

fixed effects ANOVA model. Data from recaptured eagles were excluded in this analysis. Recapture data were analyzed using a paired t-test.

JACKRABBIT TISSUE SAMPLES

Black-tailed jackrabbits (*Lepus californicus*), the major live prey of eagles wintering in our study area (pers. observ.), were collected during the winter of 1993-94. Jackrabbit liver and muscle tissue samples (≥ 5 gms each) were collected with steel shot from the Lemhi and Pahsimeroi Valleys. The samples were frozen until analyses were completed at the Holm Research Center, University of Idaho in Moscow, Idaho. Minimum detection level for tissue samples was 0.05 ppm wet weight. All of the liver samples were analyzed for lead but only muscle samples from jackrabbits with liver lead levels ≥ 0.50 ppm were analyzed.

RESULTS

GOLDEN EAGLES

Lead. From January 1990 through February 1997, data were collected from 290 wintering golden eagles. Lead was detected in the blood of 99.6% of the 281 eagles we trapped and from which blood samples were taken. The mean blood lead concentration of all the wintering golden eagles captured was 0.26 ppm \pm 0.35 ppm lead in blood (geometric mean: 0.17 ppm; range: 3.00 ppm – below the detection limit). Elevated blood lead levels (≥ 0.20 ppm) were found in 45.6% of the wintering birds sampled. Of these eagles, most were sub-clinically affected (0.20-0.60 ppm; Figure 2). However, a small group (5.4%) of birds had blood lead levels in the two highest categories (>0.60 ppm lead).

Although 52% of the golden eagles sampled in the Lemhi Valley and only 37% of the eagles captured in the Pahsimeroi Valley had elevated blood lead levels, there was no significant difference in golden eagle blood lead levels between the two valleys ($P=0.351$). The sample obtained ($n=5$) from the Birch Creek Valley was too small for statistical analysis. However, lead levels in golden eagles trapped there appeared similar to those in the other two valleys. All of the birds sampled in the Birch Creek Valley had detectable levels of lead in the blood and two of the five (40%) had blood lead levels elevated above background.

There was a highly significant difference in golden eagle blood lead levels among years ($P=0.000$; Table 1). Also, there was a significant valley by age interaction ($P=0.030$); more subadult males (39.5%) were captured in the Pahsimeroi than any other age class of eagle (Figure 3). More subadult golden eagles were captured in the study area than adults and more males than females but neither of these differences were significant ($P=0.488$ and $P=0.689$, respectively).

Table 1. Mean blood lead levels (ppm, wet weight) of golden eagles captured in east central Idaho from January 1990 through February 1997.

Year	n	Arithmetic mean	Geometric mean
1989-90	14	0.42	0.31
1990-91	16	1.10	0.11
1991-92	34	0.14	0.10
1992-93	42	0.20	0.16
1993-94	33	0.41	0.21
1994-95	34	0.16	0.08
1995-96	32	0.31	0.24
1996-97	33	0.32	0.28

Recaptures/Sightings of Color-marked Eagles. We recaptured (n = 39) or recovered dead (n = 4), 43 golden eagles during the winters from 1991-1997. Four of the eagles we recaptured were caught the second time during the same winter in which they were first caught. Fifteen were recaptured the winter following first capture. Eight were recaptured two years later and two of these birds had originally been banded as nestlings in the study area. Eight birds were recaptured three winters later. Three birds were recaptured four winters after first capture (one of these birds was first banded as a nestling in the study area), and one was recaptured five winters later. Most of the recaptured birds were retrapped only once. However, three were recaptured twice and two golden eagles were recaptured three times.

Most of the golden eagles we captured the second time were at, or within a mile of, the original capture site. However, two of the eagles were recaptured in the valley adjacent to the valley in which they were first caught.

In addition to recapturing birds we banded, we also captured one golden eagle on 6 December 1994 that was originally banded as a nestling by another biologist on the Egaksrak River in the Arctic National Wildlife Refuge on 17 July 1990 (C. McIntyre pers. comm.) Interestingly, another eagle that was captured and equipped with a PTT as a nestling in Denali National Park, Alaska in 1991 spent part of the following winter in the Lemhi Valley (C. McIntyre, pers. comm.).

The causes of mortality for birds that were recovered dead, were: electrocution (2), collision with an automobile (1), and undetermined poisoning (1). One banded eagle was recaptured alive but died of secondary sodium pentothal poisoning shortly after capture.

