TOXICITY AND BIOAVAILABILITY STUDIES OF LEAD
AND OTHER ELEMENTALS IN THE LOWER COEUR D'ALENE RIVER

Prepared for

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Acknowledgements

...Bunker Hill Strike in 1885... development of "Bunker Hill", the "Morning", the "Hecla", the "Star", the "Golconda", and the "Sunshine" and other mining properties... the tonnage of rock handled annually rose rapidly and the quantities of mine tailings and mine waters poured into the South Fork of the Coeur d'Alene River....

Ellis 1938

...at Harrison Coeur d'Alene Lake receives the muddy waters of the Coeur d'Alene River which drains an immense area including the famous Coeur d'Alene mining district. These waters are so laden with silt that they may be traced far out into the clear water of the lake.

Kemmerer 1911

In April and May of 1924 a number of wild whistling swan... visited the lower Coeur d'Alene valley... and at least 25 of these beautiful large, white waterfowl sickened and died in that locality.

Coeur d'Alene Press

In review of the reopening of the pollution problem by residents along the lower part of the Coeur d'Alene River during the last few years as a result of the increase in mine slimes and alleged toxic substances reaching their lands....

Ellis 1938

Outbreaks of waterfowl mortality have been report in northern Idaho's Coeur d'Alene River Valley for decades.

Chupp and Dalke, 1964

In 1982 approximately 200 swans of an estimated 1,200 died.

Idaho Department of Fish and Game, 1982

About Units

Throughout this report, the amount of elemental in an animal or plant tissue, water, or sediment is expressed as a weight ratio, ug/g (micrograms per gram). These data are most readily converted to dosages when exposure (food, soil, and water) is known. Micrograms/gram are numerically equivalent to parts per million (ppm). By using ug/g and stressing dose (ug/organism), I hope to minimize the tendency of persons to attach biological significance to an environmental pollutant simply because it is expressed in whole numbers that sound familiar.

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Introductory Perspective

Waterborne wastes from the intensively mined Silver Valley of Northern Idaho have been extensively deposited over an estimated 10,000 acres of wetlands (Neufeld 1987). It has been estimated that 72 million tons of mining and milling waste was discharged to the Coeur d'Alene River and Lake system. The residual waste deposits along the Lower Coeur d'Alene River (LCDAR) contain extremely high amounts of lead as well as other elementals including cadmium, zinc, arsenic, copper, and iron. In excessive amounts these can cause harmful biologic effects. Lead and cadmium are particularly well-known "toxic metals," lead even being termed an "ancient metal" in some authoritative references (Finkel 1983). The vastness of the distribution of the potentially toxic wastes and the magnitude of contamination of the wetlands are the basis of much interest in this region.

Observations of the effects of excessive exposure of wildlife to mining and milling wastes appeared early (Kemmerer 1923) and have continued to the present time (Chupp 1956) including the work reported here. It is important to note, with respect to the toxicological significance of the wastes, that the nature of the exposure has changed dramatically during the past hundred years. The River is no longer "so laden with silt that it can be traced far out into the clear waters of Lake Coeur d'Alene" (Kemmerer et al. 1923). Likewise, suspended solids that "had made the river uninhabitable to most aquatic life" (Ellis 1938) have most likely become part of today's alluvium or moved through system as solutes. Later Chupp (1956) noted that, "The expected spring mortality of these creatures, particularly the swans, is a subject rivaling the weather as a topic of conversation." Chupp also reported that the die-off was locally ascribed to "leadwater" which was said to come down the LCDAR during the spring high water period (Chupp 1956).

The significance of the "lead water" associated with earlier deaths and illness in water cannot be estimated with certainty, but it is clear that vast amounts of mining and milling wastes remain deposited in LCDAR sediments. Periodically, and perhaps chronically, on a small scale, migrating waterfowl succumb to elemental poisoning, mostly likely lead. The toxicological significance of the sediments to wildlife, and indirectly to humans, is incompletely understood.

This work developed following reported high waterfowl deaths and illnesses in Spring 1982. Idaho Department of Fish and Game (Bruner 1982 personal communication) estimated that 200 tundra swans had died of a flight of 1,200. Approximately a dozen Canada geese also died. This was the first reported episode subsequent to implementation of a series of measures to reduce contamination of LCDAR that included measures such as settling ponds for mill tailings (1968) and taller smelter stacks (1974). In 1981, the lead smelter, electrolytic zinc plant, phosphoric acid and fertilizer plants, cadmium plant, and sulfuric acid plants at the Bunker Hill complex were closed (Savage 1986). Notwithstanding, these measures and in spite of the obvious changes in the nature and extent of contamination of the LCDAR, waterfowl remained at risk.

Nature of Studies

It is important to describe how the work reported here was performed,

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since it has substantial, but understandable, limitations in scope. At the outset, the mortality in the swans presented strictly a diagnostic problem. In early 1982, conversations with Robert White, a St. Maries veterinarian, and Al Bruner, a Manager with Idaho Department of Fish and Game, called the problem to the attention of Tony Gallina, Director, Washington Animal Disease Diagnostic Laboratory (WADDL), Washington State University. I served that Laboratory as a toxicologist and was responsible for the direction of the analytical chemistry section. Elemental analyses were performed at WADDL (atomic absorption) by Bill Warner and at the Agricultural Services Laboratory (inductively-coupled plasma emission spectroscopy, ICP) by Dale Marcy and co-workers. These details are important since my work was diagnostic rather than fundamental research. Research will continue to clarify issues and some critical work will be suggested at the end of this report.

1.0. Waterfowl Deaths and Illnesses Recurred in 1982

In 1982, the annual flight of 1,200 tundra swans was subjected to the harsh conditions of a North Idaho winter. Two hundred birds were estimated to have died plus an additional 10 Canada geese (Bruner, personal communication). Dead birds were observed between Rose Lake and Harrison, Idaho. Some were in good flesh, other were emaciated.

Doctor Robert White of the St. Joe's Veterinary Clinic contacted the Washington Animal Disease Diagnostic Laboratory concerning the deaths of the swans. Arrangements were made to obtain tissues and frozen birds from Al Bruner, Wildlife Land Manager, Idaho Department of Fish and Game. They both suspected lead poisoning and provided considerable insight into the problem which recurred periodically in the area. Mr. Bruner provided a set of livers for lead analysis.

1.1. Initial Observations

Initial necropsies were performed on a female swan collected by the author April 23, 1982, at the Washington Animal Disease Diagnostic Laboratory (82-4329) by Doctor Cho. He reported "Extreme emaciation; preventriculus and gizzard packed with grass; engorged gall bladder; empty GI tracts with bile". There were a few E. coli colonies isolated from the liver. Starvation was the diagnosis and tissue analyses revealed toxic levels of lead (Table 1) and the presence of other elementals including cadmium (Table 2). Lead poisoning was the diagnosis of Doctor Erik Stauber who made substantial contributions to early efforts to describe factors associated with the mortality of the swans and geese that occurred during the Winter 1982.

A complex of biological responses can be present in lead poisoning. Most body systems may be adversely effected including neurological, gathrointestinal, hematogical, endocrine, immunological, reproductive and renal systems. Under field conditions during the 1982 episode, neurologic responses including tremors, weakness, paralysis, and convulsions were observed. Elevated lead levels were observed in tundra swans and Canada geese which died (Tables 1, 2, 3, and 4). At necropsy some were severly

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emaciated whilst other birds in better flesh had proventriculi that were packed full of plants (notably Equisetum sp.) and dark grit.

Microscopic examination by Diane Stone of the Washington Animal Disease Diagnostic Laboratory of the kidneys of a bird which died in 1985 (Tables 5 and 6) revealed nephrosis with intranuclear inclusion bodies in karyomegalic proximal tubular epithelia (Stone, personal communication). A preliminary report of findings was presented at the 1986 meeting of the Society of Toxicology (Krieger et al. 1986).

The above observations were similar to several earlier reports. In 1931, the Bureau of Biological Survey reported that "the gross pathological appearances of the swans indicated metallic poisoning". Only two lead shot were recovered and they were not considered sufficient to cause lead poisoning. Excessive elementals were found in Food and Drug Laboratory analyses (all mg/g): lead 0.07-1.9; copper 1.2-1.9; and, zinc 14.0-27.0. The Bureau report concluded that it was conceivable that lethal concentrations of toxic metals could have been deposited on the aquatic vegetation upon which swans fed. This report is summarized by Chupp and Dalke (1964).

Chupp and Dalke (1964) noted that lead, zinc and copper were found on each sample but that quantitative comparisons are not possible since sample weights were not reported.

Adler (1944) reported tissue levels of lead in poisoned Canada geese. The birds had ingested three to 81 lead shot. Liver lead levels were 27, 20, 9 and 16 ug/g compared to two control birds with 0 and 1 ug/g. The necropsy revealed enlarged liver with greenish discoloration and disintegration, friable and ulcerated gizzard lining, dark caeca, enlarged spleen and kidneys, and watery, green feces. Similar observations were made in birds collected during the present investigation.

Similar findings have been reported by Bagley et al. (1967) following deaths of Canada geese in Delaware where the problem was judged to be a recurring one ascribed to lead shot ingestion. Protrusion of the nictating membrane and a high pitched call of ill birds were two unusual observations. Liver leads ranged from 6 to 20 ug/g in a 1965 collection and 12 to 53 ug/g later that winter in 1966. Tibia levels were 17 to 56 ug/g and 19 to 102 ug/g, respectively. Pen-raised geese had 0.4 to 0.8 ug lead/g liver (mean 0.5 ug/g).

Kendall and Driver (1982) reported severe pectoral muscle atrophy and bile stained feces, enlarged gall bladder, impacted proventriculus, and erosion of the gizzard lining in lead-shot poisoned trumpeter swans _Cygnus buccinator_ (3) and a junvenille whistling swan _Olor columbianus_. They had ingested seven to 68 lead shot and had liver levels (converted to fresh weight) of 22, 20, and 15. Each of these reports involved lead shot exposures rather than mortality and morbidity associated with sedimentary lead. Available measurements show a similar level of lead in poisoned geese and swans. It would be diagnostically helpful to include a tibia sample in future studies since the level may be used to indicate duration of the excessive exposure.

A series of 13 swans from the Mission Lake area between Kellogg and Coeur d'Alene were found dead and later analyzed by Benson et al. (1976). Lead shot was found in two of the swans. Liver levels ranged 7 to 43 ug/g and leg bone <1 to 94 ug/g. The respective means were 23 ug/g and 40 ug/g. Signs typical of lead poisoning were observed at necropsy. The authors ascribed the deaths to lead poisoning as a result of ingestion of lead contaminated vegetation (Tables 8 and 9).

There are few experimental studies of the dose-response relationship in lead poisoning. Coburn et al. (1951) used captured wild mallards dosed orally with an aqueous solution of lead nitrate $[Pb(NO_3)_2]$. Dosage levels were 3, 6, 8, and 12 mg lead nitrate/kg body weight which are equivalent to 1.9, 3.7, 5.0, and 7.5 mg lead/kg body weight. (If sediment from the Thompson Lake area contained 2500 ppm (ug/g) were used in similar studies and if all sedimentary lead were assumed to be "available" (dissolved), this dosage range would be equivalent to 1200, 2400, 3200, and 4800 mg lake sediment/kg body weight. These amounts of sediment are not unreasonably large with respect to amounts of sediment that could be ingested during feeding, but the assumption of 100 percent availability of lead is extreme.)

In Coburn's work, soluble lead was virtually all retained (>95 percent) in two birds each dosed at 8 and 12 mg/kg during the first week of the study. At later times (two to three weeks), the excretion rate was 43 to 80 percent of the administered dose.

Daily dosage of 6 mg/kg produced no toxic effects during a test period of 137 days. Total exposure of the two mallards was 441 and 628 mg. At the highest dosage (12 mg/kg), death due to lead poisoning occurred in 14 to 22 days. The toxic threshold appeared to be between 6 to 8 mg/kg/day. Small scale experiments in ducks could be used to determine the toxic threshold for sediments containing lead. A steep dose-response curve is an important characteristic of lead poisoning in waterfowl.

Tissue deposition at 8 and 12 mg/kg did not differ. Mean liver lead levels in treated ducks were 20.8 mg percent (dry; 52 ug/g) and 0.5 mg percent (dry; 1 ug/g). In unexposed controls, corresponding "skeleton" levels were 46.9 mg percent (117 ug/g) and 6.8 mg percent (7 ug/g). Even though the data were collected nearly 40 years ago, they remain very useful in consideration of the exposure and absorption of lead in ducks. Lead nitrate $[Pb(NO_3)_2]$ is unquestionably more available than in sediment lead, presumably PbS. The experimental approach used by Coburn et al. (1951) would produce valuable insight into the availability of lead in the River Basin.

Lead toxicity resulting from lead shot ingestion has been thoroughly documented dating from the extensive work of Bellrose (1959).

Table 1. Summary of Initial Analyses
Tundra Swan and Canada Goose Tissues: Lead Poisoning

Year	Lead Poisoned Birds Analyzed	Provent. Contents	Tissue Lead (ug/g) Kidney	Liver	Brain
1982	15	-	14-151(6)	6-30(15)	-
1984	3	5-76	100-167	15-34	2.3-4.2
1985	11	15-216	16-98	6-41	-

Acid digests (HNO₃) of bird tissues analyzed by inductively coupled plasma emission spectroscopy. The 1982 samples were part of a large series given to federal officials by Idaho Department of Fish and Game personnel. Results are not available. Number of analyses noted in parentheses.

Table 2. Elemental Analyses of Coeur d'Alene River
Tundra Swan Tissues, 1982

Sample	Tissue	Tissue Element (ug/g) As	Cd	Cr	Hg	Pb	Se	Zn	Cu
4880-Swan	Liver-1	ND	2.3	0.5	ND	18	ND	170	27
	-2	ND	1.8	0.6	ND	13	ND	247	54
	-3	ND	1.5	0.4	ND	15	ND	90	7
	Kidney-1	ND	2.8	0.2	ND	22	ND	55	20
	-2	ND	2.1	NS	ND	137	ND	188	17
	-3	ND	1.6	0.4	ND	34	ND	51	5
Proventriculus Contents		ND	3.6	2.5	ND	687	ND	NS	NS
-Goose	Liver-1	ND	3.8	0.4	ND	12	ND	313	33
	-2	ND	2.5	0.3	ND	8	ND	183	16
	Kidney-1	ND	14	ND	ND	10	ND	128	26
	-2	ND	9.4	0.3	ND	29	ND	122	28
4329-Swan	Liver-1a	ND	3.7	ND	ND	20	ND	333	33
	-1b	ND	3.1	0.3	ND	27	ND	203	118
	-1c	ND	4.1	0.6	ND	25	ND	206	267
	-1d	ND	2.3	ND	ND	25	ND	143	59
	-1e	ND	4.1	0.5	ND	27	ND	242	105
	-1f	ND	4.3	0.5	ND	26	ND	325	71
	-1g	ND	2.5	0.5	ND	20	ND	281	19
	-1h	ND	3.0	0.4	ND	32	ND	233	187
	-1i	ND	3.8	0.3	ND	40	ND	322	14
	-1J	NS	3.2	NS	NS	30	NS	288	9
	Kidney-1	NS	4.8	NS	NS	106	NS	135	17
Blank		<2	<0.1	<0.2	7	<1	<3	2	<0.01
Scan 5/17/82		No	Yes	No	No	Yes	No	Yes	Yes

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Table 3. Additional Elemental Analyses of Coeur d'Alene River
Swan Tissues, 1982

	Tissue Element (ug/g)					
	As	Cd	Cr	Hg	Pb	Se
4329-Proventriculus-1	ND	0.5	ND	ND	ND	ND
Contents-1[a]	ND	0.8	1.0	ND	72	ND
Proventriculus-2	ND	0.2	1.0	ND	ND	ND
Contents-2	ND	0.6	1.1	ND	4	ND
Proventriculus-3	ND	0.2	0.8	ND	ND	ND
Contents-3	ND	0.7	2.1	ND	·34	ND
Proventriculus-4	ND	0.5	1.2	ND	<1.2	ND
Contents-4	ND	1.1	2.1	ND	18	ND
Proventriculus-5	ND	0.9	1.0	ND	1.6	ND
Contents-5	ND	1.2	2.9	ND	16	ND
Proventriculus-6	ND	0.6	1.1	ND	ND	ND
Contents-6	ND	1.0	1.9	ND	24	ND
Proventriculus-7	ND	0.4	1.5	ND	<1.1	ND
Contents-7	ND	1.5	1.1	ND	75	ND
Proventriculus-8	ND	0.4	0.8	ND	ND	ND
Contents-8	ND	1.1	1.4	ND	29	ND
Proventriculus-9	ND	0.3	0.9	ND	ND	ND
Contents-9	ND	0.6	1.1	ND	223	ND
Blank	4.8	0.15	0.5	4.7	<1	7.6
Scan	No	Yes	No	No	Yes	No
5/24/82						

[a] Contents of each part of the proventriculus to detect whether "hot spot"
of an element was ingested. Contents includes plant, animal, and soil
constituting the ingesta.

