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Ecotoxicological Profiles for Selected Metals and Other Inorganic Chemicals

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PREFACE

The purpose of this technical memorandum is to present profiles for chemicals of potential ecological concern. This work was performed under Work Breakdown Structure 1.4.12.2.3.04.05.02 (Activity Data Sheet 8304). These profiles contain information concerning the relationship between exposure and response for the chemicals of potential ecological concern that are used to perform the risk characterization. Inclusion of these profiles in the assessment document will provide reviewers, stakeholders, and risk managers with the information needed to independently evaluate the risk characterization.

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TABLES

ABBREVIATIONS

ACGIH	Annual Conference of Governmental Industrial Hygienists
APHA	American Public Health Association
ATP	adenosinetriphosphate
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	biological concentration factor
BW	body weight
CEC	cation exchange capacity
CV	chronic value
DNA	deoxyribonucleic acid
EC	effective concentration
EPA	U.S. Environmental Protection Agency
FAV	final acute value
FCV	final chronic value
GER	granular endoplasmic reticulum
HCN	hydrocyanic acid
LC	lethal concentration
LCT	lowest concentration tested
LCV	lowest chronic value
LD	lethal dose
LDH	lactic dehydrogenase
LOAEL	lowest-observed-adverse-effect level
LOEC	lowest-observed-effect concentration
NAS	National Academy of Science
NAWQC	National Ambient Water Quality Criteria
NOAEL	no-observed-adverse-effect level
NOEC	no-observed effect concentration
NRC	National Research Council
NRCC	National Research Council of Canada
OC	organic carbon
OECD	Office of Environmental Compliance and Documentation
PR	population reduction
RNA	ribonucleic acid
SAV	secondary acute value
SCV	secondary chronic value
SQC	sediment quality criteria
USFWS	U.S. Fish and Wildlife Service

EXECUTIVE SUMMARY

Ecological Risk Assessments for contaminated sites screen the chemicals detected on the site to determine chemicals of potential ecological concern (COPECs). Those COPECs are then assessed in detail to determine which are causally associated with significant risks to assessment endpoints. The effects assessment component of this definitive assessment must develop ecotoxicological profiles for the COPECs. The profiles contain the information concerning the relationship between exposure and response for the COPECs that is used to perform the risk characterization. Inclusion of the profile in the assessment document provides reviewers, stakeholders, and risk managers with the information needed to independently evaluate the risk characterization.

This document contains generic ecotoxicological profiles for several elements that have been COPECs for sites on the U.S. Department of Energy's (DOE's) Oak Ridge (Suter et al. 1995), Portsmouth (Oak Ridge National Laboratory 1994), or Paducah (DOE 1994) reservations. Each profile contains the following elements: background, toxicity to freshwater aquatic life, toxicity to benthic invertebrates in sediment, toxicity to plants, toxicity to wildlife, toxicity to soil invertebrates and heterotrophic processes, and references. The report also explains how these generic ecotoxicological profiles are adapted for use in individual assessments.

1. INTRODUCTION

Ecological Risk Assessments for contaminated sites screen the chemicals detected on the site to determine chemicals of potential ecological concern (COPECs). Those COPECs are then assessed in detail to determine which are causally associated with significant risks to assessment endpoints. In the effects assessment component of a definitive assessment, ecotoxicological profiles for the COPECs must be developed and presented. The profiles contain the information about the relationship between exposure and response for the COPECs that is used to perform the risk characterization. Inclusion of the profile in the assessment document provides reviewers, stakeholders, and risk managers with the information needed to independently evaluate the risk characterization.

Note that this document is complementary to the ecotoxicological benchmarks documents. The benchmarks are used in screening the contaminants for COPECs, and these profiles contain more detailed toxicological information to allow interpretation of risks. These ecotoxicological profiles are developed, reviewed, and revised on a schedule that is independent of the schedule for the benchmarks documents. Thus, occasionally the user will find that data and data sets are incorporated into the profiles but the benchmarks are not, or the converse.

This document contains generic ecotoxicological profiles for several elements that have been COPECs for sites on the U.S. Department of Energy's (DOE's) Oak Ridge (Suter et al. 1995), Portsmouth (Oak Ridge National Laboratory [ORNL] 1994), or Paducah (DOE 1994) reservations. Each profile consists of the following seven sections:

- **Background**—Properties of the element, including natural occurrence, sources, forms that occur in the environment, and the influence of environmental conditions on forms and concentrations, are briefly described.

- **Toxicity to Freshwater Aquatic Life**—Acute and chronic toxicity of the chemical to aquatic animals and plants, bioaccumulation in aquatic organisms, mode(s) of action, and water quality criteria are summarized. If extensive toxicity data are available, the data are summarized in tabular form.

- **Toxicity to Benthic Invertebrates in Sediment**—Information concerning toxicity of the chemical in sediment to benthic infauna and epifauna is summarized. The data include both laboratory tests with spiked sediments and field studies. Although these profiles are for freshwater sites, toxicity data for estuarine sediments are included because few data are available for freshwater and because U.S. Environmental Protection Agency Region IV has judged that the estuarine data are relevant to freshwater sites. If extensive toxicity data are available, they are summarized in tabular form.

- **Toxicity to Plants**—Information concerning toxicity of the chemical in soil to terrestrial vascular plants is summarized. The data include information on toxicity in soils and in soil solutions (compiled separately). The latter are relevant to cases in which plants, such as those growing near seeps, are exposed to contaminated groundwater. If extensive toxicity data are available, the data are summarized in tabular form.

- **Toxicity to Wildlife**—Information concerning toxicity of the chemical to avian and mammalian wildlife through oral exposure (e.g., food, water, and soil ingestion) is summarized. Information concerning mode of action and bioaccumulation, if available, is also summarized.

- **Toxicity to Soil Invertebrates and Heterotrophic Processes**—Information concerning toxicity of the chemical in soil to soil invertebrates (i.e., almost entirely earthworms) and to soil microbial processes such as decomposition (e.g., carbon mineralization) and nutrient cycling processes (e.g., nitrification and sulfur reduction) is summarized. If extensive toxicity data are available, they are summarized in tabular form.

- **References**—References are presented separately for each chemical to facilitate use of the profiles.

These ecotoxicological profiles are intended to be generic and therefore must be adapted for use in individual assessments. The following are suggested modifications:

- Only relevant sections should be included in the individual asssessments. For example, a chemical may be a COPEC for the fish community and an avian species, but not other endpoints. In such a case, the assessment should delete sections of the chemical's profile that deal with toxicity to sediment and soil invertebrates, soil heterotrophic processes, and mammalian wildlife.

- Relevant chemical, ecological, or ecotoxicological information that is available for the site should be incorporated. For example, studies performed by the Biological Monitoring and Abatement Program at ORNL in Bear Creek found nickel to be toxic at lower concentrations than had been reported previously. Similarly, the peculiar environmental chemistry of Bear Creek led to the occurrence of nitrate salts of metals, which, even in the absence of site-specific testing, would cause the interpretation of toxicity data to be modified. All such site-specific information that influences the exposure–response relationship for a chemical should be incorporated into the profile for the assessment.

- Some ecotoxicological responses are known to be influenced by environmental parameters. The data presented in the profiles are intended to be relevant to the DOE's Oak Ridge, Paducah, and Portsmouth reservations. However, these data may need to be modified or certain data excluded for sites with conditions different from those at these three sites. In particular, toxicity of many metals is known to be influenced by water hardness. If hardness at a site differs significantly from the 100 mg/L level—a realistically conservative value for the three DOE sites—then more appropriate data should be selected or formulas such as those used to calculate the National Ambient Water Quality Criteria for cadmium, chromium, copper, lead, and zinc should be used to correct for differences in hardness. Similarly, sediment benchmarks based on equilibrium partitioning should be adjusted for site-specific organic matter content if organic matter content is significantly different from 1%.

- Ecotoxicological issues may arise at a site that require additional researching of the literature. The resulting information should be added to the profile.

Although these generic ecotoxicological profiles should save considerable effort in ecological risk assessment, they should not be mindlessly cut and pasted into documents.

The amount and types of information available for a chemical constrain the choice of techniques for risk characterization. Therefore, assessors should carefully review the information presented in these toxicity profiles to determine which analytical techniques best characterize the information available

concerning the relationship between exposure and effects for each endpoint. For example, if there are consistent, standard toxicity data for several species within an endpoint community, species sensitivity distributions are potentially appropriate. In contrast, if data are sparse or inconsistent, it may be more appropriate to select the test results that are most relevant to the endpoint and focus on the exposure–response relationship found in that test. The toxicity profiles are meant to be used as input to the risk characterization, and not simply as supporting narrative.

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2. ALUMINUM

2.1 BACKGROUND

Aluminum (Al) is the third most abundant element, accounting for 8.1% of the earth's crust by weight (Sparling and Lowe 1996). Its natural occurrence is largely restricted to highly insoluble complex minerals (Freeman and Everhart 1971). It can be highly toxic to aquatic biota under some circumstances, but its toxicity is strongly dependent on pH, water hardness, and organic matter content. In general, the more sensitive an organism is to a low pH, the stronger the toxic response to aluminum concentrations with falling pH levels (Baker and Schofield 1982). Aluminum is amphoteric, i.e., it is more soluble in both acidic and basic solutions than in approximately neutral solutions, with its highest toxicity occurring around pH 5.5 (Freeman and Everhart 1971; Baker and Schofield 1982). The U.S. Environmental Protection Agency's (EPA's) National Ambient Water Quality Criteria (NAWQC) for aluminum (EPA 1988) assume a pH range of 6.5 to 9.0 within which aluminum occurs primarily in the form of monomeric, dimeric, and polymeric hydroxides. Toxicities of aluminum in the field may be substantially lower than indicated by dissolved aluminum values because of complexation with humic and fulvic acids. At pH values below 6.5, however, aluminum may be substantially more toxic than indicated by the EPA criteria because low pH favors the formation and solubilization of cationic aluminum (Al^{3+}). The aquatic toxicity tests used to calculate the NAWQC for aluminum are relevant to situations in which an acidic solution of aluminum is neutralized in ambient water, forming the hydroxide flocs as reported in the tests. Similar situations may occur when an acidic waste or an acidified tributary enters a well-buffered stream or lake. Their relevance to other situations, however, is questionable.

2.2 TOXICITY TO AQUATIC LIFE IN FRESH WATER

2.2.1 Acute Toxicity

Between pH 6.5 and 9.0, invertebrate acute toxicities (i.e., LC_{50} or EC_{50}) of aluminum reported by EPA (1988) ranged between 1.8 mg/L for the nematode *(Caenorhabiditis elegans)* and >79.9 mg/L for midge larvae (*Tanytarsus dissimilis*). Acute toxicities for fish ranged from 3.6 mg/L to >50.0 mg/L (Table 2-1). Acutely lethal concentrations at acidic pHs (<6), particularly in soft waters, can be much lower. If these conditions occur at a site, the appropriate literature should be consulted (Sparling and Lowe 1996; Baker and Schofield 1982; Baker et al. 1990).

2.2.2 Chronic Toxicity

Chronic values (i.e., results of life cycle or early life stage tests) for aluminum in circumneutral water reported by EPA (1988) ranged from 0.742 mg/L for *Daphnia magna* to 3.29 mg/L for fathead minnows (Table 2-1). Rainbow trout exposed for 45 days to 0.5 and 5.1mg/L Al (nominal pH 7.0) averaged 32.5 and 24.1% of the control weight, respectively (Freeman and Everhart 1971). However, at pH 4.5 and 5.5 aluminum (0.3 mg/L for 67 days) induced fatal behavioral abnormalities in brook trout, including decreased swimming response, ability to feed, and ability to evade predators (Cleveland et al. 1986). Mortality of brook trout eggs was not influenced by the aluminum concentration (0.3 mg/L) at pH between 5.5 and 7.2. However, at pH 4.5, 54.4% of the eggs did not survive (0.3 mg/L Al) after 67d (Cleveland et al. 1986). Baker and Schofield (1982) found that aluminum (0.1–0.5 mg/L) in acidic

Table 2-1. Toxicity of aluminum to aquatic organisms in circumneutral water

Conc.[1] (mg/L)	Species/Effect	Reference[2]
>79.9	Midge larvae (*Tanytarsus dissimilis*) LC_{50}	
79.0	Nematode (*Caenorhabditis elegans*) 24hr LC_{50}	
>50.0	Green sunfish (*Lepomis cyanellus*) LC_{50}	AQUIRE 1996
>49.8	Yellow perch (*Perca flavescens*) LC_{50}	
>47.9	Channel catfish (*Ictalurus punctatus*) LC_{50}	
>40.0	Chinook salmon (*Oncorhynchus tshawytsca*) LC_{50}	
38.2	Water flea (*Daphnia magna*) EC_{50}	
36.9	Water flea (*Ceriodaphnia* sp.) 48h LC_{50}	AQUIRE 1996
35.0	Fathead minnow (*Pimephales promelas*) LC_{50}	
30.6	Snail (*Physa* sp.) LC_{50}	
>23.0	Planaria (*Dugasia tigrina*) LC_{50}	
>22.6	Stonefly (*Acroneuria* sp.) LC_{50}	
22.0	Scud (*Gammarus pseudolimnaeus*)LC_{50}	
10.5	Rainbow trout (*Oncorhynchus mykiss*) 48h LC_{50}	
8.60	Rainbow trout (*Oncorhynchus mykiss*) 96h LC_{50}	AQUIRE 1996
3.60	Brook trout (*Salvelinus fontinalis*) LC_{50}	

Table 2-1 (continued)

Conc.[1] (mg/L)	Species/Effect	Reference[2]
3.29	Fathead minnow (*Pimephales promelas*) CV	
>3.00	Rotifer (*Brachionus calyciforus*) 24h LC_{50}	AQUIRE 1996
2.00	Nematode (*Caenorhabditis elegans*) 48hr LC_{50}	AQUIRE 1996
1.91	Water flea (*Ceriodaphnia dubia*) CV	
1.90	Nematode (*Caenorhabditis elegans*) 72hr LC_{50}	AQUIRE 1996
1.80	Nematode (*Caenorhabditis elegans*) 96hr LC_{50}	AQUIRE 1996
0.75	Acute NAWQ	
0.74	Water flea (*Daphnia magna*) CV	
0.09	Chronic NAWQ	

[1]Concentrations given as Al, not the compound.
[2]EPA 1988 unless otherwise cited.

solutions (pH 4.6–7.06) decreased survival and growth of white sucker larvae after 13d exposure. In contrast, 95% of all post-larval brook trout survived at pH 4.2–5.5 when no aluminum was added.

2.2.3 Toxicity to Aquatic Plants

Aluminum inhibited the growth of the diatom, *Cyclotella meneghiniana*, at 0.810 mg/L and caused mortality at 6.48 mg/L (Rao and Subramanian 1982, cited in EPA 1988). Concentrations of aluminum ranging from 0.40 to 0.90 mg/L were chronically toxic to the green alga, *Selenastrum capricornutum*. At concentrations ranging from 0.02 to 0.20 mg/L Al^{3+} the growth and activity of acid photophase of *S. capricornutum* were significantly inhibited. The activity of G6PDH was decreased at Al^{3+} levels higher than 0.02 mg/L (Kong and Chen 1995).

2.2.4 Bioaccumulation

Estimated steady state bioconcentration factors for aluminum in brook trout, which were inversely related to pH, were 215 at pH 5.3, 123 at pH 6.1, and 36 at pH 7.2 (Cleveland et al. 1991). Aluminum was found at 350, 3,000, and 11,000 μg/g BW at exposure concentrations of 0.02, 0.32, and

1.02mg/L (pH 6.5) in *D. magna* (Havas 1985). Based on body dry weight, Pynnonen (1990) found a bioconcentration factor ranging from 166.8 to 414.3 in freshwater clams, *Unio pictorum*. After a 3-week exposure to 0.3 mg/L Al, high accumulation of aluminum in the kidney suggests that it is the final target organ, while low level presence of aluminum on the gills identifies the presence of metals in the sample area.

2.2.5 Aquatic Mode of Action

The exact mechanisms of aluminum's physiological effects are still under investigation and not completely understood. Mortality of fish exposed to aluminum and low pH may be the result of (1) respiratory stress caused by gill damage and mucous clogging of the gill membrane, (2) the loss of sodium and chloride ions (Brumbaugh and Kane 1985, as cited in Lewis et al. 1990), and (3) the adsorption and nucleation of aluminum polymers at surface interfaces (Baker and Schofield 1982). Aluminum may hamper the activity of calmodulin which is active in numerous biochemical processes (Siegel and Haung 1983, as cited in Lewis et al. 1990). It has been shown that trace metals, such as aluminum, compete with H^+ for exchange sites on the gill surface (Pagenkopf 1983, as cited in Havas 1985). Muniz and Levistad (1980, as cited in Lewis et al. 1990) found that the presence of aluminum increased the loss of chloride from plasma and decreased blood oxygen tension in the brown trout. The fact that aluminum uptake and mortality increase with high pH levels may indicate that the aluminum ion may bind or precipitate on biological membranes (Freda and McDonald 1990). Verbost et al. (1992) showed that the inhibition of calcium uptake by the common carp is time and doseage dependent (>0.1 mg/L Al). In highly acidic waters, aluminum decreases RNA syntheses which may be due to disruption or deactivation of metabolic pathways (Cleveland et al. 1986). Observable effects include coughing response, hyperventilation, and excessive mucus clogging of gills. Sublethal concentrations of aluminum have been shown to cause histopathological changes in the liver, kidney, skin, muscle, and gills and interfere with reproductive physiology in trout. Many studies have shown that aluminum may inhibit or render useless the hatching enzyme that digests the vitelline membrane in *Rana pipens* embryos (Katagiri 1976; Yoshizaki 1978; Freda and Dunson 1984, all as cited in Freda and McDonald 1990; Clark and LaZerte 1985, as cited in Freda et al. 1990). In *Daphnia magna*, it has been shown that aluminum interferes with salt regulation and leads to death when sodium and chlorine concentrations are reduced (Havas 1985).

2.2.6 Water Quality Criteria

NAWQC indicated that freshwater aquatic organisms and their uses should not be affected unacceptably, when the pH is between 6.5 and 9.0, if the 4-day average concentration of aluminum does not exceed 0.087 mg/L more than once every 3 years on the average and if the 1-hour average concentration does not exceed 0.750 mg/L more than once every 3 years on the average (EPA 1988).

2.3 TOXICITY TO BENTHIC INVERTEBRATES IN SEDIMENT

Information was not found for the toxicity of aluminum in sediment. There are no sediment quality criteria (SQC) for aluminum.

2.4 TOXICITY TO PLANTS

2.4.1 Toxicity to Plants in Soil

Aluminum uptake is dependent on pH and plant species. In acidic soils, aluminum levels are greater in roots and older leaves than in younger foliage (Will and Suter 1995a). Very few studies of aluminum toxicity have been conducted in soil, compared to the number of conducted in solution. Seedling establishment of white clover in a silt loam soil (pH 5.0) was reduced by approximately 30% by the addition of 50 mg/kg Al as $Al_2(SO_4)_3$, the lowest concentration tested (Mackay et al. 1990).

2.4.2 Toxicity to Plants in Solution

Will and Suter (1995a) report no-observed-effect concentrations (NOEC) and lowest-observed-effect concentrations (LOEC) for the effects of aluminum in solutions on growth of trees, horticultural and field crops, and grasses. The NOECs for tree growth range from 0.11mg/L for citrange to 162 mg/L for pine and the LOECs range from 2.7 mg/L for citrange to 270 mg/L for pine. Trees tend to exhibit symptoms of aluminum toxicity in the roots. At acidic concentrations, mean root weight and length decreased from 21 to 42% with the addition of 2.7–270.0 mg/L Al (Görransson and Eldhuset 1991). The NOECs for horticultural and field crops and grasses range from 0.05 mg/L for asparagus to 8 mg/L for barley, and the LOECs range from 0.05 mg/L for onions to 10 mg/L for barley (Will and Suter 1995). Field crops exhibit symptoms of toxicity in the roots as well as in the seedling shoot and leaf. One maize cultivar had a reduction of 23–37% root and shoot length after one day of exposure to 0.54 mg/L Al. After an exposure of 56 days at a concentration of 2.7 mg/L Al, lettuce body weight decreased by 55%. Severity of the reaction in plants depends on plant species, pH, and length of exposure (Table 2-2).

2.4.3 Phytotoxic Mode of Action

Aluminum interferes with cell division in roots; decreases root respiration; fixes P in unavailable forms in roots; interferes with uptake, transport, and use of Ca, Mg, P, K, and water; and interferes with enzyme activities (Foy et al. 1978). Symptoms of toxicity include stubby, coralloid, damaged and brittle roots; stunting; late maturity; collapse of growing points; purpling of stems; death of leaf tips and dark green leaves (Aller et al. 1990). Such damage to the roots inhibits water and nutrient absorption. Seedlings are more susceptible to damage from aluminum toxicity than are older plants. Tolerance to aluminum toxicity has been attributed to the ability of certain cultivars of wheat, barley, soybean, and snap bean to resist aluminum-induced nutrient deficiency or reduced nutrient transport (Foy 1974a, 1974b; Foy et al. 1972, as cited in Foy et al. 1978). Organic acid production has been associated with aluminum tolerance (de la Fuente et al. 1997). Aluminum has been shown to form an insoluble phosphate in the cortex of the roots inducing phosphorus deficiency (Hutchinson et al. 1971, as cited in Foy et al. 1978).

2.5 TOXICITY TO WILDLIFE

2.5.1 Toxicity to Mammals

Relative to other metals, the toxicity of aluminum is low (Sorensen et al. 1974). The principal effect of aluminum is to interfere with phosphorous metabolism; in the alimentary canal, aluminum forms insoluble compounds with phosphorous, resulting in an imbalance of calcium and phosphorous (Carriere et al. 1986). The toxicity of aluminum is sharply increased when dietary aluminum levels

Table 2-2. Phytotoxicity data for the toxicity of aluminum derived from experiments conducted in solution (Will and Suter 1995a)

Chemical form	Plant species	NOEC (mg/L)[1]	LOEC (mg/L)[2]	Parameter growth	Reference
$Al_2(SO_4)_3$	onion	-	0.05 LCT	root & shoot weight	Wheeler and Follet 1991
$Al_2(SO_4)_3$	asparagus	0.05	0.13	root & shoot weight	Wheeler and Follet 1991
$AlCl_3$	wheat	-	0.14 LCT	root elongation	Sasaki et al. 1994
$AlCl_3$	wheat	0.14	0.27	root elongation	Sasaki et al. 1994
$Al_2(SO_4)_3$	squash	0.13	0.27	root weight	Wheeler & Follet 1991
$AlCl_3$	maize	-	0.54 LCT	root elongation	Llugany et al. 1995
$KAl(SO_4)_2$	ryegrass	-	0.63 LCT	length longest root	Wong & Bradshaw 1982
$Al_2(SO_4)_3$	lettuce	0.54	1.1	air dry weight shoot	McLean & Gilbert 1927
$AlCl_3$	maize	0.54	1.35	root elongation	Llugany et al. 1995
$Al_2(SO_4)_3$	beet	-	1.8 LCT	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	beet	-	1.8 LCT	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	barley	-	1.8 LCT	air dry weight root/shoot	McLean & Gilbert 1927
$Al_2(SO_4)_3$	rye	-	1.8 LCT	air dry weight root	McLean & Gilbert 1927
$Al_2(SO_4)_3$	beet	-	1.8 LCT	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	lettuce	0.9	1.8	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	citrange	0.11	2.7	root length	Lin & Myhre 1991
$Al_2(SO_4)_3$	rice	0.27	2.7	root & shoot weight	Wallace & Romney 1977

Table 2-2 (continued)

Chemical form	Plant species	NOEC (mg/L)[1]	LOEC (mg/L)[2]	Parameter growth	Reference
$Al_2(SO_4)_3$	soybean	0.27	2.7	leaf weight	Wallace & Romney 1977
$Al_2(SO_4)_3$	lettuce	1.8	2.7	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	rye	-	3.6 LCT	air dry weight root	McLean & Gilbert 1927
$Al_2(SO_4)_3$	radish	1.8	3.6	air dry weight root/shoot	McLean & Gilbert 1927
$Al_2(SO_4)_3$	carrot	-	3.6 LCT	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	Norway Spruce	1.4	5.4	root elongation	Godbold & Kettner 1991
$AlCl_3$	barley	4	6	root & shoot weight	Macleod & Jackson 1967
$Al_2(SO_4)_3$	turnip	3.6	7.2	air dry weight shoot	McLean & Gilbert 1927
$Al_2(SO_4)_3$	oat	3.6	7.2	air dry weight root/shoot	McLean & Gilbert 1927
$Al_2(SO_4)_3$	cabbage	-	7.2 LCT	air dry weight plant	McLean & Gilbert 1927
$Al_2(SO_4)_3$	Douglas fir	4	8	root weight	Keltjens 1990
$AlCl_3$+$Al(NO_3)_3$	spruce	5.4	8.1	growth rate root	Görransson & Eldhuset 1991
$Al_2(SO_4)_3$	lemon	4.8	8.3	fresh weight; root length	Lin & Myhre 1991
$Al_2(SO_4)_3$	orange	4.8	8.3	fresh weight; root length	Lin & Myhre 1991
$Al_2(SO_4)_3$	citrumelo	4.8	8.3	fresh weight plant	Lin & Myhre 1991
$Al_2(SO_4)_3$	barley	8	10	root & shoot weight	Macleod & Jackson 1967
$Al_2(SO_4)_3$	orange	8.3	24.4	fresh weight; root length	Lin & Myhre 1991

Table 2-2 (continued)

Chemical form	Plant species	NOEC (mg/L)[1]	LOEC (mg/L)[2]	Parameter growth	Reference
$AlCl_3$	Douglas fir	16	32	root weight & length	Keltjens 1990
$AlCl_3$+$Al(NO_3)_3$	pine	162	269.8	growth rate shoot	Görransson & Eldhuset 1991

[1]All chemical concentrations in solutions and plants are expressed in milligrams of the element per liter of solution.
[2]LCT = lowest concentration tested.

reach 50% or more of the dietary P levels (Deobald and Elvehjem 1935, as cited in Scheuhammer 1987). Other effects of aluminum include neurotoxicity. Rats exposed to aluminum displayed behavioral abnormalities and had reduced acetylcholinesterase activity (Krueger et al. 1984, as cited in Scheuhammer 1987). The oral LD_{50} for mice ranges from 770 to 980 mg aluminum/kg body weight (Ondreicka et al. 1966). Mice consuming diets containing 500 to 1000 mg/kg aluminum displayed ataxia and paralysis of the hind limbs (Golub et al. 1987). Ondreicka et al. (1966) evaluated the effects of a single dose level of aluminum on mammalian reproduction. Mice received 19.3 mg aluminum/kg body weight/day (as $AlCl_3$) in drinking water for three generations. While the number of litters and offspring per litter was not reduced, growth was significantly reduced among all rat offspring in the second and third generations. Sample et al. (1996) considered the 19.3 mg/kg/d to be a chronic LOAEL for reproductive effects; a chronic NOAEL of 1.93 mg/kg/d was estimated by multiplying the LOAEL by an uncertainty factor of 0.1. In a similar study, rats received daily intragastric doses of 0, 180, 360, or 720 mg aluminum/kg body weight/day (Domingo et al. 1987) for one generation. Growth and survival of young were reduced among the groups that received 360 and 720 mg aluminum/kg/day. Other studies also report that while aluminum does not appear to affect the number of litters or number of offspring/litter, growth and survival of offspring of aluminum-exposed parents are reduced (Golub et al. 1987; Paternain et al. 1988).

Sample et al. (1997) report median soil-small mammal uptake factors for herbivore, omnivore, and general small mammals (i.e., herbivores and omnivores combined) to be 0.0088, 0.0049, and 0.0052, respectively. These factors are unitless quotients of concentration in the whole animal/concentration in soil, all on a dry weight basis.

2.5.2 Toxicity to Birds

Under normal kidney function, the retention of aluminum in body tissues and bones of birds is minimal. However, due to its interference with phosphorous and calcium metabolism, it has been suggested that aluminum may impair eggshell formation by birds, resulting in eggshell thinning (Nyholm 1981). To test this hypothesis, Carriere et al. (1986) fed breeding ring doves (*Streptopelia risoria*) a diet containing 1000 mg/kg aluminum [as $Al_2(SO_4)_2$] and adequate but reduced calcium and phosphorous, and observed reproduction. While no reproductive effects or embryonic malformations were observed at the single dosage level considered, significant reproductive effects resulted when birds were fed a diet deficient in calcium and phosphorous that contained 750 mg/kg aluminum. Sample et al. (1996) estimated a chronic NOAEL of 109.7 mg Al/kg/d based on the 1,000 mg/kg diet. Because adverse effects were not observed under an adequate nutritional regime, a chronic LOAEL for

of 3 weeks. Therefore, among birds it appears that the manifestation of toxic effects of aluminum are dependent upon the nutritional quality of their diet.

2.6 TOXICITY TO SOIL HETEROTROPHIC PROCESSES

The role of soil characteristics in determining effects of aluminum (as $AlCl_3$) toxicity to arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979) for microbes (Table 2-3). Soils were chosen with a range in pH, organic matter, and clay contents. In all soils, a concentration of 675 mg/kg Al reduced the enzyme activity between 24 and 43%. The least inhibition occurred in the soil with the highest contents of organic matter and clay. Juma and Tabatabai (1977) used three soils to test effects of aluminum on acid phosphatase activity of microbes; pH, organic matter, and clay ranged from 5.8 to 7.8%, 5.2 to 11%, and 23 to 30, respectively. For aluminum in a loam soil (pH 5.8; 5.2% organic matter), acid phosphatase activity was reduced, and alkaline phosphatase activity was reduced in another loam soil (pH 7.4, 11% organic matter) by a concentration of 675 mg/kg Al (Table 2-3). Aluminum had no effect on the activity of either enzyme in an alkaline soil (pH 7.8, 7.4% organic matter).

