CHAPTER 6
WILDLIFE RESOURCES

This chapter describes injuries to wildlife resources of the Coeur d'Alene River basin that have resulted from exposure to hazardous metals released from mining and mineral processing operations. Section 6.1 describes the wildlife resources of the Coeur d'Alene River basin. Section 6.2 provides an overview of the toxic effects of lead on wildlife. Section 6.3 describes the injuries evaluated in the Coeur d'Alene, Section 6.4 summarizes the testing and sampling approaches, and Section 6.5 summarizes the results of the injury assessment studies. Section 6.6 presents the injury determination evaluation, Section 6.7 summarizes the conclusions of the assessment of injuries to wildlife resources, and Section 6.8 provides references cited.

6.1 DESCRIPTION OF WILDLIFE RESOURCES

The Coeur d'Alene River basin is located in the Pacific flyway for migratory waterfowl and provides important habitat for a diverse assemblage of aquatic and terrestrial wildlife species (Figure 6-1). The Coeur d'Alene River and lateral lakes area of the basin (Figure 6-2) provide abundant and diverse riparian, wetland, and lake habitats that support diverse wildlife uses, including feeding, resting, and reproduction (Figure 6-3). Historically, the riparian zones of the South Fork Coeur d'Alene River provided feeding, resting, and reproductive habitat as well. Wildlife known to inhabit or suspected to visit the lower Coeur d'Alene area include over 280 migratory and nesting bird species (Ridolfi, 1993), as well as many mammals, reptiles (e.g., snakes, turtles), and amphibians (e.g., frogs, salamanders).

Migratory birds in the Coeur d'Alene River basin include waterfowl, birds of prey, songbirds, and other neotropical species. Ducks nesting in the basin include mallards (*Anas platyrhynchos*), wood ducks (*Aix sponsa*), green-winged teal (*Anas crecca*), ring-necked ducks (*Aythya collaris*), lesser scaup (*Aythya affinis*), northern shovelers (*Anas clypeata*), ruddy ducks (*Oxyura jamaicensis*), cinnamon teal (*Anas cyanoptera*), and redheads (*Aythya americana*) (Burch et al., 1996; Audet et al., 1999c). Other waterbirds nesting in the wetland and lateral lakes area include Canada geese (*Branta canadensis*), red-necked grebes (*Podiceps grisegena*), western grebes (*Aechmophorus occidentalis*), American coots (*Fulica americana*), pied-billed grebes (*Podilymbus podiceps*), black terns (*Chlidonias niger*), common snipe (*Gallinago gallinago*), and sora (*Porzana carolina*) (Chupp and Dalke, 1964; IDFG, 1987).

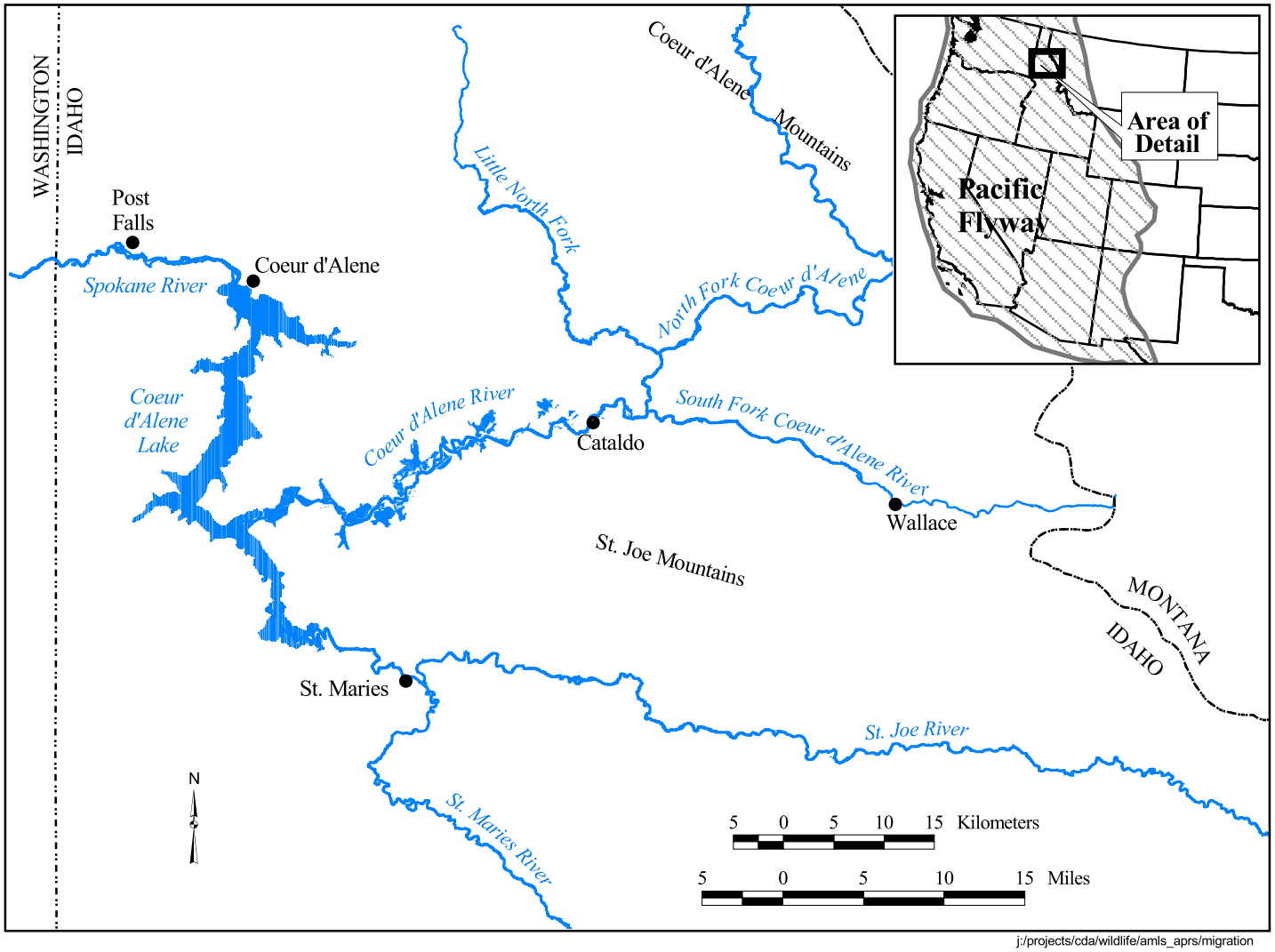


Figure 6-1. Map of the Coeur d'Alene River basin, St. Joe River basin, and surrounding areas. Inset shows area of assessment in relation to the Pacific flyway for migratory waterfowl.

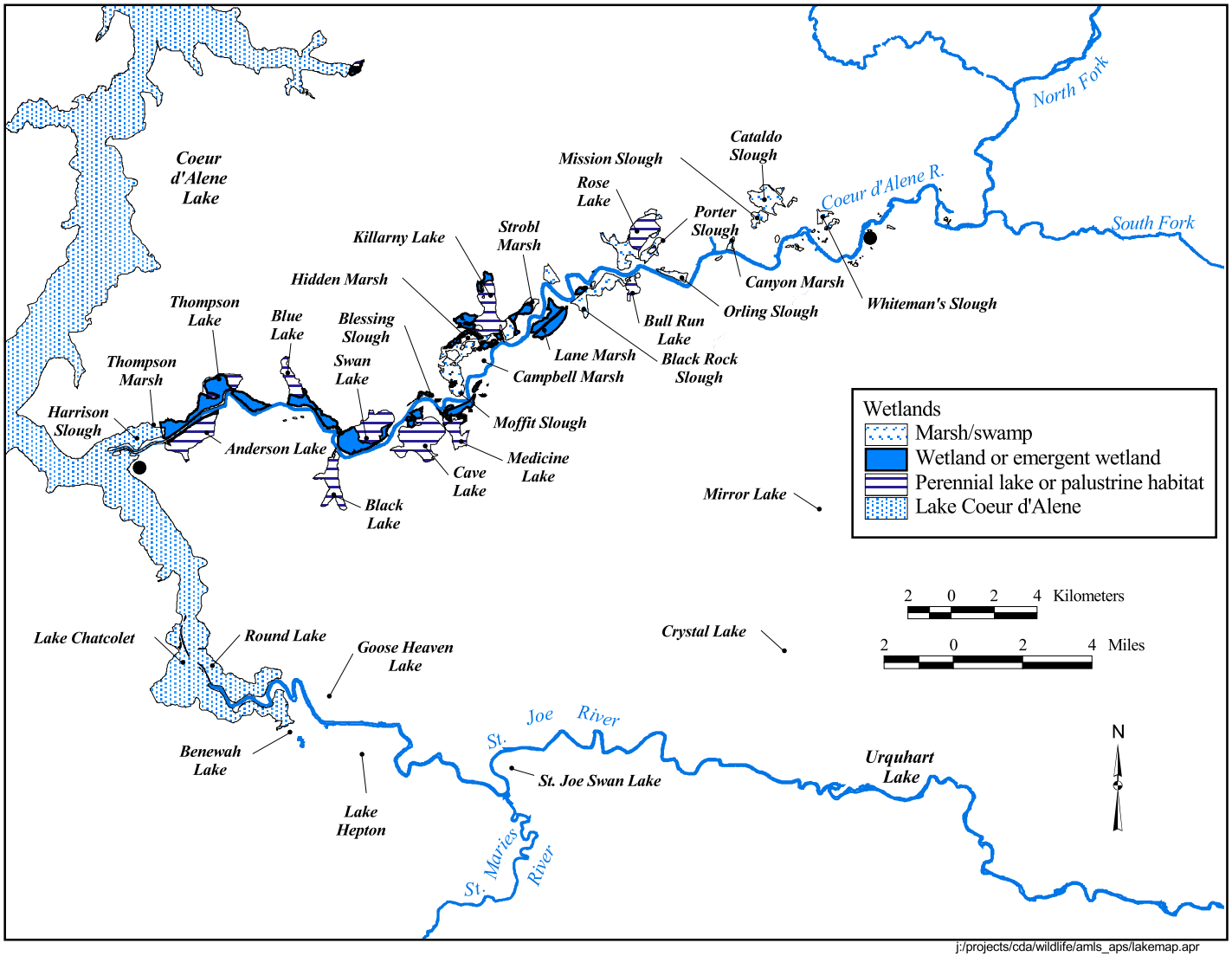


Figure 6-2. Map of the lower Coeur d'Alene basin showing the wetland and lake system adjacent to the Coeur d'Alene River (lateral lakes area).

Figure 6-3. Examples of bird usage in the Coeur d'Alene River basin. Top: tundra swans in Lane Marsh; bottom: mallards and other waterfowl in Canyon Marsh.

Waterfowl are most abundant in the Coeur d'Alene River basin during the spring migration. An estimated 270 tundra swans, 2,060 Canada geese, and 3,000 to 4,000 ducks were observed in the 1955 spring migration (Chupp and Dalke, 1964). Neufeld (1987) reported that flocks of 800 to 2,000 tundra swans and 2,000 to 10,000 Canada geese arrive in the basin during late February or early March, and remain for three to five weeks before flying to more northern breeding grounds. Blus et al. (1991) estimated a partial count of 900 tundra swans in the basin in 1987. Peak one-day waterfowl counts estimated during surveys in 1994 through 1997 were 3,758 tundra swans, 13,230 Canada geese, and 1,730 mallards (Audet et al., 1999c).

Birds of prey that inhabit the Coeur d'Alene River basin include bald eagle *(Haliaeetus leucocephalus)*, osprey *(Pandion haliaetus)*, American kestrel *(Falco sparverius)*, red-tailed hawk *(Buteo jamaicensis)*, northern harrier *(Circus cyaneus)*, sharp-shinned hawk *(Accipter striatus)*, northern goshawk *(Accipiter gentilis)*, great-horned owl *(Bubo virginianus)*, barred owl *(Strix varia)*, and western screech owl *(Otus kennicottii)*. Upland game birds such as ruffed grouse *(Bonasa umbrellus)*, California quail *(Callipepla californica)*, ring-necked pheasant *(Phasianus colchicus)*, and wild turkey *(Meleagris gallopavo)* also inhabit the floodplain and upland habitats.

Songbirds and other neotropical species that inhabit the Coeur d'Alene River basin include thrushes, sparrows, kingbirds, warblers, flycatchers, swallows, hummingbirds, and blackbirds. Amphibians present in the basin include Colombian spotted frogs *(Rana luteiventris)*, bullfrogs *(Rana catesbeiana)*, Pacific treefrogs *(Hyla regilla)*, western toads *(Bufo boreas)*, long-toed salamanders *(Ambystoma Macrodactylum)*, Giant salamanders *(Dicamptodon atterimus), and* tailed frogs *(Ascaphus truei)* (Beck et al., 1997; Howard et al., 1998).

The basin's riparian zones, wetlands, and lateral lakes also provide habitat for beaver *(Castor canadensis)*, mink *(Mustela vison)*, muskrat *(Ondatra zibethicus)*, raccoon *(Procyon lotor)*, and river otter *(Lutra canadensis)*. Larger mammals inhabiting the Coeur d'Alene River basin include black bear *(Ursus americanus)*, bobcat *(Felis rufus)*, cougar *(Felis concolor)*, coyote *(Canis latrans)*, elk *(Cervus elaphus)*, gray wolf *(Canis lupus)*, moose *(Alces alces)*, mule deer *(Odocoileus hemionus)*, and white-tailed deer *(Odocoileus virginianus)* (IDFG, 1987). Small mammals in the basin include meadow voles *(Microtus pennsylvanicus)*, shrews *(Sorex spp.)*, deer mice *(Peromyscus maniculatus)*, and others, which are hunted by birds of prey and larger mammals.

6.2 TOXIC EFFECTS OF LEAD ON WILDLIFE

Although other hazardous metals such as cadmium and zinc are present in the Coeur d'Alene River basin at elevated concentrations (see previous chapters discussing contamination in surface water and sediments) and can be toxic to wildlife, this review focuses on lead because (1) concentrations of lead in sediment and floodplain soils are extremely elevated, and (2) domestic and wildlife sicknesses and deaths in the basin have been diagnosed as lead poisoning (Chupp and Dalke, 1964; Neufeld, 1987; Blus et al., 1991; Audet et al., 1999c).

6.2.1 Literature Review

Exposure to lead can result in adverse effects to multiple tissues and organs that are critical to the viability and reproduction of wildlife (Figure 6-4). Lead affects hematological (blood), renal (kidney), muscular, behavioral, nervous, and reproductive systems (Eisler, 1988; Pain, 1996). Increasing exposure to lead typically results in an increase in the number and severity of adverse effects, from physiological malfunctions to physical deformations and eventually to death (Figure 6-5). Adverse effects of lead on wildlife include the following general categories: mortality and morbidity, disease, behavioral abnormalities, physiological malfunctions, and physical deformations (Table 6-1).

Mortality and morbidity. Clinical signs of lead poisoning in birds have been well documented in the scientific literature and include torpor; vomiting; impaction of esophagus, proventriculus, and gizzard with food leading to starvation; sloughing of gizzard; loss of coordination; accumulation of pericardial fluid; gall bladder enlargement/bile stains on gizzard lining, feces, and perianal plumage; anorexia, emaciation, and muscular atrophy; paralysis in wings and legs; loss of vision; convulsions; coma; and death (Trainer and Hunt, 1965; Venugopal and Luckey, 1978; Friend, 1987; Eisler, 1988; Franson, 1996; Pain, 1996). In general, nestlings are more sensitive than older life stages of birds, and severity of pathology increases with increasing lead exposure (Eisler, 1988).

Lead poisoning in mammals results in a similar suite of effects, including vomiting, loss of appetite, uncoordinated body movements, convulsions, stupor, coma, diarrhea, anemia, and blindness (WHO, 1995; Ma, 1996). Lead poisoning in amphibians includes sloughing of integument, sluggishness, and decreased muscle tone (Eisler, 1988). Death generally results from one or a combination of these physical and physiological impairments.

Lead can bioaccumulate in the tissues of prey organisms because it is excreted slowly, resulting in secondary poisoning of predators (Eisler, 1988).

Disease. Lead affects multiple organ systems of animals, resulting in a general decrease in health and susceptibility to disease or pathogen exposure (Eisler, 1988). Decreased host resistance to pathogens due to lead exposure has been demonstrated in laboratory tests with several species of mammals (McCabe, 1994). For example, sublethal lead exposure in mice reduced the resistance to bacterial infection by *Salmonella typhimurium* (Hemphill et al., 1971). Dietary lead (100 ppm) exposure altered immune responses of chicken, including phagocytosis and antibody titres (Hill and Oureshi, 1998). Although data are limited, lead appears to impair antibody production, reduce disease resistance, and increase mortality in animals infected with bacterial and viral agents (WHO, 1995).

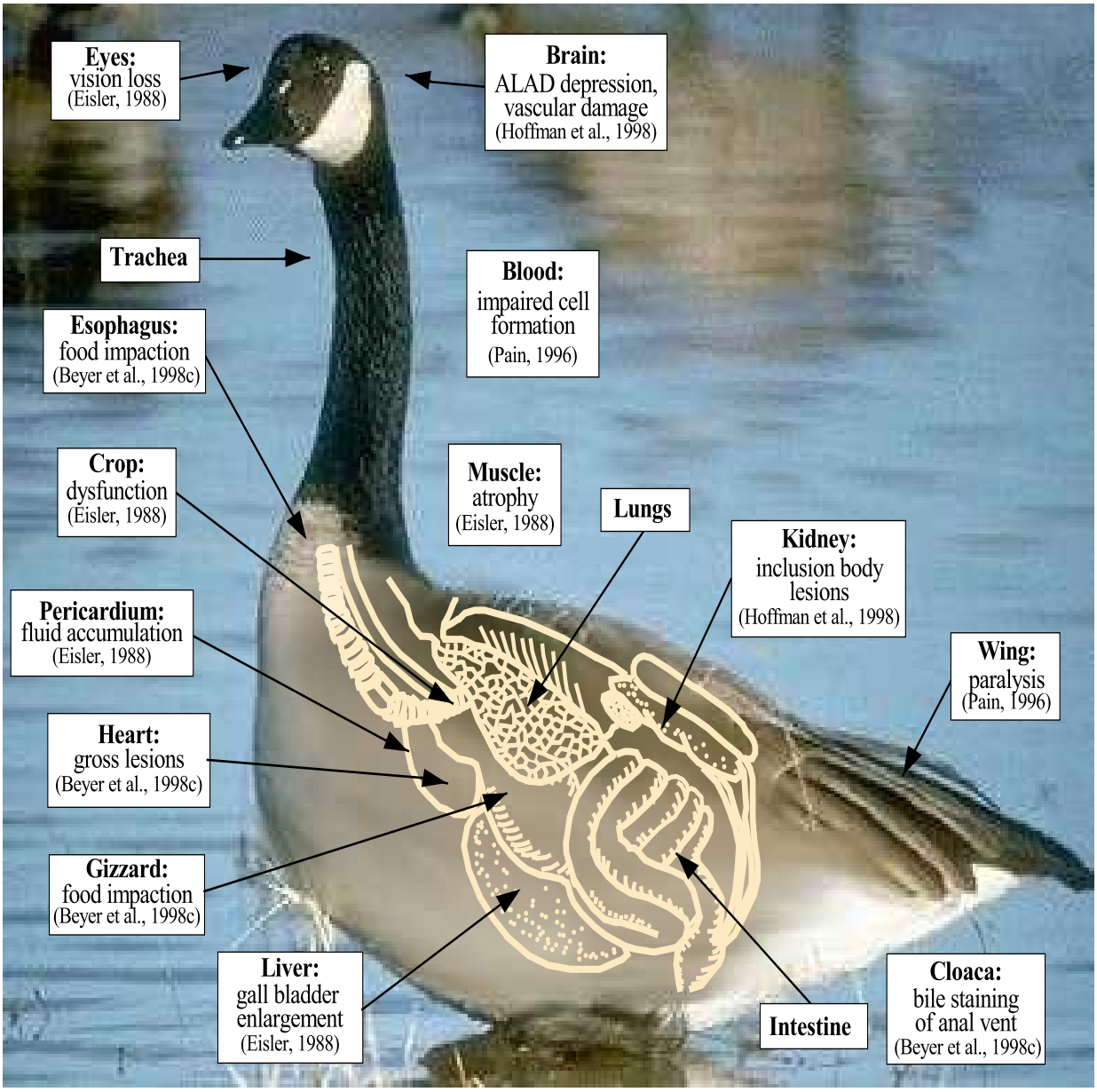


Figure 6-4. Generalized anatomy of birds showing major organs and tissues affected by lead exposure. Adapted from Hickman et al., 1974.

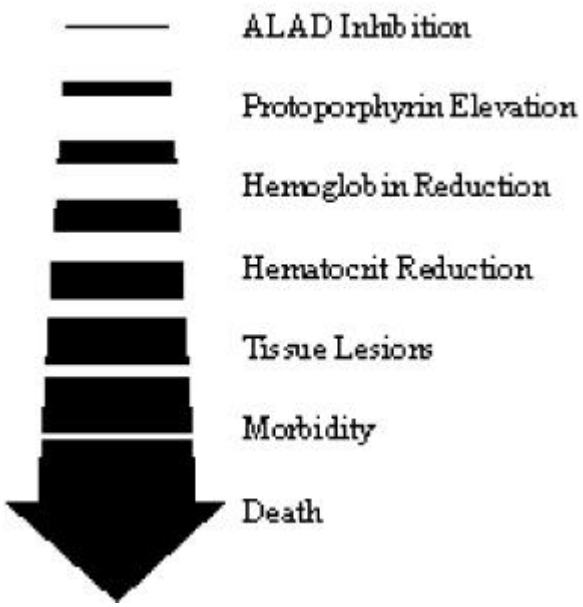


Figure 6-5. Increasing lead exposure results in an increase in the number and severity of adverse effects in wildlife from biochemical changes to death. ALAD is an enzyme involved in blood formation. Protoporphyrin, hemoglobin, and hematocrit also are components of blood that can be affected by lead.

Table 6-1
Adverse Effects of Lead on Wildlife

General Effect	Specific Effects	Information Sources
Mortality and morbidity	Torpor, vomiting, starvation, loss of coordination, paralysis in wings and legs, loss of vision, coma, death	Eisler (1988), Franson (1996), Pain (1996)
Disease	Impaired immunological responses; lower resistance to infections and pathogens	Hemphill et al. (1971), Hill and Oreshi (1998)
Behavioral abnormalities	Hyperactivity, impaired learning and memory, impaired avoidance behavior	Eisler (1988), Burger (1998)
Physiological malfunctions	Delta-aminovulinic acid dehydratase (ALAD) depression; weight loss, reproductive impairment	Dieter and Finley (1979), Hoffman et al. (1985), Eisler (1988), Pain (1996), Kelly et al. (1998)
Physical deformations	Impacted esophagus, emaciation, bile staining of gizzard lining, gall bladder enlargement, muscular atrophy; renal intranuclear inclusion bodies	Trainer and Hunt (1965), Kendall and Driver (1982), Eisler (1988), Franson (1996), Pain (1996)

Behavioral abnormalities. Lead can cause neurotoxicity and behavioral impairments in amphibians, birds, and mammals, including disruption of social behavior, hyperactivity, distractibility, impaired learning ability, and impaired predator avoidance. For example, Burger (1998) reported impaired sibling recognition in herring gulls experimentally exposed to lead. In rats, lead exposure alters development, affects specific motor skills, and can result in long-term cognitive deficits (Kuhlmann et al., 1997; Mello et al., 1998). Acutely toxic doses of lead may cause loss of coordination and paralysis. Retarded brain growth of laboratory mammals has also been reported (Eisler, 1988). Laboratory studies with mammalian species have demonstrated that lead impairs learning and memory functions in nearly all life stages of animals (WHO, 1995). Sublethal lead exposure during early development of animals produces behavioral change and deficits in learning ability that persist beyond the period of exposure (Rice, 1985; Rice and Karpinski, 1988; Ma, 1996; ATSDR, 1997; Stewart et al., 1998). Eisler (1988) concluded that lead causes neurobehavioral deficits such as learning impairment at very low blood lead levels, and at levels less than those that cause more overt signs of toxicity. The ecological importance of lead induced behavioral abnormalities may include death of wildlife caused by increased susceptibility to predation, and reduced reproductive success from altered nest building, parenting behavior, or maternal imprinting (Eisler, 1988; Lefcort et al., 1998).

Physiological malfunctions. Physiological malfunctions caused by lead exposure include hematological responses associated with inhibition of the formation of red blood cells, impairment of renal function, weight loss, and impaired reproduction.

Hematological responses are among the first measurable biochemical changes in animals exposed to lead, including inhibition of delta-aminolevulinic acid dehydratase (ALAD) activity, elevation of protoporphyrin, and reductions in hemoglobin and hematocrit levels. Increasing lead exposure leads to a cascade of biochemical changes measurable in blood, from ALAD inhibition, to protoporphyrin elevation, to reduction in hemoglobin and hematocrit, and ultimately to reduced viability of the animal from the reduced capacity to transport oxygen to the brain and other tissues (Figure 6-6).

Inhibition of blood ALAD activity after exposure to lead has been demonstrated in multiple species of wildlife (Pain, 1996). ALAD inhibition also occurs in brain, spleen, liver, kidney, and bone marrow (Hoffman et al., 1985). Anemia, or reduced hematocrit and hemoglobin resulting from inhibition of the enzymes ALAD and ferrochelatase involved in hemoglobin synthesis (Figure 6-6), may occur following a >75% inhibition of ALAD activity (Hoffman et al., 2000). ALAD inhibition by lead in mallard ducks has been associated with an increase in brain levels of the enzyme butrylcholinesterase. The increase in butrylcholinesterase may cause destruction of neural cells in the cerebellum (Dieter and Finley, 1979). Even a partial loss of cerebellum tissue is severely debilitating in waterfowl because functions critical to survival (e.g., visual, auditory, motor, and reflex responses) are integrated in this region of the brain (Dieter and Finley, 1979).

Lead exposure impairs kidney function by accumulating in the proximal convoluted tubule cells of the renal cortex in mammals, resulting in waste product (urea and uric acid) accumulation in the blood (Quarterman, 1986).

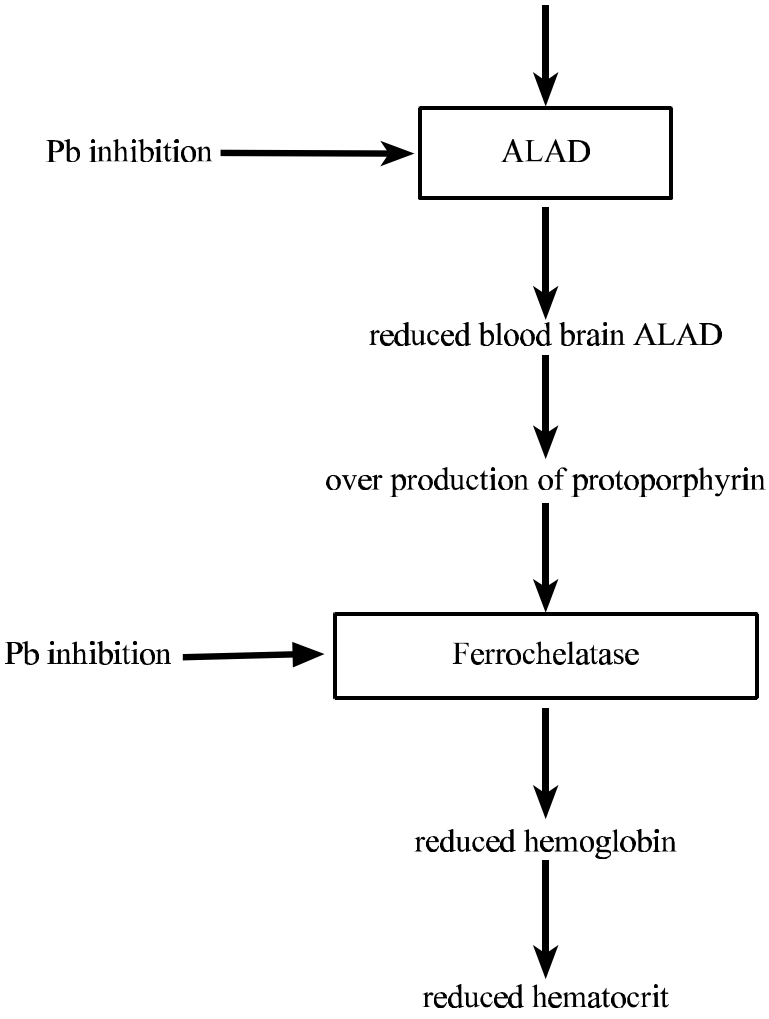


Figure 6-6. Biochemical pathway showing lead effects on blood formation.

The growth and development of animals also can be impaired following toxic exposure to lead. Emaciation is a common effect observed in lead poisoned waterfowl (Beyer et al., 1998c). Altered growth can affect the viability and reproductive success of birds (O'Connor, 1984), and delayed development may preclude metamorphosis of amphibians living in temporary water bodies (Lefcort et al., 1998).

Lead can impair wildlife reproduction at very low dietary exposures. For example, Edens et al. (1976) observed reduced egg production in Japanese quail *(Coturnix coturnix)* at dietary concentrations of lead between 1 and 100 ppm.

Physical deformations. Physical deformations include both external signs and gross pathological lesions of lead poisoning (Wetmore, 1919; Trainer and Hunt, 1965; Cook and Trainer, 1966; Bagley et al., 1967; Karstad, 1971; Sileo et al., 1973; Clemens et al., 1975; Wobeser, 1981; Pain, 1992; Locke and Thomas, 1996).

Physical deformations of lead-poisoned waterfowl include impaction of the esophagus, proventriculus, or gizzard with food (Figure 6-7), leading to starvation, emaciation, and atrophy of skeletal muscles and viscera. Lead exposure causes roughening or sloughing of the lining of the gizzard, gall bladder engorgement, and regurgitating and bile staining of the gizzard lining, feces, and the perianal plumage. Excessive pericardial fluid may accumulate, and the heart may develop white streaks that are presumed to be necrotic tissue and are associated with abnormal heart function. Tissues in general may appear pale, suggesting anemia, and the subcutaneous tissues of the submandibular area may become edemic.

Lead causes histological deformations such as interstitial fibrosis, edema, formation of acid-fast renal intranuclear inclusion bodies (RIIBs; Figure 6-8), and hemosiderosis in the liver and kidney. RIIBs, which are diagnostic of lead poisoning, are kidney lesions containing a lead-protein complex in the cell nucleus (L. Sileo, USGS-BRD, National Wildlife Health Center, pers. com., December 10, 1999). RIIBs can be seen under a microscope when treated with an acid-fast stain. Pathogens may cause other kinds of inclusion bodies in cells, but they do not react with the acid-fast stain (Locke et al., 1966). Hemosiderosis is the presence of excessive amounts of the iron-containing pigment hemosiderin (Figure 6-8). Hemosiderin results from the metabolic breakdown of hemoglobin from red blood cells (L. Sileo, USGS-BRD, National Wildlife Health Center, pers. com., December 10, 1999). Lead also causes blood vessel damage in the brain, nerve cell and ganglia damage, and demyelinating lesions (loss of nerve cell sheath) (Eisler, 1988).

