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

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SUMMARY

Cadmium contamination of the environment is especially severe in the vicinity of smelters and urban industrialized areas. There is no evidence that cadmium, a relatively rare heavy metal, is biologically essential or beneficial; on the contrary, cadmium is a known teratogen and carcinogen, a probable mutagen, and has been implicated as the cause of severe deleterious effects on fish and wildlife. The freshwater biota is the most sensitive group; concentrations of 0.8 to 9.9 ug Cd/L (ppb) in water were lethal to several species of aquatic insects, crustaceans, and teleosts, and concentrations of 0.7 to 570 ppb were associated with sublethal effects such as decreased growth, inhibited reproduction, and population alterations. These effects were most pronounced in waters of comparatively low alkalinity. Marine organisms were more resistant than freshwater biota. Decapod crustaceans, the most sensitive saltwater group, died at concentrations of cadmium in seawater ranging from 14.8 to 420 ppb. Sublethal effects to marine animals recorded at Cd concentrations of 0.5 to 10 ppb included decreased growth, respiratory disruption, altered enzyme levels, and abnormal muscular contractions; effects were usually most obvious at relatively low salinities and high temperatures. Freshwater and marine aquatic organisms accumulated measurable amounts of cadmium from water containing Cd concentrations not previously considered hazardous to public health or to many species of aquatic life; i.e., 0.02 to 10 ppb.

Mammals and birds are comparatively resistant to the biocidal properties of cadmium. The lowest oral doses producing death in rats and guinea pigs ranged from 150 to 250 mg Cd/kg body weight (ppm). Although mallards and chickens tolerated 200 ppm of cadmium in diets for protracted periods, kidney cadmium exceeded 130 ppm fresh weight under this regimen, a concentration considered life-threatening to some organisms. Sublethal effects of cadmium in birds, which were similar to those in other animals, included growth retardation, anemia, and testicular damage; however, these effects were observed at higher concentrations than in aquatic biota. Although the evidence is incomplete, wildlife populations, especially migratory birds that feed on crops growing on fields fertilized with municipal sewage sludges, may be exposed to considerable risk of harmful effects from cadmium.

It is now conservatively estimated that adverse effects on fish or wildlife are either pronounced or probable when cadmium concentrations exceed 3 ppb in fresh water, 4.5 ppb in saltwater, 100 ppb in the diet, or 100 g Cd/m^3 in air. Cadmium residues in vertebrate kidney or liver that exceed 10 ppm fresh weight or 2 ppm whole body fresh weight should be viewed as evidence of probable Cd contamination; residues of 200 ppm fresh weight kidney, or more than 5 ppm whole animal fresh weight, are probably life-threatening to the organism. (Eisler, R. 1985. Cadmium hazards to fish, wildlife , and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85 (1.2), Contaminant Hazard Reviews Report No. 2. 46 pp.)

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INTRODUCTION

There is no evidence that cadmium (Cd) is biologically essential or beneficial; on the contrary, it has been implicated as the cause of numerous human deaths and various deleterious effects in fish and wildlife. In sufficient concentration, it is toxic to all forms of life, including microorganisms, higher plants, animals, and man. It is a relatively rare metal, usually present in small amounts in zinc ores, and is commercially obtained as an industrial by-product of the production of zinc, copper, and lead. Major uses of cadmium are in electroplating, in pigment production, and in the manufacture of plastic stabilizers and batteries. Anthropogenic sources of cadmium include smelter fumes and dusts, the products of incineration of Cd-bearing materials and fossil fuels, fertilizers, and municipal wastewater and sludge discharges; concentrations are most likely highest in the localized regions of smelters or in urban industrialized areas (Hammons et al. 1978; Hutton 1983b). Industrial consumption of cadmium in the United States, estimated at 6,000 metric tons in 1968, is increasing; projected use in the year 2000 is about 14,000 tons, primarily for electroplating of motor parts and in the manufacture of batteries. The cadmium load in soils and terrestrial biota in other industrialized countries also appears to be increasing; it is currently of great concern in Scandinavia (Tjell et al. 1983), Germany (Markard 1983), and the United Kingdom (Hutton 1983a).

This account summarizes ecological and toxicological aspects of cadmium in the environment, with special reference to game fish and migratory waterfowl and their predators and prey. It also provides recommendations for the protection of sensitive species of wildlife and aquatic biota. It is part of a continuing series of synoptic reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

ENVIRONMENTAL CHEMISTRY AND BACKGROUND RESIDUES

Cadmium is a silver-white, blue-tinged, lustrous metal that melts at 321°C and boils at 765°C. This divalent element has an atomic weight of 112.4 and an atomic number of 48. It is insoluble in water, although its chloride and sulphate salts are freely soluble (Windholz et al. 1976). The availability of Cd to living organisms from their immediate physical and chemical environs depends on numerous factors, including adsorption and desorption rates of cadmium from terrigenous materials, pH, Eh, chemical speciation, and many other modifiers. The few selected examples that follow demonstrate the complex behavior of Cd in freshwater systems.

Adsorption and desorption processes are likely to be major factors in controlling the concentration of cadmium in natural waters and tend to counteract changes in the concentration of cadmium ions in solution (Gardiner 1974). Adsorption and desorption rates of cadmium are rapid on mud solids and particles of clay, silica, humic material, and other naturally occurring solids. Concentration factors for river muds varied between 5,000 and 500,000 and depended mainly on the type of solid, the particle size, the concentration of cadmium present, the duration of contact, and the concentration of complexing ligands; humic material appeared to be the main component of river mud responsible for adsorption (Gardiner 1974). Changes in physicochemical conditions, especially pH and redox potential, that occur during dredging and disposal of Cd-polluted sediments may increase chemical mobility and, hence, bioavailability of sediment-bound Cd (Khalid et al. 1981). For example, cadmium in Mississippi River sediments spiked with radiocadmium was transformed from potentially available organic forms to more mobile and readily available dissolved and exchangeable forms (i.e., increased bioavailability) under regimens of comparatively acidic pH and high oxidation (Khalid et al. 1981). The role of dissolved oxygen and aquatic plants on Cd cycling was studied in Palestine lake, a 92-ha eutrophic lake in Kosciusko County, Indiana, a long-term recipient of cadmium and other waste metals from an electroplating plant. The maximum recorded concentration of dissolved Cd in the water column was 17.3 ppb; for suspended particulates, it was 30.3 ppb (Shephard et al. 1980). During anaerobic conditions in the lake's hypolimnion, a marked decrease in the dissolved fraction and a corresponding increase in the suspended fraction were noted. The dominant form of cadmium was free, readily bioavailable, cadmium ion, Cd^{2+}; however, organic complexes of Cd, which are comparatively nonbioavailable, made up a significant portion of the total dissolved Cd. Cadmium levels in sediments of Palestine Lake ranged from 1.5 ppm in an uncontaminated area of the lake to 805 ppm near the outlet of a metal-bearing ditch that entered the lake (McIntosh et al. 1978). The dominant form of Cd in sediments was a carbonate. Levels of Cd in water varied over time and between sites, but usually ranged from 0.5 to 2.5 ppb. It is possible that significant amounts of cadmium are transferred from the sediments into rooted aquatic macrophytes and later released into the water after macrophyte death (natural or herbicide-induced), particularly in heavily contaminated systems. In Palestine Lake, Cd levels in pondweed

(*Potomogeton crispus*), a rooted aquatic macrophyte, were about 90 ppm dry weight; a maximum burden of 1.5 kg was retained by the population of *P*. *crispus* in the lake (McIntosh et al. 1978). Release of the total amount could raise water concentrations by a maximum of 1 ppb. This amount was considered negligible in terms of the overall lake Cd budgets; however, it might have limited local effects. As judged by these and other complexities regarding Cd bioavailability, it appears that the organism remains the ultimate arbiter of its environment, regardless of the source of cadmium and its geophysical surroundings.