Table 2-3. Toxicity of aluminum to soil heterotrophic processes (Will and Suter 1995b)

Chemical form	Organisms	Medium growth	pH	%OC	EXP (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$AlCl_3$	native soil microflora	clay loam	6	2.7	0.01	Arylsulfatase activity	–	675 LCT	43	Al-Khafaji & Tabatabai 1979
$AlCl_3$	native soil microflora	loam	7	5.3	0.1	Arylsulfatase activity	–	675 LCT	24	Al-Khafaji & Tabatabai 1979
$AlCl_3$	native soil microflora	loam	7	2.9	0.1	Arylsulfatase activity	67.5	675	34	Al-Khafaji & Tabatabai. 1979
$AlCl_3$	native soil microflora	clay loam	8	3.2	0.1	Arylsulfatase activity	67.5	675	42	Al-Khafaji & Tabatabai 1979
$AlCl_3$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	67.5	675	34	Juma & Tabatabai 1977
$AlCl_3$	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	67.5	675	25	Juma & Tabatabai 1977

Note: Chemical concentrations are expressed in grams of element per kilogram of growth medium.
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls.
EXP (D) = exposure in days.
% OC = % organic carbon.

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3. ANTIMONY

3.1 BACKGROUND

Antimony (Sb) is a naturally occurring metalloid element (displaying both metallic and nonmetallic properties) existing in valence states of 3 and 5 [Budavari 1989; Agency for Toxic Substances and Disease Registry (ATSDR) 1990]. Metallic antimony and a few trivalent antimony compounds are the most significant regarding exposure potential and toxicity (ATSDR 1990). Antimony is used in metallurgical processes, paints and enamels, various textiles, rubber, and fire retardation (antimony trioxide). Some antimonials such as potassium antimony tartrate have been used medicinally as parasiticides (Beliles 1979).

3.2 TOXICITY IN WATER TO AQUATIC LIFE

3.2.1 Acute Toxicity

Acute toxicity values for antimony ranged from a 96-hour EC_{50} of 0.76 mg/L for green algae (*Selenastrum capricornutum*) (EPA 1978) to a 48-hour LC_{50} of >530.0 mg/L for water fleas (*Daphnia magna*) (LeBlanc 1980; EPA 1978) (Table 3-1).

3.2.2 Chronic Toxicity

Kimball (1978) reported a chronic value (CV) of 1.6 for fathead minnows *(Pimephales promelas)*. Fathead minnows showed no reaction to concentrations up to 0.006 mg/L Sb(III), the highest concentration tested (LeBlanc and Dean 1984).

3.2.3 Toxicity to Aquatic Plants

Suter and Tsao (1996) reported a lowest chronic value(LCV) of 0.610 mg/L for aquatic plants.

3.2.4 Bioaccumulation

Little information is available for the bioaccumulation of antimony. In a 28-day exposure, bluegill (*Lepomis macrochirus*) did not accumulate antimony above concentrations in control fish, and the bioconcentration factor was <1 (EPA 1980).

3.2.5 Aquatic Mode of Action

Little information is available for the mode of action of antimony. However, it is believed to inhibit phosphorylation.

3.2.6 Water Quality Criteria

National Ambient Water Quality Criteria (NAWQC) are not available for antimony, however, final acute value (FAV) and final chronic value (FCV) were calculated at 0.18 and 0.03 mg/L (EPA 1980).

Table 3-1. Toxicity of antimony to aquatic organisms

Compound[1]	Conc.[2] (mg/L)	Species	Endpoint	Reference
Sb_2O_3	>530.0	Water flea (*Daphnia magna*)	48h LC_{50}	LeBlanc 1980; EPA 1978
Sb_2O_3	>440.0	Bluegill (*Lepomis macrochirus*)	96h LC_{50}	AQUIRE 1996
$C_4H_4KO_7Sb$	37.0	Rainbow trout (*Oncorhynchus*)	96h LC_{50}	AQUIRE 1996
$SbCl_3$	21.9	Fathead minnow (*Pimephales promelas*)	96h LC_{50}	Kimball 1978
$SbCl_3$	20.0	Nematode *(Caenorhabditis elegans)*	96h LC_{50}	AQUIRE 1996
$SbCl_3$	18.8	Water flea (*Daphnia magna*)	48h EC_{50}	Kimball 1978
SbK tartrate	9.0	Water flea (*Daphnia magna*)	48h EC_{50}	EPA 1980
Sb_2O_3	1.6	Fathead minnow *(Pimephales promelas)*	CV	Kimball 1978
Sb_2O_3	0.76	Green algae (*Selenastrum capricornutum*)	96h EC_{50}	AQUIRE 1996
Sb(III)	0.18	-	FAV	EPA 1988
Sb(III)	0.03	-	FCV	EPA 1988

[1]Compounds listed as "Sb" were not specified.
[2]Concentrations are as Sb, not the compound.

3.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Data for the toxicity of antimony in freshwater sediment is not available. Long and Morgan (1991) reported data for the toxicity of antimony in sediments from two marine bays. The data from Commencement Bay, Washington, indicated that toxicity to the amphipod *Rhepoxynius abronius* and the larvae of the oyster *Crassostrea gigas* increased with increasing sediment antimony concentrations. Sediments that were moderately toxic to both species had average antimony concentrations of 2.0 ± 5 mg/kg (note: authors did not indicate whether the variance estimate was a standard error or standard deviation). Sediments that were highly toxic to *R. abronius* had a mean antimony concentration of 91.5 $\pm$ 184 mg/kg. Sediments highly toxic to *C. gigas* had mean a concentration of 27.5 ± 101.5 mg/kg. However, data collected from San Francisco Bay showed no relation between toxicity to *R. abronius* and antimony concentrations. That is, sediments that were least toxic or not toxic had higher antimony concentrations than sediments that were most toxic or significantly toxic. The available data suggest that the toxicity of antimony in sediments is relatively uncertain and that sediment characteristics other than antimony concentrations have a considerable influence upon the observed responses.

3.4 TOXICITY TO PLANTS

3.4.1 Toxicity to Plants in Soil

Primary reference data for the toxicity of antimony to plants grown in soil are unavailable; however, the secondary reference data on the phytotoxicity of plants in soil noted undefined, qualitative phytotoxic effects on plants grown in surface soil with 5 mg/kg Sb (Kloke 1979). Antimony is considered a nonessential metal and is easily taken up by plants if available in the soil in soluble forms (Kabata-Pendias and Pendias 1984).

3.4.2 Toxicity to Plants in Solution

No information was found on the toxicity of antimony to plants in solution.

3.4.3 Phytotoxic Mode of Action

No information was found on the phytotoxic mode of action of antimony.

3.5 TOXICITY TO WILDLIFE

3.5.1 Toxicity to Mammals

Antimony is only slowly absorbed from the gastrointestinal tract. Based on animal data, gastrointestinal absorption of antimony was estimated to be 2 to 7% (Felicetti et al. 1974; Gerber et al. 1982). The specific chemical form will determine the absorption efficiency of ingested antimony. The primary target organ for acute oral exposure to antimony appears to be the gastrointestinal tract (irritation, diarrhea, vomiting) and targets for long-term exposure are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR 1990). Inhalation exposure to antimony affects the respiratory tract (pneumoconiosis, restrictive airway disorders), with secondary targets being the cardiovascular system (altered blood pressure and electrocardiograms) and kidneys (histological changes) (Renes 1953; Breiger et al. 1954). Only limited evidence exists for reproductive disorders due to antimony exposure (Belyaeva 1967).

Toxic effects ranging from gastrointestinal disorders to death have been documented for animals following acute oral exposure to antimonials. Bradley and Frederick (1941) reported that a single dose (300 mg Sb/kg) of the organic antimonial, potassium antimony tartrate, induced myocardial infarction and death in rats. However, several studies using inorganic antimonials (metallic antimony, antimony oxide, or antimony trioxide) reported that doses as high as 27,410 mg Sb/kg were not fatal to rats (ATSDR 1990).

Subchronic (24-week) exposure of rats to metallic antimony at doses of 500 to 1,000 mg/kg/day decreased plasma protein levels, hemoglobin levels, and hematocrit (Sungawa 1981; Hiraoka 1986). Sungawa (1981) also reported mild hepatotoxicity in rats receiving 418 mg Sb/kg/day (as antimony trioxide) or 500 mg Sb/kg/day (as metallic antimony). A decrease in red blood cell count was reported by Sungawa (1981) for rats given 418 mg Sb/kg/day (as antimony trioxide) for 24 weeks, and an increase reported by Smyth and Thompson (1945) in rats treated with the same compound at a dose of 894 mg Sb/kg/day for 30 days. Fleming (1982) reported that dogs receiving antimony trioxide (6644 mg Sb/kg/day) for 32 days exhibited severe weight loss, vomiting, and muscle weakness and dyskinesia of the hind limbs. A lower dose (84 mg Sb/kg/day) produced severe diarrhea. Using data from Fleming

(1982), ATSDR (1990) reported a no-observed-adverse-effect-level (NOAEL) of 501 mg Sb/kg for rats receiving the compound for 20 days. Exposure of CD-1 mice to potassium antimony tartrate in the drinking water (5 mg/L for 540 days) significantly reduced the lifespan of both males and females (Kanisawa and Schroeder 1969). Schroeder et al. (1968) also provided data showing that lifetime exposure of mice to 5 mg/L potassium antimony tartrate in the drinking water resulted in a decreased lifespan. Sample et al. (1996) estimated a chronic lowest-observed-adverse-effect level (LOAEL) of 1.25 mg/kg/d for reduced longevity based on the 5 mg/L level in drinking water. A chronic NOAEL of 0.125 mg/kg/d was estimated by multiplying the LOAEL by an uncertainty factor of 0.1.

3.5.2 Toxicity to Birds

Information on the toxicity of antimony to birds was not found.

3.6 REFERENCES

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4. ARSENIC

4.1 BACKGROUND

Arsenic (As) is a naturally occuring metaloid found in air and all living organisms. It is present in the earth's crust at approximately 2 mg/kg and is sparingly soluble in water and body fluids. It occurs as two forms in ambient media, As(III), usually the most toxic, and As(V) (EPA 1985) with its magnitude of bioavailability and toxicity dependent upon the oxidation state and temperature (McGeachy and Dixon 1992). The valence state is dependent on environmental conditions, including Eh, pH, organic content, suspended solids, and sediment. The relative toxicities of the various forms of arsenic vary from species to species. Arsenic may be released into aquatic ecosystems by anthropogenic sources including the manufacture and use of arsenical defoliants and pesticides, electric generating stations, manufacturing companies, mineral or strip mines, steel production, fossil fuel combustion, and smelting operations (Sorensen 1991; McGeachy and Dixon 1989; Ferguson and Gavis 1972, as cited in McGeachy and Dixon 1989; NRCC 1978), and natural leaching of the soils. Arsenic levels in a river ecosystem were found to be dependent upon the availability of arsenic, rainwater dillution, extent of complexation with dissolved organic matter, and possibly the metabolic activity of aquatic plants (Koranda et al. 1981). As soil clay concentration increases, arsenic adsorption onto the soil increases as a function of soil pH, texture, iron, aluminum, organic matter, and time (Woolson 1977). Arsenic is known as one of the most toxic elements to fish with acute exposures resulting in immediate death (Sorensen 1991).

4.2 TOXICITY IN WATER TO AQUATIC LIFE

4.2.1 Acute Toxicity

Acute toxicity of As(III) to six invertebrate species ranged from 0.81 mg/L for a water flea (*Daphnia magna*) (EPA 1985) to 97.0 mg/L for a midge (*Tanytarsus dissimilis*) (Holcombe et al. 1983). Acute values for fish ranged from 11.0 mg/L for the chum salmon (NAS 1977) to 82.3 mg/L for the fathead minnow (AQUIRE, 1996). Acute toxicity of As(V) ranged from 0.69 mg/L for the alga *(Selenastrum capricornutum)* (EPA 1985) to 49.6 mg/L for the water flea *Daphnia pulex* (Passino and Novak 1984). The relative toxicities of each arsenic compound appears to vary by species: the rainbow trout was slightly more sensitive to As(III) than to As(V), whereas the fathead minnow was nearly twice as sensitive to As(III) (Table 4-1).

Gill and muscle tissues of the fathead minnow exposed to 25.0 mg/L for 8 hours responded by producing stress proteins (Dyer et al. 1993). During a 48-hour LC_{50} test on *D. magna*, Burton et al. (1987) discovered that the presence of sediment significantly increased suvival in the presence of arsenite. Weir and Hine (1970, as cited in Oladimeji et al. 1984) found that arsenic caused behavioral impairments in goldfish *(Carassius auratus)*; at 0.1 mg/L As the fish showed 48% impairment in avoidance reaction.

Comparing concentration and temperature, McGeachy and Dixon (1992) held two groups of rainbow trout at separate temperatures, 5 and 15 °C at two concentration levels within each temperature group. The authors found no interaction of temperature and concentration, but higher temperatures resulted in more rapid loss of equilibrium due to more rapid uptake.

Table 4-1. Arsenic toxicity to aquatic organisms

Chem.	Conc.[1] (mg/L)	Species	Effect	Reference
Arsenic	3.80	Water flea *(Daphnia magna)*	48h LC_{50}	AQUIRE 1996
	1.70	Water flea *(Simocephalus vetulus)*	48h LC_{50}	AQUIRE 1996
As(III)	97.00	Midge *(Tanytarsus dissimilis)*	48h LC_{50}	Holcombe et al. 1983
	82.30	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	AQUIRE 1996
	45.00	Spottail shiner *(Notropis hudsonius)*	25h LC_{50}	NAS 1977
	38.00	Stone fly *(Pteronarcys californica)*	96h LC_{50}	Johnson & Finley 1980
	30.90	Barb *(Barbus javanicus)*	24h LC_{50}	AQUIRE 1996
	30.90	Featherback *(Notopterus notopterus)*	96h LC_{50}	AQUIRE 1996
	30.00–35.00	Bluegill *(Lepomis macrochirus)*	96h LC_{50}	NAS 1977; Johnson & Finley 1980
	25.90	Channel catfish *(Ictalurus punctatus)*	96h LC_{50}	NAS 1977
	24.50	Snail *(Aplexa hypnorum)*	96h LC_{50}	Holcombe et al. 1983
	23.00–26.60	Rainbow trout *(Oncorhynchus mykiss)*	96h LC_{50} [2]	Spehar et al. 1980
	24.20	Barb *(Barbus javanicus)*	96h LC_{50}	AQUIRE 1996
	22.00	Catfish *(Mystus vittatus)*	96h LC_{50}	AQUIRE 1996
	15.00	Brook trout *(Salvelinus fontinalis)*	96h LC_{50}	EPA 1985
	14.70	Snake-head catfish *(Channa punctatus)*	48h LC_{50}	AQUIRE 1996
	14.40	Flagfish *(Jordanella floridae)*	96h LC_{50}	Lima et al. 1984
	14.10	Fathead minnow *(Pimphales promelas)*	96h LC_{50}	Lima et al. 1984
	14.00	Two-spot barb *(Barbus sophore)*	48h LC_{50}	AQUIRE 1996
	12.16	Giant gourami *(Colisa fasciata)*	24h LC_{50}	AQUIRE 1996
	11.00	Chum salmon *(Oncorhynchus keta)*	48h LC_{50}	NAS 1977

Table 4-1 (continued)

Chem.	Conc.[1] (mg/L)	Species	Effect	Reference
	9.28	Snail *(Aplexa hypnorum)*	24h LC_{50}	Holcombe et al. 1983
	4.50	Marbled salamander *(Ambystoma opacum)*	8d EC_{50}	EPA 1985
	4.30	Water flea *(Daphnia magna)*	96h EC_{50}	Lima et al. 1984
	4.00	Bluegill *(Lepomis macrochirus)*	42% PR[3]	NAS 1977
	3.03	Fathead minnow *(Pimephales promelas)*	CV	Lima et al. 1984
	3.00	Water flea *(Daphnia pulex)*	48h EC_{50}	Johnson & Finley 1980
	2.96	Flagfish *(Jordanella floridae)*	CV	Lima et al. 1984
	2.32	Alga (*Cladophora* sp.)	2wk LC_{100}	EPA 1985
	2.32	Alga (*Spirogyra* sp.)	2wk LC_{100}	EPA 1985
	2.32	Alga (*Zygnema* sp.)	2wk LC_{100}	EPA 1985
	2.32	Submerged plant (*Potamogeton* sp.)	1mo. LC_{95}	EPA 1985
	1.30	Water flea *(Daphnia pulex)*	96h LC_{50}	EPA 1985
	1.20	Zooplankton	PR[3]	NRCC 1978
	0.91	Water flea *(Daphnia magna)*	CV	Lima et al. 1984
	0.87	Scud *(Gammarus pseudolimnaeus)*	96h EC_{50}	Lima et al. 1984
	0.81	Water flea *(Simocephalus serrulatus)*	96h LC_{50}	EPA 1985
	0.54	Rainbow trout *(Oncorhynchus mykiss)*	28d LC_{50}	EPA 1980
	0.49	Goldfish *(Carassius auratus)*	7d EC_{50}	EPA 1985
	0.37	Frog *(Rana hexadactyla)*	24h LC_{50}	AQUIRE 1996
	0.25	Frog *(Rana hexadactyla)*	96h LC_{50}	AQUIRE 1996
As(V)	49.60	Water flea *(Daphnia pulex)*	48h EC_{50}	Passino & Novak 1984
	42.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	AQUIRE 1996

Table 4-1 (continued)

Chem.	Conc.[1] (mg/L)	Species	Effect	Reference
	40.50	Striped bass *(Morone saxatilis)*	96h LC_{50}	AQUIRE 1996
	30.76	Alga *(Selenastrum capricornutum)*	14d EC_{50}	EPA 1985
	28.00	Rainbow trout *(Oncorhynchus mykiss)*	96h LC_{50}	AQUIRE 1996
	25.60	Fathead minnow *(Pimphales promelas)*	96h LC_{50}	EPA 1985
	24.6-41.6	Goldfish *(Carassius auratus)*	7d LC_{50}	NRCC 1978
	7.40	Water flea *(Daphnia magna)*	96h LC_{50}	Biesinger & Christensen 1972
	2.03	Eurasian milfoil *(Myriophyllum spicatum)*	32d EC_{50}	EPA 1985
	0.89	Fathead minnow *(Pimphales promelas)*	CV	EPA 1985
	0.85	Water flea *(Bosmina longirostris)*	96h EC_{50}	Passino & Novak 1984
	0.69	Alga *(Selenastrum capricornutum)*	4d EC_{50}	EPA 1985
	0.26	Alga *(Ankistrodesmus falcatus)*	14d EC_{50}	EPA 1985
	0.07		SAV	Suter & Tsao 1996
	0.05	Alga *(Scenedesmus obliquus)*	14d EC_{50}	EPA 1985
	0.003		SCV	Suter & Tsao 1996

[1]Concentrations are as As, not the compound.
[2]Adults.
[3]Population reduction.

4.2.2 Chronic Toxicity

The three chronic toxicity values for As(III) range from 0.914 mg/L to 3.026 mg/L for *D. magna* and fathead minnow, respectively (EPA 1985). The only chronic test that could be used to calculate a chronic value for As(V) used the fathead minnow and resulted in a value of 0.8916 mg/L (Table 4-1). Rainbow trout have exhibited abilities to acclimate themselves to the presence of arsenite in the water. The values for 144h LC_{50} increased by 47% from 13.2 to 19.7 mg/L after 21 days of preexposure to 0.22 of the incipient lethal level (Dixon and Sprague 1981). After a 6-week exposure, 10 and 20 mg/L As did not significantly affect rainbow trout growth while the highest concentration tested, 30 mg/L, did. Mean corpuscular hemoglobin concentration was greatly reduced at all

concentrations tested (Oladimeji et al. 1984). Bluegills showed a decrease in body and fat weight after 21 days of exposure to 1 mg/L As(III) (Speyer 1975, as cited in Dixon and Sprague 1981; Speyer and Leduc 1975, as cited in Spehar et al. 1980).

4.2.3 Toxicity to Aquatic Plants

Chronic toxicity to aquatic plants is dependent on what form of arsenic is present. Plant values for As(III) range from 2.32 mg/L to >59.20 mg/L, while values for As(V) range from 0.048 mg/L to 202.0 mg/L. Endpoints for As(V) tended toward effects concentrations while As(III) endpoints tended toward lethality concentrations (EPA 1985). Sodium arsenate showed no impact on growth curves of planktonic alga at all concentrations tested (0.05–2.0 mg/L As) (Maeda et al. 1983).

4.2.4 Bioaccumulation

Arsenic is not readily bioconcentrated by fish nor biomagnified up the food chain. Algae may accumulate higher arsenic residues than fish (Maeda et al. 1983; Woolson 1977; Spehar et al. 1980). In general, accumulation of arsenic depends upon arsenic concentration, rate of uptake, age of organs, geography, and proximity to anthropogenic sources (Hong et al. 1989). Target sites include (in order of significance) the liver, skin, and muscle. In fish, absorption appears to be irreversible as body levels remained high after arsenic concentration in the water fell (Sandhu 1977). Arsenic accumulation in *D. magna* is concentration dependent and highest with As(III). The biological concentration factors (BCFs) are 50 at 0.97 mg/L and 219 at 0.097 mg/L As(III). At a concentration of 1.0 mg/L As(V), the stonefly had a BCF of 33–35 and at 0.1 mg/L the BCF increased to 131. In another experiment using As(V) at 0.1 mg/L, the snails *Helisoma campanulata* and *Stagnicola emarginata* had BCFs of 99 and 92 respectively (Spehar et al. 1980).

4.2.5 Aquatic Mode of Action

Arsenic acts primarily by inhibiting enzymatic reactions. Growth impairment in the rainbow trout has been linked to the inhibition of enzymes that reduces the oxygen-carrying capacity of the blood and leads to the inefficient utilization of assimilated food (Oladimeji et al. 1984). Arsenic(III) is thought to react with thiol and sulfhydryl groups of amino acids, which are building blocks of numerous enzymes and coenzymes. Arsenic(III) has also been shown to interrupt oxidative metabolic pathways and may cause morphological damage to the liver mitochondria (Hong et al. 1989). The uncoupling of glycolytic phosphorylation at glyceraldehyde-3-phosphate dehydrogenase inhibits energy metabolism (McGeachy and Dixon 1989).

Hematologic abnormalities may occur as a result of injury to formed elements of the blood, damage to bone marrow stem cells, and/or interference with maturation and development of progenitors into circulatory blood cells (Hong et al. 1989).

Penrose (1975, as cited in Dixon and Sprague 1981) and Sorensen (1976, as cited in Dixon and Sprague 1981) suggest that inorganic arsenic is not converted to organic compounds in fish but is excreted directly into the bile. However, Oladimeji (1980, as cited in Oladimeji et al. 1984) suggested that arsenic is converted in fish and stored in tissue and that bile accounts for a minor fraction of total excretion.

The toxicity of arsenate to fish has been explained by two different senerios. Cairns et al. (1975, as cited in McGeachy and Dixon 1989) suggest that an increase in water temperature increases the rate of toxic metabolism and/or elimination of arsenic. On the other hand, Heath (1973, as cited in

McGeachy and Dixon 1989) hypothesize that higher temperatures result in increased toxicity due to increased uptake and distribution of the toxicant to target sites.

4.2.6 Water Quality Criteria

The acute and chronic National Ambient Water Quality Criteria (NAWQC) forAs(III) are 0.360 and 0.190 mg/L, respectively (EPA 1985). The secondary acute values and chronic values forAs(V) are 0.066 and 0.0031 mg/L respectively (Suter and Tsao 1996).

4.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Most of the available data for arsenic toxicity are from tests of marine and estuarine sediments (Table 4-2). The most sensitive tested organism was the sandworm *Nereis virevs*: exposure to 1.88 mg/kg was associated with 100% mortality. Moderate toxicity was generally observed for exposure concentrations between approximately 10 and 60 mg/kg. The highest reported concentration was 2,257 mg/kg and was associated with 79% mortality of the amphipod *Rhepoxynius abronius*. Although Long and Morgan (1991) presented results from five studies of freshwater sediments, they did not use those results for the estimation of sediment quality benchmarks because they were of generally poor quality. That is, the differences in concentrations that indicated no adverse effects and the concentrations that did indicate effects were too small, there was no concordance between concentrations and effects, or the reported detection limits were too high. The reported effects were mortality or taxa richness and the average detected concentrations from these studies ranged from 2.2 to 7.4 mg/kg. These results suggest that arsenic toxicity is highly uncertain at concentrations less than approximately 10 mg/kg.

4.4 TOXICITY TO PLANTS

4.4.1 Toxicity to Plants in Soil

The tolerance of spruce seedlings to arsenic in soil was tested in field plots by Rosehart and Lee (1973). Three-year-old seedlings grown 335 days in soil to which 1000 mg/kg As was added as As(III) (lowest concentration tested) experienced a 50% reduction in height.

Soil type affected the toxicity of As(III) to cotton and soybeans grown from seed for 6 weeks (Deuel and Swoboda 1972). In a sandy loam soil, shoot weights of both crops were reduced (cotton 22%; soybeans 45%) by 11 mg/kg As (the lowest concentration tested). Soybean growth in a clay soil was reduced 28% by 22.4 mg/kg As (lowest concentration tested). Cotton growth in this soil was reduced 29% by 89.6 mg/kg As. The chemical form of As(V) has been shown to influence the effect on corn grown from seed for 4 weeks in a loamy sand (pH 7.1). Plant weight reductions were almost 100% for NaH_2AsO_4, over 75% for $Al(H_2AsO_4)_3$, and about 65% for $Ca(H_2AsO_4)_2$ with the addition of 100 mg/kg As (Woolson et al. 1971).

Will and Suter (1995a) reported soil no-observed-effect concentration (NOEC) and soil lowest-observed-effect concentration (LOEC) values for the effects of arsenic derived from experiments conducted in soil. The soil NOEC values range from 10 to 62.7 mg/kg, and the soil LOEC values range from 2 mg/kg (barley) to 1000 mg/kg (spruce) for the phytotoxicity of arsenic (Table 4-3).

Table 4-2. Arsenic toxicity to benthic invertebrates in marine and estuarine sediments (MacDonald et al. 1994)

Conc. (mg/kg)	Endpoint	Species
1.88	14d LC_{100}	Sandworm *(Nereis virens)*
4.33	1h EC_{98} (fertilization)	Sea urchin *(Arbacia punctulata)*
4.65	48h LC_{25}	Sea urchin *(Arbacia punctulata)*
10.10	LC_{33}	Mysid shrimp *(Mysidopsis bahia)*
10.20	10d LC_{16}	Amphipod *(Ampelisca abdita)*
12.00	10d LC_{55}	Amphipod *(Hyallela azteca)*
12.80	96h LC>50	Shrimp *(Paleamonetes pugio)*
20.00	10d LC_{20}	Amphipod *(Rhepoxynius abronius)*
22.10	48h EC_{59} (abnormality)	Bivalve
43.00	20d LC_{37}	Polychaete *(Neanthes arenaceodentata)*
50.70	48h EC_{92} (abnormality)	Bivalve
58.70	48h EC_{23} (abnormality)	Oyster
690.00	48h EC_{45} (abnormality)	Oyster
2257.00	10d LC_{79}	Amphipod *(Rhepoxynius abronius)*

4.4.2 Toxicity to Plants in Solution

Mhatre and Chaphekar (1982) found no effect of As(III) (As_2O_3), up to 1 mg/kg As, on germination of seeds of sorghum, alfalfa, mung bean, cluster bean, and radish. After 5 days, reductions in root length occurred between 0.001 mg/kg As (29% reduction in cluster bean) and 1 mg/kg As (55 and 87% reductions in alfalfa and mung bean). The concentrations of As(V), from Na_2HAsO_4, required for a 50% reduction in seed germination and root length of mustard after 3-day exposure in solution (pH 7.3), was reported by Fargasova (1994) to be 30 mg/L. The EC_{50} for root length was 5.5 mg/kg As.

Will and Suter (1995a) reported NOEC and LOEC values for the effects of arsenic in solution. The NOECs range from 0.001 to 0.1 mg/kg, and the LOECs range from 0.001 mg/kg (lowest concentration tested for cluster bean) to 30 mg/kg (LC_{50} for mustard) (Table 4-4).

Table 4-3. Phytotoxicity data for the toxicity of arsenic derived from experiments conducted in soil (Will and Suter 1995a)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$NaAsO_2$	sand	barley	-	2	grain yield	Jiang & Singh 1994
As_2O_3	sandy loam	cotton	-	11.2	shoot weight	Deuel & Swoboda 1972
As_2O_3	sandy loam	soybean	-	11.2	shoot weight	Deuel & Swoboda 1972
As_2O_3	black clay	soybean	-	22.4	shoot weight	Deuel & Swoboda 1972
$NaAsO_2$	loam	barley	10	50	grain yield	Jiang & Singh 1994
$NaAsO_2$	sand	ryegrass	10	50	grain yield	Jiang & Singh 1994
$NaHAsO_4$	sand	barley	10	50	grain yield	Jiang & Singh 1994
As_2O_3	black clay	cotton	67.2	89.6	shoot weight	Deuel & Swoboda 1972
Na_2HAsO_4	sandy loam	corn	10	100	fresh weight	Woolson et al. 1971
$Al(H_2AsO_4)_3$	loamy sand	corn	10	100	fresh weight	Woolson et al. 1971
$Ca(H_2AsO_4)_2$	loamy sand	corn	10	100	fresh weight	Woolson et al. 1971
$NaAsO_2$	loam	ryegrass	50	250	grain yield	Jiang & Singh 1994
Na_2HAsO_4	loam	ryegrass	50	250	grain yield	Jiang & Singh 1994
Na_2HAsO_4	sand	ryegrass	50	250	grain yield	Jiang & Singh 1994
Na_2HAsO_4	loam	barley	50	250	grain yield	Jiang & Singh 1994
As_2O_3		spruce	-	1000	height	Rosehart & Lee 1973

Table 4-4. Phytotoxicity data for the toxicity of arsenic derived from experiments conducted in solution (Will and Suter 1995a)

Chemical form	Plant species	NOEC (mg/kg)	LOEC (mg/kg)	Growth parameter	Reference
As_2O_3	cluster bean	-	0.001 LCT	root length	Mhatre & Chaphekar 1982
As_2O_3	radish	.001	0.01	root length	Mhatre & Chaphekar 1982
As_2O_3	alfalfa	0.1	1	root & shoot lengths	Mhatre & Chaphekar 1982
As_2O_3	mung bean	0.1	1	root & shoot lengths	Mhatre & Chaphekar 1982
Na_2HAsO_4	mustard	-	5.5 EC_{50}	root length	Fargasova 1994
Na_2HAsO_4	mustard	-	30 LC_{50}	seed germination	Fargasova 1994

4.4.3 Phytotoxic Mode of Action

Arsenic is not essential for plant growth. It is taken up actively by roots, with arsenate being more easily absorbed than arsenite. Arsenic and phosphate ions are likely taken up by the same carrier (Asher and Reay 1979). The phytotoxicity is strongly affected by the valance state in which arsenic occurs in soils. Arsenite(III) is more toxic than arsenate(V), and both are considerably more toxic than organic forms (Peterson et al. 1981). Symptoms include wilting of new-cycle leaves, retardation of root and top growth, violet coloration, root discoloration, cell plasmolysis, leaf necrosis, and death (Aller et al. 1990). Arsenic is chemically similar to phosphorus. It is translocated in the plant in a similar manner and is able to replace phosphorus in many cell reactions. Arsenic(III) probably reacts with sulphydryl enzymes leading to membrane degradation and cell death. Arsenic(V) is known to uncouple phosphorylation and to affect enzyme systems (Peterson et al. 1981).