Table 4. Duck Proventriculus and Content Analyses for Lead, 1982

| | Tissue Lead (ug/g) | |
Specimen	Proventriculus	Proventriculus Content
1	<1	72
2	<1	4
3	<1	34
4	<1	17
5	2	16
6	<1	24
7	<1	75
8	<1	29
9	<1	223

Samples supplied by Mr. Al Bruner, Spring 1982. Birds died earlier in season and were frozen prior to necropsy. Contents of the proventriculus were fluidized in some cases. In other instances, the crop was fully distended and contents in good condition. Mineral (soil) and plant material were present.

Table 5. Tundra River Swan Tissue Analyses, 1984-85
(ppm fresh weight)

| | | Tissue Lead (ug/g) | |
Date	Tissue	ppm	Location
5/4/84	Brain	3.5	Highway 97 dike near
	Proventriculus	76	Harrison, Al Bruner
	Kidney	167	
	Liver	15	
4/25/84	Brain	2.3	Highway 97 dike near
	Proventriculus	25	Harrison, Al Bruner
	Liver	15	
4/24/84	Brain	4	Unknown, Al Bruner
	Proventriculus	5	
	Kidney	100	
	Liver	34	
5/22/85	Proventriculus	0.5	Woodland Bottoms, John
	Liver	0.4	Nigh. Accidental death,
	Kidney	0.4	not lead poisoning.
5/22/85	Liver	25	Woodland Bottoms, John
	Kidney	49	Nigh.
5/22/85	Proventriculus	0.05	Woodland Bottoms, John
	Liver	0.3	Nigh. Accidental death,
	Kidney	0.06	not lead poisoning.

Table 6. Trumpeter Swan Tissue Analyses, 1985

			Tissue Lead (ug/g)						
	Kidney	Liver	Esophagus Contents			Proventriculus Contents			Gizzard Contents
Trumpeter Swan[2,3,4]			1	2	3	1	2	3	
Lower Coeur d'Alene (11-2-85; #15, male)	16	15	22	25	16	55	21	83	24
Lower Coeur d'Alene (11-2-85; #16, male)	21	20	303	-	78	-	42	-	90
Low lead environment[5] Bonner's Ferry[1] (12-6-85; #17)	0.3	0.5					-		
High lead environment Lower Coeur d'Alene River near Thompson Lake (11-9-85; #18)	18	19					19[6]		

[1] Esophagus and proventriculus each sectioned into three parts. Both parts of upper gastrointestinal tract were severly impacted with plant and mineral matter.
[2] Esophagus and proventriculus contained no lead shot (x-ray).
[3] Femur: #15, 20 ppm; #16, 21 ppm
[4] Collected dead by John Nigh, Idaho Department of Fish and Game
[5] Swan found by hunter (location unknown).
[6] No impaction of preventriculus or gizzard.

Table 7. Summary of 1985 Coeur d'Alene River Swan Tissue Analyses

| Sample | Tissue Lead (ug/g) | | | Comments |
	Liver	Kidney	Proventriculus[a]	
1	4.0	NS	NS	No necropsy
2	3.9	6.1	215.9	No necropsy
85-3	11.5	97.5	6.8	No necropsy
85-4	NS	NS	NS	Carcass in very poor condition.
85-5	7.1	22.6	NS	Pale kidneys, calcium plagues on epicardium.
85-6	5.1	NS	5.2	Fibrotic liver.
85-7	5.9	NS	14.5	Intestinal mucosa reddened, distended gall bladder.
85-8	19.6	NS	20.0	Emaciated; filarial worms on epicardium.
85-9	24.7	47.4	107.8	Found weak; emaciated.
85-10	41.2	80.5	42.0	Impacted proventriculus.
85-11	.6	.6	1.9	-
85-12	37.3	38.4	236.4	Moderate impaction of proventriculus; Whole Eq. present.
85-13	18.2	34.7	47.3	Emaciated; impacted proventriculus.

NS - no sample
[a] Swans collected by Idaho Department of Fish and Game dead in Thompson Lake area.
[b] Proventriculus contents.

Table 8. Lead Levels in Tissues from Swans Found
Dead, Mission Lake Area, Idaho, 1974[a]

Tissue	Range	Tissue Lead Mean Dry Weight (ug/g)
Bone	<1-94	40.4
Brain	<1-39	8.9
Feathers	<1-84	27.9
Flesh	<1-73	11.0
Heart	<1-28	8.7
Kidney	16-113	45.1
Liver	7-43	23.1
Spleen	<1-185	74.5

[a]Benson et al., 1976

Table 9. Coeur d'Alene River Tundra Swan Liver Tissue Analyses of
Idaho Bureau of Mines, 1971 and 1974

	Dry Weight Tissue Element (ug/g)		
Sample	Pb	Zn	Cu
1971			
A	4.2	19	
B	12.5	19	
C	2.1	13	
D	4.4	14	
E	4.3	28	"Insignificant
F	3.7	29	amounts"
G	5.6	23	
H	6.0	17	
I	5.7	21	
J	4.3	11	
Mean±S.D.	5.3±2.8	19.4±6.0	
1974			
1	17	460	140
2	33	435	37
3	21	332	159
4	22	585	100
5	35	456	390
6	15	720	8
7	17	105	86
8	22	412	15
9	29	252	189
10	11	255	28
11	43	5840*	13
12	31	19*	299
13	7	350	47
Mean±S.D.	23.3±10.4	396.6±167.8	116.2±118.6

*Exclude; outside 2 S.D.

This analysis of Idaho Fish and Game samples of swan liver was supplied by Mr. Al Bruner, Spring 1982. The work was apparently performed by the State of Idaho, Bureau of Mines and Geology. Method is included in Appendix I.

The liver data for 1974 were also reported by Benson et al. (Table 8).

1.2. Other Waterfowl Work

It is often suggested that high concentrations of lead in the liver are diagnostic of recent acute exposure to lead such as ingestion of spent lead shot (Scanlon et al. 1980; Dieter 1979). Scanlon et al. (1980) found liver leads of 43.2 ± 12.8 ppm dry weight in Mallard ducks positive for lead shot and 8.0 ± 1.6 ppm dry weight in ducks negative for lead shot. This would correspond in this report to 14 ug/g (fresh weight) for mallards with lead shot and 2.7 ug/g for mallards negative for lead shot. Liver leads exceeded 3.3 ppm in 29 percent of ducks with ingested lead shot. Scanlon et al. (1980) suggested that 2 ppm lead might represent the background level for ducks (white-winged scoter *Melanitta deglandi*) that have not ingested shot and whose diet is composed of invertebrates. Scanlon et al. (1980) clearly demonstrated the elevation of liver lead as a consequence of lead shot ingestion.

Longcore et al. (1974) determined that brain lead exceeding 3 ppm (fresh weight) and 6 to 20 ppm in kidney or liver were indicative of acute lead exposure. Other liver measures of toxic exposure include 23 ppm in bald eagle *Haliaetus leucocephalus* liver (Jacobson et al. 1977), 0.5 to 32 ppm lead in Canada geese *Branta canadensis* liver (Cook and Trainer 1966), and 0.6 to 20 ppm lead in livers of lead poisoned Mallard ducks *Anas platyrhynchos* (Longcore et al. 1974).

Delta-aminolevulinic acid dehydratase is an extremely sensitive indicator of excessive exposure. Finley et al. (1976) found that blood delta-aminolevulinic acid dehydratase (ALAD) inhibition occurred three weeks after feeding 25 ppm lead nitrate in duck breader mash. The 40 percent inhibition persisted through the 12 week experiment. Only 1 ppm lead accumulated in liver. After three weeks on a control diet, ALAD returned to control levels. This response contrasts sharply with that seen in ducks following ingestion of lead shot. Following ingestion of four shot ALAD was inhibited 88 percent and the effect persisted for three months (Finley et al. 1976). This work with mallard ducks cautions against overinterpreting field data and supports the need for controlled studies using sedimentary lead from the Coeur d'Alene River Basin. Canada goose feces (12) obtained by John Nigh, at Thompson Lake in 1989 and analyzed by the author, contained a mean of 85 ug/g lead. This amount is approximately 1/30th the content of sediments in the region. Whether there is a relationship between fecal excretion in geese and sediment levels is not clear, but the question invites study as an index of availability. Interpretation of these data would require knowledge of absorption and excretion kinetics that is not available at this time.

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Table 10. Coeur d'Alene Basin Duck Liver Levels of
Cadmium, Lead, and Zinc, 1986

Sample	Tissue Elements (ug/g)		
	Cadmium	Pb	Zn
1	0.2	1.0	160
2	0.1	-	74
3	0.1	-	90
4	1.0	3.1	168
5	0.5	-	165
6	1.0	-	85
7	0.8	2.5	298
8	2.0	3.9	599
9	0.5	-	438
10	[a]	-	141
11	0.4	1.3	114
12	0.4	8.4	70
13	0.5	1.2	58
14	1.0	2.4	55
15	0.4	11.9	70
16	1.1	18.0	65
17	0.9	2.1	55
18	0.7	6.1	65
19	0.5	1.2	42
20	0.4	-	55
22	0.4	-	50
23	0.6	3.6	68
24	0.4	1.6	55
25	0.9	6.1	60
26	0.4	10.4	88
27	0.3	13.6	55
28	0.6	4.0	71
29	0.2	-	80
30	0.2	-	154
31	4.2	10.5	77
32	0.2	2.6	41
33	1.3	12.1	84
34	0.8	8.3	85
35	1.4	4.4	62
36	0.3	3.4	49
37	0.8	6.9	43
38	0.9	3.1	42
39	0.8	3.1	76
40	0.7	12.9	54
41	0.3	3.7	44
42	1.2	10.3	63
43	0.8	27.8	105
44	0.3	1.7	62
45	0.5	4.1	44
46	1.6	4.0	45
47	0.4	5.9	55
48	0.1	2.2	41
49	0.5	2.0	49

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Table 10. Coeur d'Alene Basin Duck Liver Levels of
Cadmium, Lead, and Zinc, 1986 -   (Continued)

| Sample | Tissue Element (ug/g) | | |
	Cadmium	Pb	Zn
50	0.7	10.5	62
51	1.2	5.2	47
52	0.5	3.2	57
53	1.7	12.0	46
54	1.4	7.1	55
55	6.7	7.1	113

Idaho Department of Fish and Game, August 1986

[a] MDL <0.1 ug/g
[b] MDL <1.5 ug/g
[c] Inductively-coupled plasma emission spectroscopy (see footnote on Table 11).

Table 11A.   Coeur d'Alene Basin Duck Kidney Levels of
Cadmium, Lead, and Zinc, 1986

Sample	Tissue Element (ug/g)		
	Cadmium	Pb	Zn
1	0.2	2.7	74
2	_b	_c	23
3	0.1	1.9	41
4	1.2	3.5	62
5	1.1	1.8	35
6	1.2	-	32
7	1.6	3.5	77
8	1.8	3.9	111
9	-	-	26
10	-	2.5	43
11	0.4	2.0	47
12	0.8	7.0	28
13	2.7	2.1	38
14	1.9	3.3	30
15	2.3	11.9	31
16	6.9	14.9	32
17	5.5	2.0	42
18	5.4	6.8	36
19	5.5	-	36
20	0.5	-	28
21	NS[a]	NS	NS
22	0.4	2.4	21
23	1.8	3.6	37
24	1.3	2.6	28
25	1.3	7.0	30
26	NS	NS	NS
27	1.1	8.7	34
28	0.8	13.9	48
29	-	1.4	29
30	0.2	2.0	32
31	21.1	17.3	35
32	0.6	14.2	24
33	3.5	19.9	29
34	2.9	4.7	29
35	4.8	2.4	28
36	0.4	-	22
37	5.8	4.5	40
38	5.6	1.9	30
39	0.7	-	33
40	1.6	4.4	27
41	1.3	2.8	25
42	14.0	9.1	33
43	2.8	61.8	45
44	0.3	-	27
45	1.5	3.9	31
46	16.4	4.1	33
47	1.1	4.5	29
48	0.2	1.4	30

Table 11A. Coeur d'Alene Basin Duck Kidney Levels of
Cadmium, Lead, and Zinc, 1986 - (Continued)

Sample	Tissue Element (ug/g)		
	Cadmium	Pb	Zn
49	0.7	-	28
50	1.0	6.9	26
51	6.9	9.4	35
52	6.5	2.9	32
53	25.6	15.7	45
54	2.1	2.3	33
55	12.4	2.1	35

Idaho Department of Fish and Game, August 1986

[a] NS - no sample
[b] MDL <0.2 ug/g
[c] MDL <1.5 ug/g
[d] Inductively-coupled plasma emission spectroscopy, Washington Animal
Disease Diagnostic Laboratory, Toxicology Section, Washington State
University and University of Idaho.

