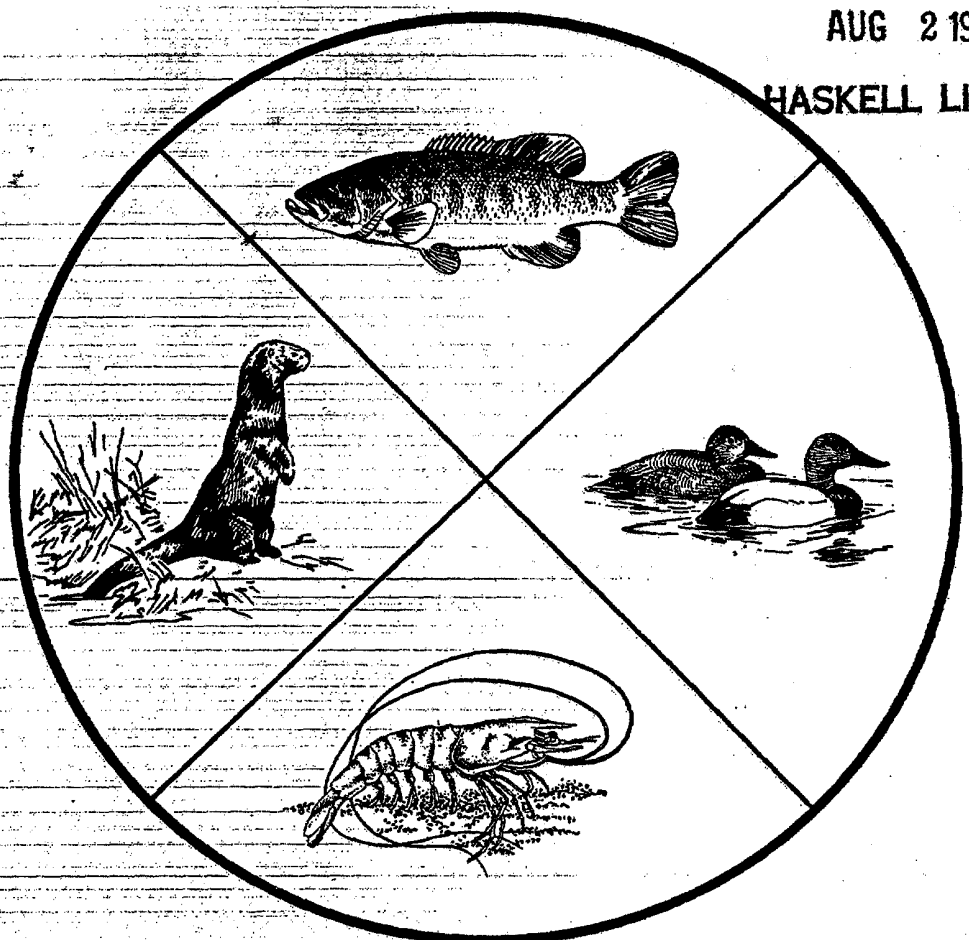
BIOLOGICAL REPORT 85(1.14)
APRIL 1988

CONTAMINANT HAZARD REVIEWS
REPORT NO. 14

LEAD HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW



Fish and Wildlife Service
U.S. Department of the Interior

Other publications in the "Hazards" series[a]

Subject	Publication date	Publication number
Mirex	March 1985	85(1.1)
Cadmium	July 1985	85(1.2)
Carbofuran	August 1985	85(1.3)
Toxaphene	August 1985	85(1.4)
Selenium	October 1985	85(1.5)
Chromium	January 1986	85(1.6)
Polychlorinated Biphenyls	April 1986	85(1.7)
Dioxins	May 1986	85(1.8)
Diazinon	August 1986	85(1.9)
Mercury	April 1987	85(1.10)
Polycyclic Aromatic Hydrocarbons	May 1987	85(1.11)
Arsenic	January 1988	85(1.12)
Chlorpyrifos	March 1988	85(1.13)

[a]Copies of individual reviews, if available, can be obtained by writing the Section of Information Management, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708.

J-7714

Biological Report 85(1.14)
April 1988

Contaminant Hazard Reviews
Report No. 14

LEAD HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

Ronald Eisler

U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, MD 20708

Fish and Wildlife Service
U.S. Department of the Interior

SUMMARY

Lead (Pb) and its compounds have been known to man for about 7,000 years, and Pb poisoning has been recognized for at least 2,500 years. All credible evidence indicates that Pb is neither essential nor beneficial to living organisms, and that all measured effects are adverse--including those on survival, growth, reproduction, development, behavior, learning, and metabolism.

Various living resources are at increased risk from Pb: migratory waterfowl that frequent hunted areas and ingest shot; avian predators that eat game wounded by hunters; domestic livestock near smelters, refineries, and Pb battery recycling plants; captive zoo animals and domestic livestock held in enclosures coated with Pb-based paints; wildlife that forage extensively near heavily traveled roads; aquatic life in proximity to mining activities, areas where Pb arsenate pesticides are used, metal finishing industries, organolead industries, and areas of Pb aerosol fallout; and crops and invertebrates growing or living in Pb-contaminated soils.

Adverse effects on aquatic biota reported at waterborne Pb concentrations of 1.0 to 5.1 ug/l included reduced survival, impaired reproduction, reduced growth, and high bioconcentration from the medium. Among sensitive species of birds, survival was reduced at doses of 50 to 75 mg Pb^{2+}/kg body weight (BW) or 28 mg organolead/kg BW, reproduction was impaired at dietary levels of 50 mg Pb^{2+}/kg, and signs of poisoning were evident at doses as low as 2.8 mg organolead/kg BW. In general, forms of Pb other than shot (or ingestible Pb objects), or routes of administration other than ingestion, are unlikely to cause clinical signs of Pb poisoning in birds. Data for toxic and sublethal effects of Pb on mammalian wildlife are missing. For sensitive species of domestic and laboratory animals, survival was reduced at acute oral Pb doses of 5 mg/kg BW (rat), at chronic oral doses of 5 mg/kg BW (dog), and at dietary levels of 1.7 mg/kg BW (horse). Sublethal effects were documented in monkeys exposed to doses as low as 0.1 mg Pb/kg BW daily (impaired learning at 2 years postadministration) or fed diets containing 0.5 mg Pb/kg (abnormal social behavior). Signs of Pb exposure were recorded in rabbits given 0.005 mg Pb/kg BW and in mice given 0.05 mg Pb/kg BW. Tissue Pb levels were elevated in mice given doses of 0.03 mg Pb/kg BW, and in sheep given 0.05 mg Pb/kg BW. In general, organolead compounds were more toxic than inorganic Pb compounds, food chain biomagnification of Pb was negligible, and younger organisms were most susceptible. More research seems merited on organolead toxicokinetics (including effects on behavior and learning), and on mammalian wildlife sensitivity to Pb and its compounds.

TABLES

ACKNOWLEDGMENTS

I thank Nancy A. Bushby, Lynda J. Garrett, and Joyce E. Haber for technical services; Julia Armstrong and Jean Higgis for secretarial help; Louis N. Locke, Jerry R. Longcore, Keith A. Morehouse, Oliver H. Pattee, Matthew C. Perry, and Christopher J. Schmitt for reviewing the manuscript; and Paul H. Eschmeyer and James R. Zuboy for editorial services.

accumulative metabolic poison that affects behavior, as well as the hematopoietic, vascular, nervous, renal, and reproductive systems. In humans, Pb causes stillbirths, miscarriages, inhibited development of fetuses, decreased male fertility, and abnormal sperm. Severe damage to the central nervous system from exposure to large amounts of Pb may result in stupor, convulsions, coma, and death. Children that survive Pb poisoning are often permanently retarded or have permanent neurological handicaps. At subclinical injury levels, Pb causes slight, but irreversible, damage to the brain development of growing children.

Natural resources are also affected by environmental Pb contamination, and some wildlife species numbers may be reduced as a result. For example, waterfowl deaths resulting from the ingestion of spent Pb shot pellets from shotgun shells were discovered more than 100 years ago in Italy and in the United States; since then Pb poisoning of waterfowl has occurred in 15 countries (Street 1983). In North America alone, approximately 3,000 tons of Pb shot are expended annually into lakes, marshes, and estuaries by several million waterfowl hunters (FWS 1986, 1987). Spent pellets are eaten by waterfowl and other birds, either in mistake for seeds or as pieces of grit. These pellets may be retained in the gizzard for weeks, where they are reduced chemically and mechanically, form soluble toxic salts, and cause characteristic signs of Pb intoxication--especially lethargy and emaciation (Street 1983). At least 2% of all North American waterfowl--or about 2 million ducks and geese (Lumeij 1985)--die each year as a direct result of ingestion of Pb shot (Bellrose 1959). These deaths contribute to the decline of some species, such as the canvasback, Aythya valisineria (Dieter 1979), pintail, Anas acuta (White and Stendell 1977), and black duck, Anas rubripes (Pain and Rattner 1988). Up to 7X more waterfowl died from Pb toxicosis as a result of ingesting spent pellets than from wounding by hunters (Zwank et al. 1985). In addition, Pb-poisoned waterfowl show delayed mortality from Pb-induced starvation, are readily captured by predators, are susceptibile to disease, and reproduce poorly (Dieter 1979). Susceptibility is markedly influenced by species, by the number and size of shot ingested, and by the types of foods eaten (White and Stendell 1977). Swans are among the more vulnerable waterfowl. In England, Pb poisoning through the ingestion of discarded Pb fishing sinkers is the major cause of death in the mute swan, Cygnus olor (Birkhead 1983); for all species of swans in England, about half died as a direct result of Pb poisoning (Demayo et al. 1982). In Washington State, 30% of the endangered trumpeter swans (Cygnus buccinator) found dead had died of Pb poisoning from ingestion of Pb shot (Kendall and Driver 1982). Lead toxicosis caused by ingestion of spent shot and other Pb objects has also been reported for sandhill crane, Grus canadensis (Windingstad et al. 1984); Canada goose, Branta canadensis (Szymczak and Adrian 1978); mourning dove, Zenaidura macroura (Locke and Bagley 1967); and wild turkey, Meleagris gallopavo (Stone and Butkas 1978). Secondary poisoning has been documented in at least five species of raptors that ate food containing Pb shot (especially hunter-wounded animals): Andean condor, Vultur gryphus (Locke et al. 1969); bald eagle, Haliaeetus leucocephalus (Pattee and Hennes 1983); honey buzzard,

In this report, I summarize available data on lead in the environment, with emphasis on fishery and wildlife resources, and review current recommendations for the protection of sensitive species. This account is part of a continuing series of brief reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

Table 1. World Pb production, consumption, and principal end uses (modified from Harrison and Laxen 1981; Demayo et al. 1982).

Production, consumption, and use	Metric tons, in thousands
PRODUCTION, 1978	
Mined Pb	3,625
Refined Pb	4,202
CONSUMPTION	
1977	2,995
1980	3,801
PRINCIPAL END USES OF REFINED Pb	
1977	
Storage batteries	1,478
Pigments and chemicals	369
Tetraalkyllead	292
Cable covering	216
Pipe and sheeting	160
Other	480
1980	
Storage batteries	1,330
Tetraethyllead	380
Cable covering	380
Solder	380
Litharge	190
Building construction	190
Caulking	190
Other	760

atmospheric Pb concentrations increased sharply due to massive increases in Pb emissions from automobiles; since then, increased Pb emissions to the atmosphere have matched trends in gasoline Pb content and consumption (Eisenreich et al. 1986; Smith et al. 1987). In 1957, the United States was overtaken by Australia and the USSR in domestic mine production of Pb; however, in 1967, the opening of the "New Lead Belt" in Missouri revived mining in the United States, and subsequently Pb was produced at the annual rate of 450,000 to 550,000 metric tons (EPA 1979). In 1975, the United States was again the leading Pb producer from mine sources, accounting for 16% of the world total; at that time, about 70% of the world Pb production came from the USA, the USSR, Australia, Canada, Peru, Mexico, China, Yugoslavia, and Bulgaria (Tsuchiya 1979). In 1986, world mine production of lead was 2,352,000 tons of which USA mine production was 353,000 tons, or 15% of the world total, and production in Missouri was 308,000 tons, or 87% of the USA total (personal communication, R. L. Amistadi, Doe Run Company, St. Louis, Missouri).

naturally, and their rate of formation may be affected by man-made organoleads (Nriagu 1978a). In surface waters, Pb exists in three forms: dissolved labile (e.g., Pb^{2+}, $PbOH^{+}$, $PbCO_3$), dissolved bound (e.g., colloids or strong complexes), or as a particulate (Benes et al. 1985). The labile forms represent a significant part of the Pb input from washout of atmospheric deposits, whereas particulate and bound forms were common in urban runoff and ore-mining effluents (Benes et al. 1985). The solubility of Pb compounds in water is pH dependent, and ranges from about 10 g Pb/l at pH 5.5, to less than 1 ug Pb/l at pH 9.0 (EPA 1980); little detectable Pb remains in solution at pH >8.0 (Prause et al. 1985). At pH 6.5 and water alkalinity of 25 mg $CaCO_3$/l, elemental Pb is soluble to 330 ug/l; however, Pb^{2+} under the same conditions is soluble to 1,000 ug/l (Demayo et al. 1982). In acidic waters, the common forms of dissolved Pb are salts of $PbSO_4$ and $PbCl_2$, ionic Pb, cationic forms of lead hydroxide, and (to a lesser extent) the ordinary hydroxide $Pb(OH)_2$. In alkaline waters, common species include the anionic forms of Pb carbonate and hydroxide, and the hydroxide species present in acidic waters (NRCC 1973). Unfortunately, the little direct information available about the speciation of Pb in natural aqueous solutions has seriously limited our understanding of Pb transport and removal mechanisms (Nriagu 1978a).

Most Pb entering natural waters is precipitated to the sediment bed as carbonates or hydroxides (May and McKinney 1981). Lead is readily precipitated by many common anions; desorption and replacement by other cations is extremely slow (Boggess 1977). In some acidic lakes, the deposition of particulate Pb was strongly correlated with the deposition of aluminum and carbon, especially during periods of increasing pH (White and Driscoll 1985). Precipitation of sparingly soluble Pb compounds is not a primary factor controlling the concentration of dissolved Pb in stream waters. Migration and speciation of Pb was strongly affected by water flow rate, increasing flow rate resulting in increased concentrations of particulate and labile Pb and a decrease in bound forms. At low stream flow, Pb was rapidly removed from the water column by sedimentation (Benes et al. 1985).

In the sediments, Pb is mobilized and released when the pH decreases suddenly or ionic composition changes (Demayo et al. 1982). However, there was no significant release of Pb from dredge spoils suspended in estuarine waters of different salinities for 4 weeks (Prause et al. 1985). Some Pb^{2+} in sediments may be transformed to tetraalkyllead compounds, including TML, through chemical and microbial processes. There is also the possibility of methylation of ionic Pb in vivo by fish and other aquatic biota, but the mechanisms are unclear (May and McKinney 1981). Methylation of Pb in sediments was positively related to increasing temperatures, reduced pH, and microbial activity, but seemed to be independent of Pb concentration (Demayo et al. 1982). In general, the concentration of tetraalkylleads in sediments is low, representing less than 10% of total Pb (Chau et al. 1980).

amino acid transport, uncouple oxidative phosphorylation, and inhibit cerebral glucose metabolism (Hong et al. 1983).

Biochemically, Pb exerts deleterious effects on hematopoiesis through derangement of hemoglobin synthesis, resulting in a shortened life span of circulating erythrocytes, often resulting in anemia. Two essential enzymes in heme formation that are extremely sensitive to Pb are delta aminolevulinic acid dehydratase (ALAD), which catalyzes the dehydration of delta amino levulinic acid (ALA) to form porphobilinogen (PBG), and ferrochelatase (= heme synthetase), which catalyzes the insertion of Fe^{2+} into protoporphyrin IX (PP). This second reaction requires the presence of glutathione and ascorbic acid. Some of the intermediates in heme follow sequentially: ALA, PBG, uroporphyrinogen III, coproporphyrinogen III, protoporphyrinogen IX, and PP. It is now well established that ALAD and ferrochelatase are the most sensitive biochemical indicators of Pb exposure, the net result being lowered ALAD activity and elevated PP activity (Barth et al. 1973; Nriagu 1978b; EPA 1979, 1980; Tsuchiya 1979; Harrison and Laxen 1981; Hoffman et al. 1981; De Michele 1984; Schmitt et al. 1984; Lumeij 1985).

