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Lead concentrations in Golden and Bald eagles.—In recent years, lead (Pb) was found in elevated concentrations in free-ranging Bald Eagles (*Haliaeetus leucocephalus*), sometimes in high enough quantities to cause mortality (Kaiser et al. 1980, Reichel et al. 1984). Published reports of mortality due to lead poisoning in other free-ranging raptors are few. California Condors (*Gymnogyps californianus*), Rough-legged Hawks (*Buteo lagopus*), Golden Eagles (*Aquila chrysaetos*), and a Red-tailed Hawk (*Buteo jamaicensis*) have died due to lead poisoning (USFWS 1985, Wiemeyer et al. 1988). Additionally, other Golden Eagles which died from diseases or unknown causes contained elevated lead concentrations (USFWS 1985). We report lead concentrations in the livers of 16 Golden and six Bald eagles and in blood samples from two Golden and two Bald eagles found dead or moribund in Idaho.

Study area and methods.—From 1977 through 1984, the Idaho Department of Fish and Game (IDFG) sent eagles to the National Wildlife Health Research Center (NWHRC),

TABLE 1

FREQUENCY DISTRIBUTIONS AND MEANS OF LEAD CONCENTRATIONS IN LIVERS FROM BALD AND GOLDEN EAGLES COLLECTED IN CENTRAL AND SOUTHERN IDAHO, 1977–1986

Species	N	$\bar{x}$ (SD)[a]	Frequency distribution (ppm wet wt.)		
			<2.0	2.0–8.0	>8.0
Golden Eagle	16	8.86 (9.02)	7	2	7
Bald Eagle	6	25.70 (17.04)	1	0	5
Total	22		8	2	12

[a] Standard deviation in parentheses.

Madison, Wisconsin, for necropsy if the cause of death was not apparent. After learning that some of these eagles died of lead poisoning, the IDFG made a concerted effort during 1985 and 1986 to send eagles that they obtained in central and southern Idaho to the NWHRC for necropsy.

Habitats in central and southern Idaho vary from flat to rolling, cool desert dominated by sagebrush (*Artemisia tridentata*)-grass associations to mountainous areas containing major river systems. Vegetation changes with elevation from cool desert to subalpine.

Lead concentrations in liver were determined by the NWHRC following necropsy. In addition, one blood sample from a live Bald Eagle was sent to the NWHRC for analysis. Five ml blood samples were taken by brachial puncture and sent to the Washington Animal Disease Diagnostic Laboratory, Washington State Univ., Pullman, Washington for analysis from an additional three sick birds (two Golden Eagles and one Bald Eagle). All samples were reported on a wet weight basis (minimum detection level = 0.10 ppm) as are all literature values reported herein.

Results and discussion.—Five Golden Eagles and one Bald Eagle were recovered from 1977 to 1984. Eleven Golden Eagles and four Bald Eagles were recovered in 1985 and 1986, and one Bald Eagle was recovered in 1987. In addition, blood samples were collected from two live Bald Eagles and two Golden Eagles in 1985 and 1986. Both a liver and a blood sample are reported for one of the Golden Eagles. Lead concentrations in livers of Golden Eagles ranged from 0.19 to 26 ppm and in Bald Eagles from 0.19 to 51 ppm. A greater proportion of Bald Eagles (83%) had elevated (>8.0 ppm) concentrations of lead in liver than Golden Eagles (44%) (Table 1).

Pattee et al. (1981) suggested that Bald Eagles with lead concentrations in liver >10 ppm were acutely exposed whereas NWHRC has suggested that 8.0 ppm lead in liver be used as the threshold level in Bald Eagles (letter on file). Feierabend and Myers (1984) considered Bald Eagles with lead concentrations in liver between 2.0 and 10.0 ppm to be sublethally poisoned and concentrations below about 2.0 ppm to be uncontaminated.

Five of 16 Golden Eagles and five of six Bald Eagles were confirmed by NHWRC to have died of lead poisoning. The liver of one Golden Eagle had a concentration of 16 ppm lead, although a blood sample taken 9 days before its death contained only 0.54 ppm lead. This bird died of lead poisoning and aspergillosis. Feierabend and Myers (1984) considered Bald Eagles with >0.6 ppm lead in blood to be sublethally poisoned.

