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Environmental Contaminants and Cholinesterase in Blood of Vernal Migrant Bald and Golden Eagles in Montana

Abstract

Environmental contaminants typically have the greatest impact on upper trophic level predators such as eagles. We collected whole blood and plasma from 123 migrant bald (Haliaeetus leucocephalus) *and golden eagles* (Aquila chrysaetos) *captured between 1985 and 1993 in west-central Montana for analysis of lead (Pb), mercury (Hg), selenium (Se), and organochlorine compounds, and to determine baseline values of cholinesterases (ChE) in free ranging, apparently healthy eagle populations. Elevated concentrations ($\bar{x}$ = 0.32 ppm) of Pb were detected in 97 percent of bald eagles. Pb was detected in 85 percent of golden eagles but at lower ($P<0.05$) concentrations ($\bar{x}$ = 0.18 ppm) than bald eagles. The source of Pb in both species was most likely Pb shot in waterfowl and Pb projectile fragments in ground squirrels* (Spermophilus *spp.). Hg was detected in 94 percent of bald eagles but only 22 percent of golden eagles. Sources of Hg were most likely fish for bald eagles and fish or gulls* (Larus *spp.) for golden eagles. Se was detected in 94 percent of bald eagles and in 88 percent of golden eagles. Bald eagles had higher ($P<0.05$) Se concentrations (x = 0.55 ppm) than golden eagles ($\bar{x}$ = 0.31 ppm). Aquatic oriented prey were the probable sources of Se for both species of eagles. Heavy metal and Se concentrations were not correlated with age or sex in either species. DDE was detected more often in bald eagles (67%) than golden eagles (48%). Organochlorine concentrations were very low and not related to age or sex for either species. Total ChE activity was higher ($P=0.0001$) in golden eagles than bald eagles. Mean acetylcholinesterase activity was lower ($P< 0.05$) in bald than golden eagles ($\bar{x}$ = 171 vs $\bar{x}$ = 296), but percent butyrylcholinesterase was higher in bald eagles. Despite fairly pervasive incidence and occasionally high concentrations of potentially toxic contaminants, populations from which both eagle species originated appear stable or increasing.*

Key words: acetylcholinesterase, bald eagle, butyrylcholinesterase, environmental contaminants, golden eagle, heavy metals, Montana, organochlorines, pesticides, selenium.

Introduction

The impact of environmental contaminants on wildlife has been well documented (Hall 1987, Porter 1993, Hoffman et al. 1995). Upper trophic level predators such as raptors may be more affected by some environmental contaminants than lower trophic level species due to biomagnification and species sensitivity. Contaminant induced mortality, reduced reproduction, and sublethal morbidity have been recorded for bald and golden eagles and remain management concerns. Moreover, habitat and prey preferences of eagles may increase their risk of exposure to environmental contaminants because both species exploit habitats and prey known to biomagnify contaminants (reviews by Newton 1979, Stalmaster 1987, Palmer 1988). Currently, the extent of adverse

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impacts on eagles from contaminants in the western U.S. is unknown (Henny and Anthony 1989), thus effects on the health of populations cannot be evaluated.

Most analysis of heavy metal and organochlorine contaminant exposure to raptorial birds has been conducted on tissues of dead birds (Reichel et al. 1984) or eggs (Wiemeyer et al. 1993). Analysis of these contaminants in free-ranging eagles may be a more representative indicator of exposure to populations than analysis of tissue of dead birds.

Detecting and evaluating extent of exposure to contaminants by use of biological markers has gained prominence recently (Fossi et al. 1992). One such nondestructive marker most frequently used for determining exposure to organophosporus and carbamate pesticides is measurement of cholinesterase (ChE) activity in plasma (Ludke et al. 1975). Depressed butyrylcholinesterase (BChE) in plasma is more indicative of recent exposure to anticholinergic pesticides than brain acetylcholinesterase (AChE) activity (Hill and Fleming 1982, Hill 1989). Only one published account providing ChE activity data in bald eagle plasma exists (Dieter and Wiemeyer 1978) and only one for golden eagle nestlings (Taira and McEwen, In press). Our objectives were to determine the incidence and severity of heavy metal, Se, and organochlorine contamination in vernal migrant bald and golden eagles and develop baseline data on ChE activity for each species.

Study Area and Methods

Each spring, a significant portion of the continental population of bald and golden eagles pass through and use habitats in Montana during vernal migrations (Swenson et al. 1981, Meyer 1992, Tilly 1995). An eagle migration corridor exists in west-central Montana near the towns of Wilsall, Park County and Ringling, Meager County where eagles concentrate each spring to feed on recently emerged Richardson's ground squirrels (*Spermophilus richardsonii*), white-tailed jackrabbits (*Lepus townsendii*), and domestic and wild ungulate carcasses. This phenomenon presents a unique opportunity to sample a large portion of the continental eagle population for exposure to environmental contaminants.