Inhibition of blood ALAD activity after exposure to Pb has been documented in many species of freshwater and marine teleosts (Hodson 1976; Hodson et al. 1977, 1980; Johansson-Sjobeck and Larsson 1979; Krajnovic-Ozretic and Ozretic 1980; Demayo et al. 1982; Schmitt et al. 1984; Haux et al. 1986), in the freshwater cladoceran, Daphnia magna (Berglind et al. 1985), in ducks, quail, doves, swallows, raptors, and songbirds (Finley et al. 1976; Dieter and Finley 1978; Dieter 1979; Hoffman et al. 1981; Franson and Custer 1982; Kendall et al. 1982; Kendall and Scanlon 1982; Eastin et al. 1983; Franson et al. 1983; Hoffman et al. 1985a, 1985b; Beyer et al. 1988), and in humans, sheep, mice, rats, rabbits, and calves (Barth et al. 1973; Boggess 1977; Nriagu 1978b; Tsuchiya 1979; Hejtmancik et al. 1982; Hayashi 1983; Peter and Strunc 1983; Schlick et al. 1983; Gietzen and Wooley 1984; Zmudzki et al. 1984). Lead-induced ALAD inhibition has been recorded not only in blood, but also in brain, spleen, liver, kidney, and bone marrow (Johansson-Sjobeck and Larsson 1979; Hoffman et al. 1981, 1985a, 1985b; Schlick et al. 1983; Friend 1985). Time for ALAD recovery to normal levels is dose dependent, organ specific, and usually directly correlated with blood Pb concentrations (Finley et al. 1976; Hodson et al. 1977; Dieter 1979; Hayashi 1983; Friend 1985). ALAD activity levels in Pb-stressed teleosts were normal 3 to 11.7 weeks postadministration (Hodson et al. 1977; Johansson-Sjobeck and Larsson 1979; Krajnovic-Ozretic and Ozretic 1980; Demayo et al. 1982); this range was 2 to 14 weeks in birds (Dieter and Finley 1978; Kendall et al. 1982; Kendall and Scanlon 1982; Friend 1985), and 3 to 12 weeks in mammals (Barth et al. 1973; Schlick et al. 1983). The physiological significance of depressed blood ALAD activity levels, except perhaps as an early indicator of Pb exposure, is debatable. Aside from a few instances of moderate anemia in workers at lead smelters, other abnormalities noted were not regarded as serious (Barth et al. 1973). Lead-induced depression in ALAD activity in mallard (Anas platyrhynchos) ducklings and ring-necked pheasant (Phasianus

number of difficulties for the usual multicompartmental loss-rate models. Some Pb in bones of high medullary content, such as the femur and sternum, have relatively long retention times--i.e., Tb 1/2 of >20 years in humans--whereas Pb stored in bones of low medullary content have Tb 1/2 values of 20 to 200 days, similar to the values for Pb in soft tissues and blood (Tsuchiya 1979; Marcus 1985). In birds, medullary bone undergoes sequences of bone formation and destruction associated with the storage and liberation of calcium during eggshell formation, indicating that sex and physiological condition primarily influence Pb kinetics in avian bone (Finley and Dieter 1978). Marcus (1985) endorsed the use of diffusion models based on the exchange of Pb between blood in canaliculi and the crystalline bone of the osteon to account for retention and bioavailability. More research is needed on the role of bone in Pb kinetics.

Lead damages nerve cells and ganglia, and alters cell structure and enzyme function. Axonal degenerative changes, especially in neuronal cell bodies, were recorded in Pb-poisoned freshwater snails (Viviparus ater), leading to altered protein synthesis (Fantin et al. 1985). Mallards dosed orally with Pb shot developed demyelinating lesions in vagal, branchial, and sciatic nerves, and showed vascular damage in the cerebellum; lesions were similar to those in Pb-intoxicated guinea pigs (Cavia sp.), rats, and guinea hens, Gallus sp. (Hunter and Wobeser 1980). Crop stasis in birds, which is characterized by paralysis of the alimentary tract, impaction of food in the gizzard and proventriculus, and regurgitation of crop fluid, has been produced by Pb shot or Pb acetate solutions. Lead induces crop dysfunction by acting either directly on the smooth muscle or on associated nerve plexuses of crop tissue, depending on the route of administration (Clemens et al. 1975; Boyer et al. 1985; Boyer and Di Stefano 1985). Mammals, including humans, undergo similar alimentary distress following intakes of lead (Boyer et al. 1985).

Effects of Pb on the nervous system are both structural and functional, involving the cerebellum, spinal cord, and motor and sensory nerves; the result may be deterioration of intellectual, sensory, neuromuscular, and psychological functions (Nraigu 1978b). The pathogenesis of Pb-induced injury to the nervous system is poorly understood, but may be mediated through vascular damage, the direct action of Pb on neurons, or alterations in porphyrin metabolism (Hunter and Wobeser 1980). Retarded brain growth in prenatal guinea pigs has been recorded at subclinical levels of Pb (i.e., at concentrations producing no elevation in blood Pb and no change in body weight), and this effect is potentiated at temperatures of 42 °C (Edwards and Beatson 1984). Lead may cause a transient disturbance in the blood-brain barrier during early postnatal growth of rats. This effect is associated with the presence of hemorrhagic lesions, suggesting focal damage to the vessels as an important event in the pathogenesis of Pb encephalopathy to suckling rats (Sundstrom et al. 1985). Brain histopathology has been recorded in Pb-poisoned chickens (Narbaitz et al. 1985) and cattle (Osweiler and Van Gelder 1978). Brain Pb concentrations are usually among the lowest in body organs, but the brain is one of the main sites of action. During chronic Pb

BACKGROUND CONCENTRATIONS

GENERAL

Lead concentrations were usually highest in ecosystems nearest Pb mining, smelting, and refining activities; Pb storage battery recycling plants; areas of high vehicular traffic; urban and industrialized areas; sewage and spoil disposal areas; dredging sites; and areas of heavy hunting pressure. In general, Pb does not biomagnify in food chains. Older organisms usually contain the greatest body burdens, and Pb accumulations are greatest in bony tissues. It seems that resources that are now at high risk (i.e., increased mortality, reduced growth, or impaired reproduction) from Pb include the following: migratory waterfowl that congregate at heavily-hunted areas; raptors that eat hunter-wounded game; domestic livestock near smelters, refineries, and recycling plants; wildlife that forage extensively near heavily traveled roads; aquatic life in proximity to mining activities, Pb arsenate pesticides, metal finishing industries, lead alkyl production, and Pb aerosol fallout; and crops and invertebrates growing or living in Pb-contaminated soils. Data on background concentrations in nonbiological and living resources are cited extensively in Bernhard and Zattera (1975), Nriagu (1978a,b), Wong et al. (1978), Branica and Konrad (1980), Jenkins (1980), Eisler (1981), Harrison and Laxen (1981), and Demayo et al. (1982).

NONBIOLOGICAL SAMPLES

Average Pb concentrations in nonbiological materials worldwide were much higher in sediments (47,000 ug/kg), soils (16,000), and sediment interstitial waters (36) than in atmospheric and other hydrospheric compartments (Table 3). Most of the lead discharged into surface waters is rapidly incorporated into suspended and bottom sediments, and most will ultimately be found in marine sediments (Harrison and Laxen 1981). Sediments now constitute the largest global reservoir of Pb; sediment interstitial waters and soils constitute secondary reservoirs (Table 3).

Lead concentrations were elevated in certain nonbiological materials as a result of nonhunting human activities and natural processes (Table 4). In sediments, Pb concentrations ranged from 3 mg/kg in carbonate marls off the Florida coast to more than 11,000 mg/kg at Sorfjord, Norway, the site of massive discharges of Pb-containing industrial and domestic wastes (Nriagu 1978a). Lead contaminates sediments from sources as diverse as steelworks, shipyards, crude oil refineries, cement and ceramic factories, Pb storage

Table 4. Lead concentrations in selected nonbiological materials.

Material (units)	Concentration[a]	Reference[b]
AIR (ug/m^3)		
Nonurban areas	0.1	EPA 1980
Urban areas	(0.3 - 2.5)	NRCC 1973
Metropolitan areas	(2 - 10)	EPA 1980
Rural roads	6	NRCC 1973
Heavy traffic	40	
Near industrial sources	May exceed 1,000	EPA 1980
RAIN (ug/l)		
Minnesota		
1979		
Rural	6	Eisenreich
Urban	29	et al. 1986
1983		
Rural	2	
Urban	4	
ATMOSPHERIC DEPOSITION (g/ha)		
New Jersey Pine Barrens		
1978-1979	350	Turner et al. 1985
1980-1982	140	
ICE (ug/l)		
Greenland		
800 BC	0.001	NRCC 1973
1750	0.01	
1940	0.07	
1973	>0.2	
SOILS (mg/kg dry weight)		
Near Pb smelter		
Missouri	128	Burrows 1981
British Columbia	>1,000	

Table 4. (Continued)

Material (units)	Concentration[a]	Reference[b]
SOLIDS ENTERING SURFACE WATERS (mg/kg dry weight)		
Street dust		
Urban	(1,000-4,000)	Harrison and Laxen 1981
Rural	440	
Highway runoff		
Suspended sediments	(3,100-5,800)	
Settleable solids	16,000	
Sewage sludge	(100-1,400)	
Suspended sediments in mineralized areas	(1,000-8,000)	
INTEGRATED STUDY (ug/kg)		
Tennessee stream		
Water	(0.01-0.019)	Demayo et al. 1982
Dissolved solids	(30-84)	
Coarse particles	(124-653)	
Colloidal particles	(62-2,820)	
SEDIMENTS (mg/kg dry weight)		
Egypt, Nile River		
Industrialized area	Max. 1,800	Fayed and Abd-El-Shafy 1985
Greece		
Near major industries	(500-600)	Scoullos 1986
Several km distant	40	
Preindustrial levels	10	
Norway		
Sorfjord	Max. 11,000	Nriagu 1978a
Sweden		
Polluted lake	(2,000-2,500)	Haux et al. 1986
Reference lake	110	
USA		
Chesapeake Bay, 1979-1981	(1-134)	Di Giulio and Scanlon 1985
Upper Mississippi River Southeastern Missouri, Big River, 1979-1981	13 (0.4-86)	Wiener et al. 1984

battery recycling plants, and heavy traffic (Scoullos 1986). Mining activities are also important. High concentrations of Pb were measured in sediments (up to 2,200 mg/kg) and detritus (up to 7,000 mg/kg) of the Big River in southeastern Missouri (Czarneski 1985). The Big River drains what was once the largest Pb-mining district in the world; commercial mining was extensive between the early 1700's and 1972. During this period more than 200 metric tons of tailings accumulated within the Big River watershed as a result of seepage from tailings ponds, from erosion of tailings piles on the banks, and through accidental discharges (Niethammer et al. 1985).

In soils, Pb concentrates in organic-rich surface horizons (NRCC 1973). In one instance, only 17 mg of soluble Pb/kg was found in soils 3 days after the addition of 2,784 mg of Pb (as lead nitrate)/kg (NRCC 1973). The estimated residence time of Pb in soils is about 20 years; complete turnover in topsoil is expected every few decades (Nriagu 1978a). In forest litter, however, the mean residence time of Pb is lengthy; estimates range from 220 years (Turner et al. 1985) to more than 500 years (Friedland and Johnson 1985).

Lead deposited on roadways is removed in drainage water, and later accumulated in roadside soils (Harrison et al. 1985). Amounts of Pb in roadside soils are increased as a direct result of the combustion of gasoline containing organolead additives. In general, the amounts of Pb were greatest along roads with the highest density of vehicular traffic, and amounts decreased rapidly with increasing distance from the roadway (Harrison and Dyer 1974; Boggess 1977; Chmiel and Harrison 1981; Way and Schroder 1982; Table 4). Elevated levels of Pb in soils also were recorded from the vicinity of storage battery reclamation plants, smelting activities, and mining and milling operations (Boggess 1977; Burrows 1981; Kisseberth et al. 1984). Fly ash from coal burned in homes or privately hauled from power plants, which contains 100 to 450 mg Pb/kg and is frequently used to reclaim land for the growth of forage and pasture crops and as an alkaline amendment in the reclamation of strip mined areas (Nriagu 1978a), is considered another source of soil Pb. Two additional sources of Pb in soils are municipal sewage sludge and lead-arsenate pesticides (Nriagu 1978a). Sewage sludge, which contains up to 100 mg Pb/kg and is applied as a fertilizer and soil conditioner at the rate of 50 million tons annually, may increase top soil levels by as much as 25 mg Pb/kg. Lead arsenate, a pesticide used to reduce bird hazards near airport runways by controlling earthworm abundance, and also to control pests in fruit orchards, represents another local source of lead contamination to soils.

Lead reaches the aquatic environment through industrial and municipal discharges, in atmospheric deposition, from weathering processes in areas of natural Pb mineralization, and in highway runoff (EPA 1980; Harrison and Laxen 1981; Birdsall et al. 1986). Industrial Pb input to aquatic environments is estimated at 10X that introduced by natural weathering processes (Scoullos 1986); sewage and aerosols are major sources (Harrison and Laxen 1981).

Table 5. Lead concentrations in field collections of selected species of flora and fauna. Values shown are in mg Pb/kg (ppm) fresh weight (FW), or dry weight (DW).

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
FUNGI, MOSSES, AND LICHENS		
Fungi, 4 species		
Near metal smelter	4 DW	Beyer et al. 1985
Control site	2 DW	
Moss, Brachythecium rivulare		
Near lead mines	(1,330 - 8,206) DW	McLean and Jones 1975
Moss, Hypnum cupressiforme		
Sweden, museum specimens		
Year of collection		
1860	(18 - 27) DW	Ruhling and
1880	(20 - 37) DW	Tyler 1968
1900	(40 - 70) DW	
1920	(22 - 90) DW	
1940	(15 - 70) DW	
1960	(65 - 75) DW	
1968	(70 - 90) DW	
Vicinity urban industry	Max. 11,611 DW	Goodman and Roberts 1971
Lichen, Parmelia baltimorensis		
Washington, DC		
1938	106 DW	Jenkins 1980
1958	270 DW	
1970	(950 - 1,371) DW	
Connecticut, 1971	198 DW	
ALGAE AND MACROPHYTES		
Acorns and berries, 4 species		
Near metal smelter	4 DW	Beyer et al. 1985
Control site	3 DW	
Aquatic macrophytes, whole		
Nile River, Egypt		
Industrialized area	Max. 22 DW	Fayed and Abd-El-Shafy 1985
Aquatic plants, 7 species		
From lead shot seeded area		
Roots	19 DW	Behan et al. 1979
Shoots	3 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Foliage, 8 species		
Near metal smelter	21 DW	Beyer et al. 1985
Control site	10 DW	
Alga, Fucus distichus		
Distance from Pb deposit		
1 km	1 DW	Bohn 1979
2 km	0.6 DW	
Alga, Fucus vesiculosus		
Whole, Raritan Bay, New Jersey		
Water Pb content		
0.002 mg/l	8 DW	Seeliger and
0.01 mg/l	38 DW	Edwards 1977
Lettuce, Lactuca sativa		
Pb-contaminated areas	71 FW	Demayo et al. 1982
Uncontaminated areas	0.5 FW	
Mule deer forage, Colorado,		
Roadside		
1978	59 DW	Harrison and
1979	42 DW	Dyer 1984
Rice, Oryza sativa		
Grown 10 m from highway		
Grain	0.2 DW	Ter Haar 1970
Straw	5.8 DW	
Grown 230 m from highway		
Grain	0.2 DW	
Straw	2.1 DW	
Spruce, Picea abies, Germany, 1984		
Declining spruce forest		
Litter	416 DW	Backhaus and
Needles	13 DW	Backhaus 1986
Nondeclining stand		
Litter	213 DW	
Needles	2 DW	
Shortleaf pine, Pinus echinata		
Missouri, leaf		
Distance from smelter, km		
0.8	3,546 (420-11,750) DW	Bolter et al. 1973
0.8 - 1.6	497 (101-1,475) DW	
1.6 - 2.4	274 (52 -1,050) DW	
2.4 - 3.2	142 (62 - 412) DW	
3.2	123 (22 - 661) DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Foliage	4 DW	
Wood	0.5 DW	
Near roadway, UK 1979		
Grass	63 DW	Chmiel and
Grass seeds	99 DW	Harrison 1981
Hawthorn, Crataegus spp.		
Leaves	146 DW	
Fruit	4 DW	
Control site, UK 1979		
Grass	2 DW	
Grass seeds	4 DW	
Hawthorn		
Leaves	4 DW	
Fruit	2 DW	
Grass		
Near factory	(830 - 1,840) DW	Edwards and
1,000 m distant		Clay 1977
Growing	(120 - 1,200) DW	
Dead and litter	(370 - 1,570) DW	
1,700 m distant		
Growing	(240 - 420) DW	
Dead and litter	(170 - 1,970) DW	
Near Pb smelter, forage		
Missouri	979 FW	Burrows 1981
British Columbia	(100 - 200) FW	
Kansas, vegetation		
Near highway	11 DW	Robel et al. 1981
Distant site	3 DW	
INVERTEBRATES		
Limpet, Acmaea digitalis		
California		
Near bridges		
Soft parts	931 DW	Graham 1972
Shell	108 DW	
Pb-free area		
Soft parts	8 DW	
Shell	9 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Insects, various species		
Distance from highway		
0-7 meters		
Sucking	16 DW	Anderson 1977
Chewing	27 DW	
Predatory	31 DW	
13 - 20 meters		
Sucking	9 DW	
Chewing	10 DW	
Predatory	20 DW	
>20 meters		
Sucking	5 DW	
Chewing	5 DW	
Predatory	6 DW	
Kansas, 1978		
Near roadway	50 DW	Udevitz et al. 1980
Control site	15 DW	
Lepidopteran larvae, UK, 1979		
Near roadway	118 DW	Chmiel and
Control site	<1 DW	Harrison 1981
Earthworm, Lumbricus terrestris		
Whole, Maryland		
Distance from highway, meters		
3.0	269 DW	Gish and
6.1	113 DW	Christensen 1973
12.2	80 DW	
24.4	43 DW	
48.8	52 DW	
Eastern tent caterpillar, Malacosoma americanum		
Whole, 1978		
Near roadway	7 DW	Beyer and Moore 1980
>10 m distant	<5.3 DW	
Millipedes, Diplopoda		
UK, 1979		
Near roadway	162 DW	Chmiel and
Control site	34 DW	Harrison 1981

