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CADMIUM AND WILDLIFE

(A LITERATURE REVIEW)

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Abstract

This paper reviews the literature concerning cadmium and wildlife. Aspects considered include the sources, production and use, distribution in the environment and wildlife, bioaccumulation, metabolism and toxicity of cadmium. Levels in various species of birds and mammals from different geographical locations are presented. From data available, there appears to be little danger at this time of widespread poisoning of wildlife with cadmium.

Résumé

Ce rapport revoit la literature sur le cadmium et la faune. Les aspects suivants on été considérés, soi les sources, la production et l'utilisation, la distribution dans l'environnement et la faune, la bioaccumulation, le métabolisme et la toxicité du cadmium. Les concentrations dans diverses espèces d'oiseaux et de mammifères de différentes régions sont présentées. D'après les données disponibles, il semble y avoir, en ce moment, très peu de danger d'empoisonnement général de la faune par le cadmium.

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Properties and Uses

Cadmium is a soft silver-white metal, first discovered in 1817 as cadmium sulfide, a minor component of zinc ore. It is ubiquitous in nature and always found in close association with zinc, to which it is chemically and physically similar. Unlike zinc, cadmium is a non-essential trace element with no known biological function. It is an insidious poison and extremely toxic to animals and man, considered by Christensen and Olsen (1957) to "have probably more lethal possibilities than any of the other metals". Cadmium has been commercially available for about 100 years but it has only been since the 1920's that large quantities have been produced. Production in the free world peaked at 17,576 metric tons in 1969 (Fleischer et al. 1974). While production has decreased in recent years, it is estimated that use in the U.S.A. will nearly double by the year 2000 to a level of about 13,600 metric tons. In the U.S.A., an estimated 2,518-3,588 metric tons were emitted into the environment in 1968 by industry (Nobbs and Pearce 1976). Emissions of this magnitude, combined with projected production statistics and the persistent nature of cadmium in the environment, make the problem of cadmium pollution of considerable concern.

The major uses for cadmium are plating, pigments, stabilizers, nickel-cadmium batteries and alloys. Cadmium also occurs in fungicides and fertilizers, nuclear control rods, television tubes, motor oil and automobile tires (Lymburner 1974, Environment Directorate

1975, Stubbs 1978). In the U.S.A. and Canada, about 50 percent and 75 percent respectively, of the cadmium consumed is used in the electroplating industry (Hiatt and Huff 1975, NRC Canada 1979).

Sources

I Natural

Cadmium occurs naturally throughout the environment with an average concentration of 0.15-0.20 ppm in the earth's crust (Hiatt and Huff 1975). It is slightly concentrated in shales, lacustrine and oceanic sediments, in manganese nodules and in marine phosphorites. All cadmium production is from sulfide ores of zinc, lead and copper, from which it is recovered as a by-product. Background levels in soils range from 0.4-3.0 ppm cadmium (Fleischer et al. 1974, Wixson 1978). Concentrations in coal vary from 0.2-30.0 ppm (Fleischer et al. 1974, Wixson 1978). Background levels of cadmium in air are generally low, ranging from tenths of a nanogram to 10 ng/m^3 in rural areas and 2 to 420 ng/m^3 in urban areas (Fleischer et al. 1974). Nearly all airborne cadmium is due to man's activities. Seawater contains about 0.15 ppb on average (though data probably include contaminated samples) and most fresh waters contain less than 1 ppb (Fleischer et al. 1974).

II Industrial

1. Air Contamination

a) Zinc Smelting

Cadmium metal is a by-product of zinc production. The Cd/Zn ratio in nonferrous ore is about 0.5 percent (Hiatt and Huff 1975). Smelting and production methods are described by Fleischer et al. (1974). Ore concentrates are roasted to oxidize the sulfide and then treated by either pyrometallurgical or electrolytic processes. From 10-25% of the cadmium is vaporized during the roasting process and the dust is collected in electrostatic precipitators or bag filters. The roasted ore is then sintered and dust from this operation is led to collectors and then vented to the atmosphere. About 15-20% of the initial cadmium accumulates in the dust obtained during sintering. The agglomerate from the sinter plants will typically retain more than 50 percent of the cadmium content of the ore. In the pyrometallurgical process, the agglomerate is reduced with carbon and carbon monoxide and the resulting metals are distilled; in the electrolytic process, the minerals are dissolved in acids (Lymburner 1974). Plant design and efficiency of the particulate collection devices determine the amount and physical and chemical properties of the cadmium emitted by smelters. Mainly oxides (or chlorides when chlorides are used in the sinter unit) are emitted from the sinter units and sulfates from the roasters. Particle size varies from the submicron range to about 8 or 10μ (Fleischer et al. 1974). Cadmium recovery

in zinc smelting can exceed 90 percent (Chizhikov 1966) but the extent of emissions varies with individual operations. The processing and refining of cadmium-bearing ores in the zinc, lead, copper and cadmium industries released an estimated 2.1 million pounds of cadmium into the air in 1968, 45 percent of the total 4.6 million pounds discharged that year (McCaul 1971). The single largest source of atmospheric emission in this category was the roasting and sintering of zinc concentrates.

b) Metal Processing and Incineration of Wastes

Metal treatment furnaces and incinerators account for more than half the total atmospheric emissions of cadmium. Fulkerson and Goeller (1973 in Fleischer et al. 1974) estimate that 1,000 tons of cadmium were volatilized during the smelting of scrap steel in 1968. In addition, the melting down and/or disposal of zinc and copper products such as automobile radiators also produces large emissions of cadmium as does the incineration of solid waste made of polyvinyl chloride containing cadmium compounds as colouring agents or stabilizers.

c) Coal Combustion

Significant quantities of cadmium are released during the combustion of coal. This source of contamination may become more important as coal consumption increases.

2. Water Contamination

The major inputs of cadmium into surface and coastal waters are from the leaching of mine tailings, particularly by acid waters, atmospheric emissions of soluble sulfates and chlorides, disposal of wastes from hydrometallurgical and cadmium-plating installations, surface runoffs in urban areas and from the discharge of sewage sludge and effluent into waterways.

3. Soil Contamination

Unacceptably high levels of cadmium in soils may occur in areas surrounding smelters or furnaces (Munshower 1977, Gale and Wixson 1979). Phosphate fertilizers and sewage sludge also contribute to cadmium contamination of soils. Concentrations of cadmium in sewage sludge from the Chicago area varied from 14 to 470 ppm (Tenny and Stanley 1967 in Fleischer et al. 1974) and from 9 to 883 in Indiana (Yost 1978 in Metal Bull. Ltd. 1978).