Lead concentrations associated with toxicity. According to the scientific literature, blood concentrations greater than 0.5 ppm and liver concentrations of 6 to 15 ppm (wet weight) have been associated with clinical poisoning in birds, including overt signs of poisoning such as muscle wasting, weakness, anemia, and incoordination (Table 6-2). Clinical poisoning in mammals occurs at similar tissue levels as in birds. For example, Ma (1996) suggested that clinical poisoning in mammalian species occurs at blood lead levels of greater than 0.35 ppm to greater than 0.6 ppm, which is within the range of blood levels causing clinical poisoning in birds (0.5 to >5 ppm; Table 6-2). Liver lead concentrations of greater than 10 ppm wet weight are often associated with clinical signs of lead poisoning (Zook et al., 1972; Osweiler et al., 1978; Ma, 1996), although signs of lead poisoning have occurred at lower liver lead concentrations (Clarke, 1973; Osweiler et al., 1978; Ma, 1996). Kidney lead concentrations greater than 27 ppm wet weight also are associated with clinical signs of lead poisoning (Ma, 1996). Liver lead concentrations greater than 3 ppm wet weight and kidney lead concentrations greater than 7.5 ppm wet weight are considered diagnostic for lead poisoning in wild mammals (Ma, 1996). These lead concentrations are also within the range reported for birds (6 to 20 ppm; Table 6-2). Blood concentrations greater than 1 ppm and liver concentrations greater than 15 ppm are associated with death and morbidity in birds (Table 6-2).

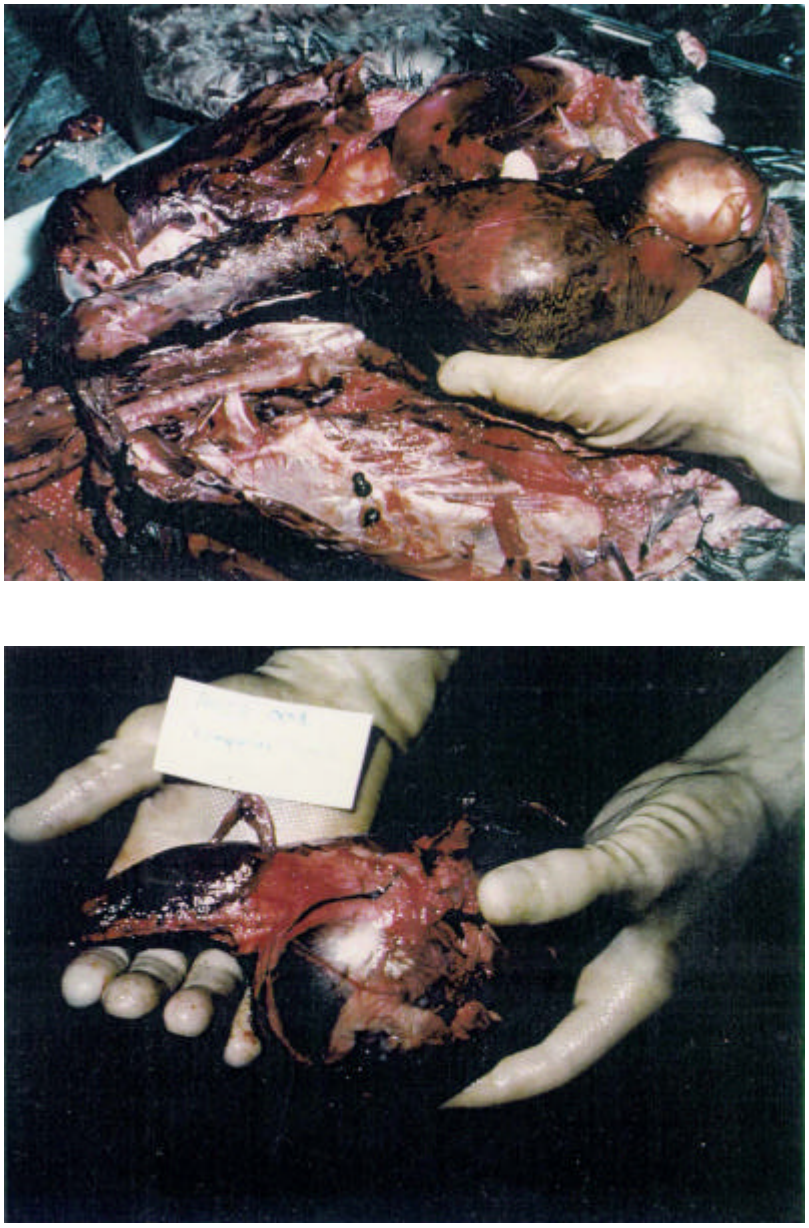


Figure 6-7. Top: Distended esophagus and proventriculus of a lead-poisoned Canada goose from the Coeur d'Alene Basin, ID. The proventriculus (in the gloved hand) is abnormally packed with food. Liver lead: 15.02 ppm. Photo date: April 15, 1997. **Bottom: Normal esophagus and proventriculus of a trumpeter swan from Wisconsin.** Cause of death unknown. Liver lead: below detection. Photo date: December 9, 1998.
Source: L. Sileo, USGS, National Wildlife Health Center.

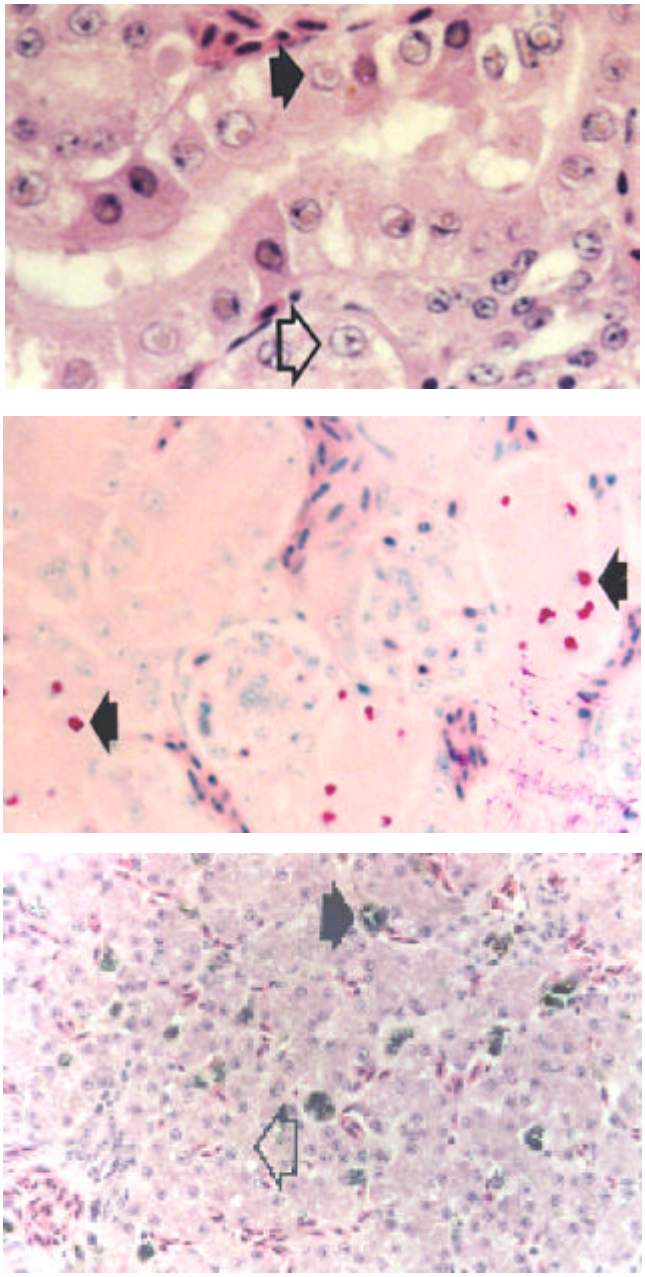


Figure 6-8. Histological deformations. Top: Microscopic section of a kidney (hematoxylin/eosin stained) from a mute swan (*Cygnus olor*) experimentally fed Coeur d'Alene River sediment (Day et al., 1998). Open arrow marks normal nuclei and closed arrow marks renal intranuclear inclusion body (RIIB). Middle: Microscopic section of kidney (acid fast stain) from a mallard duck experimentally fed Coeur d'Alene sediment (Heinz et al., 1999); closed arrows mark RIIBs. Bottom: Liver from mute swan (hematoxylin/eosin stained) experimentally fed sediment from the Coeur d'Alene River (Day et al., 1998). The prominent, irregular, dark masses (closed arrow) are deposits of hemosiderin. In many other liver cells are tiny brown granules (open arrow) that are also hemosiderin deposits. Hemosiderin is present in normal livers, but deposits this prominent are abnormal.
Source: L. Sileo, USGS, National Wildlife Health Center.

Table 6-2
Lead Concentrations (ppm, wet weight) in Bird Tissues Associated with Toxicity

Tissue	Poisoning Category	Waterfowl (Pain, 1996)	Birds of Prey (Franson, 1996)	Doves, Pigeons (Franson, 1996)	Quail, Pheasant (Franson, 1996)
Blood	No effects	< 0.2	< 0.2	< 0.2	< 0.2
	Subclinical[a]	0.2 to 0.5	0.2 to 1.5	0.2 to 2.5	0.2 to 3
	Clinical/toxic[b]	0.5 to 1	> 1	> 2	> 5
	Severe clinical/death[c]	1	> 5	> 10	> 10
Liver	No effects	< 2	< 2	< 2	< 2
	Subclinical	2 to 6	2 to 4	2 to 6	2 to 6
	Clinical/toxic	6 to 15	> 3	> 6	> 6
	Severe clinical/death	> 15	> 5	> 20	> 15
Kidney[d]	No effects	-	< 2	< 2	< 2
	Subclinical	-	2 to 5	2 to 20	2 to 20
	Clinical/toxic	-	> 3	> 15	> 15
	Severe clinical/death	-	> 5	> 40	> 50

a. Physiological effects (e.g., ALAD depression) without overt signs of poisoning.
b. Overt signs of poisoning, including muscle wasting, weakness, anemia, incoordination.
c. Mortality and morbidity.
d. Kidney: birds of prey; doves, pigeons; quail, pheasant.

Reproduction is impaired at dietary levels between 1 and 100 ppm lead in sensitive bird and mammal species (Eisler, 1988). For example, Edens et al. (1976) observed reduced egg production in Japanese quail *(Coturnix coturnix)* at dietary concentrations between 1 and 1,000 ppm lead. Pattee (1984) observed no effect on reproduction of American kestrels at dietary concentrations of 10 and 50 ppm. Lowest observed adverse effect level (LOAEL)[1]-based toxicity benchmarks for lead proposed by Sample et al. (1996) ranged from 9.4 to 1,182 ppm in diet for bird and mammal species.

1. LOAEL-based toxicity benchmarks represent lead-ingestion thresholds at which adverse effects are likely to become evident (Sample et al., 1996).

6.2.2 Data Collected Previously in the Assessment Area

There have been numerous reports of the environmental contamination, exposure, and adverse effects of metals on wildlife resources of the Coeur d'Alene River basin (Table 6-3).

Table 6-3
Chronology of Documentation of Exposure and Effects of Hazardous Substances in Coeur d'Alene Wildlife[a]

Observation Year	Species	Observations	Information Source
1924-1956	Waterfowl[b]	Exposure, deaths	Chupp and Dalke (1964)
1924-1986	Waterfowl	Exposure, deaths, lesions	Neufeld (1987)
1931	Tundra swan	Deaths	Bureau of Biological Survey (1931)
1955	Waterfowl, birds of prey	Deaths; eagle predation	Chupp (1956)
1971	Waterfowl	Deaths	Bruner (1971)[f]
1974	Tundra swan	Deaths	Benson et al. (1976)
1975	Mice, vegetation	Exposure	Herman et al. (1975)
1981-1983	Mammals[c]	Exposure	Blus et al. (1987)
1982-1989	Waterfowl, mammals, vegetation	Exposure, deaths	Krieger (1990)
1982	Waterfowl	Exposure, lesions	Stroud (1982)
1986-1987	Mink	Exposure	Blus and Henny (1990)
1986-1987	Wood duck	Exposure, sublethal effects	Blus et al. (1993)
1986-1987	Osprey	Exposure, sublethal effects	Henny et al. (1991)
1986-1987	Birds of prey,[d] mammals	Exposure	Henny et al. (1994)
1987	Waterfowl, songbirds[e]	Exposure, sublethal effects	Blus et al. (1995)
1987-1989	Tundra swan	Exposure, deaths, lesions	Blus et al. (1991)
1993	Waterfowl	Exposure, sublethal effects	Mullins and Burch (1993)
1994-1995	Aquatic biota, mallard duck	Exposure, sublethal effects	Burch et al. (1996)
1994-1995	Tundra swan	Exposure, deaths, lesions	Blus et al. (1999)
1994-1995	Mammals	Exposure	Szumski (1999)
1997	Waterfowl	Exposure	Audet et al. (1999a)

a. Adapted from Audet, 1997, which provides complete documentation.
b. Multiple waterfowl species (e.g., tundra swans, mallards).
c. Multiple mammal species (e.g., mink, deer mice, muskrat).
d. Multiple birds of prey species (e.g., American kestrel, red-tailed hawk, western screech owl).
e. Multiple songbird species (e.g., tree swallow, American robin).
f. Waterfowl Mortality on the Lower Coeur d'Alene River. Idaho Department of Fish and Game. Unpublished Report. 16 pp.

Note: Supplemental studies conducted by the Trustees between 1992 and 1997 provide additional documentation of exposure, deaths, lesions, and sublethal effects (summarized in Section 6.5).

Migratory Bird Exposure and Effects

Waterfowl deaths in the Coeur d'Alene River basin have been reported since 1924, and bird carcasses collected from the basin consistently have shown evidence of lead exposure and lesions indicative of lead poisoning (e.g., Chupp and Dalke, 1964; Neufeld, 1987). Waterfowl deaths in the basin have been investigated and reported by various agencies and university researchers, including the Bureau of Biological Survey (1931); Idaho Department of Health and Welfare (Benson et al., 1976); Idaho Department of Fish and Game (Bruner, 1971; Neufeld, 1987); U.S. Bureau of Sport Fisheries and Wildlife (Chupp and Dalke, 1964); U.S. DOI Bureau of Land Management, Washington State University, and University of Idaho (Krieger, 1990); and the U.S. Fish and Wildlife Service (Blus et al., 1991; Audet et al., 1999c).

In 1948, about 100 of an estimated 400-600 swans died in the basin, despite attempts by wildlife biologists to disperse them from the area (Chupp and Dalke, 1964). In 1954 and 1955, Chupp and Dalke (1964) recorded dead waterfowl in the Coeur d'Alene River basin, including tundra swans, Canada geese, eight species of duck, and American coots. They concluded that the dead tundra swans they examined had died from lead poisoning and that the source of the lead was the river sediments laden with mine waste that coated the aquatic vegetation eaten by the waterfowl. Chupp and Dalke (1964) suggested that waterfowl mortality increased in the Coeur d'Alene River basin when weather conditions caused migrating birds to stay longer in the area.

In 1974, Benson et al. (1976) found 13 lead-poisoned tundra swans at Mission Slough and concluded that their exposure and death resulted from ingestion of lead-contaminated vegetation. Five of six waterfowl carcasses from the Coeur d'Alene River basin examined by Stroud (1982) were diagnosed as lead poisoned without ingested lead shot. An estimated 200 tundra swans (17%) of a group of 1,200 died in the basin in 1982 (Krieger, 1990). The swans examined were diagnosed with lead poisoning based on emaciation, engorged gall bladder, impacted proventriculus and gizzard, empty gastrointestinal tracts with bile, and toxic levels of lead in tissues (Krieger, 1990). Thirty-two dead tundra swans diagnosed as lead poisoned were collected from the basin between 1987 and 1989 (Blus et al., 1991). Neufeld (1987) reported swan mortality throughout the Coeur d'Alene River Wildlife Management Area (lower Coeur d'Alene River basin) associated with the deposition of contaminated sediment on vegetation by high water just before the spring waterfowl migration.

Blus et al. (1995) measured highly elevated lead concentrations in blood and livers of American robins from the Coeur d'Alene River basin, compared to concentrations in American robins from reference areas. Liver lead concentrations were slightly elevated in tree swallows *(Tachycineta bicolor)*. Blus et al. (1995) found that lead had accumulated to potentially toxic levels in nestling robins (maximum concentrations of 0.87 µg/g in blood and 5.6 µg/g in liver) and mallards (maximum concentrations of 10.2 µg/g in blood and 2.8 µg/g in liver).

Studies of the physiological effects of lead on birds of prey in the Coeur d'Alene River basin (1986-1987) indicated that despite the fact that raptors in the basin are less exposed to lead than waterfowl, some still exhibit reductions in ALAD activity greater than 50% (Henny et al., 1991; Henny et al., 1994). Adult and nestling osprey *(Pandion haliaetus)* along the Coeur d'Alene River had elevated blood lead concentrations and exhibited greater than 50% reduction in ALAD activity compared to osprey from reference areas (Henny et al., 1991). In addition, ALAD activity was reduced by 35% in nestling northern harriers *(Circus cyaneus)*, by 55% in nestling American kestrels *(Falco sparverius)*, and by 81% in adult American kestrels of the Coeur d'Alene River basin (Henny et al., 1994).

In 1995, Burch et al. (1996) reported blood lead concentrations ranging from 0.29 to 1.37 ppm (mean 0.85 ppm) in mallard ducks from the Page Pond wetlands (Bunker Hill Superfund Site). Blood lead concentrations in mallards collected in 1997 from the Page Pond Wastewater Treatment Plant ranged from 0.67 to 10 ppm (mean 2.68 ppm) (Audet et al., 1999a). Audet et al. (1999a) found that the mean blood lead concentration in adult mallard ducks (3.7 ppm) was twice that of hatch year mallards (1.73 ppm). Blood lead concentrations in mallard ducks collected in 1997 from the Page Ponds Wastewater Treatment Plant area were significantly greater ($p = 0.0095$) than blood lead concentrations at the same site in 1995 (mean 0.846 ppm, range 0.29 to 1.37 ppm) (Audet et al., 1999a).

Mammal Exposure and Effects

Studies of lead concentration in wild mammals of the Coeur d'Alene River basin have shown elevated concentrations of lead in tissues and ingesta of muskrats, mink, raccoons, beaver, deer, voles (*Microtus* spp.), and deer mice (Blus et al., 1987; Blus and Henny, 1990; Audet, 1997; Szumski, 1999).

Liver and kidney lead concentrations in mink collected from the lateral lakes of the Coeur d'Alene River basin were elevated relative to concentrations in mink from reference sites (Blus et al., 1987; Blus and Henny, 1990; Szumski, 1999). Liver lead concentrations in lower Coeur d'Alene River basin mink were significantly greater ($p < 0.05$) than concentrations in mink collected from the North Fork Coeur d'Alene River and from Washington. Liver lead concentrations averaged 4.1 and 3.2 ppm wet weight in 1981-82 and 1986-1987, respectively, and ranged up to 34 ppm wet weight, which was the highest liver lead concentration ever recorded in a mink (Blus et al., 1987; Blus and Henny, 1990). Liver lead concentrations were positively correlated with lead concentrations of stomach contents, which ranged up to 51 ppm. Blus and Henny (1990) concluded that lead concentrations in mink in the Coeur d'Alene River basin were sufficient to cause adverse effects.

Tissue concentration data indicated no decrease in exposure to lead of mink between 1981 and 1987 ($p > 0.05$) (Blus et al., 1987; Blus and Henny, 1990), and mean liver lead concentrations in juvenile mink collected from the lateral lakes in 1994 and 1995 (Szumski, 1999) and adult mink collected in 1996 (National Wildlife Health Center Necropsy Report #WM96CO83, USGS-BRD, Madison, WI) (2.1 ppm wet weight) were only slightly lower than the 1980s averages.

Mean lead concentrations in mink livers from the Coeur d'Alene River basin (2 to 4 ppm wet weight) were much greater than mean concentrations reported in mink from state-wide surveys in Virginia (0.05 ppm wet weight; Ogle et al., 1985) and New York (0.27 ppm wet weight; Foley et al., 1991), and across Ontario (0.10 to 0.35 ppm wet weight; Wren et al., 1988), including areas affected by mining and smelting. Lead concentrations in Coeur d'Alene River mink were also much greater than concentrations measured in mink collected downstream of a copper mining and smelting region in Montana (mean 0.26 ppm wet weight; Szumski, 1998).

Geometric mean lead concentrations in the livers of muskrat collected from the Coeur d'Alene River basin (n = 72) ranged from 0.2 ppm wet weight to 1.5 ppm wet weight (Blus et al., 1987; Krieger, 1990; Audet, 1997; Szumski, 1999). The maximum liver lead concentration (16.3 ppm wet weight) was reported in a muskrat collected during the 1994-1995 trapping season from the lateral lakes area (Szumski, 1999). Lead concentrations in muskrat tissues reported by both Blus et al. (1987) and Szumski (1999) greatly exceeded concentrations in muskrats collected at reference sites on the Big Hole River in Montana (mean liver lead 0.04 ppm wet weight).

Mean liver lead concentrations in Coeur d'Alene River muskrats (1.13 ppm wet weight) were also much greater than concentrations in livers of muskrats collected in the Missouri lead mining district (0.69 ppm wet weight; Niethammer et al., 1985), areas of Pennsylvania (0.002 to 0.15 ppm wet weight; Everett and Anthony, 1976), and near an ore smelter in Manitoba (0.16 ppm wet weight; Radvanyi and Shaw, 1981), but were lower than concentrations reported in muskrats from a tidal marsh in Pennsylvania receiving industrial and municipal wastes (3.7 to 5.3 ppm wet weight; Erickson and Lindzey, 1983).

Beaver collected from the Coeur d'Alene River as part of a reconnaissance study that preceded injury studies (Audet, 1997) had a mean liver lead concentration of 1.32 ppm wet weight, which is similar to liver lead concentrations in muskrats from the same area (Szumski, 1999). The mean liver lead in Coeur d'Alene River beaver was also similar to concentrations reported in beaver collected near a metal smelter in Ontario (2.7 ppm wet weight; Hillis and Parker, 1993).

Liver lead concentrations means were also elevated in Coeur d'Alene River basin raccoons (1.10 ppm wet weight) compared to concentrations in reference raccoons from an undisturbed area in Montana (0.07 ppm wet weight; Szumski, 1999).

Deer kidneys collected in 1987 and 1988 near the former Bunker Hill smelters and along the South Fork Coeur d'Alene River contained significantly elevated ($p < 0.05$) lead (1.7 ppm wet weight) compared to deer from reference areas (1.08 ppm wet weight; Dames & Moore, 1990).

Herman et al. (1975) evaluated whole body metal concentrations and species abundance and diversity of small mammals with distance from the smelters near Kellogg. Lead concentrations in deer mice collected near the smelters were greatly elevated relative to concentrations in deer mice from reference sites to the north and south of the smelters. In mice collected within 5 miles of the smelters, geometric mean concentrations of whole body lead ranged from 7.3 to 332.5 ppm wet weight. Geometric mean lead concentration in reference mice was <5 ppm wet weight. Small

mammal diversity increased with distance from the smelters; this was attributed to the adverse effects of metals on plant diversity.

Deer mice and voles collected near tailings ponds in the Kellogg area in 1982 and 1983 (shortly after the closure of the smelters) contained greatly elevated whole body, kidney, and liver concentrations of lead (Blus et al., 1987). Whole body lead concentrations in deer mice averaged 55.3 ppm wet weight (geometric mean) and in voles, 54.7 ppm wet weight (geometric mean). Deer mice and voles collected along the South Fork and mainstem Coeur d'Alene rivers between Kellogg and Thompson Lake in 1986 also contained greatly elevated whole body lead concentrations (geometric mean >40 ppm wet weight in deer mice; Henny et al., 1994). Concentrations declined with distance downstream from Kellogg, but even as far as 40 km and 60 km downstream from the smelter, concentrations in whole body deer mice still averaged 22.8 ppm wet weight and 19 ppm wet weight, respectively.

Whole body and kidney lead concentrations were also significantly elevated in deer mice collected in 1988 near the smelter and in the Kellogg area relative to mice collected from a reference area ($p < 0.01$; Dames & Moore, 1990). Kidneys in several mice collected near the smelter evidenced renal pathologies consistent with damage produced by lead and cadmium, but no intranuclear inclusion bodies were observed.

Audet (1997) measured whole body lead concentrations in voles collected in 1992 from Kellogg and from Killarney Lake. Mean lead concentrations (13.7 ppm wet weight) were significantly greater ($p < 0.0001$) than lead concentrations in voles from the St. Joe River (0.36 ppm wet weight). Mean concentrations of liver lead (1.43 ppm wet weight) were also greater in deer mice collected from the Coeur d'Alene River than in deer mice collected from the St. Joe River (0.25 ppm wet weight).

Mean whole body concentrations of lead in deer mice collected from several sites in the vicinity of the smelters at Kellogg ranged from 7.3 to 111.5 ppm wet weight (Herman et al., 1975; Dames & Moore, 1990; Henny et al., 1994) and were 8 to 124 times concentrations reported for deer mice from an unmined area in Wisconsin (<0.1 to 0.9 ppm wet weight; Smith and Rongstad, 1981). Mean whole body concentrations of lead in voles collected from the Coeur d'Alene River basin (2.6 to 54.7 ppm wet weight; Blus et al., 1987; Henny et al., 1994; Audet, 1997) were 4 to 78 times concentrations reported for voles from an unmined area in Wisconsin (0.45 to 0.7 ppm wet weight; Smith and Rongstad, 1981). While lead concentrations in the Kellogg area apparently have declined, they still remain greatly elevated.

Amphibian Exposure and Effects

Tadpoles collected from East Page Swamp (Bunker Hill Superfund Site) contained 271 ppm (dry weight) of lead, which is substantially greater than lead concentrations in tadpoles from uncontaminated sites (e.g., 14 to 23 ppm dry weight; Mullins and Burch, 1993). Lefcort et al. (1998) and Lefcort et al. (1999) observed reduced survival, reduced growth and altered development (delayed metamorphosis), and behavioral abnormalities (altered predator avoidance

and competitive interactions) in amphibians (spotted frog *Rana luteiventris* tadpoles) exposed to stream bank soil from the Coeur d'Alene River basin.

6.3 INJURIES EVALUATED IN THE ASSESSMENT AREA

Injuries to wildlife resources in the Coeur d'Alene River basin were assessed in accordance with the DOI guidance for determination of injury to biological resources [43 CFR § 11.62 (f)(4)]. The following injury categories were evaluated: death [43 CFR § 11.62 (f)(4)(i)], physiological malfunctions [43 CFR § 11.62 (f)(4)(v)], and physical deformation [43 CFR § 11.62 (f)(4)(vi)]. These injuries were selected for assessment because existing information indicated that lead was the cause of observed bird mortalities in the basin and that lead had caused physiological malfunctions such as ALAD inhibition, and because lead is known to cause these types of adverse effects in wildlife, as discussed in Section 6.2.1.

Assessment of associated injuries to the supporting ecosystem (surface water, sediments, riparian resources) is described in separate chapters of this report.

Injury Category: Death

The following injury definitions apply:

- Injury has occurred when a significant increase in the frequency or number of dead or dying birds can be measured in a population sample from the assessment area as compared to a population sample from a control area [43 CFR § 11.62 (f)(4)(i)(C)].

To address this injury definition, the number and frequency of dead and dying birds were determined during field investigations in both the lower Coeur d'Alene River basin and reference areas. Field investigations included waterfowl and mortality surveys and laboratory diagnosis of the cause of wildlife deaths.

- Injury has occurred when a statistically significant difference can be measured in the total mortality and/or mortality rates between population samples of test organisms placed in laboratory exposure chambers containing concentrations of hazardous substances and those in a control chamber [43 CFR § 11.62 (f)(4)(i)(E)].

Toxicity tests were conducted under controlled laboratory conditions. The response of birds exposed to Coeur d'Alene River basin sediments was compared to the response of birds exposed to reference area sediment.

Injury Category: Physiological Malfunctions

The following injury definition applies:

- Injury has occurred when the activity level of whole blood ALAD in a sample from the population of a given species at an assessment area is significantly less than mean values for a population at a control area, and ALAD depression of at least 50% can be measured [43 CFR § 11.62 (f)(4)(v)(C)].

  Blood ALAD activity in waterfowl, birds of prey, and songbirds in the Coeur d'Alene and reference areas was quantified. Controlled laboratory studies were conducted to determine the relationship between waterfowl ingestion of Coeur d'Alene sediment, lead exposure, and ALAD depression.

Additional physiological malfunctions assessed included parameters indicative of impaired blood formation (protoporphyrin elevation, hemoglobin suppression, and hematocrit reduction) and weight loss.

- ***Protoporphyrin elevation.*** This chemical becomes elevated in blood following lead exposure because lead inhibits the enzyme ferrochelatase (also known as heme synthetase) (Figure 6-6). Normally ferrochelatase converts protoporphyrin to heme, which is a step in the biochemical pathway to formation of hemoglobin. In the presence of lead, the ferrochelatase enzyme is inhibited, the conversion of protoporphyrin is reduced, and protoporphyrin levels in blood become elevated.

- ***Hemoglobin suppression.*** Hemoglobin is the component of blood that carries and transfers oxygen to the cells of animals. Lead exposure decreases hemoglobin levels through the blockage of the biochemical pathway producing heme (Figure 6-6). Inhibition of the enzyme ferrochelatase reduces the amount of heme available for conversion to hemoglobin, eventually causing anemia.

- ***Hematocrit reduction.*** Hematocrit is an index of the red blood cell content of blood, and is measured by determining the packed cell volume (primarily red blood cells) of a blood sample. Lead exposure causes a decrease in hematocrit (Figure 6-6). Hematocrit reduction lowers the oxygen carrying capacity of blood, which can result in anemia and tissue hypoxia.

Blood levels of protoporphyrin, hemoglobin, and hematocrit were quantified in multiple species of wildlife collected from the Coeur d'Alene River basin and reference areas, including waterfowl, songbirds, and birds of prey. In addition, controlled laboratory studies were conducted to determine the relationship between waterfowl ingestion of Coeur d'Alene sediment, lead exposure, and changes in these blood parameters.