Table 11B. Coeur d'Alene Basin Duck Tibia Levels of
Cadmium, Lead, and Zinc, 1986

| Sample | Tissue Element (ug/g) | | |
	Cadmium	Pb	Zn
1	0.4[a]	2.8	75
2	-	-	65
3	-	1.7	66
4	-	7.5	107
5	-	-	84
6	-	0.4	74
7	-	5.2	105
8	-	3.7	91
9	-	2.4	59
10	-	2.0	67
11	-	10.2	173
12	-	6.2	70
13	-	18.2	60
14	-	29.9	89
15	-	14.6	64
16	-	18.3	137
17	-	50.4	65
18	0.3	23.4	96
19	0.1	12.3	58
20	-	6.1	53
21	NS	NS	NS
22	-	14.2	50
23	-	11.4	101
24	-	7.8	65
25	-	112.0	75
26	0.6	15.4	43
27	-	3.9	46
28	-	37.2	86
29	-	2.2	61
30	0.1	-	57
31	0.2	63.6	93
32	-	2.9	46
33	0.1	15.1	70
34	-	16.2	71
35	0.1	16.8	82
36	-	10.8	50
37	0.1	41.2	49
38	-	11.8	60
39	-	-	56
40	0.2	9.8	60
41	0.1	19.6	62
42	0.1	28.1	62
43	-	36.0	57
44	-	2.2	66
45	NS	NS	NS
46	0.2	33.9	63
47	-	12.2	63
48	-	3.9	64

Table 11B. Duck Tibia Levels of
Cadmium, Lead, and Zinc, 1986 - (Continued)

Sample	Cadmium	(ppm) Pb	Zn
49	-	4.4	55
50	-	57.0	70
51	NS	NS	NS
52	-	13.7	62
53	-	29.7	50
54	0.2	30.5	58
55	0.1	28.2	85

Idaho Department of Fish and Game, August 1986

[a] Inductively-coupled plasma emission spectroscopy.
[b] MDL <0.4 mg/g (minimum detectable level).
NS - No sample

Table 12. Coeur d'Alene Basin Duck Breast Tissue Levels of
Cadmium, Lead, and Zinc, 1986[a]

	Tissue Element (ug/g)		
Sample	Cadmium	Pb	Zn
1	-b	-c	18
2	-	-	24
3	-	-	24
4	-	-	28
5	-	-	25
6	-	-	28
7	0.1	0.9	20
8	-	-	24
9	-		15
10	-	-	21
11	-	-	27
12	-	-	16
13	-	-	17
14	-	-	13
15	-	-	14
16	-	-	15
17	-	-	11
18	0.1	-	13
19	0.1	-	14
20	-	-	14
21	NS	NS	NS
22	-	-	13
23	-	-	14
24	-	-	13
25	-	-	12
26	-	-	15
27	-	-	14
28	-	-	15
29	-	-	19
30	-	-	30
31	-	-	14
32	-	1.6	10
33	-	-	10
34	-	-	11
35	-	-	12
36	-	-	11
37	-	-	16
38	-	-	14
39	-	-	26
40	-	-	16
41	-	-	11
42	-	-	16
43	-	-	15
44	-	-	16
45	-	-	14
46	-	-	17
47	-	-	14
48	-	-	12

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Table 12. Coeur d'Alene Basin Duck Breast Tissue Levels of
Cadmium, Lead, and Zinc, 1986 - (Continued)

Sample	Cadmium	(ppm) Pb	Zn
49	-	-	13
50	-	-	14
51	-	-	14
52	-	-	16
53	-	-	12
54	0.1	-	9
55	0.1	-	12

Idaho Department of Fish and Game, August 1986

a Inductively-coupled plasma emission spectroscopy.
b MDL <0.1 mg/g (minimum detectable level).
c MDL <1.0 ug/g
NS - No sample

Table 13. Lead Levels in Duck Liver, Kidney and
Cooked and Uncooked Breast, 1986

Sample	Liver	Kidney	Breast Uncooked	Breast Cooked
1[a]	1.5	2.2	0.5	<0.5
2[a]	0.8	0.7	0.4	<0.4
3[a]	10.7	11.4	0.4	<0.4
4[a]	5.5	8.1	0.3	<0.2
5[b]	3.6	3.4	0.4	1.3
6[b]	2.4	3.4	0.5	<0.3
7[b]	1.6	1.4	0.2	<0.2
8[b]	1.6	4.2	0.3	1.4
9[b]	5.1	1.5	0.8	<0.3
10[b]	9.4	2.1	0.4	<0.2

Idaho Department of Fish and Game, August 1986

[a]Inductively-coupled plasma emission spectroscopy.
[b]Atomic absorption spectroscopy.

All analyses performed at the Washington Animal Disease Diagnostic
Laboratory, Toxicology Section, Washington State University and University
of Idaho.

Table 14. Cadmium, Lead, and Zinc in Duck Liver, Kidney,
and Cooked and Uncooked Breast[a], 1986

| Sample | Tissue Element (ug/g) | | |
	Cadmium	Pb	Zn
Liver			
5	0.7	1.8	63
6	0.2	<1	51
7	0.6	<1	58
8	0.7	3.4	71
9	0.6	6.1	80
10	0.5	7.3	97
Kidney			
5	0.7	1.8	63
6	0.4	<2	24
7	1.1	<1	27
8	1.1	5.2	29
9	<0.4	<5	32
10	<0.4	<5	27
Breast/Cooked Breast			
5	<0.1/<0.1	<1/<2	15/25
6	<0.1/<0.9	<2/<1	19/27
7	<0.05/<0.09	<0.3/<1	6/42
8	0.1/0.1	1.5/2.2	15/29
9	0.2/0.2	<1/1.7	15/22
10	0.1/NS	<2/<1	18/31

Idaho Department of Fish and Game, August 1986

[a] Atomic absorption spectroscopy
[b] NS - no sample

Table 15. August 1986 Survey of Columbia Basin, Washington, Mallards: Lead

	Liver	Kidney	Breast	Leg Bone
		Tissue Lead (ug/g)		
1	1.1	-a	-b	1.4
2	-a	-	-	4.3
3	0.9	-	-	1.7
4	-	-	-	1.7
5	1.2	0.6	-	19.1
6	1.4	-	-	1.6
7	0.9	-	-	18.5
8	1.1	-	-	2.0
9	-	-	-	6.6
10	-	-	-	3.5
11	1.9	7.2	-	3.6
12	-	-	-	1.6
13	-	-	-	1.2
14	1.4	2.0	-	37.8
15	-	-	-	4.0
16	-	1.5	-	1.4
17	-	-	-	1.7
18	-	-	-	1.8
19	-	-	-	2.0
20	-	-	-	1.4
21	-	-	-	2.2
22	-	-	-	1.5
23	-	-	-	1.8
24	-	-	-	NS
25	-	-	-	2.1
26	-	-	-	1.5
27	-	-	-	8.3
28	-	-	-	NS
29	-	-	-	NS
30	-	-	-	1.3

a Minimum detectable level for liver and kidney, <0.4 ug/g.
b MDL <0.2 ug/g
c NS - No sample

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Table 16. Composite of 1983 and 1984
Oregon Duck Liver Leads: Savvie Island

Sample (Set 1)	Tissue Lead (ug/g)	Sample (Set 2)	Tissue Lead (ug/g)	Sample (Set 3)	Tissue Lead (ug/g)
1M	3.0	2P	-	57X	-
3M	5.7	4P	-	58X	35.7
6M	‑a	5P	-	59X	2.2
8M	-	7P	15.2	60X	64.8
9M	4.5	11P	9.6	61X	73.9
15M	2.5	14P	-	62X	62.9
17M	6.1	17P	9.2	63X	-
28M	70.4	20P	63.5	64X	-
29M	36.8	21P	26.4	65X	-
31M	-	21P	-	66X	-
45M	4.8	22P	-	67X	-
48M	-	27P	51.0	68X	17.4
51M	-	30P	7.0	69X	33.5
51M	-	31P	-	70X	-
55M	-	36P	-	71X	-
56M	-	39P	67.5	72X	6.1
62M	10.3	40P	22.9	73X	43.4
64M	8.6	47P	38.1	74X	57.7
64M	-	48P	-	75X	39.3
				76X	40.4
66M	-	50P	34.3	77X	37.3
68M	40.7	55P	44.0	78X	43.8
69M	31.5	56P	4.7	79X	34.8
70M	7.3	56P	-	80X	20.9
74M	30.2	57P	41.6	81X	63.1
76M	28.2	61P	2.0	82X	68.2
78M	-	62P	15.1	83X	1.5
79M	41.3	66P	-	86X	-
79M	12.2	67P	-	180X	47.7
81M	-	70P	-	181X	40.7
85M	-	72P	5.5	182X	15.6
87M	12.5	74P	9.0	183X	69.1
87M	-	75P	31.3	184X	-
92M	4.9	75P	16.8	185X	42.0
93M	-	76P	22.3	186X	103.5
96M	-	81P	9.3	187X	34.7
99M	39.6	87P	-	188X	63.6
99M	-	96P	4.5	189X	37.7
100M	43.1	97P	32.4	190X	52.0
		98P	3.2	191X	42.3
		99P	41.3	192X	12.7
				193X	54.8
				194X	103.2
				195X	35.1
				196X	70.3
				197X	-

Table 16. Composite of 1983 and 1984
Oregon Duck Liver Leads (ug/g): Savvie Island - (Continued)

Sample (Set 1)	Tissue Lead (ug/g)	Sample (Set 2)	Tissue Lead (ug/g)	Sample (Set 3)	Tissue Lead (ug/g)
				198X	37.3
				199X	-
				200X	25.4

Oregon Department of Fish and Game, Savvie Island Road, Portland Oregon

Note: Sets M, P, and X were collected at differt times. Set X contains livers from sets M and P.

a - sample at or below minimum detectable level, <1.5 ug/g

Table 17. Composite of 1984 and 1985 Oregon Duck Liver Leads:
Umatilla National Wildlife Refuge

Sample	Tissue Lead (ug/g)	Sample	Tissue Lead (ug/g)	Sample	Tissue Lead (ug/g)
118	- a	43	-	45	-
121	39.9	49	-	48	11.1
124	-	52	-	51	-
127	-	55	-	54	-
130	-	58	-	57	-
133	-	61	-	60	-
136	-	64	-	63	-
137	-	67	-	66	-
138	-	70	-	69	-
139	-	73	-	72	-
141	-	76	-	78	-
142	-	79	-	81	-
143	-	85	-	111	-
144	-	88	-	114	-
145	-	91	-	117	-
146	-	94	-	120	-
147	-	97	-	123	-
		100	-	126	-
		106	-	129	-
		109	-	132	-
		112	-	135	-
		115	-	138	-
				141	-
				144	-
				147	-

a - sample at or near minimum detectable level, <1.5 ug/g

Samples analyzed by the Washington Animal Disease Diagnostic Laboratory,
Toxicology Section, Washington State University and University of Idaho.

Table 18A. 1986 Early Hunting Season Waterfowl Survey in
Coeur d'Alene Basin: Liver Lead

Sample	Number	Tissue Lead (ug/g)
Medimont Station		
Widgeon Hen	1	1.7
	2	5.6
	3	8.5
Blue-Winged Teal	1	1.6
Mallard Male	1	22.0
	2	5.6
	3	23.0
	4	9.4
	5	11.0
	6	21.0
	7	8.4
	8	4.4
	9	8.5
	10	210.0
Mallard Hen	1	5.4
	2	4.1
	3	72.0
	4	0.9
Pintail Hen	1	1.3
	2	1.9
	3	2.9
Pintail Male	1	5.5
	2	5.4
	3	1.9

Table 18A. 1986 Early Hunting Season Waterfowl Survey
in Coeur d'Alene Basin: Liver Lead - (Continued)

Sample	Number	Tissue Lead (ug/g)
Wood Duck Male	1	6.6
	2	4.2
	3	3.2
	4	2.2
	5	7.8
	6	3.5
	7	10.9
Wood Duck Hen	1	5.2
	2	10.0
	3	6.5
	4	3.8
	5	3.7
	6	4.6
	7	11.0
	8	4.3
	9	3.2
	10	1.5
Green-Winged Teal Hen	1	5.5
	2	18.0
	3	3.9
	4	5.8
	5	6.0
	6	2.5
	7	1.4
	9	0.9
	10	1.4
Green-Winged Teal Male	1	3.7
	2	9.1
	3	6.2
	4	5.7
	5	4.0
	6	6.2
Green-Winged Teal	1	1.8
	2	1.7
	3	2.0
Pintail	1	4.4
	2	0.7
	3	0.6
	4	2.6

Table 18A. 1986 Early Hunting Season Waterfowl Survey in
Coeur d'Alene Basin: Liver Lead - (Continued)

Sample	Number	Tissue Lead (ug/g)
Killarney Lake (Continued)		
Coot	1	0.17
	2	<0.14
Widgeon	1	1.1
	2	0.7
Wood Duck	1	2.3
	2	3.3
	3	3.2
	4	2.1
	5	0.7
	6	2.7
	7	2.2
	8	3.9
	9	2.2
	10	1.7
	11	9.3
	12	11.0
	13	2.8
	14	14.0
	15	1.3
	16	2.0
	17	5.3
Mallard Male	1	5.0
	2	3.5
	3	1.0
	4	4.7
	5	4.2
	6	3.5
	7	0.9
	8	5.6
	9	5.2
	10	2.4
	11	41.8
	12	4.5
	13	1.4
	14	0.1
	15	0.9

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Table 18B.   1987 Early Hunting Season Waterfowl Survey in
Coeur d'Alene Basin: Liver Lead

Sample	Number	Shot	Tissue Lead (ug/g)
Killarney Lake			
Mallard	1	6	10.0
	2		2.4
	3		1.0
	4	1	2.7
	5		0.9
	6	1	12.0
	7		8.6
	8		3.4
	9		4.4
	10		3.5
	11		2.6
	12		6.6
	13		0.5
	14	1	5.6
	15		9.2
	16	3	17.0
	17		3.7
	18		1.8
	19	6	22.0
	20	2	4.9
	21		1.6
	22		2.1
	23		5.5
	24		0.6
	25		11.0
	26	3	16.0
	27		0.9
	28		0.5
	29		3.9
	30		15.0
	31	2	14.0
	32		2.6
Wood Duck	1		1.0
	2		11.0
	3		3.5
	4		5.1
	5		3.9
	6		0.9
	7		1.6
	8		0.8

Table 18B. 1987 Early Hunting Season Waterfowl Survey in Coeur d'Alene Basin: Liver Lead - (Continued)

Sample	Number	Shot	Tissue Lead (ug/g)
Killarney Lake			
Green-Winged Teal	1		6.1
	2		1.2
Gadwall	1		3.5
	2		1.5
	3		2.0
	4		1.7
Medimont Station			
Mallard	1	10	36.0
	2		7.9
	3	1	4.1
	4		0.7
	5	3	10.0
	6		3.0
	7		0.3
	8		3.6
	9	9	18.0
	10	6	38.0
	11		1.5
	12		4.0
	13		2.0
	14		1.5
	15		20.0
	16		4.6
	17		9.5
	18	6	32.0
	19		12.0
	20		6.5
	21		27.0
	22		4.1
	23	1	4.2
	24	1	7.1
	25	3	6.1
	26		1.9
	27		1.8
	28		7.0
	29		1.9
	30		1.5
	31		2.9
	32	9	8.4
	33		
	34		5.8
	35		1.5

Sample	Number	Shot	Tissue Lead (ug/g)
Medimont Station (Continued)			
Mallard	36	4	6.0
	37		6.6
	38		1.5
	39	2	5.9
	40		1.6
Green-Winged Teal	1		3.2
	2		10.0
	3		9.4
Pintail	1	5	13.0
	2		3.1
	3		5.2
	4		2.0
Wood Duck	1		6.0
Widgeon	1		0.7
	2		4.6
	3		0.6
	4		0.4
	5		0.4
Widgeon	6		5.7
	7		2.8

Tissues analyzed by Stan Casteel, Washington Animal Disease Diagnostic
Laboratory, for Idaho Department of Fish and Game. Samples provided by John
Nigh, Idaho Department of Fish and Game Land Manager.

Table 19. 1988 Early Hunting Season Waterfowl Survey in Coeur d'Alene Basin: Liver Lead

Sample	Number	Tissue Lead (ug/g)
Mallard (unclassified)	1	2.1
	2	4.6
	3*	13.3
	4	20.9
	5	0.6
	6	0.9
	7*	1.9
	8	1.1
	9	3.3
	10	1.2
	11*	2.4
	12	2.8
	13	0.6
	14	0.8
	15	1.1
	16*	0.8
	17	3.9
	18	1.2
	19*	2.9
	20*	11.9
	21	9.9
	22	7.6
	23	0.4
	24	2.3
	25	9.0
	26*	21.3
	27	3.7
	28	4.1
	29	4.2
	30	4.5
	31*	3.9
	32	3.8
	33	4.5
	34	3.4
	35	4.7
	36*	53.0
	37*	10.9
	38	3.1
	39	6.0
	40	1.3
	41*	34.9
	42	14.6
	43	4.3
	44	9.2
	45	15.0
	46	5.1
	47	12.0

Table 19.   1988 Early Hunting Season Waterfowl Survey
in Coeur d'Alene Basin: Liver Lead - (Continued)

Sample	Number	Tissue Lead (ug/g)
Mallard (unclassified)	48	5.2
Continued	49	9.3
Green-Winged Teal (unclassified)	1	10.3
	2*	3.7
	3	6.4
	4	0.9
	5	7.2
	6	16.0
	7	9.8
	8	2.4
	9	2.6
	10	3.7
	11	4.6
	12	6.5
	13	3.2
	14	0.6
	15	21.3
	16	5.0
Wood Duck (unclassified)	1	8.2
	2	0.9
	3	1.4
	4	8.6
	5	4.8
	6	4.0
	7	4.8
	8	5.3
	9	5.9
	10	9.5
Coot (unclassified)	1	3.4
	2	8.1
	3	4.3
Pintail (unclassifed)	1*	21.1
	2*	10.6
	3	2.7
	4*	27.6
	5	1.8
	6	3.3
	7	4.0

Table 19. 1988 Early Hunting Season Waterfowl Survey
in Coeur d'Alene Basin: Liver Lead - (Continued)

Sample	Number	Tissue Lead (ug/g)
Widgeon (unclassified)	1	14.3
	2	15.9
	3	1.5
	4	3.8
	5	1.5
	6	1.1
	7	1.4
	8	4.4
Blue-Winged Teal	1*	4.0
	2	3.2
	3	1.3
	4	4.5
	5	4.0
	6	7.5
	7	2.9
Shoveler	1	38.3
	2	1.0
Ringnecked	1*	20.9

Tissues analyzed at the Washington Animal Disease Diagnostic Laboratory,
Toxicology Section, Washington State University and University of Idaho.
Samples provided by John Nigh, Idaho Department of Fish and Game Land
Manager.