Background levels of cadmium in uncontaminated, nonbiological compartments extended over several orders of magnitude (Korte 1983). Concentrations (ppb) of cadmium reported ranged from 0.05 to 0.2 in freshwater, up to 0.05 in coastal seawater, from 0.01 to 0.1 in open ocean seawater, up to 5,000 in riverine and lake sediments, 30 to 1,000 in marine sediments, 10 to 1,000 in soils of nonvolcanic origin, up to 4,500 in soils of volcanic origin, 1 to 600 in igneous rock, up to 100,000 in phosphatic rock, and 0.001 to 0.005 $\mu g/m^3$ in air (Korte 1983). Where Cd is comparatively bioavailable, these values are very near those that have been shown to produce harmful effects in sensitive biological species, as will be discussed later.

Cadmium, unlike synthetic compounds, is a naturally occurring element, and its presence has been detected in more than 1,000 species of aquatic and terrestrial flora and fauna. Concentrations of cadmium in a few selected species of biota are shown in Table 1; more extensive documentation was presented by Hammons et al. (1978), NRCC (1979), Jenkins (1980), and Eisler (1981). At least six trends are evident from Table 1. First, marine biota generally contained significantly higher cadmium residues than their freshwater or terrestrial counterparts, probably because total cadmium levels are higher in seawater. Second, cadmium tends to concentrate in the viscera of vertebrates, especially the liver and kidneys. Third, concentrations of Cd are higher in older organisms than in younger stages; this relationship is especially pronounced in carnivores and marine vertebrates (Eisler 1984). Fourth, higher concentrations reported for individuals of a single species collected at several locations are almost always associated with proximity to industrial and urbanized areas or to point source discharges of Cd-containing wastes. Fifth, background levels of cadmium in crops and other plants are usually <1.0 mg/kg (ppm). Little is known about the Cd concentrations required to reduce plant yields; however, plants growing in cadmium-contaminated soils contain abnormally high residues that may be detrimental to plant growth and to animal and human consumers. And finally, it is apparent from Table 1 that species analyzed, season of collection, ambient Cd levels, and sex of organism all probably modify Cd concentrations.

The relationship between reported tissue cadmium concentrations of “unstressed” populations and hazard to the organism or its consumer is not well documented. For example, cadmium in eggs of successful nests of Cooper's hawks collected in Arizona and New Mexico ranged from 0.015 to 0.24 ppm fresh weight (FW); concentrations were higher in eggs from unsuccessful nests (Snyder et al. 1973). Cadmium concentrations in livers of breeding birds were higher in two declining colonies of puffins in St. Kilda and Clo Mor (12.9-22.3 ppm, dry weight) than in colonies of puffins from other areas, or in livers of other seabirds examined (Parslow et al. 1972); however, the link to cadmium requires elucidation. Among marine teleosts, whole body levels exceeding 5 ppm FW or 86 ppm ash weight (AW) in laboratory-stressed fish suggested that death would follow within 4 weeks (Eisler 1971). Marine bivalve molluscs occasionally contain more than 13 ppm of Cd in soft parts FW (Table 1), a level considered acutely toxic to human consumers (Zaroogian and Cheer 1976). The significance of cadmium residues to organism health is further developed later.

Table 1. Cadmium concentrations in field collections of selected species of flora and fauna. Values shown are in mg Cd/kg fresh weight (FW), dry weight (DW), or ash weight (AW).

Ecosystem, taxonomic group, organism, tissue, location, and other variables	Concentration (mg/kg or ppm)	Reference[a]
Marine		
Algae and Macrophytes		
Brown alga, *Ascophyllum nodosum*		
Whole		
Norway locations:		
Sorfjorden	6.0-15.0 DW	Melhuus et al. 1978
Eikhamrane	3.5-7.7 DW	Myklestad et al. 1978
Flak	<1.0 DW	
Transferred from Eikhamrane to Flak, 120 days	<1.0-4.0 DW	
Lofoten	<0.7 DW	Haug et al. 1974
Trondheimsfjord	<0.7-1.0 DW	
Hardangerfjord	0.7-16.0 DW	
United Kingdom locations		
Menai Straits	1.8 DW	Foster 1976
Dulas Bay	1.5 DW	
Bladder wrack, *Fucus vesiculosus*		
Whole		
Sorfjorden, Norway	8.6-10.6 DW	Melhuus et al. 1978
Tamar estuary, UK	1.8-9.0 DW	Bryan and Uysal 1978
Menai Straits, UK	2.1 DW	Foster 1976
Dulas Bay, UK	1.8 DW	
Irish Sea	1.4 DW	Preston et al. 1972
Severn estuary, UK	220.0 DW	Butterworth et al. 1972
Molluscs		
Sydney rock oyster, *Crassostrea commercialis*		
Soft parts	0.4-18.6 FW	Ratkowsky et al. 1974
Soft parts	0.1-1.0 FW	Mackay et al. 1975
Pacific oyster, *Crassostrea gigas*		
Soft parts	0.2-2.1 FW	Pringle et al. 1968
Soft parts	0.0-30.7 FW	Ratkowsky et al. 1974
Soft parts	1.1 FW	Kopfler and Mayer 1967
Soft parts	3.7-9.0 DW	Watling and Watling 1976
Red abalone, *Haliotis rufescens*		
Gill	4.0-10.0 DW	Anderlini 1974
Mantle	2.8-12.8 DW	
Digestive gland	183.0-1163.0 DW	
Foot	0.2-0.5 DW	
Periwinkle, *Littorina littorea*		
Soft parts	0.9-1.5 DW	Leatherland & Burton 1974
Soft parts	0.0-0.5 FW	Topping 1973
Soft parts	210.0 DW	Butterworth et al. 1972
Squid, *Ommastrephes bartrami*		
Liver	80.6-782.0 DW	Martin and Flegal 1975; Hamanaka et al. 1977
Muscle	0.7 DW	
Gonad	0.4 DW	