- ***Weight loss.*** Changes in body weight of waterfowl exposed to Coeur d'Alene River basin sediment in controlled laboratory studies were assessed because loss of body weight can affect the viability and reproductive success of birds (O'Connor, 1984). In addition, the growth of juvenile bald eagles in a field investigation was assessed by comparing the increase in body weight of eaglets from nests in the Coeur d'Alene River basin to a reference area nest.

Although not specifically identified as injury categories in the DOI regulations at [43 CFR § 11.62 (f)(4)] (with the exception of ALAD inhibition), the physiological malfunction responses described above satisfy the four acceptance criteria for injury outlined at [43 CFR § 11.62(f)(2)(i-iv)]. Specifically, the measured biological responses are:

- ***Often the result of exposure to hazardous substances, as shown in scientific studies.*** Numerous studies have shown that parameters related to blood formation are altered by lead exposure, and that lead exposure causes elevation of protoporphyrin, hemoglobin suppression, and hematocrit reduction (Eisler, 1988; Pain, 1996; Kelly et al., 1998). Numerous scientific studies have demonstrated that changes in body weight (e.g., emaciation) are caused by lead exposure in both laboratory and field studies (Eisler, 1988; Franson, 1996; Pain, 1996). These biological responses are known to be the result of lead exposure and to increase in severity with increase in lead exposure.

- ***Caused in free-ranging organisms by exposure to hazardous substances.*** Numerous field investigations have demonstrated blood protoporphyrin elevation, hemoglobin suppression, and hematocrit reduction in wildlife populations exposed to lead (Eisler, 1988; Pain, 1996). Numerous studies have demonstrated that changes in body weight are caused by lead exposure in both the laboratory and the field (Eisler, 1988; Franson, 1996; Pain, 1996).

- ***Found in controlled laboratory experiments by exposure to hazardous substances.*** Numerous controlled laboratory studies have shown that parameters related to blood formation are altered by lead exposure, including elevation of protoporphyrin, hemoglobin suppression, and hematocrit reduction (Eisler, 1988; Pain, 1996). Growth impairment and weight loss have also been observed in controlled laboratory feeding studies with lead (e.g., Edens et al., 1976; WHO, 1995).

- ***Demonstrated by routine measurements that are practical to perform and produce scientifically valid results.*** The procedures used to collect and analyze protoporphyrin, hemoglobin, and hematocrit are standard methods that have been used for years by wildlife biologists and toxicologists (see citations in proceeding paragraphs). Growth measurements, quantified as change in the weight or length of specific body parts or the whole animal, are also routine and simple procedures used in ecology and toxicology. For both laboratory tests and field investigations, written protocols, standard procedures, and quality assurance plans were used, instruments were calibrated before use and regularly during use, and quality assurance/quality control procedures were followed.

Injury Category: Physical Deformation

The following injury definition applies:

- A statistically significant difference can be measured in the frequency of tissue or cellular lesions when comparing samples from populations of wildlife species from the assessment area and a control area [43 CFR § 11.62 (f)(4)(vi)].

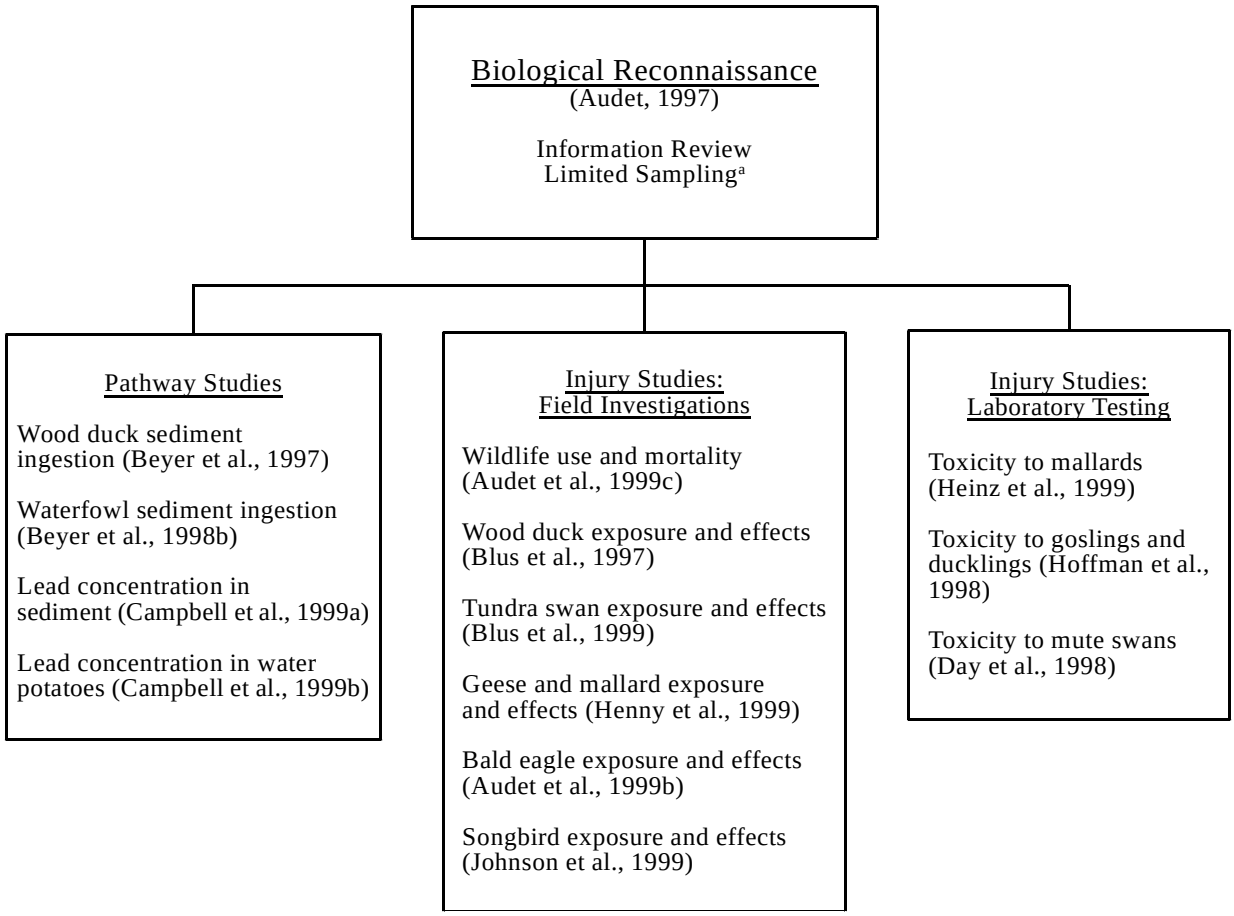
The assessment of physical deformations included quantifying differences between the frequency of gross and histopathological lesions in waterfowl populations in the Coeur d'Alene River basin and their frequency in reference areas, and between laboratory treatment groups exposed to Coeur d'Alene River basin sediment and reference area sediment. Gross lesions assessed included emaciation, abnormal bile, bile staining, and impactions of the upper gastrointestinal tract. Histopathological lesions assessed included hepatic and renal hemosiderosis, myocardial necrosis, arterial fibrinoid necrosis, and RIIBs. These gross and histopathological lesions are characteristic of lead exposure and are routinely assessed during necropsy and diagnostic examination of carcasses by trained pathologists to determine the cause of death.

6.4 INJURY DETERMINATION: TESTING AND SAMPLING APPROACHES

The injury assessment for wildlife resources in the Coeur d'Alene River basin began with an initial biological reconnaissance investigation. This investigation included a comprehensive review of existing data on the exposure and effects of metals in the Coeur d'Alene River basin and limited field sampling to determine food web exposure, to facilitate the design of pathway and injury studies, sampling methods, and quality control procedures, and to identify reference areas. The results of the biological reconnaissance investigation were used to design subsequent injury evaluation studies that focused on pathway determination and injury determination studies, as described below (Figure 6-9).

6.4.1 Pathway Studies

Pathway studies were conducted to determine the route and magnitude of exposure of biological resources to hazardous substances [43 CFR § 11.63] and to determine whether sufficient concentrations of hazardous substances were present in sediment, forage, and wildlife prey items to cause injury to biological resources [43 CFR § 11.63 (a)(2)]. Comparisons were performed to determine whether exposure of wildlife to hazardous substances in the Coeur d'Alene River basin differed from exposure of wildlife to hazardous substances at reference locations. The pathway studies characterized (1) routes of hazardous substance exposure from sediment, forage, and prey items to wildlife and (2) the degree of exposure of wildlife in the basin to pathway resources.



a. Component of pathway studies.

Figure 6-9. Flow diagram of wildlife injury assessment studies.

Pathway studies included the following (Table 6-4):

- ***Characterization of hazardous substance concentrations in sediments in areas used by wildlife.*** Surface sediments from multiple wetland and lake locations in the lower Coeur d'Alene River basin and reference areas known to be used by wildlife were collected and analyzed for hazardous substance concentrations. Details of the study are presented in:

  - Metal Contamination of Palustrine and Lacustrine Habitats in the Coeur d'Alene River basin, Idaho (Campbell et al., 1999a). Waterfowl use of the Coeur d'Alene River basin wetland habitats is documented in Audet et al. (1999c).

Table 6-4
Pathway and Injury Studies (field investigations, laboratory experiments) for Wildlife Resources Performed in the Coeur d'Alene River Basin and Reference Areas

Study Focus (study number[a])	Study Date	Measurement Objectives	Reference Area	Study Authors
Pathway Studies				
Biological reconnaissance (B1; B2)	1992-1993	Literature review; metal exposure, contamination	SJ[b]	Audet (1997)
Wood ducks (B1)	1992	Sediment ingestion by wood ducks	SJ	Beyer et al. (1997)
Water potato contamination (B1)	1994	Metal contamination in tubers[d]	SJ	Campbell et al. (1999b)
Tundra swans, Canada geese, mallard ducks (B1)	1994-1996	Sediment ingestion by waterfowl	SJ; MWMA[c]	Beyer et al. (1998b)
Sediment contamination (B1)	1995	Metal contamination in wetland/lateral lake sediments	SJ	Campbell et al. (1999a)
Injury Studies: Field Investigations				
Waterfowl; other species (B3)	1992-1997	Lead exposure; mortality/morbidity; gross/histopathological lesions; habitat use	SJ	Audet et al. (1999c)
Wood ducks (B3)	1992, 1995	Lead exposure; blood parameters	SJ	Blus et al. (1997)
Canada geese; mallards (B3)	1994-1995	Metal exposure; blood parameters; pathology	SJ; MWMA; Snake River; TNWR[e]	Henny et al. (1999)
Tundra swans (B3)	1994-1995	Metal exposure; blood parameters; pathology	MNWR[f]; KNWR[g]	Blus et al. (1999)
Bald eagles (B3)	1994	Lead exposure; metal contamination in prey; blood parameters; chick growth	MWMA	Audet et al. (1999b)
Song sparrows; American robins (B3)	1995	Lead exposure; blood parameters	SJ; Little North Fork River CdA	Johnson et al. (1999)

Table 6-4 (cont.)
Pathway and Injury Studies (field investigations, laboratory experiments) for Wildlife Resources Performed in the Coeur d'Alene River Basin and Reference Areas

Study Focus (study number[a])	Study Date	Measurement Objectives	Reference Area	Study Authors
Injury Studies: Laboratory Experiments				
Mallards (B3)	1994, 1995[h]	Toxicity of ingested sediment	SJ	Heinz et al. (1999)
Canada geese; mallards (B3)	1995[h]	Toxicity of ingested sediment	SJ	Hoffman et al. (1998)
Mute swans (B3)	1995[h]	Toxicity of ingested sediment	SJ	Day et al. (1998)

a. Refers to study identification number provided in NRDA Assessment Plan (Natural Resources Trustees, 1993).
b. SJ: St. Joe River basin, including the St. Maries River and adjacent wetlands and lakes.
c. MWMA: McArthur Wildlife Management Area (northern ID).
d. *Sagittaria* spp., a major food source of waterfowl and a traditional subsistence item for Coeur d'Alene tribal members.
e. TNWR: Turnbull National Wildlife Refuge (eastern WA).
f. MNWR: Malheur National Wildlife Refuge (central OR).
g. KNWR: Lower Klamath National Wildlife Refuge (southern OR).
h. Dates sediment samples were collected for use in laboratory feeding studies.

- ***Quantification of sediment ingestion by waterfowl.*** Sediment ingestion by waterfowl was quantified by collecting and analyzing fecal samples and the contents of the digestive system from representative species, including wood ducks, tundra swans, Canada geese, and mallard ducks from the lower Coeur d'Alene River basin and reference areas. Details are presented in:

  - The Role of Sediment Ingestion in Exposing Wood Ducks to Lead (Beyer et al., 1997)

  - Lead Exposure of Waterfowl Ingesting Coeur d'Alene River Basin Sediments (Beyer et al., 1998b).

- ***Metal contamination in the forage, prey items, and tissues of wildlife.*** Forage and prey items of waterfowl (e.g., vegetation, invertebrates) and birds of prey (e.g., fish, small mammals, waterfowl), and tissues and fecal samples from wildlife, were collected in both the Coeur d'Alene River basin and reference areas and analyzed for metal concentrations. Details are presented in:

  - Coeur d'Alene Basin Natural Resource Damage Assessment Biological Reconnaissance Investigation (Audet, 1997).

- ***Metal contamination of vegetation.*** Metal concentrations were measured in tubers of *Sagittaria latifolia* and *S. cuneata* (water potatoes) collected from the lower Coeur d'Alene River basin and the St. Joe River basin reference area. Water potatoes are an important food item of waterfowl and a traditional food of the Coeur d'Alene tribe. Metal concentrations in whole tubers (including skin and adhering sediment) were compared to metal concentrations in tubers with skin and adhering sediment removed. Details are presented in:

  - Heavy Metal Concentrations in *Sagittaria* spp. Tubers (Water Potato) in the Coeur d'Alene Basin (Campbell et al., 1999b).

Results of the pathway studies are discussed in Section 6.5. Full reports are provided in Volume II of this report: Studies Conducted as Part of the Injury Assessment.

6.4.2 Injury Studies

Supplemental injury studies were conducted to assess the biological responses of lead-exposed birds of the Coeur d'Alene River basin and to evaluate whether a relationship exists between the degree of sediment exposure and the frequency and degree of biological responses in waterfowl. Injury studies included field investigations of hazardous substances exposure and effects in

waterfowl, bald eagles, and songbirds, as well as controlled laboratory toxicity tests designed to evaluate the toxicity of ingested sediment to waterfowl.

Results of individual injury studies are summarized in Section 6.5. Full reports are provided on discs 2 and 3 of this report.

Injury studies included the following (Table 6-4):

- ***Waterfowl habitat use and diagnostic evaluation of the causes of waterfowl deaths.*** Waterfowl surveys were conducted to determine areas of use and major types of activities (feeding and comfort). Carcass searches were conducted, and dead and dying waterfowl were collected and submitted for necropsy and diagnostic evaluation. Necropsy and diagnostic evaluation included gross and histopathological examination of lesions, inspection for disease and lead artefacts, analysis of lead concentrations in tissues and ingesta, and determination of causes of deaths. In addition, food items in ingesta were identified. The number, frequency, and causes of waterfowl mortality and morbidity were determined. Details are presented in:

    - Wildlife Use and Mortality Investigation in the Coeur d'Alene Basin 1992-1997 (Audet et al., 1999c).

- ***Lead exposure and effects in waterfowl.*** Concentrations of metals in blood and other tissues, hematological responses (changes in blood ALAD activity and in levels of blood protoporphyrin, hemoglobin, and hematocrit), and physical deformations (gross and histopathological lesions) in representative waterfowl species from the Coeur d'Alene River basin and reference areas were measured and compared. Results were also compared to available historical information on lead exposure and effects in the Coeur d'Alene River basin. Details are presented in:

    - Persistence of High Blood Lead Concentrations and Associated Effects in Wood Ducks Captured near a Mining and Smelting Complex in Northern Idaho (Blus et al., 1999)

    - Field Evaluation of Lead Effects on Canada Geese and Mallards in the Coeur d'Alene River Basin, Idaho (Henny et al., 1999)

    - Persistence of High Blood Lead Concentrations and Associated Effects in Tundra Swans Captured near a Mining and Smelting Complex in Northern Idaho (Blus et al., 1999).

- ***Lead exposure and effects in bald eagles.*** Lead residues in blood and prey items, hematological responses (blood ALAD activity, hemoglobin, hematocrit), and growth were measured in young bald eagles from the Coeur d'Alene River basin and reference areas. Dead bald eagles from northern Idaho and eastern Washington were necropsied to determine causes of death. Details are presented in:

    - Lead Exposure of Bald Eagles and Prey Items in Northern Idaho and Eastern Washington (Audet et al., 1999b).

- ***Lead exposure and effects in songbirds.*** Lead residues in liver and hematological responses (changes in blood ALAD activity and hematocrit levels) were measured in song sparrows and American robins from the lower Coeur d'Alene River basin and reference areas. Details are presented in:

    - Lead Exposure in Passerines Inhabiting Lead-Contaminated Floodplains in the Coeur d'Alene River basin, Idaho (Johnson et al., 1999).

- ***Toxicity of lead-contaminated sediment to waterfowl.*** Controlled laboratory tests were conducted to assess the toxicity of ingested sediment from the Coeur d'Alene River basin to representative waterfowl species (mallards, Canada geese, mute swans), relative to the toxicity of ingested sediment from the St. Joe River basin. Biological responses evaluated included death, physiological malfunctions (e.g., changes in blood parameters, body weight), and physical deformations (gross and histological lesions). Relationships between the degree of sediment ingestion and biological responses of waterfowl were quantified. Details are presented in:

    - Toxicity of Lead-Contaminated Sediment to Mallards (Heinz et al., 1999)

    - Toxicity of Lead-Contaminated Sediment to Canada Goose Goslings and Mallard Ducklings (Hoffman et al., 1998)

    - Toxicity of Lead-Contaminated Sediment to Mute Swans (Day et al., 1998).

The biological responses selected for measurement in the injury studies are known to be responsive to lead exposure, have been used previously in scientific studies, are practical to perform, and produce scientifically valid results [43 CFR § 11.62(f)(2)(iv)]. The studies were conducted according to the quality assurance guidelines specified in the NRDA Quality Assurance Plan (USFWS, 1995).

6.4.3 Reference Areas

For each injury and pathway study, one or more reference areas appropriate for comparison to the study endpoints were selected (Table 6-4). For each of the studies, data from the reference areas were collected using methods comparable to the methods used in the Coeur d'Alene River basin [43 CFR § 11.72 (d)(5)]. Reference areas are described below and shown in Figure 6-10.

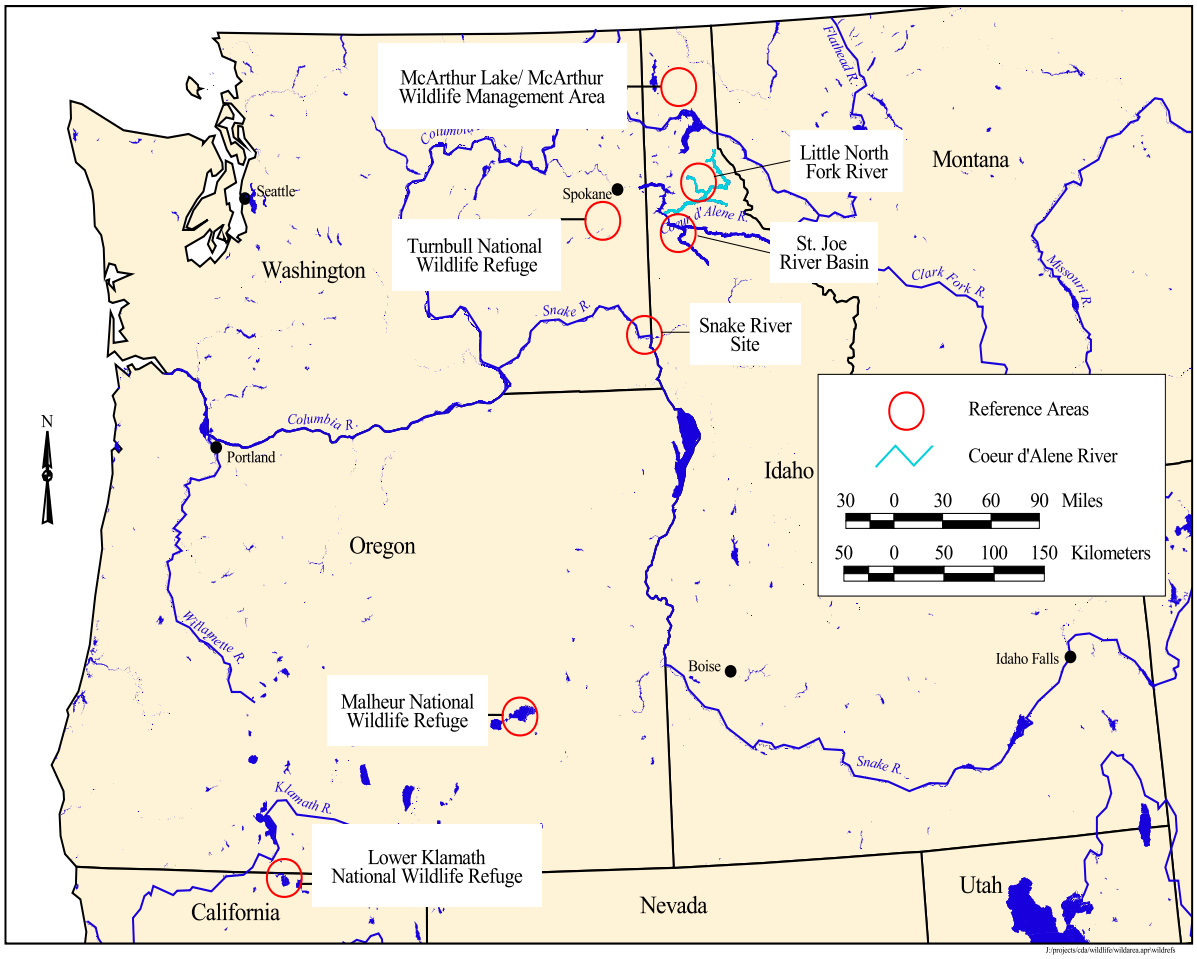


Figure 6-10. Locations of reference areas used in the Trustees' injury assessment studies.

St. Joe River basin. The St. Joe River basin is the drainage basin to the south of the Coeur d'Alene River basin. The St. Joe River basin includes the St. Joe River, the St. Maries River, and adjacent wetlands and lakes, including the southern Coeur d'Alene Lake (Figure 6-2). The basin was used as a reference area for the majority of wildlife pathway and injury studies for the following reasons:

- Absence of known releases of hazardous substance related to mining activities, including lead.

- Similarity of climate and seasonal environmental variability, and major vegetation types.

- General morphological and geographical similarity to the Coeur d'Alene River basin. The St. Joe River flows from the Montana/Idaho border, through the St. Joe Mountains, and discharges to Coeur d'Alene Lake at the southern end of the lake. The St. Joe River basin and Coeur d'Alene River basin have generally similar headwater geology, ranges in elevations and stream gradients, and stream flow rates.

- Similar wildlife species assemblages. The St. Joe River basin is on the same migration corridor (part of the Pacific flyway) as the Coeur d'Alene River basin, and thus provides similar migration routes and timing. For example, northward migrating waterfowl typically first stop in the St. Joe River basin before arriving in the Coeur d'Alene River basin.

- Similar types of wildlife habitats. The St. Joe River basin contains lacustrine, palustrine, and riparian habitats. Habitat abundance and diversity are lower in the St. Joe River basin than in the Coeur d'Alene River basin. Data collected as part of the injury assessment confirmed that habitat use by waterfowl (average number of waterfowl feeding per survey) is similar in the St. Joe River and Coeur d'Alene River basins, although the average feeding use per acre is slightly higher in the St. Joe River basin (Audet et al., 1999c).

- Similar wildlife management activities, including hunting activity. The exposure of wildlife to lead shot and other lead artifacts in the St. Joe River basin was expected to be similar to the exposure in the Coeur d'Alene River basin because management activities and hunting access are generally similar.

Data collected from the St. Joe River basin included (1) wildlife kill investigation data, including carcass counts for comparison of the frequency of wildlife kills and collection of carcasses for evaluation of the causes of mortality; (2) necropsy data to identify the frequency and severity of physical deformations and causes of death in the St. Joe River basin; (3) blood samples for comparison of endpoints related to physiological malfunctions such as inhibition of ALAD activity, and measurements of levels of protoporphyrin, hemoglobin, and hematocrit; (4) fecal samples for evaluation of the degree of exposure of reference organisms to dietary sediment; (5) hazardous substance concentrations in wildlife blood and tissues; and (6) hazardous substance concentrations in pathway items, including water potato and sediment.

Several other reference areas were selected and sampled because of one or more of the following: (1) similarity of waterfowl species that occur in the Coeur d'Alene River basin, (2) proximity to the Coeur d'Alene River basin, (3) location within the same migratory pathway (Pacific flyway), (4) expected low concentrations of hazardous substances because of absence of known mining related activities, and (5) expected similar exposure of hazardous substances from other sources (e.g., automobile emissions, lead artifacts).

McArthur Wildlife Management Area (MWMA). The MWMA, an approximately 310 ha area that includes McArthur Lake, is located approximately 115 km north of the Coeur d'Alene River basin in northern Idaho and includes habitat similar to habitat of the Coeur d'Alene River basin (Beyer et al., 1998b). The MWMA was a reference area for sediment ingestion studies (Beyer et al., 1998b) and exposure and effects studies (Henny et al., 1999). Data collected included (1) fecal samples for the determination of hazardous substances exposure to waterfowl; (2) blood samples for comparison of endpoints related to physiological malfunctions (inhibition of ALAD activity, and levels of protoporphyrin, hemoglobin, and hematocrit); (3) blood, liver, and kidney samples for comparison of hazardous substances exposure; and (4) physical deformation data to identify the frequency and severity of lesions in reference areas.

McArthur Lake was the reference area for the bald eagle study. Audet et al. (1999b) evaluated 20 potential nest locations in Kootenai, Bonner, and Boundary counties, Idaho, and ultimately selected McArthur as the reference area. Selection criteria included (1) presence of eaglets 45-55 days old, (2) nest accessibility, and (3) ability to collect data without harming either field personnel or eagles. Data collected included (1) blood samples for comparison of endpoints related to physiological malfunctions (inhibition of ALAD activity, levels of hemoglobin and hematocrit, growth); (2) blood samples for comparison of hazardous substances exposure; and (3) body weight data.

Malheur National Wildlife Refuge (MNWR) and Lower Klamath National Wildlife Refuge (KNWR). The MNWR (central Oregon) and KNWR (southern Oregon) were used as reference for studies of lead exposure and effects in tundra swans (Blus et al., 1999). Rationale for selecting these reference areas included (1) presence of the same species of birds; (2) similarity of hunting access and wildlife management activities, and thus similar exposure to lead shot; (3) same general region of Pacific flyway, and thus similar arrival and departure times at breeding grounds and wintering areas, and similar migration routes; (4) absence of known releases of hazardous substances related to mining activities, including lead; and (5) locations south of the Coeur d'Alene River basin, so birds had not recently been in the Coeur d'Alene River basin. Data collected included blood samples for comparison of endpoints related to physiological malfunctions (inhibition of ALAD activity, levels of hemoglobin and hematocrit); and blood and liver samples for comparison of hazardous substances exposure.

Turnbull National Wildlife Refuge (TNWR) and Snake River site. The TNWR, near the Coeur d'Alene River basin in eastern Washington state, and the Snake River site, south of the Coeur d'Alene River basin near Lewiston, Idaho, were used as reference areas for the field evaluation of lead effects on Canada geese and mallards (Henny et al., 1999). Henny et al. collected data for mallards from the TNWR, St. Joe River basin, and MWMA reference areas and for Canada geese from the Snake River reference area. Rationale for selecting these reference areas included (1) presence of the same species of birds; (2) similar hunting access, so similar exposure to lead shot; (3) same general region of Pacific flyway, thus similar arrival and departure times at breeding grounds and wintering areas and migration routes; and (4) absence of known releases of hazardous substance related to mining activities, including lead. Data collected included

(1) blood samples for comparison of endpoints related to physiological malfunctions (inhibition of ALAD activity, and levels of protoporphyrin, hemoglobin, and hematocrit); (2) blood, liver, and kidney samples for comparison of hazardous substances exposure; and (3) physical deformation data to identify the frequency and severity of lesions in reference areas.

Little North Fork Coeur d'Alene River. The Little North Fork Coeur d'Alene River is a tributary to the North Fork of the Coeur d'Alene River and is not exposed to mining related contamination. The Little North Fork Coeur d'Alene River and the St. Joe River basin were used as reference areas for the field investigation of exposure and effects on songbirds (Johnson et al., 1999). Selection criteria included (1) similar wildlife management activities (lands owned and managed by the Idaho Department of Fish and Game); (2) location on public or private land accessible by vehicle; (3) proximity to the Coeur d'Alene River basin; (4) similar habitat for the target species (e.g., riparian areas or near wetlands or lakes; and (5) presence of the target species. Data collected from the Little North Fork Coeur d'Alene River included (1) blood samples for comparison of endpoints related to physiological malfunctions (inhibition of ALAD activity, and hematocrit levels); and (2) liver samples for comparison of hazardous substances exposure.

6.5 INJURY ASSESSMENT STUDIES: RESULTS

This section presents the results of the pathway studies in Section 6.5.1. The injury study results are presented according to field investigations (Section 6.5.2), and laboratory toxicity tests (Section 6.5.3), with conclusions in Section 6.5.4.

6.5.1 Pathway Studies

Metal Concentrations in Sediments in Wildlife Use Areas

Concentrations of hazardous substances in sediments from Coeur d'Alene River basin areas used by wildlife were compared to concentrations in sediments from St. Joe River basin areas used by wildlife (Campbell et al., 1999a). Mean lead concentrations in Coeur d'Alene River basin sediments (1,075 to 5,826 ppm) were significantly ($p < 0.001$) greater than lead concentrations in reference area sediments (all reference area averages less than 20 ppm; Campbell et al., 1999a) (Figure 6-11). Zinc, cadmium, and arsenic concentrations were also significantly greater in Coeur d'Alene River basin sediments ($p < 0.001$); concentrations ranged from 2 to 100 times greater in Coeur d'Alene River basin sediments.

The results confirm that sediment in habitats used by wildlife of the Coeur d'Alene River basin is contaminated with lead and other metals and that concentrations of hazardous substances are substantially elevated relative to reference areas (Campbell et al., 1999a). These results are consistent with historical data showing that concentrations of lead are elevated in Coeur d'Alene River basin sediments.