Sediments in the vicinity of sewage outfalls may be greatly enriched with cadmium (Galloway 1972, Young et al. 1973 in Fleischer et al. 1974). Heavy metals, including cadmium, also accumulate in the sediments around offshore oil rigs (Newbury 1979).

Bioaccumulation and Food Chains

Evidence for the biomagnification of cadmium in food chains is not conclusive. Plants accumulate cadmium through the roots and leaves and the amount accumulated varies with the species and ambient levels (Hiatt and Huff 1975). Most plants have a cadmium content similar to normal soil (Friberg et al. 1971), but some species accumulate significant quantities of cadmium. Mosses and lichens, because of their ability to absorb and retain airborne metals, serve as indicators of environmental quality (Fleischer et al. 1974, NRC 1979). Many plant species which accumulate large quantities of cadmium are important foods for wildlife. Hence, a potential danger exists for cadmium poisoning of some wildlife species. Makhoninia and Gileva (1968 in NRC 1979) determined the bioaccumulation factors of Chara fragilis (a large alga) and Myriophyllum spicatum (water milfoil) to be 22,900 and 10,000 respectively. Muskgrasses (Chara sp.) are among the top-ranking foods of ducks, particularly the coot (Fulica americana), redhead (Aythya americana), ring-necked duck (Aythya collaris), Greater Scaup (Aythya marila), American widgeon (Mareca americana) and green-winged teal (Anas carolinensis) (Martin, Zim and Nelson 1951). According to Cearley and Coleman (1973), the southern naiad (Najas quadulepensis Spreng.) is capable of introducing potentially toxic quantities of cadmium into the food chain of higher organisms such as sunfish and waterfowl. Water hyacinth (Eichlornia

crassipes) and the bulrush (Scirpus americanus) accumulate heavy metals to such an extent that they have been suggested as means of clearing polluted waterways of their heavy metal content (Carbonneau and Tremblay 1972, Wolverton 1975 in NRC 1979). Bulrushes are an important food source of several species of ducks and geese and the muskrat (Ondatra zibethica). Carbonneau and Tremblay (1972) suggest that migrating snow geese (Chen hyperborea altantica) eating Scirpus while in the vicinity of Cap-Rouge, Quebec, may suffer from heavy metal toxicosis. Aspens are utilized extensively by beaver (Castor canadensis), varying hare (Lepus americanus), white-tailed deer (Odocileus virginianus), moose (Alces americana) and ruffed grouse (Bonasa umbellus) (Martin et al. 1951). Cannon et al. (1972 in NRC 1979) found that aspen accumulated ten times more cadmium than did other trees. Most cultivated crops are used to some extent by wildlife. Grains and corn are eaten by geese, upland game birds and raccoons (Procyon lotor). In areas where excessive amounts of sewage sludge or inorganic fertilizers have been applied to soils, a potential danger of cadmium poisoning may exist for some wildlife species. Lettuce and celery accumulated cadmium even when grown on soils containing only natural cadmium levels (Haghiri 1973, 1974 in NRC 1979). Munshower (1977) found that pasture grasses, alfalfa and barley reflected elevated soil cadmium levels and that liver and kidney concentrations in cattle and swine revealed the effect of excess cadmium in the animal's diet. Melsted et al. (1976) report similar findings from feeding pheasants corn grown on sludge-amended

soil as do Williams et al. (1978) in studies with voles. Note, however, that only about 1 percent of the cadmium in the corn grain was injested by the pheasants. The availability of the cadmium to the next successive animal in the food chain, a secondary consumer, has not been investigated. Several authors agree that there is little evidence for concentration of cadmium in most food chains (Preston 1973, Fleischer et al. 1974, Hiatt and Huff 1975, Anderson et al. 1978). However, the bioaccumulation of cadmium is quite significant in pelagic zooplankton and plankton-eating birds (Fleischer et al. 1974), fish (Fleischer et al. 1974) and mollusca, particularly oysters (Frazier 1979, Engel and Fowler 1979). Concentration factors of 1,000 and 3,000 for cadmium in the American oyster have been reported by Schuster and Pringle (1969 in Frazier 1979) and Zaroogian and Cheer (1976 in Frazier 1979) respectively. Roberts and Johnston (1978) conclude that "whilst cadmium is present only as a trace contaminant in most mine wastes, its mobility through terrestrial food chains is greater than that of the dominant spoil metals lead and zinc. Examinations of carnivores, herbivores and their estimated diets indicated that cadmium has a greater potential for accumulation through lower terrestrial trophic levels than both lead and zinc". The outbreak of Itai-itai disease in Japan also provides evidence of the accumulation of cadmium in a food chain. Irrigation water for rice paddies was contaminated by cadmium from a mine. The consumption of rice containing high cadmium concentrations is

is considered an important factor in the outbreak of the disease (Shigematsu 1978, Kato and Abe 1978).

Metabolism

a) Ingestion

The gastrointestinal tract and the respiratory system are the main routes of entry of cadmium into the body. Absorption through the skin is probably insignificant. Various animal studies show that the absorption rates for inhaled and injested cadmium are about 10-40% and 1-3% respectively (Friberg et al. 1971, Fleischer et al. 1974). A diet deficient in protein or calcium increases the absorption rate of cadmium (Friberg et al. 1971, Gontzea and Popescu 1978, Cousins 1979).

Several forms of cadmium can be ingested. The sulfide (CdS) and carbonate ($CdCo_3$) are less soluble than the corresponding zinc compounds but the hydroxide ($Cd(OH)_2$) is more soluble than the hydroxide ($Zn(OH)_2$). Cadmium forms a wide variety of soluble complexes, notably with cyanides and ammines (Fleischer et al. 1974). Its interactions with other nutrients including Zn, Cu, Fe, Hg, Se, Ca, Co, dietary protein and chelating agents alter its rate of absorption (Willoughby 1978). In experimental studies, the salt $CdCl_2$ is commpnly injected or fed to animals. The airborne particles from smelting and incineration consisting mainly of oxides, chlorides and sulfates may be injested.

b) Transport in the Blood and Tissue Distribution

After absorption from the intestine or lungs, most cadmium is initially transported in the plasma, bound to gammaglobulin, but a rapid clearance from plasma takes place with most of the cadmium being bound to proteins such as hemoglobin and metallothionein in the blood cells (Friberg et al. 1971, Neathery and Miller 1976). Even after intravenous injections or large oral doses, cadmium disappears rapidly from the blood and is deposited in the liver, followed by increasing concentrations in the kidneys (Friberg et al. 1971). Consequently, blood levels of cadmium are of virtually no diagnostic value. While cadmium can occur in most any body tissue, the highest concentrations are generally in the kidney followed by the liver (Miller et al. 1969, Friberg et al. 1971, Doyle et al. 1972, Shaikh and Lerces 1972, Anderson and Van Hook 1973, Smith et al. 1976, White and Finley 1978).