Two other Golden Eagles with elevated concentrations of lead in their livers were necropsied in Idaho. One contained an apparently ingested #7½ lead shot pellet in the lower gastrointestinal tract, and the other contained a fragment of a copper-jacketed lead bullet in its proventriculus. The birds were sent to the NWHRC for secondary examination; their

livers contained 23 and 18 ppm lead, respectively. However, lead poisoning could not be confirmed because of the previous necropsy.

Waterfowl which carry embedded pellets are frequently the pathway of lead exposure to raptors (Pattee and Hennes 1983). However, a wild California Condor, which died of lead poisoning, contained a fragment of a copper-jacketed lead bullet in its gizzard (Wiemeyer et al. 1988). Neither projectile (#7½ shot or rifle bullet) recovered from the two eagles in our study area are associated with waterfowl hunting. Platt (1976) found lead pellets in castings of Bald Eagles in Utah and thought that the birds were eating hunter-killed black-tailed jackrabbits (*Lepus californicus*). We suggest that in both instances, hunter-killed game other than waterfowl may have been the source of exposure to the two Idaho eagles.

Another Golden Eagle, collected in east-central Idaho, was sublethally poisoned by lead. Its liver contained 6.3 ppm lead, and the NWHRC suggested that lead may have contributed to its death which resulted primarily from necrotic colitis (NWHRC, letter on file).

Three morbid eagles contained elevated concentrations of lead in blood samples, but X-rays revealed no lead projectiles in the birds' gastrointestinal tracts. One, an immature Bald Eagle, which exhibited symptoms of lead poisoning (ataxia, convulsions, anorexia, and absence of trauma) had 0.14 ppm lead in blood, suggesting past exposure (E. Stauber, pers. comm.). The blood sample from another Bald Eagle (age unreported) contained 0.23 ppm lead. The third bird, a Golden Eagle, was suffering from malnutrition and had an injured wing. No symptoms of lead poisoning were apparent in this bird, but a blood sample contained 0.21 ppm lead. Concentrations of 0.1 ppm or less are expected in uncontaminated birds (Feierabend and Myers 1984).

East-central Idaho is a historic lead mining and milling area. However, Custer et al. (1983) have suggested that lead poisoning in wild raptors is probably not due to ingestion of biologically incorporated lead (e.g., an eagle eating an animal whose flesh was contaminated by feeding on plants growing on tailings at a lead mine).

Relatively large populations of wintering Golden and Bald eagles and nesting Golden Eagles inhabit parts of southern Idaho (Craig et al. 1984). The nesting areas and migration routes of these Bald Eagles are not well known, although band recoveries indicate a direct southerly route from northern nesting areas to Idaho for some (USFWS 1985). Data on the migration routes of Golden Eagles wintering in eastern Idaho are lacking. Because symptoms of lead poisoning may be delayed following ingestion (Hoffman et al. 1981), eagles can travel extended distances before indications of poisoning occur. Therefore, the source of lead contamination in Idaho eagles is not known.

Ten of the 17 Golden Eagles and seven of the eight Bald Eagles examined during this study were exposed to lead. These data suggest that both species may frequently be exposed to lead, sometimes to lethal concentrations, and that lead poisoning in Golden Eagles may be a more serious problem than previously thought. However, our results may be biased because all eagles analyzed were either sick or dead. Further study is needed to determine the portion of the eagle populations in Idaho which suffer lead contamination.

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Body temperatures of migrant accipiter hawks just after flight.—Body temperature (T_b) during flight or shortly after flight has been recorded in many studies. Red-tail Tropicbirds (*Phaethon rubri cauda*) captured within 10 sec of returning to land from extended flights had a mean T_b of 40.9°C, almost 2°C higher than the mean for these birds during incubation (Howell and Bartholomew 1962). Platania et al. (1986) measured T_b of 250 seabirds of 23 species within two min after shooting the birds from a boat 30 to 60 km off the coast. The T_b of these birds flying at sea was about 1°C higher than that of individuals studied at nesting sites. Rock Doves (*Columba livia*) shot immediately after landing at air temperatures (T_a) ranging from 13 to 26°C had body temperatures between 41.5–43.8°C (Pearson 1964), and Hart and Roy (1967) measured a T_b of 44.5°C on pigeons during free flights at air temperatures between 4–30°C. The T_b of pigeons flying in a wind tunnel increased 1.5–3.0°C in the first min of flight and remained at this higher level (Hirth et al. 1987) and at T_a above