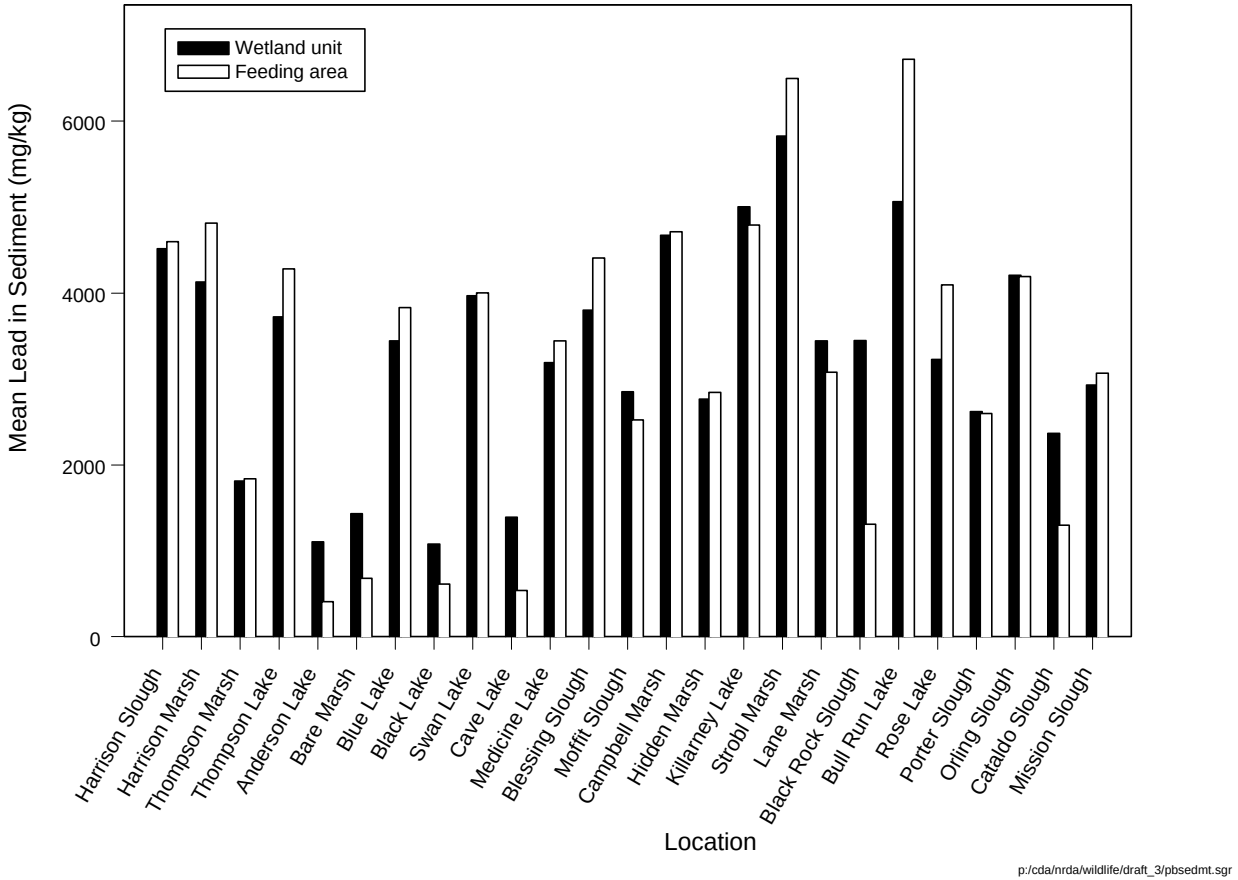


Figure 6-11. Lead concentrations in sediments of Coeur d'Alene lacustrine and palustrine habitats. Mean lead concentrations in sediment from reference area wetland units and feeding areas were 17.07 and 16.04 mg/kg, respectively.
Sources: Audet et al., 1999c; Campbell et al., 1999a.

The concentrations of lead and other hazardous substances in Coeur d'Alene River basin sediments are sufficient to provide a direct pathway to the wildlife resources of the Coeur d'Alene [43 CFR § 11.63(e)] through direct sediment ingestion [see Beyer (1997) and Beyer et al. (1998b)].

Sediment Ingestion by Waterfowl

Sediment ingestion by waterfowl was quantified to determine the degree of exposure of wildlife to sediment and to lead in sediment. Digesta (dietary contents of the digestive system) and excreta (excretory products, including feces) from representative species, including wood ducks (Beyer et al., 1997), tundra swans, Canada geese, and mallard ducks (Beyer et al., 1998b) from the Coeur d'Alene River basin and reference areas, were collected and analyzed.

Wood ducks. Since wood ducks feed on the water surface and, in the Coeur d'Alene River basin, ingest less than 2% sediment in their diet (Beyer et al., 1997), they were studied as a species representative of waterfowl expected to be less exposed to metal contaminated sediments than species that feed on wetland or lakebed surfaces. Results of digesta analyses confirmed that Coeur d'Alene River basin wood duck digesta contains elevated concentrations of lead (mean of 32 ppm) relative to digesta of wood ducks from reference areas (8 ppm). Lead concentrations in wood duck digesta were correlated with lead concentrations in the sediment in areas where the ducks feed, and most of the lead in digesta came from ingested sediment rather than from plant material (Beyer et al., 1997). The results of the wood duck study demonstrate that the contaminated sediments of the Coeur d'Alene River basin can serve as an important pathway of hazardous substances exposure even in waterfowl that have low rates of sediment ingestion.

Tundra swans, Canada geese, mallards. Analysis of the excreta of tundra swans, Canada geese, and mallard ducks from the Coeur d'Alene River basin confirmed that these species ingest large amounts of sediment and that sediment is the primary source of the lead ingested by these species (Beyer et al., 1998b). Sediment ingestion rates were determined by the relationship between the acid insoluble ash content of feces (i.e., the "mineral" component), food digestibility, and the sediment content of diets (Beyer et al., 1994). Estimated average sediment ingestion rates for both Canada geese and tundra swans were 9%, and for mallards, approximately 5% (Beyer et al., 1998b). The ninetieth percentile for sediment ingestion of tundra swans was estimated to be 22% sediment in the diet (i.e., an estimated 90% of tundra swans ingest 22% or less sediment, and 10% of tundra swans ingest more than 22%).

The average lead concentration (dry weight) in the excreta of tundra swans was 880 ppm in the Coeur d'Alene River basin and 2 ppm in reference areas (Figure 6-12). In the Coeur d'Alene River basin, lead concentrations in excreta up to 3,900 ppm (Canada goose) and 3,300 ppm (tundra swan) were found, whereas in the reference areas maximum values measured in excreta

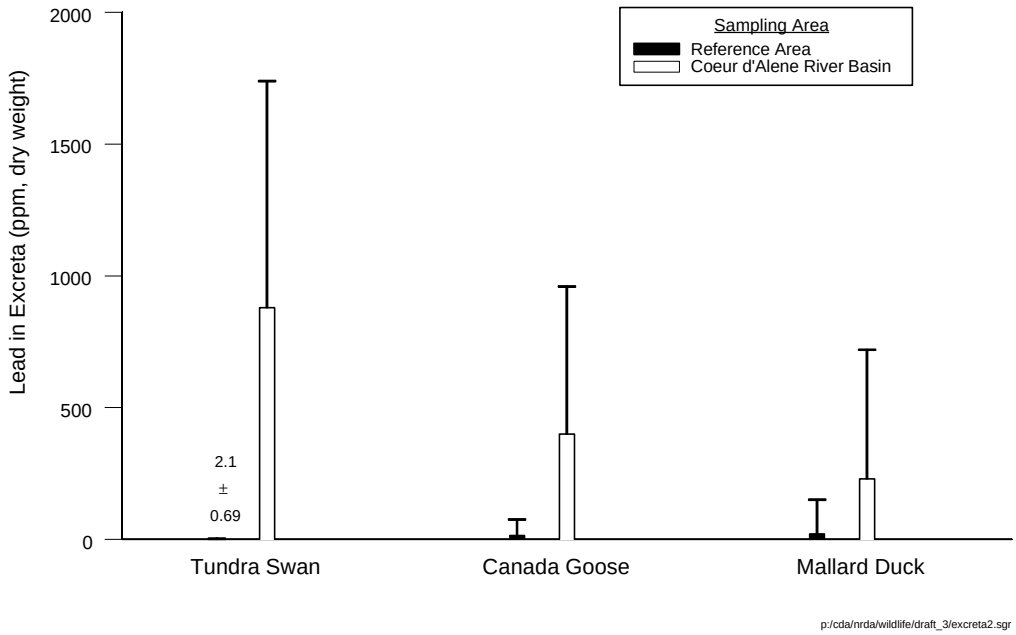


Figure 6-12. Mean (plus standard deviation) lead concentrations in the excreta of tundra swans, Canada geese, and mallard ducks from the Coeur d'Alene River basin and reference areas.
Source: Beyer et al., 1998b.

were 930 ppm (Canada goose) and 3.1 ppm (tundra swan). The average concentration of lead in mallard duck excreta was 230 ppm in the Coeur d'Alene River basin compared to 21 ppm in reference areas. The degree of elevation in lead concentrations in Canada goose, tundra swan, and mallard duck excreta demonstrates substantially elevated dietary exposure of waterfowl in the Coeur d'Alene River basin (Figure 6-12).

Lead concentrations in tundra swan feces were significantly correlated ($p < 0.05$; Spearman's rho = 0.74) with the amount of sediment ingested (Beyer et al., 1994), demonstrating that ingestion of the contaminated sediment was the source of the lead in the waterfowl (Beyer et al., 1998b). Fecal lead concentrations of all waterfowl were also significantly correlated ($p < 0.05$; $r^2 = 0.83$) with lead concentrations in sediment in the Coeur d'Alene River basin (Beyer et al., 1998b). Feces with very low lead concentrations had correspondingly low acid-insoluble ash content, which demonstrates that the primary source of the lead in the waterfowl was lead in sediment rather than lead in ingested plant material (Beyer et al., 1998b).

The results confirm that direct ingestion of contaminated Coeur d'Alene sediment is the principal exposure pathway of waterfowl to lead and other hazardous substances in the Coeur d'Alene River basin (Beyer et al., 1998b).

Metal Contamination of Vegetation

Aquatic vegetation from waterfowl use areas in the Coeur d'Alene and St. Joe river basins was collected to determine if Coeur d'Alene River basin vegetation serves as a pathway of waterfowl exposure to hazardous substances (Audet, 1997; Campbell et al., 1999b). Important components of waterfowl diets, including tubers of *Sagittaria* spp. and horsetail (*Equisetum fluviatile*), were analyzed for lead and other metals. The large, starchy tubers of *Sagittaria* spp. (water potatoes) are found throughout the Coeur d'Alene and the St. Joe river basins and are a food source for waterfowl and a traditional food of the Coeur d'Alene Tribe. Metal concentrations in whole tubers (with skin and adhering sediment) were compared to tubers with the skin and adhering sediment removed. Concentrations in Coeur d'Alene River basin tubers were compared to concentrations in St. Joe River basin tubers.

Mean lead concentrations in *Equisetum* and other aquatic vegetation that are consumed by waterfowl in the Coeur d'Alene River basin ranged from 13.78 ppm in arrowhead (*Sagittaria* spp.) to 60.29 ppm in coontail *(Ceratophyllum demersum)* (Figure 6-13) (Audet, 1997; Campbell et al., 1999b). The mean lead concentration in Coeur d'Alene River basin whole tubers (with skin and adhering sediment; 30 ppm) was significantly greater than both the mean lead concentration in whole tubers from the St. Joe River basin (0.3 ppm; $p < 0.05$) and the mean lead concentration in Coeur d'Alene River basin tubers with the skin and adhering sediment removed (0.4 ppm lead; $p = 0.185$). The results indicate that lead contamination of Coeur d'Alene River basin tubers is associated with the outside surface of the tuber, and that hazardous substances contaminate forage of Coeur d'Alene River basin wildlife.

The elevated concentrations of lead in *Equisetum*, water potatoes, and other vegetation from the Coeur d'Alene River basin are sufficient to provide a direct pathway to waterfowl.

Metal Contamination in the Wildlife Food Web

Reconnaissance sampling was conducted to assess the extent of exposure of Coeur d'Alene River basin wildlife to hazardous substances by the food chain (Audet, 1997). The reconnaissance sampling and analysis revealed exposure of dietary items of birds of prey, including small mammals (deer mouse and meadow vole), aquatic species (brown bullhead, yellow perch, and tench), and avian species (tundra swan and Canada goose); dietary items of fish (aquatic invertebrates) (Figure 6-14); and dietary items of dabbling and diving ducks (aquatic vegetation species, to lead and other heavy metals. Samples of some species were collected from the St. Joe River basin for comparison to the Coeur d'Alene River basin samples. Metals concentrations for all sample matrices collected from the St. Joe River basin were consistently low (Figure 6-14).

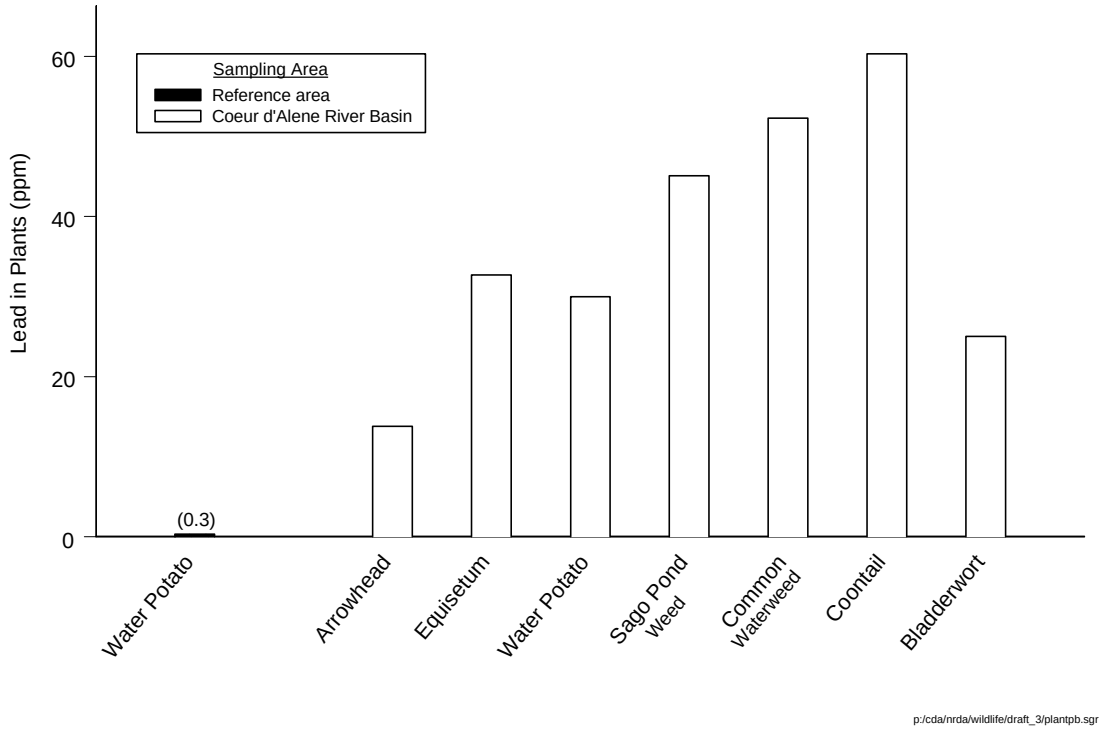


Figure 6-13. Mean lead contaminations in aquatic vegetation (ppm, wet weight; with skin and adhering sediment) consumed by waterfowl from the Coeur d'Alene and reference areas.
Sources: Audet, 1997; Campbell et al., 1999b.

Comparison of the reconnaissance sampling results to previous sampling results in the Coeur d'Alene River basin revealed little change in lead concentrations in various matrices with time. No evidence of attenuation of exposure from 1982 to 1992 for mammals (Figure 6-15), fish, or waterfowl was detected. Livers of deer mice and mink also exceeded the 7.5 ppm threshold for lead poisonings in mammals proposed by Ma (1996).

The results of this study show that multiple components of the Coeur d'Alene River basin food web are contaminated with lead. Lead concentrations are elevated in important forage and prey items of Coeur d'Alene River basin wildlife, including aquatic vegetation, fish, small mammals, and waterfowl. Contamination of the biological resources of the Coeur d'Alene River basin by hazardous substances is pervasive and sufficient to provide a food chain pathway to Coeur d'Alene wildlife resources.

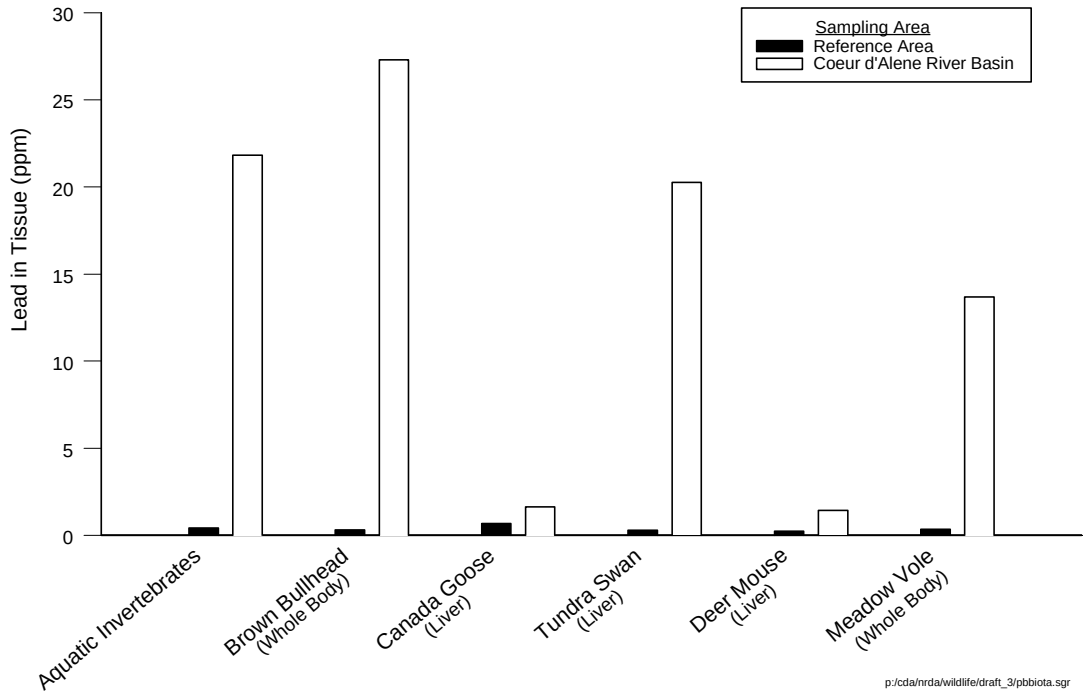


Figure 6-14. Mean lead concentrations in food web resources of the Coeur d'Alene River basin and the St. Joe River basin reference areas.
Source: Audet, 1997.

Pathway Study Conclusions

The results of the pathway studies demonstrate that Coeur d'Alene River basin sediments are contaminated with lead and other hazardous substances and that concentrations of these substances in sediments are substantially elevated relative to concentrations in St. Joe River basin sediments. Waterfowl in the Coeur d'Alene River basin are directly exposed to lead and other hazardous substances by ingestion of contaminated sediments during foraging activities.

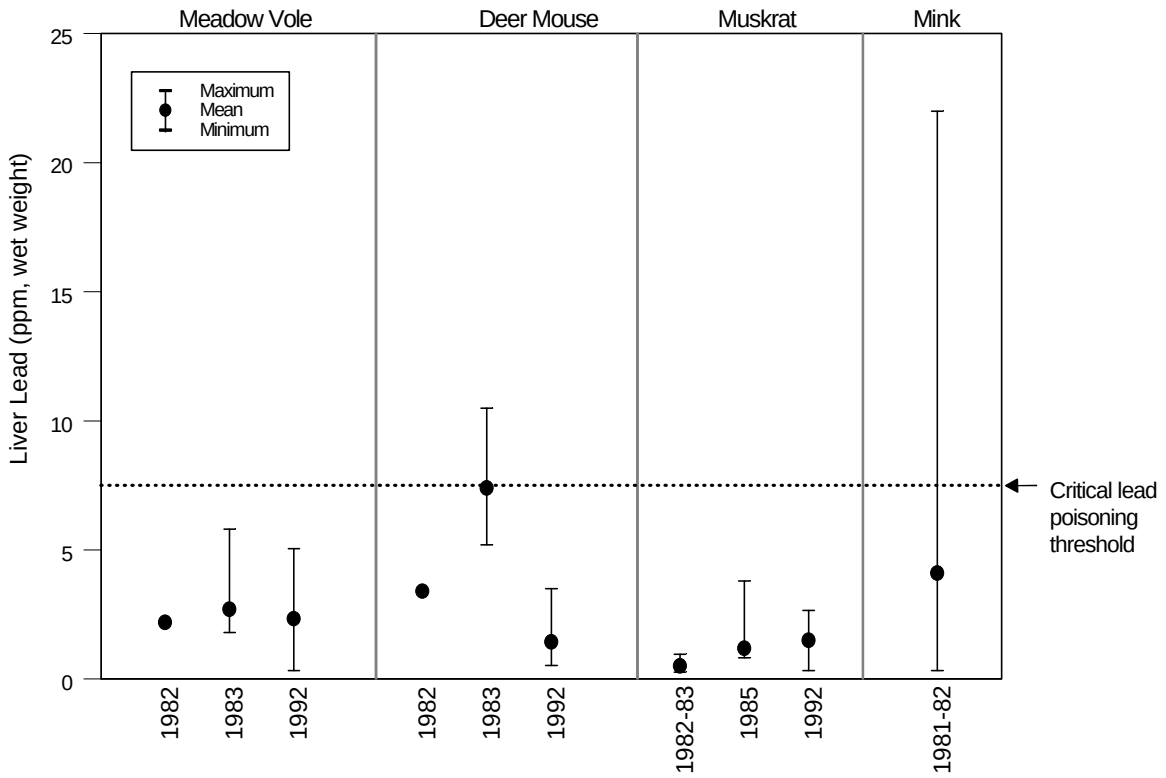


Figure 6-15. Mean lead concentrations in the tissues of Coeur d'Alene River basin wildlife collected in 1982, 1983, 1985, and 1992. Critical lead poisoning threshold of 7.5 ppm for mammals based on Ma (1996). Sources: Blus et al., 1987; Krieger, 1990; Ma, 1996; Audet, 1997.

Waterfowl, songbird, invertebrate, fish, small mammal, and aquatic vegetation tissues contain elevated concentrations of lead and provide a food chain exposure pathway to wildlife predators. Contamination of the sediment and biological resources of the Coeur d'Alene River basin by hazardous substances is pervasive and sufficient to provide both direct and indirect pathways to Coeur d'Alene River basin wildlife resources.

6.5.2 Injury Field Studies

Field investigations included determination of waterfowl habitat use and diagnosis of the causes of waterfowl deaths; evaluation of the persistence of elevated lead exposure and effects in waterfowl; and evaluation of lead exposure and effects in bald eagles and songbirds. Laboratory studies involved exposing representative species of waterfowl to sediment from either the Coeur d'Alene River basin or reference areas. The laboratory experiments included determination of the bioavailability and toxicity of contaminated sediment to mallards, Canada geese, and mute swans (a surrogate test species for tundra swans).

The biological responses investigated included death, physiological malfunctions, and physical deformations. As noted previously, the specific responses measured, which meet the acceptance criteria of the DOI regulations, are characteristic of lead exposure and effects.

Waterfowl Habitat Use and Causes of Waterfowl Deaths

Surveys were performed in the lower Coeur d'Alene area and the St. Joe River basin reference area to quantitatively evaluate waterfowl habitat use (1995 to 1997) and to determine the causes of waterfowl deaths (1992 to 1997). Areas used by wildlife were characterized by the type of use (i.e., feeding, resting) (Audet et al., 1999c), and searches for dead and dying wildlife were conducted. Recovered carcasses were submitted for necropsy examination to determine the cause of death. Diagnostic veterinary procedures included evaluation of gross and histopathological lesions, inspection for disease and lead artifacts (such as lead shot), and analysis of lead residues in tissues and ingesta.

Waterfowl surveys indicated that migratory birds, specifically tundra swans and mallard ducks, stop in the St. Joe River basin before stopping in the Coeur d'Alene River basin during their northward migration in the spring (Figure 6-1) (Audet et al., 1999c). From 1995 through 1997, tundra swan peak counts were similar in the Coeur d'Alene River basin and in the St. Joe River basin. Canada goose peak counts were 2 to 6 times higher in the Coeur d'Alene River basin, and mallard abundance, basin preference, and seasonal use varied greatly year to year (Audet, 1999c). There was no significant difference ($p > 0.05$) between waterfowl feeding use or feeding use per acre in the lower Coeur d'Alene River basin and the St. Joe River basin. However, there was a significantly greater number and frequency of dead and dying birds found in the Coeur d'Alene River basin ($p < 0.0001$) (Audet et al., 1999c).

During the 1992 to 1997 surveys, 682 animals, including 29 species of birds and 6 species of mammals, were found dead or sick in the Coeur d'Alene River basin (Audet et al., 1999c). In contrast, only 40 animals (9 species of birds, 2 species of mammals) were found dead or sick in the St. Joe River basin during the same period (Table 6-5). Animals found dead or sick included waterfowl (e.g., tundra swans, Canada geese, mallards, wood ducks), songbirds (e.g., American robin, swallows), birds of prey (e.g., bald eagles, osprey, red-tailed hawk), amphibians and reptiles (e.g., frogs and turtles), meadow vole, muskrat, mink, and beaver (Audet et al., 1999c).

Table 6-5
Animals Found Dead or Sick in the Coeur d'Alene and St. Joe River Basins, 1992-1997
[total # found (# submitted for necropsy examination)]

Species	1992	1993	1994	1995	1996	1997	Total
Coeur d'Alene River Basin							
Tundra swan	11 (11)	43 (37)	12 (5)	25 (18)	28 (19)	170 (112)	289 (202)
Canada goose	1 (1)	3 (2)	14 (3)	22 (7)	45 (16)	93 (26)	178 (55)
Mallard	1 (1)	-	14 (4)	4 (0)	10 (4)	26 (4)	55 (13)
Unknown	-	-	6 (0)	8 (0)	4 (0)	6 (0)	24 (0)
Wood duck	1 (1)	-	2 (1)	4 (0)	7 (2)	4 (2)	18 (6)
American coot	-	-	3 (3)	1 (0)	3 (1)	10 (0)	17 (4)
Muskrat	-	-	5 (5)	1 (0)	7 (4)	3 (2)	16 (11)
Violet-green swallow	2 (2)	1 (0)	-	5 (2)	2 (2)	-	10 (6)
Northern pintail	-	-	1 (0)	1 (0)	6 (1)	2 (0)	10 (1)
Barn swallow	-	5 (0)	-	3 (2)	-	-	8 (2)
Great blue heron	-	-	2 (0)	-	4 (0)	1 (0)	7 (0)
Meadow vole	2 (2)	-	1 (1)	1 (1)	1 (1)	1 (1)	6 (6)
Green-winged teal	-	-	1 (1)	2 (0)	1 (0)	1 (0)	5 (1)
Western painted turtle	-	-	1 (0)	2 (0)	-	1 (0)	4 (0)
American wigeon	-	-	-	1 (1)	2 (2)	-	3 (3)
Common goldeneye	-	-	-	-	1 (0)	2 (1)	3 (1)
Gull spp.	-	-	1 (0)	-	1 (0)	-	2 (0)
Beaver	-	-	2 (0)	-	-	-	2 (0)
Common merganser	-	-	-	-	-	2 (1)	2 (1)
American robin	-	1 (1)	1 (0)	-	-	-	2 (1)
Osprey	-	-	-	-	2 (0)	-	2 (0)
Bull frog	-	-	-	1 (0)	1 (0)	-	2 (0)
Redhead	-	1 (1)	-	-	-	1 (0)	2 (1)
Dark-eyed junco	-	-	-	1 (1)	-	-	1 (1)
Coyote	-	1 (0)	-	-	-	-	1 (0)
Common snipe	-	-	1 (1)	-	-	-	1 (1)
Grebe spp.	-	-	-	1 (0)	-	-	1 (0)
Wild turkey	-	-	-	-	-	1 (1)	1 (1)
Northern flicker	-	-	1 (1)	-	-	-	1 (1)
Canvasback	-	-	1 (1)	-	-	-	1 (1)

Table 6-5 (cont.)
Animals Found Dead or Sick in the Coeur d'Alene and St. Joe River Basins, 1992-1997
[total # found (# submitted for necropsy examination)]

Species	1992	1993	1994	1995	1996	1997	Total
Coeur d'Alene River Basin (cont.)							
Red-tailed hawk	-	-	-	-	1 (0)	-	1 (0)
Shrew spp.	-	-	-	1 (1)	-	-	1 (1)
Sora rail	-	-	-	1 (0)	-	-	1 (0)
Trumpeter swan	-	-	1 (1)	-	-	-	1 (1)
Bald eagle	-	1 (1)	-	-	-	-	1 (1)
Mink	-	-	-	-	1 (1)	-	1 (1)
Swainson thrush	1 (1)	-	-	-	-	-	1 (1)
American crow	-	-	1 (1)	-	-	-	1 (1)
CdA River Basin Total	**19 (19)**	**56 (42)**	**71 (28)**	**85 (33)**	**127 (53)**	**324 (150)**	**682 (325)**
St. Joe River Basin							
Canada goose	-	1 (1)	2 (1)	5 (4)	5 (3)	1 (0)	14 (9)
Tundra swan	-	2 (2)	1 (0)	-	1 (1)	4 (1)	8 (4)
Muskrat	-	-	-	3 (1)	1 (1)	-	4 (2)
Unknown	-	-	2 (0)	1 (0)	-	-	3 (0)
Mallard	-	-	-	-	-	2 (0)	2 (0)
Unidentified ducklings	-	-	2 (2)	-		-	2 (2)
Bald eagle	-	-	-	1 (0)	-	-	1 (0)
Bufflehead	-	-	-	-	-	1 (0)	1 (0)
Beaver	-	-	-	-	-	1 (0)	1 (0)
Varied thrush	-	-	-	-	-	-	1 (1)
Gull spp.	-	-	1 (1)	-	1 (1)	-	1 (1)
American robin	-	-	1 (0)	-	-	-	1 (0)
Redhead	-	-	-	-	1 (1)	-	1 (1)
St. Joe River Basin Total	**-**	**3 (3)**	**9 (4)**	**10 (5)**	**9 (7)**	**9 (1)**	**40 (20)**

The actual number of dead and sick animals in the Coeur d'Alene basin is greater than the number of animals observed during the surveys. Only a fraction of animal carcasses can actually be detected in the wild because all areas are not surveyed, sick and moribund animals are reclusive and/or immobile, and carcasses may be scavenged by predators or hidden in vegetation. Detection rate studies in the Coeur d'Alene River basin showed that 0% to 57% of carcasses were found by field observers (Audet et al., 1999c).