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Near roadway	141 DW	Chmiel and
Control site	27 DW	Harrison 1981
Spiders, Aranea, UK, 1979		
Near roadway	560 DW	
Control site	<1 DW	
Tubificid worms		
Rural streams	16 DW	Boggess 1977
Urban streams	367 DW	
Woodlice, Isopoda		
UK, 1979		
Near roadway	152 DW	Chmiel and
Control site	19 DW	Harrison 1981
USA		
Near highway	(380 - 682) DW	Beyer and Moore 1980
FISH		
Spotted wolffish, *Anarhichas minor*		
Near Pb mine, Greenland		
Liver	Max. 1.8 FW	Bollingberg and
Muscle	Max. 0.12 FW	Johansen 1979
Coastal marine fishes, USA		
Liver		
5 species	(<0.1 - 0.2) FW	Hall et al. 1978
20 species	(0.2 - 0.4) FW	
33 species	(0.4 - 0.6) FW	
13 species	(0.6 - 0.8) FW	
6 species	(0.8 - 1) FW	
5 species	(1 - 3) FW	
Muscle		
5 species	(0.1 - 0.3) FW	
92 species	(0.3 - 0.5) FW	
51 species	(0.5 - 0.7) FW	
7 species	(0.7 - 1) FW	
4 species	(1 - 3) FW	
Whitefish, *Coregonus* spp., Sweden		
Liver		
Polluted lake	(6 - 7) DW	Haux et al. 1986
Reference lake	<1 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Age 2+	14 DW	
Age 3+	16 DW	
Age 4+	18 DW	
Age 5+	19 DW	
INTEGRATED STUDIES		
Great Lakes, Lake Ontario		
Plankton	4 DW	Demayo et al. 1982
Zooplankton	(1 - 5) DW	
Fish	(0.1 - 0.13) FW	
Marine food chain, Central Pacific		
Seawater	0.006 FW	Flegal 1985
Phytoplankton	0.05 FW	
Zooplankton	0.04 FW	
Carnivores, muscle		
Intermediate (anchovy)	0.02 FW	
Top (tuna)	0.0003 FW	
Oklahoma pond		
Water	0.013 FW	Demayo et al. 1982
Sediments		
Surface	529 DW	
12 cm depth	206 DW	
Plankton	281 DW	
Benthos	37 DW	
Mosquitofish, Gambusia sp.	11 DW	
AMPHIBIANS AND REPTILES		
Amphibians, whole		
Near metal smelter	No species found	Beyer et al. 1985
Control site, 5 species	12 DW	
Frog, Rana sp., tadpole, whole		
Missouri, tailings pond	4,139 DW	Gale et al. 1976
Distance downstream from tailings pond		
1 km	552 DW	
25 km	37 DW	
Southeastern Missouri, 1981-1982, Big River		
Bullfrog, Rana catesbeiana, carcass		

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Blood		
Chesapeake Bay, 1974		
Normal	(0.059 - 0.064) FW	Dieter et al. 1976
Abnormal (17%)	0.263 FW	
Wingbone		
La Crosse, Wisconsin		
1976		
Males	18 (6-56) DW	Fleming 1981
Females	5 (1-20) DW	
Immatures		
Males	0.8 (0.1 - 4) DW	
Females	1 (0-21) DW	
1977		
Males	11 (9 - 12) DW	
Females	8 (1 - 48) DW	
Immatures		
Males	0.8 (<0.1 - 7) DW	
Females	<0.5 DW	
Keokuk, Iowa		
1976		
Males	6 (4 - 10) DW	
Females	5 (1 - 20) DW	
Immatures		
Males	0.5 (0.1 - 2) DW	
Females	1 (0.1 - 22) DW	
1977		
Males	2 (0.2 - 19) DW	
Females	4 (1 - 19) DW	
Birds		
Galveston Bay, Texas, 1980-1981, 3 species, liver	(0.1 - 0.5) FW	King and Cromartie 1986
Texas		
Probers with Pb shot in gizzards		
Bone	11 FW	Hall and Fisher 1985
Feather	4 FW	
Liver	0.3 FW	
Probers without Pb shot in gizzards		
Bone	6 FW	
Feather	5 FW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Juveniles	0.11(0.07-0.39)FW	
Peregrine falcon, Falco peregrinis		
Baltimore, Maryland, Age 7+		
Liver	0.8 FW	De Ment et al. 1986
Kidney	1.4 FW	
Prey organism		
Rock dove		
Urban		
Blood	1 (0.3 - 17) FW	
Liver	3 FW	
Kidney	9 FW	
Whole	5 FW	
Rural		
Blood	<0.1 FW	
Liver	0.4 FW	
Kidney	0.5 FW	
Whole	0.3 FW	
Common loon, Gavia immer, Pb poisoned		
Liver	(21 - 39) FW	Locke et al. 1982
Bald eagle, Haliaeetus leucocephalus		
Nationwide, 1978 - 1981, found dead, suspected Pb poisoning		
Liver	28 (11-61) FW	Reichel et al. 1984
Liver		
Control	0.6 FW	Bagley and Locke 1967
Pb-poisoned	21 FW	Mulhern et al. 1970
Barn swallow, Hirundo rustica		
Near Baltimore-Washington Parkway, 1979		
Feather		
Male	67 (55-82) DW	Grue et al. 1984
Female	54 (43-68) DW	
Nestling	2 (2 - 3) DW	
Carcass		
Male	5 (4 - 6) DW	
Female	9 (6 -12) DW	
Nestling	2 (1 - 2) DW	
Stomach contents		
Male	5 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
1972		
Georgia	0.1 FW	
Florida	0.1 FW	
1973		
South Carolina	0.3 FW	
Florida	0.2 FW	
Shot, 1970		
Florida	0.1 FW	
South Carolina	0.1 FW	
Sora rail, Porzana carolina		
Maryland		
Lead shot in gizzard		
Liver	(0.1 - 17) FW	Stendell et al. 1980
Bone	(1 - 127) DW	
No lead shot in gizzard		
Liver	(<0.01 - 0.08) FW	
Bone	(<0.4 - 42) DW	
Songbirds, carcass		
Near metal smelter, 10 species	56 (9 - 240) DW	Beyer et al. 1985
Control site, 9 species	15 (6 - 25) DW	
Southeastern Missouri, 1981-1982, Big River		
Green-backed heron, Butorides striatus		
Liver		
Upstream from mine site	0.1 (Max. 0.3) FW	Niethammer
Downstream	0.5 (Max. 1.5) FW	et al. 1985
Northern rough-winged swallow, Stelgidopteryx serripennis		
Carcass		
Upstream from mine site	0.5 (Max. 5) FW	
Downstream	1 (Max. 15) FW	
European starling, Sturnus vulgaris		
Nesting near highway, Maryland		
Carcass	(4 - 10) DW	Grue et al. 1986
Feathers	(7 - 52) DW	
Stomach contents	(84 - 94) DW	
Control site		
Carcass	(1 - 3) DW	
Feathers	(3 - 14) DW	
Stomach contents	(6 - 7) DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
California		
Merced	15 DW	
Sacramento	38 DW	
Other	25 DW	
Northern pintail, Anas acuta		
Adult	7 DW	
Immature	6 DW	
Mottled duck, Anas fulvigula		
Adult	48 DW	
Immature	40 DW	
Canvasback		
Adult	17 DW	
Immature	8 DW	
Redhead, Aythya americana		
Adult	26 DW	
Immature	24 DW	
Lesser scaup, Aythya affinis		
Adult	3 DW	
Immature	2 DW	
Black duck, Anas rubripes		
Adult	8 DW	
MAMMALS		
Field mouse, Apodemus sylvaticus		
Near abandoned Pb mine		
Whole body	(9 - 14) DW	Roberts et al. 1978
Kidney	(39 - 46) DW	
Liver	(12 - 13) DW	
Bone	(189 - 352) DW	
Brain	(6 - 13) DW	
Muscle	(7 - 10) DW	
Control area		
Whole body	1 DW	
Kidney	(9 - 13) DW	
Liver	(5 - 8) DW	
Bone	(11 - 21) DW	
Brain	(3 - 4) DW	
Muscle	(5 - 6) DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Dog, Canis familiaris		
Blood		
Healthy	(0.01 - 0.05) FW	NRCC 1973
Pb-poisoned	(0.06 - 0.15) FW	
Big brown bat, Eptesicus fuscus		
Whole, minus GI tract and large embryos		
Males	47 (20-90) FW	Clark 1979
Females	32 (20-56) FW	
Guano	61 DW	
Stomach contents	4 DW	
Horse, Equus caballus		
Near smelter, British Columbia		
Liver	18 FW	Burrows 1981
Kidney	16 FW	
Bone	88 FW	
Near Pb smelter (some deaths), California		
Liver	(15 - 222) FW	Knight and
Kidney	(14 - 80) FW	Burau 1973
Blood	(0.4 - 0.5) FW	
Control areas		
Blood	(0.1 - 0.3) FW	Jenkins 1980
Bank vole, Clethrionomys glareolus		
Whole body		
Near abandoned Pb mine	(16 - 21) DW	Roberts et al. 1978
Control area	(2 - 3) DW	
Chipmunk, Eutamias townsendii		
Hair		
Roadside location	235 DW	Raymond and
Control area	6 DW	Forbes 1975
Prairie vole, Microtus ochrogaster		
Illinois, whole body		
Near heavy traffic	8 DW	Getz et al. 1977b
Control area	3 DW	
Little brown bat, Myotis lucifugus		
Whole	17 (11-29) FW	Clark 1979
Guano	65 DW	
Stomach contents	26 FW	
Bats, Myotis spp., Florida 1981-1983		
Guano	(3 - 6) DW	Clark et al. 1986

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Kidney	3 DW	
Liver	1 DW	
Brain	0.1 DW	
Feces	7 DW	
Roadside locations		
Brain	(0.6 - 0.8) DW	Jenkins 1980
Liver	(0.9 - 3) DW	
Kidney	(2 - 8) DW	
Bone	(14 - 52) DW	
Hair	235 DW	
Control areas		
Brain	0.1 DW	
Liver	1 DW	
Kidney	3 DW	
Bone	5 DW	
Hair	6 DW	
Illinois, 1982		
Distance from lead battery reclamation plant		
100 m		
Liver	4 FW	Kisseberth et al. 1984
Kidney	13 FW	
Bone	79 FW	
1,000 m		
Liver	1 FW	
Kidney	3 FW	
Bone	2 FW	
Whole, 1978-1979		
Near Cu-Zn mine		
Juveniles	4 FW	Smith and Rongstad 1982
Adults	5 FW	
Control site		
Juveniles	0.5 FW	
Adults	0.7 FW	
Raccoon, Procyon lotor		
Connecticut, Pb-intoxicated		
Liver, kidney	>35 FW	Diters and Nielsen 1978
Commensal rat, Rattus norvegicus		
Houston, Texas, 1978-1979		
Urban		

Damage to plants with elevated Pb contents is usually negligible, but varies widely among species. Atmospheric Pb may have contributed to the decline of European spruce forests. The mean Pb content of needles and litter was significantly higher where tree decline was most pronounced than in areas where forests were unaffected (Backhaus and Backhaus 1986). Lead can have deleterious effects on plant growth processes at current Pb levels in urban areas and may similarly affect plants in rural areas in the future (Rolfe and Reinbold 1977). A reduction in yield of corn or soybeans is expected in low-binding capacity soils with Pb levels greater than 200 mg/kg (Rolfe and Reinbold 1977). Hay grown near roadsides may be toxic to horses and cattle (Rolfe and Reinbold 1977). In extreme cases, reforestration has been initiated in areas where forage is so heavily contaminated with Pb that it has become necessary to slaughter domestic livestock because the amounts of Pb in their livers and kidneys became unacceptably high (Edwards and Clay 1977). Typical area reforestration includes removal of contaminated forage by cutting, bailing, and burying native grasses; burning of stubble and litter; and adding of agricultural lime at the rate of 2,244 kg/ha (2,000 pounds/acre) to all soils within 1,525 m (5,000 feet) of sites where Pb levels exceed 175 mg/kg (Edwards and Clay 1977).

TERRESTRIAL INVERTEBRATES

In earthworms, lead levels were highest in those closest to highways and in areas with high volumes of traffic (Goldsmith and Scanlon 1977; Table 5). Various species of insects and soil invertebrates from roadsides, from areas receiving sewage sludge, and from metal smelter environs also contain high amounts of Pb (Table 5). Amounts of Pb in whole body were higher in earthworms, millipedes, and woodlice collected from soil and plant litter near highways than away from highways; soil and litter seem to be major reservoirs of Pb in roadside communities (Beyer and Moore 1980). In contrast, Pb concentrations in the eastern tent caterpillar (Malacosoma americanum) were lower than those reported for roadside soil and litter invertebrates, and were about 76% of that in leaves of its host, the black cherry Prunus serotina (Beyer and Moore 1980).

The use of terrestrial invertebrates as sentinel organisms has been suggested for monitoring Pb. The spider Araneus umbricatus, for example, contained Pb body burdens that correlated with that in a lichen (Lecanora conizaeoides) that is currently used to monitor atmospheric Pb (Clausen 1984). Similarly, the woodlouse (Porcellio scaber) seems to reflect Pb concentrations in adjacent soil or leaf litter (Hopkin et al. 1986).

AQUATIC BIOTA

Freshwater algae, invertebrates, and fish had comparatively elevated Pb concentrations when collected near industrialized areas, ponds with high numbers of Pb shot, urban areas, Pb mines, and tailings ponds (Table 5). For

Whitefish, Coregonus spp., from Pb-contaminated Swedish lakes, showed depressed blood ALAD and blood chemistry derangement when compared to fish from a reference lake--suggesting that Pb affects natural populations of fish in a manner similar to that observed in laboratory studies (Haux et al. 1986).

The significance of organolead residues in aquatic life is unknown, and merits additional research. In Ontario, Canada, about 16% of all fish sampled contained tetraalkyllead compounds, although none were recorded in water, vegetation, or sediments from the collection sites (Chau et al. 1980). Tetramethyllead reportedly was produced from biological and chemical methylation of several inorganic and organic Pb compounds in the aquatic environment, and has been detected at low concentrations in marine mussels, lobsters, and bony fishes (Wong et al. 1981).

AMPHIBIANS AND REPTILES

Tadpoles of bullfrogs (Rana catesbeiana) and green frogs (R. clamitans) from drainages along highways with different daily average traffic volumes (4,272 to 108,800 vehicles per day) contained elevated amounts of Pb (up to 270 mg/kg dry weight), which were positively correlated with Pb in sediments and with average daily traffic volume. Lead in tadpoles living near highways may contribute to the Pb levels reported in wildlife that eat tadpoles. Diets with amounts of Pb similar to those in tadpoles collected near heavily traveled highways have caused adverse physiological and reproductive effects in some species of birds and mammals (Birdsall et al. 1986). Elevated Pb concentrations also were recorded in various species of amphibians and reptiles collected near Pb smelters and mines (Table 5).

BIRDS

In general, Pb concentrations were highest in birds from urban locations (perhaps reflecting greater exposure to automotive and industrial contamination) and in birds near Pb mining and smelting facilities. Lead residues also are greatest in older birds (especially in bone, because of accumulation over time), in sexually mature females, and in waterfowl that have ingested Pb shot pellets (Table 5).

Continued deposition of Pb shot by hunters into wetlands habitats exposes birds to lead. Lead shot is a substantial localized source of contamination, especially in prime waterfowl habitat (Bellrose 1951, 1959; NRCC 1973; White and Stendell 1977; Stendell et al. 1979; Wobeser 1981; Clausen et al. 1982; Longcore et al. 1982; Mudge 1983; Driver and Kendall 1984; Hall and Fisher 1985). Several million hunters are estimated to deposit more than 6,000 metric tons of Pb shot annually into lakes, marshes, and estuaries; this represents about 6,440 pellets per bird bagged. Shot densities as great as 860,000 pellets/ha (2,124,000/acre) have been estimated in some locations (Wobeser 1981), although concentrations of 34,000 to 140,000/ha are more

(Kirby et al. 1983).