* Contained unspecified number of lead shot.

Table 20. 1988 Early Hunting Season Waterfowl Survey:
Liver and Leg Bone Lead

Species	Number	Liver (ug/g)	Leg Bone (ug/g)
Mallard	49	7.1±9.4	13.5±16.6
Green-Winged Teal	16	6.5±5.6	33.9±38.3
Wood Duck	10	5.3±2.9	23.1±26.3 (N=11)
Coot	3	5.3±2.5	4.4±2.4
Pintail	7	10.1±10.3	17.1±29.2
Widgeon	8	5.5±6.1	4.6±3.3
Blue-Winged Teal	7	3.9±1.9	11.9±7.0
Shoveler	2	38.3 1.0	2.8 1.0
Ring-Necked Duck	1	20.9	19.4

Analyses completed by Stan Casteel, University of Idaho, for Idaho Department of Fish and Game, 1988. An informal report to the Idaho Department of Fish and Game also lists cadmium, zinc, copper, and silver.

2.0. Lead and Other Elementals in the Environment

The overwhelming majority of exposures of living things in the Coeur d'Alene River Basin to toxic substances such as lead and cadmium are apparently benign. Under exceptional circumstances excessive exposures occur and harmful biologic effects (see 1.1., 1.2., 3.1. - 3.7.) may occur. There is no doubt that potentially excessive levels of lead and cadmium are present in the Basin. Processes and pathways which make the elementals available for absorption by plants, animals, and humans are not well described. Knowledge of mechanisms might enable development of land management strategies which minimize exposures.

The data reported in the subsequent sections provide evidence that the exposure potential is lower today than in times past.

The studies are not presented as a comprehensive study of Basin lead. The work was limited in scope and was done to better understand the potential exposures of animals.

2.1. Soils

A recent USEPA report (Hornig et al. 1988) lists lead, cadmium and zinc levels in Coeur d'Alene Basin sediments (Table 21). Levels for the North, Central, and South parts of the Lake are included as well as Anderson and Thompson Lakes. The latter contained 2492 and 3386 ug lead/g, respectively. Data shown in Table 22 show similar lead levels for various sites including Thompson River (3177 ug/g), Killarney Lake Channel (4522 ug/g), Hidden Island Channel (7376 ug/g) and Blue Lake River (2576 ug/g). Those levels measured during 1985 are typical of values recorded during other aspects of the study. They represent the amounts of lead present in the upper part (zero to six inches) of the soil column.

No attempt was made to sample a particular soil horizon and in general soils were dark and heavy. Lighter colors were observed lower in the soil column in areas where digging had occurred.

Flood sediment. Analyses of flood sediment collected from boat launches near Thompson Lake and Swan Lake show that the River is carrying substantial amounts of lead solids (Table 22). In 1985, the deposits contained 4109 ug/g (mean, N=3) and 3572 ug/g, respectively, at Thompson and Swan Lakes. Spring flood sediments might be profitably monitored over time to obtain information about current movements of lead in the Basin.

The Bunker Hill Superfund Task Force reported lead, cadmium and zinc levels in yard soils and house dusts. Amounts measured usually range over an order of magnitude. The yard soils lead levels at Superfund sites (Table 24) are similar to lead levels in Basin sediments (Table 21) reported by Hornig et al. (1988) and in various measurements made during this work (Table 22 and others). The higher levels (lead 30,000 ug/g; cadmium 205 ug/g) reported in the Task Force report have not been observed and may be characteristic of conditions not present in the sediments of the land along the River.

Probably no part of the basin environment is without detectable and relatively elevated lead levels. The ubiquitous nature of this persistent environmental pollutant hardly needs reconfirmation. Similarly, lead is well know as a "toxic heavy metal" in excessive amounts. Dose-response relationships are established for a variety of toxicological endpoints including neurotoxicity, renal toxicity and immunotoxicity. The risks associated with the various hazards known to be linked to excessive lead exposure will be determined by dose. Dose, in turn, will be determined by the availability of lead in sediments (soil), water, plants and even salt incrustations.

Table 21.   Summary of Lead, Cadmium, Zinc Levels in
Coeur d'Alene Basin Sediments

Location	Sedimentary Elements (ug/g)		
	Lead	Cadmium	Zinc
Lake Coeur d'Alene Northern Part	1146-5732	7-8	16-87
Central Part (Coeur d'Alene River Delta)	4158	8	3680
South Part	10-367	0.6-10	77-1310
Lateral Lakes			
Anderson Lake	2492	10	2180
Thompson Lake	3386	9	2560
Lower Coeur d'Alene River	2310-3992	5-8	50-90
South Fork Coeur d'Alene River	298-7897	3-5	0.1-232
U.S.A. Median (Lyman et al. 1987)	16	1	4
95 Percent of U.S.A. sediments below	199	12	39

Hornig et al., USEPA, 1988

Table 22. 1985 Sedimentary Lead Levels

Sample	Sedimentary Lead (ug/g)	Mean
CDA River WMA #7	3489	2853
	2447	
	2624	
Thompson River Bank #6	4405	3177
	2281	
	2845	
Hidden Island Channel River Bank #2	6130	7376
	7287	
	8712	
Killarney Lake Channel #1	4857	4522
	5502	
	3207	
Blue Lake River Bank #5	2447	2576
	2688	
	2594	
Blessing Slough #3	3223	3499
	3279	
	3996	
Swan Lake Channel #4	4145	3814
	3993	
	3305	
Osprey #1	3073	3117
	3555	
	2724	
Springston #1	5803	6525
	7116	
	6654	
Springston #4	15	15
	14	
	16	

Idaho Department of Fish and Game

Samples analyzed by the Washington Animal Disease Diagnostic Laboratory, Toxicology Section, Washington State University and University of Idaho.

Table 23. Coeur d'Alene River Lead During
Spring Flood, 1985

Sample	1985	River Lead Nanograms/ml
1	3/17	10
2	3/21	40
3	4/06	10
4	4/12 (Early Flood Stage)	-[a]
5	4/14	230
6	4/16	50
7	4/18	-
8	4/20	-
9	4/22	-
10	4/24	-

Surface water samples collected by the Idaho
Department of Fish and Game near boat launch near
southwest outlet of Thompson Lake.

Grab samples of surface water were stored in
plastic bags.

[a] - MDL approximately 10 ng/g assuming 1g/ml
water.

Analyzed by the Washington Animal Disease
Diagnostic Laboratory, Toxicology Section,
Washington State University and University of
Idaho.

Table 24. Elementals in Yard Soils and House Dusts at
Bunker Hill Superfund Site

	Lead (ug/g)	Cadmium (ug/g)	Zinc (ug/g)
Yard Soils	2,500-30,000	28-205	999-8109
House Dusts	1,800-36,000	19-602	1118-20545

Presentation to Bunker Hill Superfund Task Force, July 28, 1988

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2.2. Water

Water from a small pond on the southeast edge of Thompson Lake was analyzed for lead. The sample contained 20 ppb or less. Sediment in close proximity contained 2488 ug/g. The pond was tested because there was a very large amount of sediment in contact with standing water. In spite of the apparent close contact, there was not extensive accumulation of lead in the small pond. Plans to sample the invertebrates in the pond were not further developed on the basis of the low amount of lead present in the water.

During the March-April period of 1985 when the River was high, John Nigh, Idaho Department of Fish and Game collected a series of water samples. We were interested to see whether those flood waters would cary high levels of lead. Nine samples from March 17, 1985 to April 24, 1985 were analyzed and found to contain zero (MDL about 10 ug/ml) to 230 ug/ml (Table 23). This series of samples provides inadequate evidence of elevated lead associated with the spring flood even though the two highest levels were found during the early part of the flood period. The data are very limited in scope and extent and certainly do not rule out short term changes that could be biologically or environmentally significant. More extensive time-concentration data could clarify the relationship between flooding and lead.

A site investigation conducted for the Bureau of Land Management in 1988 reported a remarkably high level of lead in Thompson Lake. A sediment sample contained 2730 ug/g and the water sample 7 mg/liter. The report states: "The water sample contained lead and zinc above the analytical detection limit - 7.0 mg/L and 0.246 mg/L respectively." It seems unusual to refer to such an <u>extremely high</u> number as being "above the analytical detection limit" unless reference to the upper limit is intended. As noted in the draft report, the value is 350,000 times above the proposed recommended maximum contaminant level, 5,384 times the chronic toxicity criteria, and 206 times the acute toxicity criteria. A sampling or analytical error seems likely.

Due to the extremes reported and the seeming incompatibility with earlier findings, a preliminary survey of water levels was made in July 1989. Results listed in Table 25 include distilled water purchased in the Basin, Killarney Lake, Thompson Lake, Coeur d'Alene River near the boat launch near Thompson Lake, Wildwood Ranch (well) near Thompson Lake, Harrison City Hall, St. Joe River in St. Maries, and the Titus residence in Saint Maries. The levels found in Thompson Lake were less than one-one thousandth (<1/1000) of those reported in the report. Levels were at or near the minimum detectable levels in the distilled water, Wildwood Ranch well water, and in the pair of samples from St. Maries. Killarney Lake (0.013 ug/ml) and Thompson Lake (0.006 ug/ml) waters contained less than Coeur d'Alene River water (0.055 ug/ml).

A carefully developed surface water sampling plan could provide useful management data concerning the magnitude of seasonal changes in water lead and other elementals. A sampling program has recently been initiated by the Bureau of Land Management (Fortier, personal communication).

The discussion of Thompson Lake lead and the <u>single</u> datum reported are not adequate. More analyses may be indicated but not based on that piece of information.

2.3. Plants

There are several observations which lead to my opinion that elementals in the sediment have very low mobility and bioavailability.

1.   Very high lead levels exist in the sediments in stable form as evidenced by the massive expanse of contaminated sediments that have been deposited over decades.

2.   The lead sediments are not being mobilized in substantial amounts by plants such as horsetail *Equisetum* sp. Note: Lead-contaminated soils may be physically moved by wind and water erosion onto plant surfaces in insignificant amounts under natural conditions. Sampling of vegetation may be complicated or compromised by transfer of soil to plant surfaces.

3.   *Equisetum* sp. removed from the proventriculus of swans at necropsy apparently contained high lead levels. Follow-up studies using plants grown in high lead soils in a variety of growth stages did not contain high lead residues. The elevated levels in the necropsy materials likely resulted from surface contamination within the proventriculus rather than from environmental sources *per se*.

4.   Water potatoes *Sagitarea cuweata* are common throughout the Basin and they are among the plants eaten by muskrats. Since data concerning liver levels in muskrats were obtained, a series of water potatoes were also analyzed. Lead, cadmium, and zinc were tested in potatoes and the sediment in which they were growing.

An additional concern was the possibility that humans would consume water potatoes as part of their diet. Some relevant experimental data were obtained to determine the relationship between plant and soil levels of lead.

The potatoes were washed free of all visible sediment in the laboratory using a light brush. In our judgement, normal food preparation of vegetables taken from the ground would have involved similar cleaning. Nine samples of sediment and six potatoes were analyzed from each of the nine sites.

The levels of lead, cadmium, and zinc showed considerable variability (Table 26). Elevated lead levels were present in all soil and potato samples. Individual potato samples ranged from <4.7 to 810 ug/g lead. The range of cadmium concentrations was less extreme.

These data indicate a high relative exposure potential associated with muskrat feeding or human eating of water potatoes, however, risk can not be established because of the extent of actual exposure. The fact that muskrat livers contained modestly elevated liver levels of lead may indicate either limited ingestion and/or limited availability of lead.   It would be of interest to monitor muskrat kidneys to determine status of cadmium -- the toxic elemental that is stored in kidney.

Native Americans have formally consumed substantial amounts of water potatoes.   During earlier times of extreme pollution of the Basin, the

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potatoes acquired an unacceptable "metallic" taste and could not be eaten. The extent of current risk would require a complete understanding of their status in the diet and methods of preparation.

Most plants will absorb some elements to excess if present in an available form in the soil (Table 27). Any process such as microbial activity or acidic water seeps that might make an elemental such as lead more available could more heavily thus contaminate plants growing in the same environment. Although no evidence of such an occurrence was found in the course of sampling to date, the unlikely possibility that plants per se could be important in particular episodes of waterfowl poisoning must remain open.

The concentrations of nitrogen, phosphorus, potassium, calcium, magnesium, sodium, and a number of trace elements including lead were determined in bracken fern Pteridium aquilinum fronds and rhizomes. Considerable differences were found among the trace elements. Generally, the trace element concentration increased during the growing season. Lead, for example, increased from 5.1 (ppm in dry matter) to 10.7 ppm from May 15 to October 5. Our sampling of horsetail fern usually occurred in the summer, intermediate between the test periods in the earlier studies with bracken.

Our studies with horsetail at later times during the year showed higher levels of lead, but not toxic ones per se. Equisetum does not have a propensity to absorb large amounts of lead. Certain forms of lead, perhaps even fine particles of lead sulfide, made be readily adsorbed to plant surfaces. That mechanism is known and probably accounts for the high leads registered on the necropsy samples, some plant samples, and Equisetum (Tables 27 and 29) and wild rice (Tables 27 and 30).

Toxicological research on the lead-plant relationship is important. On the one hand, contamination of plant samples can lead to unreliable data concerning the concentrations of elementals. At the same time, the laboratory artifact can be pointing the way to mechanism of particle adsorption by food plants.

The subject deserves further study to define how important this transfer process could be, particularly under conditions where the soil has been disturbed.

Mitchell and Reith (1966) have also reported a marked seasonal difference in the lead concentration of pasture plants. During rapid early growth, plants contained 0.3 to 1.5 ppm. In the case of Equisetum in the present study, only early season growth has been considered a potential lead source based upon observations of equisetum in the proventriculus of poisoned swans at necropsy. That early season growth is less likely to contain high lead concentrations as a result of absorption. Waterfowl exposure to adsorbed lead on plant surfaces is likely but its quantitative importance can not be ascertained with available data and insight.

A series of Equisetum shoots were collected by John Nigh, Idaho Department of Fish and Game, along the southeast edge of Thompson Lake to determine whether lead was loosely associated with plant surfaces (Table 27). Young Equisetum shoots had been collected from the proventriculus of a

swan necropsied in 1985. The shoots (five sets each) were either washed or unwashed to determine whether lead was adhering to the plant cuticle. Unwashed plants contained 108 ug lead/g dry weight (range 57 to 175) and washed plants also contained 108 ug lead/g dry weight (range 61 to 214). A second tap water washing was performed on a third set of plants and the mean lead content was 89 ug/g dry weight (59 to 104). Surface soil at the site contained 2488 ug/g lead. Dried Equisetum at the same site contained 179 ug/g perhaps indicating more prolonged contact with environmental lead or breakdown of plant parts not containing lead. The lead levels of Equisetum collected at five other places were 78, 69, 42, 17, and 38 ppm.