Eagles were captured during migration through west-central Montana (vicinity 41° 10', 110° 40') between 1 March and 15 April 1985-1993. We used the modified "Lockhart method" (Harmata 1985) with avian or mammalian carcasses as baits (mostly Richardson's ground squirrels) and a radio controlled bownet (Jackman et al. 1994) around large carcasses to capture migrant eagles. A live bald or golden eagle was used at most capture sites as a lure.

Mensural data were acquired for all eagles captured and all were banded with U.S. Fish and Wildlife Service (USFWS) pop-rivet leg bands. We evaluated residency time of eagles in the study area by radio-tagging and wing notching some eagles. Radio transmitters were attached to center tail feathers of 4 bald eagles and 2 golden eagles in 1987 and 1 bald eagle and 2 golden eagles in 1992. Wing notching consisted of an ≈6 x 6 cm portion of vanes of adjacent secondary feathers removed mid-shaft on most eagles captured. The wing notch appeared as a small but distinct hole in the wing and aided in visual identification when in flight.

Bald eagles were aged by plumage characteristics presented in McCollough (1989) and Harmata (1984). Sex of bald eagles was assigned based on size using mensural data and the formulas proposed by Garcelon et al. (1985). Age class (adult/immature) of golden eagles was determined based on amount of white and number of gray bars in tail feathers. Formulas generated by

discriminate analysis of mensural data from 74 golden eagles were used to assign sex. A FORTRAN program incorporating the *a priori* age class variable and the 5 most discriminating variables for classifying sex (Appendix A) correctly classified all known sex eagles (n=12; Harmata, unpubl. data).

We collected 3-8 cc's of blood per eagle which was deposited in sodium heparinized vacutainers. Three cc of whole blood per eagle was sequestered for heavy metal and Se analysis. If available, an additional 2-5 cc of whole blood per eagle was centrifuged to separate plasma which was pipetted off and separated into 2 equal samples. Whole blood and plasma were frozen and stored at -22° C within 1 hr. Whole blood was analyzed for Pb, Hg, and Se at Montana State University (MSU) Chemical- Analytical Laboratory. Samples for Pb analysis were digested with nitric acid and concentrations determined by Graphic Furnace Additions (GFAAS). Samples for Hg analysis were digested by nitric acid in closed vessels and analyzed by Cold Vapor Generation using $SnCl_2$ reductant Atomic Absorption Spectroscopy (Verwolf 1988). Samples for Se analysis were digested with nitric acid and concentrations determined using Palladium Matrix Modifier and GFAAS. Recovery of all 3 elements from spiked samples was >90 percent. One plasma sample (0.5 to 2 cc) from each of 18 bald and 29 golden eagles was analyzed for 16 organochlorine compounds at MSU Chemical Analytical Laboratory using methods recommended by U.S. Environmental Protection Agency (EPA)(see Thompson 1974). All results are reported on a wet weight basis. Detection limits of heavy metals, Se, and organochlorine compounds are shown in Table 1. Plasma samples (0.5-2 cc) from 15 bald and 19 golden eagles were analyzed for total ChE and AChE activity at EPA Environmental Research Laboratory, Corvallis, OR. Plasma BChE was the difference between total ChE and AChE. Not all samples were frozen or analyzed at equal intervals from collection. Some thawed briefly and were refrozen and some stored at different temperatures. All may have affected reactivity of ChE (Fairbrother et al. 1991). Because of chronic lack of funds, duplicate samples were not analyzed for any constituent.

Table 1. *Detection limits of heavy metals and selenium (ppm wet weight) analyzed in whole blood and organochlorines (ppb wet weight) analyzed in plasma of vernal migrant eagles in west-central Montana, 1985-1993.*

Compound	Detection Limit	Compound	Detection Limit
Heavy metals(ppm)			
Lead	0.06	Mercury	0.03
Selenium	0.10		
Organochlorines (ppb)			
Mirex	19	Aldrin	3
Methoxychlor	35	Heptachlor	2
p,p- DDT	13	Lindane	3
p,p- DDD	9	∂ BHC	2
p,p- DDE	3	ß-BHC	4
Endrin	4	a-BHC	1
Dieldrin	5	Hexachlorobenzene	1
Heptachlor Epoxide	4	Oxychlordane	3