During the 1992 to 1997 surveys, 325 animal carcasses from the Coeur d'Alene River basin and 20 from the St. Joe River basin were necropsied to determine the cause of sickness or death. Fourteen of the Coeur d'Alene River basin carcasses and one of the St. Joe River basin carcasses were badly decomposed or scavenged and were unsuitable for reliable diagnosis; statistics are based on 311 Coeur d'Alene River basin and 19 St. Joe River basin carcasses (Audet et al., 1999c).

Lead poisoning was the single greatest cause of sickness or death of wildlife from the Coeur d'Alene River basin (80%, Figure 6-16), and 92% of the lead poisoned animals had not ingested lead artifacts (e.g., lead shot or fishing sinkers) (Figures 6-16 and 6-17). Nine species of waterfowl were documented with lead poisoning without the presence of lead artefacts (Figure 6-7). In contrast, 47% of the 19 carcasses necropsied from the St. Joe River basin were diagnosed as lead poisoned, and 78% of them (7 of 9) had ingested lead artifacts. Ingested lead artifacts were present in only 8.4% of the lead-poisoned birds examined from the Coeur d'Alene basin compared to 87.5% of lead poisoned birds from the St. Joe River basin. The carcass survey data show that swan mortality is significantly greater in the Coeur d'Alene River basin than in the St. Joe River basin ($p < 0.0001$), and that mortality rates are approximately 7 times greater in the Coeur d'Alene River basin. These data indicate that wildlife mortality rates are elevated in the Coeur d'Alene River basin, that the principal cause of wildlife deaths is lead poisoning, and that lead poisoning does not result from the ingestion of lead artifacts.

Information summarized by Audet et al. (1999c) shows that the frequency of lead poisoning without the presence of lead artifacts as a cause of mortality to tundra swans relative to other causes of death is substantially greater in the Coeur d'Alene River basin than in either the United States as a whole or in the Pacific flyway (Figure 6-18). Lead poisoning accounts for 22% to 29% of tundra swan mortalities nationwide, as well as in the Pacific flyway (Figure 6-18). These percentages include lead poisoning caused by ingestion of lead artifacts. Mortality rates and causes of death in the St. Joe River basin are similar to other waterfowl areas that experience lead poisoning because of ingestion of lead artifacts. In contrast, 96% of the tundra swan mortality in the Coeur d'Alene River basin is caused by lead poisoning without the presence of ingestion lead artifacts (Figure 6-18).

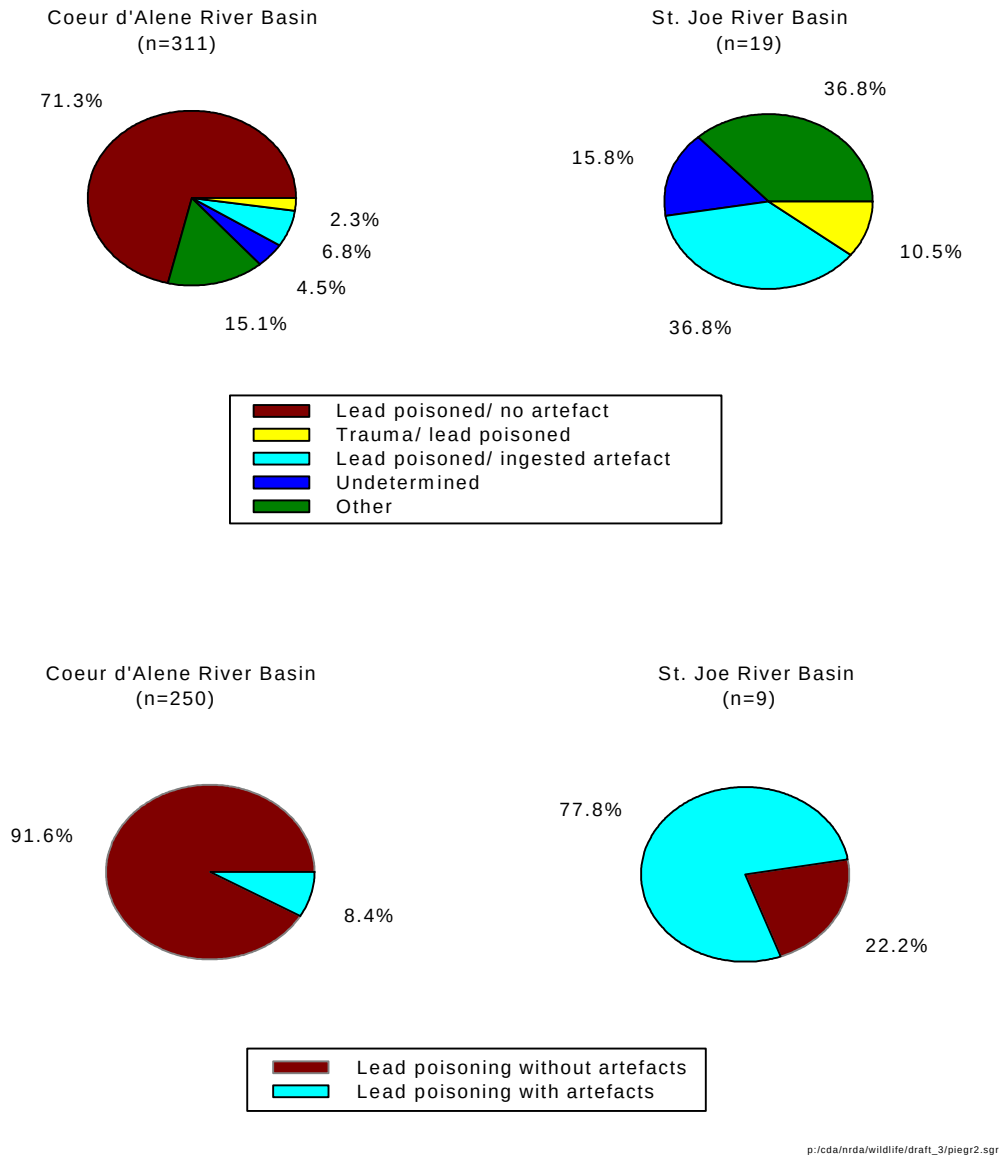


Figure 6-16. Pie charts showing causes of waterfowl deaths in the Coeur d'Alene River basin and St. Joe River basin. Top: percentage occurrence for all causes of mortality in birds submitted for necropsy; n refers to total number of dead birds found and submitted for diagnosis. Bottom: percentage occurrence of lead artifacts in birds diagnosed as lead poisoned.
Source: Audet et al., 1999b.

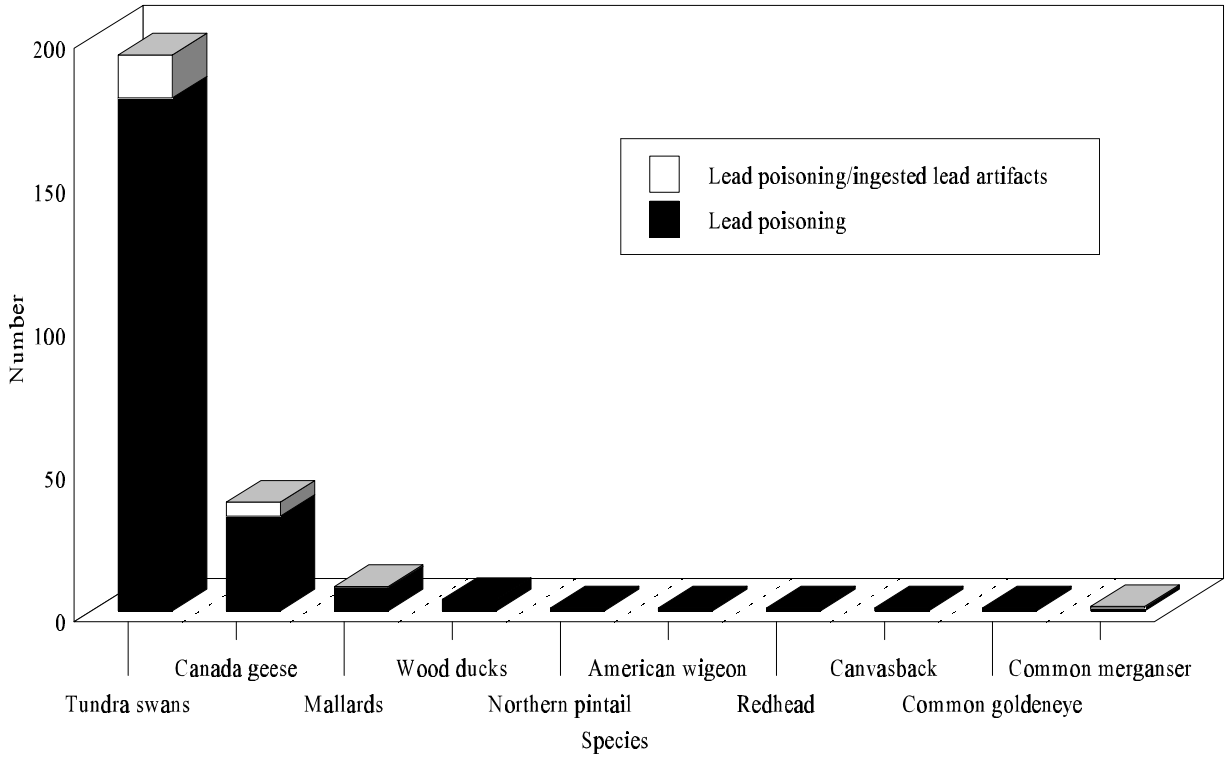


Figure 6-17. Species of waterfowl diagnosed as lead poisoned with and without lead artifacts during the 1992-1997 survey period. n = 194 for tundra swans, 38 for Canada geese, 8 for mallards, and 1 for all others. Source: Audet et al., 1999b.

The high concentrations of lead in Coeur d'Alene River basin sediments (Campbell et al., 1999a), the high rates of sediment ingestion by waterfowl (Beyer et al., 1998b), and the elevated mortality caused by lead poisoning without lead artifact ingestion (Audet et al., 1999c) all indicate that the primary source of the lead in Coeur d'Alene River basin waterfowl is ingested sediments containing lead (Audet et al., 1999c).

Altogether, from 1992 to 1997, sick and dying waterfowl diagnosed as lead poisoned without the presence of lead artifacts were found in 78% of the Coeur d'Alene River basin waterfowl habitat surveyed. Physical deformations (gross and histopathological lesions characteristic of lead poisoning) were observed in 97% of the lead-poisoned birds. Waterfowl carcasses found in the Coeur d'Alene River basin in 1997 represented the largest documented waterfowl kill in the basin since 1953 (Audet et al., 1999c). The mortality survey data indicate that waterfowl deaths occur throughout the lower Coeur d'Alene area and in areas including the South Fork Coeur d'Alene River floodplain, that poisoning occurs in multiple species, and that mortality rates have not declined from historical levels.

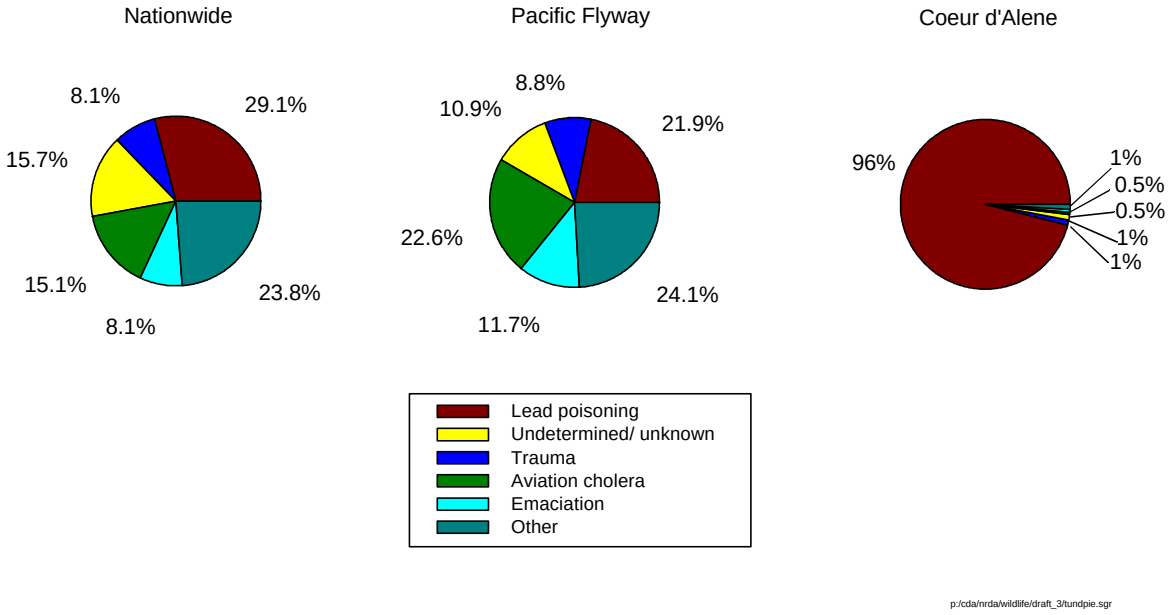


Figure 6-18. Comparison of causes of tundra swan death in the Coeur d'Alene (1992 to 1996), Pacific flyway (1980 to 1993), and nationwide (1981 to 1988).
Source: Audet et al., 1999c.

Lead Exposure and Effects in Waterfowl

Wood ducks, tundra swans, mallard ducks, and Canada geese were captured from the Coeur d'Alene River basin and reference areas to evaluate the degree and sublethal effects of lead exposure (Blus et al., 1997; Blus et al., 1999; Henny et al., 1999). These species were selected to represent migratory waterfowl species of the Coeur d'Alene River basin, and to represent a variety of foraging and feeding strategies used by waterfowl that inhabit the Coeur d'Alene River basin. Lead concentrations in blood and other tissues were measured, and hematological responses (e.g., ALAD activity, hematocrit, and hemoglobin) were quantified.

Wood ducks. Lead exposure (blood lead concentration) and hematological responses (changes in blood ALAD activity, hemoglobin, hematocrit) in wood ducks captured in 1986-1987, 1992, and 1995 from the Coeur d'Alene River basin were compared to lead exposure and hematological data from wood ducks captured in the same years from reference areas north of the Coeur d'Alene River basin (1986-1987) and the St. Joe River basin (1992 and 1995) (Blus et al., 1997).

Exposure of wood ducks collected in 1995 in the Coeur d'Alene River basin to lead was significantly greater (mean blood lead 2 ppm; range 0.63 to 4.5 ppm) than exposure of wood ducks in 1986-1987 ($p = 0.024$; mean blood lead 1.2 ppm; range below detection to 9.0 ppm). Blood lead concentrations in wood ducks collected from the Coeur d'Alene River basin in 1986-1987 and 1995 were significantly greater than blood lead concentrations in wood ducks from the St. Joe River basin ($p < 0.001$; all years combined). Eighty percent of wood ducks in the Coeur d'Alene River basin had a blood lead concentration greater than 0.25 ppm, compared to 6% in the reference area (Blus et al., 1997). Wood ducks from the Coeur d'Alene River basin exhibited physiological impairments in all years sampled, including significant reductions in ALAD activity ($p < 0.0001$), hemoglobin ($p < 0.0002$), and hematocrit ($p < 0.0001$) relative to reference area wood ducks (Figure 6-19). ALAD activity and hemoglobin reductions were significantly correlated with lead residues in blood ($p < 0.001$; ALAD $r^2 = 0.26$; hemoglobin $r^2 = 0.18$). ALAD activity was inhibited by 85 to 96% in Coeur d'Alene wood ducks. Blood lead concentrations and ALAD inhibition measured in wood ducks in 1995 and ALAD inhibition in 1992 were greater than concentrations and ALAD inhibition measured in wood ducks in 1986 and 1987 (Blus et al., 1993). These data indicate that there has been no decrease in lead exposure and effects in wood ducks from the Coeur d'Alene River basin for at least 10 years (1986-1995) (Figure 6-19) (Blus et al., 1997).

These results confirm that even bird species whose exposure to hazardous substances in the Coeur d'Alene River basin is limited by their feeding habits (e.g., water surface feeders) exhibit evidence of significantly greater lead exposure and physiological impairments than reference area birds (Blus et al., 1997).

Tundra swans. Lead exposure (lead concentrations in blood and liver) and hematological responses (changes in blood ALAD activity, hemoglobin, and hematocrit) in moribund and apparently healthy tundra swans captured in 1994-1995 from the Coeur d'Alene River basin were compared to lead exposure and hematological data from tundra swans captured in 1994-1995 in reference areas. Comparisons were also made to data from moribund and apparently healthy swans captured in 1987 from the Coeur d'Alene River basin (Blus et al., 1999). Reference areas included the Malheur National Wildlife Refuge (central Oregon) and the Lower Klamath National Wildlife Refuge (southern Oregon).

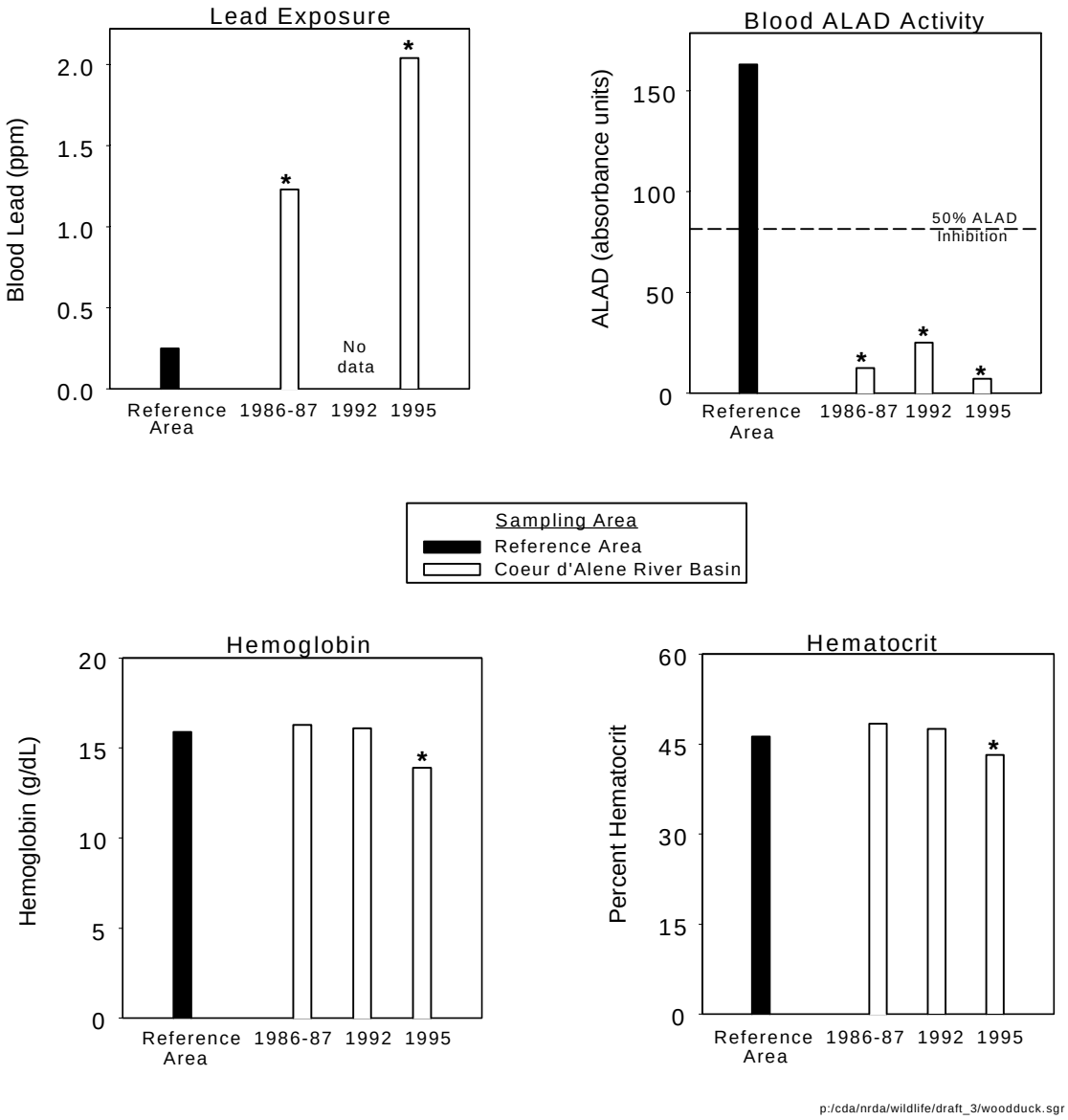


Figure 6-19. Comparison of lead exposure and effects in Coeur d'Alene River basin wood ducks over time. Reference area values are combined data for 1986 to 1995. An asterisk (*) indicates a significant difference ($p < 0.05$) between the Coeur d'Alene and reference areas. Dashed line in top right panel shows 50% ALAD inhibition level relative to reference levels. Lead exposure and ALAD activity data are geometric means.
Source: Blus et al., 1997.

Lead exposure of moribund tundra swans captured in 1994-1995 was significantly greater in the Coeur d'Alene River basin (mean blood lead of 3.3 ppm; range of 0.5 to 6.2 ppm) than in reference areas (0.11 ppm; range 0.02 to 28 ppm) ($p < 0.0001$) (Blus et al., 1999). Mean blood lead concentration of apparently healthy birds captured in the Coeur d'Alene River basin (mean 1.8 ppm, range 1.2 to 3.8 ppm) was also significantly greater than the reference mean. Excluding two extreme values that may have been related to lead shot (3 and 28 ppm), mean blood lead concentration in reference area birds was 0.08 ppm (Blus et al., 1999).

Moribund tundra swans from the Coeur d'Alene River basin exhibited physiological impairments, including statistically significant reductions in ALAD activity and hemoglobin compared to swans from reference areas. ALAD was inhibited by 93% in Coeur d'Alene tundra swans, and ALAD activity was significantly negatively correlated with the concentrations of lead in swan blood ($p < 0.001$; $r^2 = 0.75$). Hemoglobin and hematocrit levels were also significantly negatively correlated with blood levels, but appeared to show a threshold response. Hemoglobin concentrations and percent hematocrit were both relatively constant at blood lead concentrations between 0.01 and 1 ppm and declined at blood lead concentrations greater than 2 ppm lead (Blus et al., 1999).

The persistence of elevated exposure and continuing effects on swans was evaluated by comparing current data (1994 and 1995) to data collected in 1987 (Blus et al., 1991). Lead poisoning was the cause of death of 14 of 15 moribund swans collected in the Coeur d'Alene River basin in 1994-1995, and of all of 4 moribund swans collected in 1987 (Blus et al., 1991; Blus et al., 1999). Necropsy results confirmed that liver lead concentrations of the 18 lead poisoned swans ranged from 6.4 to 40 µg/g, and that all 18 swans showed signs of emaciation. Only one of the 18 lead poisoned swans contained ingested lead shot in the gizzard. There was no significant difference in liver lead concentrations between swans captured in 1994-1995 and swans captured in 1987.

Comparisons of blood and liver lead concentrations and blood parameters responsive to lead exposure indicated no reduction in lead exposure or physiological malfunctions in tundra swans from the Coeur d'Alene River basin between 1987 and 1995 (Figure 6-20) (Blus et al., 1999). Liver lead concentrations measured in lead poisoned swans in 1994-1995 by Blus et al. (1999; range of 9 to 34) were similar to liver residues in tundra swans measured in 1974 (Benson et al., 1976; range of 7 to 43 ppm; 1 of 13 birds had ingested lead shot).

These results indicate that tundra swans in the Coeur d'Alene River basin contain elevated tissue concentrations of lead, that they experience both lethal and sublethal effects characteristic of lead exposure, that most lead poisoning occurs without the presence of ingested lead artifacts, and that exposure has not diminished during the past 20 years.

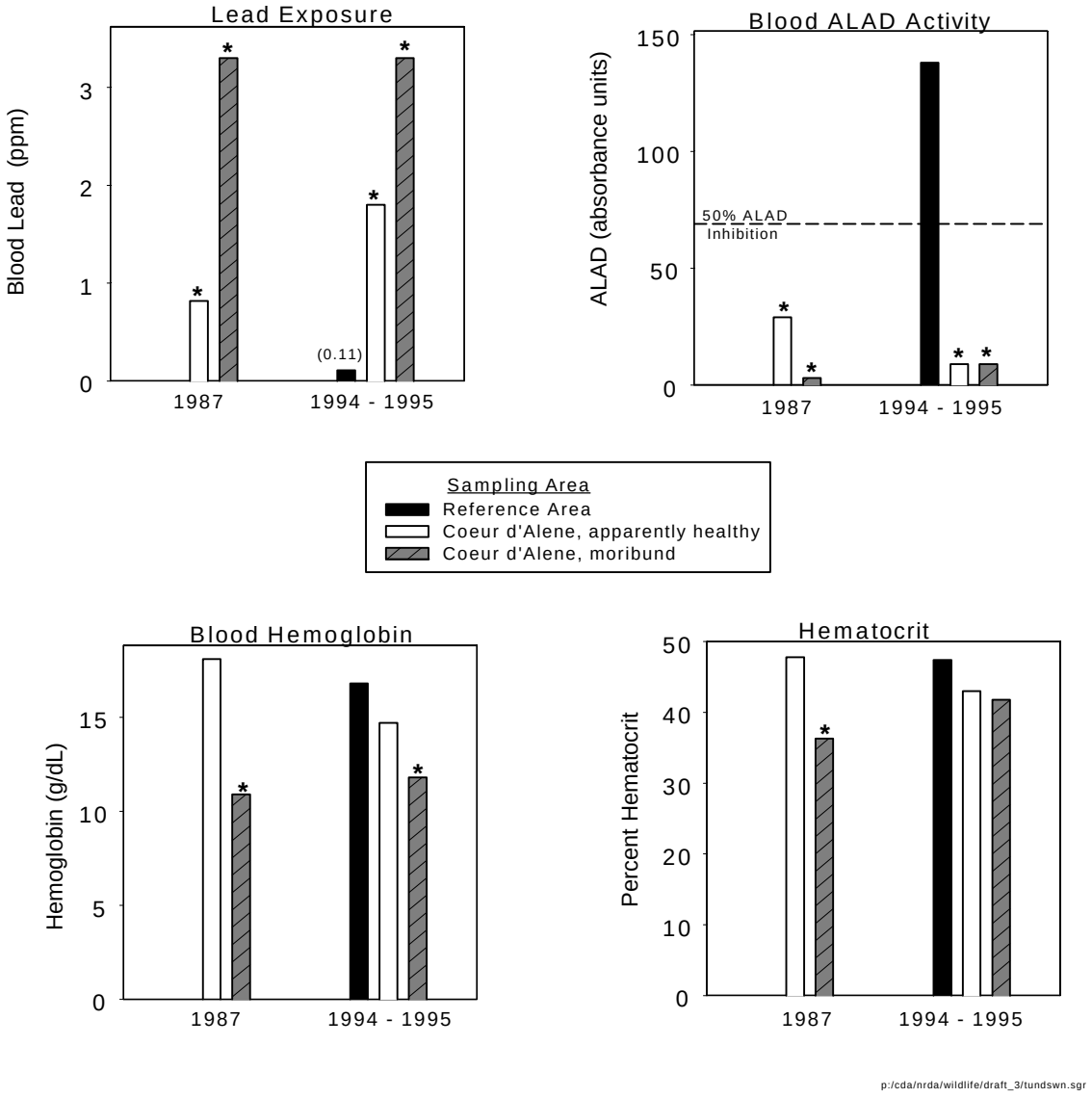


Figure 6-20. Comparison of lead exposure and effects in Coeur d'Alene River basin tundra swans over time. Reference area values are combined data for 1994 and 1995. Figure legends designate birds observed to be apparently healthy (not impaired) and sick (impaired). An asterisk (*) indicates a significant difference ($p < 0.05$) between the Coeur d'Alene and reference areas. Dashed line in the top right panel shows 50% ALAD inhibition level relative to reference levels. Lead exposure and ALAD activity data are geometric means.
Source: Blus et al., 1997.

Canada geese. Lead exposure (lead concentrations in blood, liver, and kidney) and hematological responses (changes in blood ALAD activity, protoporphyrin, hemoglobin, hematocrit) in adult and young Canada geese (goslings) from the Coeur d'Alene River basin were compared to lead exposure and hematological data from the Canada geese adults and goslings from reference areas (Henny et al., 1999). Reference areas included McArthur Wildlife Management Area (northern Idaho), a Snake River location (near Lewiston, Idaho), and the St. Joe River basin.

Lead exposure in goslings from the Coeur d'Alene area (mean blood lead 0.28 ppm; range 0.12 to 1.2 ppm) was significantly greater than in reference area goslings of comparable body mass (0.01 ppm; range <0.001 to 0.15; $p < 0.0001$) (Henny et al., 1999). Lead exposure was also significantly greater in adults from the Coeur d'Alene River basin (0.41 ppm; range 0.26 to 1.3) than in reference area adults (0.02 ppm; range 0.002 to 0.14) ($p < 0.01$). Coeur d'Alene River basin adults and goslings exhibited physiological impairments, including 65 to 86% inhibition of blood ALAD activity ($p < 0.0001$), 132 to 1523% elevation of blood protoporphyrin, and 3 to 12% reduction in hemoglobin and hematocrit relative to reference geese (Figure 6-21) (Henny et al., 1999).

In general, Coeur d'Alene goslings and adult life stages of Canada geese exhibited similar lead exposure and effects. These results indicate that both young and adult life stages of Canada geese in the Coeur d'Alene River basin contain elevated tissue concentrations of lead and that they experience effects characteristic of lead exposure.

Mallard ducks. Lead exposure (lead concentrations in blood, liver, and kidney), hematological responses (changes in blood ALAD activity, protoporphyrin, hemoglobin, hematocrit), and physical deformations (gross and histological lesions) in adult and young (hatch year; HY) mallard ducks from the Coeur d'Alene River basin were compared to lead exposure and hematological data from birds from reference areas (Henny et al., 1999). Reference areas included McArthur Wildlife Management Area (northern Idaho), Turnbull National Wildlife Refuge (eastern Washington), and the St. Joe River basin.

Lead exposure was significantly greater in Coeur d'Alene HY mallard ducks (mean blood lead 0.98 ppm; range 0.25 to 6.6 ppm) than in reference area HY mallard ducks (0.02 ppm; range of 0.007 to 0.51) ($p < 0.001$) (Figure 6-22) (Henny et al., 1999). Lead exposure was also significantly greater in adult mallard ducks from the Coeur d'Alene River basin (1.8 ppm; range 0.19 to 17.4 ppm) than in reference area adults (0.03 ppm; range 0.004 to 0.81; $p < 0.001$). Three of the 22 mallards (14%) euthanized from the Coeur d'Alene River basin contained ingested lead shot.

