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EXPERIMENTAL LEAD-SHOT POISONING IN BALD EAGLES

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Nine of 168 dead or dying bald eagles (*Haliaeetus leucocephalus*) examined in 1975–77 apparently died of lead poisoning, and exhibited liver lead levels (wet weight) of 23–38 ppm; 3 had lead shot in their digestive system (Kaiser et al. 1980). Although diagnostic criteria of acute lead exposure in experimentally dosed mallards (*Anas platyrhynchos*) exist (Longcore et al. 1974), no data are available for tissue lead levels in bald eagles experimentally dosed with lead shot. The objective of our study was to determine tissue lead values associated with lead-shot poisoning and mortality in captive bald eagles.

Documentation of lead-shot poisoning is most extensive for waterfowl (Bellrose 1964, Stout and Cornwell 1976), but has been reported in other species (Hunter and Rosen 1965, Westemeier 1966, Locke and Bagley 1967, Locke et al. 1969, Benson et al. 1974), including bald eagles (Mulhern et al. 1970, Jacobson et al. 1977, Kaiser et al. 1980). Lead shot has been reported in bald eagle castings by Dunstan (1974) and Platt (1976), but little is known about the birds' susceptibility to lead poisoning or about shot retention by eagles. American kestrels (*Falco sparverius*) dosed with lead shot regurgitated the shot and exhibited no deleterious effects (Stendell 1980), but a prairie falcon (*Falco mexicanus*) (Benson et al. 1974) and an Andean condor (*Vultur gryphus*) (Locke et al. 1969) fed hunter-killed game that contained embedded shot subsequently died exhibiting signs of lead poisoning and elevated tissue lead levels.

METHODS

Five bald eagles were selected from birds held at the Patuxent Wildlife Research Center. All were unsuitable for rehabilitation and release or captive breeding, but were otherwise healthy and in good condition. Eagles were placed in wire-mesh cages ($3 \times 3 \times 1.9$ m) elevated over a concrete slab. The first bird was moved to the test pen on the day of dosage and fed Nebraska Brand Bird of Prey diet. Subsequent birds were acclimated to the pens for 1 week and maintained on a fish diet before the initiation of dosage. A 6th bird was sacrificed to provide

Table 1. Lead levels in soft tissues (ppm, wet weight) and bone (ppm, dry weight) of a control bald eagle and eagles dosed with #4 lead shot.

Experimental status	Sex	Lead concentration (ppm)						
		Kidney	Liver	Brain	Muscle	Femur	Humerus	Tibia
Dosed-died	M	6.7	15.2	2.0	1.9	15.0	17.0	12.0
Dosed-died	F	5.4	12.5	0.5	0.4	7.8	8.4	9.1
Dosed-died	F	6.7	11.5	0.9	0.5	9.7	9.1	9.3
Dosed-died	F	5.2	27.0	2.3	0.9	9.2	6.5	9.7
Avg.		6.0	16.6	1.4	0.9	10.4	10.3	10.0
Dosed-sacrificed	F	3.2	3.4	2.1	<0.1	12.2	13.6	13.8
Control	F	0.5	0.4	0.1	<0.1	4.5	6.2	6.6

control tissues. Birds were weighed and radiographed to provide a record of the location of any body shot (4 had been crippled by gunshot) prior to the initial dosage. Subsequently birds were weighed and radiographed at intervals of 1–2 weeks.

Eagles were dosed between 16 May 1978 and 19 March 1979; only 2 birds were on dosage at any 1 time. Shot were weighed and sorted to provide groups that varied in weight by no more than 0.5 mg. Initial dosage consisted of 10 #4 lead shot. Additional groups of 10 shot were given if all of the previous 10 shot were regurgitated (1 bird was given 11th and 12th doses of 30 and 26 shot). Frequent radiographs were taken to confirm the presence or absence of shot prior to additional doses. All shot given to the 1st eagle and the initial dose to the 2nd eagle were by intubation. Subsequent dosages to this eagle and all other eagles were given by inserting the shot into a smelt (95–120 mm total length) and force-feeding the eagle. Regurgitated shot recovered from under the cage were weighed to estimate the amount of lead eroded.