4.5 TOXICITY TO WILDLIFE

4.5.1 Toxicity to Mammals

Tissues of animals generally contain an average of <0.5 mg/kg (Venugopal and Luckey 1978). Arsenic is a carcinogen and teratogen. Effects include reduced growth, hearing/sight loss, liver/kidney damage, and death (Eisler 1988). Inorganic arsenic is usually more toxic than organic arsenic compounds. Arsenic may be a required micronutrient; growth, survival, and reproduction of goats is poor if the diet contains <0.05 mg/kg As (NAS 1977). Wildlife mortality and malformations have been

observed for chronic doses of 1–10 mg As/kg body weight and dietary concentrations of 5–50 mg/kg (Eisler 1988). Acute LD_{50}s for mammals of 35–100 mg calcium arsenate/kg body weight and 10–50 mg lead arsenate/kg body weight have been reported (NRCC 1978).

Because metabolism of arsenic in rats and mice is unlike that in other animals, results of toxicity studies using rats generally should not be extrapolated to other species (Eisler 1988).

After a 14-day exposure to arsine gas, mice had a significant decrease in red blood cells, hemoglobin, and hematocrit numbers. The spleen-to-body ratio increased from 38 to 236% at 0.5 to 5.0 mg/L As (Hong et al. 1989). The solubility in organic solvents and relative nonpolarity of arsine gas allow it to tranverse biologic membranes of stem cells and/or react with sulfhydryl groups of proteins necessary for osmotic balance within erythrocytes (Graham et al. 1946 and Levinsky et al. 1970, as cited in Hong et al. 1989).

Schroeder and Mitchner (1971) exposed mice to a single dose level of 5 mg/L sodium arsenite in drinking water for three generations. While mice exposed to arsenic survived well, litter size decreased in subsequent generations. Based on the results of Schroeder and Mitchner (1971), Sample et al. (1996) estimated the chronic (no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL) for reproduction in mice to be 0.126 mg/kg/d and 1.26 mg/kg/d. (Note: the NOAEL was estimated by multiplying the measured LOAEL by an uncertainty factor of 0.1). A dose of 0.38 mg arsenic/kg over a lifetime was sufficient to cause a slight decrease in the median lifespan of laboratory mice (Schroeder and Balassa 1967), but it had no effect on growth. As little as 3 mg arsenic trioxide/kg body weight or 1 mg sodium arsenite/kg body weight can be lethal (NAS 1977).

Sample et al. (1997) report median soil-small mammal uptake factors for predator, herbivore, omnivore, and general small mammals (i.e., all trophic groups combined) to be 0.0013, 0.002, 0.0062, and 0.0038, respectively. These factors are unitless quotients of the concentration in the whole animal/concentration in soil, all on a dry weight basis. Regression analyses of the of ln-transformed whole animal concentrations on ln-transformed soil concentrations produced significant model fits for herbivores, omnivores, and general small mammals (there were insufficient data for independent analyses for predators; Sample et al. 1997). The general small mammal model most accurately predicted whole animal arsenic concentrations when applied to independent data (Sample et al. 1997).

4.5.2 Toxicity to Birds

Among birds, LD_{50}s for arsenic compounds range from 17.4 to 3300 mg/kg body weight (Eisler 1988). While no mortality was observed among mallard ducks fed a diet containing 100 mg/kg sodium arsenite for 128 days, 12% to 92% mortality was observed for ducks fed diets containing 250 to 1000 mg/kg arsenite (USFWS 1964). Based on the results reported in USFWS (1964), Sample et al. (1996) estimated the chronic NOAEL and LOAEL for mortality in mallard ducks to be 5.14 mg/kg/d and 12.84 mg/kg/d. Camardese et al. (1990) and Whitworth et al. (1991) fed mallards diets containing 30, 100, or 300 mg/kg sodium arsenate. While no effects were observed on behavior, growth was reduced for male ducks consuming 300 mg/kg arsenic and for female ducks at all exposure levels. Arsenic levels in the preen gland and kidney of Dunlin (*Calidris alpina*) paralleled each other, suggesting that excretion of arsenic is via the preen gland (Goede and de Bruin 1985). Swelling of the granular endoplasmic reticulum (GER) of the Coturnix quail suggests that ions accumulate in the interior of the GER with a subsequent influx of water due to a disruption of the normal osmotic balance across the GER membrane. ATP production is reduced through the binding of the citric acid enzymes and in turn upsets the cellular sodium pump that maintains normal osmotic balance (Bartik and Pisac 1981; Buchanan 1962; Clarke and Clarke 1967; Nystrom 1984).

4.6 TOXICITY TO HETEROTROPHIC PROCESSES AND SOIL AND LITTER INVERTEBRATES

Juma and Tabatabai (1977) evaluated the effects of two forms of arsenic on soil acid and alkaline phosphatase activities of microbes. Arsenic(III) at 1875 ppm was associated with less than a 20% reduction in acid phosphatase activity in all three soils. At a concentration of 1875 mg/kg As (lowest concentration tested), acid phosphatase activity was reduced in a loam soil (pH 7.8; percent organic matter 7.4). Arsenic(V) was more toxic than As(III) to both enzyme complexes. Alkaline and acid phosphatase activities were reduced by as little as 187.5 mg/kg As(V) (lowest concentration tested) in soils of pH 5.8 to 7.4 and percent organic matter 5.2 to 11. Frankenberger and Tabatabai (1981) investigated the effect of As(III) on microbe amidase activity in three soils in shaker flask assays. After 2½ hours, amidase activity was reduced in all three soils. Activity was almost totally inhibited in the soils at the lowest tested concentration, 1875 mg/kg. The effective concentration of 187 mg/kg (Frankenberger and Tabatabai 1981) is the lowest of the eight reported (Table 4-5).

Fischer and Koszorus (1992) tested the effects of 68 mg/kg of arsenic (as potassium arsenate) on growth and reproduction of *Eisenia fetida*. The number of cocoons produced per worm showed the highest sensitivity to arsenic with a 56% reduction at the test concentration.

Table 4-5. Toxicity of arsenic to soil heterotrophic processes (Will and Suter 1995b)

Chemical form	Organisms	Growth medium	pH	%OC	EXP (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
Na_2HAsO_4	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	–	187.3 LCT	33	Juma & Tabatabai 1977
$NaAsO_2$	native soil microflora	surface soil	6	2.6	0.1	Amidase activity	–	187.3 LCT	32	Frankenberger & Tabatabai 1981
$NaAsO_2$	native soil microflora	loam	7	4.7	0.1	Amidase activity	–	1873 LCT	97	Frankenberger & Tabatabai 1981
$NaAsO_2$	native soil microflora	clay loam	8	3.2	0.1	Amidase activity	–	1873 LCT	98	Frankenberger & Tabatabai 1981
Na_2HAsO_4	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	–	187.3 LCT	32	Juma & Tabatabai 1977
Na_2HAsO_4	native soil microflora	clay loam	8	3.7	0.1	Alkaline & acid phosphatase activity	–	1875 LCT	75,39	Juma & Tabatabai 1977
Na_2HAsO_4	native soil microflora	loam	7	5.5	0.1	Acid phosphatase activity	–	1875 LCT	62	Juma & Tabatabai 1977
$NaAsO_2$	native soil microflora	clay loam	8	3.7	0.1	Alkaline phosphatase activity	–	1875 LCT	35	Juma & Tabatabai 1977
$KH2AsO_4$	Eisenia fetida	soil & manure	–	–	56	cocoons/worm	–	68 LCT	56	Fischer & Koszorus 1992

Note: Chemical concentrations are expressed as grams of element per kilogram of growth medium.
CEC = cation exchange capacity of growth medium (milliequivalents/100 g); % DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls; EXP (D) = exposure in days; % OC = % organic carbon; LCT = lowest concentration tested.

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5. BARIUM

5.1 BACKGROUND

Barium (Ba) is found in the more common mineral forms barite ($BaSO_4$) and witherite ($BaCO_3$) (Oehme 1979). Barium fluorosilicate and carbonate are forms often used as pesticides. Approximately 400 mg/kg of barium is found in the earth's crust. Some plants accumulate barium from the soil. Barium has industrial applications in areas such as paper manufacturing, fabric printing and dyeing, synthetic rubber production, and drilling fluids. Barium in groundwater has been found to range from 0 to over 20 mg/L (Gilkeson et al. 1983).

5.2 TOXICITY IN WATER TO AQUATIC LIFE

5.2.1 Acute Toxicity

There is relatively little information on the aquatic toxicity of barium (Table 5-1). Acute values range from a 96-h LC_{50} of 6950.0 mg/L for the mosquitofish *(Gambusia affinis)* (AQUIRE) to a 96-h EC_{50} of 25.0 mg/L for the duckweed *(Lemna minor)* (Wang 1988).

5.2.2 Chronic Toxicity

No information was found on the chronic toxicity of barium to aquatic life.

5.2.3 Toxicity to Aquatic Plants

The calculated 50% inhibition concentration was 25 mg/L for duckweed (pH 6.32–8.32) in various ambient waters. The magnitude of barium's toxicity towards aquatic plants was shown to be highly dependent upon site-specific water quality characteristics and doseage (Wang 1988). Barium inhibited calcification of the freshwater green alga *Goeotaenium* at 50.0 mg/L (Prasad 1984).

5.2.4 Bioaccumulation

Barium is bioconcentrated from water by algae and vascular plants at levels dependent upon the particular species. However, Ba is not bioaccumulated through food chains and barium levels in higher species rarely exceeds 10mg/kg (Moore 1991).

5.2.5 Aquatic Mode of Action

No information was found on the mode of action of barium.

5.2.6 Water Quality Criteria

The state of Illinois has a water quality standard for indigenous aquatic life of 5.0 mg/L (Wang 1988). The Secondary Acute Value (SAV) and Secondary Chronic Value (SCV) are 0.1136 and 0.004 (Suter and Tsao 1996).

Table 5-1. Toxicity of barium to aquatic organisms

Compound	Conc.[1] (mg/L)	Species	Effect	Reference
$BaCO_3$	6950.0	Mosquitofish *(Gambusia affinis)*	96h LC_{50}	AQUIRE 1996
$BaCl_2$	3980.0	Scud *(Gammarus pulex)*	24h LC_{50}	AQUIRE 1996
$BaCl_2$	1080.0	Mosquitofish *(Gambusia affinis)*	96h LC_{50}	AQUIRE 1996
$BcCl_2$	410.0	Waterflea *(Daphnia magna)*	48h LC_{50}	LeBlanc 1980
$BaCl_2$	150.0	Brown trout (*Salmo trutta*)	96h LC_{50}	AQUIRE 1996
$BaCl_2$	122.0	Scud *(Echinogammarus berilloni)*	96h LC_{50}	AQUIRE 1996
$BaCl_2$	78.0	Crayfish *(Orconectes limosus)*	96h LC_{50}	AQUIRE 1996
$BaCl_2$	46.0	Crayfish *(Austropotamobius pallipes)*	96h LC_{50}	AQUIRE 1996
$BaSO_4$	33.7	Tubificid worm *(Tubifex tubifex)*	48h EC_{50}	Khangarot 1991
$BaSO_4$	32.0	Water flea *(Daphnia magna)*	48h EC_{50}	Khangarot & Ray 1989
$BaCl_2$	25.0	Duckweed *(Lemna minor)*	96h EC_{50}	Wang 1988
$BaCl_2$	14.5	Water flea *(Daphnia magna)*	48h EC_{50}	Biesinger & Christensen 1972
	0.114		SAV	Suter & Tsao 1996
	0.004		SCV	Suter & Tsao 1996

[1]Concentrations are as Ba, not the compound.

5.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on barium toxicity to sediment invertebrates.

5.4 TOXICITY TO PLANTS

5.4.1 Toxicity to Plants in Soil

Chaudhry et al. (1977) investigated the effects of Ba added as $Ba(NO_3)_2$ on shoot weight of barley and bush beans grown from seed for 14 days in a loam soil. Shoot growth of barley was reduced 38% after 14 days by the addition of 500 mg Ba/kg, the lowest concentration tested. Shoot growth of

bush beans was reduced 30% after 14 days by the addition of 2000 mg Ba/kg, but was not reduced at the next lowest level, 1000 mg/kg.

5.4.2 Toxicity to Plants in Solution

No information was found on the toxicity of barium to plants in solution.

5.4.3 Phytotoxic Mode of Action

Barium is commonly present in plants but is not an essential component of plant tissues. It is taken up easily from acid soils (Kabata-Pendias and Pendias 1984). Mechanisms of toxicity may include competition with Ca for root uptake (Wallace and Romney 1971).

5.5 TOXICITY TO WILDLIFE

5.5.1 Toxicity to Mammals

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system of mammals, eventually leading to paralysis. The LD_{50} for rats is 630 mg/kg for barium carbonate, 118 mg/kg for barium chloride, and 921 mg/kg for barium acetate (Lewis and Sweet 1984).

Schroeder and Mitchener (1975a, 1975b) exposed rats and mice to 5 mg barium/L in drinking water for their lifetime. There was a slight but significant reduction in longevity of treated male mice when measured as the mean age at death of the last surviving 10% of animals. The overall average life span of the group, however, was about the same as that of the control group. In another study, Perry et al. (1983) exposed rats to 0, 1, 10, or 100 mg barium/kg for up to 16 months. A significant increase in average blood pressure was observed in the highest dose group; a slight but statistically significant increase was seen in the 10 and 100 mg/kg dose groups. Because the significance of hypertension in wild populations is unclear, Sample et al. (1996) consider the maximum dose (100 mg/kg food or 5.1 mg/kg/d) to represent a chronic no-observed-adverse-effect level (NOAEL) for rats. Borzelleca et al. (1988) dosed rats with 100, 145, 209, and 300 mg/kg $BaCl_2$ in water through oral gavage for 10 days. Exposure of rats to 300 mg/kg/d $BaCl_2$ for 10 days resulted in 30% mortality to female rats. No adverse effects were observed at any other dose levels. Sample et al. (1996) therefore considered the 300 mg $BaCl_2$/kg/d dose (198 mg Ba/kg/d) to be a subchronic lowest-observed-adverse-effect level (LOAEL) for mortality in rats. A chronic LOAEL (19.8 mg Ba/kg/d) was estimated by multiplying the subchronic LOAEL by a subchronic to chronic uncertainty factor of 0.1. Information on developmental and reproductive toxicity of barium to mammals is not available.

Sample et al. (1997) report median soil-small mammal uptake factors for herbivore, omnivore, and general small mammals (i.e., herbivores and omnivores combined) to be 0.048, 0.042, and 0.042, respectively. These factors are unitless quotients of the concentration in the whole animal/ concentration in soil, all on a dry weight basis.

5.5.2 Toxicity to Birds

The LD_{50} of barium to chickens is 623 mg/kg (Johnson et al. 1960). While chickens will tolerate 1000 mg barium/kg in their diet without adverse effects, 2000 mg/kg reduces growth, 8000 mg/kg produces 50% mortality in 4 weeks, and diets containing 16,000 or 32,000 mg barium /kg are 100% lethal (Johnson et al. 1960). Sample et al. (1996) estimated a chronic NOAEL and LOAEL for mortality

in chickens, based on Johnson et al. (1960). While Ba exposures up to 2000 ppm produced no mortality, chicks in the 4000 to 32000 ppm groups experienced 5% to 100% mortality. Because 2000 ppm was the highest nonlethal dose, this dose was considered to be a subchronic NOAEL. The 4000 ppm dose was considered to be a subchronic LOAEL. A chronic NOAEL (20.8 mg/kg/d) and LOAEL (41.7 mg/kg/d) were estimated by multiplying the subchronic NOAEL and LOAEL by a subchronic to chronic uncertainty factor of 0.1.

5.6 TOXICITY TO HETEROTROPHIC PROCESS

The influence of soil characteristics on effects of Ba on microbe arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979). A 22% reduction in activity was caused by 3433 mg/kg Ba (only concentration tested).

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6. BERYLLIUM

6.1 BACKGROUND

Beryllium (Be), an alkaline earth metal, is experimentally used as a missile propellant in high-performance aerospace craft, the nuclear industry, and other industrial purposes (Finch et al. 1990; Oehme 1979). The emphasis of beryllium toxicity has recently shifted from purely industrial exposures to concern for general environmental contamination as a result of new uses. Beryllium speciation is pH dependent, with Be concentration increasing as the pH declines (Jagoe et al. 1993).

6.2 TOXICITY IN WATER TO AQUATIC LIFE

6.2.1 Acute Toxicity

Aquatic toxicity of Be is summarized in Table 6-1. Morphological changes were observed (96h) in gills of European perch *(Perca fluviatilis)* at concentrations as low as 0.01 mg/L (pH 5.5) including swelling of epithelial cell and reductions in microridges on cell surfaces (Jagoe et al. 1993). Fatal abnormalities such as the shortening of the secondary lamellae were observed at 0.05 mg/L, leading to a decrease in the surface area available for gas exchange. Beryllium at pH 4.5 damaged epithelial cells causing a disruption in the ion and osmoregulatory systems (Jagoe et al. 1993). Beryllium is approximately 100 times more toxic to guppies in soft water than in hard water (Slonim 1973, as cited in Slonim and Slonim 1973). In hard circumneutral water (400 mg/L as $CaCO_3$), guppies *(Lebistes reticulatis)* exhibited a 96h LC_{50} of 20.0 mg/L.

6.2.2 Chronic Toxicity

No information was found on the chronic toxicity of beryllium to fishes, but a standard *Daphnia magna* chronic value is available (Table 6-1).

6.2.3 Toxicity to Aquatic Plants

Beryllium inhibited growth of the green alga *Chlorella vanniellii* growing under suboptimal conditions at 100 mg/L (Karlander and Krauss 1972).

6.2.4 Bioaccumulation

A 28-day study with bluegill gave a bioconcentration factor of 19 and a half life of 1 day (EPA 1980).

6.2.5 Aquatic Mode of Action

Due to their similar chemical characteristics, it is believed that the toxicity mechanism of beryllium is similar to that of aluminum. Physiological processes of the gills such as ion regulation and gas exchange are affected, indicated by changes in chloride cell surface morphology and shortened secondary lamellae (Jagoe et al. 1993).

Table 6-1. Acute toxicity of beryllium to aquatic organisms

Compound	Conc.[1] (mg/L)	Species	Effect	Reference
$Be(NO_3)_2$	20.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[2]	Tarzwell & Henderson 1960
$Be(NO_3)_2$	0.15	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[6]	Tarzwell & Henderson 1960
$BeCl_2$	>100.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	>100.00	Flatworm *(Dugesia tigrina)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	>100.00	Oligochaete *(Lumbriculus variegatus)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	>100.00	Ramshorn snail *(Heliosoma trivolvis)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	>100.00	Aquatic sowbug *(Asellus intermedius)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	15.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[2]	Tarzwell & Henderson 1960
$BeCl_2$	5.90	Scud *(Gammarus fasciatus)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	1.00	Water flea *(Daphnia magna)*	48h LC_{50}	LeBlanc 1980
$BeCl_2$	0.40	Water flea *(Daphnia magna)*	96h LC_{50}	Ewell et al. 1986
$BeCl_2$	0.15	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[6]	Tarzwell & Henderson 1960
$BeSO_4$	40.00	Nematode *(Caenorhabditis elegans)*	24h LC_{50}	Williams & Dusenberry 1990
$BeSO_4$	31.50	Salamander *(Ambystoma opacum)*	96h LC_{50}[2]	Slonim & Ray 1975
$BeSO_4$	25.97	Tubificid worm *(Tubiflex tubiflex)*	24h EC_{50}[3]	Khangarot 1991
$BeSO_4$	22.60	Salamander *(Ambystoma maculatum)*	96h LC_{50}[2]	Slonim & Ray 1975
$BeSO_4$	20.00	Guppy *(Lebistis reticulatis)*	96h LC_{50}[2]	Slonim & Slonim 1973
$BeSO_4$	17.70	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	Kimbal 1978
$BeSO_4$	12.00	Bluegill *(Lepomis macrochirus)*	96h LC_{50}[2]	Tarzwell & Henderson 1960
$BeSO_4$	11.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[2]	Tarzwell & Henderson 1960
$BeSO_4$	10.25	Tubificid worm *(Tubiflex tubiflex)*	96h EC_{50}[3]	Khangarot 1991

Table 6-1 (continued)

Compound	Conc.[1] (mg/L)	Species	Effect	Reference
$BeSO_4$	6.49	Salamander *(Ambystoma maculatum)*	96h LC_{50}[6]	Slonim & Ray 1975
$BeSO_4$	4.80	Goldfish *(Carassius auratus)*	96h LC_{50}	Cardwell et al. 1976
$BeSO_4$	4.64	Water flea *(Daphnia magna)*	24h EC_{50}[3]	Khangarot & Ray 1989
$BeSO_4$	3.25	Flagfish *(Jordanella floridae)*	96h LC_{50}	Cardwell et al. 1976
$BeSO_4$	3.15	Salamander *(Ambystoma opacum)*	96h LC_{50}[6]	Slonim & Ray 1975
$BeSO_4$	2.50	Water flea *(Daphnia magna)*	48h LC_{50}	Kimball 1978
$BeSO_4$	2.00	Guppy *(Lebistis reticulatis)*	24h LC_{50}[6]	Slonim & Slonim 1973
$BeSO_4$	1.30	Bluegill *(Lepomis macrochirus)*	96h LC_{50}[6]	Tarzwell & Henderson 1960
$BeSO_4$	0.20	Fathead minnow *(Pimephales promelas)*	96h LC_{50}[6]	Tarzwell & Henderson 1960
$BeSO_4$	0.16	Guppy *(Lebistis reticulatis)*	96h LC_{50}[6]	Slonim & Slonim 1973
$BeSO_4$	0.14	Nematode *(Caenorhabditis elegans)*	96h LC_{50}	Williams & Dusenberry 1990
$BeSO_4$	0.07	Perch *(Perca fluviatilis)*	96h CV[4]	Jagoe et al. 1993
$BeSO_4$	0.07	Roach *(Rutilus rutilus)*	96h CV[4]	Jagoe et al. 1993
$BeSO_4$	0.005	Water flea *(Daphnia magna)*	3d CV[5]	Kimball 1978

[1]All concentrations are given as Be, not the compound.
[2]Hard water (>400mg/L Ca).
[3]Immobilization.
[4]pH 5.5.
[5]Reproduction.
[6]Soft water (20–25 mg/L Ca).

6.2.6 Water Quality Criteria

National ambient water quality criteria are not available. Calculated Tier II values are SAV- 0.035 and SCV-0.00066 (Suter and Tsao 1996).

6.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Information on the toxicity of beryllium towards benthic invertebrates was not available.

6.4 TOXICITY TO PLANTS

6.4.1 Toxicity to Plants in Soil

No primary reference data for toxicity of Be to plants grown in soil was obtained. Kloke (1979) reported unspecified toxic effects on plants grown in a surface soil with the addition of 10 mg Be/kg.

6.4.2 Toxicity to Plants in Solution

Romney et al. (1962) reported a 33% reduction in the weight of bush beans when grown for 48 days in a pH 5.3 nutrient solution containing 0.5 mg/L Be (lowest concentration tested). Barley (20- day), pea (24-day), and lettuce (28-day) weights were reduced 50, 21, and 37%, respectively, by 2 mg/L Be (as $BeCl_2$; pH 5.3) (Romney and Childress 1965). After 54 days, alfalfa weight was reduced 25% by 4 mg Be/L.

The effects of Be, from $BeSO_4$, on germination and radicle length after 3-day growth in solution of radish, cabbage, turnip, lettuce, wheat, and millet were determined by Carlson et al. (1991). There was no effect on seed germination up to 40 mg/L Be. Concentrations at which effects occurred ranged from 0.5 mg/L (63% reduction in radicle length of lettuce and turnip) to 40 mg/L Be (35% reduction in radicle length of millet). Comparing the effects on lettuce with those from the work of Romney and Childress (1965), there is an indication that early negative effects may be overcome by the plant.

Will and Suter (1995) reported no-observed-effect concentration (NOEC) and lowest-observed-effect concentration (LOEC) values for the toxicity of beryllium in solution. The NOEC values range from 1 to 30 mg/L, and the LOEC values range from 0.5 to 40 mg/L (Table 6-2).

6.4.3 Phytotoxic Mode of Action

Soluble forms of Be are easily taken up by plants, probably in a manner similar to Ca and Mg, but Be is not readily translocated from roots to shoots (Peterson and Girling 1981). Beryllium has been reported to inhibit seed germination, enzyme activation, and uptake of Ca and Mg by roots. Common symptoms are brown, retarded roots and stunted foliage (Romney and Childress 1965).

6.5 TOXICITY TO WILDLIFE

6.5.1 Toxicity to Mammals

After chronic inhalation exposure, relatively insoluble particles of Be are cleared slowly from the lungs of beagle dogs. Soluble particles are more quickly removed from the lungs. As long as 6 months post-exposure, beryllium levels in the blood increased, suggesting that a significant amount of the inhaled dose remains in the blood. The substantial residence time is possibly due to the formation of insoluble colloidal hydroxide and phosphate complexes (Reeves and Vorwald 1967, as cited in Sendelbach 1989). After deposition into the blood stream by pulmonary solubilization, beryllium is either deposited in bone or liver or excreted through the urine (Finch et al. 1990).

Table 6-2. Phytotoxicity data for the toxicity of beryllium derived from experiments conducted in solution (Will and Suter 1995)

Chemical form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	References
$BeSO_4$	lettuce	-	0.5 LCT	radicle length	Carlson et al. 1991
$BeSO_4$	turnip	-	0.5 LCT	radicle length	Carlson et al. 1991
$BeCl_2$	barley	-	2 LCT	plant weight	Romney & Childress 1965
$BeCl_2$	pea	-	2 LCT	plant weight	Romney & Childress 1965
$BeCl_2$	lettuce	-	2 LCT	plant weight	Romney & Childress 1965
$BeSO_4$	cabbage	1	2.5	radicle length	Carlson et al. 1991
$BeCl_2$	alfalfa	2	4	plant weight	Romney & Childress 1965
$BeSO_4$	radish	2.5	5	radicle length	Carlson et al. 1991
$BeSO_4$	wheat	10	20	radicle length	Carlson et al. 1991
$BeSO_4$	millet	30	40	radicle length	Carlson et al. 1991

Inhalation studies done with rats suggest that lung damage continues for as long as a year post-exposure to a single 1-h inhalation of 4.05μg Be/L. Presence of lactate dehydrogenase (LDH), a cytosolic enzyme, is indicitive of lung damage and was recorded at significant levels through 1 year post-exposure. Alkaline phosphatase, an indicator of type II pneumocyte destruction and general cell damage, continued at high levels through the 6th month post-exposure (Sendelbach et al. 1989).

Schroeder and Mitchner (1975) exposed rats to drinking water containing 5 mg/L Be (as beryllium sulfate) for their entire lifetime. While exposure to 5 ppm Be in water did not reduce longevity, weight loss by males was observed in months 2–6. Sample et al. (1996) did not consider weight loss to be an adverse effect, the 5 ppm dose level (0.66 mg/kg/d) was considered to be a chronic no-observed-adverse-effect level (NOAEL).

6.5.2 Toxicity to Birds

No information was found on beryllium toxicity to birds.

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7. BISMUTH

7.1 BACKGROUND

Bismuth (Bi), produced as a by-product of tin, lead, and copper ores, is found in the earth's crust at <1 mg/kg (Oehme 1979). Bismuth is used in the manufacture of type alloys, silvering of mirrors, low-melting solders, and heat-sensitive devices such as automatic fire extinguishers. Bismuth is found at concentrations of <0.0005 $\mu m/m^3$ in U.S. urban air.

7.2 TOXICITY IN WATER TO AQUATIC LIFE

7.2.1 Acute Toxicity

A tubificid worm *(Tubifex tubifex)* has EC_{50} values for immobilization of 14.79 and 0.662 mg/L $BeSO_4$ at 24 and 96 h, respectively (Khangarot 1991).

7.2.2 Chronic Toxicity

No information was found on the chronic toxicity of bismuth.

7.2.3 Toxicity to Aquatic Plants

No information was found on the toxicity of bismuth to aquatic plants.

7.2.4 Bioaccumulation

No information was found on the bioaccumulation of bismuth.

7.2.5 Aquatic Mode of Action

No information was found on the aquatic mode of action of bismuth.

7.2.6 Water Quality Criteria

No water quality criteria were available for bismuth.

7.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on the toxicity of bismuth to benthic invertebrates.

7.4 TOXICITY TO PLANTS

7.4.1 Toxicity to Plants in Soil

There were no reference data describing toxicity of Bi to plants grown in soil.

7.4.2 Toxicity to Plants in Solution

No primary reference data showing toxicity of Bi to plants grown in solution were available. Unspecified toxic effects on plants grown in a solution have been observed with the addition of 27 mg Bi/L (Scharrer 1955).

7.4.3 Phytotoxic Mode of Action

Although Bi has been shown to reduce the weight of some plants in solution culture (Scharrer 1955), no information on specific mechanisms of toxicity was found.

7.5 TOXICITY TO WILDLIFE

7.5.1 Toxicity to Mammals

No information was found on the toxicity of bismuth to mammals.

7.5.2 Toxicity to Birds

No information was found on the toxicity of bismuth to birds.