Lead in seeds and invertebrates within rights-of-way of major highways probably is not a hazard to adult ground-foraging songbirds, as judged from experiments with the European starling (Sturnus vulgaris). However, the effects of Pb on survival of fledglings are unknown, although Pb causes reductions in blood hemoglobin, hematocrit, ALAD activity, and brain weight (Grue et al. 1986). In another study, Pb concentrations in feather, carcass, and stomach contents of adult and nestling barn swallows (Hirundo rustica) were greater near a major U.S. highway than in a rural area; however, the number of eggs and nestlings, the body weight of nestlings at 17 days of age, and body weights of adults were similar in the two colonies, suggesting that contamination of roadsides with Pb from automobile emissions is not a major hazard to birds that feed on flying invertebrates (Grue et al. 1984).

Signs of Pb poisoning, i.e., depressed blood ALAD activity or elevated blood Pb levels, were reported for birds near a metal smelter (Beyer et al. 1985), in 17% of canvasbacks from Chesapeake Bay in 1974 (Dieter et al. 1976), and in three species of waders from the Dutch Wadden Sea living in an urban postnuptial moulting area (Goede and de Voogt 1985). The decline in submerged aquatic vegetation in Chesapeake Bay and the later shift in diet of some waterfowl species of Chesapeake Bay from the vegetation (Pb content 2.2 to 18.9 mg/kg dry weight), to the softshell clam Mya arenaria (1.3 to 7.6 mg Pb/kg dry weight), or to other bivalve molluscs (0.8 to 20.4 mg Pb/kg dry weight), probably did not increase dietary Pb burdens in these species (Di Guilio and Scanlon 1985).

The significance of trace amounts of organolead residues in birds is unknown. Trialkyllead seems to concentrate in avian kidney, but contributes less than 5% of the total amount of Pb in kidneys (Johnson et al. 1982).

MAMMALS

The highest body burdens of Pb reported in mammals were near urban areas of dense vehicular traffic, near metal mines and smelters, or near plants that reclaimed storage batteries; concentrations were higher in older organisms, especially in bone and hematopoietic tissues (Table 5; Goldsmith and Scanlon 1977; Way and Schroder 1982). A similar pattern of Pb occurrence and distribution was evident for human populations (Barth et al. 1973).

Diet provides the major pathway for Pb exposure, and amounts in bone are indicative of estimated Pb exposure and metabolism (Chmiel and Harrison 1981). Amounts of whole body Pb and feeding habits of roadside rodents were correlated: body burdens were highest in insectivores such as shrews; intermediate in herbivores, and lowest in granivores (Boggess 1977; Getz et al. 1977c). Food chain biomagnification of Pb, although uncommon in terrestrial communities, may be important for carnivorous marine mammals, such as the California sea lion (Zalophus californianus); accumulations were

The interaction effects of Pb components in smelter emissions with other components, such as zinc, cadmium, and arsenic, are unresolved (EPA 1972), and warrant additional research.

TERRESTRIAL PLANTS AND INVERTEBRATES

Fruits and vegetables acquire Pb by surface deposition from rainfall, dust, and soil, and by biological uptake through the root system (EPA 1980). Foliar absorption of Pb and transport to the root could account for a significant portion of the Pb in root tissues; however, this transport process varies widely among species. Dollard (1986) showed that this pathway accounted for 35% of the root Pb content in the radish (Raphanus sativus), but for <3% in carrots (Daucus carota) and beans (Phaseolus vulgaris). Corn (Zea mays) contained 30 mg Pb/kg dry weight when grown in soils containing Pb concentrations of 924 mg/kg, but only 17 mg/kg when grown in soils containing 786 mg Pb/kg. Sadiq (1985) concluded that contamination of soils with up to 800 mg Pb/kg probably does not elevate concentrations of Pb in corn plants. Within any plant species, however, there are Pb-resistant and Pb-sensitive breeds; some genetically fixed resistant species grow in soils containing up to 10,000 mg Pb/kg (Holl and Hampp 1975).

Plants readily accumulate Pb from soils of low pH or low organic content; however, uptake is significantly reduced after the application of lime or phosphate, which converts Pb to hydroxides, carbonates, or phosphates of relatively low solubility (Demayo et al. 1982). Lead persists for lengthy periods in forest litter; the estimated Tb 1/2 is 220 years (Turner et al. 1985). Lead seems to be tightly bound by most soils, and substantial amounts must accumulate before it affects the growth of higher plants (Boggess 1977). Although Pb is preferentially bound in soils by organics and oxides, interaction kinetics of Pb with other metals are complex and largely unknown (Bjerre and Schierup 1985). For example, uptake of Pb from soils by oat seeds (Avina sativa) was inhibited by cadmium salts, and reduced in loamy or organic soils; further, Pb in soils interfered with manganese uptake, and also increased the availability of cadmium and other heavy metals (Bjerre and Schierup 1985).

Lead inhibits plant growth, reduces photosynthesis, and reduces mitosis and water absorption (Demayo et al. 1982). Inhibition of photosynthesis is attributed to the blocking of protein sulfhydryl groups and to changes in phosphate levels in living cells (Holl and Hampp 1975). For two species of roadside weeds (Cassia spp.), pollen germination was reduced by 90% and seed germination by 87% at Pb levels of about 500 mg/kg dry weight in soil and about 300 mg/kg dry weight in foliage (Krishnayya and Bedi 1986). Normal germination rates were recorded at Pb levels of 46 mg/kg in soil and 22 mg/kg dry weight in foliage; however, some adverse effects were evident at Pb levels of 12 to 312 mg/kg in soil, and 55 to 97 mg/kg dry weight in foliage (Krishnayya and Bedi 1986). Tetraethyllead from automobile exhaust fumes is known to react in the light to produce the highly phytotoxic triethyllead cation (Backhaus and Backhaus 1986), which can freely permeate the plasma membranes of plant cells (Stournaras et al. 1984). Growth of cultures of soybean (Glycine max) cells exposed to 207 ug Pb/l (as triethyllead salts) was inhibited before the cells died (Stournaras et al. 1984). There is no

Table 6. Lethal and sublethal effects of lead[a] to selected species of aquatic organisms.

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
FRESHWATER				
Algae and macrophytes				
Alga, *Selenastrum capricornutum*	5	28 days	BCF 92,000	1
Alga, *S. capricornutum*	50	28 days	BCF 26,000	1
Alga, *Chlamydomonas reinhardii*	207	3 hours	BCF 26; some inhibition of photosynthesis	2
Alga, *C. reinhardii*	1,000	3 hours	BCF 20; 50% inhibition of photosynthesis	2
Alga, *C. reinhardii*	4,140	24 hours	Lethal	2
Alga, *Microcystis aeruginosa*	450	8 days	Immobilization	3
Invertebrates				
Daphnid, *Daphnia magna*	1	19 days	Reproductive impairment, 10%	4
Daphnid, *D. magna*	10	19 days	Reproductive impairment, 50%	4
Daphnid, *D. magna*	30	21 days	Reproductive impairment, 16%	3,5
Water hardness (mg $CaCO_3$/l) 52	9 - 16.7	Lifetime	MATC	3

Table 6. (Continued)

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
Midge, Tanytarsus dissimilis	258	10 days	LC-50	3
Isopod, Asellus meridianus				
Nontolerant strain	280	48 hours	LC-50	5
From Pb-contaminated river	3,500	48 hours	LC-50	5
Daphnid, Daphnia hyalina	600	48 hours	LC-50	6
Snail, Viviparus ater	1,000	7 days	Neuronal cytolysis	10
Snail V. ater	117,000	96 hours	LC-50	10
Aquatic insects, 5 species	3,500 to 64,000	7 to 14 days	LC-50	5
Isopod, Asellus aquaticus				
Nontolerant strain				
Pretreated for 5 days to 100 ug Pb/l	794,000	48 hours	LC-50	11
No pretreatment	330,000	48 hours	LC-50	11
Fish				
Rainbow trout, Salmo gairdneri				
Tetramethyl Pb				
Weight 1 gram	3.5	72 hours	LC-50	12

Table 6. (Continued)

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
35	71 - 146	Lifetime	MATC	3
353				
Total Pb	506,500	96 hours	LC-50	15
Dissolved Pb	1395	96 hours	LC-50	15
Dissolved Pb	120 - 360	Lifetime	MATC	15
Dissolved Pb	18.2 - 31.7	Lifetime	MATC	15
28				
Pre-hatch fry	4 - 7.6	Lifetime	MATC	5
Post-hatch fry	7.6 - 14.6	Lifetime	MATC	5
28	14.6	19 months	Vertebral deformities; caudal fin erosion	6
353	31	19 months	As above	6
28	7.2	19 months	No harmful effects	6
353	18.2	19 months	As above	6
Lake trout, Salvelinus namaycush				
Water hardness 33	48 - 83	Lifetime	MATC	16
Zebrafish, Brachydanio rerio				
Egg	50	24 hours	Pigmentation patterns of fry irreversibly altered	17
Egg	72	24 hours	Hatching inhibited	17
Brook trout, Salvelinus fontinalis				
Water hardness 44				
Total Pb	58 - 119	3 generations	MATC	3,5,18

Table 6. (Continued)

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
Cyprinid, Puntius conchonius	127	4 months	Gonadal pathology	19
Cyrinid, P. conchonius	379	96 hours	LC-50	19
Goldfish, Carassius auratus	200	4-5 days	ALAD inhibition	6
Northern pike, Esox lucius				
Water hardness 34(mg $CaCO_3$/l)	253 - 483	Lifetime	MATC	5
Threespine stickleback, Gasterosteus aculeatus	300	96 hours	LC-100	6
Smallmouth bass, Micropterus dolomieui				
Water hardness 152(mg $CaCO_3$/l)				
Fingerlings	405	90 days	No effect on growth, behavior, blood chemistry	20
Swim-up fry	2,800	96 hours	LC-50	20
Fingerlings	29,000	96 hours	LC-50	20
Egg and sac-fry	>15,900	96 hours	LC-50	20
Fathead minnow, Pimephales promelas				
Water hardness(mg $CaCO_3$/l)				
20	6,500	96 hours	LC-50	7
360	460,000	96 hours	LC-50	7
MARINE				
Algae and macrophytes				

Table 6. (Continued)

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
Pb^{2+}, larvae	476	96 hours	LC-50	3
Triethyl Pb	1,100	96 hours	LC-50; BCF 10	23
Trimethyl Pb	500	96 hours	LC-50; BCF 23	23
Tetramethyl Pb	270	96 hours	LC-50; BCF 170	23
Tetraethyl Pb	100	96 hours	LC-50; BCF 120	23
Pb^{2+}	10	63 days	BCF 12,580 for kidney and 1,580 for soft parts	25
Pb^{2+}	500	150 days	BCF 25,670 for soft parts	26
Softshell clam, Mya arenaria				
Soft parts				
Temperature, °C				
0 - 10	14	42 days	BCF 158	27
0 - 10	70	42 days	BCF 180	27
16 - 22	14	14 days	BCF 351	27
16 - 22	70	7 days	BCF 237	27
Mysid, Mysidopsis bahia	17 - 37	Lifetime	MATC	3
Sandworm, Neanthes arenaceodentata				
Salinity, o/oo				
15	20	28 days	Inhibited reproduction	28
20	3,100	28 days	Inhibited reproduction	28
Temperature, °C				
15	10,700	96 hours	LC-50	28
20	7,700	96 hours	LC-50	28
Shrimp, Crangon crangon				
Pb^{2+}	375,000	96 hours	LC-50	23
Trimethyl Pb	8,800	96 hours	LC-50; BCF 1	23
Triethyl Pb	5,800	96 hours	LC-50; BCF 2	23

Table 6. (Concluded)

Ecosystem, taxonomic group, species, and other variables	Concentration (ug Pb/l medium)	Exposure duration	Effect[b]	Reference[c]
Mummichog, Fundulus heteroclitus	315	96 hours	LC-50	3

[a]As total Pb, unless indicated otherwise.

[b]BCF = bioconcentration factor; MATC = maximum acceptable toxicant concentration. Lower value in each MATC pair indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, and metabolic upset during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

[c]References: 1, Vighi 1981; 2, Irmer et al. 1986; 3, EPA 1985; 4, Berglind et al. 1985; 5, Demayo et al. 1982; 6, Wong et al. 1978; 7, NRCC 1973; 8, Borgmann et al. 1978; 9, Spehar et al. 1978; 10, Fantin et al. 1985; 11, Fraser 1980; 12, Wong et al. 1981; 13, Johansson-Sjobeck and Larsson 1979; 14, Rombaugh 1985; 15, Davies et al. 1976; 16, EPA 1980; 17, Ozoh 1980; 18, Holcombe et al. 1976; 19, Kumar and Pant 1984; 20, Coughlan et al. 1986; 21, Rivkin 1979; 22, Schulz-Baldes and Lewin 1976; 23, Maddock and Taylor 1980; 24, Zaroogian et al. 1979; 25, Schulz-Baldes 1974; 26, Schulz-Baldes 1972; 27, Eisler 1977; 28, Reish and Gerlinger 1984; 29, Gould and Grieg 1983; 30, Kobayashi 1971.

stippling of erythrocytes; elevated Pb concentrations in blood, bone, gill, liver, and kidney; muscular atrophy; paralysis; renal pathology; growth inhibition; retardation of sexual maturity; altered blood chemistry; testicular and ovarian histopathology; and death (Aronson 1971; NRCC 1973; Adams 1975; Davies et al. 1976; Holcombe et al. 1976; Hodson et al. 1977, 1980, 1982; Johansson-Sjobeck and Larsson 1979; Reichert et al. 1979; Ozoh 1980; Demayo et al 1982; Kumar and Pant 1984; Rai and Qayyum 1984; Hodson and Spry 1985; Haux et al. 1986). The prevalence of signs is closely correlated with duration of exposure to Pb and to its uptake (Hodson et al 1982). Toxic effects of Pb uptake in fishes are increased under conditions favoring their rapid growth. Hodson et al. (1982) have shown that the rate of intoxication by Pb--as judged by uptake rates into tissues and incidence and prevalence of black tail--did not increase with fish size, but rather with growth rate.

Rooted aquatic plants, such as wild rice (Zizania aquatica), can accumulate up to 67 mg Pb/kg dry weight when cultured in tanks contaminated with high concentrations of powdered Pb (equivalent to 7,400 kg Pb/ha); however, this level is not considered hazardous to waterfowl feeding on wild rice (Behan et al. 1979). Lead content in plants collected from heavily hunted areas near refuges did not differ from those collected in the protected areas (Behan et al. 1979), which suggests that Pb bioavailability to rooted aquatics is substantially lower from shot than from powdered Pb. In another study with rooted macrophytes, Navicula sp. and Elodea canadensis rapidly accumulated Pb from solutions containing 1.0 mg Pb^{2+}/l, i.e., 70 mg Pb/kg dry weight per minute; the process was overwhelmingly passive (Everard and Denny 1985). Depuration was rapid; 90% of the Pb sorbed during the first hour by shoots of Elodea was released within 14 days after transfer to clean water, though 10% seemed to be irreversibly bound (Everard and Denny 1985).

High accumulations of Pb from ambient seawater by marine plants is well documented; concentration factors vary from 13,000 to 82,000 for algae from Raritan Bay, New Jersey (Seeliger and Edwards 1977), and from 1,200 to 26,000 for algae from Sorfjorden, Norway (Melhuus et al. 1978). Studies on the kinetics of lead uptake and retention in two species of marine algae (Phaeodactylum tricornutum, Platymonas subcordiformes) showed that both species accumulated Pb from the medium at ambient concentrations of 20 ug/l, and higher (Schulz-Baldes and Lewin 1976). In the first phase, usually completed within minutes after addition of Pb, cells of Phaeodactylum became saturated when the Pb reached a remarkable 11,640 mg/kg dry weight. In the second phase, the lead content of Platymonas continued to rise slowly, but that of Phaeodactylum declined after 2 or 3 days. In both species the content of bound Pb increased with increasing exposure time, suggesting that during prolonged exposure Pb is initially adsorbed to the cell surface, then translocated into the cell wall, the plasma membrane, and eventually the cytoplasm (Schulz-Baldes and Lewin 1976).

Sediments are not only sinks for Pb but may act as a source of Pb to aquatic biota after contamination from the original source has subsided

to the guppy. Thus, in organisms held for 28 days in solutions containing 5 ug Pb/l, Pb content was 460 mg/kg dry weight in the alga, 23 mg/kg in the grazing daphnids, and 4 to 16 mg/kg in the guppies that fed on the daphnids (Vighi 1981). Concentrations of Pb in the freshwater snail, Lymnaea peregra, collected near an abandoned Pb mine were positively correlated with the Pb content in its diet; the digestive glands contained up to 5,600 mg/kg dry weight (Everard and Denny 1984). The gut contents of eels (Anguilla anguilla) grazing on contaminated snails contained up to 4,350 mg Pb/kg, but the Pb was rapidly released; feces from both snails and eels return the Pb to the ecosystem as particulates and detritus (Everard and Denny 1984).