Dead eagles were necropsied, and portions of kidney, liver, heart, and other tissues were fixed in 10% buffered formalin and processed by American Histolabs, Inc., Silver Spring, Maryland. Sections were stained by the hematoxylin and eosin (H&E) and Kinyoun's acid-fast methods, and were examined microscopically without reference to necropsy records. Periodic acid-Schiff and Prussian blue stains also were employed. Remaining tissues were frozen for lead analysis.

Samples for lead residue analysis were dried for 2 hours at 110 C, charred for 2 hours at 200 C in a muffle furnace, then the temperature was raised to 550 C at a rate of 100 C/hour, and the samples were ashed overnight. The ash was dissolved in concentrated nitric and hydrochloric acids, and diluted to 10 ml with distilled deionized water. Determinations were made by flame atomic absorption spectrophotometry. The lower limit of quantification was 0.1 ppm, and recoveries from fortified chicken livers averaged 88%. Residues were not corrected for percent recovery, and are expressed as ppm wet weight for soft tissues and ppm dry weight for bone.

RESULTS

Four of the 5 bald eagles dosed with lead shot died; the 5th became blind and was sacrificed after 133 days. Soft-tissue lead levels were generally similar among those birds that died. Values for the dead birds were higher than those for the con-

Table 2. Characteristics of lead-shot dosages and response of eagles.

Time in captivity (years)	No. of shot given	No. shot in stomach at death	Total no. shot recovered	Total lead eroded (mg)	Shot retention (days)		Days to death	Body weight loss at death (%)
					Median	Range		
3	10	1	6	19.4	2	2–20	20	23
7	30	1	29	37.8	2	1–7	10	17
6	20	3	20	42.3	2	1–7	12	16
6	156	10	155	184.9	7	1–37	125	23
9	80	3	80	129.0	2	½–48	133	20

trol and sacrificed eagles (Table 1). Little lead accumulated in muscle of dead eagles ($\bar{x}$ = 0.9 ppm); highest levels were in the liver ($\bar{x}$ = 16.6 ppm) and kidney ($\bar{x}$ = 6.0 ppm). Brain and liver lead levels exhibited the most variation. Bone lead levels were higher in dosed eagles (including the sacrificed bird) than in the control. No apparent trends in lead levels were observed among the different bones.

Number of shot given, number retained, and amount of lead eroded varied among individuals (Table 2). As few as 10 and as many as 156 shot were given to an individual before it died. Number of days from initial dosage to death and retention time for shot also were variable (Table 2). Shot were regurgitated as quickly as 12 hours after dosage or retained as long as 48 days, with no pattern evident among or within individuals. Birds that died ceased eating several days before death, and did not regurgitate any more shot. All contained at least 1 shot in the stomach at the time of death. Due to the low bulk of the diets, it was not possible to ascertain pellet casting frequency.

Table 3. Lesions present in lead-poisoned bald eagles.

	No. affected	No. examined
Cardiovascular lesions		
Myocardial necrosis	4	4
Fibrinoid necrosis	2	5
Renal lesions		
Nephrosis	3	5
Acid-fast nuclear inclusions	0	5

Major gross lesions were emaciation and hydropericardium. Renal and cardiovascular lesions were present in the poisoned eagles (Table 3); renal tubular degeneration (nephrosis) was present in 3 of 5 eagles examined. Acid-fast intranuclear inclusions were not observed in proximal convoluted tubule epithelial cells, but there were prominent deposits of a granular brown pigment in the cytoplasm. Medial fibrinoid necrosis of small arteries was present in the gizzard and proventriculus of 1 eagle, and in the liver, spleen, heart, and kidney of another. Less pronounced arterial changes characterized by a lack of myofiber definition and swollen, irregular nuclei occurred in several vessels of a 3rd eagle. Multifocal myocardial necrosis was present in the 4 hearts examined. Splenic lymphoid depletion, gastritis, multifocal hepatitis, pancreatitis, and myositis were present but uncommon, and were probably unrelated to lead poisoning.