There is virtually no transfer of cadmium from birds to eggs (Henning et al. 1971 in Sell 1975, Sell 1975) or across the placenta in mammals (Nethery and Miller 1975).

c) Role of Metallothionein

Metallothionein plays a major part in the metabolism of cadmium. It is a low molecular weight protein with a strong affinity for cadmium and to a lesser extent zinc. Small doses of cadmium stimulate its synthesis (Bryan et al. 1979). First isolated from

equine renal cortex by Kägi and Vallee (1961), metallothionein or similar low molecular weight proteins have since been found in several species of birds and mammals (Pulido et al. 1966, Olafson and Thompson 1974, Singhal et al. 1974, Osborn 1978), fish (Olafson and Thompson 1974 in James 1978) and invertebrates (Frazier 1979). Once bound to metallothionein, cadmium is retained in the tissues for extended periods of time and in this bound state it is generally considered biologically inert, up to certain levels (Miller 1973, Willoughby 1978). Several studies have shown that animals have no known mechanism to guard against excessive uptake of cadmium (Cotzias et al. 1961, Miller et al. 1969, Friberg et al. 1971, Miller 1973). Cotzias et al. (1961) found that alimentary absorption in mice occurred irrespective of the body's Cd burden. However, the binding of cadmium to metallothionein and the increased biosynthesis of metallothionein with an increase in body burden of cadmium may represent a biological defence mechanism against toxic cadmium ions (Friberg et al. 1971, Shaikh and Lerces 1972). Bull et al. (1977) conclude that the high concentrations of cadmium found in seabirds originate from natural sources and that the birds have developed, through natural selection, mechanisms which enable them to tolerate this metal. They suggest that a mechanism involving a metallothionein-like protein may be involved. In this bound state, cadmium continues to accumulate in the body, particularly the kidneys, with only minimal quantities being excreted (Friberg 1978). After injection into mice, Cd^{109} showed negligible total-body turnover (Cotzias et al. 1961). The

task group on metal accumulation (1973) reviews the concept of biological half-times. The biological half-life of cadmium for the human kidney has been estimated from 17.6 years (Tsuchiya and Sugita 1971) to 33 years (Kjellström et al. 1971). Anderson and Van Hook, Jr. (1973) found the half-life of cadmium in the kidneys of chipping sparrows, Spizella passerina, to be 99 days. They point out that the long-term presence of cadmium in the liver and kidney increases this elements' potential for toxicity.

Toxicity

The toxic effects of cadmium are reviewed by Nilsson (1970), Flick et al. (1971), Friberg et al. (1971) and Neathery and Miller (1975). Cadmium is toxic to most living organisms (Schroeder and Balassa 1961) and is lethal to some species at very low concentrations. The lethal concentrations of cadmium in water for several species of aquatic invertebrates and fish are summarized in appendices II and III. Several of these values were taken from the review of water quality criteria by McKee and Wolf (1963). Note that the LC_{50} is much higher than the concentration resulting in no adverse effects. Cadmium concentrations ranging from 0.068 to 0.150 ppm were lethal to fathead minnows while levels of 0.037 to 0.057 ppm were tolerated with no adverse effects on survival, growth or reproduction (Pickering and Gast 1972). Eaton (1974) reported an 11 month LC_{50} of 0.080 ppm cadmium in water for the bluegill, Lepomis macrohirus Rafinesque, with a concentration of 0.031 ppm necessary for no adverse effects.

Pickering and Gast (1972) and Eaton (1974) suggest using fish embryos or larvae, the more sensitive stages in the life cycle, when estimating "no effect" or "safe" concentrations. The toxicity of cadmium is much higher in soft water than hard; Tarzwell and Henderson (1960) found the 96 hour TL_m of $CdCl_2$ toward fathead minnows to be 0.9 ppm in soft water and 5.0 ppm in hard water. There may be an antagonistic action between calcium, magnesium and perhaps other base metals and cadmium (McKee and Wolf 1963). This factor should be considered when comparing LC_{50} values from different studies.

Less data is available on the lethal concentration or dosage for mammals and birds. A cadmium chloride concentration of 250 ppm in drinking water was fatal to rats (Anon. 1950 in McKee and Wolf 1963). For rabbits, 0.15 to 0.3 gm $CdCl_2$ per kg body weight was lethal, and for cats, 125 ppm $CdCl_2$ in the diet was fatal (Anon. 1950 in McKee and Wolf 1963). Cadmium oxide fumes are very toxic. When inhaled for 15 minutes a concentration of 35 mg/m^3 caused 7 of 20 rats to die and 310 mg/m^3 for 10 minutes was lethal to 2 out of 4 dogs (Barrett et al. 1941 in Fleischer et al. 1974). Toward rabbits, the 8 hour LC_{50} for oxide dust was 15 mg/m^3 (Friberg et al. 1971). In humans, a lethal acute exposure to the oxide fume is about 5 mg/m^3 for 8 hours, whereas about 1 mg/m^3 for 8 hours is considered dangerous (Hiatt and Huff 1975).

Japanese quail receiving 75 ppm cadmium in the diet grew more slowly and had a higher mortality than control birds. Those birds fed cadmium also developed severe anemia (Jacobs et al. 1969).