Common mussel, *Mytilus edulis*		
Soft parts		
U.S. West Coast	2.3-10.5 DW	Goldberg et al. 1978
U.S. East Coast	0.6-6.2 DW	
Port Phillip Bay, Australia	0.2-1.3 FW	Phillips 1976
Western Port Bay, Australia	up to 18.2 FW	
Scottish waters	0.1-2.0 FW	Topping 1973
Looe estuary, U.K.	0.8-2.6 DW	Bryan and Hummerstone 1977
Tasmania	5.5 FW	Eustace 1974
Corio Bay, Australia	2.0-63.0 DW	Talbot et al. 1976
Mussel, *Mytilus edulis planulatus*		
Soft parts		
Mean dry weight		
0.09 g	0.6 DW	Harris et al. 1979
0.39 g	0.8 DW	
0.48 g	1.1 DW	
0.69 g	1.3 DW	
Scallop, *Pecten maximum*		
Soft parts	13.0 DW	Segar et al. 1971
Muscle	1.9 DW	
Gut and digestive gland	96.0 DW	
Mantle and gills	3.2-17.0 DW	
Gonad	2.5 DW	
Shell	0.0 DW	
Soft parts	32.5 DW	Bryan 1973
Kidney	79.0 DW	
Kidney	54.0 DW	George et al. 1980
Kidney concretion	546.6 DW	Carmichael et al. 1979
Digestive gland	321.0 DW	Bryan 1973
Edible tissues	5.1-23.0 FW	Topping 1973
Giant scallop, *Placopecten magellanicus*		
Muscle		
March	up to 8.8 DW	Reynolds 1979
Rest of year	<3.7 DW	
Viscera		
March	104.1 DW max.	
August	121.2 DW max.	
February	161.8 DW max.	
June	105.3 DW max.	
Gonad	0.5-3.2 FW	Greig et al. 1978
Visceral mass	3.7-27.0 FW	
Clam, *Scrobicula plana*		
Digestive gland		
Gannel estuary, U.K.	39.8 DW	Bryan and Hummerstone 1978
Camel estuary, U.K.	1.7 DW	
Transferred from Camel to l Ganne estuary for 352 days	5.6 DW	
Transferred from Gannel to Camel estuary for 352 days	21.0 DW	
Whelk, *Thais lapillus*		
Soft parts	425.0 DW	Butterworth et al. 1972
Crustaceans		
Rock crab, *Cancer irroratus*		
Flesh	0.1-1.0 FW	Greig et al. 1977

Digestive gland	1.1-4.8 FW	
Gills	0.7-2.7 FW	
Brown shrimp, *Penaeus* sp.		
Flesh	0.2 DW	Horowitz and Presley 1977
Exoskeleton	0.5 DW	
Viscera	2.6 DW	
Whole	<0.4 DW	Sims and Presley 1976
American lobster, *Homarus americanus*		
Whole	0.5 FW; 5.3 AW	Eisler et al. 1972
Meats	0.2 FW; 10.0 AW	
Exoskeleton	0.6 FW; 4.1 AW	
Gill	0.5 FW; 17.2 AW	
Viscera	1.2 FW; 33.8 AW	
Prawn, *Pandalus montagui*		
Tail	0.0 DW	Ray et al. 1980
Egg	0.1 DW	
Carcass	0.3 DW	
Hepatopancreas	6.4 DW	
Whole	0.5 DW	
Spiny lobster, *Panulirus interruptus*		
Muscle	0.3 FW	Vattuone et al. 1976
Hepatopancreas	5.6-29.3 FW	
Grass shrimp, *Palaemonetes pugio*		
Whole	1.4-6.2 DW	Pesch and Stewart 1980
Annelids		
Marine worm, *Nephtys hombergi*		
Whole		
March	9.0 FW	Rosenberg 1977
October	89.0 FW	
Sandworm, *Nereis diversicolor*		
Whole	0.1-3.6 DW	Bryan and Hummerstone 1973, 1977
Echinoderms		
Asteroid, *Echinus esculentus*		
Intestines	8.9 DW	Riley and Segar 1970
Remaining tissues	<0.7 DW	
Fish		
Flounder, *Platichthys flesus*		
Whole		
Barnstaple Bay, U.K.		
Age II	1.1 DW	Hardisty et al. 1974
Age III	1.4 DW	
Age IV	1.6 DW	
Age V	1.7 DW	
Oldbury on Severn, U.K. (metals-contaminated area)		
Age II	4.0 DW	
Age III	4.5 DW	
Age IV	5.1 DW	
Age V	5.2 DW	
Yellowtail flounder, *Limanda limanda*		
Liver	0.4 DW	Westernhagen et al. 1980
Skin	0.2 DW	
Otoliths	0.2 DW	
Gills	0.2 DW	
Fin	0.2 DW	

Muscle	0.1 DW	
Backbone	0.05 DW	
Blue marlin, *Makaira indica*		
Muscle	0.1-0.4 FW	Mackay et al. 1976
Liver	0.2-83.0 FW	
Striped bass, *Morone saxatilis*		
Muscle	0.03 FW	Heit 1979
Liver	0.3 FW	
Atlantic cod, *Gadus morrhus*		
Roe	0.0-0.5 DW	Julshamn & Braekkan 1978
Muscle	0.02 DW	Julshamn & Braekkan 1975
Gonad	0.0-0.07 DW	
Liver	0.09 DW	
Bluefish, *Pomatomus saltatrix*		
Muscle	up to 0.08 FW	Bebbington et al. 1977
Shorthorn sculpin, *Myoxocephalus scorpius*		
Muscle	1.4 DW	Bohn and Fallis 1978
Liver	4.1 DW	
Birds		
Adelie penguin, *Pygoscelis adeliae*		
Liver	90.0 DW	Robertson et al. 1972
Lesser scaup, *Aythya affinis*		
Liver	0.6 FW	White et al. 1979
Kidney	2.3 FW	
New Zealand estuaries, 5 spp.		
Liver	0.1-1.5 FW	Turner et al. 1978
Kidney	0.1-14.8 FW	
Corpus Christi, Texas, 7 spp.		
Kidney	0.4-22.7 FW	White et al. 1980
Puffins, 2 spp.		
St. Kilda, Scotland		
Males		
Liver	14.6-29.4 DW	Bull et al. 1977
Kidney	67.0-133.0 DW	
Females		
Liver	14.1-39.9 DW	
Kidney	75.1-231.0 DW	
Sea gull, *Larus atricilla*		
Downy young		
Kidney	0.5 FW	Hulse et al. 1980
Other tissues	<0.05 FW	
Adults		
Muscle	0.1 FW	
Heart	0.1 FW	
Brain	0.5 FW	
Bone	0.4 FW	
Liver	0.6 FW	
Kidney	5.0 FW	
Common eider, *Somateria mollissima*		
Egg	1.0 DW	
Lande 1977		
Muscle	2.0 DW	
Liver	13.0 DW	
Kidney	25.0 DW	

Brown pelican, *Pelecanus occidentalis*		
Florida		
Liver	1.3-2.4 FW	Jenkins 1980
Muscle	0.2-0.3 FW	
California		
Liver	0.6-13.6 FW	
Muscle	0.2-0.4 FW	
Common tern, *Sterna hirundo*		
Liver	3.8 FW	
Kidney	21.3 FW	
Mammals		
Northern fur seal, *Callorhinus ursinus*		
Kidney	0.1-15.6 FW	Anas 1974
Liver	0.5-4.6 FW	
Pilot whale, *Globicephala macrorhynchus*		
Blubber	0.4-0.8 FW	Stoneburner 1978
Liver	11.3-19.0 FW	
Kidney	27.1-41.8 FW	
California sea lion, *Zalophus californianus*		
Liver	2.0-2.6 FW	Buhler et al. 1975
Kidney	10.2 FW	
Cerebellum	0.6 FW	
Other tissues	<0.2 FW	
Sea otter, *Enhydra lutris*		
Kidney	89.0-300.0 DW	Jenkins 1980
Walrus, *Odobenus rosmarus*		
Kidney	51.6 FW	
Liver	7.7 FW	
Muscle	0.3-0.7 FW	
Freshwater		
Macrophytes		
Water lily, *Nuphar luteum*		
Whole	0.5-1.8 DW	Jenkins 1980
Pondweed, *Potamogeton richardsoni*		
Leaf and stem	0.6-4.9 DW	
Root	1.3-6.7 DW	
Molluscs		
Clams, Illinois River		
Soft parts, 3 spp.	0.2-1.4 FW	Hammons et al. 1978
Annelids		
Whole, Illinois River	0.5-3.2 FW	
Fish		
United States, Nationwide, 1976-1977		
Whole	0.07 FW (0.01-1.04)	May and McKinney 1981
Upper Clark Fork River, western Montana		
Muscle, 3 spp.	0.2-0.6 FW	Hammons et al. 1978
Liver, 7 spp.	0.3-0.8 FW	
Great Lakes		
Whole, 3 spp.	0.0-0.14 FW	
Liver, 10 spp.	0.1-1.4 FW	
Illinois River		
Whole, 10 spp.	<0.08 FW	
New York State, various locations		
Whole		
Adirondacks region	0.02-0.05 FW	Lovett et al. 1972
Hudson River	up to 0.14 FW	