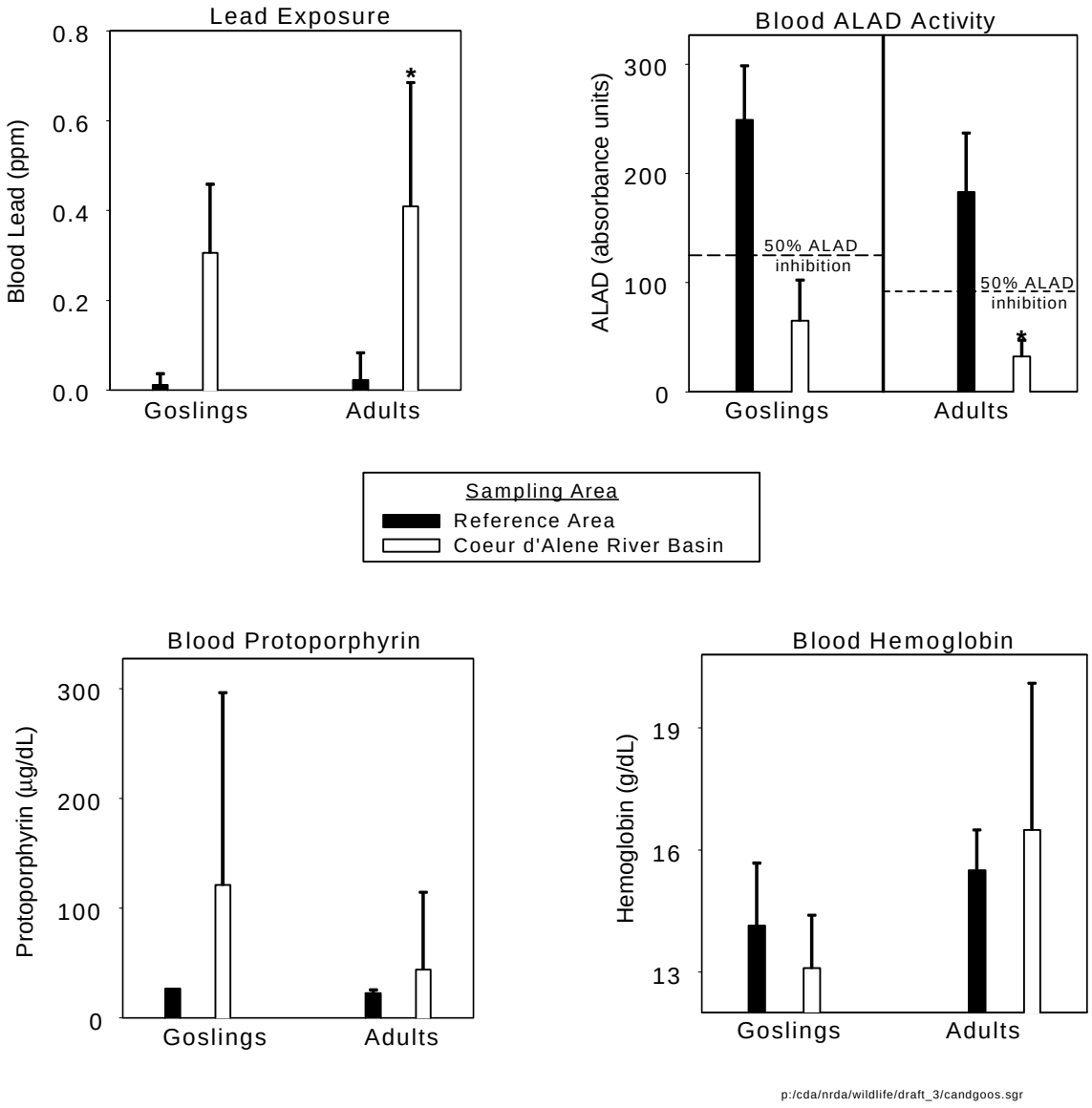


Figure 6-21. Comparison of lead exposure and effects in Canada geese goslings and adults from the Coeur d'Alene River basin and reference areas. An asterisk (*) indicates a significant difference ($p < 0.05$) between the Coeur d'Alene and reference areas. Dashed lines in top right panel show 50% ALAD inhibition level relative to reference levels.
Source: Henny et al., 1999.

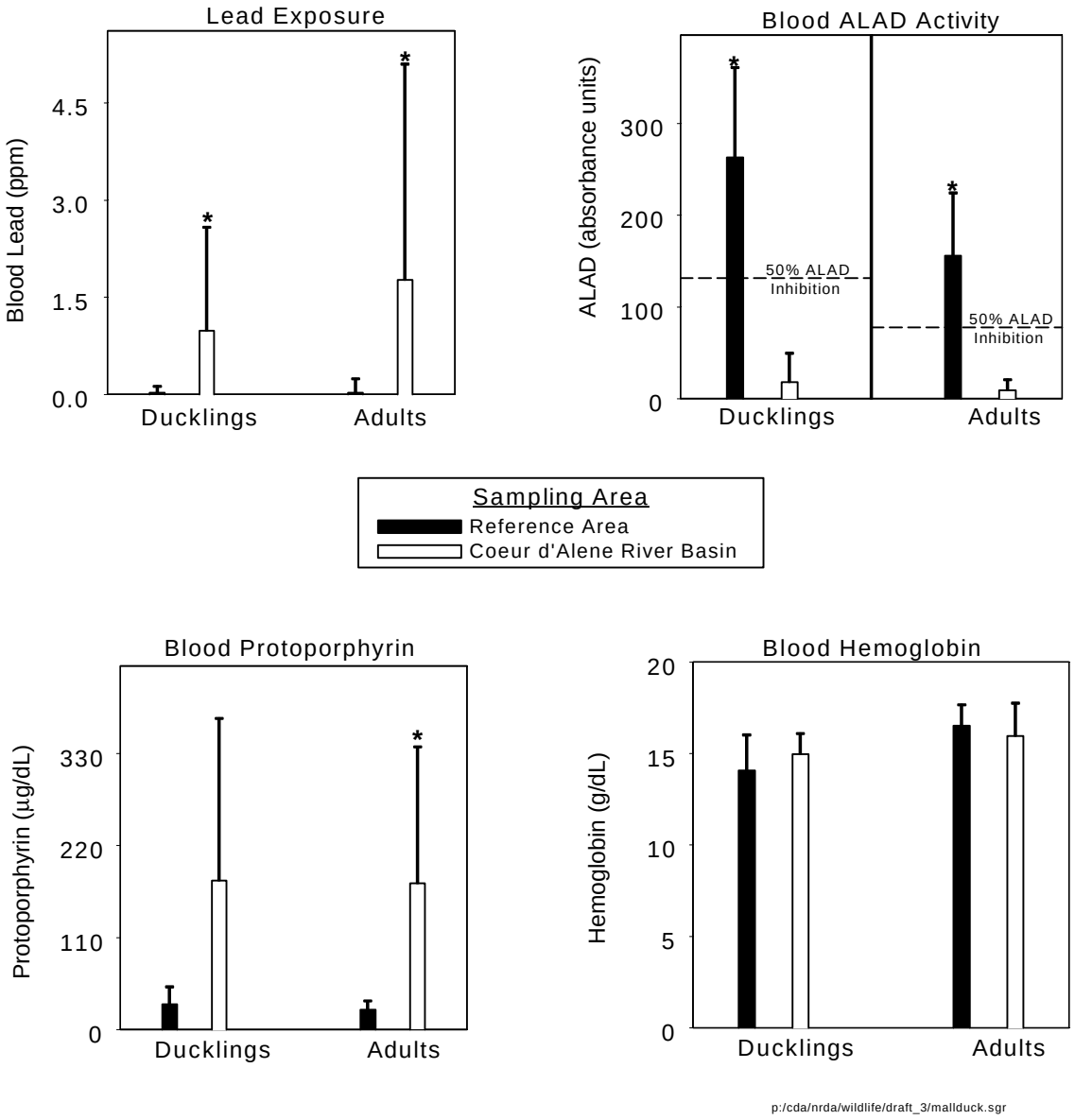


Figure 6-22. Comparison of lead exposure and effects in hatch year mallards and adults from the Coeur d'Alene and reference areas. An asterisk (*) indicates a significant difference ($p < 0.05$) between the Coeur d'Alene and reference areas. Dashed lines in top right panel show 50% ALAD inhibition level relative to reference levels. Source: Henny et al., 1999.

Ninety-four percent of the Coeur d'Alene River basin HY mallard ducks exhibited greater than 50% ALAD inhibition. Ninety percent of Coeur d'Alene River basin adults exhibited significant ALAD inhibition relative to reference adult mallard ducks ($p < 0.0001$), and all showed greater than 50% ALAD inhibition. Protoporphyrin concentrations were significantly elevated in Coeur d'Alene HY mallard ducks and in adult mallard ducks compared to birds from reference areas ($p < 0.0004$ for HY, $p < 0.002$ for adults). In Coeur d'Alene River basin HY mallard ducks, protoporphyrin was elevated by approximately 590%, and in adults, by approximately 740%.

The majority of the young and adult mallard ducks captured from the Coeur d'Alene River basin also exhibited physiological deformations related to lead exposure, including poor body condition, hepatic hemosiderosis, and renal necrosis (Henny et al., 1999). In general, lead exposure and effects were similar in mallard duckling and adult life stages.

The study results indicate that young and adult mallard ducks from the Coeur d'Alene River basin contain elevated tissue concentrations of lead, that they experience physiological effects characteristic of lead exposure, and that lead poisoning typically occurs without the presence of ingested lead artifacts.

Lead Exposure and Effects in Bald Eagles

To evaluate lead exposure and effects on bald eagles in the Coeur d'Alene River basin, blood samples were collected from young bald eagles and hematological parameters (blood ALAD activity, hemoglobin and hematocrit levels) and growth were measured. Blood samples and growth data were collected from eagles in nests in the Coeur d'Alene River basin and at McArthur Lake, MWMA (Audet et al., 1999b). Food chain exposure was evaluated by measuring lead concentrations in eagle prey items, including muskrats *(Ondatra zibethicus)*, brown bullheads (*Ameriurus nebulosus*), and other fish species collected from the Coeur d'Alene and St. Joe river basins, and by measuring lead concentrations in lead-poisoned waterfowl (without ingested lead shot) from the Coeur d'Alene River basin (Audet, 1997*;* Audet et al., 1999b).

Blood lead levels were higher in Coeur d'Alene River basin eaglets (0.03 to 0.18 ppm) than in reference eaglets (0.01, 0.02 ppm) (Audet et al., 1999b). Blood protoporphyrin and hemoglobin were similar in Coeur d'Alene River basin and reference area eaglets. ALAD was inhibited by 35 to 65% in Coeur d'Alene River basin eaglets. The average weight of Coeur d'Alene eaglets was lower than the average weight of reference area eaglets of similar age. For both blood and growth measurements, sample sizes were small, so statistics were not provided.

Prey items of eagles in the Coeur d'Alene River basin were contaminated with lead (Figure 6-14). For example, lead concentrations were significantly greater in brown bullheads from the Coeur d'Alene River basin (range 3.8 to 122 ppm) than in brown bullheads from the St. Joe River basin (range <0.1 to 2.9 ppm) ($p < 0.0001$). Lead concentrations in tissues of lead-poisoned waterfowl prey items from the Coeur d'Alene area were elevated and ranged from 1.64 to 38.0 ppm in liver and from <0.09 to 0.76 ppm in muscle (Audet, 1997).

For comparison, dead bald eagles from northern Idaho and eastern Washington were necropsied to determine causes of death. Lead poisoning without the presence of ingested lead shot was the most common diagnosis of dead bald eagles collected in northern Idaho/eastern Washington. Of the 13 carcasses documented, 10 were suitable for necropsy. Six of the 10 carcasses necropsied, including 2 of the 4 carcasses collected from the Spokane River basin (which includes the Coeur d'Alene River basin), were lead-poisoned without ingested lead shot (Audet et al., 1999b).

The results of the blood parameter and growth comparisons and the food chain exposure studies indicate that bald eagles of the Coeur d'Alene River basin are exposed to elevated concentrations of lead in prey items, have elevated blood lead concentrations, and have reduced blood ALAD activity. Four types of lead exposure were considered possible: ingestion of lead shot embedded in the tissues of waterfowl prey; ingestion of lead sinkers in fish; ingestion of lead in offal; and ingestion of lead in sediments, either directly or in prey (Audet et al., 1999b). Lead poisoning without ingested artifacts was documented as a cause of bald eagle death in northern Idaho and eastern Washington.

Lead Exposure and Effects in Songbirds

Biological reconnaissance sampling conducted in 1992 indicated that floodplain songbirds in the Coeur d'Alene River basin were exposed to elevated concentrations of lead (Audet, 1997). Songbirds, which feed on insects, worms, and other invertebrates, are exposed to lead in the Coeur d'Alene River basin by routes other than ingestion of lead artifacts and incidental consumption of soil while feeding. To evaluate lead exposure (liver lead) and effects (changes in blood ALAD activity and hematocrit) in songbirds, song sparrows and American robins were sampled in 1995 in the floodplain of the lower Coeur d'Alene River basin and in reference areas (Johnson et al., 1999). Reference areas included the North Fork Coeur d'Alene River and the St. Joe River basin.

Liver lead concentrations were significantly greater in song sparrows collected from the lower Coeur d'Alene River basin than in sparrows from reference areas (mean 1.9 ppm versus 0.10 ppm, $p = 0.0079$; Johnson et al., 1999). Blood ALAD activity in song sparrows and robins from the Coeur d'Alene River basin was significantly inhibited relative to ALAD activity in reference birds ($p = 0.004$). Inhibition of blood ALAD activity averaged 51% in Coeur d'Alene

River basin song sparrows and 75% in robins. ALAD activity was inhibited by greater than 50% in 43% of Coeur d'Alene River basin song sparrows and in 84% of Coeur d'Alene River basin robins (Johnson et al., 1999).

These data indicate that songbirds inhabiting the floodplain of the Coeur d'Alene River basin are exposed to elevated lead concentrations and exhibit physiological malfunctions from lead exposure.

6.5.3 Injury Laboratory Studies

In addition to the field studies described above, the Trustees performed a series of controlled laboratory feeding experiments to examine the relationship between ingestion of lead-contaminated Coeur d'Alene River basin sediments and lead exposure and effects. In each experiment, sediments from the Coeur d'Alene River basin were mixed with waterfowl feed (to simulate naturally occurring sediment ingestion) and fed to representative species of waterfowl, including mallards, Canada geese, and mute swans (a surrogate for tundra swans) (Table 6-6). Measurement endpoints included death, physiological malfunctions (e.g., changes in blood parameters, body weight), and physical deformations (gross and histological lesions).

Table 6-6
Experimental Design of Laboratory Studies[a]

Test Species	Life Stage	Exposure (% sediment)	Food Matrix Tested	Duration (weeks)	Study Authors
Mallard duck	Subadult	Ingested dose: 3% to 20% (Experiment 1)	Commercial diet (pelletized)	5 to 10	Heinz et al. (1999)
		Ingested dose: 14%, 17% (Experiment 3)	Commercial diet (mash) or ground corn diet	15	
	Juvenile	Nominal exposure: 12%, 24%	Commercial diet (mash) or 2/3 ground corn/commercial diet (mash)	6	Hoffman et al. (1998)
Canada goose	Juvenile	Nominal exposure: 12% to 48%	Commercial diet (mash)		
Mute swan	Juvenile	Nominal exposure: 12%, 24%	Commercial diet (pelletized) or ground rice diet (pelletized)	7	Day et al. (1998)

a. Measurements: ALAD, blood lead, hematocrit, hemoglobin, metals in kidney and liver, protoporphyrin, survival, tissue lesions, body weight, lead in feces (Heinz et al. and Day et al. only), and blood enzymes (Hoffman et al. and Day et al. only).

The controlled laboratory experiments enabled investigators to (1) expose animals in a controlled setting to field collected sediments in the absence of lead shot; (2) alter the type of diet mixed with the sediment to investigate food matrix effects; and (3) expose animals to increasing concentrations of lead in sediment to evaluate exposure or dose-response relationships. Although laboratory studies allow for precise testing of dietary exposure conditions, they may result in an underestimate of the actual toxicity of lead contaminated sediments since laboratory studies are generally conducted under less stressful conditions than animals encounter in the wild (e.g, absence of food limitations, predators, temperature extremes).

Waterfowl were exposed to laboratory diets prepared with sediment from either the Coeur d'Alene River basin (average 3,700 ppm lead, range 3,400-4,000 ppm) or the St. Joe River basin reference area (8 ppm lead, range 6.3-9.7 ppm). All sediments were sieved (1 mm) to remove lead artifacts (Heinz et al., 1999). Lead exposure concentrations in the laboratory diets were produced by mixing sediments with feed. For example, the commercial and corn diets prepared from 24% Coeur d'Alene River basin sediment in the Heinz et al. (1999) study contained 950 and 870 ppm lead, respectively.

Nominal exposure levels (expressed as the percent sediment in bird feed) were selected based on the amount of sediment that waterfowl ingest in the field (Beyer et al., 1998b). The highest exposure levels were selected to approximate the upper ninetieth percentile of sediment ingestion of tundra swans in the Coeur d'Alene River basin, or 22% sediment ingestion (Beyer et al., 1998b). Fecal samples were collected in the laboratory studies by Heinz et al. (1999) and Day et al. (1998) to allow comparison to sediment content and lead concentrations measured in fecal samples collected in the field.

Feeding experiments were conducted with either a nutritionally complete commercial waterfowl feed or less nutritious diets containing corn (mallard and Canada goose studies) or rice (swan study) that are more representative of natural diets in the field. The different diets were used to evaluate the effect of the food matrix (pelleted or mash diets; commercial, corn, or rice diets) on the bioavailability of sediment lead to waterfowl. In the wild, waterfowl may ingest a diversity of food items that are less nutritious than commercial diets (Day et al., 1998). Less nutritious diets (e.g., low in calcium) have been shown to increase lead accumulation in birds (e.g., Scheuhammer, 1996).

Toxicity of Sediments to Subadult Mallard Ducks

To evaluate relationships between ingestion of lead-contaminated sediment and biological responses, subadult mallard ducks (20 to 30 weeks old) were fed either Coeur d'Alene sediment or reference area sediment in a commercial duck feed or in a nutritionally deficient ground corn diet (Heinz et al., 1999). Dietary mixtures with Coeur d'Alene sediment contained from 3 to 24%

sediment. The 24% sediment diet contained 870 to 950 ppm lead. The dietary mixture with reference sediment contained 24% sediment and approximately 3 ppm lead. Measurement endpoints included lead and zinc concentrations in blood, kidney, and liver; survival; physiological malfunctions (weight loss, changes in blood ALAD activity and protoporphyrin, hemoglobin, and hematocrit levels); and physical deformities (gross and histopathological lesions). The results are expressed as the ingested dose of sediment (% in diet), estimated from analysis of fecal samples using the same procedures used to estimate mallard duck sediment ingestion in the field.

Experimental dietary groups of mallard ducks fed increasing percentages of Coeur d'Alene sediment in a commercial diet showed corresponding increases in lead exposure (lead concentrations in blood and liver) in the absence of lead shot (Figure 6-23). In comparison, ducks that consumed reference area sediment exhibited minimal lead exposure (Figure 6-23). The positive relationship between Coeur d'Alene River basin sediment ingestion and blood and liver lead concentrations demonstrate that lead in Coeur d'Alene River basin sediment is bioavailable to waterfowl.

With the pelletized commercial diet (Experiment 1 of Heinz et al., 1999), nominal exposures (% sediment in feed) were nearly identical to the estimated ingested doses (% ingested). Mallards fed pellets containing 3, 6, 12, and 24% Coeur d'Alene sediment actually ingested an estimated average of 3.2, 6.5, 11, and 19% sediment (Heinz et al., 1999). Mallards fed the ground corn diet ingested less sediment than mallards fed the pelletized commercial diet. Mallards fed the mash commercial diet consumed the least amount of sediment, possibly because of sorting by the ducks. For example, mallards fed 24% sediment in the pelletized commercial diet (Experiment 1 of Heinz et al., 1999) ingested 27% sediment and mallards fed 24% sediment in the mash commercial diet (Experiment 2 of Heinz et al., 1999) ingested only 11% sediment. Lead concentrations in feces of mallards exposed to Coeur d'Alene sediments in the laboratory tests (means of 284 to 1660 ppm) were within the range measured in the field by Beyer et al. (1998b) (2.3 to 3,600 ppm; mean of 230; median of 98 ppm).

Even low levels of Coeur d'Alene River basin sediment ingestion (average of 3.2%) caused physiological malfunctions, including depression of ALAD activity greater than 50% and elevation of blood protoporphyrin (Figure 6-23; Heinz et al., 1999). Higher sediment ingestion rates resulted in increasing effects, including significant reductions in hemoglobin levels (Figure 6-23). Mallards that ingested an average of 19% Coeur d'Alene River basin sediment in the commercial diet exhibited physical deformations, including atrophy of the breast muscles, green staining of the feathers around the vent, viscous bile, green staining of the gizzard lining, and RIIBs (Figure 6-24). One of 10 mallards from the 19% Coeur d'Alene River basin sediment ingestion group died. Necropsy observations of atrophied breast muscles and green stained gizzard lining confirmed that the cause of death was lead poisoning (Heinz et al., 1999).

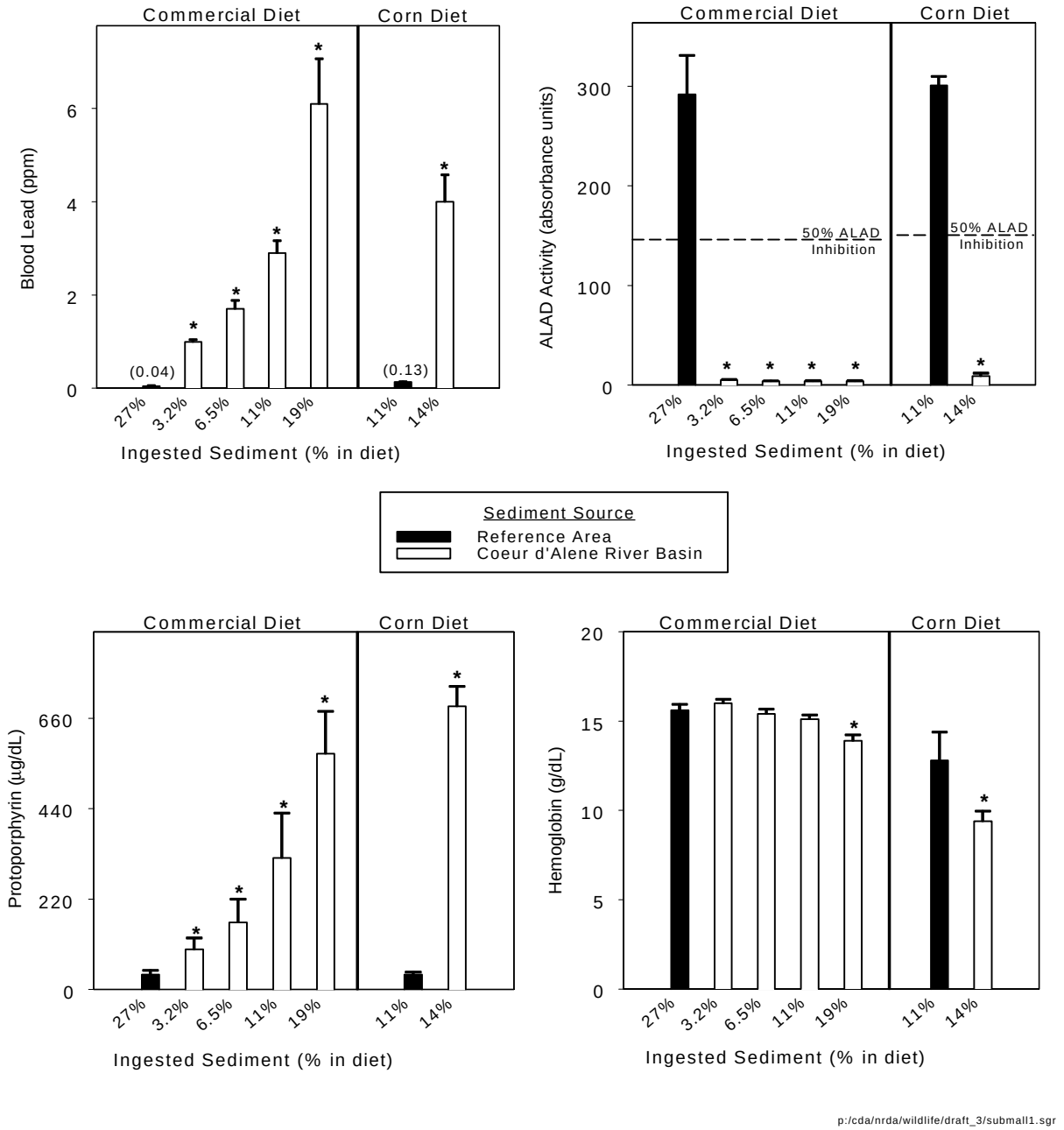


Figure 6-23. Blood liver lead residues (top left), blood ALAD activity (top right), blood protoporphyrin levels (bottom left), and blood hemoglobin levels (bottom right) of subadult mallard ducks ingesting sediment from the Coeur d'Alene River basin or the reference area. Birds were provided sediment mixed in either a commercial diet or a ground corn diet. An asterisk (*) indicates a significant difference ($p < 0.05$) in lead concentrations in birds ingesting Coeur d'Alene sediment compared to birds on the same diet ingesting reference sediment.
Source: Heinz et al., 1999; Experiments I and III.

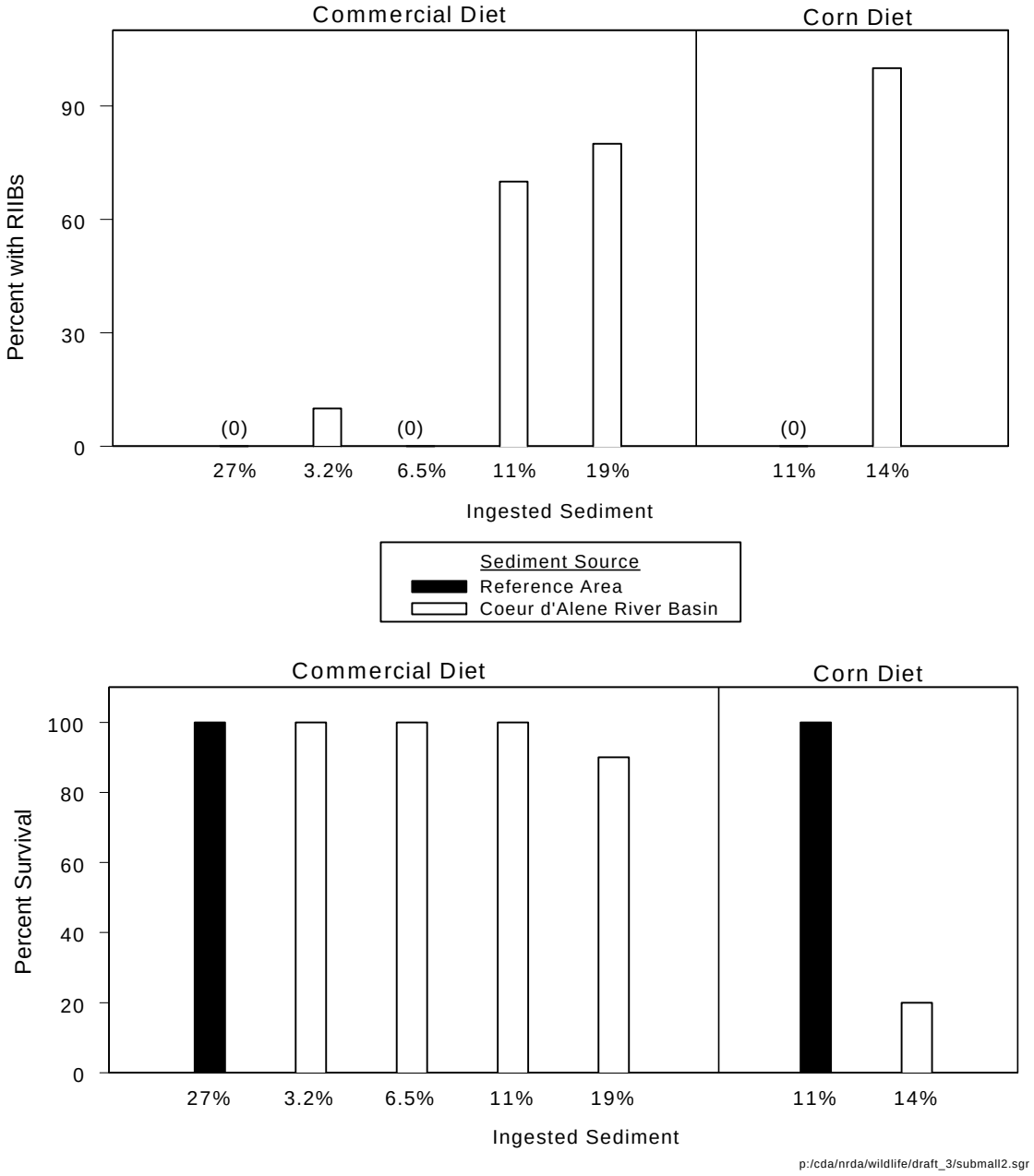


Figure 6-24. Comparison of the frequency of renal intranuclear inclusion bodies (RIIBs) (top panel) and survival (bottom panel) in subadult mallard ducks ingesting sediment from the Coeur d'Alene River basin or the reference area. Birds were provided sediment mixed in either a commercial diet or a ground corn diet. Source: Heinz et al., 1999; Experiments I and III.

When birds were fed a nutritionally deficient ground corn diet, the effects of ingestion of Coeur d'Alene River basin sediment relative to ingestion of reference area sediment were more severe (Figures 6-23 and 6-24; Heinz et al., 1999). In addition to significant changes in blood and liver lead concentrations, ALAD activity, protoporphyrin, and hemoglobin levels, mallards that ingested an average of 14% Coeur d'Alene sediment showed a 27% reduction in body weight relative to birds fed the corn diet and reference area sediment. Blood ALAD activity was depressed by 97% relative to reference birds (Figure 6-23). All birds ingesting Coeur d'Alene River basin sediment on the corn diet had physical deformations, including emaciation and RIIBs (Figure 6-24; Heinz et al., 1999). Four of the five birds (80%) ingesting the corn diet with 14% Coeur d'Alene sediment died from lead poisoning during the 15 week exposure period (Figure 6-24).

The feeding experiments show that lead in Coeur d'Alene River basin sediments is bioavailable to mallard ducks, that lead poisoning results from exposure to Coeur d'Alene River basin sediments in the absence of lead shot, and that the number and severity of effects increase as sediment ingestion increases. Effects observed in the feeding experiments were similar to responses observed during field investigations, including hematological changes, physical deformations, and death. The degree of lead exposure and the number and severity of effects were greater in mallards fed a nutritionally deficient corn diet more similar to field conditions than in those fed a nutritionally complete commercial diet.

