposure.

Data Analysis. Causes of death or morbidity were classified into three categories: (1) physical injury, including drowning, electrocution, collision, gunshot, and other trauma; (2) disease and debilitation, including avian cholera, other diseases, emaciation, and degeneration; (3) toxicosis, including pesticide and suspected pesticide poisoning, Pb poisoning, and other poisonings (mercury and strychnine). Two-way contingency table analysis was used to test the relationship between cause of death or morbidity and species. Data from both species were combined in subsequent analyses because no difference was detected among proportions of Bald and Golden eagles within the three diagnostic categories ($\chi^2 = 0.7$, $P = 0.70$, $N = 388$). This was followed by log-linear analysis using the maximum-likelihood technique (PROC CATMOD, SAS Institute 1988) to examine relationships among cause of death or morbidity, region (prairie provinces and BC), and time period. The two time periods examined were May–October, which represented the breeding season, and November–April, which represented autumn migration, wintering, and spring migration. The latter time period included months during and soon after the fall hunting season, which occurs from September–December. The log-linear model included all main effects as well as two-way interactions that included cause of death or morbidity. Separate analysis of the relationship among species and cause of death or morbidity was required because there were too few Golden Eagles ($N = 39$) to include in the multi-way log linear table.

We used analysis of variance of rank-transformed kidney Pb level data to evaluate if Pb exposure at background and sublethal levels predisposed eagles to particular causes of death or morbidity. We used logistic regression with maximum likelihood estimation (PROC CATMOD) to determine if Pb levels were related to age

class, date of recovery, waterfowl hunting intensity, or species. Only main effects were included in the model. In addition, we used two-sample Wilcoxon tests to examine relationships between Pb levels and time of year of recovery, relative to the fall hunting season (September–December). Throughout this study, *P* values less than 0.05 were considered significant.

We examined the geographic association among Pb levels in eagles and waterfowl hunting intensity. To do so, we used harvest survey data (Canadian Wildlife Service unpubl. data) from 1990–95 to estimate mean number of waterfowl-hunting-days/year. Waterfowl-hunting-days data were provided as point estimates that represented 30-min by 30-min blocks of land. Geostatistics, a point surface interpolation technique, was used to derive estimates of waterfowl hunting activity across the study area (GS+™ version 3.11.7, Gamma Design 1999). Geostatistics is an acceptable method for estimating data values for locations that were not sampled directly, by examining data from locations that were sampled. A model of spatial correlation was established and used to interpolate data values at the unknown locations.

The first step of geostatistics is to calculate the sample semivariance and use that to estimate the shape of a curve that represents the semivariance as a function of distance. The second step is to use the estimated semivariance function to determine the weights needed to define the contribution of each sampled point to the interpolation. Sample points close to the point for which an estimated value is to be generated contribute the most to the interpolation. The waterfowl-hunting-days data fit an exponential model (R^2 = 0.98 and residual sums of squares = 0.052), indicating that the variability in hunting activity among locations was a function of distance. Thus, spatial interpolation of the data was appropriate, and waterfowl-hunting-days were interpolated at a resolution of 10 km × 10 km across the study area. Interpolation of the waterfowl-hunting-days data for a specific location was done using nearest-neighbor values weighted by distance and the degree of autocorrelation present for that distance. A maximum of 16 nearest neighbors lying within a 30-km radius of the specific location was used in the interpolations. Low intensity waterfowl hunting areas were designated as those with fewer than 1000 hunting-days/year, and accounted for 87% of the 100 km^2 hunting areas. Hunting activity on high intensity waterfowl hunting areas ranged from 1000–11 178 hunting-days per year.

Eagles with high Pb levels that had been shot (N = 5) were excluded from statistical analyses of Pb contamination because the elevated Pb levels in their tissues may have resulted from embedded Pb shot fragments rather than metabolic uptake of Pb (Wayland et al. 1999). Samples from eagles that had been in rehabilitation centers for >3 wk were not used in analyses because Pb concentrations normally decline to background levels within that time period (Reiser and Temple 1981). Similarly, eagles that received chelating agents to bind Pb were not used in analyses.