Cadmium is chemically similar to mercury but its toxicity depends less on its chemical form than does the toxicity of mercury. The cadmium ion Cd^{++} is the presumed cause of physiological effects in both acute and chronic poisoning (McCaul 1971, Lorke 1978). Cadmium has a particular affinity for sulfhydryl groups and, to a lesser extent, hydroxyl groups (Nilsson 1970). The cadmium ion is a potent inhibitor of several enzyme systems; its probable action is by binding to essential sulfhydryl groups in the enzymes (Cook 1970, Nilsson 1970). It is probably through its effect on enzymes, particularly those involved in intermediary metabolism, that cadmium exerts its toxicity (Singhal et al. 1974, Gontzea and Popescu 1978). The manifestations of cadmium toxicity can be categorized into acute and chronic responses. Cases of acute poisoning are a result of a relatively high level of intake in a short time. Of greater importance from the standpoint of wildlife toxicology is the chronic poisoning which may occur after long-term ingestion of relatively small quantities of cadmium.

Kidney damage is the most typical feature of chronic cadmium poisoning by injection (Friberg 1978). Renal damage does not occur until concentrations of cadmium in the kidney reach about 200 ppm (wet weight) in renal cortex (Friberg et al. 1971, Nobbs and Pearce 1976). Cadmium affects the reabsorption functions of the proximal tubules resulting in proteinuria, an increase in the excretion of low molecular weight proteins (Hiatt and Huff 1975, Friberg 1978, Kazantzis 1979). Proteinuria is usually followed by a more generalized

renal tubular dysfunction (Kazantzis et al. 1963 in Fleischer 1974) characterized by increased excretions of aminoacids, glucose, phosphorus and calcium (Friberg 1978). With continued exposure to cadmium the buildup in the kidney stops and urinary cadmium increases (Neathery and Miller 1975). Chronic exposure via inhalation results in lung emphysema as well as kidney disease and proteinuria (Friberg 1978). Cadmium may be toxic to practically any system. Histological changes associated with cadmium toxicity have been observed in the testes, kidney, liver, gastrointestinal tract, heart, blood vessels, bone marrow and pancreas (Richardson et al. 1974). Cadmium is also suspected of having several other toxic effects including hypertension (Schroeder 1964), testicular necrosis (Lofts and Murton 1967, Friberg et al. 1971), anemia (Jacobs et al. 1969), osteomalacia (Friberg et al. 1971), a shortened life span and increased mortality (Schroeder et al. 1964, McCaul 1971) and the impairment of growth (Doyle et al. 1972, Richardson et al. 1974). The evidence for cadmium exhibiting mutagenic, carcinogenic and teratogenic effects is reviewed by Fleischer et al. (1974), Nilsson (1970) and Friberg (1978).

The toxicity of cadmium may depend on the synergistic effect of other substances or compounds (Calabrese et al. 1977, Nordberg et al. 1978, Campbell et al. 1978, Fox 1979); this topic is reviewed in depth in the section on interactions among metals (pp. 489-543 in Nordberg 1976). It is now generally accepted that while chronic cadmium poisoning was probably a major factor causing the outbreak of "Itai-itai" disease in Japan, low intakes of calcium, protein and vitamin

D may have been involved as well (Friberg et al. 1971, Tsuchiya 1978). Gontzea and Popescu (1978) found that "a quantitatively and qualitatively adequate protein supply increased the resistance of rats to cadmium, diminishing significantly the severity of the symptoms induced by the metal". The metabolism of zinc and cadmium are intimately connected (Schroeder et al. 1967, Jacobs et al. 1969, Friberg et al. 1971). Zinc is an essential metal with many enzyme systems being zinc dependent. Cadmium seems to have the ability to exchange with zinc thus interfering with enzyme activity. Supplee (1961) was able to produce the signs of zinc deficiency in turkey poults by providing dietary cadmium. The toxicity of exogenously-administered cadmium can be reduced, or even prevented in some cases, by zinc, cobalt, selenium, estrogen and thiol compounds (Flick et al. 1971). There is some evidence of a relationship between tissue concentrations of cadmium and exposure to PCB (Olsson et al. 1979). Olsson et al. (1979) state that "high levels of DDT and PCBs are often associated with high levels of heavy metals". As an example they cite Martin et al. (1976). Female California sea lions delivering premature pups have high concentrations of heavy metals, including cadmium, as well as organochlorines.

Cadmium and Wildlife

Tissue concentrations of cadmium in various species of birds and mammals from different geographical localities are summarized in

Appendix I. In general, cadmium levels are quite low. The most notable exception to this is the thrush (Turdus philomelos) with a cadmium concentration in the kidney of 387 ppm dry weight (Martin and Coughtrey 1975). Relatively high levels were also reported for several species of seabirds (Anderlini et al. 1972, Parslow and Jeffries 1977, Murton et al. 1978), the eider (Sematería mollisima L.) and the lesser black-backed gull (Larus fuscus fuscus) (Lande 1977). However, in no case other than the thrush, did the cadmium concentrations even approach the critical level of 200 ppm in renal cortex tissue. Evidence from animal data suggests that at kidney cortex concentrations above 200 ppm wet weight, tubular dysfunction and/or histopathological changes occur in the kidney (Bonnell et al. 1960, Axelsson et al. 1968, Stowe et al. 1972, Kawai et al. 1976, Nomiyama et al. 1976 in Nordberg 1976, Friberg et al. 1971). Thus, at least for the wildlife species on which data are available, it appears that cadmium poisoning is not a major problem. There is a paucity of information on normal levels in wildlife. Few authors make any attempt to interpret the levels of cadmium found. Munshower and Neuman (1979), in their study of mule deer and antelope in Montana, conclude that cadmium concentrations of 1.27 and 2.70 ppm in the kidneys of antelope and deer respectively, were reflective of levels in healthy animals in those particular populations. Only two references suggested that existing levels of cadmium present a problem to wildlife. Heavy metal toxicosis may have been a contributing factor to a particular stranding of short-finned pilot whales (Stoneburner 1978).

Cadmium concentrations in muskrats in the Saucon Creek area, Hallertown, Pennsylvania, may be high enough to cause anemia, a sign that appears in studies of chronic cadmium poisoning (Everett and Anthony 1976).

A few experimental studies have investigated the relationship between cadmium and various wildlife species. Such studies are of value in interpreting levels found in wild animals. In vitro studies revealed that the biosynthesis of steroid hormones in the gray seal (Halichoerus grypus) was altered by cadmium levels of 0.45 ppm in testicular incubates (Freeman and Sangalang 1977). They suggest that this level of contamination may have a significant effect on the physiology of the seal. Cadmium concentrations in this order of magnitude are commonly found in seals and many other species of wild animals (Appendix I).