Eagles were grouped by species, age class (immature/adult), and sex for comparisons of mean contaminant concentrations. Distributions of concentrations for all contaminants were skewed; therefore data were log transformed and geometric means computed for comparisons. Means tests were performed among groups only when ≥50 percent of samples from both groups exhibited detectable concentrations of contaminant. Samples below detection limits were assigned values equal to one half the detection limit for the respective contaminant (Wiemeyer et al. 1989). Chi-square analysis was used to compare frequency of occurrence among groups when >50 percent of samples in one group were below the detection limit. Arithmetic means were used for ChE comparisons

and analyses were performed on nontransformed data. Years with <4 samples (1985, 1986, 1989) for a given species were not included in analysis of annual differences in contaminants. SOLO, a statistical software package for personal computers (BMDP, 1440 Sepulveda, Los Angeles, CA 90025) was used for chi-square, ANOVA, and t-tests. Statistical significance was assigned at $P \leq 0.05$, but P values >0.05 were presented when differences were considered biologically significant. BMDP Neuman-Keuls Range tests identified differences detected by ANOVA. Contaminant concentrations are reported in parts per million (ppm) wet weight. ChE activity is expressed in µmoles of acetylthiocholine iodide substrate hydrolized/min/L of plasma (µmoles/min/L).

Results

We collected blood samples from 123 of 156 eagles captured between 1985 and 1993: 37 bald eagles and 86 golden eagles. Adults (>3years old) comprised 49 percent of bald eagles sampled and 58 percent of golden eagles sampled. Number of samples analyzed varied from year to year (range 3-30).

Only 1 of 4 radio-tagged golden eagles was relocated more than 1 day post capture; most left the study area within hours of release. Two of 5 radio-tagged bald eagles were detected within the study area for 3 days. No eagles with wing notches were observed >1 hr. post release.

Heavy metals and SE were detected in both species (Table 2). Blood Pb values were grouped according to 4 exposure criteria proposed by Redig (1984); <0.20 ppm = background; ≥0.20 ppm to 0.59 ppm = exposed; 0.60 ppm to 0.99 ppm = clinically affected; ≥1.00 ppm = acute Pb poisoning (Table 3). A higher proportion of bald eagles (86%) were exposed to elevated (≥0.20 ppm) environmental Pb than golden eagles (56%)(x^2=2.13, P=0.145, df=1). Bald eagles had higher Pb and Se concentrations than golden eagles (t=3.52, P=0.001; t=10.99, P=0.002, respectively). A significant difference in frequency of occurrence of detectable Hg was found between species (x^2=15.75, P=0.0001, df=1), with bald eagles exhibiting much greater exposure than golden eagles (Table 2). No differences in frequency of occurrence of detectable contaminants in blood were found across sex or age class within species (all analyses, x^2<0.42, P>0.5, 1 df).

Pb concentrations in bald eagles declined over time from 1987 ($\bar{x}$ = 0.53) to 1992 ($\bar{x}$ = 0.35), and yearly differences may have been biologically significant (F=2.32, P=0.101); no differences among years occurred for Hg concentrations. Bald eagles had higher Se concentrations in 1991 ($\bar{x}$ = 0.81) and 1992 (0.94) than in 1987 (0.42) or 1990 (0.16) (F=14.58, P<0.001). Differences in Pb and Se concentrations in golden eagles occurred among years (F=6.42, P=0.001; F=4.91, P<0.001, respectively). Frequency of occurrence of Hg in blood of golden eagles also was different among years (x^2=12.95, P=0.011, df=4), ranging from 100 percent exposed in 1991 (n=6) to none exposed in 1989 (n=1) and 1990 (n=16), but a consistent trend for either heavy metal or Se over time was not evident. There was no correlation of Se with Hg in either bald eagles (r=-0.14, P=0.43) or golden eagles (r=0.14, P=0.27).

Table 2. *Heavy metal and selenium concentrations (ppm wet weight) and frequency of occurrence in blood of vernal migrant eagles in west-central Montana, 1985-1992.*

	Pb				Hg				Se			
	Mean	Maximum Level[1]	Percent Detected	n	Mean	Maximum Level[1]	Percent Detected	n	Mean	Maximum Level[1]	Percent Detected	n
Bald eagle	0.32	1.10	97	37	0.54	1.70	94	34	0.55	2.80	94	34
Golden eagle	0.18	1.30	85	86	-[2]	1.00	22	77	0.31	1.70	88	77

[1]Minimum level was below detection limit.

[2]Greater than 50 percent of sample below detection limit.