DISCUSSION

Liver and kidney lead levels appear to be useful criteria for diagnosing lead poisoning in bald eagles. Kidney lead levels above 5 ppm and liver lead levels above 10 ppm can be used as indicators of acute

exposure to lead. Field samples of dead or dying bald eagles were found to have liver lead levels of 21–38 ppm (Mulhern et al. 1970, Jacobson et al. 1977, Kaiser et al. 1980) and a kidney level of 5 ppm (Mulhern et al. 1970). These liver lead levels are higher than 3 of 4 of the experimentally killed eagles. The lower levels in the livers of the experimental eagles may reflect the short exposure time before death occurred. Wild eagles would probably be less likely to encounter such a large number of shot in such a short time period. Brain levels were low in the birds that died in less than 12 days, when compared to those that required 20 or more days to die. Brain levels were also high in the eagle that became blind following exposure.

These experimental lead values in the kidneys and livers of poisoned eagles are similar to the 6–20-ppm levels suggested by Longcore et al. (1974) as indicative of recent acute exposure in mallard ducks, although the actual residue levels they found in dead birds were considerably higher. The experimental eagle lead residues also are comparable to those found in studies with other bird species killed by lead poisoning (Locke et al. 1966, Bagley et al. 1967, Erne and Borg 1969). The number of lead shot given at one time (10) in this study may not be typical of field exposure, but there is evidence that wild eagles may, on occasion, accumulate large numbers of shot. Jacobson et al. (1977) found 75 lead shot in the digestive system of the bald eagle they examined. It appears that the residue levels reported in this study are reasonable, but may be low in comparison to field samples. Sublethal exposure is also hazardous and could contribute to a bird's death, as evidenced by the eagle that became blind without dying or exhibiting high kidney or liver lead levels.

Microscopic examination revealed arterial, myocardial, and renal lesions probably caused by lead toxicosis. Arterial fibrinoid necrosis is not a common lesion in wild birds; its presence in free-flying bald eagles suggests the possibility of lead poisoning. The brown renal pigment was possibly a hematinlike blood-breakdown pigment from erythrocytes damaged by lead. Acid-fast inclusions are more readily found in some species of lead-poisoned birds, such as mallards (Locke et al. 1966), than in others, such as Canada geese (*Branta canadensis*) (Locke et al. 1967); bald eagles apparently resemble the latter group. The glomerular lesions were likely caused by pre-experimental events of unknown cause.

Individual response to lead-shot ingestion was quite variable. The interaction of various factors (time of retention, number of shot retained, amount of lead eroded, individual susceptibility) apparently affect survival time. Predicting the effect of shot ingestion on a particular eagle is therefore difficult, because many unknown or poorly understood variables appear to influence an eagle's ability to survive shot ingestion. Healthy eagles probably could be expected to regurgitate lead shot and survive occasional exposure. However, if additional shot are swallowed, the possibility increases that the bird will reach a threshold of exposure at which point it stops eating, shot are retained, and death occurs. This threshold may be related to an interaction between lead erosion rates and those factors that influence shot retention, but the actual causative factors are unknown. Secondary factors such as disease, predation, accidents, starvation, and exposure also could contribute to the death of a weakened individual.

Lead poisoning has been diagnosed in

a number of dead eagles found in the field, but criteria based on experimentally dosed bald eagles has not existed. Based on our study, liver lead levels greater than 10 ppm and kidney levels greater than 5 ppm may be indicative of acute lead exposure. Although acid-fast intranuclear inclusions were not observed, characteristic renal and cardiovascular lesions were present. The extent of this mortality and its impact on bald eagle populations is unknown.

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