White and Finley (1978) fed adult mallards (Anas platyrhynchos) 0, 2, 20 or 200 ppm CdCl in the diet. The liver and kidney accumulated highest levels of cadmium and residues were correlated with treatment groups. After 60 and 90 days, lesions were produced in kidneys of all birds in the group fed 200 ppm cadmium. This study also agrees with Henning et al. (1971) and Sell (1975) in showing little cadmium transfer to the eggs of birds. Compared to these experimental results, ruddy duck carcasses collected from the Delaware River in 1973 (White and Kaiser 1976) and canvasbacks from the Chesapeake Bay region (Stendell 1974 in White and Kaiser 1974) had relatively low levels of cadmium; the levels were comparable to those in ducks fed 2 ppm daily. The

results of a study by Finlay et al. (1979) show a reduction in fertility of Peromyscus exposed to 1 ppm cadmium in the drinking water. Such levels of exposure could occur in natural environments; a reduction in the fertility with subsequent declines in important prey species like Peromyscus could have profound impact on animals further up the food chain.

As discussed above, several studies have investigated the transfer of cadmium through the food chain (Melsted et al. 1976, Williams et al. 1978). Williams et al. (1978) fed meadow voles (Microtus pennsylvanicus) diets from crops grown on sludge-treated soils and concluded that diets containing 1 ppm cadmium may cause significant accumulations in animal tissues. Some species of wildlife already contain levels of cadmium as high as those found in this study (0.03-3.69 ppm); animals consuming plant material from sludge-treated areas could be exposed to excessive amounts of cadmium. At fairly low concentrations, cadmium could disrupt food chains (particularly in aquatic environments) by poisoning highly susceptible species in lower trophic levels.

The effects of cadmium on fish are important from a wildlife viewpoint since many fish species form an important link in aquatic food chains. The range of cadmium concentrations in water which are generally lethal to fish range from 10 to 10,000 ppb (McKee and Wolf 1963). However, many species are adversely affected by sub-lethal levels (Pickering and Gast 1972, Eaton 1974, Bengtsson et al. 1975, Calabrese et al. 1977). Exposure to 7.5 ppb cadmium in water for 70 days caused minnows (Phoxinus phoxinus) to develop lesions in their

spinal columns (Renatsson et al. 1975). Sangalang and O'Halloran (1972) found non-lethal levels of cadmium to cause testicular injury and alterations of androgen synthesis in brook trout (Salvelinus frontinalis). Sangalang and Freeman (1974 in NRC 1979) found that 1 ppb of cadmium in water can interfere with the reproductive process in a salmonid species. This is significant for these species spawn in rivers and streams which are quite prone to industrial pollution. The developing embryo and fish larvae are very sensitive life stages (Pickering and Gast 1972, NRC 1979). The viability of Pacific herring (Clupea pallasii) eggs exposed to 10 ppm cadmium was only 27.3% compared to 82.0% for controls (Mounib et al. 1976 in NRC 1979). Larvae of the herring (Clupea harengus), the flounder (Platichtys flesus) and the garpike (Belone belone) accumulated cadmium to a considerable degree even though acute toxic or lethal effects were not observed (Dethlefsen et al. 1975 in NRC 1979). Cadmium concentrations of 57 ppb decreased the survival of developing fathead minnow embryos (Pickering and Gast 1972).

Several recent references (Appendix IV) give more up-to-date information on the chronic toxicity of cadmium to fish, particularly salmonids. These references contain a complete literature review of the topic. Note that chronic toxicity has been observed in concentrations of 200-700 ppt in water, levels much lower than the levels cited in this report from earlier references.

In a survey of 406 fish from 49 waters in New York state, Lovett et al. (1972 in NRC 1979) found that the majority of the fish (68.5%) contained 0.02 ppm Cd (whole body wet weight) or less. An

average concentration of 0.09 ppm Cd (wet weight) was found in 19 whole fish of three species from the Great Lakes (Lucas et al. 1970). Cadmium levels were very low in yellow perch (Perca flavescens), white bass (Morone chrysops) and smallmouth bass (Micropterus dolomieui) collected from Long Point Bay, Lake Erie, in 1972, i.e., 0.063, 0.034 and 0.047 ppm whole body wet weight respectively (Kelso and Frank 1974). Based on these data it is unlikely that cadmium toxicity presents a threat to fish-eating birds. Molluscs and crustaceans appear to accumulate cadmium more readily than do fish.

In general, cadmium seems to present no major threat to wildlife except in isolated local areas where there is severe pollution such as in the vicinity of lead and zinc smelters, mining sites and sewage outfalls. Incinerators, land-fill sites and secondary industries utilizing cadmium may also be a source of local pollution. Fallout around smelters results in highly elevated cadmium levels in soils and plants; these levels decline rapidly with increasing distance from the smelter. Hence, cadmium presents a potential danger to wildlife only in the few square miles adjacent to the smelter. Near the Cu-Zn smelter at Anaconda, Montana, cadmium concentrations of 29 µg/g dry weight soil 2 km from the smelter dropped to 1.6 µg/g at 43 km. However, concentrations were still above the background range of 0.2-0.6 ppm at 60 km (Munshower 1977). Within 1 km of the zinc smelters in Palmerton, Pennsylvania, cadmium levels of 1750 ppm were measured in surface soils (Buchauer 1973). Background levels were reached at 16 to 21 km from the smelter. Beavington (1975) found that cadmium levels in both soil and lettuce were significantly

correlated with the distance from the smelter chimney. Near primary lead smelters in Missouri, leaf litter samples showing cadmium concentrations approaching 100 ppm dropped to background levels at distances of 10-15 miles (Bolter 1974 in Gale and Wixson 1979). Depending on stack height and wind conditions, cadmium pollution may be restricted to an even smaller area. Dugdale and Hummel (1978) found elevated cadmium levels (23.3 ppm) $\frac{1}{2}$ mile from a smelter at Belledune, N.B., but no difference between surface and underground soil samples 2 miles from the smelter. Only minimal levels were found beyond 1 km from a smelter in Missouri (Van Hook et al. 1978). Cadmium pollution was confined to a very localized area in a cove on the Hudson River contaminated by industrial discharge between 1952 and 1971. In the vicinity of the outfall, bottom sediments contained cadmium concentrations in excess of 18,000 ppm dry weight. Levels drop as low as 33 ppm about 1000 feet from the outfall (Kneip 1978, Kneip and Hazen 1979). Kneip (1978) concludes that "although the study area has been heavily contaminated by both cadmium and nickel for a number of years, none of the findings of this or other studies has shown a serious ecosystem effect to exist". Kneip (1978) suggests that planktonic species as well as muskrats and turtles in the area may be adversely affected but there is no conclusive evidence. There is a limited effect on benthic organisms in areas with sediment concentrations exceeding 1000 µg/g and the effect is very significant where concentrations exceed 10,000 µg/g (Kneip 1978).