Table 3. *Lead exposure levels in blood of vernal migrant eagles captured in west-central Montana, 1985-1992.*

Exposure (ppm)	Number Detected (%) Bald Eagles		Golden Eagles	
<0.20	5	(13.5)	38	(44.2)
0.20-0.59	27	(73.0)	37	(43.0)
0.60-0.99	3	(8.1)	9	(10.5)
≥1.00	2	(5.4)	2	(2.3)
	37	(100.0)	86	(100.0)

Only 6 of 16 scanned organochlorine compounds were detected in blood of migrant eagles, but at very low concentrations (Table 4). Frequency of occurrence of DDE and DDD was no different in bald eagles than golden eagles (x^2=0.43, P=0.51, df=1; x^2=0.03, P=0.86, df=1, respectively). No differences between sex or age classes within species were found for any organochlorine compound detected (all analyses, x^2<0.101, P>0.75, df=1).

Table 4. *Frequency of occurrence and maximum concentrations (ppm wet weight) of organoclorine compounds detected in plasma of vernal migrant eagles in west-central Montana, 1990-1993.*

Compound	Bald Eagle (n=18) Percent Detected	Maximum[1] Level[1]	Golden Eagle (n=29) Percent Detected	Maximum Level
p, p'- DDE	67[2]	0.087	48	0.021
p, p'- DDD	6	0.010	7	0.009
Dieldrin	11	0.007	-	-
Hexachlorobenzene	22	0.001	-	-
Heptachlor Epoxide	-	-	10	0.039
Oxychlordane	-	-	3	0.010

[1]Minimum level was below detection limit.

[2]Mean concentration was 0.007 ppm.

ChE in plasma was analyzed in blood of 34 eagles captured in 1990 and 1991 (Table 5). Differences in total ChE activity approached significance between sexes of golden eagles (t=2.09, P=0.069) and bald eagles (t=1.80, P=0.09) and age classes of golden eagles (t=1.90, 0.074). Bald eagles had lower total ChE activity (t=4.52, P=0.0001), AChE activity (t=2.43, P=0.025), and BChE (t=3.77, P=0.011) than golden eagles but higher percentages of BChE (Fig. 1). Four bald eagles and 5 golden eagles had BChE:ChE ratios ≥80 percent. Of those groups, 2 bald eagles (13.3% of total) and 2 golden eagles (10.5% of total) had total ChE activity values >2 SE below the sample mean.

Table 5. *Cholinesterase (ChE) activity (μmoles/min/L) in plasma of vernal migrant eagles captured in west-central Montana, 1990 and 1991.*

Species (n) Age/Sex (n)	Total ChE $\bar{x}$	SE	AChE[1] $\bar{x}$	SE	BChE[2] $\bar{x}$	SE
Bald Eagle (15)	691	32	171	15	520	28
Immature (8)	707	41	157	21	549	42
Adult (7)	673	55	187	23	486	34
Male (7)	631	31	149	25	481	33
Female (8)	744	49	190	17	553	42
Golden Eagle (19)	1033	61	296	31	736	67
Immature (9)	917	63	283	36	633	48
Adult (10)	1137	93	307	50	830	116
Male (7)	1209	124	324	51	885	161
Female (12)	929	48	279	39	650	41

[1]Acetylcholinesterase

[2]Butyrylcholinesterase

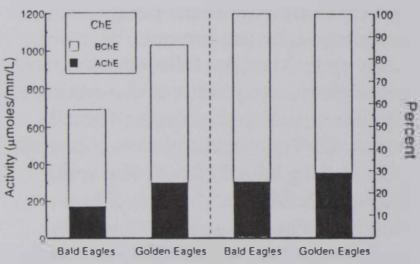

Figure 1. *Acetyl- (AChE) and butyryl-(BChE) cholinesterase activity and proportion of total cholinesterase (ChE) activity in plasma of vernal migrant bald (n=15) and golden eagles (n=19) captured in west-central Montana, 1990 and 1991.*

Discussion

Heavy Metals and Selenium

Pb apparently continues to contaminate a large portion of the Continental eagle population in spring. In Montana, a greater proportion of

vernal migrant bald eagles (97%:this study) was contaminated with Pb than autumn migrants (41.4%, Table 1, Wiemeyer et al. 1989). Proportion of golden eagles exposed to Pb in Montana (86%:this study) was less than that reported for those in California (94.6%:Pattee et al. 1990), but detection limits were lower (0.01 ppm) in California than this study and may influence interpretation.