Toxicity of Sediments to Goslings and Ducklings

The relationship between ingestion of lead-contaminated sediment and biological responses was also evaluated in laboratory experiments with Canada goslings and mallard ducklings (Hoffman et al., 1998). Birds were fed either Coeur d'Alene River basin sediment or reference area sediment in a commercial duck feed. Mallard ducklings were also fed a less nutritious corn/commercial diet mixture. Dietary mixtures with Coeur d'Alene sediment contained from 12 to 48% reference area sediment. The 48% reference sediment diet contained an estimated 1656 ppm lead. The dietary mixture with reference sediment contained 24 and 48% sediment. The 48% sediment diet contained approximately 5.5 ppm lead. Measurement endpoints included lead and zinc concentrations in blood, kidney, and liver; survival; physiological malfunctions (loss of body weight; changes in blood ALAD activity, and protoporphyrin, hemoglobin, and hematocrit levels; changes in blood and liver biochemical parameters); and physical deformations (gross and histopathological lesions). The results are expressed as a nominal exposure (% sediment in feed provided to the birds) rather than the actual dose ingested because the amount of sediment actually consumed was not measured. All birds were fed mash diets (both commercial and corn diets), so actual sediment ingestion may have been less than the nominal exposure concentration based on the results of Heinz et al. (1999) that were previously presented.

With a commercial diet, the lowest experimental exposure to Coeur d'Alene River basin sediment (12%) resulted in elevated tissue concentrations of lead and physiological malfunctions in both goslings and ducklings, including significant elevation of blood protoporphyrin levels and depression of ALAD activity greater than 50% ($p \leq 0.05$; Figures 6-23 and 6-25; Hoffman et al., 1998). Higher sediment exposure levels resulted in an increase in lead concentrations in tissues and greater frequency and degree of physiological malfunctions characteristic of lead exposure, including significant reductions in hemoglobin concentrations, changes in blood and liver biochemical parameters, and weight loss ($p \leq 0.05$; Figures 6-23, 6-24, and 6-25). Both the goslings and the ducklings exposed to Coeur d'Alene River basin sediment exhibited physical deformations, including RIIBs in ducklings (Figure 6-24). Twenty-two percent of the goslings in the highest exposure group (48% Coeur d'Alene River basin sediment) died (Hoffman et al., 1998).

With the less nutritionally complete diet containing corn (two-thirds mixture of ground corn and commercial diet), the effects of ingestion of Coeur d'Alene River basin sediment were generally more severe (Figures 6-26 and 6-27; Hoffman et al., 1998). In addition to showing significant changes in ALAD activity (96% depression) and protoporphyrin levels ($p \leq 0.05$), the ducklings exposed to 24% Coeur d'Alene River basin sediment in the corn diet showed a 20% reduction in body weight relative to control birds fed the corn diet with added reference area sediment. Ducklings ingesting Coeur d'Alene River basin sediment in the corn diet also demonstrated physical deformations, including both RIIBs and brain lesions (Figure 6-27; Hoffman et al., 1998).

Weight loss in both goslings and ducklings occurred at levels of Coeur d'Alene River basin sediment exposure that caused other physiological impairments such as reductions in hemoglobin levels. For example, in the commercial diet treatments, significant reductions in the tarsus length of goslings were observed in birds fed a diet containing 48% sediment, and reductions in the brain weight of ducklings were observed in birds fed a diet containing 24% sediment ($p \leq 0.05$). These results suggest that growth of waterfowl in the Coeur d'Alene River basin may be impaired as physiological impairments occur. Reduced growth in birds is associated with reduced viability and impaired reproduction in field populations (O'Connor, 1984; Harris et al., 1993).

The relative bioavailability of lead in Coeur d'Alene River basin sediment was evaluated by comparing tissue concentrations and effects in ducklings exposed to diets containing Coeur d'Alene River basin sediments to tissue concentrations and effects in ducklings fed reference area sediment mixed with a form of lead known to be biologically available (lead acetate).

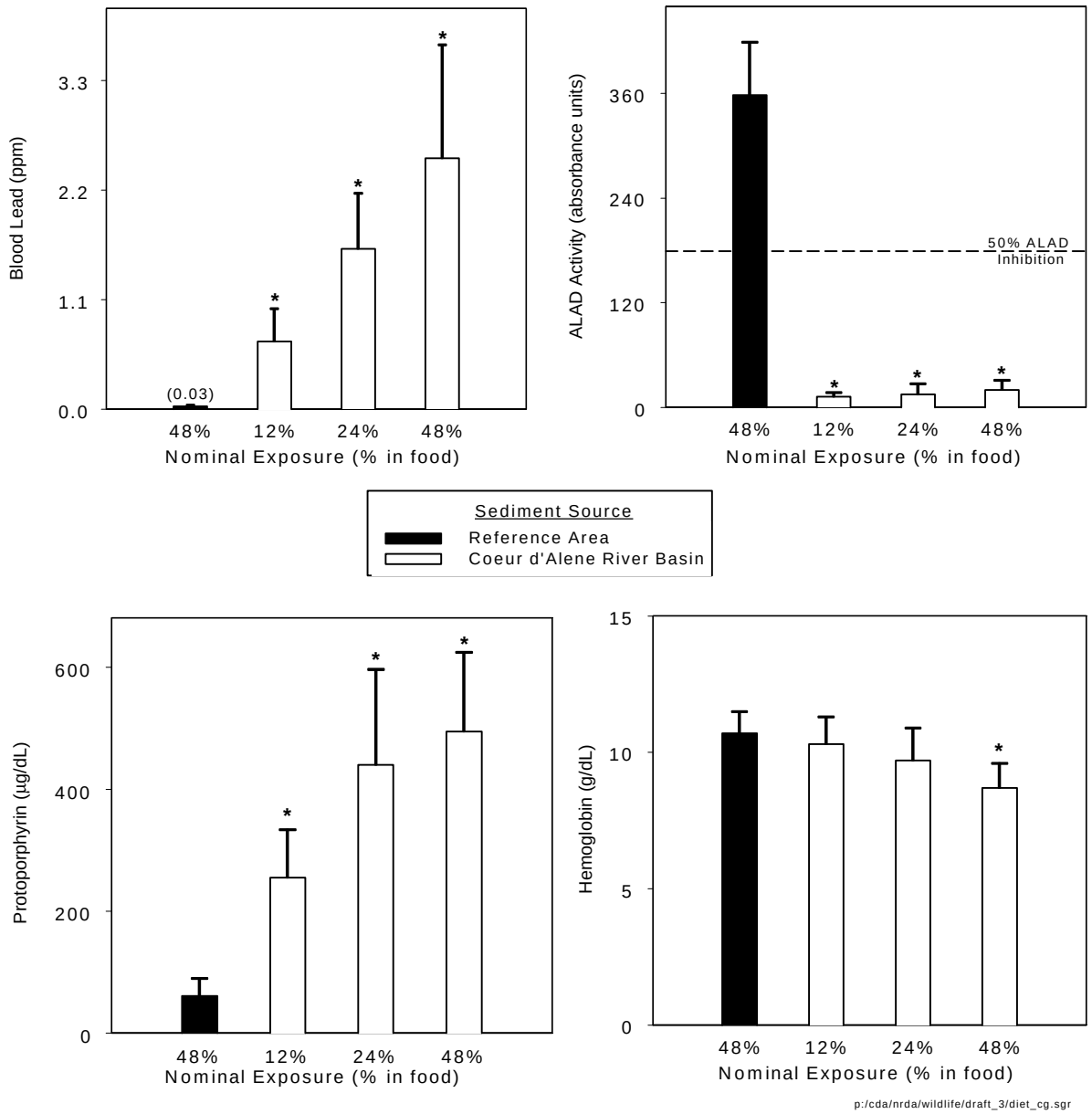


Figure 6-25. Blood lead (top left) and blood ALAD activity (top right), blood protoporphyrin levels (bottom left), and blood hemoglobin levels (bottom right) in Canada goslings exposed to sediment from the Coeur d'Alene River basin or the reference area. Birds were provided sediment mixed in a commercial diet. An asterisk (*) indicates a significant difference ($p < 0.05$) in birds ingesting Coeur d'Alene sediment compared to birds ingesting reference sediment. Blood lead and ALAD values are geometric means. All other values are arithmetic means.
Source: Hoffman et al., 1998.

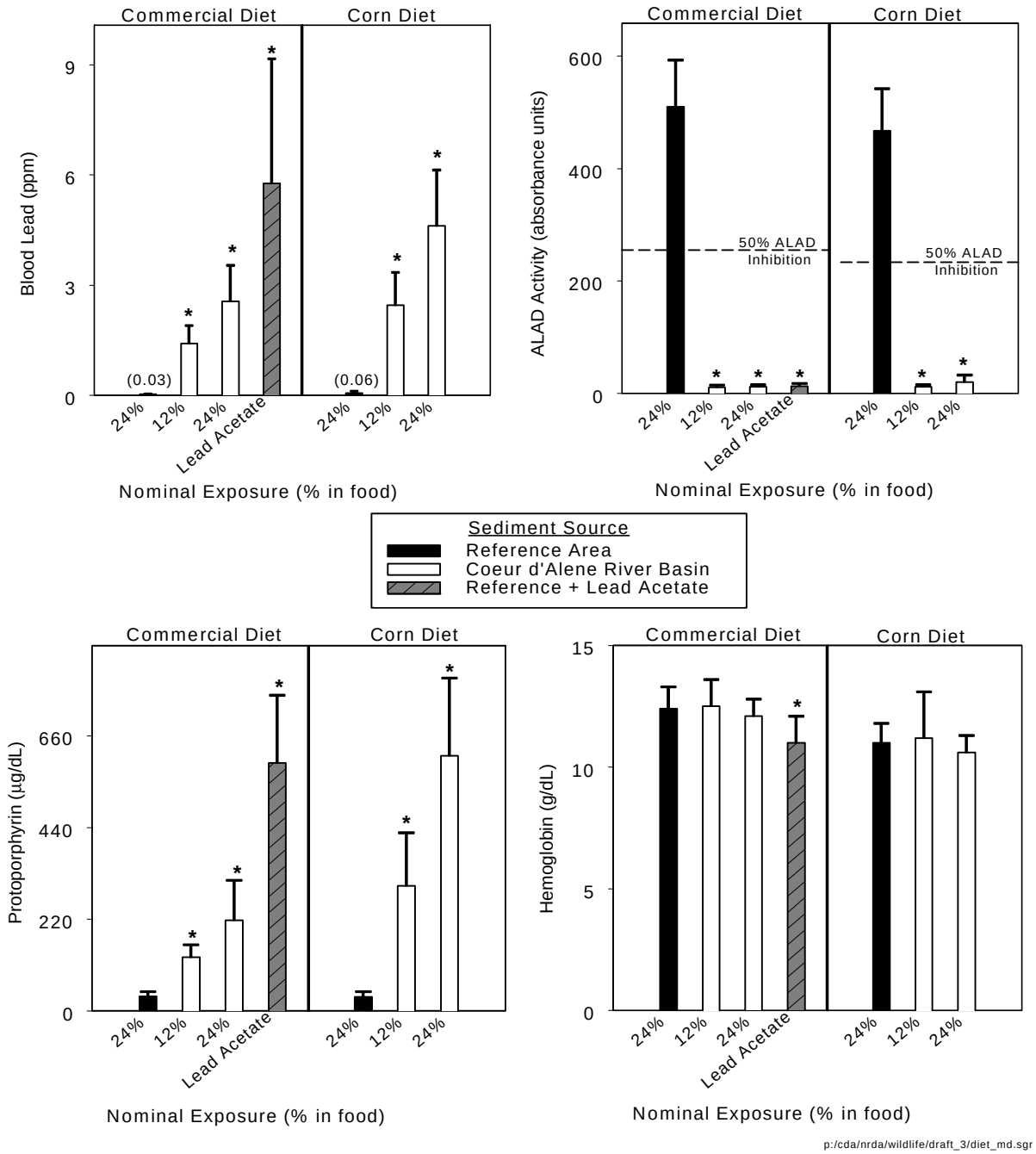


Figure 6-26. Blood lead residues (top left), blood ALAD activity (top right), blood protoporphyrin levels (bottom left), and blood hemoglobin levels (bottom right) in mallard ducklings exposed to sediment from the Coeur d'Alene River basin, the reference area, or to reference sediment spiked with lead acetate (commercial product). Birds were provided sediment mixed in either a commercial diet or a diet containing both corn and commercial feed. An asterisk (*) indicates a significant difference ($p < 0.05$) in birds ingesting Coeur d'Alene sediment compared to birds on the same diet ingesting reference sediment. Blood lead and ALAD values are geometric means. All other values are arithmetic means.
Source: Hoffman et al., 1998.

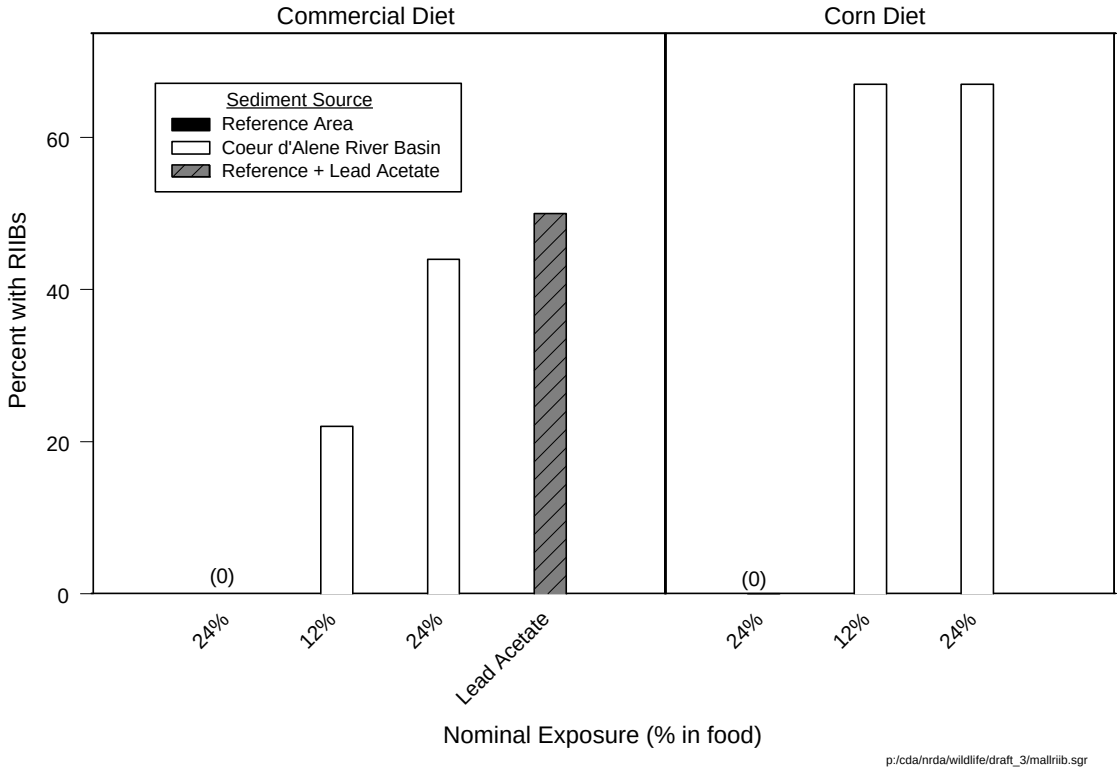


Figure 6-27. Renal intranuclear inclusion bodies (RIIBs) in mallard ducklings exposed to sediment from the Coeur d'Alene River basin, the reference area, or to reference sediment spiked with lead acetate (commercial product). Birds were provided sediment mixed in either a commercial diet or a diet containing corn and a commercial feed.
Source: Hoffman et al., 1998.

Lead acetate is a commercially produced form of lead that has been used in other laboratory toxicity studies (Eisler, 1988). The effects of lead acetate exposure on ducklings were generally similar to effects in mallards fed Coeur d'Alene River basin sediment mixed in the corn diet (Figures 6-26 and 6-27). For example, both treatment groups exhibited a similar degree of physiological malfunctions (20 to 21% reduction in weight relative to reference birds; 17- to 18-fold increase in protoporphyrin levels; greater than 95% depression of ALAD activity; Hoffman et al., 1998). The results demonstrate that lead acetate is more bioavailable than lead in Coeur d'Alene River basin sediment (as measured by tissue lead concentrations), but the types and degree of biological responses caused by both forms of lead are similar.

The feeding experiments show that lead in Coeur d'Alene River basin sediments is bioavailable to both young Canada geese and mallard ducks, that lead poisoning results from exposure to Coeur d'Alene River basin sediments in the absence of lead shot, and that the number and severity of effects increase as sediment ingestion increases. Lead exposure and effects were generally similar in the young of Canada geese and mallards. For example, goslings and

ducklings exposed to 12% Coeur d'Alene sediment (commercial diet) had similar mean blood lead concentrations (1 ppm), inhibition of ALAD activity (>95%), and elevation of protoporphyrin (400% increase). Effects observed in the feeding experiments were similar to responses observed during field investigations, including hematological changes and physical deformations. The degree of lead exposure and the number and severity of effects were greater in mallards fed a less nutritious diet containing corn than in those fed a nutritionally complete commercial diet.

Toxicity of Sediments to Juvenile Mute Swans

The relationship between ingestion of lead-contaminated sediment and biological responses of young of mute swans was evaluated by Day et al. (1998). Mute swans were used as a surrogate for tundra swans because of (1) similar size, (2) similar feeding preferences in aquatic habitats, and (3) the availability of a source of swans not previously exposed to lead. Birds were fed either sediment from the Coeur d'Alene River basin or sediment from a reference area in a commercial diet or a less nutritious rice diet. Dietary mixtures with Coeur d'Alene River basin sediment contained 12 or 24% sediment. The 24% sediment diet contained an estimated 700 to 850 ppm lead. The dietary mixture with reference sediment contained 24% sediment and approximately 4.4 to 5.8 ppm lead.

The nutritional value of the rice diet was more comparable to the preferred diet of swans in the wild (water potato tubers and wild rice) than was the commercial diet. For example, the commercial diet contained 16% protein, 20,600 ppm calcium, and 10,000 ppm phosphorus, whereas the cultivated rice used by Day et al. (1998) contained 7.1% protein, 260 ppm calcium, and 3,000 phosphorus, water potatoes contain 3.2% protein, 380 ppm calcium, and 6,100 ppm phosphorus, and wild rice contains 7.5% protein, 160 ppm calcium, and 3,100 ppm phosphorous.

Measurement endpoints included lead and zinc residues in blood, brain, and liver; survival; physiological malfunctions (weight loss, changes in blood ALAD activity, and protoporphyrin, hemoglobin, and hematocrit levels; changes in plasma and brain biochemical parameters); and physical deformations (gross and histopathological lesions). The results are expressed as a nominal exposure (% sediment in feed provided to the birds), which approximates actual ingestion rates since pelletized feeds were used.

Sediment exposure levels for mute swans (12% and 24% sediment) were selected to be similar to sediment ingestion rates determined for wild tundra swans from the Coeur d'Alene River basin (mean of 9%; 90th percentile of 22%; n = 86, Beyer et al., 1998b). Lead concentrations in feces of swans exposed in the laboratory (1,200 to 2,000 ppm; Day et al., 1998) were within the range of lead in feces measured in the Coeur d'Alene River basin (6 to 3,300 ppm; Beyer et al., 1998b). The overlap indicates that levels of ingestion and exposure to contaminated sediment in the laboratory studies were similar to exposure of wild birds.

Exposure to 12% Coeur d'Alene River basin sediment in the commercial diet caused increased concentrations of lead in tissues and physiological malfunctions, including significant elevation of blood protoporphyrin and depression of ALAD activity greater than 50% ($p \leq 0.05$; Figures 6-28 and 6-29; Day et al., 1998). Exposure to 24% Coeur d'Alene River basin sediment in the commercial diet resulted in more severe effects, including significant reductions in hemoglobin concentrations and hematocrit levels ($p \leq 0.05$; Figures 6-28 and 6-29). In addition, all swans exposed to 24% Coeur d'Alene River basin sediment exhibited physical deformations, including RIIBs (Figure 6-29; Day et al., 1998).

The effects of exposure to Coeur d'Alene River basin sediment were more severe in swans fed the rice diet than in those fed the commercial diet (Figures 6-28 and 6-29). In addition to significant changes in ALAD activity (96% inhibition) and protoporphyrin, hemoglobin, and hematocrit levels ($p \leq 0.05$), swans fed the rice diet containing 24% Coeur d'Alene River basin were significantly smaller (32% lower body weight than birds fed the rice diet and reference area sediment; $p \leq 0.05$) (Figure 6-29). Swans that ingested Coeur d'Alene River basin sediment with the rice diet were ataxic (exhibited loss of equilibrium) and lethargic and had physical deformations, including emaciation and RIIBs (Day et al., 1998).

The feeding experiments show that lead in Coeur d'Alene River basin sediments is bioavailable to swans, that lead poisoning results from exposure to Coeur d'Alene River basin sediments in the absence of lead shot, and that the number and severity of effects increase as sediment ingestion increases. Effects observed in the feeding experiments were similar to responses observed during field investigations, including hematological changes and physical deformations. The degree of lead exposure and the number and severity of effects were greater in swans fed a more environmentally comparable rice diet than in those fed a nutritionally complete commercial diet.

6.5.4 Injury Study Conclusions

The results of controlled laboratory feeding studies demonstrate that lead in Coeur d'Alene River basin sediments is bioavailable to multiple species of migratory birds, that lead poisoning results from exposure to Coeur d'Alene River basin sediments in the absence of lead shot, and that the number and severity of effects increase as ingestion of lead-contaminated sediment increases. These relationships are observed in representative life stages and species of migratory birds, including young and subadult mallards, young Canada geese, and juvenile mute swans. The effects observed in the laboratory were similar to responses observed in multiple species of migratory birds in the wild in the Coeur d'Alene River basin, including hematological changes, physical deformations, and death. The degree of lead exposure and the number and severity of effects were greater in waterfowl fed less nutritionally complete diets that are representative of natural diets in the field.

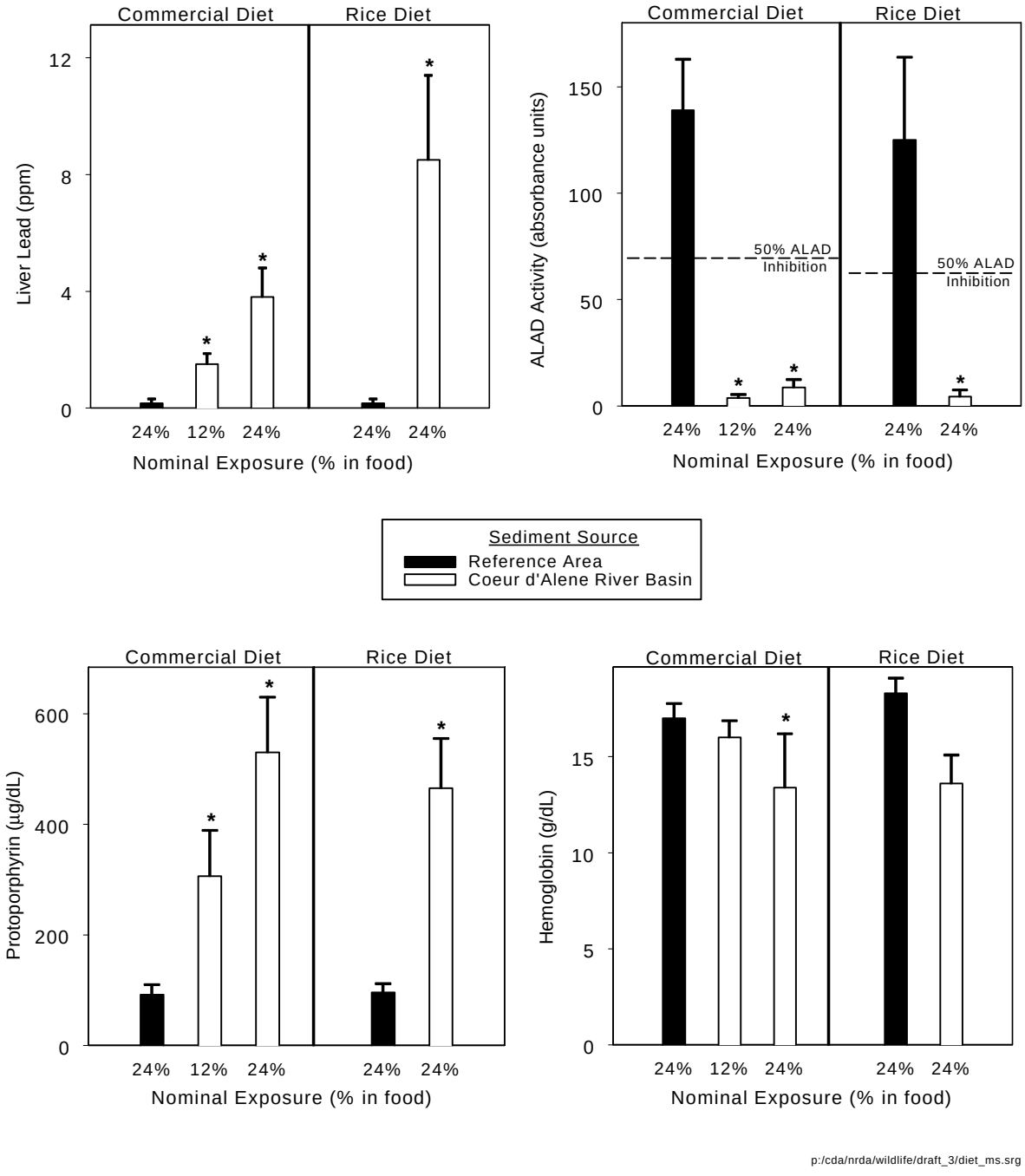


Figure 6-28. Blood and liver lead residues (top left), blood ALAD activity (top right), blood protoporphyrin levels (bottom left), and hemoglobin levels (bottom right) in mute swans exposed to sediment from the Coeur d'Alene River basin or the reference area. Birds were provided sediment mixed in either a commercial diet or a rice diet. An asterisk (*) indicates a significant difference ($p < 0.05$) in birds ingesting Coeur d'Alene sediment compared to birds on the same diet ingesting reference sediment.
Source: Day et al., 1998.

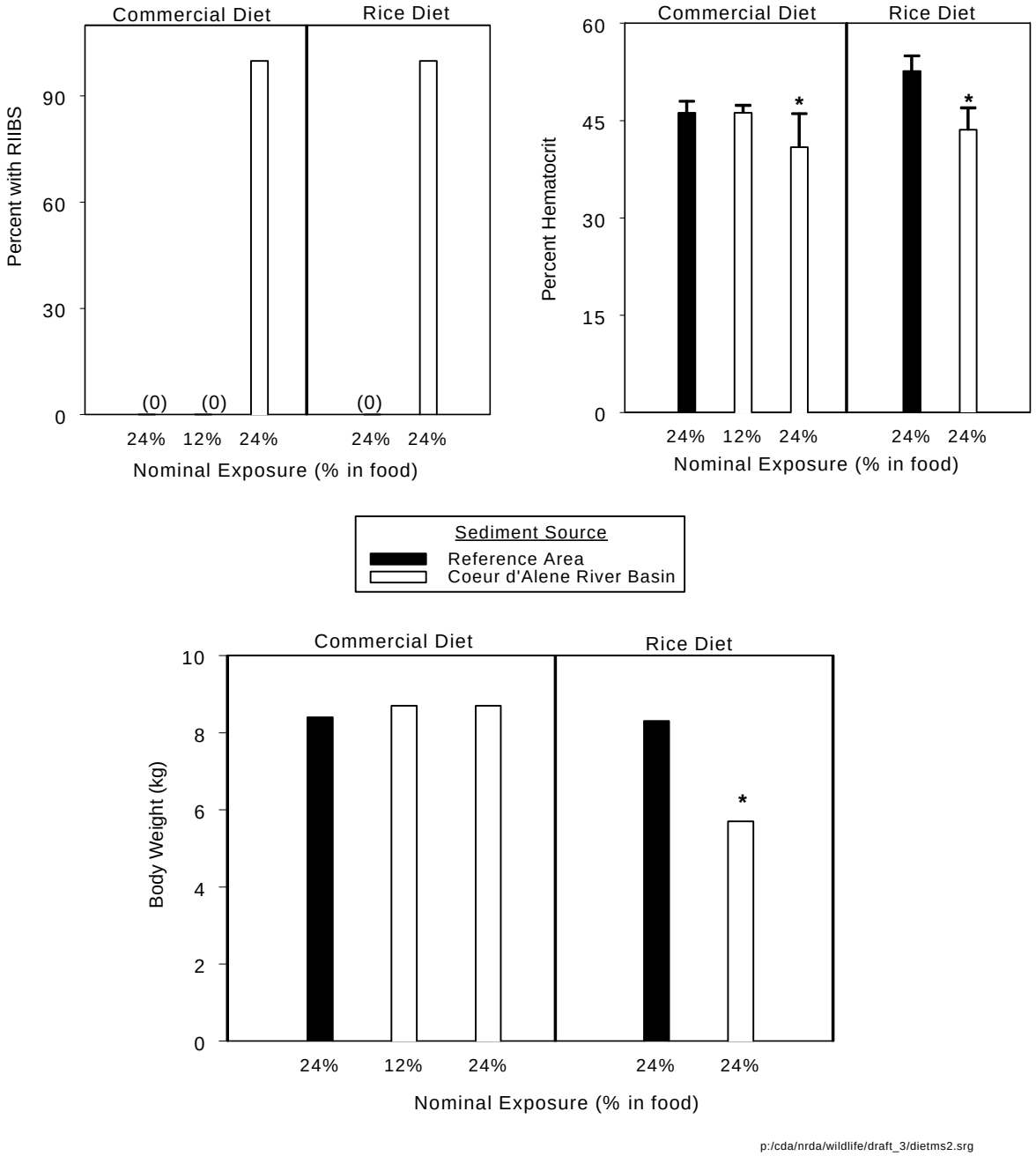


Figure 6-29. Renal intranuclear inclusion bodies (RIIBs; top left), blood hematocrit (top right), and body weight (bottom left) in mute swans exposed to sediment from the Coeur d'Alene River basin or the reference area. Birds were provided sediment mixed in either a commercial diet or a rice diet. An asterisk (*) indicates a significant difference ($p < 0.05$) in birds ingesting Coeur d'Alene sediment compared to birds on the same diet ingesting reference sediment.
Source: Day et al., 1998.