As Fleischer et al. (1974) conclude, "our ignorance of the effects of cadmium in natural or polluted systems is almost total".

Based on the scanty data available on the uptake, concentration and effects of cadmium in natural systems, four main types of effects on wildlife appear likely to occur (after Fleischer et al. 1974):

1) direct toxicity to plants and animals in locally polluted areas, for example, near smelters.

2) cumulative toxicity to predatory animals which eat the kidney and liver of their vertebrate prey.

3) cumulative toxicity to animals such as seagulls which feed regularly on molluscs or to animals eating plants from polluted areas.

4) disruption of food chains through lethal toxicity or adverse effects on reproduction in fish and lower plants and animals.

It appears cadmium does not pose a widespread threat to wildlife presently and is not likely to in the future. However, the situation of cadmium in the environment is still poorly understood. As Gale and Wixson (1979) state, "more precise definitions of toxic effects and hazardous levels for normal terrestial and aquatic organisms are badly needed". Considering its high toxicity and persistence in the environment, a close scrutiny of this metal should be maintained.

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Appendix I

Tissue levels of cadmium in wild birds and mammals from various geographic locations.

Values in standard type were cited in the literature; values in parentheses were calculated on the basis of a tissue water content of 75 percent.

Some values represent a mean for several individuals cited in the literature.

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
BIRDS						
Fulmar *(Fulmarus glacialis)*	St. Kilda, Scotland	kidney	(8.20)-(60.00)	32.80-240.00	1976	101, 22
	?	liver	(9.25)	37.00	1968-1974	115
Manx Shearwater *(Puffinus puffinus)*	St. Kilda, Scotland	kidney	(16.75)-(57.75)	67.00-231.00	1976	101, 22
Ashy Petrel *(Oceanodroma homochroa)*	California	liver	(13.30)	53.20	1968, 1969, 1971	2
		breast	(2.00)	8.00		
		bone	(0.47)	1.88		
Leach's Petrel *(Oceanodroma leucorhoa)*	St. Kilda, Scotland	kidney	(17.13)-(32.00)	68.50-128.00	1976	101, 22
Wilson's Petrel *(Oceanites oceanicus)*	Antarctica	liver	(5.08), (5.18)	20.30, 20.70	1970	2
		breast	(0.81), (0.92)	3.25, 3.67		
		bone	(0.36), (0.22)	1.42, 0.86		
Storm Petrel *(Hydrobates pelagicus)*	St. Kilda, Scotland	kidney	(7.55)-(13.23)	30.20-52.92	1976	101
Snow Petrel *(Tagodroma nivea)*	Antarctica	liver	(6.93)	27.72	1970	2
		breast	(1.43)	5.72		
		bone	(0.22)	0.88		
Brown Pelican *(Pelicanus occidentalis)*	Louisiana	egg	0.004	(0.016)	1971-1973	16
Gannet *(Sula bassana)*	Britain	liver	(1.18)	4.72	1968-1974	115
	Scotland	egg	(0.08), (0.10)	0.32, 0.40	1976	101, 22
Canada Goose *(Branta canadensis)*	New York State	muscle	2.36	(9.44)	?	10
Canvasback *(Aythya valisineria)*	New York State	liver	4.00	(16.00)	?	10

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
Greater Scaup *(Aythya marila)*	New York State	liver	2.00	(8.00)	?	10
Bufflehead *(Bucephala albeola)*	New York State	muscle	2.00	(8.00)	?	10
Common Eider *(Somateria mollissima)*	?	liver	(2.50)	10.00	1968-1974	115
	Norway	egg	(0.25)	1.00	1972-1975	79
		kidney	(6.25)	25.00		
		liver	(3.25)	13.00		
		muscle	(0.50)	2.00		
Ruddy Duck *(Oxyura jamaicensis)*	New Jersey	liver	0.61	(2.44)	1973	155
White-winged Scoter *(Melanitta deglandi)*	New York State	liver	2.0	(8.0)	?	10
Cooper's Hawk *(Accipiter cooperii)*	Arizona, New Mexico	egg	0.12	(0.48)	?	140
Sparrow Hawk *(Accipiter nisus)*	Avonmouth, England	kidney	(16.65)	66.60	?	89
		kidney	(14.33)	57.32		
		liver	(1.35)	5.40		
		lung	(0.38)	1.52		
		kidney	(0.93)	3.82		
		kidney	(0.95)	3.80		
		liver	(0.18)	0.72		
		lung	(0.05)	0.20		
Kestrel *(Falco sparverius)*	Ithaca, New York	egg	0.08	(0.32)	1969, 1970	80
Ruffed Grouse *(Banasa umbellus)*	?	liver	0.88	(3.52)	?	130
Pheasant *(Phasianus colchicus)*	Ohio	muscle	0.17	(0.68)	1971-1974	32

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry level	Date	Reference
Great Blue Heron *(Ardea herodias)*	Lake Erie	muscle	N.D.-1.98	(N.D.)-(7.92)	1972	63
American Coot *(Fulica americana)*	South Dakota	whole bird	0.08	(0.32)	1975, 1976	56
Oystercatcher *(Haematopus ostralegus)*	?	liver	(7.25)	29.0	1968-1974	115
Herring Gull *(Larus argentatus)*	Bristol Channel, England	egg	0.06, 0.03	(0.24), (0.12)	1971, 1972	117
	Great Lakes, Canada	liver	0.11-1.81	(0.44)-(7.24)	1979	a
Lesser Black-backed Gull *(Larus fuscus)*	Bristol Channel, England	egg	0.06	(0.24)	1971-1972	117
	Norway	kidney	(2.50)	10.00	1972-1975	79
		liver	(1.00)	4.00		
		muscle	(0.25)	1.00		
Franklin's Gull *(Larus pipixcan)*	South Dakota	whole bird	0.31	(1.24)	1975, 1976	56
Black-headed Gull *(Larus ridibundus)*	Mediterranean	liver	0.22-2.60	(0.88)-(10.40)	1975, 1976	152
		muscle	0.25-1.90	(1.00)-(7.60)		
		kidney	0.36-2.10	(1.44)-(8.40)		
		brain	N.D.-1.40	(N.D.)-(5.60)		
Kittiwake *(Risa tridactyla)*	Lundy, British Isles	egg	1.30, 1.70	(5.20), (6.80)	1972	114
Razorbill *(Alca torda)*	St. Kilda, Scotland	kidney	(3.65)-(4.55)	(14.6)-(18.2)	1976	101, 22
Puffin *(Fratercula arctica)*	St. Kilda, Scotland	kidney	(18.78)-(31.25)	75.12-125.00	1976	101, 22
		egg	(0.60)	2.40	1969, 1970	116
		whole bird	(1.22)	4.88		
	Scotland, East Coast	liver	(1.30), (3.63)	5.20, 14.52		
Crow *(Corvus brachyrhynchos)*	South Dakota	whole bird	0.03	(0.12)	1975, 1976	56