There was not a significant difference in blood lead levels of individual golden eagles between the first and last capture (n=33; p=0.091). Blood lead levels increased in 58.8% of the recaptured eagles and decreased or stayed the same in 41.1% of the birds (Table 2).

Figure 2. Comparison, by year, of Blood Lead Levels in Wintering Golden Eagles in East Central Idaho; 1990-97.

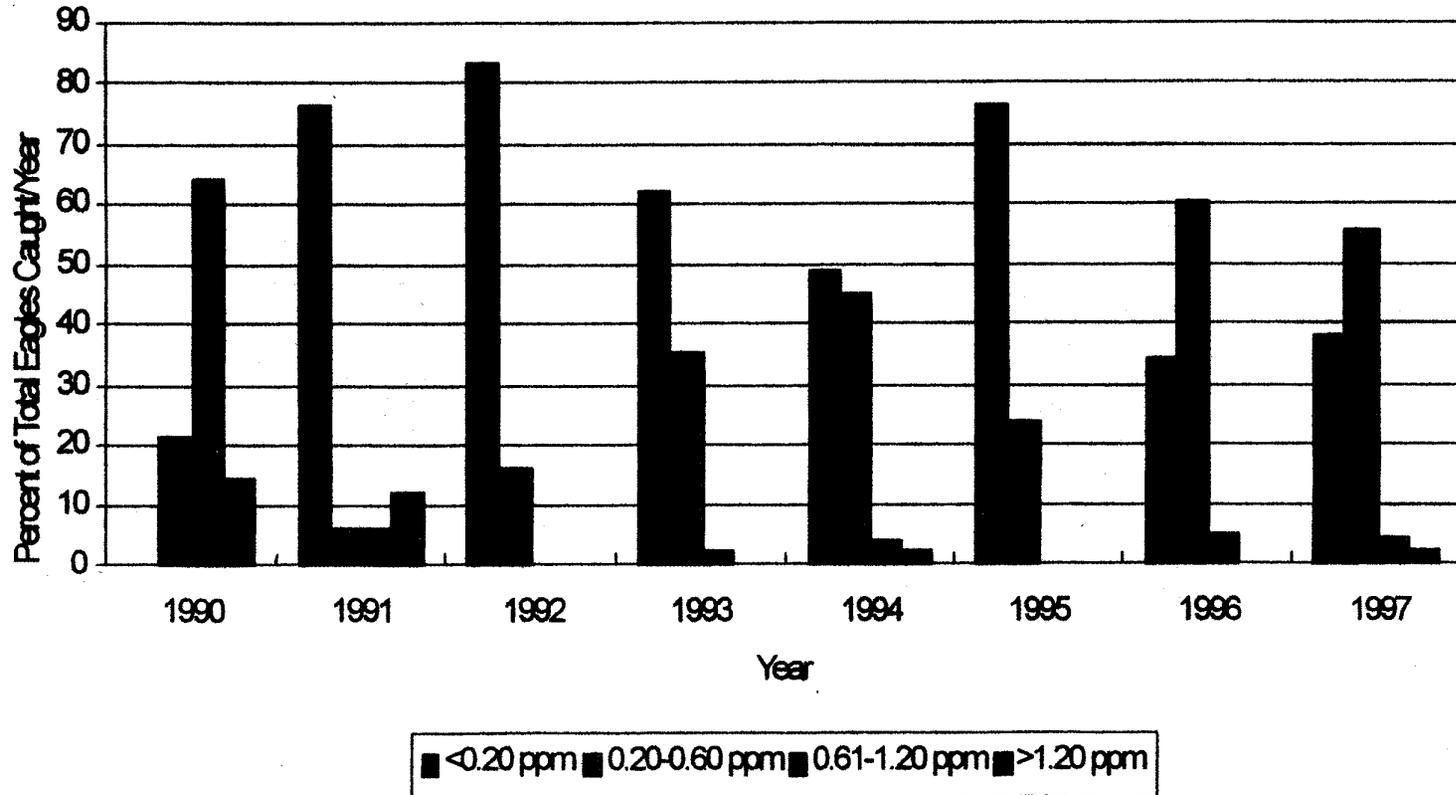
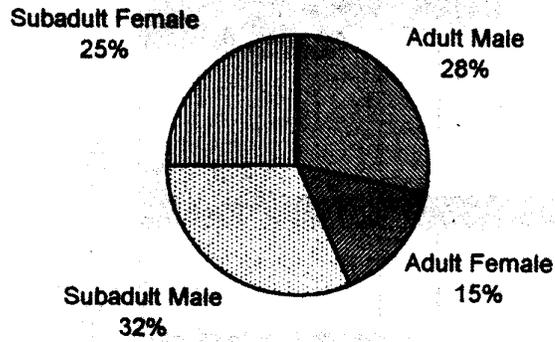