RESULTS

Causes of Death, Injury, or Illness. Cause of death, injury, or illness could not be determined for 158 of 546 eagles that were examined. Thirteen percent of eagles were diagnosed with trauma other than electrocution, drowning, collisions, or gunshot wounds; 11.5% were electrocuted; 7.6% were pesticide-poisoned; 7.3% were shot; 6.4% were Pb-poisoned, and 25% died or were debilitated by other causes (Table 1). Eagles poisoned by OP or carbamate insecticides had mean ±SD brain cholinesterase activity of 8.4 ± 6.5 μmol/min/g (N = 22). The corresponding values for eagles diagnosed with other or undetermined causes of death (N = 7) were 17.5 ± 2.8 μmol/min/g. Excluding the cases with undetermined cause of death or date of collection, proportional differences in causes of death or morbidity of eagles (N = 370) were related to region where they were found (P < 0.001; Fig. 1), but not to time of year (P = 0.09). Poisonings and suspected poisonings accounted for 40% of diagnoses of eagles from the prairie provinces, but only 19% of those from BC. In contrast, 45% of eagles from the prairies and 66% of those from BC were injured.

Table 1. Final diagnoses for 546 dead and debilitated Bald and Golden eagles in western Canada, 1986–98

	British Columbia		Prairie Provinces		
Diagnosis	Bald Eagle	Golden Eagle	Bald Eagle	Golden Eagle	Total
Degeneration or emaciation	21	3	5	3	32
Avian cholera	0	0	6	0	6
Other infectious disease	14	0	8	2	24
Pesticide poisoning	20	0	19	3	42
Lead poisoning	19	1	11	4	35
Other poisoning	2	0	3	0	5
Suspected poisoning[a]	4	0	12	2	18
Collision	34	0	1	2	37
Drowning	15	0	0	0	15
Electrocution	50	0	4	9	63
Gunshot	25	0	14	1	40
Other trauma[b]	33	1	29	8	71
Undetermined	123	4	22	9	158

[a] Poisoning suspected because of additional evidence.

[b] Category includes wing injuries, attacks by other eagles, leg injuries, trap-related injuries, unknown origin, soaked feathers, internal injuries, asphyxiation, spinal injuries, fall from nest, and exhaustion.

Lead Exposure and Poisoning. Pb levels were de-

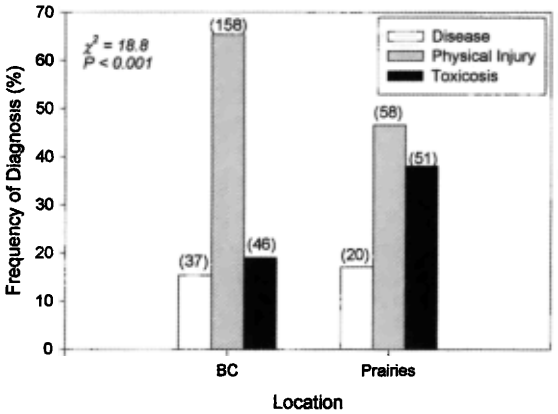

Figure 1. Percent frequency of three categories of diagnosis of death and morbidity, according to the region where eagles were found. Unknown causes of death and morbidity have been excluded. Utilization of a log-linear model showed that cause of death or morbidity was influenced by region. Numbers in parentheses indicate sample sizes.

termined in kidney, liver, or blood samples from 372 eagles (Table 2). Most eagles (85%) had background levels of Pb and 15% had high Pb levels in their tissues. The high Pb group included eagles with Pb levels consistent with poisoning (10%) and those with elevated Pb levels (5%).

Bald Eagles with toxicoses other than Pb poisoning had higher Pb levels than those diagnosed with various physical injuries, disease, or debilitation ($P = 0.037$). For Bald Eagles, median values for kidney lead levels in three categories of diagnosis were as follows: toxicoses other than lead poisoning—1.09 μg/g ($N = 29$); physical injuries—0.27 μg/g ($N = 139$); and disease and debilitation—0.26 μg/g ($N = 37$). There was no difference in kidney Pb levels among groups of Golden Eagles ($P = 0.614$). Median values for kidney Pb levels in Golden Eagles with toxicoses other than Pb poisoning ($N = 4$), physical injuries ($N = 16$), and diseases or debilitation ($N = 6$), were 0.77, 0.74, and 1.27 μg/g, respectively.