Tissue-bound Pb has been shown not to contribute to contamination at higher trophic levels (Custer 1984), so the source of contamination in vernal migrant eagles appears to be Pb shot, projectiles, or fragments in food items. Mortality and morbidity from Pb poisoning has been recorded in bald and golden eagles (Pattee et al. 1981, Redig et al. 1983, Reichel et al. 1984, Gill and Langelier 1994). In virtually all cases, the source of Pb was shot or projectiles. In western Montana, shooting is commonly used for population control of ground squirrels and "plinking gophers" is popular recreation. Both activities are intense in spring when migrant eagles are present. We have seen up to 30 eagles following shooters from pasture to pasture and some eagles appear to cue on the report of small arms. Of 32 ground squirrels shot with .22 cal. long rifle, hollow point (HP) bullets, 18 (56%) had visible Pb fragments in radiographs of whole carcasses, as did 6 of 8 (75%) shot with .22 cal. magnum HP loads (Harmata, unpubl. data). Fragments as large in profile as #7 1/2 shot were present in all squirrels with fragments and many had fragments as large as #2 shot. Four squirrels shot with magnum loads had fragments at least 4 x 5 mm, with some as large as 6 x 6 mm. Both bald eagles (38%) and golden eagles (26%) in our study had Pb concentrations in blood indicative of "significant recent exposure" i.e., ≥0.4 ppm (Wiemeyer et al. 1989) and both species commonly scavenge hunter-killed ground squirrels.

The link between Pb poisoning in bald eagles and ingestion of waterfowl wounded with Pb shot has been well established (Pattee and Hennes 1983). A similar mechanism doubtless exists with golden eagles because they commonly take waterfowl (Olendorff 1976, Palmer 1988). Although Pb shot has been banned for waterfowl hunting in the U.S., use is still legal and widespread in Canada where most migrant eagles originate (Harmata 1984, McClelland et al. 1994, Tilly 1995). Continuous exposure to contaminated waterfowl is feasible as ducks and geese wounded the previous autumn in Canada may gradually weaken or succumb throughout winter and spring in the U.S. and become available to eagles.

Determining sublethal effects of any contaminant and concentration at which they occur is problematic as is the impact on individuals or populations represented (Heinz 1989). Most toxicology studies report contaminant concentrations derived from analyses of tissues other than blood. Relating blood concentrations to other tissue concentrations for toxic thresholds of contaminants is tenuous (Henny and Meeker 1981, Hensler and Stout 1982). Regardless, the USFWS established ≥0.5 ppm Pb in blood of eagles as indicative of toxic but sublethal exposure (Redig 1985). Redig (1984) considered blood Pb concentrations of 0.2-1.00 ppm in eagles indicative of toxic, chronic, sublethal exposure and Redig (pers. comm.) felt that eagles with a blood Pb concentration of ≥0.80 ppm should be debilitated or at least appear sick. Symptoms of sublethal, toxic exposure to Pb in raptors may include anorexia (Reiser and Temple 1981), impairment of vision and motor activity (Pattee et al. 1981), and increased susceptibility to disease, capture, and collisions with vehicles (Scott and Eschmeyer 1980, Reiser and Temple 1981, Redig 1985).

Severe problems of lead poisoning in raptors have been reported and a few

involve eagles (e.g., Locke et al. 1969, Jacobsen et al. 1977, Reichel et al. 1984, Janssen et al. 1986, Gill and Langelier 1994). None of 36 eagles with blood Pb concentrations ≥0.4 ppm exhibited any sublethal symptoms. Golden and bald eagles used as lure birds in this study were fed many prey items collected using lead ammunition (shot and projectiles), often daily for weeks for up to 15 years. None ever exhibited any sickness, lethargy, or debilitation. Therefore, we feel ingestion of prey items contaminated with Pb shot and small caliber fragments may briefly elevate blood concentrations but seldom induce mortality and morbidity in healthy eagles. Under normal circumstances, it is highly unlikely a wild eagle would ingest as many Pb shot in prey in as short a duration as that which induced mortality in experimental eagles (Pattee et al. 1981), although Reichel et al. (1984) noted one eagle with 25 Pb shot in its stomach. Poisoning from ingestion of pellets and fragments may occur mostly if exacerbated by previous existing, debilitating conditions (e.g., injury, disease, starvation, other toxic contaminants) that inhibit the ability to regurgitate indigestible material (pellets). Individuals encountering Pb poisoned eagles should be vigilant for evidence of pre-existing conditions increasing sensitivity and suseptibility to Pb toxicosis.

Ecological differences between bald and golden eagles may account for higher Pb concentrations in bald eagles. Bald eagles, associated with aquatic habitats, probably consume more waterfowl containing Pb shot than golden eagles. Bald eagles also scavenge more than golden eagles and thus may encounter more animal carcasses shot and lost or abandoned. If data on residency time of marked eagles are representative, higher concentrations and incidence of Pb in bald eagles may be a result of lingering in areas of abundant hunter-killed ground squirrels more than golden eagles. Quicker movement of golden eagles through west-central Montana may result in reduced overall exposure to Pb contaminated food, reflected in lower sample concentrations than bald eagles, since both are eating similar prey in the study area.