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
Song Thrush *(Turdus philomelos)*	Avonmouth, England	kidney	(96.75)	387.00	?	89
Starling *(Sturnus vulgaris)*	U.S.A. (50 sites)	whole bird	0.48	(1.92)	1973	156
			0.04	(0.15)	1971	90
	South Dakota		0.10	(0.40)	1975, 1976	56
MAMMALS						
Common Shrew *(Sorex araneus)*	Wales	whole animal	(<0.25)	<1.00	1976	123
Polar Bear *(Thalarctos maritimus)*	Franklin District, N.W.T.	kidney	0.73	(2.92)	1972-1975	13
Raccoon *(Procyon lotor)*	Florida	kidney	2.48	(9.92)	?	62
Coyote *(Canis latrans)*	?	kidney	0.36	(1.44)	?	130
California Sea Lion *(Zalophus californianus)*	Oregon Coast	liver	1.61-3.46	(6.44)-(13.84)	1970, 1971, 1973	21
		kidney	7.22-12.00	(28.88)-(48.00)		
		muscle	0.07-0.16	(0.28)-(0.64)		
		heart	0.14	(0.56)		
		cerebellum	0.03-0.08	(0.13)-(0.32)		
		cerebrum	0.03-0.17	(0.13)-(0.68)		
		fat	0.04	(0.17)		
	?	kidney	60.00	(240.00)	1970, 1971	1
		liver	6.00	(24.00)		
Fur Seal *(Callorhinus ursinus)*	Mexico to Bering Sea	liver	0.50-4.60	(2.00)-(18.40)	1970, 1971	1
		kidney	0.10-15.60	(0.40)-(62.40)		

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
Common Seal *(Phoca vitulina)*	Wadden Sea, Netherlands	liver	0.05-0.30	(0.20)-(1.20)	?	78
	East Anglia, West Scotland	liver	0.20-1.10	(0.80)-(4.40)	1969, 1970	124
		kidney	0.10-1.90	(0.40)-(7.60)		
		spleen	0.13	(0.52)		
		brain	0.15	(0.60)		
		muscle	0.13	(0.52)		
		claw	0.52	(2.08)		
		rib bone	0.41	(1.64)		
Common Seal *(Phoca vitulina)* } pooled data Grey Seal *(Halichoerus grypus)*	British Waters	kidney	2.20-11.60	(8.80)-(46.40)	1969?-1972	60
Ring Seal *(Phoca hispida)*	Pond Inlet, N.W.T.	kidney	0.00	(0.00)	1972-1975	b
			0.00	(0.00)		
			0.00	(0.00)		
			5.58	(22.32)		
	Holman Island, N.W.T.		7.04	(28.16)	1977	c
			5.31	(21.24)		
			4.35	(17.40)		
			3.64	(14.56)		
			3.23	(12.92)		
			7.40	(29.60)		
			3.26	(13.04)		
			3.62	(14.48)		
	Rankin Inlet, N.W.T.		0.55	(2.20)	1972-1975	b
			0.36	(1.44)		
			0.59	(2.36)		
			7.60	(30.40)		
			14.40	(57.60)		
			13.70	(54.80)		
Seal *(Phocidae)*	Spence Bay, N.W.T.	kidney	1.89	(7.56)	1972-1975	b
	Franklin District, N.W.T.		1.40	(5.60)	1972-1975	13
			1.89	(7.56)		
			(4.66)	18.46		
	Hudson's Bay		5.32	(21.28)	1972-1975	13

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
Bearded Seal *(Erignathus barbatus)*	Rankin Inlet, N.W.T.	kidney	0.03	(1.20)	1972-1975	b
Rock Squirrel *(Spermophilus varigatus)* / Pack Rat *(Neotoma cineren)*	Utah, pooled samples	liver	(0.32)-(6.66)	1.3-26.70	?	135
		bone	(0.75)-(1.93)	3.0-7.70		
Squirrel *(Sciurus carolinensis)*	Jacksonville, Florida	kidney	2.04-15.91	(8.16)-(63.64)	?	93
Squirrels *(Sciurus carolinensis and S. niger)*	Ohio	muscle	0.20	(0.80)	1971-1974	32
Squirrel *(Scirus sp.)*	?	liver	0.73	(2.92)	?	130
White-footed Mouse *(Peromyscus leucopus)*	Pennsylvania	liver	0.12-0.15	(0.48)-(0.60)	1972, 1973	7
		kidney	0.18-0.66	(0.72)-(2.64)		
	Ohio	muscle	N.D.-0.50	(N.D.)-(2.00)	1971-1974	32
		liver	N.D.-0.43	(N.D.)-(1.72)		
Field Mouse *(Apodemus sylvaticus)*	North Wales, U.K.	whole animal	(<0.25)-(0.75)	<1.0-3.0	1976	123
	South Wales, U.K.	bone	(0.04)-(0.62)	0.14-2.49	?	66
		kidney	(0.10)-(9.93)	0.40-39.72		
		liver	(0.02)-(2.46)	0.08-9.84		
		brain	(0.01)-(0.20)	0.04-0.78		
		muscle	(0.003)-(0.09)	0.01-0.36		
		total body	(0.015)-(0.66)	0.06-2.64		
Bank Vole *(Clethrionomys glareolus)*	South Wales, U.K.	kidney	(0.20)-(4.20)	0.78-16.80	?	66
		liver	(0.15)-(1.28)	0.59-5.12		
		total body	(0.01)-(0.22)	0.05-0.87		
Field Vole *(Microtus agrestis)*	South Wales, U.K.	kidney	(0.07)-(2.23)	0.29-8.91	?	66
		liver	(0.01)-(0.27)	0.04-1.06		
		total body	(0.01)-(0.16)	0.04-0.64		
	North Wales, U.K.	whole animal	(<0.25)	<1.0	1976	123