As discussed earlier, Pb clearly inhibits the formation of heme at several points, adversely affects blood chemistry, and accumulates in hematopoietic organs of aquatic organisms. In addition, Pb interferes with chlorophyll formation in plants by inhibiting the conversion of coproporphyrinogen to proporphyrinogen by competing with iron, inhibits allantoise formation in annelids, inhibits alpha-glycerophosphate dehydrogenase activity in trout, increases glutamic oxalacetate transaminase activity in Daphnia, affects neural and hormonal systems that control activity and metabolic rates in fish, interacts with polar sites of glycoproteins in epidermal mucus of fish, and may inhibit vitamin C and tryptophan metabolism (Wong et al. 1978).

Some populations of freshwater isopods are tolerant to Pb. Inasmuch as nontolerant isopods from an unpolluted site can be made tolerant by exposure to low levels, it is suggested that naturally occurring tolerance may be achieved by acclimatization (Fraser 1980). Research is needed on Pb transformation mechanisms, on toxic forms of Pb and interaction effects with other compounds, and on effects of Pb-contaminated sediments on benthos (Wong et al. 1978).

AMPHIBIANS AND REPTILES

Lead poisoning in adult leopard frogs (Rana pipiens) is indicated by a series of signs: sloughing of integument; sluggishness; decreased muscle tone; decreases in red blood cells, white blood cells, neutrophils, and monocytes; erosion of the gastric mucosa; and (before death) excitement, salivation, and muscular twitching. The 30-day LC-50 value for R. pipiens was 105 mg Pb/l, but some deaths and elevated liver residues were noted at water concentrations as low as 25 mg/l (Kaplan et al. 1967). In soft water (99 mg $CaCO_3$/l), some marbled salamanders (Ambystoma opacum) exposed to 1.4 mg Pb/l died in 8 days (EPA 1985). At about 1.0 mg/l, Pb blocked synaptic transmission by competitive inhibition of calcium in the bullfrog, Rana catesbeiana (Kober and Cooper 1976). At 0.5 mg Pb/l, tadpoles of Rana utricularia required additional time to metamorphose; and at 1.5 mg Pb/l, thyroid histopathology was recorded and the delay in metamorphosis was more pronounced (Yeung 1978).

from ingestion of Pb shot in food items (Custer et al. 1984). Some raptors ingest many shot in a short time. For example, the stomach of a bald eagle suspected of dying from Pb poisoning contained 75 shot (Jacobson et al. 1977). Results of experimental Pb shot poisoning of bald eagles (Table 7) confirmed results of nationwide monitoring showing that 5.4% of all dead eagles found in 1974-1975 died of Pb poisoning, as evidenced by liver Pb levels of 23 to 38 mg/kg fresh weight (Pattee et al. 1981). Ingestion of food containing biologically incorporated Pb, although contributing to the Pb burden of carnivorous birds, is unlikely in itself to cause clinical Pb poisoning (Custer et al. 1984). A similar case is made for powdered Pb (Franson et al. 1983), and forms of Pb other than shot (Table 7); the strong indication is that the form in which Pb is ingested is crucial.

Signs of Pb poisoning in birds have been extensively documented (Bellrose 1951, 1959; Jordan and Bellrose 1951; Clemens et al. 1975; Forbes and Sanderson 1978; Hunter and Wobeser 1980; Pattee et al. 1981; Wobeser 1981; Franson and Custer 1982; Johnson et al. 1982; Eastin et al. 1983; Kendall and Scanlon 1983; Street 1983; Di Giulio and Scanlon 1984; Fimreite 1984; Gjerstad and Hanssen 1984; Hudson et al. 1984; Anderson and Havera 1985; Burger and Gochfeld 1985; Carlson and Nielsen 1985; Friend 1985; Hoffman et al. 1985a; Lumeij 1985; Beyer et al. 1988). Outwardly, Pb-poisoned birds show the following signs: loss of appetite, lethargy, weakness, emaciation, tremors, drooped wings, green liquid feces, and impaired locomotion, balance, and depth perception. Internally, Pb-poisoned birds show microscopic lesions of the proventricular epithelium, pectoral muscles, brain, proximal tubular epithelium of the kidney, and bone medullary osteocytes; an enlarged bile-filled gall bladder; anemia; elevated protoporphyrin IX levels in blood; decreased ALAD activity levels in blood, brain, and liver; reduced brain weight; abnormal skeletal development; cephalic edema; and esophageal impaction. Postmortem examination of Pb-poisoned birds may show edematous lungs; serous fluid in the pleural cavity; bile regurgitation; abnormal gizzard lining; a usually pale, emaciated, and dehydrated carcass; and elevated Pb levels in liver (>2 mg/kg fresh weight, >10 mg/kg dry weight), kidney (>6 mg/kg dry weight), and blood (> 0.2 mg/l).

Toxic and sublethal effects of Pb and its compounds on birds held under controlled conditions vary widely with species, with age and sex, and with form and dose of administered Pb (Table 7). Several generalizations are possible: decreased blood ALAD and increased protoporphyrin IX activity levels are useful early indicators of Pb exposure; Pb shot and certain organolead compounds are the most toxic forms of Pb; nestlings are more sensitive than older stages; and tissue Pb concentrations and pathology both increase in birds given multiple doses over extended periods (Table 7).

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Single oral dose of shot	Dosed birds recaptured in significantly greater numbers than controls.	7
Single oral dose of tetraethyllead	LD-50 of 107 mg/kg BW. Signs of intoxication included excessive drinking, regurgitation, hypoactivity, muscular incoordination, fluffed feathers, eyelid drooping, tremors, and loss of appetite. Regurgitation within 7 minutes, other signs as soon as 20 minutes, and death usually between 1 and 4 days posttreatment. Remission took up to 8 days.	7a
Fed diets containing 25 mg Pb/kg, as lead nitrate, for 12 weeks	No deaths; no pathology; no significant accumulations of Pb in liver, kidney, or bone; no changes in hemoglobin or hematocrit; decrease in blood ALAD activity, and increase in blood Pb levels--both returned to normal within 3 weeks on Pb-free diet.	8
Fed diets containing 100 mg Pb^{2+}/kg	Elevated levels in bone (9.6 mg/kg fresh weight vs. 0.7 in controls) and egg (1.3 vs. 0.9 in controls).	9
Fed diets containing metallic Pb for 42 days		
100 mg/kg diet dry weight	Elevated Pb levels (mg/kg dry weight) in kidney (23) liver (7), and bone (5).	10

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Rock dove, Columba livia		
Intragastric administration of 6.25 mg Pb (as lead acetate)/kg BW daily for 64 weeks	Anemia, elevation in erythrocyte porphyrin, kidney pathology; residues (mg/kg fresh weight) of 603 in kidney, 501 in bone, 8 in liver, 2 in brain, 4.4 in blood, 0.8 in sciatic nerve, and 0.1 in crop.	14
Intubation of 6.25 mg Pb (as lead acetate)/kg BW, chronic exposure	Interfered with four-step learning sequence; elevated blood Pb levels remained for 5 weeks after Pb exposure.	15
Japanese quail, Coturnix japonica		
Single oral dose of tetraethyllead	LD-50 of 24.6 mg/kg BW.	7a
Fed diets containing different forms of Pb for 5 days		
5,000 mg metallic Pb/kg	No effect on survival or food consumption.	16
5,000 mg Pb (as lead nitrate)/kg	No overt signs of toxicity.	16
5,000 mg Pb (as lead subacetate $C_4H_{10}O_8Pb_3$)/kg	No overt signs of toxicity.	16
2,761/mg Pb (as lead arsenate)/kg	LD-50.	16
Prairie falcon, Falco mexicanus		
Fed shotgun-killed pheasants and ducks	Death, preceded by vomiting, ataxia, blindness, and	

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
10 mg Pb/kg diet	Elevated Pb in bone (4 to 9 mg/kg dry weight vs. <0.8 in controls) and in liver (3 vs. <0.5 in controls).	20
Nestlings dosed orally with metallic Pb powder daily for 10 days		
625 mg/kg BW	Mortality (40% in 6 days); reduced growth; reduced kidney and liver weight; abnormal skeletal development; ALAD depression in all tissues examined; elevated burdens (mg/kg fresh weight) in kidney (15), liver (6), and brain (3).	21
125 mg/kg BW	Reduced growth, reduced brain weight, abnormal skeletal development, ALAD depressions in hematopoietic tissues, elevated burdens (mg/kg fresh weight) in kidney (7), and liver (4).	21
25 mg/kg BW	ALAD depression in all tissues examined; burdens (mg/kg fresh weight) elevated in kidney (3) and in liver 1.4).	21
Fed 60 days with homogenized cockerels (Gallus sp.) containing up to 448 mg (biologically incorporated) Pb/kg dry weight	No effect on survival, growth, hemoglobin, hematocrit, and erythrocyte number. Elevated burdens in kidney, liver, femur, brain, and blood.	22

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
3 No.6 shot (300 mg)	Some deaths between days 8 and 15 posttreatment, reduced food intake, weight loss, lethargy, diarrhea; residues of 7.3 mg/kg fresh weight liver, 139 dry weight tibia.	25,26
6 No. 6 shot (600 mg)	If shot retained in gizzard, death resulted; residues (mg/kg) 72 fresh weight in liver, 154 dry weight in tibia.	25,26
Controls	Residues (mg/kg) 0.1 fresh weight in liver, 5 dry weight in tibia.	25,26
Raptors, 4 spp.		
Fed rock doves (Columba livia) and brown hares (Lepus europaeus) containing Pb shot for 3 weeks to 6 months	Death preceded by weight loss, convulsions, and inability to fly. Residues (mg/kg dry weight) at death ranged from 57 to 175 in liver, and 34 to 221 in kidney.	27
Common tern, Sterna hirundo		
Single injection of 200 mg Pb^{2+}	Adverse effects on behavior (locomotion, balance, righting response, feeding tasks, behavioral thermoO regulation); most apparent within 5 days postinjection.	28
Ringed turtle-dove, Streptopelia risoria		
Single oral dose of 2 pellets (220 mg)	Blood Pb (mg/l) 4.69 at 24 hours, and 0.14 at 14 days (vs. control values of 0.004 to 0.012 mg/l); blood ALAD depressed from 24 hours through 14 days.	29

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
	and feather in progeny of Pb-treated parents than in controls.	34
European starling, Sturnus vulgaris		
Oral administration (capsule) of triethyllead chloride at 2,000 ug daily (28 mg/kg BW) for 11 days, or until death	Mortality 100% by day 6. Dying birds showed decreased respiration, squatting, fluffed feathers, and abnormal head posture. Average residues (mg/kg fresh weight) 6.0 in bone, 7.3 in brain 19.9 in kidney, 20.0 in muscle, and 40.2 in liver.	35
As above, but dose was 200 ug daily (2.8 mg/kg BW)	No deaths, reduced food consumption. All tissue residues <2.0 mg/kg fresh weight (vs. <0.1 in controls).	35
Oral administration (capsule) of trimethyllead chloride at 2,000 ug daily (28 mg/kg BW) for 11 days, or until death	Mortality 100% by day 6. Signs included impaired balance, tremors, fluffed feathers, uncoordinated feeding movements, weight loss, inability to fly. Residues (mg/kg fresh weight) averaged 4.3 in bone, 11.0 in muscle, 16.7 in brain, 30.2 in kidney, and 82.4 in liver.	35
As above, but dose was 200 ug daily (2.8 mg/kg BW)	No deaths, survivors hyperactive. Average tissue residues (mg/kg fresh weight) 0.4 in bone, 3.1 in muscle, 3.5 in brain, 3.7 in liver, and 5.4 in kidney.	35

Trialkyllead salts are 10 to 100X more toxic to birds than are inorganic Pb salts; they tend to accumulate in lipophilic soft tissues in the yolk and developing embryo, and have high potential as neurotoxicants (Forsyth et al. 1985); accordingly more research is needed on alkyllead toxicokinetics. Some alkyllead compounds have been implicated in bird kills. In autumn 1979, about 2,400 birds of many species were found dead or disabled on the Mersey estuary, England, an important waterfowl and marsh bird wintering area; smaller kills were observed in 1980 and 1981 (Bull et al. 1983). Affected birds contained elevated Pb concentrations in liver (>7.5 mg/kg fresh weight), mostly as organolead. Bull et al. (1983) suggested that trialkyllead compounds were discharged from a petrochemical factory producing alkylleads, into the estuary where they were accumulated (up to 1.0 mg/kg fresh weight) by clams (Macoma balthica) and other invertebrates on which the birds could feed. Birds dosed experimentally with trialkyllead compounds died with the same behavioral and internal signs found in Mersey casualties; tissue levels of trialkyllead were similar in the two groups of birds (Osborn et al. 1983). Sublethal effects that might influence survival in the wild were found in both sublethally dosed and apparently healthy wild birds when tissue levels of trialkyllead compounds were matched in the two groups of birds. It was concluded that trialkyllead compounds were the main cause of the observed mortalities and that many apparently healthy birds were still at risk (Osborn et al. 1983).

Nestlings of altricial species (those confined to the nest for some time after hatch) may be considerably more sensitive to Pb exposure than adults, and also more sensitive than hatchlings of many precocial species (Hoffman et al. 1985a). Hatchlings of precocial species, including chickens, Japanese quail (Coturnix coturnix), mallards, and pheasants, are relatively tolerant to moderate Pb exposure, i.e., there was no effect on growth at dietary levels of 500 mg Pb/kg, or survival at 2,000 mg Pb/kg (Hoffman et al. 1985a,b).

Some species of domestic birds are resistant to Pb toxicosis. for example, blood Pb levels of 3.2 to 3.8 mg/l in Pb-stressed cockerels (Gallus sp.) were much higher than residues considered diagnostic for Pb poisoning in most domestic mammals, except swine--which tolerated up to 143 mg Pb/l blood (Franson and Custer 1982).

MAMMALS

Three stages of recognizable Pb poisoning, or plumbism, have been reported in humans (NRCC 1973): (1) mild or severe dysfunction of the alimentary tract as shown by loss of appetite, constipation, abdominal cramps, headaches, general weakness, and fatigue; (2) atrophy of forearm extensor muscles, or paralysis of these muscles and more striking atrophy; and (3) lead encephalopathy, which occurs frequently in Pb-poisoned infants and young children, but only rarely in industrial workers. In general, people with hepatitis, anemia, and nervous disorders were more susceptible to Pb poisoning (Barth et al. 1973). The transfer of Pb across the human placenta and its potential threat to the fetus have been recognized for more than 100 years; women occupationally exposed to Pb showed a comparatively high abortion rate (Tachon et al. 1983). Sensitivity of the brain to the toxic effects of Pb is

Table 8. Lethal and sublethal effects of lead to selected species of mammals.