Although mean concentrations and detection rates of Pb were lower in golden eagles, negative effects may be more profound. Calcium mitigates effects of Pb in ducks (Carlson and Nielsen 1985) and a similar antagonistic effect may occur in bald eagles because their diet is calcium rich (i.e., fish). In contrast, the predominantly mammalian diet of golden eagles may contain less calcium, making them more sensitive (up to 3X) to Pb poisoning than bald eagles (Thomas, N., Nat. Wildl. Health Res. Ctr., Madison, WI, pers. comm.). Craig et al. (1990) attributed deaths of golden eagles with 0.54 and 0.23 ppm Pb in blood to Pb poisoning, although those levels are considered far too low to be associated with Pb poisoning (Wiemeyer, pers. comm.). Craig et al.. (1990) did not test for other contaminants nor did they mention presence of other conditions (e.g. injuries) that may have contributed to or caused death. Six golden eagles were recovered and treated for the effects of Pb poisoning during our study. In contrast, no bald eagles were found suffering the effects of Pb poisoning although our data indicated they were more extensively and severely contaminated.

Hg in blood of bald eagles suggests eagles fed on a moderately contaminated food supply (Eisler 1987) that was most likely aquatically associated (Wiemeyer 1991). Frenzel and Anthony (1989) found very low Hg concentrations in waterfowl but very high concentrations in bald eagles in Oregon. Although they did not analyze Hg in fish, other studies show fish often

contain high concentrations of Hg (Eisler 1987). The source of Hg in aquatic ecosystems is generally unknown but is often related to leaching Hg from hard rock mine tailings treated with the "Washoe Process" (Martin 1992). Mines with tailing piles thus treated are present throughout the northern Rocky Mountains and western Canada. Portions of the western U.S. and Canada cover mecuriferous rock (Jonasson and Boyle 1971 *in* Wiemeyer et al. 1989) and natural leaching also may be a source of Hg in watersheds. Once in aquatic ecosystems, Hg is bioaccumulated as methyl-mercury through micro-organisms (Colwell et al. 1975) to invertebrates (Hildebrand et al. 1980) to fishes or, directly into fish through gill surfaces from water (Phillips and Buhler 1980). Tissue Hg in some fishes increases exponentially with size (Phillips et al. 1987) and the contaminant is further biomagnified in predators.

Low concentrations and detection rates of Hg in blood of golden eagles probably reflect upland habitat and prey preferences. Golden eagles prefer mammalian prey which generally is less dependent on aquatic foods and thus would be less likely to accumulate Hg. However, direct consumption of fish by golden eagles may be higher than reported historically (e.g., Carnie 1954; 3.6%, Olendorff 1976; 0.4%). For example, golden eagles captured spawning rainbow trout (*Oncorhynchus mykiss*) in Arizona during March (Brown 1992) indicating that fish may be included in the diet of migrants more than previously recorded. Golden eagles nesting along the Scottish coast had lower reproductive success than those inland because they fed on contaminated seabirds (Furness et al. 1989). Gulls (*Larus* spp.), notorious for accumulating contaminants (Kozie 1986), migrate along similar routes as eagles in Montana and may be taken by eagles.

Dietary Hg affects raptorial species neurologically (Fimreite and Karstad 1971) and reproductively (Fimreite and Karstad 1971, Wiemeyer et al. 1984). Neurological effects appear to be manifested in a threshold effect resulting in overall weakness and wasting. Se may mitigate effects of Hg, or vice versa (Pellitier 1985, Eisler 1987) but no correlation between Hg and Se was found to indicate antagonistic interaction of the 2 elements. However, uptake and excretion rates may vary and any compensatory mechanism may be masked by analysis of only one sample per subject. None of the eagles sampled in this study displayed symptoms of Hg poisoning and we are unaware of any published accounts of toxic concentrations of Hg in blood of bald or golden eagles. We suspect as result of attention to Pb poisoning and lack of funds for additional analyses, some studies may have attributed Hg-induced mortality or morbidity in eagles to Pb (e.g., Craig et al. 1990).

Incidence of Se residues in both bald and golden eagles was high, with mean concentrations higher than those considered toxic in bovines (≥0.50 ppm in blood: Eisler, R., Patuxent Wildl. Res. Ctr., pers. comm.). However, applicability of this toxic concentration to birds is tenuous. Se induced mortality has been documented in waterfowl, but the effects are primarily teratogenic or manifested in reduced natality or productivity (Eisler 1985, Ohlendorf et al. 1988, Heinz et al. 1989, Hoffman et al. 1990). Although congenital mandibular deformities have been observed in bald eagles in the Great Lakes Region, Bowerman et al. (1994) stated Se was an unlikely causitive agent and similar effects have not been recorded for eagles elsewhere. None of the eagles from our study exhibited symptoms of Se poisoning.