47 other areas	<0.02 FW	
Rainbow trout, *Salmo gairdneri*		
Alaska		
Whole	<0.07 FW	Jenkins 1980
Arizona		
Whole	<0.05 FW	
White crappie, *Pomoxis annularis*		
Whole	0.0-0.3 FW	
Sauger, *Stizostedion canadense*		
Whole	<0.05 FW	
Walleye, *Stizostedion vitreum vitreum*		
Liver	0.2 FW	
Whole	up to 0.16 FW	
Terrestrial		
Plants		
Lettuce, *Lactuca sativa,* whole		
Cd in soil, mg/kg		
<2.5	2.8 DW	Jenkins 1980
2.5	11.5 DW	
10.0	27.1 DW	
Soybean, *Glycine max*, plant top		
Cd in soil, mg/kg		
10	13.0 DW	
50	24.0 DW	
100	26.0 DW	
Tobacco, *Nicotiana tobacum*	2.0 DW	
Wheat, *Triticum aestivum*, grain		
Tons sewage sludge/hectare		
6.5	119.0 DW	
58.0	257.0 DW	
Control	<0.15 DW	
Annelids		
Earthworms, whole, 4 spp.		
distance from highway, meters		
3	12.6 DW	Gish and Christensen 1973
6.1	8.8 DW	
12.2	8.3 DW	
24.4	6.9 DW	
48.8	7.1 DW	
Control	3.0 DW	
Birds		
Ring-necked pheasant, *Phasianus colchicus*		
Liver	0.9 FW	Jenkins 1980
Kidney	7.4 FW	
American robin, *Turdus migratorius*		
Kidney	2.0 FW	
Liver	0.6 FW	
European starling, *Sturnus vulgaris*		
Whole, various U.S. locations		
Bakersfield, CA	0.24 FW	Martin and Nickerson 1973
Lansing, MI	0.12 FW	
Elkins, WV	0.12 FW	
Farmington, NM	0.12 FW	
Phoenix, AZ	0.11 FW	
Other U.S. areas	<0.05 FW	

Cooper's hawk, *Accipiter cooperii*		
Egg	0.12 (0.015-0.24 FW)	Snyder et al. 1973
Mammals		
Short-tailed shrew, *Blarina brevicauda*		
Liver	1.3 FW	Jenkins 1980
Whole	0.4 FW	
Cow, *Bos bovis*		
Distance from smelter		
Liver		
0.8 km	0.9 FW	
72.4 km	0.3 FW	
Kidney		
0.8 km	3.7 FW	
72.4 km	1.4 FW	
Coyote, *Canis latrans*		
Kidney	0.4 FW	
Elk, *Cervus* sp.		
Liver	1.5 DW	
Kidney	8.1 DW	
Muscle	0.6 DW	
Porcupine, *Erethizon dorsatum*		
Heart	0.4 FW	
Meadow vole, *Microtus pennsylvanicus*		
Collected from fields near Oxford, Ohio, receiving sewage sludge for 4 years at yearly rate of 8960 kg sludge/ha.		
Liver		
Adult males	0.8 FW	Maly and Barrett 1984
Adult females	3.1 FW	
Subadult males	1.2 FW	
Subadult females	1.1 FW	
Kidney		
Adult males	6.3 FW	
Adult females	19.1 FW	
Subadult males	3.5 FW	
Subadult females	6.2 FW	
From control fields		
Liver		
Adult males	0.7 FW	
Adult females	0.1 FW	
Subadult males	0.1 FW	
Subadult females	0.1 FW	
Kidney		
Adult males	0.3 FW	
Adult females	1.1 FW	
Subadult males	0.3 FW	
Subadult females	0.3 FW	
White-tailed deer, *Odocoileus virginianis*		
Kidney	0.7-11.7 FW	Jenkins 1980
Muscle	0.0-0.3 FW	
Liver	0.0-0.7 FW	
Gray squirrel, *Sciurus carolinensis*		
Kidney		
2 years old		
Urban area	15.9 FW	

Rural	2.0-4.6 FW
Red squirrel, *Sciurus hudsonicus*	
Kidney	7.8-17.4 FW
Liver	0.7-2.0 FW
Eastern cottontail, *Sylvilagus floridanus*	
Liver	up to 2.1 FW
Kidney	up to 13.5 FW
Muscle	up to 0.5 FW

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

ACUTE TOXICITY

A substantial toxicological data base for cadmium and freshwater biota demonstrates that ambient cadmium water concentrations exceeding 10 ppb are associated with high mortality, reduced growth, inhibited reproduction, and other adverse effects. Inasmuch as the current recommended drinking water criterion for human health protection is 10 ppb cadmium (EPA 1980), it is noteworthy that several species of freshwater aquatic insects, crustaceans, and teleosts exhibited significant mortality at cadmium concentrations of 0.8 to 9.9 ppb during exposures of 4 to 33 days; mortality generally increased as exposure time increased, water hardness decreased, and organism age decreased (Table 2).

Resistance to cadmium is higher in marine than in freshwater organisms; survival usually is higher at the lower temperatures and higher salinities for any given level of cadmium in the medium. Decapod crustaceans are the most sensitive marine group in short-term tests; LC-50 (96 h) values ranged from 320 to 420 ppb for the grass shrimp (*Palaemonetes vulgaris*), the hermit crab (*Pagurus longicarpus*), and the sand shrimp (*Crangon crangon*) (Eisler 1971). Studies of longer duration demonstrated that survival of shrimp groups was low at >250 ppb during 6 weeks of exposure and that hermit crab deaths were recorded at 60 ppb after 6 weeks, although some survivors remained at 10 weeks when the studies ended (Pesch and Stewart 1980). In another study, an LC-50 range of 14.8 to 19.5 ppb Cd was reported for two species of mysid shrimp subjected to “lifetime” (i.e., 23 to 27 days) exposure to cadmium salts (Gentile et al. 1982).

Birds are comparatively resistant to the biocidal properties of cadmium. Adult drake mallards (*Anas platyrhynchos*) fed up to 200 ppm cadmium in the diet for 90 days all survived with no loss of body weight (White and Finley 1978). Laying hens fed 200 ppm dietary Cd also survived; egg production was suppressed at that concentration but not at lower concentrations tested (White and Finley 1978). Marine and terrestrial animals, including ducks, have been shown to be particularly abundant in a wildlife community associated with a marine sewer outfall (Brown et al. 1977); these animals were contaminated with high levels of cadmium, as well as zinc and copper, but were apparently protected from the deleterious effects of high metal body burdens by metallothioneins. Amounts of these metal-binding proteinaceous metallothioneins and heavy metal loading appear to depend primarily on the degree of pollution and secondarily on the species of animal and its position in the food web. Ducks contained the highest levels of metallothioneins of all groups examined (Brown et al. 1977). Mammals are also comparatively resistant to cadmium. The lowest oral dose, in mg/kg body weight of cadmium (as fluroborate) producing death, was 250 in rats and 150 (as cadmium fluoride) in guinea pigs (EPA 1980).