Species, dose, and other variables	Effects	Reference[a]
Cattle, cows, Bos spp.		
Tissue Pb (mg/kg fresh weight) 0.81 in blood, 26.4 in liver, 50.3 in kidney, and 400 in rumen contents	Signs of clinical Pb toxicosis observed.	1
Calves given 2.7 mg Pb/kg body weight (BW), as Pb acetate, for 20 days; milk diet	Death.	2
Calves given 3.0 to 3.5 mg Pb/kg BW daily for 3 months; grain and hay diet	No effect.	2
Calves given 5 mg Pb/kg BW, as Pb acetate, for 7 days; grain and hay diet	Appeared normal.	3
Calves given 5 mg Pb/kg BW, as Pb acetate, for 7 days; milk diet	Signs of Pb poisoning; some deaths.	3,4
Calves given 5 mg Pb/kg BW daily for 10 to 20 days	Blindness, 16% mortality.	4
Calves given forage containing 5 to 6 mg Pb/kg	Fatal in 2 months.	1
Calves given 5 to 6 mg Pb/kg BW daily for 3 years	Chronic toxicity.	2
Adults given 6 mg Pb/kg BW daily for 3 years	No deaths.	5
Calves given 6 to 7 mg Pb/kg BW daily for 2 months	Fatal.	2

Table 8. (Continued)

Species, dose, and other variables	Effects	Reference[a]
equivalent to about 3.5 mg Pb/kg BW	Tissue Pb levels (mg/kg fresh weight) 1.2 in brain, 1.7 in blood, 15.7 in spleen, 23.4 in liver, 32.2 in kidney, and 735 in bone.	9
Total dose of 10 to 25 grams	Toxic.	6
Goat, Capra sp.		
Total dose of 20 to 25 grams	Toxic.	6
Guinea pig, Cavia cobaya		
Single intraperitoneal injection of 25 mg/kg BW, as Pb acetate	Reduced brain weight of newborn pigs. Effect synergized when dams were exposed to elevated (42 °C) temperatures for 24 hours: 88% with microencephaly vs. 5% in group given 25 mg/kg without hyperthermia.	10
Horse, Equus caballus		
Tissue Pb levels, in mg/kg fresh weight, of 0.39 in blood, 18 in liver, and 16 in kidney	Signs of clinical Pb toxicosis observed.	1
Ate forage containing 1.7 mg Pb/kg	Fatal in several months.	1
Consumed 2.4 mg Pb/kg BW daily	Lethal.	4
Fed 6.25 mg Pb/kg BW daily for 105 days, as Pb acetate	No deaths; blood Pb levels of 350 to 380 ug/l at day 105.	6

Table 8. (Continued)

Species, dose, and other variables	Effects	Reference[a]
Mouse, Mus sp.		
0.05 to 0.1 mg Pb/kg BW daily	Irreversible inhibition of ALAD activity in bone marrow and red blood cells.	14
Tissue concentrations of 0.78 mg/kg femur bone marrow, 3.7 mg/l blood, 15.8 mg/kg brain, or 43 mg/kg liver	Inhibition of ALAD activity 50% within 10 minutes.	14
1.5 mg Pb/kg BW daily, as tetraethyllead chloride	Reduction in success of implanted ova.	8
2.2 mg/kg BW or 3 mg/kg BW daily, as tetraethyllead	Frequency of pregnancy reduced when dose given 3 to 5 days after mating.	8
Pregnant females given single intrauterine injection of 20 mg Pb/kg BW on day 8 of gestation	Smaller litters, increased fetal deaths.	15
800 mg Pb/l, as lead acetate, in drinking water for 11 weeks	Decrease in litter size, decreased survival of pups, and decreased birth weight.	16
1,000 mg Pb/l in drinking water for 9 months	No effect on survival or fertility.	4
Sheep, Ovis aries		
Lambs fed 50 ug Pb/kg daily (~ 3 mg)	Tissue accumulations.	5
Lambs exposed to low levels (350 ug Pb/l blood) in utero	Impaired visual discrimination and learning behavior.	17

Table 8. (Continued)

Species, dose, and other variables	Effects	Reference[a]
Single oral dose of 600 mg Pb/kg BW	Fatal.	9
Total dose of 20 to 25 grams	Toxic.	6
Primates, various species		
Cynmolgus monkey, Macaca iris		
Intramuscular injection of 1.0 mg Pb/kg BW daily during pregnancy or lactation	Fetus exposed to lead through placenta or maternal milk.	19
1.5 mg Pb/l in drinking water as lead acetate, for 9 months (equivalent to 0.5 mg Pb/kg BW daily), or 6 mg/l (2 mg/kg BW), or 15 mg/l (5 mg/kg BW).	Increasing blood Pb levels from third month, according to dose; kidney pathology. Effects more severe in animals on low calcium diets.	20
Intramuscular adminstration of 5 mg Pb^{2+}/kg BW daily during pregnancy or lactation	Abortions and death in pregnant monkeys; cerebral pathology in newborns.	19
Cynmolgus monkey, Macaca fascicularis		
Dosed orally from birth to age 200 days with 100 ug Pb (as Pb acetate)/kg BW, 5X weekly, milk substitute diet	Blood Pb concentration of 254 ug/l; declined to 131 ug/l over next 100 to 150 days. At age 3 years, impaired ability to perform motor discrimination reversal tasks.	21
As above, except dose is 50 ug Pb/kg BW	Blood Pb levels of 154 ug/l (vs. 35 ug/l in controls), declining to 109 ug/l at day 150 post-administration. At age 3 years, group showed impaired color discrimination. No overt signs of toxicity, normal blood chemistry (except Pb), normal growth and development skills.	21

Table 8. (Continued)

Species, dose, and other variables	Effects	Reference[a]
Weanling females given 0.0, 0.5, 5, 25, 50, or 250 mg Pb (as Pb acetate)/l drinking water for 6 to 7 weeks, then mated and exposed continuously through gestation and lactation	At 25 mg/l and higher, growth retardation and delayed vaginal opening observed; some maternal deaths occurred and were associated with blood Pb concentrations >200 ug/l. Some pup malformations and deaths in all groups. The 5 mg/l group had elevated blood Pb levels.	23
Single intravenous injection (mg Pb/kg BW in parentheses)		
Tetramethyllead (80)	LD-50.	24
Tetraethyllead (10)	LD-50.	24
Trimethyllead (20 to 25)	LD-50.	24
Triethyllead (8)	LD-50.	24
Single oral dose (mg Pb/kg BW)		
Tetramethyllead (108)	LD-50.	24
Tetraethyllead (12)	LD-50.	24
Single intraperitoneal injection (mg Pb/kg BW)		
Tetramethyllead (70 to 100)	LD-50.	24
Tetraethyllead (10)	LD-50.	24
Trimethyllead (17)	LD-50.	24
Triethyllead (5)	LD-50.	24
5 mg Pb/l drinking water, lifetime exposure	Lowered survival and reduced longevity.	4
Single intraperitoneal injection of 7 mg Pb/kg BW, as tetraethyllead	Depressed food intake, and hyperactivity.	25
Male weanlings exposed to age 50 days to drinking water containing 25 mg Pb/l, as Pb acetate	At day 86: behavioral deficits; blood Pb concentrations of 150 to 200 ug/l; brain Pb levels (ug/kg) 70 in treated group vs. 28 in controls.	26

Table 8. (Concluded)

Species, dose, and other variables	Effects	Reference[a]
Swine, Sus sp.		
Oral doses of 64 mg Pb/kg BW, as Pb acetate, 6X weekly for 13 weeks	No deaths, reduced blood ALAD activity, blood Pb concentration (a remarkable) 143 mg/l.	32
As above, except doses administered intraperitoneally	All died.	32
Total dose of 10 to 25 grams	Toxic.	6

[a]References: 1, Osweiler and Van Gelder 1983; 2, Zmudzki et al. 1983; 3, Zmudzki et al. 1984; 4, Demayo et al. 1982; 5, NRCC 1973; 6, Dollahite et al. 1978; 7, Kwatra et al. 1986; 8, Clark 1979; 9 Forbes and Sanderson 1978; 10, Edwards and Beatson 1984; 11, Burrows and Borchard 1982; 12, Gilmartin et al. 1985; 13, Barth et al. 1973; 14, Schlick et al. 1983; 15, Wide 1985; 16, Sharma and Kanwar 1985; 17, EPA 1980; 18, Nriagu 1978b; 19 Tachon et al. 1983; 20, Colle et al. 1980; 21, Rice 1985; 22, Hopkins 1970; 23, Kimmel et al. 1980; 24, Branica and Konrad 1980; 25, Czech and Hoium 1984; 26, Cory-Slechta et al. 1985; 27, Cory-Slechta et al. 1983; 28, Massaro et al. 1986; 29, Cory-Slechta et al. 1981; 30, Zirkin et al. 1985; 31, Barrett and Livesey 1985; 32, Lassen and Buck 1979.

Although the use of lead arsenate as an insecticide in orchards is diminishing, residues of Pb still remain in the upper soil surface and will continue to remain bioavailable almost indefinitely (Gilmartin et al. 1985).

Naturally occurring radiolead-210, which has a half-life of 22 years, is a significant contributor to the natural radiation dose in man; comparatively high levels have been reported in certain grasses and lichens, and their consumers, such as reindeer, caribou, and ptarmigan, as well as in lanternfishes (Nriagu 1978b). The implications of this finding to wildlife health are unknown.

Table 9. Proposed lead criteria for the protection of natural resources and human health.

Resource, (units), and other variables	Criterion	Reference[a]
CROPS		
Irrigation water (mg/l)		
USA		
Neutral and alkaline soils	<10	Demayo et
Acidic soils	<5	al. 1982
Chronic use	<5	Abbasi and
Short-term use	<20	Soni 1986
Canada		
Continuous use	<5	Demayo et
Intermittent use	<10	al. 1982
Australia	<5	
AQUATIC LIFE		
Freshwater (ug total Pb/l)		
USA		
Water hardness, in mg $CaCO_3$/l		
50	1.3[b], 34[c]	EPA 1985
100	3.2[b], 82[c]	
200	7.7[b], 200[c]	
Great Lakes		
Superior	<100	Harrison and
Huron	<200	Laxen 1981
Others	<250	
England	<400	
Seawater (ug total Pb/l)	5.6[b], 140[c]	EPA 1985
Water (ug/l)		
Tetraalkyllead	<1	Maddock and
Trialkyllead	<100	Taylor 1980
Sewage effluent limits (ug/l)		
California	<4,000	Harrison and
Industrial discharge limits to surface waters (ug/l)		Laxen 1981
Illinois	<100	
USA	<500	
Canada	<2,000	
Switzerland	<5,000	

Table 9. (Continued)

Resource, (units), and other variables	Criterion	Reference[a]
Domestic livestock		
Drinking water (ug/l)		
USA	<100	Demayo et al.
Australia	<250	1982
Canada		
Horse	<500	
Others	<1,000	
Forage (mg/kg fresh weight)		
Horse	<80	Edwards and
Cattle	<200	Clay 1977
Tissue residues		
Unstressed (mg/kg fresh weight)		
Blood	<0.2	Osweiler and
Liver	<1.1	Van Gelder 1978
Kidney	<1.2	
Mouse, Mus sp.		
Elevated (mg/kg body weight daily)		
Total intake	>0.05	Schlick et al. 1983
Mule deer, Odocoileus hemionus		
Excessive (mg/day)		
Total intake	>3	Harrison and Dyer 1984
Raccoon, Procyon lotor		
Elevated (mg/kg fresh weight)		
Liver	>10	Diters and Nielsen 1978
HUMAN HEALTH		
Drinking water (ug/l)		
USA		
1975	<500	Harrison and
1977	<250	Laxen 1981
1980	<50	EPA 1980; NAS 1980
South Africa	<500	Harrison and Laxen 1981
Canada, Australia	<50	Demayo et al. 1982
USSR, Japan	<100	Abbasi and
India	10 to <100	Soni 1986

Table 9. (Concluded)

Resource, (units), and other variables	Criterion	Reference[a]
Life-threatening	>1,000	
Target	<150, maximum 300	
Air (ug Pb/m^3)		
Safe, USA	<1.5 (3-month arithmetic mean)	NAS 1980
Occupational, USA	<50[f]	EPA 1979; NAS 1980
Proposed, worldwide	<2	Barrett and Howells 1984
Hazardous	2,220	Barth et al. 1973
House paints (mg/l)	<600	EPA 1979
Gasoline (mg/l)		
USA		
Recent	473 to 658[d]	EPA 1979
Proposed	131[e]	
UK		
1972	840	Harrison and
1978	450	Laxen 1981
1981	400	Barrett and
Proposed	150	Howells 1984
West Germany	150	Harrison and Laxen 1981

[a]Each reference applies to the values in the same row, and in the rows that follow for which no other reference is indicated.

[b]Four-day average, not to be exceeded more than once every 3 years.

[c]One-hour average, not to be exceeded more than once every 3 years.

[d]Equals 1.8 to 2.5 g/gallon.

[e]Equals 0.5 g/gallon.

[f]Average 8-hour period

[g]Blood Pb levels, usually expressed as ug/deciliter, have been converted to ug/liter, for uniformity, in the present work.

1980, 1985; Rice 1985). Behavioral deficits have been reported for young rats when blood Pb levels exceeded 0.1 mg/l, and in children with blood Pb concentrations of 0.4 to 0.5 mg/l (EPA 1980; Rice 1985), and in birds when Pb was administered early in development (Burger and Gochfeld 1985). Recently, behavioral impairment was recorded in 3-year-old monkeys that received 50 or 100 ug Pb/kg BW from birth to age 200 days. Blood Pb levels immediately after exposure, and at time of testing, were 0.15 to 0.25 mg/l (age 200 days), and 0.11 to 0.13 mg/l (age 3 years); this is the first report of behavioral impairment in a primate species at blood Pb concentrations that are considered to be well within the bounds of safety for children (Rice 1985). This subject appears to constitute a high priority research need for wildlife species of concern.

Teratol. 5:557-563.

Barrett, J., and P.J. Livesey. 1985. Low level lead effects on activity under varying stress conditions in the developing rat. Pharmacol. Biochem. Behav. 22:107-118.

Barth, D., A. Berlin, R. Engel, P. Recht, and J. Smeets (eds.). 1973. Proceedings international symposium. Environmental health aspects of lead. Commis. European Commun., Luxembourg. 1,168 pp.

Beeby, A. 1985. The role of Helix aspersa as a major herbivore in the transfer of lead through a polluted ecosystem. J. Appl. Ecology 22: 267-275.

Behan, M.J., T.B. Kinraide, and W.I. Selser. 1979. Lead accumulation in aquatic plants from metallic sources including shot. J. Wildl. Manage. 43:240-244.

Bellrose, F.C. 1951. Effects of ingested lead shot upon waterfowl populations. Trans. North Am. Wildl. Conf. 16:125-135.

Bellrose, F.C. 1959. Lead poisoning as a mortality factor in waterfowl populations. Illinois Nat. Hist. Surv. Bull. 27:235-288.

Benes, P., M. Cejchanova, and B. Havlik. 1985. Migration and speciation of lead in a river system heavily polluted from a smelter. Water Res. 19:1-6.

Beresford, W.A., M.P. Donovan, J.M. Henninger, and M.P. Waalkes. 1981. Lead in the bone and soft tissues of box turtles caught near smelters. Bull. Environ. Contam. Toxicol. 27:349-352.

Berglind, R., G. Dave, and M.L. Sjobeck. 1985. The effects of lead on aminolevulinic acid dehydratase activity, growth, hemoglobin content, and reproduction in Daphnia magna. Ecotoxicol. Environ. Safety 9: 216-229.

Bernhard, M., and A. Zattera. 1975. Major pollutants in the marine environment. Pages 195-300 in E.A. Pearson and E.D. Frangipane (eds.). Marine pollution and marine waste disposal. Pergamon Press, New York.

Beyer, W.N., and A. Anderson. 1985. Toxicity to woodlice of zinc and lead oxides added to soil litter. Ambio 14:173-174.

Beyer, W. N., and E. J. Cromartie. 1987. A survey of Pb, Cu, Zn, Cd, Cr, As, and Se in earthworms and soil from diverse sites. Environ. Monitor. Assess. 8:27-36.

Beyer, W. N., G. W. Miller, and E. J. Cromartie. 1984. Contamination of the

Boyer, I.J., and V. Di Stefano. 1985. An investigation of the mechanism of lead-induced relaxation of pigeon crop smooth muscle. J. Pharmacol. Exp. Ther. 234:616-623.

Boyer, I.J., D.A. Cory-Slechta, and V. Di Stefano. 1985. Lead induction of crop dysfunction in pigeons through a direct action on neural or smooth muscle components of crop tissue. J. Pharmacol. Exp. Ther. 234:607-615.

Braham, H.W. 1973. Lead in the California sea lion (Zalophus californianus). Environ. Pollut. 5:253-258.

Branica, M., and Z. Konrad (eds.). 1980. Lead in the marine environment. Pergamon Press, Oxford, England. 353 pp.

Buerger, T.T., R.E. Mirarchi, and M.E. Lisano. 1986. Effects of lead shot ingestion on captive mourning dove survivability and reproduction. J. Wildl. Manage. 50:1-8.

Bull, K.R., W.J. Avery, P. Freestone, J.R. Hall, D. Osborn, A.S. Cooke, and T. Stowe. 1983. Alkyl lead pollution and bird mortalities on the Mersey estuary, UK, 1979-1981. Environ. Pollut. 31A:239-259.

Bunzl, K., and W. Kracke. 1984. Distribution of ^{210}Pb, ^{210}Po, stable lead and fallout ^{137}Cs in soil, plants and moorland sheep of a heath. Sci. Total Environ. 39:143-159.

Burger, J., and M. Gochfeld. 1985. Early postnatal lead exposure: behavioral effects in common tern chicks (Sterna hirundo). J. Toxicol. Environ. Health 16:869-886.

Burrows, G.E. 1981. Lead toxicosis in domestic animals: a review of the role of lead mining and primary lead smelters in the United States. Vet. Human Toxicol. 23:337-343.

Burrows, G.E., and R.E. Borchard. 1982. Experimental lead toxicosis in ponies: comparison of the effects of smelter effluent-contaminated hay and lead acetate. Am. J. Vet. Res. 43:2129-2133.

Carlson, B.L., and S.W. Nielsen. 1985. Influence of dietary calcium on lead poisoning in mallard ducks (Anas platyrhynchos). Am. J. Vet. Res. 46:276-282.

CEP. 1979. International conference. Management and control of heavy metals in the environment. CEP Consultants, Edinburgh, UK. 664 pp.

Chasko, G.G., T.R. Hoehn, and P. Howell-Heller. 1984. Toxicity of lead shot to wild black ducks and mallards fed natural foods. Bull Environ. Contam. Toxicol. 32:417-428.

Custer, T.W., J.C. Franson, and O.H. Pattee. 1984. Tissue lead distribution and hematologic effects in American kestrels (Falco sparverius L.) fed biologically incorporated lead. J. Wildl. Dis. 20:39-43.

Czarneski, J.M. 1985. Accumulation of lead in fish from Missouri streams impacted by lead mining. Bull. Environ. Contam. Toxicol. 34:736-745.

Czech, D.A., and E. Hoium. 1984. Some aspects of feeding and locomotor activity in adult rats exposed to tetraethyl lead. Neurobehav. Toxicol. Teratology 6:357-361.