7.6 REFERENCES

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8. BORON

8.1 BACKGROUND

Boron (B) is found in the earth's crust at an average concentration of 16 mg/kg (Oehme 1979). Borax ($Na_2B_4O_7$) is used in soldering and welding to remove oxide film, for softening water, in soaps, and in glass, pottery, and enamels. Boron has medicinal properties as sodium borate and borax is used as a common cleaner (Dixon et al. 1976). Agricultural runoff from the application of boric acid as an insecticide and nonselective herbicide acts as a non-point source that severely affects the ecology of wetlands (Sander et al. 1991; Smith and Anders 1989). Boron is found in natural water supplies at levels lower than 1.0 mg/L, in soils of the Western United States at 10–300 mg/kg and is essential to plants (Smith and Anders 1989). Boron can also be found in vegetables, fruits, cereals, and breads (Valdes-Dapena and Arey 1962, as cited in Lee et al. 1978).

Boron is released by weathering processes, exists in sediments as borosilicates, and is an inert compound with regard to the metabolism of living animals (Butterwick et al. 1989). Boron speciation is dependent on water quality parameters such as boron concentration and pH (Maier and Knight 1991). Boric acid, $B(OH)_3$, and $B(OH)^{4-}$ are the active forms of boron and are highly soluble and stable, however, it is not certain which form of the metal is most toxic. Unlike a number of other metals, boron toxicity is not affected by water hardness (Butterwick et al. 1989).

8.2 TOXICITY IN WATER TO AQUATIC LIFE

8.2.1 Acute Toxicity

Relatively little aquatic toxicity information is available for boron. The only standard data are LC_{50}s and chronic values (CVs) for *Daphnia magna* exposed to boric acid in relatively hard water. Lewis and Valentine (1981) reported a 48h LC_{50} of 226 mg/L for *Daphnia magna*. The lethal threshold concentration was <200 mg/L of boron. Gersich (1984) reported a LC_{50} of 133 mg/L. Neonate stages of *D. magna* had a slightly higher 48h LC_{50} of 141 mg/L. Fourth instar *Chironomus decorus* exhibited a 48h LC_{50} of 1376.0 mg B/L (Maier and Knight 1991). Hamilton and Buhl (1990) found that chinook salmon *(Oncorhynchus tshawytscha)* have a higher LC_{50}, 725 mg/L, than do coho salmon *(O. kisutch)*, 447 mg/L. The 24h LC_{50}s for both species were greater than 1000.0mg/L, the highest concentration tested.

8.2.2 Chronic Toxicity

Gersich (1984) reported a CV of 9.33 mg/L for *D. magna* while Lewis and Valentine (1981) reported a slightly lower CV of 8.83 mg/L for the same species. Chronic exposures to sodium tetraborate significantly inhibited midge larvae growth at 20.0 mg/L (Maier and Knight 1991).

8.2.3 Toxicity to Aquatic Plants

Root growth of *Myriophyllum spicatum* was inhibited by 50% after a 32-day test at 40.3mg/L boron as tetraborate salt (Butterwick et al. 1989).

8.2.4 Bioaccumulation

Boron can be bioaccumulated at higher concentrations but there is no biomagnification up the trophic levels (Ohlendorf et al. 1986, as cited in Whitworth et al. 1991). Coho salmon, *Oncorhynchus kisutch,* have a BCF of 22.4–2.7 (Thompson et al. 1976, as cited in Butterwick et al. 1989). Due to its polarity, boron does not bioaccumulate in fat tissue (Moseman 1994). Instead, the target areas include the brain, spinal cord, and liver (Whitworth et al. 1991). In the San Joaquin Valley of California, an area of high boron run-off from agricultural fields, boron was found at concentrations of 371, 501, and 1860 mg/kg in the wigeon grass, algae, and grass seeds, respectively (Whitworth et al. 1991). Boron levels in aquatic vegetation seed have been as high as 3500 mg/kg, a concentration sufficient to adversely affect birds that feed upon it (Schuler 1987, as cited in Smith and Anders 1989).

8.2.5 Aquatic Mode of Action

No information was found on the bioaccumulation of boron.

8.2.6 Water Quality Criteria

No water quality criteria data are available. Calculated Tier II values are SAV-0.0297; SCV-0.0016 mg/L (Suter and Tsao 1996).

8.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on boron toxicity to sediment invertebrates.

8.4 TOXICITY TO PLANTS

8.4.1 Toxicity to Plants in Soil

Boron is an essential nutrient for plants. Uptake of the element is proportional to the boron concentration in the soil (Moseman 1994), and boron becomes highly toxic at elevated levels (Butterwick et al. 1989). John et al. (1977) investigated the effects of boron added as H_3BO_3 on shoot weight of corn seedlings grown 7 weeks in muck or two silt loam soils. Addition of 50 mg B/kg to the muck soil (pH 4.5; 56% organic matter) resulted in a 56% reduction in plant growth (10 mg B/kg had no effect). Growth was reduced 37% by the addition of the lowest concentration tested (0.5 mg/kg) in one silt loam soil (pH 5.7; 6% organic matter), and 83% by the addition of 50 mg B/kg in the other (pH 5.7; 3 % organic matter) (10 mg/L had no effect). Symptoms of boron toxicity include yellowing of leaf tip, chlorosis, necrosis of chlorotic tissue, and leaf abscisis (Butterwick et al. 1989).

In a field study of the fate and toxicity of emissions from a geothermal cooling tower, effects of boron on trees were observed (Lang et al. 1986). The threshold for toxic effects (10% visible injury) on big leaf maple (*Acer macrophyllum*) and interior live oak (*Quercus wislizeni*) were 529 and 488 mg/kg B in foliage, respectively. The threshold for Digger pine (*Pinus sabiniana*) was 272 mg/kg B in foliage. Relationships between soil boron and foliage boron were presented.

8.4.2 Toxicity to Plants in Solution

Wallace et al. (1977) evaluated the effect of boron (as H_3BO_3) on leaf, stem, and root weights of bush bean seedlings in solution. After 16 days, root and leaf weights were reduced 35 and 45% by 5.4 mg B/L, while 1.1 mg/L had no effect. Bowen (1979) reported unspecified toxic effects on plants grown in a solution with the addition of 1.0 mg B/L. *Pinus radiata* has a lowest observed effect concentration (LOEC) of 50.0 mg/L when exposed to various boron compounds (Marzo Munoz Cobo and de Lanuza 1970, as cited in Butterwick et al. 1989).

8.4.3 Phytotoxic Mode of Action

Boron is a plant micronutrient involved in transport of sugars across membranes, synthesis of nucleic acids, and protein utilization. It is rapidly taken up, mainly as the neutral $B(OH)_3$ molecule, and equally distributed between roots and shoots (Wallace and Romney 1977). Toxicity symptoms include needle tip necrosis and discoloration in pines (Neary et al. 1975) and burning of leaf edges in other plants. Grasses and legumes appear to have greater than average tolerance to high boron concentrations (Gupta 1984), and pines appear to be particularly sensitive (Stone and Baird 1956).

8.5 TOXICITY TO WILDLIFE

8.5.1 Toxicity to Mammals

According to the US EPA, 5.0 mg B/L in drinking water is the maximum safe level of boron for livestock (Butterwick et al. 1989). Sheep develop enteritis with naturally occurring boron at concentrations of 130–300 mg B kg/L (Koval'skii 1965, as cited in Butterwick et al. 1989). Sisk et al. (1990) fed goats 2.0g/kg BW of a boron fertilizer which had proven toxic to cows and observed behavioral effects that included stargazing (staring), spontaneous charging, avoidance behavior from phantom attacks, tail wagging and prancing after 40–48 hours post-exposure. At 48–72 hours post-exposure, they had hung heads, tremors, ear flicking, and head jerking, and they showed signs of anorexia (Sisk et al. 1990).

Most mammalian boron toxicity studies have been conducted using rats and dogs. LD_{50}s for rats range from 2.66 to 6.08 B/kg body weight (as sodium borate) and 3.16 to 5.14 B/kg BW (as boric acid); dogs have a somewhat lower tolerance to boric acid, with LD_{50}s ranging from 1.78 to 2.0 B/kg BW (Pfeiffer et al. 1945, as cited in Sisk et al. 1990; Weir and Fisher 1972; Smyth et al. 1969, as cited in Sisk et al. 1990; Lewis and Sweet 1984, as cited in Sisk et al. 1990). Food uptake and body weight of rats were not affected by exposure to borax at 2000 mg/L after 60 days (Lee et al. 1978). Five months post-exposure to a diet containing 9000 mg/kg diet of boric acid for 28 days, bone of exposed rats retained boron at a level three-fold greater than that in control rats (Moseman 1994).

Boron has been known to decrease male fertility in rats and dogs (Lee et al. 1978). Chronic exposure of both rats and dogs leads to testicular atrophy, spermatogenic arrest, and germinal aplasia (Bouissou and Castagnol 1965, as cited in Lee et al. 1978). Lee et al. (1978) investigated germinal aplasia induced by boron exposure and found that accumulation in the testicles increased with dose concentration and dose length. At 30 to 60 days post-exposure, there was a significant drop in germinal elements in the 1000mg/L borax group. At 60 days post-exposure, there was a drop in liver (13.79 to 10.41g), testicular (1.81 to 0.63g) and epidermis (1.23 to 0.8g) weights in both the 1000 mg/L and 2000 mg/L groups (Lee et al. 1978). Testicular atrophy was seen 90 days post-exposure at 1170 mg/L in a similar study conducted by Weir and Fisher (1972).

Weir and Fisher (1972) fed rats diets containing 117, 350, and 1170 mg B/kg (as either boric acid or borax) for three generations. No adverse effects were observed among individuals on the 117- and 350-kg/kg diets; reproductive performance, as measured by fertility and lactation indices, exceeded those for controls (Weir and Fisher 1972). In contrast, rats consuming the 1170 mg/kg diet were sterile; atrophied testes were observed among males while females displayed decreased ovulation. Based on the results of Weir and Fisher (1972), Sample et al. (1996) estimated a chronic no observed adverse effect level (NOAEL) and lowest observed adverse effect level (LOAEL) for reproduction in rats to be 28 mg/kg/d and 93.6 mg/kg/d, respectively.

While not completely understood, a number of theories concerning the mechanisms of boron toxicity in mammals have been proposed. It is believed that involuntary hyperactive movements expressed during boron toxosis are due to boron interference in the extrapyramidal system. Boron may also interact with estrogen and testosterone by influencing mineral metabolism through endocrine mechanisms (Nielsen et al. 1987, as cited in Sisk et al. 1990). Blood glucose levels have been shown to increase following boron dosing, suggesting its involvement in the cessation of glucose metabolism (Sisk et al. 1990).

8.5.2 Toxicity to Birds

Sander et al. (1991) report a mean oral LD_{50} of boric acid to 1-day-old chickens of 2950 mg/kg. Once administered orally, boric acid distributes throughout body water and tends to accumulate in the brain, liver, kidney, and white muscle of the chicken (Sander et al. 1991). Among chickens consuming 2500 and 5000 mg boric acid/kg in feed for 3 weeks, tissue levels were significantly higher (50 to 124% greater) than the controls. The 48-hour symptoms of boron toxosis include diarrhea, ataxia, incoordination, hypertonia, and at times death. Dermal absorption was not evident (Sander et al. 1991).

High concentrations of boron have been found in aquatic food webs associated with agricultural drainwater (Hoffman et al. 1991). As a result, mallard ducks and other waterfowl may be at risk of boron toxicosis. Hoffman et al. (1991) observed that diets containing 1000 mg/kg boric acid caused a decrease in growth by 20% over 4 weeks; diets containing 1600 mg/kg caused 10% mortality and decreased growth by 30%. Consumption of a diet supplying adequate protein and 1000 mg boron/kg decreased hematocrit and hemoglobin by 9 and 10%, respectively, and decreased liver and spleen weights with respect to those of the controls (Hoffman et al. 1991). Whitworth et al. (1991) observed significant behavioral effects among mallard ducklings consuming diets containing 1600 mg/kg B (as boric acid) for 6 weeks. Exposed ducklings spent significantly more time resting and under the heat lamps than did controls. These behavioral effects may be due to decreased brain energy from lower brain ATP production and may reduce individual survival (Whitworth et al. 1991). Smith and Anders (1989) fed mallard ducks diets containing 8, 35, 288, and 1000 mg B/kg (as boric acid) for 3 weeks prior to, during, and 3 weeks post reproduction. Consumption of 1000 mg/kg diet reduced egg fertility by 48%, increased embryo mortality 7.5-fold and increased duckling mortality by 81% at 7 days. While all diets reduced weight gain by ducklings, no adverse reproductive effects were observed among the other dose levels. Based on the results of Smith and Anders (1989), Sample et al. (1996) estimated the NOAEL and LOAEL for reproductive effects in mallards to be 28.8 and 100 mg/kg/d, respectively.

Because the study considered exposure throughout reproduction, the 288-ppm dose was considered to be a chronic NOAEL and the 1000-ppm dose was considered a chronic LOAEL. It should be noted that histology may not prove boron toxicosis in birds, as toxic effects such as decreased hatching success and body weight are observed before any histologic pathologies are evident (Smith and Anders 1989).

8.6 TOXICITY TO HETEROTROPHIC PROCESSES

The influence of soil characteristics on effects of boron on microbe arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979). No clear relationship was evident between (1) the magnitude of reduction in activity and (2) soil pH and organic matter. Juma and Tabatabai (1977) evaluated the effects of boron on soil acid and alkaline phosphatase activities. Acid phosphatase activity was reduced in the two lower pH soils at a concentration of 270 mg/kg. Alkaline phosphatase activity was not affected by boron in the soils tested. The effective concentration of 27 mg/kg (Al-Khafaji and Tabatabai 1979) is the lowest of the six reported (Table 8-1).

Table 8-1. Toxicity of boron to soil heterotrophic processes (Will and Suter 1995)

Chemical form	Organisms	Growth Medium	pH	%OC	EXP (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$Na_2B_4O_7$	native soil microflora	loam	7	2.9	0.1	Arylsulfatase activity	–	27 LCT	31	Al-Khafaji & Tabatabai 1979
$Na_2B_4O_7$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	27	270	33	Juma & Tabatabai 1977
$Na_2B_4O_7$	native soil microflora	clay loam	8	3.2	0.1	Arylsulfatase activity	27	270	65	Al-Khafaji & Tabatabai 1979
$Na_2B_4O_7$	native soil microflora	loam	7	5.3	0.1	Arylsulfatase activity	–	270 LCT	60	Al-Khafaji & Tabatabai 1979
$Na_2B_4O_7$	native soil microflora	clay loam	6	2.7	0.1	Arylsulfatase activity	–	270 LCT	70	Al-Khafaji & Tabatabai 1979
$Na_2B_4O_7$	native soil microflora	loam	7	5.5	0.1	Acid phosphatase activity	–	270 LCT	22	Juma & Tabatabai 1977

Note: Chemical concentrations are expressed in grams of element per kilogram of growth medium.
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared with controls.
EXP (D) = exposure in days.
% OC = % organic carbon.

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9. BROMINE

9.1 BACKGROUND

Bromine (Br) as CH_3Br is used as a common soil fumigant to control a wide variety of insects, fungi, and weeds (Nazer et al. 1982).

9.2 TOXICITY IN WATER TO AQUATIC LIFE

9.2.1 Acute Toxicity

LeBlanc (1980) found LC_{50}s for *Daphnia magna* to be 1.5 and 1.0 mg Br/L and during 24- and 48-hour tests. The no-discernible-effect level in the same test was 0.46mg Br/L.

9.2.2 Chronic Toxicity

No information was found on the chronic toxicity of bromine.

9.2.3 Toxicity to Aquatic Plants

No information was found on the toxicity of bromine to aquatic plants.

9.2.4 Bioaccumulation

No information was found on the bioaccumulation of bromine.

9.2.5 Aquatic Mode of Action

No information was found on the aquatic mode of action of bromine.

9.2.6 Water Quality Criteria

No water quality criteria were available for bromine.

9.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on the toxicity of bromine to benthic invertebrates.

9.4 TOXICITY TO PLANTS

9.4.1 Toxicity to Plants in Soil

No primary reference data were available for toxicity of Bromineto plants grown in soil. Kloke (1979) reported unspecified toxic effects on plants grown in a surface soil with the addition of 10 mg Br/kg. Newton and Toth (1952) found no toxicity symptoms or reduction in weight of tomato plants at concentrations up to 20 mg Br/kg in soil. Similar results from Nazer et al. (1982) showed that the

highest concentration tested by fumigation, 17.3 mg Br/L, inflicted toxic effects upon a number of fresh fruits and crops.

9.4.2 Toxicity to Plants in Solution

No primary reference data were available for toxicity of bromine to plants grown in solution. Martin (1966) reported unspecified toxic effects on plants grown with the addition of 15 mg Br/L.

9.4.3 Mechanism of Phytotoxicity

Bromine can substitute for part of the Cl^- requirement of plants. Symptoms of excess bromine are similar to those of excess salt: leaf edge necrosis and poor seed germination (Martin et al. 1956).

9.5 TOXICITY TO WILDLIFE

9.5.1 Toxicity to Mammals

No information was found on the toxicity of bromine to mammals.

9.5.2 Toxicity to Birds

No information was found on the toxicity of bromine to birds.

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10. CADMIUM

10.1 BACKGROUND

Cadmium (Cd) occurs predominately in the form of free divalent cations in most well-oxygenated, low-organic-matter, fresh waters (EPA 1985). However, both particulate matter and dissolved organic matter can bind cadmium in biologically unavailable forms. There is no evidence that cadmium is a biologically essential or beneficial element (Eisler 1985). Cadmium toxicity is related to water hardness, with a reduction in toxicity associated with increased water hardness (EPA 1985). Therefore, the cadmium toxicity values presented in this chapter that are not from tests conducted in waters of moderate hardness are normalized to 100 mg/L using the slopes calculated by the EPA (1985).

10.2 TOXICITY IN WATER TO AQUATIC LIFE

10.2.1 Acute Toxicity

See Table 10-1.

10.2.2 Chronic Toxicity

An unusually large number of chronic values (16) is available for cadmium (Table 10-1).

10.2.3 Toxicity to Aquatic Plants

Aquatic plant toxicity data for cadmium are relatively abundant. The range of plant toxicity values from 23 studies is 0.002 to 20 mg/L, with a geometric mean of 0.26 EPA (1985). These values are higher than those for sensitive aquatic animals.

10.2.4 Bioaccumulation

Aquatic organisms are able to bioconcentrate cadmium , although there is evidence that only the lower trophic levels can biomagnify this element. Bioconcentration data for cadmium are relatively abundant; the following summary is based on EPA (1985). The range of bioconcentration factors for whole fish from five studies is 33 to 2,213 mg/L, with a mean of 991. The range of bioconcentration factors for whole invertebrates from 21 studies is 164 to 4,190 mg/L, with a mean of 1,328. The range of bioconcentration factors for aquatic plants from four studies is 580 to 960 mg/L, with a mean of 716.

10.2.5 Aquatic Mode of Action

Cadmium is a highly toxic metal with no known nutrient properties. It is toxic by a variety of mechanisms in numerous tissues (Sorensen 1991). The most conspicuous injury in most tests is irregular ventilation associated with damage to the gills. Histological injury also occurs in the kidneys, liver, reproductive organs, and blood cells. In chronic studies, cadmium weakens bones, causing spinal deformities in fish.

Table 10-1. Hardness-normalized toxicity of cadmium to aquatic organisms calculated from EPA (1985)

Concentration (mg/L)[1]		Effect
100 mg/L hardness	200 mg/L hardness	
24.6	72.8	Goldfish (*Carassius auratus*) LC_{50}
18	18	Stonefly (*Plecoptera* sp.) LC_{50}[2]
17.7	38.7	Mayfly (*Ephemerella grandis*) LC_{50}
17.3	37.8	Tubificid worm (*Rhyacodrilus montana*) LC_{50}
16.8	36.7	Mosquitofish (*Gambusia affinis*) LC_{50}
16.5	36	White perch (*Morone americana*) LC_{50}
15.1	33	Tubificid worm (*Stylodrilus heringlanus*) LC_{50}
12.7	23.2	Bluegill (*Lepomis macrochirus*) LC_{50}
12.5	27.3	Channel catfish (*Ictalurus punctatus*) LC_{50}
12.4	27	Tubificid worm (*Spirosperma nikolskyl*) LC_{50}
10.9	23.8	Threespine stickleback (*Gasterosteus aculeatus*) LC_{50}
10.4	22.8	Tubificid worm (*Varichaeta pacifica*) LC_{50}
9.62	21.02	Tubificid worm (*Spirosperma ferox*) LC_{50}
8.79	19.22	Tubificid worm (*Quistadrilus multisetosus*) LC_{50}
8.79	19.22	Tubificid worm (*Tubifex tubifex*) LC_{50}
8.3	18.15	Snail (*Amicola* sp.) LC_{50}
7.8	17.5	Guppy (*Poecilla reticulata*) LC_{50}
7.68	16.79	White sucker (*Catostomus commersoni*) LC_{50}
7.43	16.24	Caddisfly (*Trichoptera* sp.)LC_{50}
6.86	12.85	Green sunfish (*Lepomis cyanellus*) LC_{50}
6.59	14.41	Tubificid worm (*Branchiura sowerbyl*) LC_{50}
6.31	13.79	Flagfish (*Jordanella floridae*) LC_{50}
5.24	11.46	Northern squawfish (*Ptychochellus oregonensis*) LC_{50}
5.05	11.04	Mayfly (*Paraleptophlebia praepedita*) LC_{50}
4.67	10.21	Tubificid worm (*Limnodrilus hoffmelsterl*) LC_{50}
3.71	8.12	Worm (*Nais* sp.) LC_{50}
2.94	6.43	Pumpkinseed (*Lepomis gibbosus*) LC_{50}
2.62	5.73	Midge (*Chironomus* sp.) LC_{50}
1.61	3.52	American eel (*Anguilla rostrata*) LC_{50}
0.875	1.913	Isopod (*Asellus bicrenata*) LC_{50}

Table 10-1 (continued)

Concentration (mg/L)[1]		Effect
100 mg/L hardness	200 mg/L hardness	
0.705	1.542	Crayfish (*Orconectes limosus)* LC_{50}
0.484	1.06	Bryozoan (*Plumatella emarginata*) LC_{50}
0.471	1.03	Common carp (*Cyprinus carpio*) LC_{50}
0.448	0.979	Amphipod (*Hyalella azteca*) LC_{50}
0.343	0.749	Snail (*Physa gyrina*) LC_{50}
0.311	0.681	Bryozoan (*Pectinatella magnifica*) LC_{50}
0.227	0.497	Snail (*Aplexa hypnorum*) LC_{50}
0.216	0.472	Banded killifish (*Fundulus diaphanus*) LC_{50}
0.181	0.397	Water flea *(Ceriodaphnia reticulata)* EC_{50}
0.153	0.334	Scud (*Gammarus* sp.) LC_{50}
0.132	0.288	Scud (*Gammarus pseudolimnaeus*) LC_{50}
0.122	0.266	Water flea *(Daphnia pulex)* EC_{50}
0.1	0.219	Water flea (*Simocephalus serralatus*) EC_{50}
0.0935	0.2044	Isopod (*Lirceus alabamae*) LC_{50}
0.0891	0.1948	Water flea (*Molina macrocopa*) EC_{50}
0.072	0.1699	Fathead minnow (*Pimephales promelas*) LC_{50}
0.0667	0.1459	Bryozoan (*Lophopodella carteri*) LC_{50}
0.0281	0.0485	Bluegill (*Lepomis macrochirus*) CV
0.0266	0.0582	Water flea (*Daphnia magna)* EC_{50}
0.0262	0.0452	Fathead minnow (*Pimephales promelas*) CV
0.0141	0.0243	Atlantic salmon (*Salmo salar*) CV
0.0141	0.0243	Smallmouth bass (*Micropterus dolomieui*) CV
0.014	0.0242	Northern pike (*Esox lucius*) CV
0.014	0.0242	Lake trout (*Salvelinus namaycush*) CV
0.0135	0.0233	White sucker (*Catostomas commersoni*) CV
0.0129	0.0282	Coho salmon (*Oncorhynchus kisutch*) LC_{50}
0.0127	0.0219	Brown trout (*Salmo trutta*) CV
0.0093	0.02032	Chinook salmon (*Oncorhynchus tshawytscha*) LC_{50}
0.0092	0.01586	Flagfish (*Jordanella floridae*) CV
0.00834	0.01438	Snail (*Aplexa hypnorum*) CV
0.00784	0.01714	Rainbow trout (*Oncorhynchus mykiss*) LC_{50}
0.00739	0.01274	Coho salmon (*Oncorhynchus kisutch*) CV
0.00678	0.01168	Water flea (*Ceriodaphnia reticulata*) CV
0.00464	0.008	Chinook salmon (*Oncorhynchus kisutch*) CV

Table 10-1 (continued)

Concentration (mg/L)[1]		Effect
100 mg/L hardness	200 mg/L hardness	
0.00407	0.00701	Brook trout (*Salvelinus fontinalis*) CV
0.0039	0.0086	Acute NAWQC
0.00358	0.00782	Brown trout (*Salmo trutta*) LC_{50}
0.0011	0.002	Chronic NAWQC
0.00033	0.00057	Water flea (*Molna macrocopa*) CV
0.000233	0.000401	Water flea (*Daphnia magna*) CV

[1]Concentrations given as Cd, not the compound.
[2]The stonefly acute test did not specify the hardness of the water.

10.2.6 Water Quality Criteria

Aquatic toxicity is dependent on hardness, so the acute National Ambient Water Quality Criterion is defined as $e^{(1.128[\ln(\text{hardness})]-3.82)}$, and the chronic criterion is defined as $e^{(0.785[\ln(\text{hardness})]-3.49)}$ (EPA 1985). Criteria values for Cd are 0.0039 and 0.0011mg/L for the acute and chronic toxicity of cadmium at 100 mg/L hardness (Suter and Tsao 1996).

10.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Most of the available data for cadmium toxicity are from tests of marine and estuarine sediments (Table 10-2). The lowest toxic concentration was 0.070 mg/kg, which was associated with a 25% mortality in tests with the sea urchin *Arbacia punctulata*. Most of the concentrations reported to be toxic ranged from 0.267 mg/kg to 41.6 mg/kg. The highest reported concentrations were 1,070 mg/kg, 2,580 mg/kg, and 2,850 mg/kg, each of which was associated with a 50% mortality in 10-day tests with the amphipod *Ampelisca abdita*. Although Long and Morgan (1991) presented results from several studies of freshwater sediments, they did not use those results for the estimation of sediment quality benchmarks because they were of generally poor quality. That is, the differences in concentrations that indicated no adverse effects and the concentrations that did indicate effects were too small, there was no concordance between concentrations and effects, or the reported detection limits were too high. The reported effects were mortality, density, or taxa richness, and the average detected concentrations from these studies ranged from 0.05 to 2.8 mg/kg. These results suggest that cadmium toxicity is somewhat uncertain at concentrations less than approximately 3 mg/kg.

10.4 TOXICITY TO PLANTS

10.4.1 Toxicity to Plants in Soil

A number of researchers have measured reductions in growth of a variety of plants in different soils with 10 mg/kg or less of cadmium added to soil as soluble salts. Plants tested include sycamore and spruce trees, wild flowering plants, and crops and horticultural plants (corn, lettuce, radish, wheat). Soils range from light sands to heavy silty clay loams in the acid to neutral pH range. No clear trends in responses are evident that indicate that any particular type of plant is more sensitive to cadmium

Table 10-2. Cadmium toxicity to benthic invertebrates in marine and estuarine sediments (MacDonald et al. 1994)

Conc (mg/kg)	Endpoint	Species
0.070	48h LC_{25}	sea urchin *(Arbacia punctulata)*
0.267	1h EC_{80} (fertilization)	sea urchin *(Arbacia punctulata)*
0.300	10d LC_{100}	amphipod *(Corophium volutator)*
0.300	10d LC_{100}	amphipod *(Rhepoxynius abronius)*
0.300	10d LC_{62}	amphipod *(Rhepoxynius abronius)*
0.346	1h EC_{56} (fertilization)	sea urchin *(Arbacia punctulata)*
0.450	10d LC_{20}	amphipod *(Rhepoxynius abronius)*
0.490	10d LC_{16}	amphipod *(Ampelisca abdita)*
0.606	10d EC_{97} (failure to emerge)	amphipod *(Rhepoxynius abronius)*
0.709	low density	echinodermata
0.710	low density	rhynchocoela
0.720	low density	arthoropoda
0.726	low richness	benthic species
0.756	10d LC_{55}	amphipod *(Hyalella azteca)*
0.815	48h LC_{60}	oyster *(Crassostrea gigas)*
0.988	15m EC_{50}	phosphoreum *(Microtox)*
1.010	10d LC_{50}	amphipod *(Rhepoxynius abronius)*
1.040	48h EC_{33} (abnormality)	oyster *(Crassostrea gigas)*
1.200	Sediments devoid of feral clams	bivalve *(Macoma balthica)*
1.310	EC_{50}	phosphoreum *(Microtox)*
1.460	1h EC_{98} (fertilization)	sea urchin *(Arbacia punctulata)*
1.490	48h LC_{51}	bivalve *(Mulinia lateralis)*
1.570	EC_{50}	phosphoreum *(Microtox)*
1.600	14d LC_{100}	sand worm *(Nereis virens)*
1.650	20d EC<90 (failure to emerge)	amphipod *(Hyalella azteca)*
1.650	20d EC<90 (failure to emerge)	amphipod *(Lepidactylus dytiscus)*
1.650	10d LC_{29}	amphipod *(Lepidactylus dytiscus)*
1.650	10d EC_{90} (reburial)	amphipod *(Lepidactylus dytiscus)*
1.810	48h EC_{11} (development)	bivalve *(Mulinia lateralis)*
1.900	20d LC_{37}	polychaete *(Neanthes arenaceodentata)*
1.930	EC_{50}	phosphoreum *(Microtox)*
2.060	EC_{50}	phosphoreum *(Microtox)*
3.090	10d LC_{32}	amphipod *(Ampelisca abdita)*
4.000	10d EC_{70} (emergence)	amphipod *(Rhepoxynius abronius)*
4.300	low abundance	arthoropoda
4.700	low richness	benthic species

Table 10-2 (continued)

Conc (mg/kg)	Endpoint	Species
5.400	72h EC_{98} (avoidance)	amphipod *(Eohaustorius sencillus)*
5.600	72h LC_{40}	amphipod *(Rhepoxynius* spp. *)*
5.600	72h EC_{70} (avoidance)	amphipod *(Rhepoxynius* spp. *)*
6.200	low abundance	echinodermata
6.500	10d EC_{50} (failure to rebury)	amphipod *(Rhepoxynius abronius)*
6.500	72h EC_{84} (avoidance)	amphipod *(Rhepoxynius abronius)*
6.900	10d LC_{50}	amphipod *(Rhepoxynius abronius)*
7.610	low abundance	amphipod *(Rhepoxynius abronius)*
8.000	10d EC_{30} (failure to emerge)	amphipod *(Rhepoxynius abronius)*
8.200	10d LC_{50}	amphipod *(Rhepoxynius abronius)*
8.320	low abundance	benthic species
8.400	72h EC_{94} (avoidance)	amphipod *(Eohaustorius sencillus)*
8.400	72h LC_{98}	amphipod *(Eohaustorius sencillus)*
8.500	72h LC_{76}	amphipod *(Rhepoxynius abronius)*
8.680	low abundance	amphipod *(Rhepoxynius abronius)*
8.680	low abundance	phoxocephalid
8.7–11.5	10d LC_{50}	amphipod *(Rhepoxynius abronius)*
9.070	10d EC_{50} (failure to rebury)	amphipod *(Rhepoxynius abronius)*
9.720	10d EC_{50} (failure to emerge)	amphipod *(Rhepoxynius abronius)*
12.900	low density	echinoderm
15.300	48h EC_{45} (abnormality)	oyster
16.000	10d EC_{60} (failure to emerge)	amphipod *(Rhepoxynius abronius)*
18.800	low density	polychaeta
19.300	low density	amphipod
19.300	low density	phoxocephalid
19.900	low density	brittle star *(Ophiuroidea)*
19.900	low density	echinodermata
19.900	low density	sponge *(Foraminifera)*
20.800	4d EC_{50} (failure to rebury)	amphipod *(Rhepoxynius abronius)*
23.000	low richness	benthic species
25.900	4d LC_{50}	amphipod *(Rhepoxynius abronius)*
27.000	low richness	amphipod
27.000	low density	crustacea
27.000	low richness	macrobenthos
27.000	low density	phoxocephalid
27.200	72h LC_{50}	amphipod *(Rhepoxynius* spp.)
28.700	low density	mollusca
28.700	10d LC_{21}	amphipod *(Rhepoxynius abronius)*
41.600	10d LC_{79}	amphipod *(Rhepoxynius abronius)*
290.000	10d LC_{50}	amphipod *(Rhepoxynius hudsoni)*
1070–2850	10d LC_{50}	amphipod *(Ampelisca abdita)*

(reductions in growth range from 23 to 45%), or that growth conditions (pH and soil texture) consistently affect toxicity. Will and Suter (1995a) reported a large range of soil no-observed-effect concentration (NOEC) and (lowest-observed-effect concentration (LOEC) values for the toxicity of cadmium in soil. The NOEC values range from 1 to 56.3 mg/kg, and the LOEC values range from 1 to 300 mg/kg (Table 10-3).