Age class and species differentiated between eagles with background or high Pb levels ($P \leq 0.01$). Adult and subadult eagles had a higher percentage with high Pb levels than did immature eagles. This difference was evident for Bald Eagles ($P = 0.005$) and when data from both species were combined ($P = 0.002$). High Pb levels were detected in 17.6% of adult and subadult Bald Eagles, but in only 5.9% of immature Bald Eagles. When data from both species were combined, 19.5% of adult and subadult eagles, and 7.0% of immature eagles had high Pb levels. In Golden Eagles, the percent of adult and subadult birds with high Pb (36.3%) was not different from the percent of immature eagles with high Pb (20.0%, $P = 0.58$). In comparison with Bald Eagles, a higher percentage of Golden Eagles had high Pb levels. The percent of Bald Eagles with high Pb levels in the immature and subadult/adult age classes were 5.9% and 17.6%, respectively. The percent of Golden Eagles with high Pb levels in these age classes were 20.0% and 36.3%, respectively.

Date of recovery and waterfowl hunting intensity failed to provide additional discrimination between the background and high Pb groups ($P \geq 0.19$). However, Golden Eagles with high Pb levels tended to be found during or soon after the fall hunting season (Fig. 2), while those with background Pb levels were more often found several months later. This seasonal difference in date of recovery approached significance (Wilcoxon two-sample test, $P = 0.08$). Date of recovery of Bald Eagles, as related to hunting season, did not differ

Table 2. Pb levels in tissues from 372 immature, subadult, adult, and unknown age Bald and Golden eagles collected from western Canada, 1986–98.

	Bald Eagle			Golden Eagle			
Pb Level	Immature	Subadult and Adult	Unknown	Immature	Subadult and Adult	Unknown	Total
Background[a]	111	163	15	8	14	6	317
Elevated[b]	1	11	1	1	4	0	18
Poisoned[c]	6	24	1	1	4	1	37

[a] Pb <6 μg/g dry weight kidney or liver; <0.2 μg/ml blood.
[b] Pb 6–20 μg/g dry weight kidney; 6–30 μg/g dry weight liver; 0.2–1.0 μg/ml blood.
[c] Pb >20 μg/g dry weight kidney; >30 μg/g dry weight liver; >1.0 μg/ml blood.

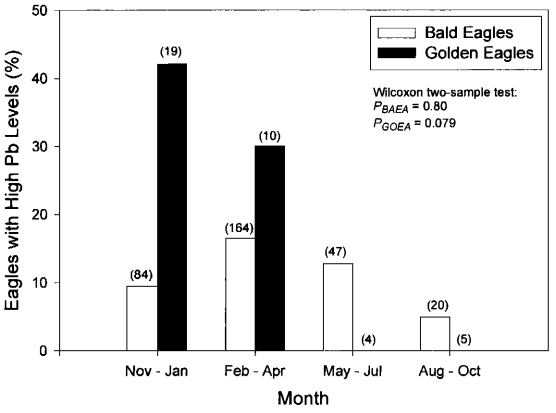

Figure 2. Temporal changes in the percent of Bald and Golden eagles with high Pb levels, in relation to the fall hunting season (September–December). High Pb levels were defined as >0.2 μg/ml blood or >6 μg/g dry weight liver or kidney tissue. Numbers in parentheses indicate sample sizes.

between those with background and high Pb levels ($P = 0.8$). Waterfowl hunting intensity did not affect the percent of Bald or Golden eagles with high Pb levels ($P \geq 0.48$). In areas of low intensity waterfowl hunting, 12.8% of Bald Eagles and 32.0% of Golden Eagles had high Pb levels. High Pb levels were evident in 14.1% of Bald Eagles and 21.4% of Golden Eagles recovered from high intensity waterfowl hunting areas.

Median and quartile Pb levels in kidneys of five eagles with Pb shot in their gastrointestinal tracts were 23.1 μg/g and 20.8–76.8 μg/g, respectively, and were higher than in 329 eagles without shot in their gastrointestinal tracts (0.4 μg/g, 0.08–1.4 μg/g, Wilcoxon two-sample test: $P < 0.001$).