Organochlorines

DDE (a metabolite of DDT) was the primary contaminant reducing reproductive success of bald eagles in

North America with the majority of exposure from the avian portion of the diet (Wiemeyer 1991). Although DDE is less toxic to birds than most organochlorines, it can elicit abnormal behavior, egg shell thinning and adult and embryonic mortality (Risebrough 1986). Low concentrations found in our study reflect the ban on DDT and subsequent decline in use, but continued presence (i.e., detection frequency) indicates persistence of the chemical.

DDE concentrations in wintering bald eagles in Colorado in 1977 were higher than those for autumn migrant eagles in Montana in 1979-1981 (Henny et al. 1979, Wiemeyer et al. 1989) all of which were higher than eagles sampled in our study between 1990 and 1993. DDE concentrations in plasma may be expected to be up to 2 times greater than that of whole blood (Wiemeyer, pers. comm.). Early samples with highest concentrations (Colorado) were plasma as were those with lowest concentrations and most recent collection dates (this study). Mid-range samples, both in chronology and DDE concentrations (autumn migrants in Montana) were whole blood. Differences among studies are probably a function of history, origin, and movements of eagles sampled, and laboratory techniques. Regardless, considering the sequence of studies, sample type and magnitude relationships of contaminant concentrations, and that eagles from all studies may move along similar migration routes (n.b. McClelland et al. 1994), results of all 3 studies indicate a progressive, dramatic decline in organochlorine residues in bald eagles, punctuating the efficacy of bans and use restrictions on organochlorine pesticides.

Organochlorine residues were detected in a surprising number of golden eagles sampled. Nearly half of the golden eagle plasma samples (48%) had detectable concentrations of DDE. The source was most likely waterfowl.

Cholinesterases

Organophosphorus and carbamate poisons used as rodenticides, insecticides and predacides have caused significant mortality in bald and golden eagles (Franson et al. 1985, Henny et al. 1987, USFWS 1991). Such poisonings continue throughout the west (USFWS 1991, Associated Press 1991A and B, 1993A and B, Garber 1992) because of mounting frustration among stockmen due to unavailability of effective predacides (e.g., 1080). The primary mechanism of poisoning is through illegal use of registered, legal insecticides (carbofuran, carbaryl, famphur, fenthion). These compounds are mixed with ethylene glycol (vehicle anti-freeze) and applied to large ungulate carcasses, ostensibly to kill coyotes. Several instances were noted in spring 1991 where one illegally poisoned bait carcass killed 10 eagles along the Rocky Mountain Front in Montana (USFWS 1991).

ChE activity in all bald eagle samples from our study was >20 percent lower than that found by Dieter and Wiemeyer (1978). Lower AChE activity of samples generated by this study may be a result of captive, controlled situation of eagles in their study or use of different substrate between studies. However, difference is more likely a function of less than optimal sample preservation procedures dictated by field studies in remote areas employed in our study.

Taira and McEwen (In press) found higher BChE activity in nestling golden eagles in areas treated with an anticholinergic pesticide than nestlings in an untreated control area. They attributed increased BChE activity to a rebound effect caused by release of large amounts of enzyme from the liver (Thompson 1991), suggesting the ratio of AChE to BChE may be more indicative of exposure to anticholinergic

North America with the majority of exposure from the avian portion of the diet (Wiemeyer 1991). Although DDE is less toxic to birds than most organochlorines, it can elicit abnormal behavior, egg shell thinning and adult and embryonic mortality (Risebrough 1986). Low concentrations found in our study reflect the ban on DDT and subsequent decline in use, but continued presence (i.e., detection frequency) indicates persistence of the chemical.

DDE concentrations in wintering bald eagles in Colorado in 1977 were higher than those for autumn migrant eagles in Montana in 1979-1981 (Henny et al. 1979, Wiemeyer et al. 1989) all of which were higher than eagles sampled in our study between 1990 and 1993. DDE concentrations in plasma may be expected to be up to 2 times greater than that of whole blood (Wiemeyer, pers. comm.). Early samples with highest concentrations (Colorado) were plasma as were those with lowest concentrations and most recent collection dates (this study). Mid-range samples, both in chronology and DDE concentrations (autumn migrants in Montana) were whole blood. Differences among studies are probably a function of history, origin, and movements of eagles sampled, and laboratory techniques. Regardless, considering the sequence of studies, sample type and magnitude relationships of contaminant concentrations, and that eagles from all studies may move along similar migration routes (n.b. McClelland et al. 1994), results of all 3 studies indicate a progressive, dramatic decline in organochlorine residues in bald eagles, punctuating the efficacy of bans and use restrictions on organochlorine pesticides.