10.4.2 Toxicity to Plants in Solution

Several crops and horticultural plants experienced reduced growth when grown in solution containing 0.1 mg Cd/L or less. These include carrot, soybean, corn, and tomato. Growth reductions range from 21 to 73%. No clear trends in responses are evident that indicate that any particular type of plant is more sensitive to cadmium. In different experimental settings, the same plants exhibit greater tolerance to cadmium with no effects apparent up to 28 mg Cd/L for corn in one experiment. Length of experiment and growth solution nutrient content differences may be responsible for differences in experimental results with the same plant.

Will and Suter (1995a) reported a large range of soil NOEC and LOEC values for the toxicity of cadmium in solution. The NOEC values range from 0.01 to 11.2 mg/L, and the LOEC values range from 0.01 to 692 mg/L (Table 10-4).

10.4.3 Phytotoxic Mode of Action

Cadmium is not essential for plant growth. If present in available form, it is readily taken up by the roots and translocated through the plant, and accumulated. Cadmium is chemically similar to Zn, an essential element. Competition between the two for organic ligands may explain some of the toxic effects of cadmium and the ameliorative effects of Zn on cadmium toxicity. Cadmium depresses uptake of Fe, Mn, and probably Ca, Mg, and N (Wallace et al. 1977; Iwai et al. 1975). Cadmium is toxic at low concentrations, compared with other heavy metals. Symptoms resemble Fe chlorosis, and include necrosis, wilting, reduced Zn levels, and reduction in growth. The mechanisms of toxicity include reduced photosynthetic rate, poor root system development, reduced conductivity of stems, and ion interactions in the plant. Agronomic crops are more sensitive than trees to cadmium toxicity (Adriano 1986).

10.5 TOXICITY TO WILDLIFE

10.5.1 Toxicity to Mammals

While there is little information to indicate that this relatively rare metal is biologically essential or beneficial, cadmium has been suggested as the cause of various deleterious effects to wildlife (Eisler 1985). Mammals and birds are comparatively resistant to the biocidal properties of cadmium, which include growth retardation, anemia, and testicular damage. Cadmium tends to bioaccumulate in the liver and kidney, eventually acting as a cumulative toxin. Cadmium residues of 2 mg/kg whole body fresh weight are evidence of cadmium contamination, and residues >5 mg/kg whole animal fresh weight may be life-threatening (Eisler 1985).

The lowest oral dose resulting in death for rats was 250 mg Cd/kg body weight (EPA 1980a). Weigel et al. (1987) fed rats 0.24, 0.85, or 2.25 mg Cd/kg in diet for 8 weeks. Concentrations $\geq$0.85 mg/kg resulted in reduced food intake, reduced body weights, and reduced enzyme activity, but no hematological effects were noted. Ma et al. (1991) determined that an average cadmium intake of 15

Table 10-3. Phytotoxicity data for the toxicity of cadmium derived from experiments conducted in soil (Will and Suter 1995a)

Chemical form	Soil Type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$CdCl_2$	silt loam	soybean	-	1	shoot weight	Miller et al. 1976
$CdCl_2$	sand + peat	soybeans	-	1.25	plant weight	Strickland et al. 1979
$CdCl_2$	soil + sand	spruce	1	2	root & shoot weights	Burton et al. 1984
$CdCl_2$	sand + peat	soybeans	1.25	2.5	plant weight	Strickland et al. 1979
$CdCl_2$	silty clay loam	radish	-	2.5	root weight	Haghiri 1973
$CdCl_2$	silty clay loam	lettuce	-	2.5	plant weight	Haghiri 1973
	loamy sand	corn	-	2.5	shoot weight	Miller et al. 1977
	loamy sand	spinach	2	4	plant weight	Sadana & Singh 1987b
$CdSO_4$	silt loam	spinach	-	4	leaf weight	Bingham et al. 1975
$CdSO_4$	silt loam	soybean	-	5	bean weight	Bingham et al. 1975
$CdCl_2$	silty clay loam	sycamore	-	5	leaf weight	Carlson & Bazzaz 1977
$CdCl_2$	silty clay loam	wheat	2.5	5	shoot weight	Haghiri 1973
$Cd(NO_3)_2$	sand:peat: soil	beech	-	-	annual ring width	Hagemeyer et al. 1993
$CdSO_4$	silt loam	curley cress	-	8	leaf weight	Bingham et al. 1975
$CdCl_2$	sand	black-eyed susan	-	10	germination; root&shoot weights	Miles & Parker 1979a
$CdCl_2$	sand	blazing star	-	10	root & shoot weights	Miles & Parker 1979a
$CdCl_2$	sand	thimbleweed	-	10	shoot weight	Miles & Parker 1979a

Table 10-3 (continued)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$CdCl_2$	sand	bergamot	-	10	root weight	Miles & Parker 1979a
$CdCl_2$	silt loam	soybean	1	10	shoot weight	Miller et al. 1976
$CdCl_2$	silt loam	soybean	1	10	shoot weight	Miller et al. 1976
$CdCl_2$	silt loam	soybean	1	10	shoot weight	Miller et al. 1976
$CdCl_2$	loamy sand	soybean	1	10	shoot weight	Miller et al. 1976
$CdCl_2$	Brown earth	radish	-	10	root & shoot weights	Khan & Frankland 1983
$CdCl_2$	Brown earth	oats	-	10	root weight	Khan & Frankland 1984
	loamy sand	wheat	-	10	grain yield	Sadana & Singh 1987a
$CdCl_2$	sand	bluestem	-	10	root & shoot weights	Miles & Parker 1979b
$CdCl_2$	silty clay loam	soybean	5	10	shoot weight	Haghiri 1973
$CdCl_2$	surface soil	soybean	5	10	seeds/plant	Aery & Sakar 1991
$CdCl_2$	sand + peat	soybeans	5	10	plant weight	Strickland et al. 1979
$CdSO_4$	silt loam	lettuce	-	13	head weight	Bingham et al. 1975
$C_4H_6CdO_4$	acid Cambisol	wheat	7	14.1	shoot weight	Reber 1989
$CdCl_2$	humic sand	tomato	3.2	16	fresh shoot weight	Adema & Henzen 1989
$CdSO_4$	silt loam	corn	-	18	grain yield	Bingham et al. 1975

Table 10-3 (continued)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$CdSO_4$	silt loam	carrot	-	20	tuber weight	Bingham et al. 1975
$CdCl_2$	sandy loam	red oak	10	20	plant weight	Dixon 1988
$CdCl_2$	sandy+clay loam	wheat	10	20	grain & straw yields	Singh et al. 1991
$CdCl_2$	sand + peat	soybeans	10	20	plant weight	Strickland et al. 1979
$CdCl_2$	loamy sand	corn	15	25	root length	Hassett et al. 1976
$CdCl_2$	sand	corn	-	28	plant weight	Traynor & Knezek 1973
$CdSO_4$	silt loam	turnip	-	28	tuber weight	Bingham et al. 1975
$CdCl_2$	sand	Ky bluegrass	10	30	root & shoot weights	Miles & Parker 1979a
$CdCl_2$	sand	bluestem	10	30	root & shoot weights	Miles & Parker 1979a
$CdCl_2$	sand	poison-ivy	10	30	root & shoot weights	Miles & Parker 1979a
CdO	alluvial	wheat	10	30	grain yield	Muramoto et al. 1990
$CdCl_2$	loam	lettuce	3.2	33	fresh shoot weight	Adema & Henzen 1989
$CdCl_2$	silt loam	spinach	-	40	root & leaf weights	John 1973
$CdCl_2$	silt loam	peas	-	40	seed, pod, vine weights	John 1973
$CdCl_2$	silt loam	oats	-	40	grain yield	John 1973
$CdCl_2$	silt loam	radish	-	40	tuber & top weights	John 1973
$CdSO_4$	silt loam	field bean	-	40	bean weight	Bingham et al. 1975
$CdCl_2$	Brown earth	wheat	-	50	root weight	Khan & Frankland 1984

Table 10-3 (continued)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$CdSO_4$	silt loam	wheat	-	50	grain yield	Bingham et al. 1975
$CdCl_2$+CdO	Brown earth	radish	-	50	root weight	Khan & Frankland 1984
$CdSO_4$	silt loam	radish	-	96	tuber weight	Bingham et al. 1975
$CdCl_2$	humic sand	oats	10	97	fresh shoot weight	Adema & Henzen 1989
$CdCl_2$	silt loam	rye	50	100	shoot weight	Carlson & Rolfe 1979
$CdCl_2$	surface soils	radish	-	100	top & root weights	John et al. 1972b
CdO	alluvial	rice	30	100	root & shoot weights	Muramoto et al. 1990
CdO	Brown earth	wheat	-	100	root weight	Khan & Frankland 1984
CdO	Brown earth	radish	-	100	root & shoot weights	Khan & Frankland 1983
$CdCl_2$	silt loam	soybean	10	100	shoot weight	Miller et al. 1976
$C_4H_6CdO_4$	Phaeosem	wheat	56.3	113	shoot weight	Reber 1989
$CdCl_2$	humic sand	lettuce	32	136	fresh shoot weight	Adema & Henzen 1989
$CdCl_2$	loam	oats	10	159	leaf weight	Adema & Henzen 1989
$CdSO_4$	silt loam	tomato	-	160	fruit weight	Bingham et al. 1975
$CdSO_4$	silt loam	zucchini	-	160	fruit weight	Bingham et al. 1975
$CdSO_4$	silt loam	cabbage	-	170	head weight	Bingham et al. 1975

Table 10-3 (continued)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$CdCl_2$	loam	tomato	32	171	fresh shoot weight	Adema & Henzen 1989
$CdCl_2$	silt loam	lettuce	40	200	root & leaf weights	John 1973
$CdCl_2$	silt loam	broccoli	40	200	leaf weight	John 1973
$CdCl_2$	silt loam	cauliflower	40	200	root & leaf weights	John 1973
$CdCl_2$	silt loam	carrot	40	200	root, tuber,top weight	John 1973
	loam	cotton	-	300	leaf & stem weights	Rehab & Wallace 1978
	loam	cotton	-	300	leaf & stem weights	Rehab & Wallace 1978

mg/kg/day by common shrews corresponded with critical renal metal loads of 120 mg/kg, a level indicative of adverse health effects. Rats on a diet with 5 mg Cd/kg suffered shortened lifespans (Schroeder et al. 1965). Cd at 50 mg/kg in the diet depleted iron from rat livers (Whanger 1973). Rats eating diets with 7.15 mg Cd/kg (as CdO) exhibited growth reductions, but those consuming a diet with 2.80 mg Cd/kg did not (Weigel et al. 1984).

Several studies have been performed to evaluate effects of cadmium on mammalian reproduction. Ferm and Layton (1981) observed that rats receiving >6 mg Cd/kg body weight daily during pregnancy gave birth to malformed fetuses. In a three-generation reproductive study, the population of mice exposed to 1 mg/kg $CdCl_2$ in their drinking water died out after the second generation (Schroeder and Mitchner 1971). Wills et al. (1981) fed rats diets containing 0.08, 0.1, and 0.125 mg/kg $CdCl_2$ for four generations. While no reduction in the growth or survivorship of offspring was observed at any dose level, fertility (no. litters/no. females) was reduced by 63% in rats receiving the 0.125-ppm-Cd diet; fertility was not reduced in the 0.1-ppm-Cd diet. Machemer and Lorke (1981) exposed rats to 1.82, 6.13, 18.39, and 61.32 mg Cd/kg/d (as $CdCl_2$) through oral gavage during days 6–15 of gestation. Rats exposed to 61.32 mg Cd/kg/d did not reproduce. At the next lower dose, 18.39 mg/kg/d, the number of stunted and malformed fetuses were significantly greater than controls and fetal weights were significantly decreased. No adverse effects on reproduction were observed at the 6.13 mg/kg/d dose level. In contrast, Baranski et al. (1983) observed no adverse effects among rats exposed to up to 4 mg Cd/kg/d, 5 days a week for 5 weeks. While fetal weights were significantly reduced (6% to 13%) among mice exposed to 10, 20, or 40 mg/L Cd (as $CdCl_2$) in drinking water during days 1 to 19 of pregnancy, no other adverse effects were reported (Webster 1978). Sutou et al. (1980) exposed rats to 0.1, 1, and 10 mg Cd/kg/d (as $CdCl_2$) via oral gavage for 6 weeks during mating and gestation. While no adverse effects were observed at the 1 mg/kg/d dose level, fetal implantations were reduced by 28%, fetal survivorship was reduced by 50%, and fetal resorptions increased by 400% amongst the 10 mg/kg/d group. Sample et al. (1996) considered the 1 and 10 mg/kg/d to be the chronic no-observed-adverse-

Table 10-4. Phytoxicity data for the toxicity of cadmium derived from experiments conducted in solution (Will and Suter 1995a)

Chemical form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	Reference
$CdCl_2$	carrot	-	0.01 LCT	shoot weight	Turner 1973
$Cd(NO_3)_2$	soybeans	-	0.05 LCT	root & leaf weights	Cunningham 1977
	soybean	-	0.05 LCT		Cunningham et al. 1975
$CdCl_2$	corn	0.01	0.1	plant weight & grain yield	Iwai et al. 1975
$CdSO_4$	bean	-	0.1 LCT	plant weight	Page et al. 1972
$CdSO_4$	turnip	-	0.1 LCT	plant weight	Page et al. 1972
$CdSO_4$	beet	-	0.1 LCT	plant weight	Page et al. 1972
$CdCl_2$	tomato	0.01	0.1	shoot weight	Turner 1973
$CdSO_4$	giant endive	-	0.1 LCT	root & weights	Garate et al. 1993
$CdCl_2$	wheat	-	0.1 LCT	shoot weight	Jalil et al. 1994
$CdSO_4$	Norway spruce	-	0.112 LCT	root length	Lamersdorf et al. 1991
$CdSO_4$	chrysanthemum	-	0.112 LCT	root & stem weights	Patel et al. 1976
$CdSO_4$	corn	-	0.112 LCT	fresh plant weight	Stiborova et al. 1986
$CdSO_4$	corn	0.25	0.5	plant weight	Page et al. 1972
$CdSO_4$	Norway spruce	-	0.56 LCT	root elongation	Godbold & Huttermann 1985
$CdCl_2$	lettuce	-	0.84 EC_{50}	fresh shoot weight	Adema and Henzen 1989
$CdCl_2$	corn	0.1	1	plant weight	Iwai et al. 1975
$CdCl_2$	swiss chard	0.1	1	shoot weight	Turner 1973
$CdSO_4$	tomato	-	1 LCT	plant weight	Page et al. 1972
$CdSO_4$	pepper	-	1 LCT	plant weight	Page et al. 1972
$CdSO_4$	barley	-	1 LCT	plant weight	Page et al. 1972
$CdSO_4$	lettuce	-	1 LCT	plant weight	Page et al. 1972
$CdCl_2$	beetroot	0.1	1	shoot weight	Turner 1973
$CdSO_4$	sesame	-	1.1 LCT	root growth	Inouhe et al. 1994

Table 10-4 (continued)

Chemical form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	Reference
$CdSO_4$	pea	-	1.1 LCT	root growth	Inouhe et al. 1994
$CdSO_4$	radish	-	1.1 LCT	root growth	Inouhe et al. 1994
$CdSO_4$	cucumber	-	1.1 LCT	root growth	Inouhe et al. 1994
$CdSO_4$	tomato	-	1.1 LCT	root growth	Inouhe et al. 1994
$CdSO_4$	Azuki bean	-	1.1 LCT	root growth	Inouhe et al. 1994
$CdSO_4$	cotton	-	1.12 LCT	plant weight	Rehab and Wallace 1978
$CdSO_4$	ryegrass	-	1.25 LCT	longest root & shoot	Wong and Bradshaw 1982
$CdCl_2$	rice	-	1.4 EC_{50}	radicle weight	Wang 1994
$CdCl_2$	corn	0.2	2	plant weight	Iwai et al. 1975
$CdCl_2$	corn	0.2	2	plant weight	Iwai et al. 1975
$CdCl_2$	corn	0.2	2	plant weight	Iwai et al. 1975
$CdSO_4$	cabbage	1	2.5	plant weight	Page et al. 1972
$CdCl_2$	tomato	1.1	3 EC_{50}	fresh shoot weights	Adema and Henzen 1989
$CdSO_4$	lettuce	1.1	3.4	root growth	Inouhe et al. 1994
$CdSO_4$	barley	1.1	3.4	root growth	Inouhe et al. 1994
$CdCl_2$	broad bean	4	6	root length	Misra et al. 1994
$CdCl_2$	oat	-	6 EC_{50}	fresh shoot weight	Adema and Henzen 1989
$CdSO_4$	oats	3.4	6.8	rootgrowth	Inouhe et al. 1994
$CdSO_4$	bean	0.11	11	root & leaf weights	Wallace 1979
$Cd(NO_3)_2$	corn	11.2	28.1	root & shoot lengths	Rascio et al. 1993
$CdCl_2$	maize	-	45 LCT	seed germination, radicle length, & plant weight	El-Enany 1995
$CdCl_2$	mustard	-	48 EC_{50}	root length	Fargasova 1994
$CdCl_2$	mustard	-	692 LC_{50}	seed germination	Fargasova 1994

effect level (NOAEL) and the lowest-observed-effect level (LOAEL), respectively, for reproductive effects in rats.

10.5.2 Toxicity to Birds

No mortality was observed among adult mallard ducks fed diets containing 0, 1.6, 16.2, and 210 mg Cd/kg for 90 days, however egg production was significantly reduced in the group consuming 210 mg Cd/kg (White and Finley 1978). In addition, the testes of males in the 210 mg/kg Cd group atrophied and the spermatogenic process was disrupted (White et al. 1978). Based on the results from White and Finley (1978), Sample et al. (1996) estimated the NOAEL and LOAEL for reproduction in mallards to be 1.45 and 20 mg/kg/d, respectively. Among mallard ducklings, 20 mg/kg Cd in the diet for 12 weeks produces mild to severe kidney lesions, and reduces packed cell volume and hemoglobin concentrations in the blood (Cain et al. 1983). Avoidance behavior of black ducklings is impaired by consumption of diets containing 40 mg/kg Cd for 4 months (Heinz et al. 1983).

10.6 TOXICITY TO HETEROTROPHIC PROCESSES AND SOIL AND LITTER INVERTEBRATES

The influence of soil characteristics on effects of cadmium on microbe arylsulfatase activity was evaluated by Al-Khafaji and Tabatabai (1979). A reduction in activity was measured in four soils (23 to 55%) at 2810 mg Cd/kg, with the greatest reduction in the soil with the lowest clay content. Juma and Tabatabai (1977) evaluated the effects of cadmium on soil acid and alkaline phosphatase activities. Acid phosphatase activity was reduced in three soils (44 to 51%) at 2810 mg/kg. Alkaline phosphatase activity was reduced 27% in the loam soil at a concentration of 281 mg/kg and 78% at 2810 mg/kg in a clay loam soil in which this was the only concentration tested. Haanstra and Doelman (1991) investigated short- and long-term effects of metals on arylsulfatase activity, urease activity (Doelman and Haanstra 1986), and total phosphatase activity (Doelman and Haanstra 1989) by native soil microflora in five soils. For all three enzyme systems, the highest EC_{50}s were found in the soil with the highest clay content. The lowest EC_{50}s were 1888 mg/kg for arylsulfatase, and 840 and 340 mg/kg in the sand for phosphatase and urease activities. The highest EC_{50}s were 230 mg/kg in the soil with the highest pH for phosphatase, 30 mg/kg Cd in the sandy loam soil for urease, and 121 mg/kg in the sand (lowest pH, organic matter, and clay) for arylsulfatase (Table 10-5).

The effects of several elements on dehydrogenase activity of the native soil microflora in a composite soil sample from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). A concentration of 30 mg/kg Cd reduced dehydrogenase activity by 47%. Lighthart and Bond (1976) investigated the effects of cadmium (as $CdCl_2$) added to a small soil and litter microcosm. A 43% reduction in respiration (O_2 uptake) was observed in microcosms inoculated with 6.1 mg/kg Cd. Threshold levels of cadmium (as cadmium acetate) for soil respiration of native microflora in three soils were determined by Reber (1989). There was no clear relationship between these soil characteristics and the magnitude of reduction in soil respiration at the concentrations tested. The highest LOEC concentration (56.3 mg/kg) for cadmium was associated with the soil containing the lowest percentage of organic matter and the lowest pH. Liang and Tabatabai (1977) investigated the effects of various metals on N mineralization by native soil microflora in four soils with varying pH and organic matter content. Only one concentration of each metal was tested. Cadmium reduced N mineralization in two soils at 562 mg/kg, but no relationship between soil characteristics and effects of cadmium could be discerned.

Table 10-5. Toxicity of cadmium to heterotrophic processes and soil invertibrates (Will and Suter 1995b)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$C_4H_6CdO_4$	native soil microflora	sandy hortisol	7	1.5	84	Respiration	7	14	23	Reber 1989
$CdCl_2$	native soil microflora	sandy soil	7	1	42	Urease activity	–	340 ED	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy loam	6	3	548	Urease activity	–	30 ED	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy loam	6	3	548	Phosphatase activity	–	9869 ED_{50}	50	Doelman & Haanstra 1989
$CdNO_4$	native soil microflora	surface soil		1.3	1	Dehydrogenase activity	–	30 LCT	47	Rogers & Li 1985
$CdCl_2$	native soil microflora	clay	8	1.5	42	Phosphatase activity	–	9779 ED_{50}	50	Doelman & Haanstra 1989
$Cd(NO_3)_2$	*Pseudomonas denitrificans*	silt loam	7	2	4	Denitrification	10	50	22	Bollag & Barabasz 1979
$Cd(NO_3)_2$	*Pseudomonas aeruginosa*	silt loam	7	2	4	Denitrification	10	50	25	Bollag & Barabasz 1979
$CdCl_2$	native soil microflora	silt loam	5	–	–	Nitrification	–	50	>20	Suter and Sharples 1984
$CdCl_2$	native soil microflora	clay	8	1.5	42	Arylsulfatase activity	–	9520 ED_{50}	50	Haanstra & Doelman 1991
$CdCl_2$	native soil microflora	silty loam	8	1	42	Phosphatase activity	–	5485 ED_{50}	50	Doelman & Haanstra 1989
$CdCl_2$	native soil microflora	clay	8	1.5	548	Phosphatase activity	–	5305 ED_{50}	50	Doelman & Haanstra 1989

Table 10-5 (continued)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$CdCl_2$	native soil microflora	clay	8	1.5	42	Urease activity	–	4460 ED_{50}	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy peat	4	6.5	42	Urease activity	–	3260 ED_{50}	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy peat	4	6.5	42	Arylsulfatase activity	–	3192 ED_{50}	50	Haanstra & Doelman 1991
$CdSO_4$	native soil microflora	clay loam	8	3.2	0.1	Arylsulfatase activity	281	2810	23	Al-Khafaji & Tabatabai 1979
$CdSO_4$	native soil microflora	loam	7	2.9	0.1	Arylsulfatase activity	281	2810	55	Al-Khafaji & Tabatabai 1979
$CdSO_4$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	281	2810	44	Juma & Tabatabai 1977
$CdSO_4$	native soil microflora	loam	7	5.3	0.1	Arylsulfatase activity	–	2810 LCT	42	Al-Khafaji & Tabatabai 1979
$CdSO_4$	native soil microflora	clay loam	6	2.7	0.1	Arylsulfatase activity	–	2810 LCT	27	Al-Khafaji & Tabatabai 1979
$CdSO_4$	native soil microflora	loam	7	5.5	0.1	Acid phosphatase activity	–	2810 LCT	48	Juma & Tabatabai 1977
$CdSO_4$	native soil microflora	clay loam	8	3.7	0.1	Alkaline & acid phosphatase activity	–	2810 LCT	78, 51	Juma & Tabatabai 1977
$CdCl_2$	native soil microflora	sandy soil	7	1	42	Arylsulfatase activity	–	2214 ED_{50}	50	Haanstra & Doelman 1991

Table 10-5 (continued)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$CdCl_2$	native soil microflora	silt loam	8	1	42	Arylsulfatase activity	–	1888 ED_{50}	50	Haanstra & Doelman 1991
$CdCl_2$	native soil microflora	sandy loam	6	3	548	Arylsulfatase activity	–	1798 ED_{50}	50	Haanstra & Doelman 1991
$CdCl_2$	native soil microflora	clay	8	1.5	548	Arylsulfatase activity	–	1016 ED_{50}	50	Haanstra & Doelman 1991
$CdSO_4$	native soil microflora	surface soil	6	2.9	35	Nitrification	500	1000	62	Bewley & Stotzky 1983
$CdCl_2$	native soil microflora	silty loam	8	1	42	Urease activity	–	970 ED	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	soil/litter microcosm	–	–	–	Respiration	–	920 LCT	61	Lighthart et al. 1977
$CdCl_2$	native soil microflora	sandy soil	7	1	42	Phosphatase activity	–	840 ED	50	Doelman & Haanstra 1989
$C_4H_6CdO_4$	native soil microflora	acid cambisol	6	1	84	Respiration	28	56	23	Reber 1989
$CdCl_2$	native soil microflora	Brown earth	5	–	30	Cellulolytic activity	50	100	35	Khan & Frankland 1984
$Cd(NO_3)_2$	native soil microflora	silt loam	7	2	21	Denitrification	50	100	27	Bollag & Barabasz 1979
$CdCl_2$	native soil microflora	sandy soil	7	1	548	Urease activity	–	120 ED	50	Doelman & Haanstra 1986
$CdSO_4$	native soil microflora	clay loam	8	4	20	N mineralization	–	562 LCT	39	Liang & Tabatabai 1977
$CdCl_2$	native soil microflora	silty loam	8	1	548	Urease activity	–	520 ED	50	Doelman & Haanstra 1986

Table 10-5 (continued)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$CdSO_4$	Native soil microflora	silty clay	7	3	20	N mineralization	–	562 LCT	27	Liang & Tabatabai 1977
$CdCl_2$	native soil microflora	clay	8	1.5	548	Urease activity	–	520 ED	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy soil	7	1	548	Arylsulfatase activity	–	121 ED	50	Haanstra & Doelman 1991
$CdCl_2$	native soil microflora	silty loam	8	1	548	Arylsulfatase activity	–	137 ED	50	Haanstra & Doelman 1991
$CdCl_2$	native soil microflora	soil/litter microcosm	–	–	24	Respiration	0.006	6.1	43	Lighthart & Bond 1976
$Cd(NO_3)_2$	*Pseudomonas* sp.	silt loam	7	2	4	Denitrification	–	10 LCT	23	Bollag & Barabasz 1979
$C_4H_6CdO_4$	native soil microflora	phaeosem	7	1	84	Respiration	7	14	22	Reber 1989
$CdCl_2$	native soil microflora	silty loam	8	1	548	Phosphatase activity	–	230 ED	50	Doelman & Haanstra 1989
$CdSO_4$	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	–	281 LCT	27	Juma & Tabatabai 1977
$CdCl_2$	native soil microflora	sandy peat	4	6.5	548	Urease activity	–	490 ED	50	Doelman & Haanstra 1986
$CdCl_2$	native soil microflora	sandy soil	7	1	548	Phosphatase activity	–	330 ED	50	Doelman & Haanstra 1989
$CdCl_2$	*Eisenia andrei*	OECD soil	6	5	84	growth	10	32	40	van Gestel et al. 1991

Table 10-5 (continued)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$CdCl_2$	*Eisenia andrei*	OECD soil	6	5	84	sexual development EC_{50}	–	27	50	van Gestel et al. 1991
$Cd(NO_3)_2$	*Eisenia fetida*	OECD soil	6	–	56	cocoon production EC_{50}	–	46.3	50	Spurgeon et al. 1994
$C_4H_6CdO_4$	*Eisenia fetida*	horse manure	–	–	56	cocoon production	–	25LCT	52	Malecki et al. 1982
$CdCl_2$	*Eisenia andrei*	OECD soil	6	5	21	cocoons/worm; juveniles/worm	10	18	23,22	van Gestel et al. 1992
$CdCl_2$	*Lumbricus rubellas*	sandy loam	7	4	84	survival	150	1000	82	Ma 1982
$CdCl_2$	*Eisenia fetida*	sandy soil	4	0.9	14	survival LC	–	440	50	van Gestel & van Dis 1988
$CdCl_2$	*Eisenia andrei*	OECD soil	6	5	84	sexual development EC_{50}	–	108	50	van Gestel et al. 1991
	Dendrobaena rubida	soil & dung	7	5.7	120	% cocoon hatching success;	10	100	47	Bengtsson et al. 1986
						hatchlings/coc; total hatchlings			38,30	
$C_4H_6CdO_4$	*Eisenia fetida*	horse manure			140	cocoon production		50LCT	24	Malecki et al. 1982
Soluble forms	*Eisenia fetida*	horse manure			42	growth;cocoon production		100LCT	25,10 0	Neuhauser et al. 1984

Table 10-5 (continued)

Chemical form	Organisms	Growth medium	pH	% OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$CdCl_2$	*Eisenia andrei*	OECD soil	6	5	84	growth	32	100	44	van Gestel et al. 1991
	Dendrobaena rubida	soil & dung	5	5.7	120	cocoons/worm	10	100	62	Bengtsson et al. 1986
	Dendrobaena rubida	soil & dung	6	5.7	120	cocoons/worm; hatchlings/cocoon	10	100	78,71	Bengtsson et al. 1986
						total hatchlings			74	
$CdNO_3$	*Eisenia fetida*	OECD soil	6	5	14	survival LC	–	1843	50	Neuhauser et al. 1985

Note: Chemical concentrations are g of element/kg of growth medium.
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared with controls.
EXP (D) = exposure in days.
Growth medium:OECD soil (% dry weight): sphagnum peat, 10; kaolin clay, 20; fine sand, 69; $CaCO_3$, 1; pH 6.0.
% OC = % organic carbon.