Species	Location	Tissue	Cadmium level ppm wet weight	Cadmium level ppm dry weight	Date	Reference
Muskrat *(Ondatra zibethica)*	Pennsylvania	kidney	0.17-1.07	(0.68)-(4.28)	1974	43
Porcupine *(Erethizon dorsatum)*	?	kidney	0.85-7.30	(3.40)-(29.20)	?	130
		heart	0.35	(1.40)		
Rabbit *(Sylvilagus floridanus)*	Ohio	muscle	0.06	(0.24)	1971-1974	32
		kidney	0.46-1.86	(1.84)-(7.44)		
	Pennsylvania	liver	1.21-1.67	(4.84)-(6.67)	1972, 1973	7
		kidney	6.72-9.41	(26.88)-(37.64)		
		bone	7.83-7.92	(31.31)-(31.67)		
White-tailed Deer *(Odocoileus virginianus)*	Ohio	muscle	0.09	(0.36)	1971-1974	32
	?	kidney	2.07	(8.28)	?	130
Mule Deer *(Odocoileus hemionus)*	Montana	kidney	(0.68)	2.70	?	100
Whale *(Cetacea)*	Franklin District, N.W.T.	kidney	(15.75)	62.99	1972-1975	13
		kidney	1.05	(4.20)		
		liver	0.02	(0.08)		
		liver	1.98	(7.92)		
		liver	0.03	(0.12)		
Dolphin *(Delphinus delphinus)*	French Mediterranean Coast	blubber	N.D.	N.D.	1976	153
Short-finned Pilot Whale *(Globicephala macrorhynchus)*	Georgia	blubber	0.34-0.75	(1.36)-(3.00)	1977	142
		liver	11.30-19.80	(45.20)-(79.20)		
		kidney	27.10-41.80	(108.40)-(167.20)		

N.D. - Non detectable

a - Andy Gilman, 1979, personal communication. Unpublished data from the National Registry, Toxic Chemical Residues.

b - Fisheries and Environment Canada (1972-1975), Commercial and Lake Survey Monitoring Data. Personal communication with G.W. McGregor, Industry Services Branch, Freshwater Institute, Winnipeg. as cited in Sergy, G. 1978. Environmental Distribution of Cadmium in the Prairie Provinces and Northwest Territories, Report EPS 3-NW-78-2.

c - Health and Welfare Canada, 1977. Personal communication with Dr. R. Eaton, Northern Medical Research Unit, Charles Camsell Hospital, Edmonton. as cited in Sergy, G. 1978. Environmental Distribution of Cadmium in the Prairie Provinces and Northwest Territories, Report EPS 3-NW-78-2.

Appendix II. Some LC_{50} concentrations of cadmium in water for miscellaneous species of aquatic invertebrates and fish

Species	Cadmium concentration in water LC_{50} (ppm)	Duration	Reference
INVERTEBRATES			
Grass shrimp *Palaemontes vulgaris*	0.42	96 hr.	Eisler 1971
Sand shrimp *Crangon septemspinosa*	0.32	"	"
Hermit crab *Pagurus longicarpus*	0.32	"	"
Green crab *Carcinus maenus*	4.10	"	"
Common soft-shell clam *Mya areraria*	2.20	"	"
Common starfish *Asterias forbesi*	0.82	"	"
Atlantic oyster drill *Urosalpinx cinerea*	6.60	"	"
Eastern mud snail *Nassarius obsoletus*	10.50	"	"
Sandworm *Nereis virens*	11.0	"	"
Blue mussel *Mytilus edulis*	25.0	"	"
FISH			
Striped killifish *Fundulus majalis*	21.0	96 hr.	Eisler 1971
Sheepshead minnow *Cyprinodon variegatus*	50.0	"	"
Mummichog *Fundulus fundulus*	55.0	"	"
Fathead minnow *Pimephales promelas*	0.150	5 wk.	Pickering & Gast 1972
	0.089	9 wk.	" "
	0.068	9 mo.	" "

Fathead minnow *Pimephales promelas* (cont'd)	0.9	96 hr.	Tarzwell & Henderson 1960
	5.0	96 hr.	" "
	1.05	96 hr.	Pickering & Henderson 1966
	72.6	96 hr.	" "
Bluegill *Lepomis macrohirus Rafinesque*	.080	11 mo.	Eaton 1974
	1.94	96 hr.	Pickering & Henderson 1966
Goldfish *Carassius auratus*	2.34	96 hr.	Pickering & Henderson 1966
Guppy *Lebistes reticulatus*	0.056	?	Shaw & Lowrance 1956
	1.27	96 hr.	Pickering & Henderson 1966
Green sunfish *Lepomis cyanellus*	2.84	96 hr.	Pickering & Henderson 1966
	66.0	96 hr.	" "
Rainbow trout *Salmo gairdnerii*	0.008 to 0.01	7 day	Ball 1967

Appendix III. Lethal concentrations* of cadmium in water in some species of invertebrates and fish

Species	Lethal conc. ppm Cd in water	Duration	Reference
Eels *Anguilla japonica*	0.18	50 hr.	Doudoroff & Katz 1953
	1.83	18.4 hr.	" "
Flatworm *Polycelis nigra*	2.7	?	Jones 1940
Goldfish *Carassius auratus*	0.0165	$8\frac{1}{2}$-18 hr.	Ellis 1937
Stickleback *Gasterosteus aculeatus*	0.42	?	Anderson 1948
	0.3	7 days	Jones 1957
	0.7	4 days	"
	3.0	2 days	"
	7.0	1 day	"
Mummichog *Fundulus fundulus*	6.0	36 hr.	Doudoroff & Katz 1953

* Not known if lethal concentrations refer to LC_{50}, LC_{90}, LC_{100}, etc.

Appendix IV. Recent references on water quality and chronic toxicity of cadmium to fish

U.S. E.P.A. 1976. Quality Criteria for Water. U.S. E.P.A. (Issued as EPA 440-9-76-023).

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