Organochlorine residues were detected in a surprising number of golden eagles sampled. Nearly half of the golden eagle plasma samples (48%) had detectable concentrations of DDE. The source was most likely waterfowl.

Cholinesterases

Organophosphorus and carbamate poisons used as rodenticides, insecticides and predacides have caused significant mortality in bald and golden eagles (Franson et al. 1985, Henny et al. 1987, USFWS 1991). Such poisonings continue throughout the west (USFWS 1991, Associated Press 1991A and B, 1993A and B, Garber 1992) because of mounting frustration among stockmen due to unavailability of effective predacides (e.g., 1080). The primary mechanism of poisoning is through illegal use of registered, legal insecticides (carbofuran, carbaryl, famphur, fenthion). These compounds are mixed with ethylene glycol (vehicle anti-freeze) and applied to large ungulate carcasses, ostensibly to kill coyotes. Several instances were noted in spring 1991 where one illegally poisoned bait carcass killed 10 eagles along the Rocky Mountain Front in Montana (USFWS 1991).

ChE activity in all bald eagle samples from our study was >20 percent lower than that found by Dieter and Wiemeyer (1978). Lower AChE activity of samples generated by this study may be a result of captive, controlled situation of eagles in their study or use of different substrate between studies. However, difference is more likely a function of less than optimal sample preservation procedures dictated by field studies in remote areas employed in our study.

Taira and McEwen (In press) found higher BChE activity in nestling golden eagles in areas treated with an anticholinergic pesticide than nestlings in an untreated control area. They attributed increased BChE activity to a rebound effect caused by release of large amounts of enzyme from the liver (Thompson 1991), suggesting the ratio of AChE to BChE may be more indicative of exposure to anticholinergic

compounds than depressed AChE alone. If so, up to 13 percent of bald eagles and 10 percent of golden eagles in west-central Montana may have been exposed to anticholinergic compounds.

Evaluation of the proportion of migrant eagles exposed to anticholinergic compounds is tenuous and should be viewed with extreme caution without more data on baseline ChE activity in normal wild eagles collected under optimal conditions. If AChE:BChE ratio is more indicative of exposure than absolute values, preservation techniques may not have seriously affected interpretation of results.

Conclusion

Despite fairly pervasive incidence and occasionally high concentrations of potentially toxic contaminants, populations from which both eagle species originated appear stable or increasing. Bald eagles migrating through Montana originate in central to northern, western Canada (Harmata 1984, McClelland et al. 1994) and population status and productivity there is normal (n.b. Gerrard et al. 1994). Additionally, bald eagles in the Continental U.S. were reclassified from endangered to threatened on 12 July 1995 (Fed. Reg. 60:36000-36010). Status of wintering populations (primarily composed of migrants) contributed to reclassification. Studies on population status of golden eagles have indicated healthy and stable populations in the Continental U.S. (e.g., Harlow and Bloom 1989, Phillips et al. 1990) but we are aware of no similar studies in western Canada. However, counts of vernal migrant golden eagles at Rogers Pass, Montana and Mt. Lorette, Alberta between 1987 and 1994 showed little difference in magnitude among years (Tilly 1995), suggesting at least population stability.

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Appendix A. *Classification formulas for sex assignment of captured golden eagles, as determined by discriminate analysis. See text for classification of age class.*

Adults:

M = -1427.744 + 13.17478(TWAP[1]) + 17.59463(BD)+ 29.76324(WC) + 2.846877(WS) - 1.139853(HL)

F = -1696.418 + 15.47448(TWAP) + 19.89797(BD)+ 32.67586(WC) + 3.188033(WS) - 1.833955(HL)

Immatures:

M = -1140.714 + 9.111182(TWAP) + 48.47891(BD)+ 10.07384(TL) + 2.364044(WS) + 0.6223608(AWT)

F = -1349.390 + 10.62534(TWAP) + 51.91983(BD)+ 10.96982(TL) + 2.532903(WS) + 5.069061(AWT)

IF 1 > 2 THEN THE EAGLE IS A MALE, OTHERWISE A FEMALE.

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[1]TWAP = Tarsal Width, anterio-posterior, BD = Bill Depth, WC = Wing Chord, WS = Wing Span, HL = Hallux Length, AWT = Weight (without crop contents), TL = tail length.