Since Equisetum is a potentially important foodplant, it is important to summarize observations concerning its lead content. The hardy marsh plants thrives in soils that contain several thousand ug/g lead. Roots apparently washed free of adhering soil still contained extremely high lead levels, but lead was not apparently transported to the aerial parts of the plant to an appreciable extent. Dried plant parts had higher lead levels due to loss of water. There is no evidence that Equisetum has a special capacity to concentrate and transport lead. Since contaminated soils can adhere to the plant under certain conditions, it is possible that some plants might expose foragers to elevated and even toxic lead levels. This possibility is hypothetical but warrants consideration when Equisetum is discovered by necropsy of waterfowl or other wildlife.

Table 25. Preliminary Water Levels (July 1989) of Lead from
Coeur d'Alene River Basin

| Sample | Preliminary Lead (ppm) | | | |
	1	2	3	Mean
Distilled water	0.004	0.003	0.004	0.003
Killarney Lake[a]	0.015	0.014	0.009	0.013
Thompson Lake[b]	0.006	0.006	0.006	0.006
Coeur d'Alene River[c]	0.060	0.056	0.048	0.055
Wildwood Ranch[d]	0.000	0.008	0.001	0.003
Harrison City Hall[e]	0.000	0.000	0.000	0.000
St. Joe River[f]	0.000	0.000	0.000	0.000
Titus residence[g]	0.000	0.002	0.000	0.001

Krieger, 1989

Levels of 0.005 ug/ml are near limit of detectability (approximately 0.002 ug/ml). All samples obtained in triplicate July 24, 1989. ppm = ug/ml.

[a] Public boat launch (Township 49N Range 2W Sec 10, 11, 13, 14: Boise Meridian).
[b] Midpoint along north shore (49N 3W 21).
[c] 100 yards upstream from boat launch on channel from Thompson Lake.
[d] Well approximately 300' up mountain from site (3); about 1/4 mile from Thompson Lake shore.
[e] Harrison, Idaho.
[f] St. Maries, Idaho. Public boat launch in Aqua Park.
[g] Two miles west of St. Maries; 250 foot well (46N 2W 20).

Table 26. Lead, Cadmium, and Zinc in Water Potato and
Corresponding Sediments

Sample		Tissue Elements (ug/g)		
		Pb	Cd	Zn
Water Potatoe				
1	Mean ± SD	411 ± 220	3.2 ± 1.4	367 ± 127
2		463 ± 236	1.2 ± 0.7	242 ± 93
3		30 ± 23	1.4 ± 1.0	155 ± 155
4		121 ± 97	1.2 ± 0.6	165 ± 77
5		18 ± 16	2.6 ± 3.6	150 ± 134
6		14 ± 6	0.5 ± 0.3	28 ± 6
7		254 ± 131	1.3 ± 0.7	272 ± 99
8		104 ± 79	1.3 ± 0.8	79 ± 39
9		19 ± 14	1.1 ± 0.5	45 ± 25
1	Median	397	3.0	358
2		460	0.9	240
3		33	1.1	116
4		88	1.3	159
5		11	0.9	97
6		13	0.4	25
7		248	1.4	284
8		84	1.2	78
9		17	1.0	46
Sediment				
1	Mean ± SD	6364 ± 834	52.5 ± 19.7	4336 ± 549
2		5551 ± 577	32.1 ± 7.6	4259 ± 731
3		868 ± 366	13.5 ± 3.9	1076 ± 263
4		2596 ± 1399	18.6 ± 9.9	1907 ± 870
5		342 ± 115	5.6 ± 0.8	272 ± 108
6		327 ± 136	7.4 ± 1.4	305 ± 125
7		407 ± 45	83.6 ± 18.6	620 ± 227
8		2125 ± 48	30.4 ± 13.7	243 ± 60
9		617 ± 307	8.5 ± 1.7	593 ± 212
1	Median	5975	50.6	4254
2		5559	33.3	4215
3		911	12.4	1035
4		2863	16.5	1916
5		329	5.7	234
6		327	7.2	296
7		413	32.6	716
8		406	26.8	238
9		612	7.9	618

Idaho Department of Fish and Game
Bureau of Indian Affairs

Table 26. Lead, Cadmium, and Zinc in Water Potato and
Corresponding Sediments - (Continued)

Median Values		Water Potato	Sediment
Regression Output:		**Lead**	
		397	5975
Constant	240.6148	460	5559
Std Err of Y Est	1302.725	33	911
R Squared	0.723306	88	2863
No. of Observation	9	11	329
Degrees of Freedom	7	13	327
		248	413
X Coefficient(s)	11.27273	84	406
Std Err of Coef.	2.635233	17	612
		Cadmium	
		3	50.6
Constant	0.296566	0.9	33.3
Std Err of Y Est	9.913833	1.1	12.4
R Squared	0.634868	1.3	16.5
No. of Observations	9	0.9	5.7
Degrees of Freedom	7	0.4	7.2
		1.4	32.6
X Coefficient(s)	16.99383	1.2	26.8
Std Err of Coef.	4.871076	1	7.9
		Zinc	
		358	4254
Constant	-182.955	240	4215
Std Err of Y Est	1143.097	116	1035
R Squared	0.572235	159	1916
No. of Observations	9	97	234
Degrees of Freedom	7	25	296
		284	716
X Coefficient(s)	10.81154	78	238
Std Err of Coef.	3.533081	46	618

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Table 27. Preliminary Soil and Plant (Dry) Survey at Thompson Lake, 1985

Sample	Lead (ug/g)			Mean
Site 1 Springston				
Mud/silt	1980	1627	1245	1618
Reed/Grass (not identified)	1.9	1.3	1.3	1.5
Site 2 Springston				
Deep roots and silt "a little silt"	3443	3167	3533	3381
Equisetum roots		349		349
upper parts	5.1	5.0	3.9	4.7
Site 1 Osprey				
Silt/roots	2076	3000	1815	2297
Equisetum roots	1992	2112	2049	2051
upper parts	13	13	12	13
Reed/Grass (not identified)	12	7	8	9
Rice cutgrass (Leersim oryzoldecs)	39	30	25	31
Site 2 Osprey				
Silt/roots	2486	3347	3734	3222
Equisetum roots	1573	2207	2003	1928
upper parts	2.3	2.7	2.4	2
Wild rice roots	2.8	3.6	1.5	3
upper parts	638	833	992	821
Site 3 Osprey				
Silt/roots	2551	2302	2497	2450
Equisetum roots	not sampled			
upper parts	10	6	7	8
Wild rice upper parts	2.2	1.9	2.4	2

Collected and analyzed by the Washington Animal Disease Diagnostic Laboratory, Toxicology Section, Washington State University and University of Idaho.

Table 28. Vegetation (Dry Weight) and Soil Lead From Deltas of
St. Maries and South Coeur d'Alene Rivers, 1986

	Sample	Vegetation Lead (ppm)	Soil Lead (ppm)
St. Maries River			
A	Grass-1	<4	NA
		<4	
		<4	
B	Reed	<4	NA
		<4	
		<4	
C	Grass-2	<4	29
		<4	35
		<4	44
South Coeur d'Alene River			
D	Grass-3	20	3193
		19	2316
		15	2814
E	Grass-4	21	NA
		18	
		20	
F	Grass-5	24	NA
		43	
		72	
G	Grass-6	35	1743
		141	2147
		48	2113
H	Grass-7	17	2813
		12	3638
		112	3280
I	Grass-8	12	3501
		9	3181
		8	3558
J	Grass	6	2613
		12	2121
		13	2434
K	Reed	<4	2763
		<4	2617
		5	3222
L	Reed	4	2695
		9	2947
		9	3062

Idaho Department of Fish and Game

Analyzed by the Washington Disease Diagnostic Laboratory, Toxicology
Section, Washington State University and University of Idaho.

Table 29. July 1989 Survey of **Equisetum** at Thompson Lake

Sample	Collection Depth (inches below surface)	Lead (ug/g) Wet	Dry
1[a]	6	11	116
2[a]	18	7	26
3[a]	18	2	8
4[b]	26	2	7
5[b]	36	1	16
6[b]	30	3	30
7[b]	32	10	85
8[a]	12	3	1
9[a]	12	3	1
10[a]	6	2	16

Krieger, 1989

[a] Sample collected by John Nigh, Idaho Department of Fish and Game, four to six inches above marsh bottom.
[b] Samples collected 12 inches below water surface in deeper water.

Table 30. July 1989 Survey of Wild Rice at Thompson Lake[a]

Sample	Lead (ug/g) Wet	Dry
1	0.4	3.8
2	0	0
3	8.5	28.5
4	1.1	4.5
5	0.7	14.4
6	0.2	1.7
7	4.2	64.0
8	1.5	25.8
9	1.3	28.5
10	1.9	12.5

Krieger, 1989

[a] Samples collected by John Nigh, Idaho Department of Fish and Game.

2.4. Salt Incrustations

Ellis (1938) in his report "Pollution of the Coeur d'Alene River and Adjacent Waters by Mine Wastes", noted the presence of salt incrustations on the banks and flats of the River. A portion of Ellis' text is quoted below:

> "Many areas, some of them acres in extent, along the banks and flats between Cataldo and the mouth of the Coeur d'Alene River were covered at the time of this survey (July, 1932) with crustations of a crystalline material varying from pure white to dull gray or dirty brown in color. This material, as has been discussed in the section on bottom and shore conditions (page 64) was always found on deposits of mine wastes and mine slimes, or in soil heavily impregnated with these slimes. Careful investigation in the field left no doubt but that these incrustations had been produced by the weathering of the exposed mine wastes which are subjected to a variety of conditions conducive to chemical changes as moisture, light, heat and oxidation."

Incrustations from Thompson Flat and Bradley were analyzed by the United States Department of Agriculture and Bureau of Fisheries personnel during 1932 and 1933. The total elemental composition was reported (Table 31). Physically similar (but much smaller in size) incrustations were noted during present studies. These, too, were analyzed (Table 31). The analysis was done due to the very extensive distribution and potentially toxic nature of the earlier salt incrustations. At the present time, it is quite clear that the salts that currently form on the soil surface contain substantially lower amounts of lead and other elements (assuming chemical analyses to be comparable). Samples designated "clean-1" and "clean-2" were collected near Thompson Lake within 300 yards of each other. Their similar chemical composition is perhaps reflective of similar environmental conditions at each site. Other elements are reported in the legend of Table 31. The incrustations could present significant hazard to wildlife since they represent a readily available source of salts, but the risk seems small due to their small size and very limited distribution in the environment.

Table 31. Elemental Analyses of Salt Incrustations

| Sample | Micrograms/Grams (ppm) | | | | | |
	Zinc	Lead	Iron	Manganese	Copper	Arsenic
Thompson Flat and Bradley (1931-32)[a]	50-120,000	3-8,000	90,000	40,000	Trace	Trace
Thompson Lake (1985)[d]						
salt/"clean-1"[b]	22	12	234	52	0.4	<2
salt/soil[b]	207	134	2137	468	3.4	<2
salt/"clean-2"[c]	33	17	294	80	0.5	<2

[a] Ellis, 1938.

[b] Also Cd 0.2 and 2.1; Ca 119 and 111; Mg 3716 and 791; Na 61 and 139; K 7.8 and 34, respectively. Se, Tl, Hg analyzed but not detected.

[c] Also Cd 0.4; Ca 421; Mg 5607; Na 71; K 15. Se, Tl, Hg analyzed but not detected.

[d] Krieger, 1985.

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3.0. Lead and Animals

Excessive lead exposures were once relatively common in the Coeur d'Alene Basin, but today as a result of natural chemical and biological processes such excesses occur much less frequently. Those processes are poorly understood, virtually unknown, in molecular terms. The early description of Ellis (1938) depicts the physical deposition process and early biological consequences:

"the rock powder deposited on their lands by the river, that is the mine wastes, was of a highly toxic nature and that normal vegetation, crops and hay were killed out and that the stock particularly horses, and to some extent cattle, dogs and chickens were killed by the substances left upon the grass and along the shore of the river after the water receded.

and Ellis continued...

... the substances brought down from the mines and deposited by the river on the lands, produced certain toxic substances when exposed to the air, for which the stock, particularly the horses, developed a fondness."

3.1. Cattle and Horses

Feed contaminated via surface deposition of lead has resulted in losses of cattle and horses (Hammond and Aronson 1964). More recently elevated lead levels on foliage near urban highways have been recognized and studied to generate understanding of lead-plant relations (see Nriagu 1978). In general, lead is poorly systemic in plants. Chronic toxicity in horses in the vicinity of a smelter due to atomospheric deposition of lead has also been reported by Knight and Burau (1973).

Foliage levels of 150 ppm or more are associated with toxicity in livestock (Hammond and Aronson 1964). Burrows and Borchard (1982) diagnosed lead toxicity in horses in the Kellogg area. Several follow-up studies which confirmed the toxicity of lead to cattle and horses were performed.

Earlier Washington State University undergraduates surveyed lead in 118 horses and eight cattle in the Kellogg area of North Idaho. Less than 2 ug/ml lead was found in water from the region (Burrows et al. 1981).

The findings are summarized in Table 32. Also listed are regional norms published by Puls (1981) that are widely used in veterinary and animal science (Table 33). No horses with signs of lead toxicity were observed. One horse with a high lead level (0.70 ug/ml) had previously received chelation therapy.

Laboratory studies performed at Washington State University (Burrows and Borchard 1981, 1982) included lead acetate feed of three ponies and five calves (62.8 and 94.2 mg/kg/day). At the higher dosage, all calves died in four days or less. In the second trial, anorexia, depression, ataxia, circling and blindness wee seen in less than seven days. One calf survived

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beyond seven days following treatment with calcium EDTA (6.6 percent; 1 ml/2 lbs. body weight, i.v.).

The condition of ponies gradually declined associated with decreased feed and water consumption and hind limb ataxia. Apparent pharyngeal paralysis in the more advanced stages of the test feeding (seven to 10 days before death). Excessive salivation and nasal discharge related to difficulty in swallowing were observed. Two ponies were fed 20 g/day for the 91 days of the experiment. A third pony was fed 10 g/day on days one through 59 and 20 g/day for the remainder of the test period. Two died from foreign body inhalation pneumonia and the other from tubular nephrosis.

Baled hay from Smelterville was contaminated in 1974 by surface deposition of lead. The hay contained 345 to 540 ug/g whilst control hay of unspecified origin contained only 3 to 4 ug/g. The contaminated ground was west of Kellogg, approximately two miles west of Bunker Hill. Horses were killed by feeding on these pastures during the early 1970s. The hazardous condition did not persist following implementation of emission control measures. These levels in hay are particularly high and similar to those reported by Hammond and Aronson (1964) in forages in Minnesota contaminated by smelter emissions.

Hammond and Aronson (1964) estimated the minimum cumulative fatal dosage for cattle as 6 to 7 mg/kg/day. Allcroft (1951) has reported that 5 to 6 mg/kg/day may be fatal or continued almost indefinitely without toxic effects. Assuming 540 ug/g lead in hay from the Smelterville episode and 20 pounds per day hay consumption, the daily dose would be about 5 g or 20 mg/kg (250 kg cattle). This dosage is clearly well above that which might be achieved by ingestion of plants surveyed during the course of this work.

The data presented in Table 34 can only be used qualitatively due to uncertainty about the limit of detectability. The samples were obtained in August 1986 from two groups of horses in good physical condition. Each was on overgrazed pasture and provided well water. Lead levels were at or very slightly above the minimum limit of detectability. Larger samples would be required to reliably compare the blood leads in the two groups. The delta soil contained about 2000 ug/g lead. No soil measurements are available for the Avery site.