6.6 INJURY DETERMINATION EVALUATION

The results of the Trustees' studies and other existing data from the Coeur d'Alene River basin demonstrate the following:

- ***Wildlife resources in the Coeur d'Alene are exposed to elevated concentrations of lead.*** Types of biota confirmed to contain elevated concentrations of lead include:

  - aquatic vegetation, including waterfowl forage such as water potatoes
  - aquatic biota, including invertebrates, amphibians, and fish
  - small mammals, including meadow voles and deer mice
  - larger mammals, including muskrat, beaver, mink, and deer
  - birds of prey, including bald eagles, osprey, kestrel, and prey items
  - floodplain songbirds, including song sparrows and American robins
  - waterfowl, including wood ducks, Canada geese, mallards, and tundra swans.

  Exposure was confirmed by the extremely high concentrations of lead in Coeur d'Alene River basin sediments (e.g., 500 to 20,000 ppm), sediment ingestion by wildlife, bioaccumulation of lead in the blood and tissues of multiple species of wildlife, and documentation of biological responses in multiple species of Coeur d'Alene River basin wildlife that are characteristic of lead exposure.

- ***Wildlife exposure occurs as a result of ingestion of contaminated sediments and from consumption of lead-contaminated food items.*** Lead exposure in migratory birds has been found to increase with increasing sediment ingestion rates and increasing sediment contamination with lead.

- ***Multiple adverse effects caused by lead exposure have been observed in Coeur d'Alene wildlife in the field.*** The biological responses observed in Coeur d'Alene wildlife include:

  - death, in numerous species of migratory birds

  - physiological malfunctions, including changes in parameters related to impaired blood formation in migratory birds

  - physical deformations, including gross and histopathological lesions in multiple tissues of migratory birds

  - lead and cadmium concentrations in tissues of multiple species of mammal that exceed concentrations associated with clinical signs of metal poisoning

  - reduced survival, reduced growth, delayed development, and behavioral abnormalities of amphibians.

- ***Controlled laboratory studies have confirmed that the lead contained in Coeur d'Alene River basin sediments is bioavailable and causes the adverse effects observed in the field.*** The number and severity of adverse effects was found to increase with increasing lead exposure.

6.6.1 Pathway Determination

The purpose of pathway determination is to identify the route or media by which hazardous substances have been transported from sources to the wildlife resources of the Coeur d'Alene River basin [43 CFR § 11.63(dd)].

Pathways were determined by demonstrating that sufficient concentrations exist in pathway resources now, and have existed in the past, to carry hazardous substances to Coeur d'Alene River basin wildlife and their supporting habitats [43 CFR § 11.63 (a) (2)]. The critical pathways for wildlife exposure in the Coeur d'Alene River basin are sediment and dietary (food chain) pathways.

Sediment Pathway

The sediment exposure pathway involves exposure to hazardous substances through ingestion of contaminated sediment, followed by absorption in the gastrointestinal tract during digestion of food items [43 CFR § 11.63(b) and (e)]. Sediment was found to be the principal pathway of lead exposure to migratory birds in the Coeur d'Alene, as evidenced by the following:

- ***Sediments are contaminated with lead.*** The sediments in the floodplains, beds, and banks of the lower Coeur d'Alene River basin contain extremely elevated lead concentrations. For example, lead concentrations in surface sediments in wetlands of the lower Coeur d'Alene area range as high as 19,900 ppm (i.e., nearly 2% of sediment by weight is lead) (Campbell et al., 1999a). Additional information documenting the extent of sediment contamination is presented in Chapter 5, Sediment Resources, and Chapter 10, Injury Quantification.

- ***Wildlife ingest sediment.*** Sediment ingestion can be substantial for many wildlife species (Beyer et al., 1994). Many migratory birds species ingest sediment while feeding on roots, tubers, and submergent and emergent vegetation. In addition, birds may deliberately ingest sediment to aid digestion.

  On average, Canada geese and tundra swans ingest an estimated 9% sediment in diet, and an estimated 10% of tundra swans ingest more than 22% sediment in diet (Beyer et al., 1998b). Moreover, the contaminated sediments of the Coeur d'Alene River basin serve as an important pathway of hazardous substances exposure even in surface-feeding waterfowl such as wood ducks that have low rates of sediment ingestion (Beyer et al.,

1997). Consistent with observations of the sediment pathway in the Coeur d'Alene River basin, Nelson et al. (1998) reported that lead exposure in filter-feeding waterbirds in Lake Nakuru, Kenya, occurred predominately through ingestion of lead-contaminated suspended solids.

- ***Sediment lead is bioavailable.*** Controlled laboratory experiments with mallards (ducklings and subadults), Canada geese (goslings), and mute swans (juveniles) have demonstrated that the lead in the sediment from the lower Coeur d'Alene River basin is bioavailable to migratory birds (Day et al., 1998; Hoffman et al., 1998; Heinz et al., 1999). Lead residues in the blood, liver, and kidney tissues of these species increased with increasing sediment exposure. Biological responses sensitive to and diagnostic of lead exposure also increased with increasing sediment exposure.

Food Chain Pathway

The food chain pathway [43 CFR § 11.63(f)] involves contact with hazardous substances through consumption of contaminated food. Hazardous substances in sediments are accumulated in plants, invertebrates, fish, mammals, and birds, which are consumed by other species of birds and mammals in the Coeur d'Alene River basin. Food chain exposure is an important pathway for lead and other metals in the Coeur d'Alene River basin, as evidenced by the following:

- ***Sediment lead contaminates vegetation.*** Lead contamination of vegetation in the Coeur d'Alene River basin is caused by sediments adhering to the surface of plants (Campbell et al., 1999b). Waterfowl are exposed to high lead concentrations when feeding on vegetation that holds contaminated sediment on leaf surfaces or when they consume vegetative parts that are partially buried in the sediment (Beyer et al., 1998b). Waterfowl may also ingest some lead incorporated in plant tissues, independent of adhering sediment.

- ***Wildlife forage and prey items are contaminated.*** Lead and other metals accumulate in dietary items of fish (aquatic invertebrates) (Woodward et al., 1997; Farag et al., 1998) and dietary items of dabbling and diving ducks (aquatic vegetation) (e.g., Krieger, 1990; Audet, 1997; Farag et al., 1998). Lead and other metals accumulate in dietary items of birds of prey and carnivorous mammals, including small mammals, fish, and avian species. Concentrations of lead in prey items are substantially elevated in the Coeur d'Alene River basin compared to concentrations in reference area prey items. For example, lead concentrations in meadow voles and brown bullheads were 38 and 85 times higher, respectively, in the Coeur d'Alene River basin than in the St. Joe River basin (Audet, 1997).

- ***Wildlife tissues are contaminated.*** Lead and other metals have bioaccumulated in the wildlife of the Coeur d'Alene River basin, including multiple species of waterfowl (without the presence of lead artifacts), bald eagles, mammals, species of cultural

significance (cutthroat trout, beaver, muskrat, and deer), and songbirds (robins). In contrast, lead levels in tissues of wildlife (without the presence of lead artifacts) from reference areas are generally low. Many of the wildlife species with elevated tissue concentrations are species that do not ingest lead shot. Songbirds, for example, feed on organisms that live in sediment and floodplain soils, and muskrats and beavers feed on vegetation.

6.6.2 Injury Determination

Wildlife injuries resulting from exposure to lead that were specifically evaluated included:

- death [43 CFR § 11.62 (f)(4)(i)
- physiological malfunctions [43 CFR § 11.62 (f)(4)(v)]
- physical deformations [43 CFR § 11.62 (f)(4)(vi)].

Other types of injuries, such as behavioral abnormalities [43 CFR § 11.62 (f)(4)(iii)] and disease [43 CFR § 11.62 (f)(4)(ii)], were not evaluated explicitly for wildlife resources but can be caused by lead exposure (Section 6.3). Behavioral abnormalities have been observed in the field in the Coeur d'Alene River basin and in wildlife exposed to Coeur d'Alene River basin sediment and soil in controlled laboratory studies. Douglas-Stroebel (1997) reported altered activity levels in mallards fed Coeur d'Alene sediment, and Lefcort et al. (1998) observed altered predator avoidance and competitive interactions in amphibians exposed to Coeur d'Alene River basin sediment/bank soil. Nevertheless, because of the relatively large amount of available and relevant data, injury determination focused on death, physiological malfunctions, and physical deformations.

Death [43 CFR § 11.62 (f)(4)(i)]

Wildlife in the Coeur d'Alene River basin have died from exposure to lead. Death from lead poisoning has been documented in both field investigations and controlled laboratory studies in which waterfowl were fed diets containing lead-contaminated sediment.

Wildlife kill investigations. The wildlife kill investigations confirmed that the number and frequency of dead and dying birds in the Coeur d'Alene River basin are significantly greater than the number and frequency in the St. Joe River basin [43 CFR § 11.62 (f)(4)(i)(C)]. Of the carcasses collected in the Coeur d'Alene River basin, 71% were diagnosed as lead poisoned without lead artifacts, and 78% of the areas of the Coeur d'Alene River basin that were investigated contained dead or dying waterfowl diagnosed with lead poisoning without the presence of lead artifacts. In comparison, 19% of waterfowl diagnosed as lead poisoned from the St. Joe River basin contained no ingested lead artifacts. The results of wildlife kill investigations demonstrate death injuries to wildlife in the Coeur d'Alene River basin as defined by the DOI regulations.

Laboratory toxicity testing. Laboratory toxicity testing demonstrated that ingestion of lead-contaminated sediments from the Coeur d'Alene River basin causes waterfowl deaths [43 CFR § 11.62 (f)(4)(i)(E)]. A greater number of deaths occurred within treatments groups that ingested Coeur d'Alene River basin sediments than within treatment groups that ingested reference sediment. The laboratory experiments were conducted using standard test methods, and waterfowl were exposed to the same substances to which wild populations are exposed [43 CFR § 11.62 (f)(4)(i)(E)]. The results of laboratory toxicity testing demonstrate death injuries to wildlife in the Coeur d'Alene River basin as defined by the DOI regulations.

Physiological Malfunctions [43 CFR § 11.62 (f)(4)(v)]

Physiological malfunctions in migratory birds caused by lead were documented in field investigations and in controlled laboratory studies in which waterfowl were fed diets containing lead-contaminated sediment. Physiological malfunctions related to lead exposure include ALAD inhibition, other physiological and biochemical changes, and reduced growth.

ALAD inhibition. Injury has occurred when the activity level of whole blood ALAD in a sample from the population of a given species at an assessment area is significantly less than mean values for a population at a control area, and ALAD depression of at least 50% can be measured [43 CFR § 11.62 (f)(4)(v)(C)].

Field studies confirm that ALAD inhibition in birds from the Coeur d'Alene River basin is prevalent, that ALAD activity in birds of many species from the Coeur d'Alene River basin is significantly inhibited relative to reference bird populations, and that for many species, ALAD inhibition relative to reference populations exceeds 50% [43 CFR § 11.62 (f)(4)(v)(D)]. Relative to reference populations, ALAD activity is significantly reduced in Coeur d'Alene wood ducks, tundra swans, Canada geese goslings and adults, mallard juveniles and adults, osprey juveniles and adults, kestrel juveniles and adults, American robins, and song sparrows. Injury studies confirmed that ALAD activity in Coeur d'Alene wood ducks was inhibited by 85 to 96%; in Coeur d'Alene tundra swans, by 93%; in Coeur d'Alene Canada geese goslings and adults, by >50%; in Coeur d'Alene juvenile and adult mallards, by >50%; in Coeur d'Alene American robins and song sparrows, by >50%; and in Coeur d'Alene bald eagle chicks, by 35% to 65%. Previous studies the basin confirmed that in Coeur d'Alene juvenile and adult osprey, ALAD activity was inhibited by >52% (Henny et al., 1991), and in Coeur d'Alene juvenile and adult kestrels, by >55% and > 81%, respectively (Henny et al., 1994). Injury studies confirmed that ALAD activity was inversely correlated with lead concentration in the blood of wood ducks and tundra swans, and ALAD activity in song sparrows was inversely correlated with soil lead concentrations (i.e., increasing inhibition with increasing sediment/soil contamination).

Laboratory injury studies confirmed that ingestion of lead-contaminated sediment causes ALAD inhibition in waterfowl species representative of the Coeur d'Alene River basin waterfowl that exhibited ALAD inhibition in the field. ALAD inhibition greater than 50% was demonstrated for multiple species of waterfowl in controlled laboratory experiments in which test species ingested

sediment collected from the lower Coeur d'Alene River basin. ALAD activity was lower in all subadult mallards that ingested lead-contaminated sediments (3% to 19% sediment ingestion) than in control birds. ALAD inhibition >90% was observed in Canada geese goslings and mallard ducklings at all doses of contaminated sediment (12% to 48% sediment exposure; actual dose not measured). ALAD activity was inhibited by >95% in mute swans at all exposure levels (12% to 24% Coeur d'Alene sediment in feed; both commercial and rice diets).

The laboratory results confirm the field results and explain the cause of the ALAD inhibition. Significant ALAD inhibition observed in both field investigations and controlled laboratory experiments demonstrates injury to wildlife in the Coeur d'Alene as defined by the DOI regulations [43 CFR § 11.62 (f)(4)(v)(D)].

Responses associated with impaired blood formation. Other physiological malfunctions caused by hazardous substances that satisfy the acceptance criteria for biological responses [43 CFR § 11.62(f)(2)(i-iv)], including increases in protoporphyrin and decreases in hemoglobin and hematocrit, were demonstrated in field and laboratory studies.

- ***Protoporphyrin.*** Protoporphyrin concentrations increase in blood following lead exposure because of inhibition of the enzyme ferrochelatase, which is involved in hemoglobin formation. Field investigations results confirmed that protoporphyrin levels in multiple species of Coeur d'Alene River basin migratory birds are significantly elevated relative to levels in birds from reference areas. In controlled laboratory experiments, protoporphyrin was significantly greater in waterfowl fed lead-contaminated sediment than in waterfowl fed reference sediment. Protoporphyrin levels increased in proportion to the percentage of lead-contaminated sediments in the diet.

- ***Hemoglobin.*** This biochemical is the component of blood that carries and transfers oxygen to the cells of animals. Lead exposure decreases hemoglobin levels through the blockage of the biochemical pathway producing heme. In field studies, hemoglobin levels of wood ducks and tundra swans were inversely correlated with blood lead concentrations, indicating that increased lead exposure results in decreased hemoglobin. Hemoglobin was significantly lower in multiple species of Coeur d'Alene waterfowl, including tundra swans and wood ducks, relative to birds in reference areas. In controlled laboratory tests, hemoglobin was significantly reduced in mallards, Canada geese, and mute swans fed Coeur d'Alene River basin sediment.

- ***Hematocrit.*** Hematocrit is an index of the red blood cell content of blood and is measured by determining the packed cell volume (primarily red blood cells) of a blood sample. Lead exposure causes a decrease in hematocrit via inhibition of the early steps of red blood cell formation. In field studies, hematocrit levels of wood ducks and tundra swans were inversely correlated with blood lead concentrations, indicating that increased lead exposure results in decreased hematocrit. Hematocrit was significantly reduced in

waterfowl from the Coeur d'Alene River basin and in waterfowl fed lead-contaminated Coeur d'Alene River basin sediment.

Loss of body weight. Weight loss, a physiological malfunction caused by hazardous substances that satisfies the acceptance criteria for biological responses [43 CFR § 11.62(f)(2)(i-iv)], was demonstrated in controlled laboratory studies. The body weights of mallard ducks, Canada geese, and mute swans fed Coeur d'Alene River basin sediments were 20 to 30% lower than the weights of waterfowl fed diets containing reference area sediment. Loss of body weight occurred at exposure levels similar to those causing hemoglobin and hematocrit reductions.

The ecological significance of changes in blood parameters and weight loss is reduced viability of wildlife caused by impaired blood formation and other physiological malfunctions. The results of field investigations and controlled laboratory experiments demonstrate physiological malfunction injuries to wildlife in the Coeur d'Alene River basin as defined by the DOI regulations [43 CFR § 11.62(f)(4)(v)].

Physical Deformation [43 CFR § 11.62 (f)(4)(vi)]

Physical deformations caused by lead were demonstrated in both field investigations and controlled laboratory studies. Physical deformations caused by lead exposure include internal gross and histological lesions.

Gross lesions. Gross lesions caused by lead exposure include emaciation, atrophy of breast muscles, abnormal bile, bile staining, and impactions of the upper gastrointestinal tract. These lesions were observed in Coeur d'Alene River basin waterfowl and in waterfowl that ingested Coeur d'Alene River basin sediments in controlled laboratory experiments.

Histopathological lesions. Histopathological lesions caused by lead exposure include hepatic and renal hemosiderosis, myocardial necrosis, arterial fibrinoid necrosis, and RIIBs. RIIBs, which are lesions diagnostic of lead exposure, were observed in Coeur d'Alene River basin waterfowl and in waterfowl ingesting Coeur d'Alene sediments in controlled laboratory experiments. Additionally, mallard ducklings ingesting Coeur d'Alene sediment exhibited brain lesions (myelin swelling of the brain and nerve fiber degeneration) (Hoffman et al., 1998). Ducklings ingesting reference area sediment did not exhibit these lesions.

6.6.3 Summary of the Injury Determination Evaluation

- ***Sufficient concentrations of hazardous substances exist in pathway resources to expose wildlife resources.*** The source of hazardous substance exposure to wildlife is releases of lead and other metals from mining and mineral processing activities. Hazardous substances are transported from the South Fork Coeur d'Alene River basin in surface water, soil, and sediment to the lower Coeur d'Alene River basin.

Hazardous substance concentrations in pathway resources are sufficient to expose wildlife via ingestion of contaminated sediment and forage and prey items. Concentrations of cadmium and lead in tissues of wildlife from the Coeur d'Alene River basin greatly exceed concentrations in tissues of wildlife from reference areas. Exposure to lead artifacts is not the principal pathway of lead exposure to waterfowl in the Coeur d'Alene River basin.

- ***Exposure of wildlife species in the Coeur d'Alene River basin to hazardous substances causes injury.*** The results of field investigations and controlled laboratory experiments demonstrate that death, physiological malfunctions, and physical deformation injuries to wildlife of the Coeur d'Alene River basin result from dietary exposure to hazardous substances. Injuries have occurred and continue to occur as a result of exposure to lead and other hazardous substances in Coeur d'Alene River basin sediments, wildlife forage items, and prey items. In addition, sediments, vegetation, and biota are injured, as defined by the DOI regulations [e.g., 43 CFR § 11.62(b)(i)(v)], because they serve as pathways of injury to other aquatic biological resources.

  Birds in the Coeur d'Alene River basin that ingest sediment, forage, or prey contaminated with lead (tundra swans, Canada geese, mallard ducks, wood ducks, northern pintails, American wigeons, redhead ducks, canvasback ducks, osprey, American kestrels, American robins, song sparrows and eagles) and mammals that ingest sediment or prey contaminated with lead (mice, voles, and mink) are injured by exposure to lead. The number of dead and dying swans and geese diagnosed as lead poisoned (without the presence of lead artifacts; normalized for population sizes) was significantly greater in the Coeur d'Alene River basin than in reference areas. Field studies confirm that ALAD inhibition in birds from the Coeur d'Alene River basin is prevalent, that ALAD activity in birds from the Coeur d'Alene River basin is significantly inhibited relative to reference bird populations, and that ALAD inhibition relative to reference populations exceeds 50%. The frequency of gross and histopathological lesions diagnostic of lead poisoning was substantially greater in carcasses collected from the Coeur d'Alene River basin than in carcasses collected from reference areas.

  Controlled laboratory experiments confirmed that birds die after ingesting sediments from the Coeur d'Alene River basin. Laboratory studies confirmed that ingestion of Coeur d'Alene sediment causes ALAD inhibition in waterfowl representative of wild species that exhibit ALAD inhibition in the Coeur d'Alene River basin. The laboratory results confirm the field results and confirm the cause of the ALAD inhibition. Other physiological malfunctions also occur in wildlife ingesting Coeur d'Alene sediment, including weight loss and changes in blood parameters associated with blood formation (e.g., protoporphyrin, hemoglobin, hematocrit). Laboratory feeding studies confirmed that birds fed lead-contaminated sediments developed gross and histopathological lesions characteristic of lead poisoning.

Laboratory studies have determined that there is a dose-response relationship between the magnitude of exposure to Coeur d'Alene River basin sediment and physiological malfunctions such as biochemical changes in migratory birds. The injury assessment studies demonstrated a causal relationship between increasing sediment ingestion by multiple species of waterfowl and 1) elevation of protoporphyrin levels in blood and 2) reduction in hemoglobin and hematocrit levels. Exposure of amphibians to floodplain sediment from the Coeur d'Alene River basin causes death, physiological malfunctions (impaired development and growth), and behavioral abnormalities.

Field and laboratory studies have demonstrated that there is a dose-response relationship between lead in sediments and the injuries described above. Ingestion of lead-contaminated sediments is the most plausible pathway and cause of the injuries to waterfowl in the basin. Deaths and sublethal injuries cannot be explained by other agents, including lead artifacts (e.g., shot or sinkers), disease (e.g., aspergillosis, avian cholera), or other factors (e.g., trauma).

The above conclusions all indicate the presence of multiple and pervasive injuries to the wildlife resources of the Coeur d'Alene River basin caused by hazardous substance releases associated with mining related activities. Contaminated sediments are the source of lead exposure to wildlife and serve as either direct (sediment ingestion) or indirect (food web contamination) exposure pathways. The injuries are caused by lead-contaminated sediment; thus the supporting habitat for wildlife in the basin, which serves as an exposure pathway, is injured.

6.6.4 Consideration of Lead Artifacts as Cause of Lead Poisoning

Outside of the Coeur d'Alene River basin, the principal source of lead exposure to wildlife is lead shot, lead bullet fragments, and lead sinkers (lead artifacts) ingested by or embedded in the tissues of game animals (Anderson and Havera, 1985; Sanderson and Bellrose, 1986; Beyer et al., 1998c; Wayland and Bollinger, 1999). Beyer et al. (1998c) examined data describing over 1,000 dead and dying waterfowl from hunting areas throughout the United States and found that 29% contained at least one lead shot in the gizzard, and 94% of the waterfowl with ingested lead shot were diagnosed as lead poisoned. Lead shot was detected in 23.5% of trumpeter and tundra swans found dead and dying in western Washington between 1986 and 1992 (Lagerquist et al., 1994). Lead shot accounted for about 20% of the known deaths of trumpeter swans in Idaho, Montana, and Wyoming (survey years 1976 to 1987; Blus et al., 1989). These data indicate that over the last 20 to 25 years, 20 to 24% of the waterfowl carcasses examined in the United States contained ingested lead artifacts, and lead poisoning is the major cause of death of dead and dying waterfowl that contain ingested lead shot.

Overall, the incidence of ingestion of lead artifacts by waterfowl in Coeur d'Alene River basin is similar to the incidence of ingestion in other hunting areas in the United States. During the 1984-1985 hunting season, 29% of the duck carcasses collected in the Coeur d'Alene River basin contained ingested lead shot, and during the 1985-1986 hunting season, 25% of the duck carcasses collected in the Coeur d'Alene River basin had ingested lead shot (Shipley, 1985 and Krieger 1986, as cited in Neufeld, 1987). Casteel et al. (1991) reported that in 1987, 23 of 70 (33%) mallard ducks collected from the Coeur d'Alene River basin had ingested lead shot. Use of lead shot for waterfowl hunting in the Coeur d'Alene River basin was prohibited in 1986, and thus the percentage of waterfowl examined that contain lead artifacts may be declining. For example, during each year of the 1990 to 1997 waterfowl hunting seasons, 4 to 14% of the waterfowl gizzards examined from the Coeur d'Alene River Wildlife Management Area contained lead shot (IDFG, 1993, and unpublished reports; Audet et al., 1999c), and 15% of dead waterfowl collected from the Coeur d'Alene and St. Joe river basins in 1987 contained ingested lead shot (Blus et al., 1995).

In contrast to patterns in other areas of the United States, the lead-poisoned waterfowl of the Coeur d'Alene River basin contain a relatively low incidence of ingested lead artifacts. In 1974, 13 dead tundra swans collected in the Coeur d'Alene area were diagnosed as lead poisoned based on liver lead concentrations, but only one of the 13 (8%) contained lead shot (Benson et al., 1976). Only 13% of 32 lead-poisoned swans (1987-1989 collections) examined by Blus et al. (1991) contained lead artifacts, whereas 95% of lead poisoned tundra swans outside of the Coeur d'Alene area contain lead artifacts (Blus et al., 1991). Audet et al. (1999c) reported that only 12.5% of mallards diagnosed with lead poisoning in the Coeur d'Alene River basin contained lead shot. These data indicate that only 8 to 13% of Coeur d'Alene waterfowl diagnosed with lead poisoning contain lead shot. In contrast, 78% of lead poisoned birds collected from the St. Joe River basin between 1992 and 1997 contained lead artifacts (Audet et al., 1999c).

The frequency of lead poisoning of waterfowl is substantially elevated in the Coeur d'Alene River basin (96% of deaths) compared to the frequency of lead poisoning deaths reported for the Pacific flyway (23%) and nationwide (29%; Figure 6-18; Audet et al., 1999c). However, in contrast to most other areas in the United States, the principal pathway of lead exposure to wildlife in the Coeur d'Alene area is not ingestion of lead artifacts. Ingested lead artifacts were observed in only 8.4% of lead poisoned birds from the Coeur d'Alene area between 1992 and 1997 (Audet et al., 1999b). The incidence of lead artifact ingestion in the Coeur d'Alene River basin is similar to or possibly lower than in the Pacific flyway and nationwide, and, therefore, lead artifact ingestion in the Coeur d'Alene River basin does not provide an explanation for the elevated rate of lead poisoning mortality there.

Finally, the controlled laboratory studies documented adverse effects similar to effects observed in the field (death, ALAD inhibition, changes in other blood parameters, presence of lesions). The studies were performed by sieving Coeur d'Alene sediments to remove lead shot or other artifacts. Therefore, the laboratory studies provide additional evidence that injuries to Coeur d'Alene wildlife are not caused by artifacts. Overall, the evidence indicates that injuries are

caused by exposure to hazardous substances released from mining and mineral processing activities.

6.6.5 Causation Evaluation

Injuries to wildlife result from exposure to lead-contaminated sediments, forage, and prey items in the Coeur d'Alene River basin. Injuries resulting from exposure to lead are demonstrated by the following:

- ***Wildlife are exposed to lead.*** The Coeur d'Alene River basin ecosystem is contaminated with lead, and wildlife in it ingest lead in sediment, forage, and prey items. Lead concentrations in these pathway resources are sufficient to expose wildlife to injurious levels of lead. Multiple species of wildlife, wildlife forage, and wildlife prey in the Coeur d'Alene area, including invertebrates, fish, amphibians, songbirds, waterfowl, birds of prey, and small and large mammals, have elevated tissue lead concentrations.

- ***Lead is known to cause the same biological responses observed in Coeur d'Alene wildlife.*** Multiple scientific studies of amphibians, birds, and mammals have shown that lead causes death, increased disease susceptibility, behavioral abnormalities, physiological malfunctions, and physical deformations. These same effects have been observed in field investigations of Coeur d'Alene wildlife and in wildlife exposed to Coeur d'Alene sediment in the laboratory.

- ***Lead exposure exceeds toxicity thresholds.*** Concentrations of lead in the tissues of Coeur d'Alene wildlife are greater than the toxicity thresholds recommended by Pain (1996) for waterfowl, and Ma (1996) for mammals (Table 6-7). Lead residues in both blood and liver tissues of Coeur d'Alene River basin waterfowl exceed both clinical and severe poisoning thresholds (Table 6-7). Clinical poisoning (e.g., physiological malfunctions) thresholds are exceeded in songbirds and mammals. The threshold values in Table 6-7 are consistent with field observations in the Coeur d'Alene River basin, where extensive waterfowl deaths have been observed (severe poisoning), physiological malfunctions are observed in songbirds (clinical poisoning), and ALAD inhibition is observed in eagles (subclinical poisoning).

- ***Lead exposure and effects are spatially consistent.*** Lead exposure, sediment contamination, and biological responses are significantly correlated in multiple species of wildlife (i.e., lead exposure and effects increase in proportion to sediment and soil contamination levels). Species with high sediment ingestion rates (i.e., tundra swans) exhibit the most adverse effects (death). Waterfowl feeding in areas with the highest lead concentrations in sediment have the highest lead exposure.

Table 6-7
Comparison of Tissue-Residue Toxicity Values for Lead with Lead Residues in Coeur d'Alene River Basin Wildlife

Parameter	Blood Lead				Liver Lead (ppm, wet wt.)				
	Waterfowl		Bald Eagles		Waterfowl		Songbirds		Mammals
Toxicity Value	0.5-1 ppm[a]	>1 ppm[a]	1-5 ppm[b]	>5 ppm[b]	6-15 ppm[a]	>15 ppm[a]	5-8 ppm[c]	>8 ppm[d]	>7.5 ppm wet wt.[e]
Effect	Clinical poisoning	Severe poisoning	Clinical poisoning	Severe poisoning	Clinical poisoning	Severe poisoning	Clinical poisoning	Severe poisoning	Clinical poisoning
Exceeded in CdA Wildlife Tissues	✓	✓	No	No	✓	✓	✓	✓	✓
Data Source	1, 2, 3	1, 2, 3	4	4	1, 5	1, 5	5	5	5, 6, 7

Toxicity values: a. Pain, 1996 b. Franson, 1996 c. Friend, 1987 d. Blus et al., 1995 e. Ma, 1996.

Data source: 1. Blus et al., 1999 2. Henny et al., 1999 3. Blus et al., 1997 4. Audet et al., 1999b 5. Audet et al., 1997 6. Kreiger, 1990 7. Blus et al., 1987.

- ***Lead exposure causes injury.*** Controlled laboratory experiments demonstrate that increasing lead exposure results in an increase in biological responses, from biochemical alterations, to physical deformations, to death. Waterfowl ingesting lead contaminated sediment from the Coeur d'Alene River basin exhibit injuries from lead, whereas waterfowl ingesting reference area sediment do not. The laboratory experiments enabled elimination of lead artifacts — as well as other factors such as trauma, predation, etc. — as a possible cause of the lead poisoning.

- ***Evaluation of alternatives.*** Necropsy reports of pathologists from the U.S. Fish and Wildlife Service National Wildlife Health Center have identified lead as the principal cause of death of waterfowl in the Coeur d'Alene River basin. Deaths and sublethal injuries cannot be explained by other agents, including lead artifacts (e.g., shot or sinkers), disease (e.g., aspergillosis, avian cholera), or other factors (e.g., trauma). Lead poisoning was the greatest single cause of sickness or death (80%) of Coeur d'Alene wildlife, and 92% of those lead-poisoned animals had no ingested lead artifacts (e.g., lead shot or fishing sinkers). In contrast, 47% of the carcasses necropsied from the St. Joe River basin reference area were diagnosed as lead poisoned, and 78% of those contained lead artifacts (Audet et al., 1999c).

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