Table 2. Lethal concentrations (LC) of cadmium to freshwater biota during various exposure intervals. Concentrations shown are in µg Cd/L (ppb) of medium fatal to 10% or 50% of test organisms.

Group, taxon, or life state	Water hardness, in mg $CaCO_3$/L	LC values, in ppb	Exposure interval	Reference[a]
Insects				
Ephemerella sp.	44-48	LC-50, <3.0	28 days	Spehar et al. 1978
Tanytarsus dissimilis	47	LC-50, 3.8	10 days	Anderson et al. 1980
Cladocerans				
Daphnia magna	51	LC-50, 9.9	96 h	EPA 1980
Daphnia magna	51	LC-50, 5.0	21 days	Biesinger and Christian 1972
Daphnia magna	"soft"	LC-50, 0.7	20 days	Canton and Slooff 1982
Simocephalus serrulatus	11	LC-50, 3.5-8.6	96 h	Giesy et al. 1977
Fish				
Threespine stickleback, *Gasterosteus aculeatus*	--	LC-50, 0.8	33 days	Pascoe and Mattey 1977
Striped bass, *Morone saxatilis*				
Larvae	70	LC-50, 1.0	96 h	Hughes 1973
Fingerlings	70	LC-50, 2.0	96 h	
Chinook salmon, *Oncorhynchus tshawytscha*				
Swimup	23	LC-10, 1.2	200 h	Chapman 1978
Swimup	23	LC-50, 1.8	96 h	
Parr	23	LC10, 1.3	200 h	
Parr	23	LC-50, 3.5	96 h	
Smolt	23	LC-10, 1.5	200 h	
Juveniles	--	LC-50, 0.6-1.6	96 h	Finlayson and Verrue
Coho salmon, *Oncorhynchus kisutch*				
Juveniles	22	LC-50, 2.0	217 h	Chapman and Stevens 1978
Adults	22	LC-50, 3.7	215 h	
Rainbow trout, *Salmo gairdneri*				
Swimup	23	LC-10, 1.0	200 h	Chapman 1978
Swimup	23	LC-50, 1.3	96 h	
Parr	23	LC-10, 0.7	200 h	
Parr	23	LC-50, 1.0	96 h	
Smolt	23	LC-10, 0.8	200 h	
Age 2-months	82-132	LC-50, 6.6	96 h	Hale 1977
Age 2-months	31	LC-50, 1.8	96 h	Davies 1976
Age 2-months	--	LC-50, 6.0-7.0	96 h	Kumada et al. 1973, 1980
Adult	54	LC-50, 5.2	17 days	Chapman

				and Stevens 1978
		LC-50, 5.0-7.0	10 days	Kumada et al. 1973
Brook trout, *Salvelinus fontinalis*				
	330-350	LC-50, 3.8-4.4	96 h	Carroll et al. 1979
	44	LC-50, 2.4	96 h	

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

SUBLETHAL EFFECTS

Studies of 30 to 60 days duration with three comparatively sensitive species of freshwater fishes demonstrated that concentrations of >1 and <3 ppb cadmium in water of low alkalinity caused reductions in growth, survival, and fecundity of brook trout, the most sensitive species tested (Table 3). Under conditions of increasing alkalinity, the maximum allowable cadmium concentration range for brook trout increased to >7 and <12 ppb; a similar case was made for the walleye (Table 3).

Among all species of freshwater biota examined, cadmium concentrations of 0.47 to 5.0 ppb were associated with decreases in standing crop, decreases in growth, inhibition of reproduction, immobilization, and population alterations (Table 4). There is an abundant technical literature documenting numerous sublethal effects at higher Cd concentrations; however, these were excluded from the present account if the effects were observed at >10.0 ppb, the currently recommended criterion for drinking water.

For marine organisms, ambient Cd levels between 0.5 and 10.0 ppb resulted in decreases in growth, respiratory disruption, molt inhibition, shortened life span of F1 generation crustaceans, altered enzyme levels, and abnormal muscular contractions. Effects, in general, were more pronounced at the lower salinities and higher temperatures tested (Table 4).

Table 3. Maximum allowable toxicant concentrations (MATC) of cadmium to sensitive species of freshwater teleosts (after Brungs et al. 1978).

Organism and exposure period (days)	Water alkalinity, in mg $CaCO_3$/L	MATC, in µg Cd/L or ppb medium
Brook trout, *Salvelinus fontinalis*		
60	30	>1-<3
60	177	>7-<12
Channel catfish, *Ictalurus punctatus*		
60	34	>11-<17
60	172	>12-<17
Walleye, *Stizostedion vitreum vitreum*		
30	33	>9-<25
30	172	>86.7

Sublethal effects in birds are similar to those in other species and include growth retardation, anemia, and testicular damage (Hammons et al. 1978). However, harmful damage effects were observed at higher concentrations when compared to aquatic biota. For example, Japanese quail fed 75 ppm Cd in diet developed bone marrow hypoplasia, anemia, and hypertrophy of both heart ventricles at 6 weeks (Richardson et al. 1974). In zinc-deficient diets, effects were especially pronounced and included all of the signs mentioned plus testicular hypoplasia; a similar pattern was evident in cadmium-stressed quail on an iron-deficient diet. In all tests, 1% ascorbic acid in the diet prevented Cd-induced effects in Japanese quail (Richardson et al. 1974). Adult male white leghorn chickens given 2 ppm $CdSO_4$ daily by intraperitoneal injection for 15 to 22 days, or a total dose of 60 mg Cd per chicken, developed anemia, an enlarged heart, myocardial infarction, and other abnormalities (Sturkie 1973). Testicular damage was observed in ringdoves 20 days after intramuscular injection of 6.6 ppm Cd body weight (Richardson et al. 1974); in domestic pigeons, however, testicular damage was observed after a single subcutaneous injection of only 0.5 ppm (Sarker and Mondal 1973), and cardiovascular disease developed after exposure to 600.0 ppb in drinking water (Revis et al. 1981). In mallard ducklings fed 20 ppm dietary cadmium for 12 weeks, blood chemistry was altered, and mild to severe kidney lesions developed (Cain et al. 1983). Altered avoidance behavior in the form of hyperresponsiveness was observed in young American black ducks (*Anas rubripes*) produced from parents fed 4 ppm dietary cadmium for about 4 months before egg laying; this behavioral effect was observed only at comparatively low dietary cadmium levels and is considered harmful to wild birds (Heinz and Haseltine 1983). Cadmium readily reacts with sulfhydral groups and may compete, especially with zinc, for binding sites on proteins and, thus, may inhibit a variety of enzymatic reactions. The addition of zinc, iron, ascorbic acid, calcium, or selenium to diets ameliorated Cd damage effects, whereas the addition of lead or mercury exacerbated them (Hammons et al. 1978).