Based upon their condition, the horses are apparently thriving on the delta near Harrison. The pasture contained elevated lead levels relative to soil background levels (2000 ug/g versus 16 ug/g), but it's availability is very limited.

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Table 32. 1972 Summary of Analyses of Horse and Cattle Blood: Lead

Sample	N	Mean Blood Lead[a] (ug/ml)	Range
Kellogg Horse	118	0.23	0.05-0.70
Control Horse	10	0.21	0.05-0.50
Kellogg Cattle	8	0.22	0.20-0.25
Control Cattle	2	0.18	0.15-0.20

Burrows et al. 1981

[a] Minimum detectable level was about 0.1 ug/ml.

Table 33. Tissue Lead Levels in Animals

Species	Liver	Tissue (ug/g) Kidney	Blood	Bone
Waterfowl				
Normal	0.05-0.5	0.1-1.0	0.02-0.5	2-32
Toxic				
Ducks and Swans	10-64	60-1600	-33	20-300
Geese	9-102	8-55	3.3-16	
Sheep				
Normal	0.05-0.8	0.1-0.8	0.02-0.25	1-3
High	5-25	5-100	0.7-0.9	10-40
Toxic	10-100	5-200	1-5	-
Poultry (Chickens)				
Normal	0.1-0.5	0.1-1.0	0.04-0.05	<50
High	5-10	5-12	2-6.2	150-400
Toxic	18-90	20-150	4-12	>400
Horse				
Normal	0.5	0.5	0.04-0.25	3-4
High	3-5	3-5	0.3-0.6	8-10
Toxic				
Acute	4-50	5-140	0.3-0.5	40-200 (dry weight)
Chronic	10-500	20-200	0.6-2.5	
Cattle				
Normal	0.1-1	0.2-2.0	0.02-0.2	1.0-7.0
High	2-10	3-20	0.3-0.4	-
Toxic	5-300	10-700	0.35-32	-
Dog				
Normal	0.1-3.5	0.1-2.5	0.01-0.2	
High	3.6-5	5-10	0.3-0.8	
Toxic	50-200	10-50	0.6-7.4	

Puls 1981

Table 34. 1986 Delta Horse Blood and
Soil Levels of Lead (ug/ml)

Horse	Lead (ug/ml)
Delta	
1	0.5
2	<0.4
3	<0.4
4	0.4
5	0.5
6	<0.4
Avery - Background Exposure	
1	<0.4
2	<0.4
3	<0.4
Delta Surface Soil (ug/g)[a]	
1	2114
2	1872

Krieger 1986

[a]Upper 1-3 inches of soil from area where horses were
pastured.

All analyses performed at the Washington Animal
Disease Diagnostic Laboratory, Toxicology Section,
Washington State University and University of Idaho.

3.2. Pets

Hunting dogs in the Basin have been poisoned when their owners have used sediments in the animals exercise runs (Robert White, St. Joe Veterinary Clinic, St. Marie's, personal communication). Lead poisoning developed after several months of exposure which included digging along the fence line. The weakened animals responded to calcium - EDTA therapy which was repeated on two additional occasions after exposure had stopped.

On the same property, the owner had used soil/sediments in a child's sandbox. The owner was informed of the risk, and advised to consult local health authorities. No evidence of excessive exposure was reported.

These uses of soil/sediments containing potentially toxic levels of lead and cadmium present excessive risk. Persons who might consider use of such sediments for home improvement projects must be warned to prevent movement of the elementals into domestic environments.

3.3. Muskrat

Muskrat Ondatra zibethica are a potentially valuable biological monitor of lead bioavailability in that carcasses are available each winter and the entire carcass can be obtained. The animals are prominent herbivores and some are particularly active around areas being developed by Fish and Game to promote species diversity. Water potato may be an important foodplant of muskrats and it is also eaten by some Native Americans (Table 26).

An early specimen (roadkill) in 1985 near Thompson Lake had liver and kidney levels of 0.8 and 1.0, respectively. Subsequently, a series of 66 livers obtained from trappers were analyzed at the Washington Animal Disease Diagnostic Laboratory, Toxicology Section, Washington State University and University of Idaho Department of Fish and Game from specimens provided by John Nigh, Idaho Fish and Game. All contained measurable lead which ranged from 0.2 to 3.9 ug/g. The mean $\pm$ S.D. was 1.2 $\pm$ 0.8 ug/g. These levels (Table 35) are higher than many of the norms recorded in Table 32. Values for unexposed muskrats are not available.

Muskrats may be very useful biological monitors of trends in lead bioavailability. Their biology makes them attractive monitors since they have close contact with environmental lead and their diet includes lead-exposed foods. They are trapped each winter and their carcasses presently lack commercial value. Thus, use of muskrats as environmental lead monitors would not require specific trapping and killing of these aquatic rodents for tissue analysis. Muskrats from other regions could be used as controls to discern background levels. It must be emphasized that levels above control values do not necessarily imply a harmful biologic effect. A review of this report also noted that muskrat monitoring might be a means to differentiate lead bioavailability between lakes.

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Table 35.   1985 Muskrat Liver: Lead

Sample	Tissue Lead (ug/g)		Sample	Tissue Lead (ug/g)
1	1.6		34	0.6
2	1.2		35	0.6
3	1.4		36	3.1
4	1.0		37	1.8
5	1.2		38	0.3
6	1.5		39	3.8
7	0.9		40	1.9
8	1.9		41	1.3
9	0.4		42	2.3
10	0.7		43	1.8
11	0.8		44	2.4
12	1.0		45	0.2
13	1.6		46	0.3
14	0.8		47	1.1
15	1.6		48	0.6
16	0.9		49	2.1
17	1.5		50	0.8
18	0.9		51	1.2
19	2.1		52	0.2
20	0.6		53	0.3
21	3.1		54	0.2
22	1.7		55	0.2
23	1.2		56	1.6
24	1.1		57	0.4
25	0.6		58	0.2
26	1.1		59	0.6
27	1.1		60	04
28	1.9		61	0.5
29	0.3		62	0.2
30	0.6		63	0.6
31	2.6		64	0.9
32	0.6		65	0.7
33	1.7		66	2.7
		Mean $\pm$ SD		1.18$\pm$0.82

Idaho Department of Fish and Game

All analyses performed at the Washington Animal Disease Diagnostic Laboratory, Toxicology Section, Washington State University and University of Idaho.

3.4. Mouse

A 10.9 g mouse <u>Microtus pennsylvanicus</u> was found dead in 1985 in the marsh of Thompson Lake north of the Springston Mill site. The mouse was thin, stomach and lung were hyperemic, and lower intestine impacted. The liver was grossly normal and contained 2.2 ug/g lead. Kidney, small intestine and colon contained 4.1, 1.7, and 7.0 ug/g, respectively. None of these levels were considered life threatening <u>per se</u>.

Following the initial finding a series of mice were trapped by Jane Homan in May 1985 in the marshy area along the southeast shore of Thompson Lake. Kidney and liver leads were measured and are presented in Table 36. An attempt was also made to assess tail lead but sample size precluded reliable measurements. In general, kidney lead was greater than liver lead, and the difference was significant $p<0.05$.

The mouse liver samples provided ample evidence of accumulation of elevated tissue levels, but no observations to suggested toxicity were made.

Roberts et al. (1978) measured lead in soil, vegetation, ground-living invertebrates and indigenous small mammal populations in Wales. Small mammal body and tissue lead were elevated. Control bodies ranged 0.9-2.8 ug/g fresh weight whilst small mammals from four contaminated sites contained (ug/g fresh weight) 8.6-31.3, 16.3-20.7, 42.8-45.3, and 11.2.

Small mammals could be a suitable bioindicator species in the Coeur d'Alene River Basin. Since the relative sensitivities of indices of poisoning are well established in the laboratory (Goyer et al. 1970) progress with wild species would likely be facilitated.

3.5. Fish

Very limited pilot studies of the occurrence of lead in fish liver were conducted. In two series of Kokanee of about 10 and 20 fish each provided by Diane Stone, 1985-86, levels were barely detectable at background. If elevated levels had been present, the analysis could have been quantitative, however, at the low environmental levels present, the amounts were too low to be reliably measured.

The Agency for Toxic Substances and Disease Registry documented the occurrence of lead and other elementals in a small sample of Coeur d'Alene Lake fish (Margolis 1986). Fishes included yellow perch, cutthroat and rainbow trout, Kokanee, large-mouth bass, and brown bullhead. The mean and the median levels of lead were 0.8 and 0.45 ug/g (range 0.06 - 3.3). Exposure assessments for adults and children were performed and projected exposures using a "worst case" scenario exceeded an ADI established by the Environmental Protection Agency. Note: ADI is an estimate of the maximum daily intake that will not produce adverse chronic or subchronic noncarcinogenic human health effects. Derivation includes a safety factor.

The Idaho Department of Health and Welfare cooperated with the Agency for Toxic Substances and Disease Registry to determine the extent of exposure among high, moderate, low, and nonconsumers of fish. The

investigators surveyed consumers and measured lead and cadmium blood levels. Exposure was lower than earlier predicted and lead and cadmium blood levels were within norms for the general population (Bashor 1988).

The most recent available information concerning elementals in fish in the Hornig et al. (1988) report titled "Coeur d'Alene Basin Water Quality Monitoring." Samples from the Lake as well as Wolf Lodge Creek, Mission Slough, Killarney Lake, Round Lake, and the South Fork above Mullan. The conclusions are quoted in part as follows:

> "The Region 10 Laboratory conducted heavy metal analyses on composites of several species of game fish collected by the Idaho Department of Fish and Game from Coeur d'Alene Lake, the mainstem lateral lakes, and the South Fork at Mullan. The purpose of these analyses was not to provide a full human health risk assessment, but rather to get a general picture of the degree of contamination in edible tissues, and to locate which species and locations would be candidates for follow-up analyses. The preliminary results from these analyses indicate that the heavy metals, which are at very elevated levels in the sediments, are not accumulating in edible fish tissue at levels considered dangerous to sports fishermen (Table 11). High levels of cadmium, however, were found in liver and kidney organs, which indicates that this metal remains bioavailable and points to the potential of using these organs for assessing long term trends in cadmium levels."

3.6. Turtle

A turtle (species unknown) was caught by the author in Thompson Lake in June 1986. The turtle was held without food overnight. Feces which contained grit and chitinous insect parts were analyzed for lead. The sample contained 380 ug/g. It is of interest that 12 goose feces samples also obtained at Thompson Lake contained 85 ug/g lead (mean). Dietary exposure is assumed. It is proposed that analysis of turtle feces might be a biological means to compare the availability of lead in stable and disturbed environments. Data such as this collected over a prolonged period of time might allow scientists and land managers to develop unique insight into factors effecting the availability of lead in the region. Due to their long lives and relatively small range, turtles may also be useful for bone and soft tissue analysis of lead accumulation (Beresford et al. 1981).

Table 36. Mouse Liver and Kidney Lead, Thompson Lake

Sample	Tissue Lead (ug/g) Liver	Kidney	Sample	Tissue Lead (ug/g) Liver	Kidney
1	2.8	5.4	26	6.2	17.6
2	7.2	18.9	27	1.5	2.4
3	2.1	3.4	28	1.5	3.0
4	6.1	6.3	29	0.1	4.4
5	2.4	10.8	30	8.7	41.7
6	9.8	1.5	31	7.0	6.8
7	4.3	5.7	32	0.9	2.4
8	10.0	11.0	33	0.7	2.5
9	2.5	8.2	34	1.2	5.0
10	1.8	12.4	35	7.9	16.8
11	2.4	8.2	36	1.3	5.4
12	2.0	0.6	37	9.0	6.1
13	4.5	4.5	38	0.9	1.4
14	1.2	6.8	39	1.0	2.9
15	6.0	9.9	40	3.2	1.0
16	10.2	11.2	41	5.2	7.2
17	4.5	9.9	42	0.1	1.4
18	3.6	18.4	43	QNS	QNS
19	1.7	5.4	44	0.1	2.4
20	21.9	8.7	45	QNS	QNS
21	0.6	3.5	46	QNS	QNS
22	9.3	14.1	47	QNS	NS
23	2.6	0.9	48 Fetus/14	QNS	NS
24	0.1	2.7	49 Fetus/15	0.2 ppm	NS
25	3.3	3.4			

Idaho Department of Fish and Game

QNS - Quantity not sufficient
NS - no sample

Collected and analyzed at the Washington Animal Disease Diagnostic Laboratory, Toxicology Section, Washington State University and University of Idaho.

3.7. American Osprey

An attempt was made by the author in 1986 to analyze American Osprey _Pandion haliaetus_ feces and debris from a nesting site along the south shore of Thompson Lake. Owing to the fish diet of the osprey it was thought that the samples might be useful monitors of environmental lead. The debris had fallen from the nest and contained 85 to 109 (mean 99) ug/g. Whether the lead resulted from the diet or from sediment adhering to the samples can not be determined.

Three regurgitated pellets were obtained in 1986 in the vicinity of another osprey nest. They were collected from the ground and after nitric acid digestion found to contain 30, 55, and 254 ug/g lead. Pellets collected to assure minimal sediment contamination might be useful in monitoring changes in lead bioavailability. (The species involved in this case may or may not have been osprey.)

Feces-contaminated grasses below the osprey nests contained 8.4 ug/g lead. Uncontaminated grasses contained 2.7 ug/g. The fish diet and accessibility of osprey make them attractive birds for monitoring.

All samples were collected and analyzed under the supervision of the author.

3.8. Great Blue Heron

A young (one year old) Great Blue Heron was apparently killed in 1985 by an automobile near Thompson Lake. Tissues provided by John Nigh, Idaho Department of Fish and Game, were analyzed in the Diagnostic Laboratory. Liver and kidney lead levels were 0.17 and 0.23 ug/g, respectively. The stomach (preventriculus) contained 389 ug/g lead. If that level is common to the diet, the lead that is being ingested is probably poorly available for absorption, distribution and deposition in tissues such as liver and kidney.

4.0. Supplementary Notes on Lead as an Environmental Hazard

Discussion of hazards and risks associated with the occurrence of lead in the Lower Coeur d'Alene River Basin requires a careful separation of terms. Hazard in this context refers to the various harmful biological effects that are associated with excessive exposures to the chemical under study. Dose-response relationships in laboratory animals are the most common form of knowledge. Both experimental and epidemiological studies in animals and humans can contribute to the description of hazard. Risk on the other hand relates to the likelihood that exposure will be associated with a harmful effect. Thus, as is the case with lead, the hazard to wildlife is present (this study and many others) but the risk seems relatively low due to the poor availability of the lead substrates in sediments (Tables   ). Probably underlying this fact is the stability and extreme insolubility of lead sulfide species (Nriagu 1978).

The work contained in the previous sections for the most part followed the initial tissue analyses that led to the conclusion that the 1982-83 swan deaths resulted from lead poisoning and focused upon the general issue of lead availability to plants and wildlife in the Lower Coeur d'Alene River Basin. The work is reported in six sections. <u>1.0. Waterfowl Deaths and Illnesses Recurred in 1982. 2.0. Lead and Other Elementals in the Environment, 3.0. Lead and Animals. 4.0. Supplementary Notes on Lead as an Environmental Hazard, 5.0. Mitigating Hazards of Sedimentary Lead and Areas for Additional Study. and 6.0. Summary.</u> Historical and contemporary data concerning exposure and toxicity of lead in waterfowl comprised the opening section (1.1. and 1.2.). Subsurface soil (2.1.), water (2.2.), and plant (2.3.) samples were analyzed for lead and occasionally for other elements. This work provided current estimates of subsurface soil levels and further showed very low levels relative to acute toxicity thresholds of lead to be present in water and plants. More data about concentrations of lead in water and amounts in plants would be necessary to definitively characterize any chronic risk. It would seem to be low based the limited studies available <u>3. Lead and Animals</u>. This work concerning Cattle and Horses (3.1.), Pets (3.2.), Muskrats (3.3.), Mouse (3.4.), Fish (3.5.), Turtle (3.6.), Osprey (3.7.), and Heron (3.8.) coupled with the waterfowl work reported above (1.1. and 1.2.) clearly shows that the alluvial deposits constitute an everpresent hazard of low apparent risk. This hazard-risk issue will be the central one for persons charged to manage the wetlands impacted by the mining and milling wastes.