Bollag and Barabasz (1979) evaluated the effects of several metals on denitrification in autoclaved soil by three species of soil-dwelling *Pseudomonas* species of bacteria and on denitrification in soil by native soil microflora. In the autoclaved soil, two of the three *Pseudomonas* species had reductions in activity at 50 mg Cd/kg, while the third was more sensitive to the toxic effects of this metal (and Cu and Zn) on denitrification. The native soil population in unautoclaved soil was more tolerant of the cadmium, with reductions in activity at 100 mg Cd/kg. It is not clear whether this difference is due to changes in the chemical and physical nature of the soil during autoclaving or to the greater tolerance of the natural soil population to cadmium.

Khan and Frankland (1984) used a dyed cellophane film technique to evaluate the effects of cadmium on cellulolytic activity of native soil microflora in a Brown earth soil. A 35% reduction in percent cellulose decomposition was measured in pots containing 100 mg Cd/kg, while 50 mg/kg had no effect. Lighthart et al. (1977) evaluated the effects of a number of metals at single concentrations on respiration of native soil microflora in small coniferous forest soil/litter microcosms. Cadmium at 920 mg/kg reduced respiration 61%. In a study of the effects of cadmium on N mineralization and nitrification by native soil microflora in a moderately acid soil, Bewley and Stotzky (1983) found N mineralization to be unaffected by cadmium levels up to 1000 mg/kg, the highest concentration tested. Nitrification was reduced 62% by 1000 mg/kg. The effects of cadmium as $CdCl_2$ on carbon and nitrogen mineralization and nitrogen transformations in alfalfa-amended sieved soil were determined by Suter and Sharples (1984). Respiration was reduced by 20% at 500 mg/kg, ammonia levels were increased by as much as a factor of 12, and nitrate levels were reduced by >21% at 50 mg Cd/kg and higher. These results suggest that nitrification is more sensitive than mineralization to cadmium.

van Gestel et al. (1992) have established a fairly standard procedure for testing the toxicity of chemicals to earthworms (*Eisenia andrei*) in an artificial soil mixture. The EC_{50} for clitella development (indicating sexual maturity) was 108 mg Cd/kg for dung placed in a center hole in the substrate, and 27 mg/kg for dung mixed in with substrate (van Gestel et al. 1991). Spurgeon et al. (1994) tested the effects of cadmium [as $Cd(NO_3)_2$] on survival and growth of *E. fetida*. The calculated LC_{50} was greater than 300 mg Cd/kg. The EC_{50} for cocoon production was 46.3 mg/kg. Malecki et al. (1982) tested the effects of cadmium added to horse manure (as cadmium acetate) on *E. fetida*. In the 8-week test, the lowest concentration tested, 25 mg Cd/kg, caused a 52% decrease in cocoon production. In the 20-week test, the lowest concentration tested, 50 mg Cd/kg, caused a 24% decrease in cocoon production.

Neuhauser et al. (1984) looked at the effects of cadmium added as various soluble salts on growth and reproduction of *E. fetida*. After 6 weeks, both growth (weight) and cocoon production were decreased (25 and 100%) by 100 mg Cd/kg, the lowest concentration tested.

Bengtsson et al. (1986) report the effects of cadmium on reproduction in the earthworm *Dendrobaena rubida* when grown in a substrate at varying acidity. The metal was added to a 1:2 (volume) combination of sandy soil and well-decomposed cattle dung with a resulting organic carbon of about 6%. After 4 months at pH 4.5, the number of cocoons produced per worm was reduced 62% by 100 mg Cd/kg, while 10 mg/kg had no effect. The percentages of hatched cocoons, hatchlings/cocoon, and total number of hatchlings were not affected. At pH 5.5, the number of cocoons produced per worm, hatchlings/cocoon, and total number of hatchlings were reduced 78, 71, and 74%, respectively, by 100 mg Cd/kg, while 10 mg/kg had no effect. The percent hatched cocoons was not affected. At pH 6.5, the percent hatched cocoons, hatchlings/cocoon, and total number of hatchlings were reduced 47, 38, and 30%, respectively, by 100 mg Cd/kg, while 10 mg/kg had no effect. The number of cocoons/worm was not affected. van Gestel and van Dis (1988) investigated the effects of acidity on acute toxicity of cadmium ($CdCl_2$) to adult *E. andrei*. The LC_{50} was between 320 and 560

mg Cd/kg after 14 days at pH 4.1 and >1000 (no effect) at pH 7. The LC_{50} concentration in OECD soil, with 10% organic matter at pH 7, was also >1000 (no effect).

Neuhauser et al. (1985) used OECD artificial soil (pH 6) to determine LC_{50} of cadmium (added as cadmium nitrate) for adult *E. fetida*. After 14 days, the LC_{50} was calculated to be 1843 mg Cd/kg. Ma (1982) used a sandy loam soil spiked with $CdCl_2$ to determine the effects of cadmium on survival of adult *Lumbricus rubellus*. After 12 weeks, 1000 mg Cd/kg caused an 82% decrease in survival, while 150 mg/kg had no effect.

Kammenga et al. (1994) compared the acute toxicity of cadmium ($CdCl_2$) to seven terrestrial nematode species belonging to different taxonomic and ecological groups. The nematodes *T. elegans* and *A. avenae* had LC_{50} values of greater than 90 mg/kg after 96 hours; they were essentially unaffected by the experimental treatment. Among the bacterial feeders the LC_{50} ranged from approximately 3 to 60 mg Cd/kg after 72 hours. The nematode *C. elegans* was used by van Kessel et al. (1989) to test the effects of cadmium (as $CdCl_2$) on growth (length) and reproduction (number of juveniles per adult) of this soil-dwelling group. Reproduction, the more sensitive parameter, was reduced 36% by 3.6 mg/kg. The influence of cadmium (as $CdCl_2$) on life-history characteristics of the collembolan *Folsomia candida* was investigated by Crommentuijn et al. (1993). A soil concentration ($HCl+HNO_3$ extractable) of 326 mg Cd/kg reduced the number of offspring produced by 21% after 42 days. Representatives of Collembola and mites were used by van Straalen et al. (1989) to assess the effect of cadmium in diets of soil microarthropods on population growth rates. A concentration of 15 mg Cd/kg caused an approximate 56% reduction in calculated population growth rate after 61 days, while 4.7 mg/kg had no effect. Adult mites (*P. peltifer*) fed the same diet for 84 days experienced a 23% reduction in population growth rate at a concentration of 9 mg Cd/kg, while 3 mg/kg had no effect. Hopkin and Hames (1994) investigated the effects of cadmium (as $CdNO_3$) in food on survival and reproduction of the terrestrial isopod *Porcellio scaber*. The number of juveniles produced was decreased 47% by 10 mg Cd/kg (lowest concentration tested) while 50 mg/kg was required to reduce total survival. The effects of cadmium on individual weight, new shell growth, and reproductive behavior of the snail *Helix aspersa* was evaluated by Russell et al. (1981). Reproductive activity was reduced 28% by 25 mg Cd/kg, while 10 mg/kg had no effect. New shell growth and weight were not affected at this concentration. There is agreement between the two studies on the effects of cadmium on nematodes in that approximately 3 mg/kg in solution is detrimental to the organisms.

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11. CALCIUM

11.1 BACKGROUND

Calcium (Ca) is the third most abundant metal and makes up an average concentration of 3.64 mg/kg of the earth's crust. It is an essential nutrient and occurs in surface waters primarily as calcium carbonate ($CaCO_3$). Calcium, as dissolved cations in fresh water, is chiefly responsible for water hardness and reduces the toxicity of several other metals (Rand and Petrocelli 1984). Calcium, essential to plant and animal life, is present in bones, teeth, eggshell, coral, and many soils.

11.2 TOXICITY IN WATER TO AQUATIC LIFE

11.2.1 Acute Toxicity

Although calcium is rarely toxic in ambient waters, acute toxicity values for *Daphnia magna* were determined in laboratory tests by Biesinger and Christensen (1972). The 48-hour LC_{50}s for $CaCl_2$ were 464.0 with food added and 52.0 mg/L without food added.

11.2.2 Chronic Toxicity

Three-week exposure to *D. magna* of 11.6 mg/L $CaCl_2$ with food added caused a 16% decrease in reproduction. Calcium chloride is likely to result in a higher rate of dissociation than calcium carbonate. Therefore, measured concentrations of calcium carbonate in ambient waters are likely to overestimate the free Ca(II) fraction (Biesinger and Christensen 1972).

During the brown trout *(Salmo trutta)* life cycle, freshly fertilized eggs proved to be the most sensitive to ionic concentrations of calcium between 1.0 and 8.0 mg/L in acidic water pH 4.5 (Brown 1982). Such low pH levels may occur where acidic wastes are released.

11.2.3 Toxicity to Aquatic Plants

No information was found on the toxicity of calcium to aquatic plants.

11.2.4 Bioaccumulation

Calcium is a major nutrient and under most conditions, internal concentrations are well regulated.

11.2.5 Aquatic Mode of Action

Where calcium toxicity has been reported, the apparent mechanism of action is induction of ionic imbalances.

11.2.6 Water Quality Criteria

There are no National Ambient Water Quality Criteria or secondary values for calcium.

11.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on the toxicity of calcium to benthic invertebrates.

11.4 TOXICITY TO WILDLIFE

11.4.1 Toxicity to Mammals

No information was found on the toxicity of calcium to mammals.

11.4.2 Toxicity to Birds

No information was found on the toxicity of calcium to birds.

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12. CHROMIUM

12.1 BACKGROUND

Chromium (Cr) occurs in the environment as either chromium (III) or chromium (VI). Trivalent chromium is an essential metal in animals, playing an important role in insulin metabolism (Larngard and Norseth 1979). Hexavalent chromium is more toxic than chromium (III) because of its high oxidation potential and the ease with which it penetrates biological membranes (Steven et al. 1976; Taylor and Parr 1978). Chromium (III), the predominant form in the environment, exhibits decreasing solubility with increasing pH, and is completely precipitated at a pH above 5.5. In most soils, chromium is primarily present as precipitated chromium (III), which is not bioavailable and has not been know to biomagnify through food chains in its inorganic form (Eisler 1986). Chromium is released into the environment in the processing of chromate, electroplating, production at tanning and textile plants, pigment production, and cooling tower preservation. Cr is naturally released into the environment through the weathering of soils (Fishbein 1976).

12.2 TOXICITY IN WATER TO AQUATIC LIFE

12.2.1 Acute Toxicity

Acute toxicity is higher for hexavalent than trivalent chromium when tested in the same water and with the same species, but the ranges of reported acutely lethal levels largely overlap. Hexavalent chromium LC_{50}s range from 0.022 to 1,870 mg/L for invertebrates in 50 tests and from 17.6 to 147.5 mg/L in fishes in 80 tests (Table 12-1). Trivalent chromium LC_{50}s range from 1.2 to 64 mg/L for invertebrates in 16 tests and from 4.1 to 71.9 mg/L in fishes in 17 tests (Table 12-1).

12.2.2 Chronic Toxicity

Chronic toxicity is higher for hexavalent than trivalent chromium when tested in the same water and with the same species, but the ranges of reported acutely lethal levels largely overlap. Hexavalent chromium CVs range from <0.0025 to 0.040 mg/L for invertebrates in six tests and from 0.073 to 2.0 mg/L in fishes in four tests (Table 12-1). Trivalent chromium CVs range from <0.044 to 0.19 mg/L for invertebrates in four tests and from 0.068 to 1.02 mg/L in fishes in two tests (Table 12-1).

12.2.3 Toxicity to Aquatic Plants

Levels of hexavalent chromium that are toxic to plants range from 0.002 to 7.8 mg/L in 28 tests (EPA 1985). Levels of trivalent chromium that are toxic to plants range from 0.40 to 9.9 mg/L in three tests (EPA 1985).

12.2.4 Bioaccumulation

Concentration factors in aquatic plants tend to decrease with increasing chromium concentration (Garg and Chandra 1994). At a concentration of 2.67 mg/L the accumulation of Cr was twelve times higher in roots than in the shoots of a series of aquatic plants (Jana 1987).

Buhler et al. (1977, as cited in Sastry and Sunita 1982) found that when chronicly exposed to chromium, fish tend to retain significant amounts of the metal in the kidney, liver, gills, gall

Table 12-1. Toxicity of chromium to aquatic animals; all data are from EPA (1995-Cr) unless otherwise noted.

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
		Chromium (VI)			
Stonefly, Neophasganophora capitata	LC_{50}	Potassium dichromate	120-160	1870	1870
Crayfish Orconectes rusticus	LC_{50}	Potassium dichromate	120-160	176	176
Bluegill, Lepomis macrochirus	LC_{50}	Potassium chromate	45	170	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium chromate	44	168.8	
Green sunfish, Lepomis cyanellus	LC_{50}	Potassium dichromate	400	147.56	114.7
Bluegill, Lepomis macrochirus	LC_{50}	Potassium chromate	44	147	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	120-160	144.5	
Damselfly, Enallagma aspersum	LC_{50}	Potassium dichromate	120-160	140	140
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	135	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	171	135	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	133	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	360	133	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	133	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	133	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	20-44	132.89	132.9
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	171	130.4	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	129	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	126	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	126	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	126	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	125	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	124	119.5
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	123	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	123	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium chromate	44	120	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	20	118	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	44	113	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	44	113	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	44	113	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	110	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	—	110	
Bluegill, Lepomis macrochirus	LC_{50}	Potassium dichromate	45	110	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	109	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	102	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	98	
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	220	90	
Green sunfish, Lepomis cyanellus	LC_{50}	Potassium dichromate	400	89.16	
Striped shiner, Notropis chrysocephalus	LC_{50}	Potassium dichromate	120-160	85.6	86.6
Sand shiner, Notropis stramineus	LC_{50}	Potassium dichromate	120-160	74.6	74.6
White crappie, Pomoxis annularis	LC_{50}	Potassium dichromate	120-160	72.6	72.6
Rainbow trout, Salmo gairdneri	LC_{50}	Sodium dichromate	45	69	69
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	66	
Midge, Chironomus tentans	LC_{50}	Potassium dichromate	101	61	61
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	60	
Brook trout, Salvelinus fontinalis	LC_{50}	Sodium dichromate	45	59	59

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	120-160	58	
Midge, Tanytarsus dissimilis	LC_{50}	Potassium dichromate	47	57.3	57.3
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	56	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	55	
Bluntnose minnow, Pimephales notatus	LC_{50}	Potassium dichromate	120-160	54.225	54.22
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	53	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	53	
Fathead minnow, Pimephales promelas	LC_{50}	—	—	52	
Central stoneroller, Campostoma anomalum	LC_{50}	Potassium dichromate	120-160	51.25	51.25
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	51	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	50	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Silverjaw minnow, Ericymba buccata	LC_{50}	Potassium dichromate	120-160	49.6	49.6
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	49	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	49	
Emerald shiner, Notropis atherinoides	LC_{50}	Potassium dichromate	120-160	48.4	48.4
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	48	
Johnny darter, Etheostoma nigrum	LC_{50}	Potassium dichromate	120-160	46	46
Fathead minnow, Pimephales promelas	LC_{50}	Potassium chromate	20	45.6	
Snail, Physa heterostropha	LC_{50}	Potassium chromate	171	40.6	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	209	39.7	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	38	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	209	37.7	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Goldfish, Carassius auratus	LC_{50}	Potassium dichromate	20	37.5	
Fathead minnow, Pimephales promelas	LC_{50}	—	—	37	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	37	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	209	37	
Yellow perch, Perca flavescens	LC_{50}	Potassium dichromate	120-160	36.3	36.3
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	209	35.9	
Striped bass, Morone saxatilis	LC_{50}	Potassium dichromate	35	35	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	34	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	34	
Fathead minnow, Pimephales promelas	LC_{50}	Sodium dichromate	220	33.2	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	209	32.7	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Snail, Physa heterostropha	LC_{50}	Potassium chromate	171	31.6	23.01
Guppy, Poecilia reticulata	LC_{50}	Potassium dichromate	20	30	30
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	29	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	360	27.3	
Striped bass, Morone saxatilis	LC_{50}	Potassium dichromate	35	26.5	30.45
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	220	26	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	400	24.14	
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	400	22.58	41.05
Fathead minnow, Pimephales promelas	LC_{50}	Potassium dichromate	20	17.6	
Snail, Physa heterostropha	LC_{50}	Potassium chromate	45	17.3	
Snail, Physa heterostropha	LC_{50}	Potassium chromate	45	17.3	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Snail, Physa heterostropha	LC_{50}	Potassium dichromate	43	16.8	
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	—	3.49	
Fathead minnow, Pimephales promelas	CV	Potassium dichromate	209	1.987	
Bryozoan, Lophopodella carteri	LC_{50}	Potassium chromate	205	1.56	1.56
Bryozoan, Pectinatella magnifica	LC_{50}	Potassium chromate	205	1.44	1.44
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	45	0.9	
Cladoceran, Daphnia pulex	LC_{50}	Potassium dichromate	45	0.76	
Bryozoan, Plumatella emarginata	LC_{50}	Potassium chromate	205	0.65	0.65
Amphipod, Hyalella azteca	LC_{50}	Potassium chromate	50	0.63	0.63
Rainbow trout, Salmo gairdneri	CV	Sodium dichromate	45	0.2646	
Brook trout, Salvelinus fontinalis	CV	Sodium dichromate	45	0.2646	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	213	0.212	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	100	0.175	
Cladoceran, Daphnia magna	LC_{50}	Sodium chromate	185	0.164	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	92	0.157	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	—	0.141	
Cladoceran, Daphnia magna	LC_{50}	Potassium chromate	212	0.137	
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	185	0.131	
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	—	<0.123	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	240	0.11	
Cladoceran, Daphnia magna	LC_{50}	Sodium chromate	—	<0.103	
Amphipod, Gammarus pseudol imnaeus	LC_{50}	Potassium chromate	50	0.101	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Amphipod, Gammarus pseudol imnaeus	LC_{50}	Potassium dichromate	48	0.0941	
Cladoceran, Ceriodaphnia reticulata	LC_{50}	Sodium dichromate	45	0.0857	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	240	0.081	
Cladoceran, Daphnia magna	LC_{50}	Sodium chromate	213	0.0758	
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	196	0.0736	
Rainbow trout, Salmo gairdneri	CV	Chromium trioxide	34	0.07318	
Amphipod, Gammarus pseudol imnaeus	LC_{50}	Potassium dichromate	48	0.0671	0.067
Cladoceran, Daphnia magna	LC_{50}	Potassium chromate	188	0.0667	
Cladoceran, Simocephalus vetulus	LC_{50}	—	45	0.05	
Cladoceran, Daphnia magna	LC_{50}	Sodium chromate	—	0.05	
Cladoceran, Daphnia pulex	LC_{50}	—	45	0.048	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Cladoceran, Ceriodaphnia reticulata	LC_{50}	—	45	0.045	0.045
Cladoceran, Simocephalus serrulatus	LC_{50}	Sodium dichromate	45	0.0409	0.041
Cladoceran, Ceriodaphnia reticulata	CV	Sodium dichromate	45	0.04	
Cladoceran, Daphnia pulex	LC_{50}	Sodium dichromate	45	0.0363	0.036
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	240	0.035	
Cladoceran, Simocephalus vetulus	LC_{50}	Sodium dichromate	45	0.0323	0.032
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	45	0.0242	
Cladoceran, Daphnia magna	LC_{50}	—	45	0.022	0.023
Cladoceran, Daphnia magna	LC_{50}	Sodium dichromate	50	0.0213	
Cladoceran, Daphnia magna	LC_{50}	Sodium chromate	50	0.0206	
Cladoceran, Simocephalus serrulatus	CV	Sodium dichromate	45	0.0199	

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	50	0.0199	
Cladoceran, Daphnia magna	LC_{50}	Potassium dichromate	196	0.0199	
Cladoceran, Daphnia magna	LC_{50}	Potassium chromate	50	0.0153	
Cladoceran, Daphnia magna	CV	Sodium chromate	—	<0.01	
Cladoceran, Simocephalus vetulus	CV	Sodium dichromate	45	0.006132	
Cladoceran, Daphnia pulex	CV	Sodium dichromate	45	0.006132	
Cladoceran, Daphnia magna	CV	Sodium dichromate	45	<0.0025	
		Chromium (III)			
Bluegill, Lepomis macrochirus	LC_{50}	Chromium potassium sulfate	360	71.9	15.02
Fathead minnow, Pimephales promelas	LC_{50}	Chromium potassium sulfate	360	67.4	
Caddisfly, Hydropsyche betteni	LC_{50}	chromic chloride	44	64	71.06
Cladoceran, Daphnia magna	LC_{50}	chromic nitrate	215	58.7	16.01

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Cladoceran, Daphnia magna	LC_{50}	chromic nitrate	195	51.4	
Caddisfly, (Unidentified)	LC_{50}	—	50	50	50
Damselfly, (Unidentified)	LC_{50}	—	50	43.1	43.1
Fathead minnow, Pimephales promelas	LC_{50}	Chromium potassium sulfate	203	29	
Cladoceran, Daphnia magna	LC_{50}	chromic nitrate	99	27.4	
Fathead minnow, Pimephales promelas	LC_{50}	Chromium potassium sulfate	203	27	10.32
Cladoceran, Daphnia magna	LC_{50}	chromic nitrate	110	26.3	
Rainbow trout (2 mos), Salmo gairdneri	LC_{50}	chromic nitrate	—	24.1	
Striped bass, Morone saxatilis	LC_{50}	—	55	17.7	16.37
Pumpkinseed, Lepomis gibbosus	LC_{50}	—	55	17	15.72
Banded killifish, Fundulus diaphanus	LC_{50}	—	55	16.9	15.63

Table 12-1. (continued).

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Cladoceran, Daphnia magna	LC_{50}	chromic nitrate	52	16.8	
White perch, Morone americana	LC_{50}	—	55	14.4	13.32
Common carp, Cyprinus carpio	LC_{50}	—	55	14.3	13.23
American eel, Anguilla rostrata	LC_{50}	—	55	13.9	12.86
Snail (embryo), Amnicola sp.	LC_{50}	—	50	12.4	
Rainbow trout, Salmo gairdneri	LC_{50}	Chromium Chloride	44	11.2	
Midge, Chironomus sp.	LC_{50}	—	50	11	11
Worm, Nais sp.	LC_{50}	—	50	9.3	9.3
Snail (adult), Amnicola sp.	LC_{50}	—	50	8.4	10.21
Bluegill, Lepomis macrochirus	LC_{50}	Chromium potassium sulfate	20	7.46	
Crayfish, Orconectes limosus	LC_{50}	chromium chloride	—	6.6	
Fathead minnow, Pimephales promelas	LC_{50}	Chromium potassium sulfate	20	5.07	

Table 12-1 (continued)

Species	Effect	Chemical	Hardness (mg/L as $CaCO_3$)	Concentration (mg/L)[1]	Species mean acute value (mg/L)[1,2]
Rainbow trout, Salmo gairdneri	LC_{50}	Chromium nitrate	26	4.4	9.669
Goldfish, Carassius auratus	LC_{50}	Chromium potassium sulfate	20	4.1	8.684
Guppy, Poecilia reticulata	LC_{50}	Chromium potassium sulfate	20	3.33	7.053
Amphipod, Gammarus sp.	LC_{50}	—	50	3.2	3.2
Mayfly, Ephemerella subvaria	LC_{50}	chromic chloride	44	2	2.221
Cladoceran, Daphnia magna	LC_{50}	chromic chloride	—	1.2	
Fathead minnow Pimephales promelas	CV	Chromium potassium sulfate	203	1.025	
Cladoceran, Daphnia magna	CV	Chromic nitrate	100	0.1937	
Rainbow trout, Salmo gairdneri	CV	Chromium nitrate	26	0.06963	
Cladoceran, Daphnia magna	CV	Chromic nitrate	52	0.06611	
Cladoceran, Daphnia magna	CV	Chromic nitrate	206	<0.044	

[1] All concentrations are for chromium, not the chemical compound.
[2] For chromium(III), species mean acute values are adjusted to a hardness of 50 mg/L (EPA 1985-Cr).

bladder, and bile. Bioconcentration factors for hexavalent chromium in three studies of rainbow trout were <1, 1, and 2.8 (EPA 1985).

12.2.5 Aquatic Mode of Action

Fish gills involvement in osmoregulation and respiration is impaired by Cr(VI). Fish were unable to compensate for a loss of plasma ions as indicated by the increasing levels of sodium and plasma osmolality with increased exposure times. Damage of the gill tissue is witnessed by the formation of a film of coagulated mucus. Once entering the circulatory system through respiration in the gills, Cr(VI) is transported to the liver. Overdosing of chromium inhibits the conversion of blood lactate into liver glycogen as evidenced by the decrease in liver lactate dehydrogenase activity. Eventually, disturbed osmotic and ionic balance in the fish coupled with asphyxiation at the gills leads to mortality (Van Der Putte et al 1982, Wong et al. 1982, Sastry and Sunita 1982).

Cr was shown to increase rate of glucose absorption in the intestines of the snakefish, *Channa punctatus*, at all concentrations tested (Sastry and Sunita 1982). Symptoms of Cr(III) toxicocis in the common carp include the loss of epithelial cells on the gills, lesions of the liver, hypertrophy and congestion of the hepatic cells, disintegration of epithelial cells in the intestine and the presence of small particulates in the microvilli region and gills (Wong et al. 1982).

12.2.6 Water Quality Criteria

The acute criterion for Cr(IV) (0.016 mg/L) is not defined in terms of hardness, but the acute National Ambient Water Quality Criterion for Cr(III) is defined as $e^{(0.819[\ln(\text{hardness})]+3.69)}$ (EPA 1985). The chronic criterion for Cr(IV) (0.011 mg/L) is not defined in terms of hardness, but the chronic National Ambient Water Quality Criterion for Cr(III) is defined as $e^{(0.819[\ln(\text{hardness})]+1.56)}$ (EPA 1985).

12.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Most of the available data for chromium toxicity are from tests of marine and estuarine sediments (Table 12-2). The toxic concentrations ranged from 17.40 mg/kg, which was associated with a 25% mortality in tests with the sea urchin *Arbacia punctulata*, to 682 mg/kg, which was associated with reduced taxa richness and reduced density of classes of invertebrates. Although there incidence of effects was high in the probable-effects range calculated from these results, this was greatly influenced and exaggerated by data from multiple tests conducted in only two field surveys (Long et al. 1995). There is relatively little consistency in the effects data presented by NOAA (Long and Morgan, 1991). This may be due to the lack of chromium speciation data in the NOAA studies. All of the data were reported as total chromium, whereas the hexavalent form has been reported to be the most toxic. Long and Morgan (1991) cited the results of six freshwater sediment studies that they considered to be of sufficient quality for calculating sediment benchmarks. Five of those studies reoprted signifcant mortality of *Daphnia magna* in overlying-water, but the sediment concentrations associated with that effect ranged from 72.6 mg/kg to 980 mg/kg. In Torch Lake, Michigan, concentrations of 180 mg/kg of chromium were associated with significant mortality to *D. magna* and *Hexagenia sp.* (studies cited in Long and Morgan 1991).

Table 12-2. Chromium toxicity to benthic invertebrates in marine and estuarine sediment (MacDonald et al. 1994).