Dallinger, R., and W. Wieser. 1984. Patterns of accumulation, distribution and liberation of Zn, Cu, Cd and Pb in different organs of the land snail Helix pomatia L. Comp. Biochem. Physiol. 79C:117-124.

Davies, P.H., J.P. Goettl, Jr., J.R. Sinley, and N.F. Smith. 1976. Acute and chronic toxicity of lead to rainbow trout Salmo gairdneri, in hard and soft water. Water Res. 10:199-206.

Decker, R.A., and A.M. McDermid, and J.W. Prideaux. 1979. Lead poisoning in two captive king vultures. J. Am. Vet. Med. Assoc. 175:1009.

Demayo, A., M.C. Taylor, K.W. Taylor, and P.V. Hodson. 1982. Toxic effects of lead and lead compounds on human health, aquatic life, wildlife plants, and livestock. CRC Crit. Rev. Environ. Control 12:257-305.

De Ment, S.H., J.J. Chisolm, Jr., J.C. Barker, and J.D. Strandberg. 1986. Lead exposure in an "urban" peregrine falcon and its avian prey. J. Wildl. Dis. 22:238-244.

De Michele, S.J. 1984. Nutrition of lead. Comp. Biochem. Physiol. 78A:401-408.

Deuel, B. 1985. Experimental dosing of northern pintails in California. Calif. Fish Game 71:125-128.

Dieter, M.P. 1979. Blood delta-aminolevulinic acid dehydratase (ALAD) to monitor lead contamination in canvasback ducks (Aythya valisineria). Pages 177-191 in National Academy of Sciences. Animals as monitors of environmental pollutants. Washington, D.C.

Dieter, M.P., and M.T. Finley. 1978. Erythrocyte δ-aminolevulinic acid dehydratase activity in mallard ducks: duration of inhibition after lead shot dosage. J. Wildl. Manage. 42:621-625.

Dieter, M.P., and M.T. Finley. 1979. Delta aminolevulinic acid dehydratase enzyme activity in blood, brain, and liver of lead-dosed ducks. Environ. Res. 19:127-135.

of forage. Vet. Human. Toxicol. 19:247-249.

Eisenreich, S.J., N.A. Metzer, N.R. Urban, and J.A. Robbins. 1986. Response of atmospheric lead to decreased use of lead in gasoline. Environ. Sci. Technol. 20:171-174.

Eisler, R. 1977. Toxicity evaluation of a complex metal mixture to the softshell clam Mya arenaria. Mar. Biol. 43:265-276.

Eisler, R. 1981. Trace metal concentrations in marine organisms. Pergamon Press, New York. 687 pp.

Eisler, R. 1984. Trace metal changes associated with age of marine vertebrates. Biol. Trace Elem. Res. 6:165-180.

EPA. 1972. Helena Valley, Montana, area environmental pollution study. U.S. Environ. Protection Agency Rep. AP-91. 179 pp. Avail. from U.S. Environ. Protection Agency, Office of Air Programs, Research Triangle Park, North Carolina 27711.

EPA. 1979. The health and environmental impacts of lead and an assessment of a need for limitations. U.S. Environ. Protection Agency Rep. 560/2-79-001. 494 pp.

EPA. 1980. Ambient water quality criteria for lead. U.S. Environ. Protection Agency Rep. 440/5-80-057. 151 pp. Avail. from Natl. Tech. Infor. Serv., 5285 Port Royal Road, Springfield, Virginia 22161.

EPA. 1985. Ambient water quality criteria for lead - 1984. U.S. Environ. Protection Agency Rep. 440/5-84-027. 81 pp. Avail. from Natl. Tech. Infor. Serv., 5285 Port Royal Road, Springfield, Virginia 22161.

Eskildsen, J., and P. Grandjean. 1984. Lead exposure from lead pellets: age-related accumulation in mute swans. Toxicol. Lett. 21:225-229.

Everard, M., and P. Denny. 1984. The transfer of lead by freshwater snails in Ullswater, Cumbria. Environ. Pollut. 35A:299-314.

Everard, M., and P. Denny. 1985. Flux of lead in submerged plants and its relevance to a freshwater system. Aquatic Bot. 21:181-193.

Fantin, A.M.B., A. Franchini, E. Ottaviani, and L. Benedetti. 1985. Effect of pollution on some freshwater species. II. Bioaccumulation and toxic effects of experimental lead pollution on the ganglia in Viviparus ater (Mollusca, Gastropoda). Basic Appl. Histochem. 29:377-387.

Fayed, S.I., and H.I. Abd-El-Shafy. 1985. Accumulation of Cu, Zn, Cd, and Pb by aquatic macrophytes. Environ. Int. 11:77-87.

Washington, D.C.

Franson, J.C., L. Sileo, O.H. Pattee, and J.F. Moore. 1983. Effects of chronic dietary lead in American kestrels (Falco sparverius). J. Wildl. Dis. 19:110-113.

Frape, D.L., and J.D. Pringle. 1984. Toxic manifestations in a dairy herd consuming haylage contaminated by lead. Vet. Rec. 114:615-616.

Fraser, J. 1980. Acclimation to lead in the freshwater isopod Asellus aquaticus. Oecologia 45:419-420.

Friedland, A.J., and A.H. Johnson. 1985. Lead distribution and fluxes in a high-elevation forest in northern Vermont. J. Environ. Qual. 14:332-336.

Friend, M. 1985. Interpretation of criteria commonly used to determine lead poisoning problem areas. U.S. Fish Wildl. Serv., Fish Wildl. Leafl. 2. 4 pp.

FWS. 1986. Migratory bird hunting; availability of a final supplemental environmental impact statement (SEIS) on the use of lead shot for hunting migratory birds in United States. Federal Register 51(124):23443-23447.

FWS. 1986a. Use of lead shot for hunting migratory birds in the United States. Final supplemental environmental impact statement. 535 + xx pp. Avail. from U.S. Fish Wildlife Service, Office of Migratory Bird Management, Washington, D.C. 20240.

FWS. 1987. Migratory bird hunting; zones in which lead shot will be prohibited for the taking of waterfowl, coots and certain other species in the 1987-88 hunting season. Federal Register 52(139):27352-27368.

Gale, N.L., E. Bolter, and B.G. Wixson. 1976. Investigation of Clearwater Lake as a potential sink for heavy metals from lead mining in southeast Missouri. Pages 95-106 in D.D. Hemphill (ed.). Trace substances in environmental health. Vol. X. Univ. Missouri, Columbia.

Geist, C.R., S.W. Balko, M.E. Morgan, and R. Angiak. 1985. Behavioral effects following rehabilitation from postnatal exposure to lead acetate. Percep. Motor Skills 60:527-536.

Getz, L.L., L.B. Best, and M. Prather. 1977a. Lead in urban and rural song birds. Environ. Pollut. 12:235-239.

Getz, L.L., L. Verner, and M. Prather. 1977b. Lead concentrations in small mammals living near highways. Environ. Pollut. 13:151-157.

Getz, L.L., A.W. Haney, R.W. Larimore, J.W. McNurney, H.V. Leland, P.W.

Hall, R.A., E.G. Zook, and G.M. Meaburn. 1978. National Marine Fisheries Service survey of trace elements in the fishery resource. U.S. Dep. Commerce, NOAA Tech. Rep. NMFS SSRF-721. 313 pp.

Hall, S.L., and F.M. Fisher, Jr. 1985. Lead concentrations in tissues of marsh birds: relationship of feeding habits and grit preference to spent shot ingestion. Bull. Environ. Contam. Toxicol. 35:1-8.

Hamir, A.N. 1981. Lead poisoning of dogs in Australia. Veety Rec. 108:438-439.

Hamir, A.N., N.D. Sullivan, and P.D. Handson. 1982. The effects of age and diet on the absorption of lead from the gastrointestinal tract of dogs. Aust. Vet. J. 58:266-268.

Hardisty, M.W., S. Kartar, and M. Sainsbury. 1974. Dietary habits and heavy metal concentrations in fish from the Severn Estuary and Bristol Channel. Mar. Pollut. Bull. 5:61-63.

Harrison, P.D., and M.I. Dyer. 1984. Lead in mule deer forage in Rocky Mountain National Park, Colorado. J. Wildl. Manage. 48:510-517.

Harrison, R.M., W.R. Johnston, J.C. Ralph, and S.J. Wilson. 1985. The budget of lead, copper and cadmium for a major highway. Sci. Total Environ. 46:137-145.

Harrison, R.M., and D.P.H. Laxen. 1981. Lead pollution. Causes and control. Chapman and Hall, New York. 168 pp.

Hastings, L., H. Zenick, P. Succop, T.J. Sun, and R. Sekeres. 1984. Relationship between hematopoietic parameters and behavorial measures in lead-exposed rats. Toxicol. Appl. Pharmacol. 73:416-422.

Haux, C., and A. Larrson. 1982. Influence of inorganic lead on the biochemical blood composition in the rainbow trout, Salmo gairdneri. Ecotoxicol. Environ. Saf. 6:28-34.

Haux, C., A. Larsson, G. Lithner, and M.L. Sjobeck. 1986. A field study of physiological effects on fish in lead-contaminated lakes. Environ. Toxicol. Chem. 5:283-288.

Hayashi, M. 1983. Lead toxicity in the pregnant rat. I. The effect of high-level lead on δ-aminolevulinic acid dehydratase activity in maternal and fetal blood or tissues. Environ. Res. 30:152-160.

Hejtmancik, M.R., Jr., E.B. Dawson, and B.J. Williams. 1982. Tissue distribution of lead in rat pups nourished by lead-poisoned mothers. J. Toxicol. Environ. Health 9:77-86.

Hong, J.S., H.A. Tilson, P. Hudson, S.F. Ali, W.E. Wilson, and V. Hunter. 1983. Correlation of neurochemical and behavorial effects of triethyl lead chloride in rats. Toxicol. Appl. Pharmacol. 69:471-479.

Hopkin, S.P., G.N. Hardisty, and M.H. Martin. 1986. The woodlouse Porcellio scaber as a 'biological indicator' of zinc, cadmium, lead and copper pollution. Environ. Pollut. 11B:271-290.

Hopkin, S.P., and M.H. Martin. 1984. Assimilation of zinc, cadmium, lead and copper by the centipede Lithobius variegatus (Chilopoda). J. Appl. Ecol. 21:535-546.

Hopkins, A. 1970. Experimental lead poisoning in the baboon. Brit. J. Indust. Med. 27:130-140.

Howard, D.R., and R.A. Braum. 1980. Lead poisoning in a dairy herd. Annu. Proc. Am. Assoc. Vet. Lab. Diag. 23:53-58.

Hudson, R.H., R. K. Tucker, and M.A. Haegele. 1984. Handbook of toxicity of pesticides to wildlife. U.S. Fish Wildl. Serv. Resour. Publ. 153. 90 pp.

Hunter, B., and G. Wobeser. 1980. Encephalopathy and peripheral neuropathy in lead-poisoned mallard ducks. Avian Dis. 24:169-178.

Ireland, M.P. 1977. Lead retention in toads Xenopus laevis fed increasing levels of lead in contaminated earthworms. Environ. Pollut. 12:85-92.

Irmer, U., I. Wachholz, H. Schafer, and D.W. Lorch. 1986. Influence of lead on Chlamydomonas reinhardii Danegard (Volvocales, Chlorophyta): accumulation, toxicity and ultrastructural changes. Environ. Exper. Bot. 26:97-105.

Jacobson, E., J. W. Carpenter, and M. Novilla. 1977. Suspected lead toxicosis in a bald eagle. J. Am. Vet. Med. Assoc. 171:952-954.

Janssen, D.L., J.E. Oosterhuis, J.L. Allen, M.P. Anderson, D.G. Kelts, and S.N. Wiemeyer. 1986. Lead poisoning in free-ranging California condors. J. Am. Vet. Med. Assoc. 189:1115-1117.

Jenkins, D.W. 1980. Biological monitoring of trace metals. Vol. 2. Toxic trace metals in plants and animals of the world. Part II. U.S. Environ. Protection Agency Rep. 600/3-80-091:619-778.

Johansson-Sjobeck, M-J., and A. Larsson. 1979. Effects of inorganic lead on delta-aminolevulinic acid dehydratase activity and hematological variables in the rainbow trout, Salmo gairdnerii. Arch. Environ. Contam. Toxicol. 8:419-431.

lead concentrations in tissues of adult male ringed turtle doves that ingested lead shot. J. Toxicol. Environ. Health 8:649-658.

Kimmel, C.A., L.D. Grant, C.S. Sloan, and B.C. Gladen. 1980. Chronic low-level lead toxicity in the rat. I. Maternal toxicity and perinatal effects. Toxicol. Appl. Pharmacol. 56:28-41.

King, K.A., and E. Cromartie. 1986. Mercury, cadmium, lead, and selenium in three waterbird species nesting in Galveston Bay, Texas, USA. Colon. Waterbirds 9:90-94.

Kirby, R.E., H.H. Obrecht III, and M.C. Perry. 1983. Body shot in Atlantic brant. J. Wildl. Manage. 47:527-530.

Kisseberth, W.C., J.P. Sundberg, R.W. Nyboer, J.D. Reynolds, S.C. Kasten, and V.R. Beasley. 1984. Industrial lead contamination of an Illinois wildlife refuge and indigenous small mammals. J. Am. Vet. Med. Assn. 185:1309-1313.

Knight, H.D., and R.G. Burau. 1973. Chronic lead poisoning in horses. J. Am. Vet. Med. Assoc. 162:781-786.

Knowlton, M.F., T.P. Boyle, and J.R. Jones. 1983. Uptake of lead from aquatic sediment by submersed macrophytes and crayfish. Arch. Environ. Contam. Toxicol. 12:535-541.

Kobayashi, N. 1971. Fertilized sea urchin eggs as an indicatory material for marine pollution bioassay, preliminary experiments. Publ. Seto Mar. Biol. Lab. (Japan) 18:379-406.

Kober, T.E., and G.P. Cooper. 1976. Lead competitively inhibits calcium-dependent synoptic transmission in the bullfrog sympathetic ganglion. Nature (Lond). 262:704-705.

Krishnayya, N.S.R., and S.J. Bedi. 1986. Effect of automobile lead pollution in Cassia tora L. and Cassia occidentalis L. Environ. Pollut. 40A:221-226.

Krajnovic-Ozretic, M., and B. Ozretic. 1980. The ALA-D activity test in lead exposed grey mullet Mugil auratus. Mar. Ecol. Prog. Ser. 3:187-191.

Kumar, S., and S.C. Pant. 1984. Comparative effects of the sublethal poisoning of zinc, copper and lead on the gonads of the teleost Puntius conchonius Ham. Toxicol. Lett. 23:189-194.

Kwatra, M.S., B.S. Gill, R. Singh, and M. Singh. 1986. Lead toxicosis in buffaloes and cattle in Punjab. Indian J. Anim. Sci. 56:412-413.

sediment-bound lead to the estuarine bivalve Scrobicularia plana. J. Mar. Biol. Assoc. U.K. 58:793-802.

Macdonald, J.W., C.J. Randall, H.M. Ross, G.M. Moon, and A.D. Ruthven. 1983. Lead poisoning in captive birds of prey. Vet. Rec. 113:65-66.

Mackay, D.W., W. Halcrow, and I. Thornton. 1972. Sludge dumping in the Firth of Clyde. Mar. Pollut. Bull. 3:7-10.

Maddock, B.G., and D. Taylor. 1980. The acute toxicity and bioaccumulation of some lead alkyl compounds in marine animals. Pages 233-261 in M. Branica and Z. Konrad (eds.). Lead in the marine environment. Pergamon Press, Oxford, England.

Mahaffey, K.R., J.I. Rader, J.M. Schaefer, and S.N. Kramer. 1980. Comparative toxicity to rats of lead acetate from food or water. Bull. Environ. Contam. Toxicol. 25:541-546.

Marchetti, R. 1978. Acute toxicity of alkyl leads to some marine organisms. Mar. Pollut. Bull. 9:206-207.

Marcus, A.H. 1985. Multicompartment kinetic models for lead. I. Bone diffusion models for long-term retention. Environ. Res. 36:441-458.

Martin, W.E., and P.R. Nickerson. 1973. Mercury, lead, cadmium, and arsenic residues in starlings - 1971. Pestic. Monitor. J. 7:67-72.

Massaro, T.F., G.D. Miller, and E.J. Massaro. 1986. Low-level lead exposure affects latent learning in the rat. Neurobehav. Toxicol. Teratol. 8:109-113.

May, T.W., and G.L. McKinney. 1981. Cadmium, lead, mercury, arsenic, and selenium concentrations in freshwater fish, 1976-77 - National Pesticide Monitoring Program. Pestic. Monitor. J. 15:14-38.

McDonald, L.J. 1986. Suspected lead poisoning in an Amazon parrot. Can. Vet. J. 27:131-134.

McLean, R.O., and A.K. Jones. 1975. Studies of tolerance to heavy metals in the flora of the rivers Ystwyth and Clarach, Wales. Freshwater Biol. 5:431-444.

Melhuus, A., K.L. Seip, H.M. Seip, and S. Mykelstad. 1978. A preliminary study of the use of benthic algae as biological indicators of heavy metal pollution in Sorfjorden, Norway. Environ. Pollut. 15:101-107.