Among small laboratory mammals it appears that physiologically bound cadmium is more effective than $CdCl_2$ in producing metabolic iron irregularities. For example, in young mice fed oysters containing 1.8 ppm of Cd for 28 days, hematocrit and hemoglobin values were depressed and other blood chemistry factors were altered (Siewicki et al. 1983). Diets containing intrinsic oyster Cd at 1.8 ppm were more effective in producing hematopoietic alterations than were diets containing $CdCl_2$ at 3.6 ppm Cd (Siewicki et al. 1983).

Table 4. Sublethal effects of cadmium to selected species of aquatic biota

Type of medium, taxonomic group, organism, and other variables	Ambient Cd concentration, in ppb	Exposure period	Effect	Reference[a]
Freshwater				
Algae				
Asterionella formosa	2.0	--	Decreased growth rate	Conway 1978
Arthropoda				
Daphnia pulex	1.0	20 weeks	Reduced reproduction	Bertram and Hart 1979
Daphnia galeata mendotae	4.0	22 weeks	Reduced biomass	Marshall 1978
Eucyclops agilis	5.0	52 weeks	Population reduction	Giesy et al. 1979
Cambarus latimanus	5.0	22 weeks	Increased mortality	Thorp et al. 1979
Daphnia magna	2.6	21 days	Immobilization threshold	Biesinger and Christian 1972
Daphnia magna	0.7	21 days	Decreased reproduction (50%)	
Daphnia magna	0.17	21 days	No effect	
Daphnia magna	0.37	20 days	No effect	Canton and Slooff 1982
Daphnia magna	4.7	20 days	Decreased reproduction (50%)	
Annelida				

Pristina sp.	5.0	52 weeks	Population reduction	Giesy et al. 1979
Miscellaneous				
Mixed macroinvertebrates	5.0	52 weeks	Reduction in biomass and number of taxa	
Fish				
Brook trout, *Salvelinus fontinalis*	2.0	8 weeks	Disrupted lactic dehy-drogenase activity and blood glucose levels	Christensen et al. 1977
Atlantic salmon, *Salmo salar*	0.47	12 weeks	Alevin growth reduction	Rombough and Garside 1982
Salmo salar	2.0	60 days	Cranial pathology, reduced growth, death	Peterson et al. 1983
Salmo salar	0.2	60 days	Normal growth and development	
Medaka, *Oryzias latipes*	6.0	96 h	No effect	Canton and Slooff 1982
Marine				
Algae				
Phaeodactylum tricornutum	10.0-25.0	--	Decreased growth	Cossa 1976
Skeletonema costatum	10.0-25.0	--	Decreased growth	Berland et al. 1977
Arthropoda				
Crab, Pontoporeia	6.5	265 days	Reduced F1 life span	Sundelin 1983
Fiddler crab, *Uca pugilator*	1.0	--	Reduced respiration	Vernberg et al. 1974
Mysid shrimp, *Mysidopsis* spp.	10.0	23-27 days	Molt inhibition	Gentile et al. 1982
Mysidopsis spp.	5.1	23-27 days	No effect	
Coelenterata				
Laomedea loveni				
Salinity 10 ppt	3.0	7 days	EC-50, irreversible polyp retraction	Theede et al. 1979
Salinity 15 ppt	5.6	7 days	" "	
Temperature 15° C	9.0	7 days	" "	
Temperature 17.5° C	5.6	7 days	" "	
Fish				
Striped bass, *Morone saxatilis*				
Juveniles	5.0	90 days	Enzyme disruption	Dawson et al. 1977
Juveniles	0.5-5.0	30 days	Decreased oxygen consumption	
Winter flounder,				

Pseudopleuronectes 5*americanus*	5.0	60 days	Increased gill tissue respiration	Calabrese et al. 1975

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

A study by Beyer et al. (1985) of metal contamination in wildlife from the vicinity of two zinc smelters in Palmerton, Pennsylvania, demonstrated the difficulties in interpretation of cadmium residues from biota in the presence of other potentially hazardous metal contaminants (Table 5). The soil litter horizon at Palmerton was heavily contaminated with lead (2,700 ppm), zinc (24,000 ppm), copper (440 ppm), and cadmium (710 ppm). Invertebrates that fed on soil litter or soil organic matter, such as earthworms, slugs, and millipedes, were rare or absent in the vicinity of the smelters but not at more distant sampling sites (Table 5). Concentrations of all metals tended to be higher in these invertebrates than in other invertebrate groups collected. Amphibians and reptiles were also rare or absent at the Palmerton site, but not at more distant stations. Mean concentrations, in ppm dry weight, of cadmium were highest in carrion insects (25), followed by fungi (9.8), leaves (8.1), shrews (7.3), moths (4.9), mice (2.6), songbirds (2.5), and berries (1.2). By contrast, average concentrations of lead, in ppm dry weight, were highest in shrews (110), followed by songbirds (56), leaves (21), mice (17), carrion insects (14), moths (4.3), berries (4), and fungi (3.7). Evidence for lead poisoning in shrews included high residues in kidney (280 ppm wet weight) and reduced blood enzyme levels. In addition, livers from two cuckoos from Palmerton had lead concentrations of 18 and 25 ppm wet weight; however, the cuckoos and other songbirds appeared to be healthy. Concentrations of zinc and copper tended to be highest in the same organisms that contained the highest concentrations of cadmium, emphasizing the importance of documenting organism body burdens of all suspected contaminants before significance is attributed to any single component. Beyer et al. (1985) demonstrated that only a small portion of all metals measured in the soil became incorporated into plant foliage and suggested that most of the metal contamination detected in biota came from aerial deposition.

In human tissues, there was a significant increase in cadmium burdens in the years 1897-1939 vs. 1980. cadmium content in the renal cortex portion of the kidney increased by a factor of 47 during this interval, and whole body burden increased by a factor near 5 (Drasch 1983). The significance of this increase is not fully clear; however, recent work has suggested that cadmium and lead are associated with increased risk of heart-related death, even in the light of known conventional causes of such fatalities (Voors et al. 1982). Similar data for wildlife are lacking, and this clearly indicates an area for additional research.

Table 5. Cadmium residues, in mg/kg dry weight (ppm), in soil, flora, and fauna collected near two zinc smelters in Palmerton, Pennsylvania (from Beyer et al. 1985).

Soil and category of plants and animals	Direction and distance of areas from smelter emissions: Downwind about 2.1 km	Upwind 9.7 km
Soil		
Upper litter layers	250-710	6-13
Upper mineral layers	3-35	1-3
Foliage	8.1	2.3
Acorns and berries	1.2	0.6
Fungi	9.8	2.2
Moths, 8 spp.	0.8-11.0	0.4-1.7
Caterpillars	3.3	0.8
Earthworms, 2 spp.	NF[a]	62-140
Slugs	NF	20
Millipedes	NF	2.1-4.5
Beetles	1.3	0.8
Flies, 2 spp.	29-44	NF
Hornets	NF	2.3
Centipedes	28	NF
Birds		
Carcasses, 9 spp.	--	1.2
Carcasses, 10 spp.	2.5	--
White-footed mouse, carcass	2.6	1.2
Short-tailed shrew, carcass	NF	4.8
Amphibians, 5 spp.	NF	1.4

[a]NF = organism not found.

BIOACCUMULATION

Biological half times of cadmium in humans is lengthy. Based on body burden and excretion data, Cd may remain in the human body 13 to 47 years. Although Cd is excreted primarily in urine and feces, cadmium tends to increase in concentration with age of the organism and eventually acts as a cumulative poison (Hammons et al. 1978). These phenomena have not been documented adequately in wildlife species.