Figure 3. Age and Sex of Wintering Golden Eagles Captured in East Central Idaho; 1990-97.

Lemhi Valley



Pahsimeroi Valley

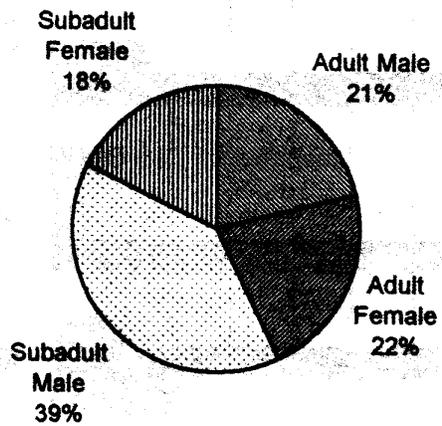


Table 2. Changes in blood lead levels of wintering golden eagles in east central Idaho recaptured one or more years after first capture; 1990-1997.

Change in Blood Lead Levels	-1	-	0	+	+1
sample size	5	7	2	9	11
mean # years between samples	1.6	2.3	2.0	2.1	2.5

Key: -1: lead level decreased by one exposure category (see methods)

-: decrease in lead level but remained in same category

0: no change in lead level

+: increase in lead level but remained in same category

+1: lead level increased by one exposure category

Nestlings. Twelve blood samples were obtained in summer 1991 and 1992 from nestling golden eagles, just prior to fledging. None of these bloods contained elevated lead.

Mercury. None of the golden eagles sampled from 1990-1995 in our study had elevated Hg levels in the blood ($\bar{x} = 0.027 \pm 0.047$). Therefore, mercury analysis of golden eagle bloods was discontinued after winter 1994-95.

BALD EAGLES

Lead. Data were collected from 39 bald eagles during the study. Blood samples were taken from 36 of these birds. Although an equal number of adult and subadult bald eagles were captured, most were males (Table 3). Most of the bald eagles were assumed to be nonresidents because there are only three known bald eagle nesting territories within the study area.

Table 3. Sex and age of wintering bald eagles captured in the Lemhi and Pahsimeroi Valleys from 1990-1997.

	ADULT MALE	ADULT FEMALE	SUBADULT MALE	SUBADULT FEMALE	TOTAL
sample size	15	3	13	5	36
Percent	41.7	8.3	36.1	13.9	100.0

The mean blood Pb level for bald eagles was 0.43 ± 0.46 ppm (geometric mean: 0.26 ppm; range: 1.90 – 0.04 ppm). Fourteen (38.9%) of the bald eagles had blood lead

levels \leq 0.20 ppm; 15 (41.7%) had blood lead levels from 0.20 – 0.60; five (13.9%) had blood lead levels from 0.61 – 1.20 ppm; and 2 (5.5%) had \geq 1.20 ppm lead in blood.

Mercury. The mean level of Hg in the sampled bald eagles was 1.10 ± 0.63 ppm (geometric mean: 0.94 ppm; range: 2.70 - 0.04 ppm).

Recaptures. We recaptured one adult male bald eagle. This bird was recaptured within two days of first capture, so no additional blood samples were collected.

One subadult male bald eagle caught on 9 January 1993 in the Pahsimeroi Valley, was found dead in Alberta, Canada on 30 April 1993. This bird's blood lead level when captured was 0.34 ppm and its Hg level was 0.55 ppm. No information was available on the cause of death or on Pb and Hg levels in the eagle at the time of its death.

A subadult female bald eagle banded on 12 January 1996 near Lemhi, Idaho in the Lemhi Valley was found electrocuted 17 May 1996 approximately 61 km southeast of Salmon, Idaho. Her blood lead level in January was in the *exposed* category (0.23 ppm lead) and there were 1.10 ppm Hg in the blood. No information is available currently on Pb or Hg levels in this eagle at the time of death.