DISCUSSION

Causes of Death, Injury, or Illness. A sampling bias existed in this study because of the way in which eagles were found and reported. Thus, relative importance of each cause of death or morbidity may not be representative of the actual proportion at the population level. Birds affected by starvation and disease may be reclusive and susceptible to depredation (Wobeser 1994, 1997), thus, decreasing the probability of encounter by humans. Many eagles during this study were found in southwestern BC near major population centers, but few were obtained from northern breeding areas where human populations are sparse.

Prevalence of Pb poisoning, electrocution, trauma, emaciation, and disease as reported in this study in western Canada were similar to those previously reported for the United States (Table 3). The present study found that Pb poisoning accounted for 6% of diagnoses, a value that is in agreement with previous reports. Interestingly, we found that carbamate and OP insecticide poisonings in western Canada accounted for a much higher percent of cases than had been reported in the United States. Gunshot and trapping diagnoses accounted for a smaller percent of cases in western Canada than in the United States. Overall, the proportion of human-induced mortality and morbidity in this study (44% of all cases and 62% of cases with known diagnoses) was similar to that in other studies (Reichel et al. 1984, National Wildlife Health Laboratory 1985, Franson et al. 1995, Harmata et al. 1999). Results suggest humans are directly responsible for a large proportion of eagle deaths, but it is uncertain whether this is impacting eagle populations. There is no evidence that populations of either Bald or Golden eagles had de-

Table 3. Causes of death or morbidity (percent of all cases) reported in eagles in the United States and in western Canada.

DIAGNOSIS	U.S.A. 1963–84[a]	U.S.A. 1960s–90s[b]	U.S.A. 1978–81[c]	WESTERN CANADA 1986–98[d]
Trauma	21	23[e] 27[f]	20	22
Pb poisoning	6	8	6	6
OP and carbamate poisonings	0.5	~3	0	8
Other poisonings	5	U[g]	1	1
Electrocution	9	12[e] 25[f]	15	11
Gunshot	23	15	19	7.6
Trapping	5	U	6	1
Emaciation	8	U	11	6
Disease	2	U	6	5 5
Undetermined	18	U	6	29

[a] N = 1429 Bald Eagles (National Wildlife Health Laboratory 1985).
[b] N ~ 4300 Bald and Golden eagles (Franson et al. 1995).
[c] N = 293 Bald Eagles (Reichel et al. 1984).
[d] N = 546 Bald and Golden eagles (this study).
[e] Bald Eagles.
[f] Golden Eagles.
[g] Unknown.

clined in western Canada through to the early 1980s (Gerrard 1983, Kirk 1996). Although no recent data are available, remedial measures to reduce the numbers of eagles dying from human-related causes may not be necessary for the conservation of eagle populations.

In this study, eagles from the prairie provinces were poisoned by insecticides more often than those from BC. This regional variation was likely due to differences in the manner in which carbamate and OP insecticides were used. Insecticide poisoning in BC likely resulted from the appropriate use of granular carbamate and OP insecticides (Elliott et al. 1996, 1997). Furthermore, insecticide poisoning in BC may have been reduced in the latter years of this study by successful efforts that were undertaken to remove the most toxic products from the market or to at least reduce their use. In contrast, insecticide poisonings in the prairie provinces have been attributed to the illegal use of carbamate and OP insecticides in baits intended to kill coyotes (*Canis latrans*; Bollinger and Mineau 1995). Twelve percent (22/177) of the eagles collected from the prairie provinces were diagnosed with pesticide poisoning. Assuming that these cases all resulted from efforts to bait and kill coyotes, the effect of this activity is greater in the prairie provinces than has been documented for North America as a whole (5%, Bortolotti 1984c). The use of poisons to kill predators of livestock continues to be a problem for eagles in the prairie provinces.

Lead Exposure and Poisoning. Although 6% of the eagles in this study were initially diagnosed as Pb-poisoned, subsequent tissue analysis showed that 10% of 372 eagles had concentrations indicative of Pb poisoning. The principal reason for this difference is that Pb poisoning cannot be properly determined without analyzing tissues for Pb. Thirteen percent of the eagles from the prairie provinces, for which post-mortem examination did not identify a cause of death, had tissue Pb levels indicative of Pb poisoning. Cause of death or morbidity was undetermined for 158 eagles in this study, and not all birds were analyzed for Pb. Thus, diagnosis without tissue analysis may have underrepresented Pb poisoning as a mortality factor. The 10% estimate based on only those eagles for which Pb levels were determined, may be more accurate.