Hazards posed by lead in the Coeur d'Alene River Basin system include adequately documented, periodic acute poisoning of waterfowl as well as uncertain chronic effects that might be apparent as enhanced susceptibility to disease and reproductive toxicity. The toxicity of lead has been reconfirmed and increasingly defined for at least 2000 years. Similarly, in the Lower River system, the occurrence of lead and associated elementals has been recognized for about 100 years. Environmental lead is a substantial environmental hazard due to its persistence, tendency to accumulate in tissues such as bone and liver, and acute and chronic toxicity in excessive amounts.

Risks associated with lead in the Lower River system are much less certain due to uncertains about the bioavailability of environmental sedimentary lead. In part, this results from the aqueous insolubility of lead sulfide which is apparently the predominant sedimentary form of the element. Low solubility becomes an even more critical issue when the PbS deposits are considered in their full depth and breadth. These extensive deposits are effectively protected from environmental factors such as water, oxidizing agents, and reducing agents that might make lead more bioavailable. Stabilization of lead by humic materials (Schiefer and Ulrich 1960) is a natural ongoing process which is probably limited by the limited interfaces between solubilized lead and organic matter.

MacLean et al. (1969) reported on the extractability of added lead in soils and its concentration in plants. They noted beneficial effects of soil organic matter, lime, and phosphate in reducing extractable lead in the soils and its uptake by plants. Those investigators clearly showed on a very small scale how management practices might be developed to minimize risks associated with excessive lead exposure.

The beneficial effect of organic matter seems to be of particular importance. Divalent metal ions (Pb^{2+}) form complexes with soil humic compounds (Schiefer and Ulrich 1960). Such lead complexes will concentrate in highly organic soils and complexed lead will have a very low order of availability.

There may be circumstances when lime and phosphate treatment can be used to an advantage on a small scale. The action of lime seems to result from more extensive lead-organic complexing at higher pH. The effect of phosphorous in reducing lead availability derives from the insolubility of lead phosphates (Schiefer and Ulrich 1960).

Activities which result in the introduction of increased amounts of organic matter will stabilize elementals such as lead and cadmium by decreasing their bioavailability via complex formation. The relationship between solubilization of elementals from their chemically stable environments and subsequent fixation of the element in an organic form represents an ongoing process which should be kept in mind by land managers. Actions which might result in excessive solubilization or diminished organic complexation should be avoided or be carefully controlled in extent and duration.

Substantial discussion has developed concerning the impact of habitat enhancement activities of the Idaho Department of Fish and game. Island building to promote species diversity needs to be evaluated with respect to its impact on the bioavailability of lead. Feces analysis may be one means of obtaining important data (in addition to work with muskrats mentioned previously, Section 3.3.).

This may be illustrated by examination of the fecal analyses obtained in July 1989 from Canada goose nesting sites on Thompson Lake. The range of lead was 14-181 ppm (mean 85 ppm) (Section 1.2.).

Similar analysis should be performed in areas impacted and unimpacted by "island building" to determine the extent of local impact. On the simple basis of mass and extent, the amounts of sediment involved are minuscule portions of the total volume of soil/sediment in the Basin. The resulting species diversity and increased fluxes of organic matter would seem to me to far outweigh any known additional risks to wildlife or the environment posed by the projects themselves.

5.0. Mitigating Hazards of Sedimentary Lead and Areas for Additional Study

The Coeur d'Alene River Basin includes some areas which contain exceptionally high amounts of sedimentary lead and other elementals, notably cadmium and zinc. Health issues related to lead must be defined in extremely broad terms due to the sheer vastness of areas in the Basin that contain elevated and potentially toxic levels. So far as wildlife are concerned at this time, toxicological issues can be defined with respect to lead rather than to other elementals or hypothetical interactions. It seems sufficient to acknowledge the presence of other potentially toxic elements, but to focus on qualitative and quantitative issues effecting lead.

In a similar fashion, I recommend focusing attention on a few key biological indicators for purposes of defining the bioavailability of lead and its toxicity. Some biological indicators which seem especially appropriate are ducks and muskrats. They acquire a lead body burden, can be easily necropsied to provide soft tissues (liver, kidney) and bone for analysis, and, importantly, are hunted and trapped in large numbers. The latter issue is important both with respect to providing a robust sample as well as to involve a large number of people in the project of sampling. That opportunity to educate the public should not be lost.

The widespread distribution of high levels of lead and other elementals such as cadmium and zinc does not need further documentation. Natural processes such as erosion and flooding as well as mining, agriculture, lumbering, land management, and recreation may alter the quantity and quality of the elemental load in any particular environmental compartment in the Basin. These merits and possible negative consequences of these processes which might alter lead availability must be carefully evaluated.

There must be active pursuit of activities which increase species diversity thereby contributing to stabilization of elementals, particularly lead. Some regulations relating to hazardous substances may actually be detrimental to remediation efforts in the vast Coeur d'Alene River Basin since most regulations were not developed to address issues of this scale.

So far as specific research needs to augment land management, I suggest the following potential areas of effort:

1. <u>Establish a biological monitoring program using an appropriate set of indicators including, and perhaps limited to, ducks and muskrats.</u>

The figures depicting the distribution of liver lead show that the residential duck population which was sampled in August 1986 has the same characteristics as samples obtained from hunters in Fall 1986 and Fall 1987, Figure 4. As shown earlier, Figures 1, 2, and 3, duck livers from environments which contain background lead (Umatilla and Columbia Basin) and tissues from a shot impacted area present sharply different patterns of lead distribution.

Analysis of the largest feasible numbers of early season duck livers should be continued on an annual basis. Consideration should also be given to collecting late season data when possible. The later season activity would be expected to show possible influence of lead shot ingestion as well as duck migration. Data summarized in Table 37 and distribution from figures 1, 2, and 3 indicate that lead-impacted populations can be distinguished from unexposed duck populations.

Very similar considerations apply to the use of muskrats as environmental monitors (Section 3.3.) of lead. In the case of muskrats, a more epidemiological approach could be employed to compare regions within the basin. Their use has the drawback that toxic levels in muskrats are not known and there is less basic toxicology data available in muskrats than in ducks. Nonetheless, comparative work would be very interesting concerning lead availability and potential toxicity.

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Coeur D'Alene 1986

Coeur D'Alene '86 Hunting

% Affected

% Affected

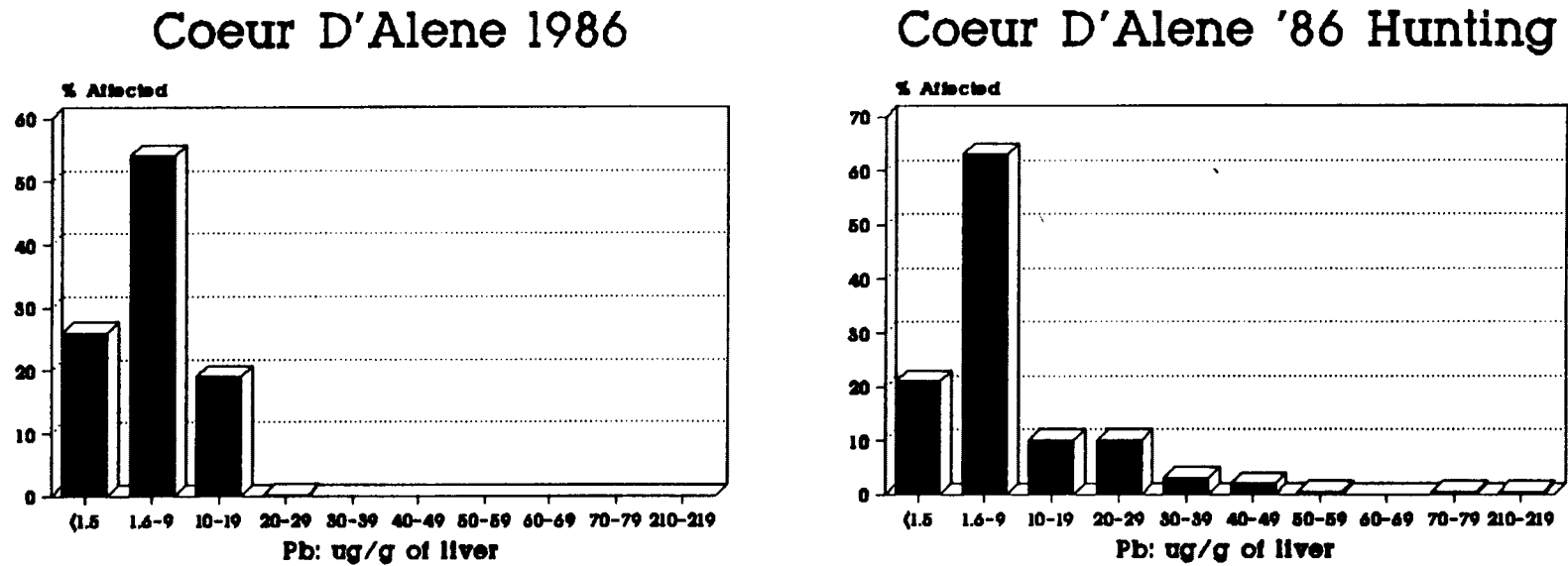**Pb: ug/g of liver**

Pb: ug/g of liver

Figure 1. Distribution of liver lead in ducks from the Coeur d'Alene River Basin. The left element shows distribution in birds collected during a controlled hunt in August 1986. The right element shows the distribution of liver lead of birds sampled at bag limit inspection stations in the fall of 1986.
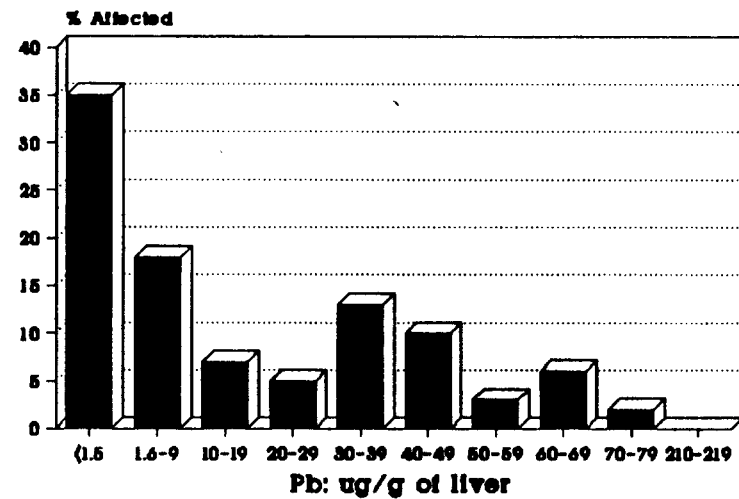
Coeur D'Alene '87 Hunting

Savvie Island

% Affected

% Affected

Pb: ug/g of liver

Pb: ug/g of liver
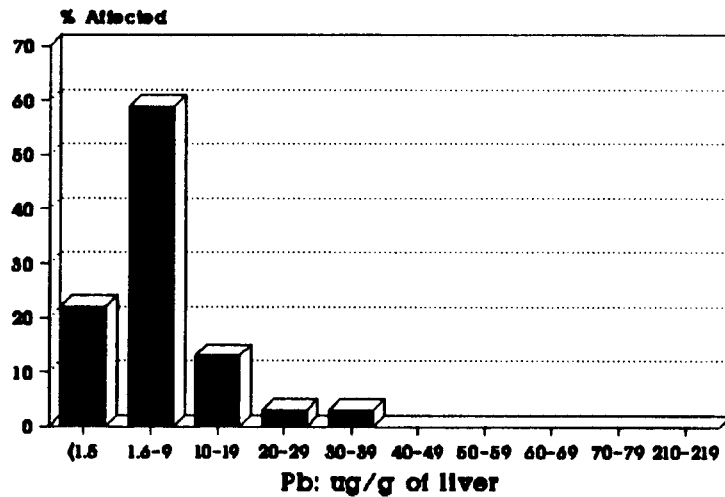
Figure 2. Distribution of liver lead in ducks sampled at bag limit inspection stations in the fall of 1987 in the Coeur d'Alene River Basin and earlier at Savvie Island, Oregon. The Savvie Island ducks represented 1983 and 1984 populations with lead shot exposure (Oregon Department of Fish and Game).

Umatilla NWR

Columbia Basin
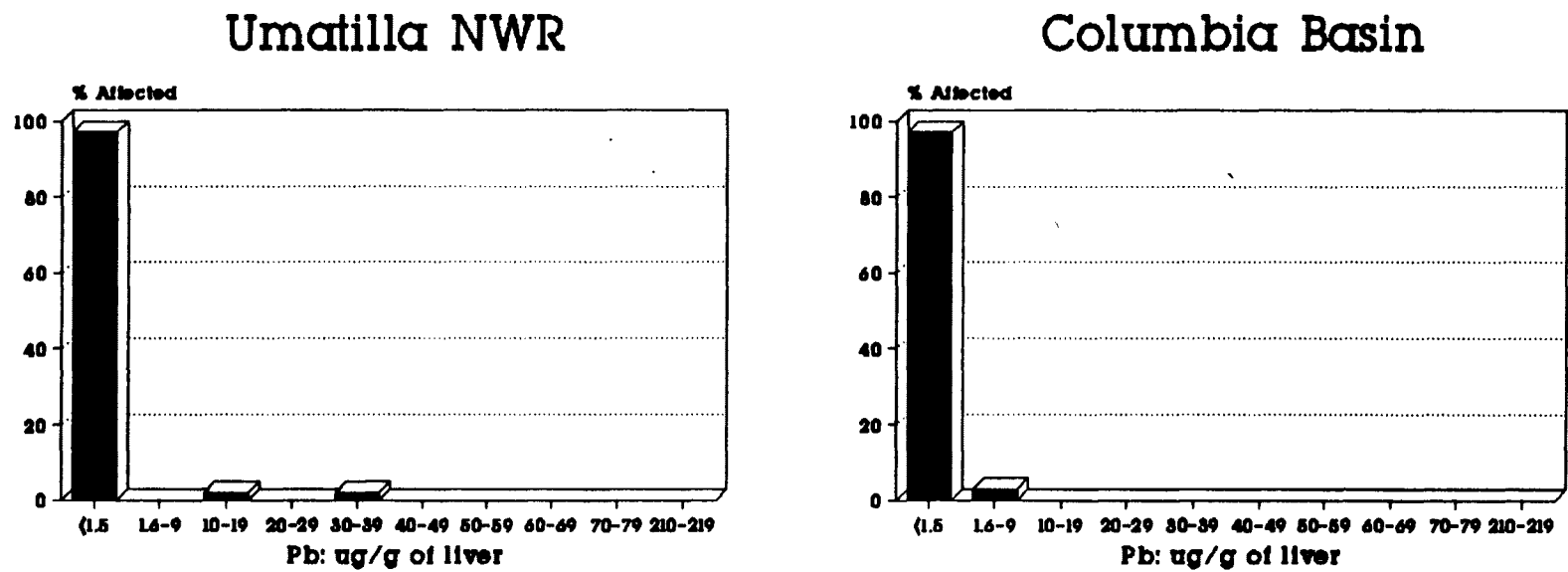
Figure 3.   Distribution of liver lead in ducks from areas not substantially impacted by environmental lead.