LEAD IN JACKRABBITS

Liver and muscle tissue samples were collected from 32 jackrabbits in the Lemhi Valley and 29 jackrabbits in the Pahsimeroi Valley during winter 1993-94. Seventy-two percent of all the jackrabbits sampled had liver lead levels below the detection limit. In the Pahsimeroi River Valley 24.1% (7) of the jackrabbits had detectable lead levels in the liver (range 0.29 – 2.10 ppm). Of these muscle samples analyzed, only one had detectable lead (0.97 ppm). In the Lemhi Valley 31.3% (10) of the jackrabbits had detectable lead in their livers (range: 0.29 – 0.79 ppm). No jackrabbit muscle tissues sampled in the Lemhi River Valley contained detectable lead.

DISCUSSION

LEAD IN GOLDEN EAGLES

The number of wintering golden eagles in our study area with elevated blood lead levels from 1990-1997 (45.6%) is slightly higher than reported by Pattee et al. (1990) in southern California (35.8%; $n = 162$). Another study on the prevalence of lead exposure and/or lead poisoning in bald and golden eagles from eleven states, ranged from 3-44% over a 16-year period; 1980-1995 (Kramer and Redig 1997). A study of migrating golden eagles in Montana reported a higher incidence of lead contamination than we found, with 56% of 86 golden eagles having blood lead levels > 0.20 ppm wet

weight (Harmata and Restani 1995). These data suggest that the sources of lead contamination to golden eagle populations are widespread.

The connection between lead-shot used in waterfowl hunting and lead poisoning in bald eagles has been well documented (Reichel et al. 1984, Feierabend and Myers 1984). Because of this link, it was expected that the number of lead-poisoned eagles would decrease after the 1991 federal ban on the use of lead shot in waterfowl hunting. Unfortunately, neither we nor Kramer and Redig (1997) have observed a decline in lead-poisoned eagles since 1991. Although we found a significant difference in lead levels among years, there has been no downward trend during our study. In fact, the number of eagles captured that had elevated lead in their blood during the winters of 1995-96 and 1996-97, is higher than in the previous four winters. This suggests that there is a continuing source of lead contamination to eagles. Kramer and Redig (1997) suggest that we should reevaluate current theories regarding the source of lead contamination in eagles, as well as, how eagles are poisoned.

The physiological conditions that affect the uptake, binding and release of Pb by tissues and organs in animals are not well known (Task Group on Metal Accumulation 1973). In addition, lead accumulations following ingestion of Pb shot can be affected by many factors, including age, geographic location, habitat, and time of year (Eisler 1988). Nonetheless, blood lead levels are thought to be an accurate indication of relatively recent exposure to lead. Lead in blood is available for distribution throughout the body, absorption into other tissues, or can be excreted from the body (Task Group on Metal Accumulation 1973). As this occurs, in the absence of a continued source of contamination, lead levels in the blood tend to decrease. Kendall et al. (1982) reported blood lead levels of a ringed turtle-dove (*Streptopelia risoria*) fed a single oral dose of 2 lead pellets to have dropped from 4.69 mg/l at 24 hours after ingestion, to 0.14 mg/l at 14 days after ingestion. We assume that blood lead levels in eagles would show a similar decline in the absence of new sources of lead contamination. However, our results from individual eagles tracked over a long period did not necessarily show a decrease in blood lead levels. In fact, birds recaptured at least one winter apart exhibited no significant difference in blood lead levels between first and last recapture. Pattee et al. (1990) also reported no significant difference in the blood lead levels of golden eagles based on age. In our study, although some golden eagle blood lead levels decreased between subsequent recaptures, blood lead concentrations increased with age in 58.8% of the golden eagles recaptured. This indicates that many of the eagles we sampled must be repeatedly exposed to lead contamination.

Biologically incorporated or tissue-bound lead has been recognized as a possible contributing factor to elevated lead levels in raptors but it is not generally considered to be the primary source of lead toxicosis (Pattee and Hennes 1983). Henny et al. (1991, 1994) have reported that lead in the tissue of prey is not readily available to raptors because it generally concentrates in bones, which are not completely digested. In addition, eagles often strip the flesh from prey and leave much of the bony material behind (pers. observ.).