Freshwater and marine aquatic organisms accumulate cadmium from water containing Cd concentrations not previously considered hazardous to public health or to many species of aquatic life (Table 6). In American oysters, held for 40 weeks in flowing seawater containing 5.0 ppb of Cd, edible meats contained 13.6 ppm Cd fresh weight, a level considered to be an emetic threshold for human consumers (Zaroogian and Cheer 1976). These oysters retained virtually all accumulated cadmium (12.5 ppm) during a 16-week posttreatment immersion in clean seawater (Zaroogian 1979). Emetic thresholds for Cd in oysters were surpassed in 5 weeks at 25 ppb and in only 2 weeks at 100 ppb (Shuster and Pringle 1969). There is considerable variation in the ability of teleost tissues to accumulate cadmium from the ambient medium. Among rainbow trout, for example, exposed for 2 weeks to 9 ppb Cd, bioconcentration factors (BCF) were 260 for gill, 17 for liver, 26 for kidney, and zero for spleen and heart tissues (Roberts et al. 1979). At slightly higher ambient Cd levels of 10 ppb and exposure for 3 months, BCF values were substantially higher: 1,740 for gill, 4,900 for liver, 740 for kidney, 160 for spleen, and 100 for heart tissues (Roberts et al. 1979). The evidence for cadmium transfer through various trophic levels suggests that only the lower trophic levels exhibit biomagnification. In the freshwater food chain extending from the alga *Chlorella vulgaris*, to the cladoceran *Daphnia magna*, to the teleost *Leucospius delineatus*, it was demonstrated that algae held 10 days in water containing 10 ppb of cadmium contained 30 ppm dry weight, up from 4.5 ppm at the start (Ferard et al. 1983). Cladocerans feeding on cadmium-loaded algae for 20 days contained 32 ppm Cd dry weight, up from 1.4 ppm at the start. However, fish fed Cd-contaminated cladocerans for 4 days showed no change in body burdens.

Table 6. Bioconcentration of cadmium from ambient medium by selected species of aquatic biota.

Type of medium, taxonomic group, and organism	Ambient concentration of Cd, in ppb	Exposure period, in weeks	Bioconcentration factor, whole organism	Reference[a]
Freshwater				
Insects				
Ephemerella sp.	5.0	52	1,630	Giesy et al. 1979
Pantala hymenea	5.0	52	736	
Ischnura sp.	5.0	52	1,500	
Fam. Pytiscidae	5.0	52	164	
Fam. Chironomidae	5.0	52	2,200	
Fam. Ceratopogonidae	5.0	52	936	
Fish				
Salmo gairdneri	4.0	10	33	Kumada et al. 1980
Gambusia affinis	0.02	8	4,100	Williams and Giesy 1978
Gambusia affinis	5.0	26	7,440	Giesy et al. 1977
Algae				
Chlorella vulgaris	10.0	1.4	2,550	Ferard et al. 1983
Marine				
Molluscs				
Aquipecten irradians	10.0	3	131	Eisler et al. 1972
Crassostrea virginica	10.0	3	116	
Crassostrea virginica	5.0	40	2,720	Zaroogian and Cheer 1976
Fish				
Fundulus heteroclitus	10.0	3	15	Eisler et al. 1972
Crustaceans				
Homarus americanus	10.0	3	21	
Pontoporeia affinis	6.5	66	3,500	Sundelin 1983

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

In laboratory studies with chipping sparrows fed radiocadmium-109 in their diets for 3 weeks, it was demonstrated that cadmium became localized in the liver and kidneys (Anderson and Van Hook 1973). During posttreatment on a radiocadmium-free diet, there was an initial rapid drop in radioactivity, and the remaining radiocadmium had an estimated biological half-life of 99 days (Anderson and Van Hook 1973). Marine killifish containing radiocadmium-115m lost 90% of the accumulated radiocadmium during a 6-month posttreatment observation period; the liver usually contained 75 to 80% of the total body dose at any time (*Fundulus heteroclitus* (L.) (Eisler 1974). Mallards fed 200 ppm Cd in the diet for about 13 weeks all survived but levels in liver and kidney were elevated at 110 and 134 ppm fresh weight, respectively (White and Finley 1978). Mallard ducklings fed only 20 ppm dietary cadmium for 12 weeks contained 42 ppm Cd in the liver (Cain et al. 1983).

The exact mechanism of acute cadmium poisoning is unknown, but, among teleosts, it depends in part on exposure period, concentration of Cd in the medium, and water temperature and salinity. Under conditions of high Cd concentration and short exposure, the gill seems to be the primary site of damage and accumulation; under conditions of prolonged exposure and low Cd levels, the intestine, kidney, and possibly other tissues were measurably affected. Retention of cadmium by teleosts depends on tissue biomagnification potential, length of postexposure recovery period, and other factors. The significance of comparatively low concentrations of cadmium in tissues of fish, other aquatic organisms, and wildlife, and the implications for organism health, is not fully understood. Although numerous physical, chemical, and biological factors demonstrably modify uptake and retention of cadmium by fish and wildlife (Hammons et al. 1978; EPA 1980; Eisler 1981, 1984), the significance of relatively high cadmium residues to animal and plant health is difficult to interpret. There is some evidence, however, that life-threatening concentrations are 200 ppm Cd fresh weight in the renal cortex portion of the mammalian kidney (Hammons et al. 1978) and 5.0 ppm fresh weight whole body of estuarine teleosts (Eisler 1974).

TERATOGENESIS, MUTAGENESIS, CARCINOGENESIS

Teratogenic effect on animals appears to be greater for cadmium than for other metals, including lead, mercury, copper, indium, and arsenic (Ferm and Layton 1981). Among amphibians, frog embryos reared in 5,000 to 7,500 ppb Cd showed nonclosure of the neural tube (Ferm and Layton 1981). In embryos of fathead minnows from adults reared in water containing 37 to 57 ppb Cd, and from eggs transferred directly to such media, percent hatching was reduced, deformities were increased, and various blood clots developed (Pickering and Gast 1972). Embryos of the bluegill (*Lepomis macrochirus*) held in water at 80 ppb Cd or more showed edema, microcephalia, and malformed caudal fins (Eaton 1974). Eggs of a marine killifish (*Fundulus heteroclitus*) were little affected at up to 10,000 ppb of cadmium (Weis and Weis 1977). Caudal and hindlimb abnormalities were observed in chickens following injection of eggs with 0.1 to 1.0 ppm of cadmium chloride; excess zinc appeared to have a protective effect (Ferm and Layton 1981). Rats subjected to >6 mg Cd per kg body weight daily during pregnancy produced fetuses with jaw defects, cleft palates, club feet, and pulmonary hyperplasia (Ferm and Layton 1981). Among hamsters, cadmium administration was associated with embryonic tail defects; effects were synergized by salts of lead or mercury and antagonized by selenium (Ferm and Layton 1981). No conclusive evidence of Cd teratogenesis in humans is available.