Coeur D'Alene '86-87

% Affected

Pb: ug/g of liver

■ Coeur D'Alene '86   ▨ Coeur D'Alene '86 Hu
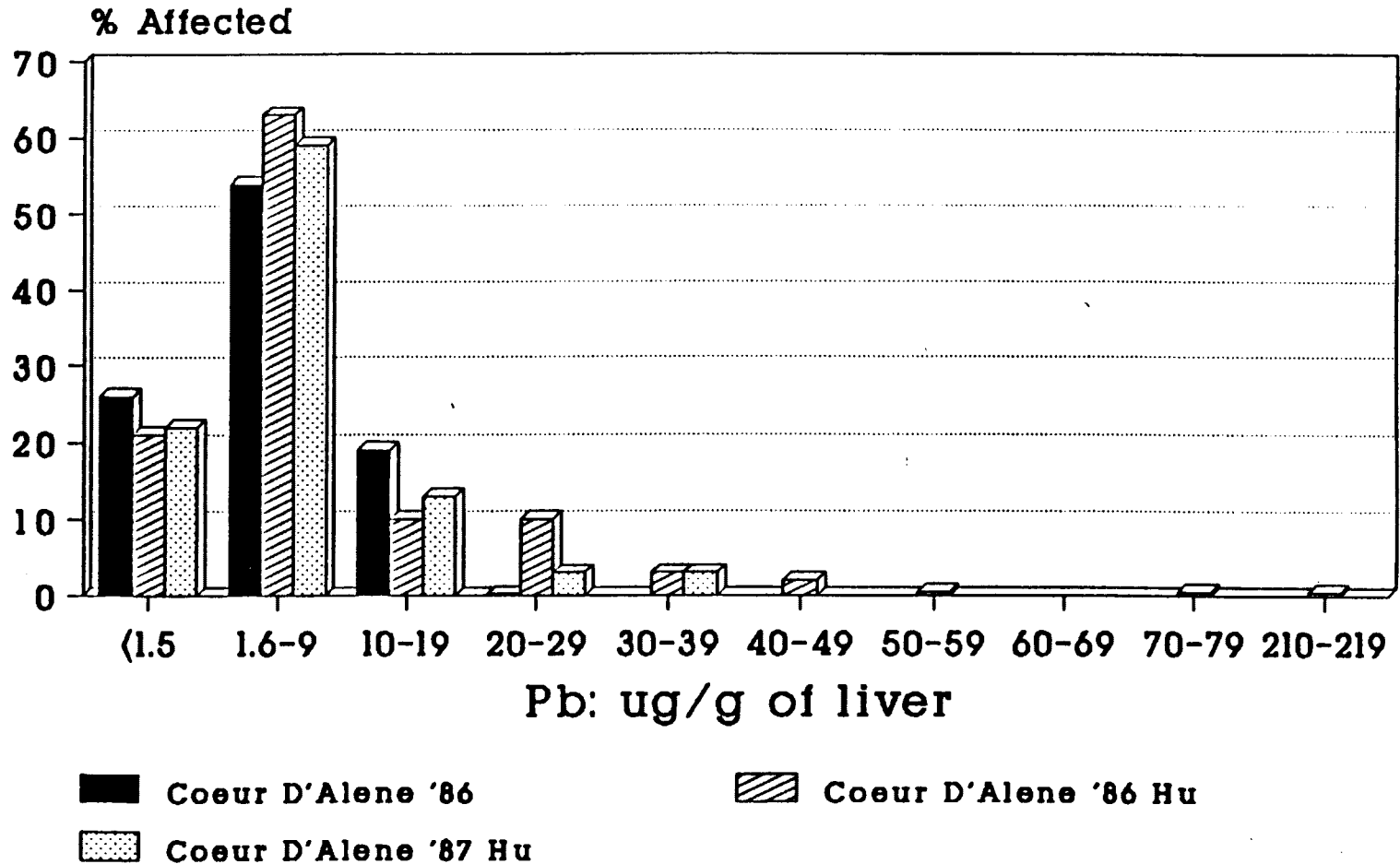
▩ Coeur D'Alene '87 Hu

Figure 4.   Summary of distribution of liver leads from three separate sets of ducks from the
Coeur d'Alene River Basin including an August 1986 controlled hunt and bag limit
inspections in fall 1986(Hu) and 1987(Hu).

Table 37. Summary of Lead Levels in Duck Livers from
Coeur d'Alene River Basin and Other Selected Sites

Site	Environmental Source	Liver Leads (ug/g) Median	Range
Coeur d'Alene River			
Bag Check 1986	Lead sediments	3.9	0-210
Bag Check 1987		3.9	0.3-38.0
Controlled Hunt 1986		3.5	0.8-27.8
Savvie Is., Oregon 1983/1984	Lead shot impacted	9.0	0.8-73.9
Umatilla National Wildlife Reserve 1984/1985	Not lead impacted	0.8	0.8-39.9
Columbia Basin 1986	Not lead impacted	0.3	0.2-1.9

Liver lead values listed in Tables 15 to 19 and distributions shown in Figures 1, 2, and 3.

2. <u>In order to evaluate the present hazard wildlife represented by the leaded sediments, I would give highest priority to experimentally evaluating the bioavailability of lead (and potentially cadmium).</u>

The importance of the bioavailability issue is both analytical and biological. Whether metallic lead (Pb^0) or sedimentary lead (PbS and other Pb^{2+} species) samples are analyzed by usual spectoscopic techniques, they yield a common result - ug lead. There is abundant evidence that both the physical and chemical forms of lead are important determinants of toxicity. We know virtually nothing about the toxicology of sedimentary PbS.

It has been estimated that 2.4 million ducks die worldwide of lead shot poisoning -- not including losses resulting from sublethal effects (Wobeser 1981). As a result, our understanding of lead toxicity in waterfowl is principally based upon studies performed using lead shot. Related issues have been studied under a variety of experimental circumstances but no work is available concerning insoluble forms of lead such as PbS. These studies provide valuable foundation, perhaps unique with respect to the toxicity to wildlife of a persistent environmental pollutant. As a result, the required additional experimental studies with sedimentary lead can be pursued with minimal methods development.

The subject is by no means fully understood and studies must be carefully planned and controlled. Whether total body burden is related to the onset of signs of clinical lead poisoning is not established. Several complicating factors are known. Dietary calcium intake may influence lead burden by changing the ratio of distribution between skeletal and soft tissues such as liver (Six and Goyer 1970). Additionally, under conditions of stress such as illness, starvation, or aging, lead may be reabsorbed from bone and distributed to soft tissues (Williams 1958). These factors should prompt caution in interpreting body burden data when field observations or histopathology are lacking.

Goyer et al. (1970) has established the relative sensitivity of indices of lead poisoning in laboratory animals.

The experimental plan would include administration of feed containing control and three (3) levels of sedimentary lead. A second "in-life control group" would be caged in a lead contaminated area at Thompson Lake. Over a two-month observation period, bird weights and blood lead, hematocrit, free erythrocyte protoporphyrin, and delta-aminolevulinic acid dehydratase activities would be measured. Although it is clear that elevated tissue levels of lead are present in ducks in the Basin, the risks associated with those levels is not known. These studies would help establish whether the exposures result in toxicity.

Published studies of Mautino and Bell (1986), Rattner et al. (1989), Pain and Rattner (1988), and Carlson and Nielsen (1985) using ducks and the same parameters as suggested above demonstrate feasibility of suggested studies. Histopathology could be an additional means to assess whether the sediment exposures are

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associated with harmful effects (Rao et al. 1989). French et al. (1987) studied the effects of zinc shot by mallard ducks using a protocol similar to that suggested here to evaluate the fate and effects of lead from the sediments.

Studies performed to evaluate risks related to lead shot ingestion provide extremely valuable data concerning lead poisoning. The fate and effects of lead derived from lead shot and that derived from lead sulfide-rich River sediments may be very different based upon chemical and temporal issues. The results of the proposed work can be used to describe no adverse effect levels and margins of safety. This will be extremely valuable management data.

3. Assess the health status of resident duck populations.

The reproductive status of Coeur d'Alene River Basin duck populations should be thoroughly evaluated and periodically assessed. If there are apparent changes in lead availability or changes in tissue levels based upon hunter surveys, studies should be intensified.

Reproductive and immunotoxicity of lead are known but not studied in ducks or other wildlife. Population studies in progress (Henny, personal communication) will contribute to knowledge of possible effects on Basin wildlife.

4. Establish a "Winter Bird Watch" citizens program in collaboration with Idaho Fish and Game, the University of Idaho Toxicology Program, and other State and Federal Agencies.

The episodic deaths of swans and geese have been reported (see frontis) and leave no doubt that under poorly understood conditions, lead-related deaths occur.

Whether ducks are similarly effected under prevailing conditions is not known and must be investigated as part of the effort to determine whether existing lead burdens (liver levels 3.5-4.0) are having a harmful effect. Deaths may be unnoticed and since lead-poisoned birds tend to seek seclusion and often die in areas of heavy cover where they may be consumed by predators and scavengers (U.S. Department of Interior 1988). These additional field studies would supplement the high priority feeding experiments noted above. Over a period of time some invaluable survey observations could be collected.

Using the media of newspapers and simple fliers, people could be instructed concerning signs of poisoning: lack of feeding, loss of weight, weakness, tremors, drooped wings, greenish liquid feces, poor coordination (difficulty walking, poor balance). Participants can be given this type of information in the form of a check list which can be mailed to an agency office at a specified time. If dead birds are collected, they must be bagged in plastic and frozen until being transferred to Toxicology Laboratory personnel for necropsy and tissue analysis.

Groups that might coordinate the "Winter Bird Watch" are Chambers of Commerce, Parent-Teachers Associations, Clubs of hunters and/or fishermen, Veterinarians, schools and other community service organizations. Community effort and involvement in the bird watch will be important to their understanding of the need for continued evaluation and any future remediation efforts. Knowledge of past lead poisonings would make it quite clear to at least a core of people (Winter Bird Watchers) that "things" are unquestionably *a lot better than they used to be.*

If the survey is effective in locating areas where excessive exposure is occurring (from either sedimentary lead or lead shot), tissue analyses and controlled hunting may be used to help clarify the nature and extent of the problem.

5. <u>Establish clear contingency plans for the collection and frozen storage of dead birds related to episodic deaths of Basin waterfowl.</u>

For a period of time all collectible birds should be taken for lead analysis if winter and spring deaths are excessive in the view of Land Managers.

Investigators should record the condition of the carcass, contents of the gastrointestinal tract, and take liver, kidney, and tibia for lead analysis.

Sick or moribund birds should be captured or killed and transported to the Toxicology Laboratory of the Washington Animal Disease Diagnostic Laboratory, Washington State University, for necropsy and histopathological analysis. Progress in understanding the cause of episodic tundra swan and Canada goose deaths to date has been made using birds that died under field conditions. As a result, a certain amount of breakdown occurred before tissues were fixed and evaluated. The disease process may be better defined if additional samples are obtained in the future.

6.0.   Summary

Abundant evidence documents the extensive contamination of the Lower Coeur d'Alene River drainage with mining and milling wastes. The deposits contain an array of potentially toxic elementals, most notably lead, cadmium and zinc. The deposits are physically and chemically stabilized to varying extents. On the basis of their sheer volume and extensive distribution, the deposits have substantial potential to disrupt living systems, but that potential is seldom expressed as a result of the apparently low biological availability of the lead. The condition could be described so far as wildlife are concerned as hazardous, low risk. This fact must not prompt complacency. There seems little reason to minimize the potential health and environmental impact. The apparently low availability of the residues provides risk and environmental managers with an opportunity for informed and thoughtful decision-making that is often lost when environmental issues become environmental crises.

I have recommended establishing an aggressive biological monitoring program for lead and cadmium. Utilization of ducks from fall bag checks of hunters should allow more reliable distinction of lead shot and environmental exposures as the use of steel shot increases. Several experimental feeding studies with ducks are essential to better support their use as biological monitors. A second key monitor is the muskrat. I suggest monitoring cadmium and lead in muskrat kidneys on at least a biannual basis to obtain basic data concerning elemental bioavailability from sediment. It may be possible to target particular areas for study as a result of activities which might alter bioavailability. Use of ducks and muskrats represent a readily available resource which can be developed into a sound biological monitoring program without labor intensive sampling. Use of hunted and trapped animals will require no additional wildlife losses.

So far as the episodic deaths of tundra swans and Canada geese are concerned, the environmental and biological studies have unfortunately provided little new insight. Each year a few of the conspicuous, white swans succumb and whenever tested, high or toxic levels of liver lead have been found. Those lead levels are a consequence of the conditions along the Lower Coeur d'Alene River Basin. They are not likely to change over the short term, but it is likely that the risk will be diminished as elementals become more biologically stabilized. A management decision to reduce swan deaths could include herding with aircraft and development of feeding areas farther from the mainstem of the River. The problem is most apparent when the birds are plentiful and run-off waters high -- both conditions which will probably recur.

Detailed population studies of ducks in the River Basin can make a substantial contribution to understanding of the possible biological significance of elevated lead (and cadmium) levels (see Neufeld 1987). Since the immune system and reproductive system are both sensitive to excessive lead, toxic effects, if any, should be revealed in population studies. That basic biological work seems of critical importance in developing further land use strategies for the Basin.

It has become increasingly apparent that decision-making in the Basin must not be too tightly constrained by economic considerations attached to a small "Superfund Site", by states political boundaries, nor by federal zeal. Environmental lead distribution in water should be better defined. Basic knowledge should be obtained to determine the availability of lead sediments to ducks, muskrats, and mice to provide biological tools to assess impacts of human activities in the Basin. Once a biological monitoring program is established, it must be adequately supported and sustained over an extended period of time to detect trends and critical events. The Coeur d'Alene River Basin is the site of deposition of lead and other elementals which can be managed with interdisciplinary scientific support and increased efforts to inform concerned sectors of the public of the nature of the problem.

There is abundant evidence that things are getting better.

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7.0. References

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APPENDIX I.

Spectrochemical Analysis of Swan Liver: Idaho Bureau of Mines

Summary:

Ten (10) samples were received, frozen, from Idaho Fish and Game Commission. Nine (9) were swan livers and one (1) was rumen. Samples were alphabetically listed A through J. A freeze-drying procedure was used to prepare samples for investigation of volatile organometallic compounds, i.e., methyl mercury. Another portion of the livers were ashed in the normal manner in preparation for regular spectroscopic analysis.

Procedure:

A. Freeze Drying

Approximately one gram of frozen liver was weighed into a crucible and placed in a vacuum desiccator. The desiccator was attached to a lyophilyzing apparatus and evacuated for 24 hours at 50 microns pressure.

The dried residue was ground in a "Wiggle-Bug" for 30 seconds, mixed with Li_2CO_3, and arced for 30 seconds in a graphite cup electrode.

B. Ashing

Approximately one gram of frozen sample was weighed into a crucible and placed in a muffle furnace at 600°C. After six hours the crucibles were removed, cooled, and weighed for ash content. The ash was mixed 1:1 with Li_2CO_3 and arced to completion in a graphite electrode.

Method used by Idaho Bureau of Mines and Geology to analyze 1971 and 1974, Fish and Game samples. Al Bruner to author, 1982.

APPENDIX II.

Lead and Other Elemental Analysis:
Washington Animal Disease Diagnostic Laboratory,
Toxicology Section, Washington State University and
University of Idaho

<u>Analysis</u>. Inductively coupled plasma emission spectroscopy (ICP) and atomic absorption spectroscopy (AA) were used for the analysis of lead and other elementals. Tissues (usually about 1 gram) were digested overnight at 70°C in concentrated nitric acid in a sealed teflon vessel. A Lead Reference Standard (Banco Standardized, Anderson Laboratories, Incl., Fort Worth, Texas) was used. Reagent blanks were used and minimum levels of detectability for liver tissue were usually about 1 ug/g using ICP and about 0.1 ug/g using AA.

Identical methods were used to analyze cadmium, zinc, and other elements. In the vast majority of cases, ICP was the method of choice.

Quality control spikes were run periodically and samples were also rerun to obtain an indication of precision. Results were within 10 percent under normal circumstances.