Mierau, G.W., and B.E. Favara. 1975. Lead poisoning in roadside populations of deer mice. Environ. Pollut. 8:55-64.

Osweiler, G.D., and G.A. Van Gelder. 1978. Epidemiology of lead poisoning in animals. Pages 143-177 in F.W. Oehme (ed.). Toxicity of heavy metals in the environment. Part 1. Marcel Dekker, New York.

Ozoh, P.T.E. 1980. Effect of lead on pigment pattern formation in zebrafish (Brachydanio rerio). Bull. Environ. Contam. Toxicol. 24:276-282.

Pain, D. J. 1987. Lead poisoning in waterfowl: an investigation of sources and screening techniques. Ph.D. thesis, Oxford University, England. 335 pp.

Pain, D. J., and B. A. Rattner. 1988. Mortality and hematology associated with the ingestion of one number four lead shot in black ducks, Anas rubripes. Bull. Environ. Contam. Toxicol. 40:159-164.

Pattee, O.H. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. Arch. Environ. Contam. Toxicol. 13:29-34.

Pattee, O.H., and S.K. Hennes. 1983. Bald eagles and waterfowl: the lead shot connection. Trans. N. Am. Wildl. Nat. Resour. Conf. 48:230-237.

Pattee, O.H., S.N. Wiemeyer, B.M. Mulhern, L. Sileo, and J.W. Carpenter. 1981. Experimental lead-shot poisoning in bald eagles. J. Wildl. Manage. 45:806-810.

Perry, M. C., and J. W. Artmann. 1979. Incidence of embedded and ingested shot in oiled ruddy ducks. J. Wildl. Manage. 43:266-269.

Perry, M. C., and P. H. Geissler. 1980. Incidence of embedded shot in canvasbacks. J. Wildl. Manage. 44:888-894.

Peter, F., and G. Strunc. 1983. Effect of ingested lead on concentration of blood and tissue lead in rabbits. Clin. Biochem. 16:202-205.

Prause, B., E. Rehm, and M. Schulz-Baldes. 1985. The remobilization of Pb and Cd from contaminated dredge spoil after dumping in the marine environment. Environ. Technol. Lett. 6:261-266.

Rai, R., and M.A. Qayyum. 1984. Haematological responses in a freshwater fish to lead poisoning. J. Environ. Biol. 5:53-56.

Raymond, R.B., and R.B. Forbes. 1975. Lead in hair of urban and rural small mammals. Bull. Environ. Contam. Toxicol. 13:551-553.

Redig, P.T., C.M. Stowe, D.M. Barnes, and T.D. Arent. 1980. Lead toxicosis in raptors. J. Am. Vet. Med. Assoc. 177:941.

poisoning in waterfowl. Illinois Nat. Hist. Surv., Spec. Publ. 4. 34 pp.

Schlick, E., K. Mengel, and K.D. Friedberg. 1983. The effect of low lead doses in vitro and in vivo on the d -ala- d activity of erythrocytes, bone marrow cells, liver and brain of the mouse. Arch. Toxicol. 53:193-205.

Schmitt, C.J., F.J. Dwyer, and S.E. Finger. 1984. Bioavailability of Pb and Zn from mine tailings as indicated by erythrocyte δ-aminolevulinic acid dehydratase (ALA-D) activity in suckers (Pisces: Catostomidae). Can. J. Fish. Aquat. Sci. 41:1030-1040.

Schmitt, C. J., and S. E. Finger. 1987. The effects of sample preparation on measured concentrations of eight elements in edible tissues of fish from streams contaminated by lead mining. Arch. Environ. Contam. Toxicol. 16:185-207.

Schulz-Baldes, M. 1972. Toxicity and accumulation of lead in the common mussel Mytilus edulis in laboratory experiment. Mar. Biol. 16:226-229.

Schulz-Baldes, M. 1974. Lead uptake from sea water and food, and lead loss in the common mussel, Mytilus edulis. Mar. Biol. 25:177-193.

Schulz-Baldes, M., and R.A. Lewin. 1976. Lead uptake in two marine phytoplankton organisms. Biol. Bull. 150:118-127.

Scoullos, M.J. 1986. Lead in coastal sediments: the case of the Elefsis Gulf, Greece. Sci. Total Environ. 49:199-219.

Seeliger, U., and P. Edwards. 1977. Correlation coefficients and concentration factors of copper and lead in seawater and benthic algae. Mar. Pollut. Bull. 8:16-19.

Settle, D. M., and C. C. Patterson. 1980. Lead in albacore: guide to lead pollution in Americans. Science 207:1167-1176.

Sharma, S., and K.C. Kanwar. 1985. Reproductive performance in mice following lead administration. Res. Bull. Panjab Univ. 36:389-394.

Sheppard, C.R.C., and D.J. Bellamy. 1974. Pollution of the Mediterranean around Naples. Mar. Pollut. Bull. 5:42-44.

Sileo, L., and W.N. Beyer. 1985. Heavy metals in white-tailed deer living near a zinc smelter in Pennsylvania. J. Wildl. Dis. 21:289-296.

Sileo, L., and S. I. Fefer. 1987. Paint chip poisoning of Laysan albatross at Midway Atoll. J. Wildl. Dis. 23:432-437.

Sirota, G.R., and J.F. Uthe. 1977. Determination of tetraalkyl lead

Street, M. 1983. The assessment of mortality resulting from the ingestion of spent lead shot by mallard wintering in South East England. Congr. Int. Fauna Cinegetica y Silvestre 15 (1981):161-167.

Sundstrom, R., K. Muntzing, H. Kalimo, and P. Sourander. 1985. Changes in the integrity of the blood-brain barrier in suckling rats with low dose lead encephalopathy. Acta Neuropathol. 68:1-9.

Szymczak, M.R., and W.J. Adrian. 1978. Lead poisoning in Canada geese in southeast Colorado. J. Wildl. Manage. 42:299-306.

Tachon, P., A. Laschi, J.P. Briffaux, and G. Brain. 1983. Lead poisoning in monkeys during pregnancy and lactation. Sci. Total Environ. 30:221-229.

Ter Haar, G.I. 1970. Air as a source of lead in edible crops. Environ. Sci. Technol. 4:226-229.

Tsuchiya, K. 1979. Lead. Pages 451-484 in L. Friberg, G.E. Nordberg, and V.B. Vouk (eds.). Handbook on the toxicology of metals. Elsevier/North Holland Biomedical Press, Amsterdam.

Turner, R.S., A.H. Johnson, and D. Wang. 1985. Biogeochemistry of lead in McDonalds Branch watershed, New Jersey Pine Barrens. J. Environ. Qual. 14:305-314.

Udevitz, M.S., C.A. Howard, R.J. Robel, and B. Curnutte, Jr. 1980. Lead contamination in insects and birds near an interstate highway, Kansas. Environ. Entomol. 9:35-36.

Varanasi, U., and D.J. Gmur. 1978. Influence of water-borne and dietary calcium on uptake and retention of lead by coho salmon (Oncorhynchus kisutch). Toxicol. Appl. Pharmacol. 46:65-75.

Veit, H.P., R.J. Kendall, and P.F. Scanlon. 1983. The effect of lead shot ingestion on the testes of adult ringed turtle doves (Streptopelia risoria). Avian Dis. 27:442-452.

Vighi, M. 1981. Lead uptake and release in an experimental trophic chain. Ecotoxicol. Environ. Safety 5:177-193.

Walsh, D.F., B.L. Berger, and J.R. Bean. 1977. Mercury, arsenic, lead, cadmium, and selenium residues in fish, 1971-73 - National Pesticide Monitoring Program. Pestic. Monitor. J. 11:5-34.

Walsh, T.J., and H.A. Tilson. 1984. Neurobehavorial toxicology of the organoleads. Neurotoxicology 5:67-86.

Ward, N.I., and R.R. Brooks. 1979. Lead levels in wool as an indication of

Yeung, G.L. 1978. The influence of lead, an environmental pollutant on metamorphosis of Rana utricularia (Amphibia: Ranidae). Arkansas Acad. Sci. Proc. 32:83-86.

Zaroogian, G.E., G. Morrison, and J.F. Heltshe. 1979. Crassostrea virginica as an indicator of lead pollution. Mar. Biol. 52:189-196.

Zirkin, B.R., R. Gross, and L.L. Ewing. 1985. Effects of lead acetate on male rate reproduction. Pages 138-145 in F. Homburger and A.M. Goldberg (eds.). Concepts in toxicology. Vol. 3. In vitro embryotoxicity and teratogenicity tests. Karger, Basel, Switzerland.

Zmudzki, J., G.R. Bratton, C. Womac, and L. Rowe. 1983. Lead poisoning in cattle: reassessment of the minimum toxic oral dose. Bull. Environ. Contam. Toxicol. 30:435-441.

Zmudzki, J., G.R. Bratton, C. Womac, and L.D. Rowe. 1984. The influence of milk diet, grain diet, and method of dosing on lead toxicity in young calves. Toxicol. Appl. Pharmacol. 76:490-497.

Zook, B.C., R.M. Sauer, and F.M. Garner. 1972. Lead poisoning in captive wild animals. J. Wildl. Dis. 8:264-272.

Zwank, P.J., V.L. Wright, P.M. Shealy, and J.D. Newsom. 1985. Lead toxicosis in waterfowl on two major wintering areas in Louisiana. Wildl. Soc. Bull. 13:17-26.

☆U.S. GOVERNMENT PRINTING OFFICE: 1986-574-765/85103

50272-101

REPORT DOCUMENTATION PAGE	1. REPORT NO. Biological Report 85(1.14)	2.	3. Recipient's Accession No.

4. Title and Subtitle
Lead hazards to fish, wildlife, and invertebrates: a synoptic review

5. Report Date
April 1988

6.

7. Author(s)
Ronald Eisler

8. Performing Organization Rept. No.

9. Performing Organization Name and Address
U.S. Fish and Wildlife Service
Patuxent Wildlife Research Center
Laurel, MD 20708

10. Project/Task/Work Unit No.

11. Contract(C) or Grant(G) No.
(C)
(G)

12. Sponsoring Organization Name and Address

13. Type of Report & Period Covered

14.

15. Supplementary Notes

16. Abstract (Limit: 200 words)

Ecological and toxicological aspects of lead (Pb) in the environment are briefly reviewed, with emphasis on fish and wildlife, their predators, and prey. Subtopics include sources and uses, chemical properties, mode of action, background concentrations, lethal and sublethal effects, and current recommendations for the protection of sensitive living resources. Resources that are at increased risk from Pb include migratory waterfowl that congregate at heavily hunted staging areas and ingest shot, avian predators that consume hunter-wounded game, domestic livestock near smelters and Pb battery recycling plants, zoo animals and livestock held in enclosures coated with Pb-based paints, wildlife that forage near heavily traveled roads, aquatic life near mining activities and areas of Pb pesticide use or aerosol fallout, and crops and terrestrial invertebrates growing or living in Pb-contaminated soils. Recent legislation limiting the content of Pb in paints, reducing the Pb content in gasoline, and (by 1991-1992) eliminating the use of Pb shot will substantially reduce environmental burdens of Pb.

17. Document Analysis a. Descriptors

Fish
Birds
Mammals
Contaminants
Natural Resources
Invertebrates
Plants
Toxicity

b. Identifiers/Open-Ended Terms

Lead
Heavy metals
Metabolism
Lead shot
Hunting
Regulations
Organoleads
Sublethal effects

c. COSATI Field/Group

18. Availability Statement
Released unlimited

19. Security Class (This Report)
Unclassified

21. No. of Pages
134

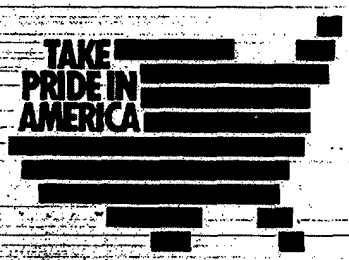
20. Security Class (This Page)
Unclassified

22. Price

(See ANSI-Z39.18) See Instructions on Reverse

OPTIONAL FORM 272 (4-77)
(Formerly NTIS-35)
Department of Commerce

DUP040008017

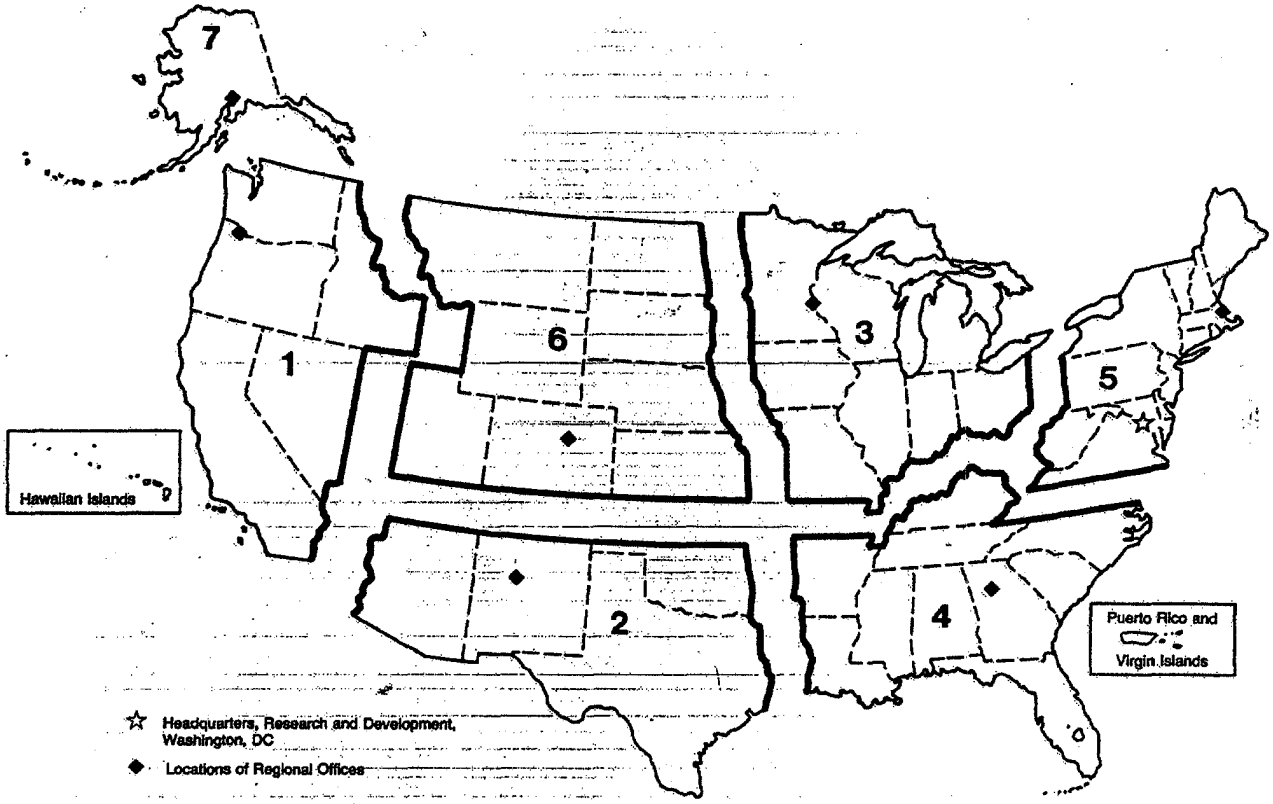


Preserve Our Natural Resources

DEPARTMENT OF THE INTERIOR
U.S. FISH AND WILDLIFE SERVICE

As the Nation's principal conservation agency, the Department of the Interior has responsibility for most of our nationally owned public lands and natural resources. This includes fostering the wisest use of our land and water resources, protecting our fish and wildlife, preserving the environmental and cultural values of our national parks and historical places, and providing for the enjoyment of life through outdoor recreation. The Department assesses our energy and mineral resources and works to assure that their development is in the best interests of all our people. The Department also has a major responsibility for American Indian reservation communities and for people who live in island territories under U.S. administration.

DUP040008018



REGION 1
Regional Director
U.S. Fish and Wildlife Service
Lloyd Five Hundred Building, Suite 1692
500 N.E. Multnomah Street
Portland, Oregon 97232

REGION 2
Regional Director
U.S. Fish and Wildlife Service
P.O. Box 1306
Albuquerque, New Mexico 87103

REGION 3
Regional Director
U.S. Fish and Wildlife Service
Federal Building, Fort Snelling
Twin Cities, Minnesota 55111

REGION 4
Regional Director
U.S. Fish and Wildlife Service
Richard B. Russell Building
75 Spring Street, S.W.
Atlanta, Georgia 30303

REGION 5
Regional Director
U.S. Fish and Wildlife Service
One Gateway Center
Newton Corner, Massachusetts 02158

REGION 6
Regional Director
U.S. Fish and Wildlife Service
P.O. Box 25486
Denver Federal Center
Denver, Colorado 80225

REGION 7
Regional Director
U.S. Fish and Wildlife Service
1011 E. Tudor Road
Anchorage, Alaska 99503