From a variety of studies in which mice and bacteria were used as models, it appears likely that cadmium has mutagenic effects. Mice injected with 3 or 6 mg $CdCl_2$/kg body weight showed changes in chromosome number 12 h later; similar changes were observed in hamsters at 1.5 to 3.0 mg/kg (Ferm and Layton 1981). Very high dosages (>100 mg/kg) produced chromosomal abnormalities in plant seeds. Also, $CdCl_2$ had a mutagenic effect on indicator strains of *Salmonella* bacteria. However, the evidence for these effects is still diffuse and often contradictory (Ferm and Layton 1981).

Laboratory studies with mice and rats have conclusively demonstrated that the injection of cadmium metal or salts causes malignancies (sarcoma) at the site of injection and testicular tumors; however, the simultaneous administration of zinc is protective against sarcoma and interstitial cell tumor development (EPA 1980). Among humans, the available epidemiological evidence is not sufficient to conclude that cadmium is definitely implicated as a carcinogen (EPA 1980; Nomiyama 1982).

CURRENT RECOMMENDATIONS

Proposed limits for cadmium in water, food, and air for protection of human health and aquatic life are shown in Table 7. It is noteworthy that the current upper limit of 10.0 ppb of cadmium in drinking water for human health protection is not sufficient to protect many species of freshwater biota against the biocidal properties of cadmium or against sublethal effects, such as reduced growth and inhibited reproduction. Ambient water quality criteria formulated for protection of freshwater aquatic life state that, for total recoverable cadmium, the criterion, in µg/L, is the numerical value given by e to the power (1.05 (ln (hardness))-8.53) as a 24-h average and the concentration, in ppb, should never exceed the numerical value given by e to the power (1.05 (ln (hardness)) -3.73). Thus, at water hardnesses of 50, 100, and 200 mg/L as $CaCO_3$, the criteria are 0.012, 0.025, and 0.051 ppb, respectively, and the concentration of total recoverable cadmium should never exceed 1.5, 3.0, and 6.3 ppb, respectively. Unfortunately, data are accumulating that demonstrate that even these comparatively rigorous criteria are not sufficient to protect the most sensitive species of freshwater insects, plants, crustaceans, and teleosts. It now appears that levels in excess of 3.0 ppb of cadmium in freshwater are potentially hazardous to aquatic biota and that levels near 1.0 ppb are cause for concern in waters of low alkalinity. Not listed in Table 7, but still recognized as proposed criteria (EPA 1973), are the comparatively high levels of 10.0 ppb allowed for agricultural use on all soils (except neutral and alkaline soils, which may be irrigated with water having levels as high as 50.0 ppb) and public water supplies for livestock purposes, which may not exceed 50.0 ppb of cadmium.

The saltwater aquatic life protection criterion of 4.5 ppb seems adequate to prevent death, but will not prevent potentially deleterious physiological effects, including disrupted respiration in crustaceans and teleost. Incidentally, at 5.0 ppb of cadmium, the lowest concentration critically examined, oysters biomagnify ambient levels to concentrations hazardous to human consumers and possibly other animal consumers. The maximum allowable saltwater concentration (MAC) during a 24-h period was recommended as 59.0 ppb (Table 7). However, death of various species of marine crustaceans was reported at 60.0 ppb after exposure for 6 weeks and at 14.8 to 19.5 ppb after 23 to 27 days. Furthermore, a MAC of 59 ppb may be met with daily discharges of 59 ppb for 2 h and no discharge of cadmium for the rest of the day. The effects of exposure of marine life to 59 ppb of cadmium salts for 2 h daily for protracted periods have not yet been investigated. Accordingly, seawater concentrations in excess of 4.5 ppb of total cadmium at any time should be considered as potentially hazardous to marine life, until additional data prove otherwise.

Food is recognized as the major source of cadmium in humans, except in comparatively rare cases of occupational air exposure. The recommended upper limit for cadmium in food is 75 µg/day (Table 7). On the basis of an absorption factor of 0.1 (EPA 1980), a total of 7.5 µg Cd will be retained daily. A 70-kg adult ingests an estimated 0.75 kg of food daily, which suggests that human diets should not exceed 100 µg/kg. Ducks, geese, and other species of wildlife, unlike adult humans, may consume 6 to 7% of their total body weight daily and may graze extensively on crops directly affected by sewage and other wastes containing high Cd residues. Feeding studies with mallards indicated that diets containing 200 mg Cd/kg produced no obvious deleterious effects after 13 weeks. At the end of that study, however, kidney cadmium levels under those conditions were about 134 ppm fresh weight, a level near the 200 ppm fresh weight designated a “critical threshold” (and presumably life-threatening) for the renal cortex portion of the human kidney. Field observations on ducks and laboratory studies are not strictly comparable. Under field conditions, birds and other wildlife may consume food containing high Cd levels, but it is almost certain that these diets also contain other potentially harmful contaminants, as well as metals or compounds that may ameliorate cadmium toxicity. The significance of foods containing complex mixtures of contaminants and their resultant toxicological interactions are imperfectly understood. Until other data become available, wildlife dietary levels exceeding 100 µg Cd/kg diet fresh weight on a sustained basis should be viewed with caution.

Current recommendations for cadmium in air and human health protection under the worst scenario (Table 7) assume that total daily air intake is 27.14 m^3 for an adult human who spends about 6.3 h in occupational exposure to air containing 100 µg Cd/m^3 (EPA 1980). Under these conditions, a 70-kg adult would retain about 361 µg Cd/day, based on an absorption factor of 0.5 (EPA 1980), and most of this Cd would probably be translocated to the kidney; a critical threshold level of 200 mg Cd/kg in the kidney would be reached in about 1.52 years. It is not now known whether respiration rates of wildlife, particularly birds, are comparable to those of humans, or whether cadmium absorption energetics are similar, or whether wildlife species that frequent point sources of air contaminated by high Cd levels for protracted periods are at greater risk than humans. Evidence given by Beyer et al. (1985) demonstrated that flora and fauna in the vicinity of industrial smelters were affected by Cd and its associated heavy metals. This strongly suggest that current recommendations for cadmium levels under occupational air exposure should be revised downward for wildlife protection.

Finally, the issue of the significance of cadmium residues in various body parts requires resolution. At this time, it appears that cadmium residues in the vertebrate kidney or liver that exceed 10.0 mg/kg fresh weight or 2.0 mg/kg in whole body fresh weight should be viewed as evidence of probable cadmium contamination. Elevated levels of 13.0 to 15.0 ppm Cd tissue fresh weight probably represent a significant hazard to animals of the higher trophic levels, and residues of 200 ppm fresh weight kidney or more than 5.0 ppm whole animal fresh weight should be considered life-threatening.

Table 7. Current recommendations for cadmium in water, food, and air (adapted from EPA 1980).

Ecosystem, environmental compartment, and other variables		Cadmium concentration[a]
Water		μg/1
Freshwater aquatic life protection		
at water hardness, in mg $CaCO_3$/1, of		
50		1.5
100		3.0
200		6.3
Saltwater aquatic life protection		
24-h average		4.5
Maximum allowable concentration		59.0
Human health protection		
Best case		0.5
Average case		1.3
Worst case		10.0
Food	μg/day	μg/kg diet/day
Human health protection[b]		
Best case	12	16
Average case	30	40
Worst case	75	100
Air		$\mu g/m^3$
Human health protection		
Best case, ambient exposure		0.001
Average case, ambient exposure		0.03
Worst case		
Occupational exposure		100.0
Ambient exposure		0.4

[a]Units of concentration shown apply to the ecosystem concerned (water, food, air).

[b]Assumes consumption of 0.75 kg food per day by 70 kg adult.

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