At concentrations below those known to cause mortality, Pb is immunotoxic and neurotoxic, can cause behavioral deficits, and impair digestion and feeding (Burger 1995). Thus, elevated concentrations of Pb in birds may increase their susceptibility to diseases and accidents, and impair their ability to hunt, obtain food, and digest food. We did not find evidence to support an association between elevated Pb levels and the prevalence of disease, emaciation, or physical injury in eagles. Similarly, the known causes of raptor mortality in Great Britain were generally unrelated to elevated Pb exposure (Pain et al. 1994).

Bald Eagles that were poisoned by other toxic agents, principally OP and carbamate insecticides, had higher kidney Pb concentrations than diseased, emaciated, or injured birds. Scheuhammer and Wilson (1990) reported that various cholinesterase-inhibiting insecticides in combination with Pb did not have an additive effect on d-aminolevulinic acid dehydratase (ALA-d) inhibition when compared with the effects of Pb alone. The ALA-d enzyme is sensitive to Pb and serves as an excellent biomarker of Pb exposure (Scheuhammer and Wilson 1990). In this study, many insecticide-poisoned Bald Eagles were found in areas where Pb exposure was also comparatively high. The association between tissue Pb levels and the prevalence of poisonings by other toxic agents probably reflects a greater possibility that birds from these areas will be exposed through their diets to both Pb and insecticides. We doubt that sublethal Pb exposure increases the susceptibility of eagles to insecticides.

In this study, high Pb levels were found in a greater proportion of adult and subadult eagles than immature eagles. In the United States, adult females comprised 47% of all Pb-poisoned Bald Eagles, but only 25% of the continental population, indicating a particular sensitivity of this age-sex group (U.S. Fish and Wildlife Service 1986). Frequent exposure to Pb may result in age-related increases in Pb concentrations in soft tissue (Pain et al. 1994), and eagles in western Canada may be exposed to Pb frequently enough to result in age-related Pb accumulation. This may be an important issue from the population perspective, because eagles are long-lived and have a low annual reproductive potential, so population levels are linked to the survival of reproducing adults (Grier 1980, Newton 1988).

Eagles with Pb shot in their gastrointestinal tracts had much higher Pb levels than those without shot in their gastrointestinal tracts. This suggests that Pb ammunition is an important source of Pb exposure in eagles in western Canada. Although the majority of Pb-poisoned eagles did not

Table 4. Summary of North American studies showing the prevalence of eagles with high Pb and Pb-poisoned tissue concentrations.

SPECIES	LOCATION	TISSUE ANALYZED (*N*)	HIGH PB[a] (PERCENT)	PB-POISONED[b] (PERCENT)	REFERENCE
Bald Eagle	Idaho	Liver (5)	83	83	Craig et al. 1990
	Montana	Blood (37)	86	5	Harmata and Restani 1995
	Minnesota	Blood (25)	96	28	Hennes 1985
	Montana & Saskatchewan	Blood (178)	19	1	Miller et al. 1998
	Western USA	Blood (120)	3[c]	NA[d]	Wiemeyer et al. 1989
	Western Canada	Blood, liver, kidney (333)	13	9	This study
Golden Eagle	Idaho	Liver (16)	56	44	Craig et al. 1990
	Idaho	Blood (178)	42	NA	Craig and Craig 1995
	Montana	Blood (86)	56	2	Harmata and Restani 1995
	California	Blood (162)	36	2	Pattee et al. 1990
	Western Canada	Blood, liver, kidney (39)	28	15	This study

[a] Pb >6 μg/g dry weight kidney or liver; >0.2 μg/ml blood.
[b] Pb >20 μg/g dry weight kidney; >30 μg/g dry weight liver; >1.0 μg/ml blood.
[c] Pb >0.4 μg/ml blood.
[d] NA—not available.