Conc. (mg/kg)	Endpoint	Species
17.40	48h LC_{25}	*Arbacia punctulata* (sea urchin)
31.30	10d LC_{83}	*Leptocheirus plumulosus* (amphipod)
35.70	20d LC_{95}	*Leptocheirus plumulosus* (amphipod)
48.3-54.0	10d LC_{55}	*Hyalella azteca* (amphipod)
57.90	1h EC_{98} (fertilization)	*Arbacia punctulata* (sea urchin)
59.90	48h EC_{85} (development)	*Arbacia punctulata* (sea urchin)
60.00	48h Significant increase in burrowing time	*Macoma balthica* (bivalve)
60.40	low density	echinodermata
61.20	low density	arthropods
62.00	low spec. richness	benthic species
77.40	EC_{50}	*Photobacterium phosphoreum* (Microtox)
87.30	Sediments devoid of feral clams	*Macoma balthica* (bivalve)
90.00	24h - avoidance	*Macoma balthica* (bivalve)
93.70	10d LC_{32}	*Ampelisca abdita* (amphipod)
108.00	20d EC<90 (emergence)	*Hyalella azteca* (amphipod)
108.00	10d LC_{29}	*Lepidactylus dytiscus* (amphipod)
108.00	10d EC_{90} (reburial)	*Lepidactylus dytiscus* (amphipod)
108.00	20d EC<90 (emergence)	*Lepidactylus dytiscus* (amphipod)
110.00	48h EC_{33} (abnormality)	*Crassostrea gigas* (oyster)
146.00	low abundance	arthropods
157.00	low spec. richness	benthic species
160.00	14d reduced growth rate	*Chromadorina germanice* (nematode)
201.00	low abundance	echinoderm
211.00	low abundance	echinoderms
227.00	low abundance	phoxocephalids
227.00	low abundance	amphipods

Table 12-2 (continued)

Conc. (mg/kg)	Endpoint	Species
369.00	14d LC_{100}	*Nereis virens* (sandworm)
383.00	low density	echinoderm
416.00	low spec. richness	benthic species
523.00	low density	echinoderm
523.00	low density	*Foraminifera* (sponge)
523.00	low density	*Ophiuroidea* (brittle star)
527.00	low density	amphipod
527.00	low density	phoxocephalid
669.00	10d LC_{21}	*Rhepoxynius abronius* (amphipod)
669.00	low density	mollusca
682.00	low density	crustacea
682.00	low density	phoxocephalid
682.00	low density	amphipod
682.00	low spec. richness	macro benthos

12.4 TOXICITY TO PLANTS

12.4.1 Toxicity to Plants in Soil

Cr(VI) is generally believed to be more toxic to plants and more mobile inside the plant than Cr(III). The valence of the Cr ion is more important in determining the distribution of the element than the specific species. There are, however, conflicting views on the differential uptake and metabolism of Cr(III) and Cr(VI). Cr(VI) may be absorbed into roots, or Cr(VI) may be reduced to Cr(III) just before absorption into the roots (Smith et al. 1989).

Turner and Rust (1971) investigated the effect of Cr added as Cr(VI) on soybean seedlings grown 3 days in a loam soil. Fresh shoot weight was reduced 30% by 30 mg Cr/L, while 10 mg/kg had no effect. Adema and Henzen (1989) calculated EC_{50} concentrations for effects of Cr added as Cr(VI) on lettuce, tomato and oats grown in a growth chamber from seed for 14d. The EC_{50} for lettuce in a humic sand soil (pH 5.1, % organic matter 3.7) was greater than 11 mg/kg while in a loam soil (pH 7.4, % organic matter 1.4) it was 1.8 mg Cr/kg. The EC_{50} for tomato in the humic sand soil was 21 mg/kg, while in the loam soil it was 6.8 mg Cr/kg. The EC_{50} for oats in the humic sand soil was 31 mg/kg, while in the loam soil it was 7.4 mg Cr/kg. Results of these experiments show the ameliorating effects of organic matter on Cr (VI) toxicity.

Farasgova (1994) studied the effects of metals on the germination and root growth of *Sinapsis alba* seeds at various pHs. 72h LC_{50}s for germination were 123.03 mg/kg at pH 2.46 and 100.0 mg/kg

at pH 7.25. 72h LC_{50}s for root growth inhibition were 5.01 mg/kg at pH 2.46 and 45.71 mg/kg at pH 7.25.

Will and Suter (1995a) reported soil NOEC and soil LOEC values for the toxicity of chromium to plants in soil. The NOEC values range from 0.35 to 11 mg/kg, and the LOEC values range from 1.8 to 31 mg/kg (Table 12-3).

12.4.2 Toxicity to Plants in Solution

Calculated EC_{50} concentrations for effects of Cr(VI) added as $K_2Cr_2O_7$ on lettuce, tomato and oats grown from seed for 14d ranged from 0.16 (lettuce) to 1.4 mg Cr/kg (oats) (Adema and Henzen 1989). The concentration of Cr(VI), from $(NH_4)_2CrO_4$, required for a 50% reduction in seed germination and root length of mustard after 3d exposure in solution (pH 7.3), was reported by Farasgova (1994) to be 100 mg/kg. EC_{50} for root length was 46 mg Cr/kg. Using a 1:1 combination of Cr(III) ($CrCl_3$) and Cr(VI) (K_2CrO_7) in nutrient solution (pH 5), Hara et al. (1976) measured a 68% reduction in weight of cabbage with 10 mg Cr/kg (2 mg/kg had no effect).

Top weight of soybean seedlings grown for 5d in nutrient solution containing Cr(VI) was reduced 21% by 1 mg Cr/kg, while 0.5 mg/kg had no effect (Turner and Rust 1971). Wallace et al. (1977) measured a 30% reduction in leaf weight of bush beans grown 11d in nutrient solution containing 0.54 mg Cr/kg as Cr (VI).

Length of the longest root of rye grass was reduced 69% by exposure to 2.5 mg Cr(VI)/kg (lowest concentration tested) in nutrient solution (pH 7) for 14d (Wong and Bradshaw 1982). Length

Table 12-3. Phytotoxicity data for the toxicity of chromium derived from experiments conducted in soil. (Will and Suter 1995a)

Chemical form	Soil type	Plant species	Soil NOEC (mg/kg)	Soil LOEC (mg/kg)	Growth parameter	Reference
$K_2Cr_2O_7$	loam	lettuce	0.35	1.8	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	loam	tomato	3.2	6.8	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	loam	oats	3.5	7.4	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	humic sand	lettuce	-	>11	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	humic sand	tomato	10	21	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	loam	soybean	10	30	fresh shoot weight	Turner & Rust, 1971.
$K_2Cr_2O_7$	humic sand	oats	11	31	fresh shoot weight	Adema & Henzen, 1989.

of the longest shoot was not affected at this concentration. Breeze (1973) found little difference in the toxicity of Cr(III) [$Cr_2(SO_4)_3$] and Cr(VI) ($K_2Cr_2O_7$) to rye grass seed germination. Seed exposed to solutions containing 50 mg/kg Cr (III) or (VI) reduced germination 37 and 38% after 2.5d.

Nutrient solution containing 0.05 mg Cr(III)/kg [$Cr_2(SO_4)_3$] reduced leaf and stem weights of chrysanthemum seedlings exposed for 2 d by 31 and 36% (Patel et al. 1976). This was the lowest concentration tested and root weight was not affected.

Based on these experiments, there is an indication that the source of the Cr affects plant response, and seed germination is not as sensitive as growth.

Will and Suter (1995a) reported NOEC and LOEC values for the toxicity of chromium to plants in solution. The NOEC values range from 0.004 to 50 mg/L and the LOEC values range from 0.052 to 100 mg/L (Table 12-4).

12.4.3 Phytotoxic Mode of Action

Chromium is not an essential element in plants. The (VI) form is more soluble and available to plants than the (III) form, and the former is considered the more toxic form (Smith, et al. 1989). In soils within a normal Eh and pH range, Cr(VI), a strong oxidant, is likely to be reduced to the less available Cr(III) form, although the (III) form may be oxidized to the (VI) form in the presence of oxidized Mn (Bartlett and James 1979). In nutrient solution, however, both forms are about equally taken up by plants and toxic to plants (McGrath 1982). Cr(VI), as CrO_4^{2-}, may share a root membrane carrier with SO_4^{2-} (Smith et al. 1989). Cr(VI) is more mobile in plants than Cr(III) but translocation varies with plant type. After plant uptake, Cr generally remains in the roots because of the many binding sites in the cell wall capable of binding especially the Cr(III) ions (Smith et al. 1989). Within the plant, Cr(VI) may be reduced to the Cr(III) form and complexed as an anion with organic molecules. Symptoms of toxicity include stunted growth, poorly developed roots, and leaf curling. Chromium may interfere with C, N, P, Fe, and Mo metabolism, and enzyme reactions (Kabata-Pendias and Pendias 1984).

12.5 TOXICITY TO WILDLIFE

12.5.1 Toxicity to Mammals

At high concentrations, chromium is a mutagen, teratogen and carcinogen (Eisler 1986). Chromium (VI) is more toxic than chromium (III); the LD_{50} for chromium (III) in mice is 260 mg/kg BW and 5 mg/kg BW for chromium (VI). Guinea pigs fed 50 mg/kg chromium (III) for 21 weeks showed no adverse effects (Preston et al. 1976). Ivankovic and Preussmann (1975), fed rats diets containining 1%, 2%, and 5% Cr_2O_3 (Cr^{+3}) for 90d to 2 years. No carcenogenicity or adverse effects on longevity or reproduction were observed at any dose level. Sample et al. (1996) considered the 5% dose (2737 mg/kg/d) to represent a chronic NOAEL for Cr^{+3} in rats.

In a study of the toxicity of chromium (VI) to rats, MacKenzie et a. (1958) exposed rats to six levels (0.45, 2.2, 4.5, 7.7, 11.2, and 25 mg/L) in water for 1 year. Because adverse effects were not observed at any level, Sample et al. (1996) considered the 25 mg/L dose (3.28 mg/kg/d) to represent a chronic NOAEL for Cr^{+6} in rats. Rats fed Cr(VI) reached a toxic threshold (mortality) at 1000 mg/L in water for 90 days (Steven et al. 1976, cited in Eisler 1986). A chronic LOAEL for mortality in rats

Table 12-4. Phytotoxicity data for the toxicity of chromium derived from experiments conducted in solution (Will and Suter 1995a).

Chemical form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	Reference
$CrSO_4$	chrysanthemum	-	0.052 LCT	stem & leaf weights	Patel et al., 1976.
$K_2Cr_2O_7$	lettuce	.004	0.16 EC_{50}	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	tomato	0.11	0.29 EC_{50}	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	bush beans	-	0.54 LCT	leaf weight	Wallace et al., 1977.
$K_2Cr_2O_7$	soybean	0.5	1	shoot weight	Turner and Rust, 1971.
$K_2Cr_2O_7$	oat	0.12	1.4 EC_{50}	fresh shoot weight	Adema & Henzen, 1989.
$K_2Cr_2O_7$	ryegrass	-	2.5 LCT	root length	Wong and Bradshaw, 1982.
$K_2Cr_2O_7$	rice	-	4.8 EC_{50}	radicle Weight	Wang, 1994.
$CrCl_3$+K_2CrO_4	cabbage	2	10	plant weight	Hara et al., 1976.
$(NH_4)_2CrO_4$	mustard	-	46 EC_{50}	root length	Fargasova, 1994.
$Cr_2(SO_4)_3$	rye grass	10	50	% seed gemination	Breeze, 1973.
$K_2Cr_2O_7$	ryegrass	10	50	% seed germination	Breeze, 1973.
$CrCl_3$	tomato	50	100	root weight and length	Moral et al., 1995.
$(NH_4)_2CrO_4$	mustard	-	100 LC_{50}	seed germination	Fargasova, 1994.

in rats (13.14 mg/kg/d) was estimated from the results of Steven et al. (1976, cited in Eisler 1986) by Sample et al. (1996).

12.5.2 Toxicity to Birds

Toxicity of chromium to aquatic birds is a concern due to the tendency of chromium to accumulate in roots, a highly selected food. If a study of chromium impacts on avian behavior, Heinz and Haseltine (1981) observed no effects on avoidance behavior from fright stimulus among 7-day-old black ducklings fed diets containing 20 or 200 mg Cr(III)/kg. Haseltine et al. (unpublished manuscript) fed black ducks diets containing 10 or 50 mg/kg Cr (III) for 10 months and evaluated reproductive performance. Although weight gain, egg laying, fertility and embryonic mortality were unaffected by either dose level, hen mortality was higher as reproductive season approached duckling survivorship was

reduced 28% by the 50mg/kg diet. Based on these results, Sample et al. (1996) estimated the NOAEL and LOAEL for reproductive effects in black ducks to be 1 and 5 mg/kg/d, respectively.

12.6 TOXICITY TO HETEROTROPHIC PROCESSES AND SOIL AND LITTER INVERTEBRATES

Liang and Tabatabai (1977) investigated the effects of various metals on N mineralization by native soil microflora in four soils. Chromium(III) at 260 mg/kg reduced N mineralization in the soil containing the highest organic matter content. The effects of Cr(III) on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). After 6 days, a concentration of 30 mg/kg Cr (the lowest concentration tested) reduced dehydrogenase activity by 54%. Juma and Tabatabai (1977) evaluated the effect of Cr on soil acid and alkaline phosphatase activities in microbes. Acid and alkaline phosphatase activities were affected at 1635 mg/kg in all three soils to about the same degree, but greater inhibition of alkaline phosphatase activity occurred in the soil with the greatest content of organic matter and clay. Ross et al. (1981) evaluated the relative toxicities of forms of Cr to respiration of native soil microflora in a loam and a sandy loam soil. Cr (III), tested at only 100 mg/kg, caused reductions of 41 and 48% in the two soils. A concentration of 10 mg/kg (the lowest concentration tested) Cr (VI) caused reductions in both soils (27 and 33%). Cr(VI) was more toxic than Cr(III) to soil respiration. Bhuiya and Cornfield (1976) investigated the effects of several metals on N mineralization and nitrification by native soil microflora in a sandy soil at different pH levels. At 6 weeks, mineralization and nitrification were reduced by 1000 mg/kg Cr at pH 7, but not at pH 6. After 12 weeks, neither mineralization nor nitrification was affected by Cr at either pH.

Haanstra and Doelman (1991) investigated short- and long-term effects of Cr on arylsulfatase activity, urease activity (Doelman and Haanstra 1986), and total phosphatase activity (Doelman and Haanstra 1989) by native soil microflora in five soils. The highest EC_{50}s were 3203, 5512, and 4470 mg/kg, respectively, for arylsulfatase, phosphatase, and urease activities found in different soils. The lowest was 17 mg/kg in the sand for arylsulfatase and 1170 and 490 mg/kg in the clay for phosphatase and urease. In an 18-month study, the highest EC_{50}s were 1798, 20020, and 1110 mg/kg Cr, respectively, for arylsulfatase, phosphatase, and urease activities found in different soils. The lowest were 12 and <1 mg/kg in the clay for arylsulfatase and urease activities and 2692 mg/kg in the sandy loam for phosphatase activity.

Abbasi and Soni (1983) assessed the effect of Cr(VI), added as $K_2Cr_2O_7$, on survival and reproduction of the earthworm *Octochaetus pattoni*. Survival was the most sensitive measure with a 75% decrease resulting from 2 mg/kg Cr, the lowest concentration tested. The number of cocoons produced was not diminished until the concentration reached 20 mg/kg Cr (highest concentration tested); the number of juveniles produced was not affected. Soni and Abbasi (1981) found no survival of *Pheretima posthuma* after 61 days in a paddy soil to which 10 mg/kg Cr(VI) (lowest concentration tested) was added. van Gestel et al. (1992) also found growth of *E. andrei* to be more sensitive to Cr than reproduction. 32 mg/kg Cr (III) reduced growth by 30% while cocoons/worm/week, percent fertile cocoons, and juveniles/worm/week were reduced 28, 22, and 51%, respectively, by 100 mg/kg Cr. Molnar et al. (1989) examined the effects of Cr(III) and Cr(VI) on growth and reproduction of *Eisenia fetida*. Reproduction after 8 weeks was the measure most sensitive to Cr(III) with a 55% decrease in the number of cocoons and hatchlings at 625 mg/kg Cr(III).

The relative toxicity of Cr(III) and Cr(VI) is not clear from these studies. Cr(VI) ions can pass through cell membranes with much greater ease than Cr(III) ions. However, it is thought that Cr(VI) is

reduced to Cr(III) inside the cell (Molnar et al. 1989); this latter may be the final active form. Without a better understanding of Cr transformations in the soil, transport across earthworm cell membranes, and reactions within the cell, the effects of the two different forms cannot be distinguished (Table 12-5).

Table 12-5. Toxicity of chromium to heterotrophic processes and soil invertebrates (Will and Suter 1995b).

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CrCl_3$	native soil microflora	clay	8	1.5	548	Phosphatase activity	–	2652 D	560	Doelman & Haanstra,. 1989.
$CrCl_3$	native soil microflora	sandy loam	6	3	548	Phosphatase activity	–	2792 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	sandy soil	7	1	548	Phosphatase activity	–	20020 ED_{50}	ED_{50}	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	sandy peat	4	6.5	42	Arylsulfatase activity	–	5928 ED_{50}	50	Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	sandy loam	6	3	42	Phosphatase activity	–	5512 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	silty loam	8	1	42	Urease activity	–	4470 ED_{50}	50	Doelman & Haanstra, 1986.
$CrCl_3$	native soil microflora	silty loam	8	1	548	Phosphatase activity	–	4139 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	sandy soil	7	1	42	Urease activity	–	3970 ED_{50}	50	Doelman & Haanstra, 1986.
$CrCl_3$	native soil microflora	silty loam	8	1	42	Phosphatase activity	–	3728 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	sandy peat	4	6.5	42	Phosphatase activity	–	3208 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	sandy peat	4	6.5	42	Urease activity	–	1360 ED_{50}	50	Doelman & Haanstra, 1986.

Table 12-5 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CrCl_3$	native soil microflora	sandy soil	7	1	42	Phosphatase activity	–	3208 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	130	1300	39	Juma & Tabatabai, 1977.
$CrCl_3$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	130	1300	30	Juma & Tabatabai, 1977.
$CrCl_3$	native soil microflora	clay loam	8	3.2	0.1	Arylsulfatase activity	130	1300	35	Al-Khafaji & Tabatabai, 1979.
$CrCl_3$	native soil microflora	loam	7	2.9	0.1	Arylsulfatase activity	130	1300	43	Al-Khafaji & Tabatabai, 1979.
$CrCl_3$	native soil microflora	loam	7	5.5	0.1	Acid phosphatase activity	–	1300 LCT	27	Juma & Tabatabai, 1977.
$CrCl_3$	native soil microflora	clay loam	8	3.7	0.1	Alkaline & acid phosphatase activity	–	1300 LCT	27,25	Juma & Tabatabai, 1977.
$CrCl_3$	native soil microflora	sandy peat	4	6.5	548	Arylsulfatase activity	–	3203 ED_{50}	50	Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	loam	7	5.3	0.1	Arylsulfatase activity	–	1300 LCT	32	Al-Khafaji & Tabatabai, 1979.
$CrCl_3$	native soil microflora	clay loam	6	2.7	0.1	Arylsulfatase activity	–	1300 LCT	54	Al-Khafaji & Tabatabai, 1979.

Table 12-5 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CrCl_3$	native soil microflora	clay	8	1.5	42	Phosphatase activity	–	1170 ED_{50}	50	Doelman & Haanstra, 1989.
$CrCl_3$	native soil microflora	silty loam	8	1	548	Urease activity	–	1110 ED_{50}	50	Doelman & Haanstra, 1986.
$CuSO_4$	native soil microflora	sandy loam	7	2	21	Nitrification	100	1000	67	Premi & Cornfield, 1969.
CrO	native soil microflora	sandy soil	7	–	42	N mineralization & nitrification	–	1000 LCT	22,24	Bhuiya & Cornfield, 1976.
$CrCl_3$	native soil microflora	sandy soil	7	1	548	Urease activity	–	630 ED	50	Doelman & Haanstra, 1986.
$CrCl_3$	native soil microflora	clay	8	1.5	548	Arylsulfatase activity	–	575 ED	500	Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	clay	8	1.5	42	Urease activity	–	490 ED	50	Doelman & Haanstra, 1986.
$CrCl_3$	native soil microflora	clay	8	1.5	548	Urease activity	–	420 ED	50	Doelman & Haanstra, 1986.
$CrCl_3$	native soil microflora	silty loam	8	1	548	Arylsulfatase activity	–	411 ED	500	Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	sandy loam	6	3	42	Arylsulfatase activity	–	309 ED	500	Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	clay	8	1.5	42	Arylsulfatase activity	–	281 ED	500	Haanstra & Doelman, 1991.

Table 12-5 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CrCl_3$	native soil microflora	loam	6	3	20	N mineralization	–	260 LCT	20	Liang & Tabatabai, 1977.
$CrCl_3$	native soil microflora	sandy soil	7	1	548	Arylsulfatase activity	–	203 ED_{50}		Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	loam	6	–	22	Respiration	–	100 LCT	41	Ross et al. 1981.
$CrCl_3$	native soil microflora	sandy loam	6	–	22	Respiration	–	100 LCT	48	Ross et al., 1981.
$CrSO_4$	native soil microflora	surface soil	–	1.3	1	Dehydrogenase activity	–	30 LCT	54	Rogers & Li, 1985.
$CrCl_3$	native soil microflora	sandy soil	7	1	42	Arylsulfatase activity	–	17 ED_{50}		Haanstra & Doelman, 1991.
$CrCl_3$	native soil microflora	sandy loam	6	3	548	Arylsulfatase activity	–	12 ED_{50}		Haanstra & Doelman, 1991.
$K_2Cr_2O_7$	native soil microflora	loam	6	–	22	Respiration	–	10 LCT	27	Ross et al., 1981.
$K_2Cr_2O_7$	native soil microflora	sandy loam	6	–	22	Respiration	–	10 LCT	33	Ross et al. 1981.
$CrCl_3$	native soil microflora	sandy loam	6	3	548	Urease activity	–	<1 ED_{50}		Doelman & Haanstra, 1986.
$Cr(NO_3)_3$	Eisenia andrei	OECD soil	6	5	21	growth	10	32	30	van Gestel et al., 1992.

Table 12-5 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$KCr(SO_4)_2$	Eisenia fetida	soil & manure	–	–	56	number cocoons and hatchlings	–	625LCT	55	Molnar et al., 1989.
$K_2Cr_2O_7$	Octochaetus pattoni	soil & dung	–	–	60	survival	–	2LCT	75	Abbasi & Soni, 1983.
$K_2Cr_2O_7$	Pheretima posthuma	paddy soil	–	–	61	survival	–	10LCT	100	Soni & Abbasi, 1981.

Note: Chemical concentrations are g of element/kg of growth medium
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls
EXP (D) = exposure in days
Growth Medium:OECD soil (% dry weight): sphagnum peat, 10; kaolin clay, 20; fine sand, 69; $CaCO_3$, 1;
pH 6.0
% OC = % organic carbon
CEC = cation exchange capacity of growth medium (milliequivalents/100 g)

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13. COBALT

13.1 BACKGROUND

Cobalt (Co), a hard, silvery white metal, is strategically important in the production of high-temperature alloys and permanent magnets (Brobst and Pratt 1973). Cobalt salts are utilized in the production of pigments and in paint dryers as catalysts (Lustigman et al. 1995). It is found in the earth's crust at an average concentration of 20 mg/kg. Cobalt is an essential element making up approximately one-half of the contents of vitamin B12, which is necessary in the prevention of pernicious anemia (Oehme 1979). Comparatively little is known about the toxicity of cobalt.

13.2 TOXICITY IN WATER TO AQUATIC LIFE

13.2.1 Acute Toxicity

Acute toxicity values for cobalt ranged from 3.75 mg/L for fathead minnows (Kimball 1978) to 139.32 mg/L for a tubificid worm (Khangarot 1991) (Table 13-1).

13.2.2 Chronic Toxicity

Chronic toxicity values (CVs) ranged from 0.0286 mg/L for fathead minnows to 0.0051 mg/L for *D. magna*. Zebrafish embryos and larvae, *Brachydanio rerio*, were exposed to $CoCl_2$ for 16 days. The no-observed-effect concentrations (NOECs) 0.06 and 3.84 mg/L were found for survival and hatching times, respectively (Dave and Xiu 1991).

13.2.3 Toxicity to Aquatic Plants

Twenty-one-day chronic tests using the unicellular alga, *Chlamydomonas reinhardtii*, exposed lethal toxic effects of cobalt(II) as $Co(NO_3)_2$ at 10–50 mg/L. Growth impairment starting at 10mg/L increased to the highest concentration tested (50 mg/L) and proved to be time and dose dependent. All cells at 50 mg/L were degraded and bleached, indicative of the inhibition of chlorophyll synthesis (Lustigman et al. 1995).

13.2.4 Bioaccumulation

No information was found on the bioaccumulation of cobalt.

13.2.5 Aquatic Mode of Action

Mechanism for cobalt toxicity in aquatic plants may be linked to the competition of cobalt with iron for active sites on the chlorophyll molecule (Lustigman et al. 1995).

13.2.6 Water Quality Criteria

Calculated Tier II values include a secondary acute value (SAV) of 1.5 mg/L and a secondary chronic value SCV) of 0.023 mg/L (Suter and Tsao 1996).

Table 13-1. Cobalt(II) toxicity to aquatic organisms

Conc. (mg/L)[1]	Species	Endpoint	Reference
139.32	Tubificid worm *(Tubifex tubifex)*	96h EC_{50}	AQUIRE 1996
>100.00	Scud *(Gammarus fasciatus)*	96h LC_{50}	Ewell et al. 1986
>100.00	Ramshorn snail *(Heliosoma trivolvis)*	96h LC_{50}	Ewell et al. 1986
>100.00	Oligochaete *(Lumbriculus variegatus)*	96h LC_{50}	Ewell et al. 1986
>100.00	Aquatic sowbug *(Asellus intermedius)*	96h LC_{50}	Ewell et al. 1986
82.70	Carp *(Cyprinus carpio)*	96h LC_{50}	AQUIRE 1996
66.80	Goldfish *(Carassius auratus)*	96h LC_{50}	AQUIRE 1996
32.00	Bdelloid rotifer *(Philodina acuticornis)*	24h EC_{50}	AQUIRE 1996
25.00	Flatworm *(Dugesia tigrina)*	96h LC_{50}	Ewell et al. 1986
22.00	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	Ewell et al. 1986
17.59	Frog *(Rana hexadactyla)*	96h LC_{50}	AQUIRE 1996
5.99	Water flea *(Daphnia magna)*	48h LC_{50}	Kimball 1978
5.10	Water flea *(Daphnia magna)*	CV	Kimball 1978
3.75	Fathead minnow *(Pimephales promelas)*	96h LC_{50}	Kimball 1978
1.48		SAV	Suter and Tsao 1996
0.29	Fathead minnow *(Pimephales promelas)*	CV	Kimball 1978
0.23		SCV	Suter and Tsao 1996

[1]All concentrations are given as Co(II), not the compound.

13.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on the toxicity of cobalt to benthic invertebrates.

13.4 TOXICITY TO PLANTS

13.4.1 Toxicity to Plants in Soil

Linzon (1978) reported unspecified toxic effects on plants grown in a surface soil with the addition of 20 mg/kg Co.

13.4.2 Toxicity to Plants in Solution

Wallace et al. (1977) evaluated the effect of cobalt as $CoSO_4$ on bush beans grown for 21 days in nutrient solution (Table 13-2). Leaf dry weight was reduced 22% by the addition of 0.06 mg/kg Co, the lowest concentration tested. Root and stem weight were not affected at this concentration. Chrysanthemum seedling root weight was reduced 55% after 21-day growth in nutrient solution containing the same concentration of cobalt as $CoSO_4$ (Patel et al. 1976). Leaf weight and stem weight were not affected at this concentration.

Will and Suter (1995) reported NOEC and LOEC values for the toxicity of cobalt to plants in solution. The NOEC values range from 1 to 50 mg/kg, and the LOEC values range from 0.06 to 100 mg/kg.

13.4.3 Phytotoxic Mode of Action

Cobalt is not known to be essential to plants except legumes in symbiosis with N_2-fixing microorganisms. When translocated from roots, the element travels in the xylem as the Co(II) ion (Tiffin 1967). Toxicity symptoms due to excess cobalt are typical of Fe deficiency-induced chlorosis and necrosis and root tip damage (Wallace et al. 1977). Apparent inhibition of mitosis and chromosome damage has been observed (Aller et al. 1990).

13.5 TOXICITY TO WILDLIFE

13.5.1 Toxicity to Mammals

Christensen and Luginbyh (1974, as cited in Speijers et al. 1982) reported rat oral LD_{50} concentrations of 1700 and 180 mg/L for Co(II) as cobalt oxide and cobalt chloride, respectively. This large divergence in LD_{50} values for the same ion suggest that Co(II) toxicity is dependent on the other complexed ions as well as its concentration (Table 13-3). Symptoms of cobalt toxicity (10 day) in rats at high concentrations include sedation, diarrhea, and decreased body temperature (2.5–7.5 °C). The decrease in body temperature is due to the inhibition of oxidative processes by cobalt and proved to be time and dosage dependent (Speijers et al. 1982). Looking at cobalt's chronic reproductive effects, Pedigo et al. (1988) found decreased fertility, sperm concentration, and testicular weight in mice fed 72.0 mg/kg/d for 13 weeks. In a similar study, Mollenhauer et al. (1985, as cited in Beyersman and Hartwig 1992) reported mouse testicular damage associated with $CoCl_2$ at 265 mg/kg body weight. Cobalt has been shown to inhibit DNA repair in mammalian cells leading to magnification of genotoxicity of primary DNA damaging agents (Beyersmann and Hartwig 1992).

Table 13-2. Phytotoxicity data for the toxicity of cobalt derived from experiments in solution (Will and Suter 1995)

Chemical form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	Reference
$CoSO_4$	bush beans	-	0.06 LCT	leaf weight	Wallace et al. 1977
$CoSO_4$	chrysanthemum	-	0.06 LCT	root weight	Patel et al. 1976
$CoSO_4$	honeysuckle	1	5	radicle elongation	Patterson & Olson 1983
$CoSO_4$	paper birch	1	5	radicle elongation	Patterson & Olson 1983
$CoCl_2$	broad bean	8	10	root elongation	Misra et al. 1994
$CoSO_4$	black spruce	5	10	radicle elongation	Patterson & Olson 1983
$CoSO_4$	jack pine	10	20	radicle elongation	Patterson & Olson 1983
$CoSO_4$	red pine	10	20	radicle elongation	Patterson & Olson 1983
$CoSO_4$	white spruce	20	50	radicle elongation	Patterson & Olson 1983
$CoSO_4$	white pine	50	100	radicle elongation	Patterson & Olson 1983

13.5.2 Toxicity to Birds

A low incidence of lesions in skeletal and smooth muscle was observed in ducklings fed 200 and 500 mg/kg for 2 weeks (van Vleet et al. 1981, as cited in Diaz et al. 1994). Diaz et al. (1994) reported lesions present in chickens exposed to 250 and 500 mg/kg for 14 days in the proventriculus, gizzard, duodenum, pancreas, liver, heart, and skeletal muscle. Mortality increased linearly in response to increasing cobalt concentrations. Chicks in the 250 and 500 mg/kg groups gained 24.4 and 79.6% less weight than the control groups and exhibited decreased feed efficiency. This decrease may be linked to observed glandular proventricular necrosis and pancreatic fibrosis due to their role in the generation of digestive enzymes. It was also shown that a low iron content in the food supply will increase cobalt uptake. Embryonic development is adversely affected at any cobalt concentration and it is suspected that cobalt increases chicks' susceptibility to infectious agents (Diaz et al. 1994). Cobalt's toxic effects are generally due to complexation of the ion with amino acids and sulfhydryl groups (Elinder and Friberg 1986, as cited in Diaz et al. 1994).