have Pb shot or fragments in their digestive tracts, there may have been ample time for regurgitation (Pattee et al. 1981) or complete digestion of the Pb fragments. Although the difference was not statistically significant, Golden Eagles with high Pb levels tended to be found during or soon after the fall hunting season, whereas those with background Pb levels were found more often long after hunting seasons had ended. Similar results have been reported for Golden Eagles in California (Pattee et al. 1990), Bald Eagles in the United States (Pattee and Hennes 1983, Wiemeyer et al. 1989) and BC (Elliott et al. 1992), and Western Marsh-Harrier (*Circus aeruginosus*) in Europe (Pain et al. 1993, 1997, Mateo et al. 1999). In our study, there was no relationship between Pb levels and the time of year when Bald Eagles were found. However, some Bald Eagles found in late winter or early spring may have died at the onset of winter, but were frozen and buried by snow for several months before being discovered. Furthermore, eagles ingesting Pb ammunition during or shortly after the hunting season may not have died until several weeks later (Pattee et al. 1981).

We tested the hypothesis that consumption of waterfowl is a major source of Pb exposure in eagles (Pattee and Hennes 1983) by examining elevated Pb levels in the context of waterfowl hunting intensity and interspecific dietary differences. We found no geographic evidence linking high Pb levels in eagles to the use of Pb shot for waterfowl hunting, suggesting that waterfowl is not their primary source of Pb. Alternatively, this lack of correlation may have resulted from eagles flying long distances in short time periods (McClelland et al. 1994, Brodeur et al. 1996) after ingesting Pb shot. Eagles may have consumed waterfowl and associated Pb shot in areas of high waterfowl hunting intensity, then flown to areas of low waterfowl hunting intensity before dying of Pb poisoning. In addition, the gradual phasing out of lead shot for waterfowl hunting in southwestern BC, beginning in 1998, may have reduced the likelyhood that lead shot-contaminated waterfowl would have been an important source of lead to Bald Eagles, many of which were recovered in that area during this study.

Bald and Golden eagles are opportunistic foragers that eat a wide variety of prey, but in North America, Bald Eagles feed mainly on fish and waterfowl (Stalmaster and Plettner 1992, Miller et al. 1998, Restani et al. 2000), while Golden Eagles

feed mainly on mammals and upland game birds (Olendorff 1976). If waterfowl were the primary source of Pb for eagles, one would expect to find a higher percentage of Bald Eagles with high Pb levels than Golden Eagles. In fact, this has been reported in several studies from the western United States (Table 4). In contrast, we found high Pb levels in 13% of Bald Eagles and 28% of Golden Eagles in western Canada. Kramer and Redig (1997) suggested that Pb poisoning in eagles in the north-central United States resulted mainly from their scavenging small mammals and large game. Pb shot and bullet fragments in game birds, small mammals, and large game carcasses constituted the major source of Pb for Golden Eagles (Pattee et al. 1990, Gjershaug 1992, Bezzel and Fünfstuck 1995, Harmata and Restani 1995, Kendall et al. 1996). Moreover, Pb-poisoned Bald Eagles in the Greater Yellowstone Ecosystem had ingested large caliber bullets while feeding on ungulate carcasses (Harmata et al. 1999). Our results, as well as those from the studies mentioned above, suggest that waterfowl is not the primary source of Pb for eagles in large areas of western North America. Banning the use of Pb shot for waterfowl hunting may not significantly reduce Pb poisoning in eagles, and the use of Pb ammunition for hunting mammals and upland game birds may continue to pose a risk to eagles and other raptor species in Canada.

In conclusion, we found that various physical injuries, electrocution, pesticide poisoning, gunshot wounds, and Pb poisoning were the most common diagnoses in Bald and Golden eagles in western Canada. Further monitoring of eagle mortality and populations are required to assess the population level impacts of these mortality factors. Although high Pb levels were associated with other toxicoses, there was no evidence to suggest that sublethal effects of Pb predisposed eagles to injury or disease. In comparison with immature eagles, a higher percent of adult and subadult eagles had high Pb levels. This difference among age classes may also have repercussions at the population level. We found no significant association between Pb levels and the fall hunting season, but we suspect that eagles in western Canada were exposed incidentally to Pb ammunition through the consumption of various prey species. Waterfowl appeared not to be the primary source of Pb for eagles in western Canada, and we suspect that Bald and Golden eagles will continue to be Pb-poisoned despite the ban on the use of Pb shot for waterfowl hunting.

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