Cobalt's toxic effects on birds, including weight loss and decreased feeding capacity, may be partially if not completely ameliorated by the application of methionine or cysteine to the diet. Cysteine at 1–18% of the diet alleviated growth depression caused by 250 mg/kg. The mechanism of cysteine

Table 13-3. Acute oral toxicity of cobalt compounds in Wistar rats (Speijers et al. 1982)

Test	LD_{50} for Co(II) ion (mg/kg BW)
$CoCl_2$	190.0
$Co_3(PO_4)_2$	187.0
$Co(CH_3CO_2)_2$	168.0
$CoSO_4$	161.0
CoO	159.0
$Co(NO_3)_2$	140.0
$CoBr_2$	109.0
CoF_2	91.0

may be their complexation of 3 molecules to one cobalt molecule, rendering it unavailable for uptake (Southern and Baker 1981).

13.6 TOXICITY TO HETEROTROPHIC PROCESS

Lighthart et al. (1977) evaluated the effects of cobalt at a single concentration on respiration of native soil microflora in soil/litter microcosms. Cobalt at 1362 mg/kg reduced respiration 23%.

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14. COPPER

14.1 BACKGROUND

Copper (Cu) occurs in natural waters primarily as the divalent cupric ion in free and complexed forms (EPA 1985). Copper is a minor nutrient for both plants and animals at low concentrations, but is toxic to aquatic life at concentrations only slightly higher. Concentrations of 0.001 to 0.010 mg/L are usually reported for unpolluted surface waters in the United States. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH 1986). Copper is soluble in nitric acid and hot sulfuric acid, slightly soluble in hydrochloric acid and ammonia, and insoluble in water (Stokinger 1981). The largest anthropogenic releases of copper to the environment result from mining operations, agriculture, solid waste, and sludge from sewage treatment plants.

14.2 TOXICITY IN WATER TO AQUATIC LIFE

14.2.1 Acute Toxicity

Reported copper LC_{50}s range from 0.0065 to 9.3 mg/L for invertebrates in 56 tests and from 0.010 to 10.2 mg/L in fishes in 177 tests (EPA 1985). Toxicity of copper appears to be a function of calcium hardness and associated carbonate alkalinity. Therefore, the concentrations in Table 14-1are corrected for hardness of 100 and 200 mg/L.

14.2.2 Chronic Toxicity

Reported copper chronic values (CVs) range from 0.0095 to 0.029 mg/L for invertebrates in seven tests and from <0.0074 to 0.060 mg/L in fishes in 15 tests (EPA 1985). The CV concentrations in Table 14-1 are corrected for 100 and 200 mg/L using the formulas provided in EPA (1985).

14.2.3 Toxicity to Aquatic Plants

Copper salts are commonly used as algicidal and algistatic agents, particularly to control blue-green algae. Reported copper toxicity values for aquatic plants range from 0.001 to 10.45 mg/L for 32 tests (EPA 1985).

14.2.4 Bioaccumulation

Copper is not known to be appreciably bioaccumulated by fish, but some algae and bivalve molluscs do bioconcentrate or bioaccumulate copper by factors of over 1000 (EPA 1985).

14.2.5 Aquatic Mode of Action

The toxicity of copper to aquatic life is related primarily to activity of the cupric (Cu^{2+}) ion, and possibly to the hydroxy complexes. The cupric ion is highly reactive, forms moderate to strong complexes and precipitates with inorganic and organic constituents (e.g., carbonate, phosphate, amino acids), and is readily sorbed onto surfaces of suspended solids. The proportion of free cupric ion may be less than 1 percent in eutrophic waters where complexation predominates. These complexes appear to be much less toxic than free cupric ions, thus reducing toxicity. Hence, even dissolved copper

Table 14-1. Hardness-normalized toxicity of copper to aquatic organisms calculated from values in EPA (1985).

CONCENTRATION (MG/L)[1]		EFFECT
100 MG/L HARDNESS	200 MG/L HARDNESS	
19.6757	37.8060	Stonefly (*Acroneuria lycorias*) LC_{50}
11.2597	21.6351	White perch (*Morone americana*) LC_{50}
8.2719	15.8940	American eel (*Anguilla rostrata*) LC_{50}
3.8237	7.3471	Crayfish (*Procambarus clarki*) LC_{50}
3.6066	6.9299	Snail (*Campeloma decisum*) LC_{50}
2.6843	5.1577	Crayfish (*Orconectes limosus*) LC_{50}
1.7293	3.3228	Snail (*Amnicola* sp.) LC_{50}
1.6653	3.1999	Blacknose dace (*Rhinichthys atratulus*) LC_{50}
1.6653	3.1999	Rainbow darter (*Etheostoma caeruleum*) LC_{50}
1.6134	3.1002	Creek chub (*Semotilus atromaculatus*) LC_{50}
1.7434	2.9887	Bluegill (*Lepomis macrochirus*) LC_{50}
1.5191	2.9189	Banded killifish (*Fundulus diaphanus*) LC_{50}
1.3414	2.5774	Brown bullhead (*Ictalurus nebulosus*) LC_{50}
1.3149	2.5264	Mozambique tilapia (*Tilapia mossambica*) LC_{50}
1.2315	2.3662	Pumpkinseed (*Lepomis gibbosus*) LC_{50}
0.6375	1.2250	Striped shiner (*Notropis chrysocephalus*) LC_{50}
0.4663	0.8960	Worm (*Lumbriculus variegatus*) LC_{50}
0.4492	0.8632	Sockeye salmon (*Oncorhynchus nerka*) LC_{50}
0.4423	0.8499	Orangethroat darter (*Etheostoma spectabile*) LC_{50}
0.3207	0.8254	Guppy (*Poecilia reticulata*) LC_{50}
0.3789	0.7281	Midge (*Chironomus tentans*) LC_{50}
0.3778	0.7258	Atlantic salmon (*Salmo salar*) LC_{50}
0.3768	0.7240	Mosquitofish (*Gambusia affinis*) LC_{50}
0.3217	0.6180	Northern squawfish (*Ptychocheilus oregonensis*) LC_{50}
0.3193	0.6136	Snail (*Goniobasis livescens*) LC_{50}
0.2644	0.6053	Fathead minnow (*Pimephales promelas*) LC_{50}

Table 14-1 (continued)

CONCENTRATION (MG/L)[1]		EFFECT
100 MG/L HARDNESS	200 MG/L HARDNESS	
0.3019	0.5800	Goldfish (*Carassius auratus*) LC_{50}
0.3013	0.5789	Common carp (*Cyprinus carpio*) LC_{50}
0.2594	0.4984	Bryozoan (*Pectinatella magnifica*) LC_{50}
0.2556	0.4910	Chiselmouth (*Acrocheilus alutaceus*) LC_{50}
0.2121	0.4076	Brook trout (*Salvelinus fontinalis*) LC_{50}
0.1729	0.3323	Worm (*Nais* sp.) LC_{50}
0.1509	0.2900	Central stoneroller (*Campostoma anomalum*) LC_{50}
0.1387	0.2664	Bluntnose minnow (*Pimephales notatus*) LC_{50}
0.1350	0.2594	Coho salmon (*Oncorhynchus kisutch*) LC_{50}
0.1270	0.2441	Northern pike (*Esox lucius*) CV
0.1217	0.2234	Cutthroat trout (*Oncorhynchus clarki*) LC_{50}
0.1080	0.2075	Snail (*Gyraulus circumstriatus*) LC_{50}
0.1020	0.1960	Tubifed worm (*Limnodrilius hoffmeisteri*) LC_{50}
0.0828	0.1590	Snail (*Physa integra*) LC_{50}
0.0787	0.1457	Rainbow trout (*Oncorhynchus mykiss)* LC_{50}
0.0712	0.1368	Bryozoan (*Lophodella carteri*) LC_{50}
0.0712	0.1368	Bryozoan (*Plumatella emarginata*) LC_{50}
0.0690	0.1326	Snail (*Physa heterostropha*) LC_{50}
0.0656	0.1260	Brook trout (*Salvelinus fontinalis*) CV
0.0649	0.1247	Brown trout (*Salmo trutta*) CV
0.0642	0.1234	Lake trout (*Salvelinus namaycush*) CV
0.0615	0.1182	Bluegill (*Lepomis macrochirus*) CV
0.0576	0.1108	Midge (*Chironomus* sp.) LC_{50}
0.0553	0.1063	Scud (*Gammarus pulex*) LC_{50}
0.0645	0.0983	Chinook salmon (*Oncorhynchus tshawytscha*) LC_{50}
0.0488	0.0939	Water flea (*Ceriodaphnia pulex*) EC_{50}

Table 14-1 (continued)

CONCENTRATION (MG/L)[1]		EFFECT
100 MG/L HARDNESS	200 MG/L HARDNESS	
0.0436	0.0900	Water flea (*Ceriodaphnia magna*) EC_{50}
0.0439	0.0844	White sucker (*Catostomus commersoni*) CV
0.0434	0.0835	Fathead minnow (*Pimephales promelas*) CV
0.0424	0.0816	Scud (*Gammarus pseudolimnaeus*) LC_{50}
0.0400	0.0769	Rainbow trout (*Oncorhynchus mykiss)* CV
0.0393	0.0756	Fathead minnow (*Pimephales promelas*) CV
0.0370	0.0710	Caddisfly (*Clistornia magnifica*) CV
0.0361	0.0693	Water flea (*Ceriodaphnia reticulata*) EC_{50}
0.0296	0.0568	Chinook salmon (*Oncorhynchus tshawytscha*) CV
0.0273	0.0524	Brook trout (*Salvelinus fontinalis*) CV
0.0236	0.0454	Snail (*Campeloma decisum*) CV
0.0236	0.0454	Snail (*Physa integra*) CV
0.0039	0.0086	NAWQC Acute Value
0.0144	0.0277	Fathead minnow (*Pimephales promelas*) CV
0.0296	0.0249	Water flea (*Daphnia magna*) CV
0.0129	0.0247	Scud (*Gammarus pseudolimnaeus*) CV
0.0150	0.0243	Water flea (*Ceriodaphnia pulicaria*) EC_{50}
0.0183	0.0219	Fathead minnow (*Pimephales promelas*) CV
0.0011	0.002	NAWQC Chronic Value
0.0098	0.0188	Brook trout (*Salvelinus fontinalis*) CV
0.0115	0.0097	Water flea (*Daphnia magna*) CV
0.0115	0.0097	Water flea (*Daphnia magna*) CV
0.0047	0.0091	Bluntnose minnow (*Pimephales notatus*) CV

[1] Concentrations given as Cu, not the compound.

measurements may overestimate copper exposure relative to laboratory test waters due to dissolved organic and inorganic ligands (EPA 1985; Benson et al. 1994).

14.2.6 Water Quality Criteria

The NAWQC for copper are functions of water hardness. The equations are $e^{(0.8545[\ln(\text{hardness})]-1.465)}$ for the chronic value and $e^{(0.9422[\ln(\text{hardness})]-1.464)}$ for the acute value (EPA 1985; Suter and Tsao 1996).

14.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Most data on copper toxicity are available from tests of marine and estuarine sediments (Table 14-2). The toxic concentrations range from 1 mg/kg, which was associated with reduced diversity of the benthic invertebrate community, to 2,820 mg/kg, which was associated with 79% mortality of the amphipod *Rhepoxynius abronius*. There is relatively little consistency in the effects data presented by NOAA (Long and Morgan 1991). They cited the results of ten freshwater sediment studies considered to be of sufficient quality for calculating sediment benchmarks. Five of those studies reported significant mortality of *Daphnia magna* in overlying water, but the sediment concentrations associated with that effect ranged from 68 mg/kg to 1,800 mg/kg. Two of these studies also found that significant mortality of mayflies (*Hexagenia sp.)* was associated with 68 mg/kg and 1,800 mg/kg (studies cited in Long and Morgan 1991). Significant mortality of the amphipod *Hyalella azteca* was associated with 19.5 mg/kg and 156.0 mg/kg in two studies cited in Long and Morgan (1991). Taxa richness was significantly depressed in two studies, but the associated concentrations ranged from 45 mg/kg to 589 mg/kg. The available data suggest that sediment characteristics other than copper concentrations have a considerable influence on the observed responses.

14.4 TOXICITY TO PLANTS

14.4.1 Toxicity to Plants in Soil

Miles and Parker (1979) found approximately 68% reductions in root and shoot weights of little bluestem grown from seed for 12 weeks in a sandy soil (pH 7.8, % organic matter 2.5), with 100 mg/kg Cu as $CuSO_4$ added (only concentration tested). Growth was reduced in a second sandy soil (pH 4.8, % organic matter 1.9) by 86% with the addition of 100 mg/kg Cu (only concentration tested). Wallace et al. (1977) evaluated the effects of Cu, added as $CuSO_4$ to a loam soil, on leaf and stem weights of bush beans grown from seed for 17 days. Leaf weight was reduced 26% by 200 mg/kg Cu, while 100 mg/kg had no effect.

14.4.2 Toxicity to Plants in Solution

The effect of Cu on stem diameter growth and plant weight of red pine, maple, dogwood, and honeysuckle was examined by Heale and Ormrod (1982). All seedlings (90-d old) grown for 110 d in nutrient solution containing 4 mg/L Cu from $CuSO_4$ (lowest concentration tested) were affected. Reductions in rate of stem diameter increase and in plant weight were 41 and 50%, 79 and 67%, and 97 and 74% for maple, dogwood, and honeysuckle, respectively. Red pine experienced a 28% decrease in plant weight at 4 mg/L Cu, but the stem diameter rate of increase was unaffected up to 20 mg/L Cu (highest concentration tested).

Table 14-2. Copper toxicity to benthic invertebrates in marine and estuarine sediment (MacDonald et al. 1994).

Conc. (mg/kg)	Endpoint	Species
1.00	moderate diversity	benthic species
3.78	48 h LC_{25}	*Arbacia punctulata* (sea urchin)
8.08	10 d LC_{55}	*Hyalella azteca* (ampipod)
10.70	low abundance	oligochaeta
11.30	low density	annelida
12.60	10 d LC_{46}	*Lepidactylus dytiscus* (amphipod)
12.70	48 h EC_{85} (development)	*Arbacia punctulata* (sea urchin)
13.60	ET_{50} (burrowing time)	*Protothaca staminea* (littleneck clam)
14.00	low density	echinodermata
14.20	10 d EC_{90} (reburial)	*Lepidactylus dytiscus* (amphipod)
14.20	low density	arthropoda
14.20	20 d EC<90 (emergence)	*Lepidactylus dytiscus* (amphipod)
14.20	20 d EC<90 (emergence)	*Hyalella azteca* (ampipod)
14.20	10 d LC_{29}	*Lepidactylus dytiscus* (amphipod)
14.50	low richness	benthic species
16.80	20 d EC<90 (emergence)	*Hyalella azteca* (ampipod)
16.80	20 d EC<80 (emergence)	*Lepidactylus dytiscus* (amphipod)
16.80	10 d EC_{83} (reburial)	*Lepidactylus dytiscus* (amphipod)
23.40	ET_{50} - 63-69h (reburial)	*Protothaca staminea* (littleneck clam)
30.00	10 d LC_{16}	*Ampelisca abdita* (amphipod)
32.40	ET_{50} - 25-300h (burial)	*Protothaca staminea* (littleneck clam)
38.20	48 d LC_{25}	*Protothaca staminea* (littleneck clam)

Table 14-2 (continued)

Conc. (mg/kg)	Endpoint	Species
38.20	48 d LC_{15}	*Protothaca staminea* (littleneck clam)
43.00	20 d LC_{52}	*Neanthes* sp. (polychaete)
43.80	1 h EC_{98} (fertilization)	*Arbacia punctulata* (sea urchin)
67.00	48 h Significant increase in burrowing time	*Macoma balthica* (bivalve)
68.20	48 h EC_{56} (abnormal)	bivalve
71.00	low abundance	arthropods
72.00	EC_{50}	*Photobacterium phosphoreum* (Microtox)
73.20	low richness	benthic species
76.00	48 h EC_{59} (abnormal)	bivalve
84.60	10 d LC_{67}	*Rhepoxynius abronius* (amphipod)
87.80	48 h EC_{92} (abnormal)	bivalve
96.70	low abundance	echinoderm
98.70	48 h LC_{92}	mussel
103.00	10 d LC_{32}	*Ampelisca abdita* (amphipod)
106.00	48 h EC_{23} (abnormal)	oyster
109.00	low abundance	echinoderms
114.00	48 h EC_{67} (abnormal)	mussel
117.00	10 d LC_{31}	*Ampelisca abdita* (amphipod)
118.00	10 d LC_{26}	*Rhepoxynius abronius* (amphipod)
121.00	low abundance	phoxocephalids
121.00	low abundance	amphipods
124.00	10 d LC_{83}	*Leptocheirus plumulosus* (amphipod)

Table 14-2 (continued)

Conc. (mg/kg)	Endpoint	Species
125.00	10 d EC_{31} (emergence)	*Rhepoxynius abronius* (amphipod)
125.00	10d EC_{77} (emergence)	*Corophium volutator* (amphipod)
130.00	10 d EC_{63} (avoidance)	amphipod
130.00	10 d LC_{95}	amphipod
135.00	Sediments devoid of feral clams	*Macoma balthica* (bivalve)
147.00	96 h LC>50	*Palaemonetes pugio* (shrimp)
150.00	24h - avoidance	*Macoma balthica* (bivalve)
157.00	20 d LC_{95}	*Leptocheirus plumulosus* (amphipod)
164.00	EC_{50}	*Photobacterium phosphoreum* (Microtox)
181.00	10 d LC_{52}	*Grandidierella japonica* (amphipod)
187.00	low density	echinoderm
192.00	10 d LC_{62}	*Rhepoxynius abronius* (amphipod)
196.00	48 h LC_{52}	*Mulinia lateralis* (bivalve)
202.00	0.25 h EC_{50} (extract)	*Photobacterium phosphoreum* (Microtox)
220.00	20 d EC_{60} (abnormal development)	*Dendraster excentricus* (echinoderm)
258.00	low richness	benthic species
259.00	10 d LC_{81}	*Rhepoxynius abronius* (amphipod)
270.00	low density	phoxocephalid
270.00	low density	amphipod
337.00	20 d LC_{37}	*Neanthes arenaceodentata* (polychaete)
361.00	10 d LC_{55}	*Hyalella azteca* (ampipod)
412.00	low density	polychaeta

Table 14-2 (continued)

Conc. (mg/kg)	Endpoint	Species
455.00	low density	*Foraminifera* (sponge)
455.00	low density	*Ophiuroidea* (brittle star)
455.00	low density	echinoderm
581.00	low density	phoxocephalid
581.00	low richness	macro benthos
581.00	low density	crustacea
581.00	low density	amphipod
592.00	10 d LC_{21}	*Rhepoxynius abronius* (amphipod)
592.00	low density	mollusca
612.00	14 d LC_{100}	*Nereis virens* (sandworm)
918.00	48 h EC_{45} (abnormal)	oyster
2820.00	10 d LC_{79}	*Rhepoxynius abronius* (amphipod)

Wong and Bradshaw (1982) measured a 71% reduction in length of longest root in rye grass grown for 14 d in nutrient solution (pH 7) to which 0.031 mg/L Cu as $CuSO_4$ was added (lowest concentration tested). After 4 d, root length of rice seedlings was reduced 64% by 64 mg/L Cu (6.4 mg/L had no effect) added as $CuSO_4$ to nutrient solution (Gupta and Mukherji 1977).

Maize seedlings germinated and grown for 10 d in solution containing $CuSO_4$ had a 40% reduction in total fresh weight in the 0.06 mg/L Cu treatment (lowest concentration tested) (Stiborova et al. 1986). This same concentration caused a 45% reduction in root weight of chrysanthemums grown for 21d in nutrient solution with $CuSO_4$ added (Patel et al. 1976).

Will and Suter (1995a) reported NOEC and LOEC values for the toxicity of copper to plants in solution. The NOEC values range from 0.5 to 50 mg/L and the LOEC values range from 0.031 to 100 mg/L (Table 14-3).

14.4.3 Phytotoxic Mode of Action

Copper is a micronutrient essential for plant nutrition. It is required as a co-factor for many enzymes and is an essential part of a copper protein involved in photosynthesis. Copper occurs as part of enzymes and enzyme systems. Root absorption appears to be passive, perhaps in organo-copper

Table 14-3. Phytotoxicity data for the toxicity of copper derived from experiments conducted in solution (Will and Suter 1995a).

Chemical form	PLANT SPECIES	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	REFERENCE
$CuSO_4$	ryegrass	-	0.031 LCT	length longest root	Wong and Bradshaw, 1982.
$CuSO_4$	corn	-	0.064 LCT	fresh plant weight	Stiborova et al., 1986.
$CuSO_4$	chrysanthemum	-	0.064	root weight	Patel et al., 1976.
$CuCl_2$	rice	-	0.22 EC_{50}	radicle weight	Wang, 1994
$CuSO_4$	paper birch	0.5	1	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	honeysuckle	-	4 LCT	stem dia increase; plant weight	Heale and Ormrod, 1982.
$CuSO_4$	dogwood	-	4 LCT	stem dia increase; plant weight	Heale and Ormrod, 1982.
$CuSO_4$	red pine	-	4 LCT	plant weight	Heale and Ormrod, 1982.
$CuSO_4$	black spruce	1	5	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	red pine	1	5	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	jack pine	5	10	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	maple	2	10	plant weight	Heale and Ormrod, 1982.
$CuSO_4$	white spruce	10	20	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	honeysuckle	20	50	radicle elongation	Patterson & Olson, 1983
$CuSO_4$	rice	6.4	64	root length	Gupta and Mukherji, 1977.
$CuSO_4$	white pine	50	100	radicle elongation	Patterson & Olson, 1983

complexes (Jarvis and Whitehead 1983), and active through a specific carrier (Fernandes and Henriques 1991). Copper may be deficient in low-copper soils because the metal is adsorbed to cells in the root system. The form in which it is taken into the root affects its binding there (Wallace and Romney 1977). Copper can be transported in the xylem and phloem of plants complexed with amino acids.

The most common toxicity symptoms include reduced growth, poorly developed root system, and leaf chlorosis (Wong and Bradshaw 1982). The basic deleterious effect of Cu is related to the root system, where it interferes with enzyme functioning (Mukherji and Das Gupta 1972). It also strongly interferes with photosynthesis and fatty acid synthesis (Smith et al. 1985).

14.5 TOXICITY TO WILDLIFE

14.5.1 Toxicity to Mammals

Copper is a component of a number of metalloenzymes such as catalase, peroxidases, and cytochrome oxidase and is essential for the utilization of iron (Goyer 1991; Stokinger 1981). Although most copper salts occur in two valence states, as cuprous (Cu^{+}) or cupric (Cu^{2+}) ions, the biological availability and toxicity of copper is most likely associated with the divalent state (ATSDR 1990).

The metabolism of copper involves mainly its transfer to and from various organic ligands, most notably sulfhydryl and imidazole groups on amino acids and proteins (ATSDR 1990). The liver is one of the primary organs involved in the storage and metabolism of copper. Absorption of ingested copper occurs primarily in the upper gastrointestinal tract (EPA 1987). Soluble copper compounds (oxides, hydroxides, citrates) are readily absorbed but water-insoluble compounds (sulfides) are poorly absorbed (Venugopal and Luckey 1978). Zinc, molybdenum, and other metals may decrease dietary copper absorption (USAF 1990).

In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of ≥100 mg/kg/day, and hematological effects at doses of 40 mg/kg/day (EPA 1986; Haywood 1985; Rana and Kumar 1978; Gopinath et al. 1974; Kline et al. 1971). Oral or intravenous administration of copper sulfate can increase fetal mortality and developmental abnormalities in experimental animals (Lecyk 1980; Ferm and Hanlon 1974). Rat oral LD_{50} values for various copper compounds are 140 mg/kg for copper chloride ($CuCl_2$); 470 mg/kg for copper oxide (Cu_2O); 940 mg/kg for copper nitrate ($Cu(NO_3)_2 \cdot 3H_2O$); and 960 mg/kg for copper sulfate ($CuSO_4 \cdot 5\ H_2O$) (Stokinger 1981). Deaths in animals given lethal doses of copper have been attributed to extensive hepatic centrilobular necrosis (USAF 1990).

In a 90-day subchronic study with copper cyanide (CuCN), high mortality, attributed to hemolytic anemia, was seen in both male and female rats receiving 50 mg/kg/day by gavage, but not in those receiving ≤5 mg/kg/day (EPA 1986). In general, male rats appeared to be more sensitive to the effects of CuCN than female rats. Rats receiving 500 mg/kg copper in their diet (about 5 mg/day) appeared normal, while rats receiving 1000 mg/kg exhibited depressed growth, those at 2000 mg/kg hardly grew at all, and those on a 4000 mg/kg diet lost weight rapidly and died (Boyden et al. 1938). Salt licks containing 5–9% copper sulfate caused anorexia, hemolytic anemia, icterus, and hemoglobinuria, followed by death within 2 days in sheep using the licks (Gopinath et al. 1974). The estimated ingested dose was 40–49 g over a 25- to 86-day period. Lecyk (1980) observed reduced litter size, decreased fetal weights, and skeletal abnormalities in the offspring of mice fed diets supplemented with 3000 or 4000 mg/kg copper sulfate (155 or 207 mg copper/kg/day, respectively) for one month prior to gestation and on days 0–19 of gestation.

Aulerich et al. (1982) reported an increased mortality rate (≥58%) in the offspring of mink fed a diet supplemented with >50 mg copper/kg as copper sulfate for 50 weeks; no adverse effects were observed at 25 mg/kg supplemental Cu. Based on the results of this study, Sampl et al. (1996)

estimated the NOAEL and LOAEL for reproductive effects of copper on mink to be 11.7 and 15.14 mg/kg/d, respectively. Lifetime exposure to 42.4 mg copper/kg/day (as copper gluconate) in drinking water caused a 12.8% decrease in the maximal lifespan in mice (Massie and Aiello 1984).

14.5.2 Toxicity to Birds

Domestic chicks on diets ≥324 mg/kg copper grew slowly; mortality increased with dietary copper concentrations of 1270 mg/kg (Mayo et al. 1956). Arthur et al. (1958) observed no ill effects in chicks fed ≤500 mg/kg copper in diet up to 8 weeks of age. Dietary copper levels from 588–1176 mg/kg for 10 weeks exerted a toxic effect on chick growth; the minimum toxic level of copper appeared to be about 500 mg/kg (Mehring et al. 1960). Turkey poults tolerated 676 mg/kg copper in starter diets for 21 days with no deleterious effects, but copper was definitely toxic at levels >1620 mg/kg (Vohra and Kratzer 1968). Chickens given a daily dose of >70 mg/kg of $CuCO_3$ died while those receiving <60 mg/kg exhibited slight symptoms of copper poisoning but survived. No symptoms of copper poisoning were observed in domestic mallards ingesting ≤29 mg/kg/day of $CuCO_3$, but daily intakes ≥55 mg/kg/day were toxic (Pullar 1940).

14.6 TOXICITY TO HETEROTROPHIC PROCESSES AND SOIL AND LITTER INVERTEBRATES

Liang and Tabatabai (1977) investigated the effects of various metals on N mineralization by native soil microflora in four soils. Copper at 320 mg/kg severely reduced N mineralization in one soil. The effects of Cu on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). A concentration of 30 mg/kg Cu (the lowest concentration tested) reduced dehydrogenase activity by 28%. Bollag and Barabasz (1979) evaluated the effects of Cu on denitrification by three species of soil-dwelling *Pseudomonas* species of bacteria in autoclaved soil and by native soil microflora. In the autoclaved soil LOECs ranged from 10 (lowest concentration tested) to 250 mg/kg (highest concentration tested). Denitrification by the native soil population was reduced 44% by 250 mg/kg Cu. The effects of adding Cu, as $CuSO_4$, to a sandy loam adjusted to three pH levels on N mineralization during a 21-day incubation was assessed by Quraishi and Cornfield (1973). Mineralization was decreased by 1000 mg/kg Cu at all three pH levels (5.1, 5.9, and 7.3), with the inhibitory effect increasing with decreasing pH from 39% to 100%.

Bhuiya and Cornfield (1972) assessed the effects of several metals on C mineralization by native microflora in a sandy soil, with or without added organic matter. After 12 weeks, soil respiration was reduced in the Cu-treated soil with amendment but not in the unamended soil. The effects of Cu (as $CuCl_2$) on carbon and nitrogen mineralization and nitrogen transformations in alfalfa-amended sieved soil were determined by Suter and Sharples (1984). Respiration was significantly reduced after exposure to 100 ppm for 3d. Ammonia concentration was decreased significantly by 58% on days 4 and 11 at 10 mg/kg, but there was no increase of effects with concentration on those dates, and ammonia concentrations were unchanged or increased at those concentrations on later dates. Ammonia concentrations were significantly increased at 500 and 1000 mg/kg from day 18 to 53, and after day 18, there were regular patterns of increasing ammonia at increasing Cu levels. Nitrate levels were reduced by 21% on day 4 at a concentration of 10 mg/kg, but not on later dates. On days 11, 25, 39, and 53, nitrate concentrations were significantly reduced at 500 mg/kg Cu. Table 14-4 summarizes the toxicity of Cu to heterotrophic processes.

Neuhauser et al. (1984) evaluated the effects of soluble forms of copper on growth and reproduction *E. fetida*. After 6 weeks, both growth (weight) and cocoon production were decreased (75 and 85%) by 2000 mg/kg Cu, while 1000 mg/kg had no effect. Neuhauser et al. (1985) used the OECD artificial soil to estimate LC_{50} of Cu (added as Cu nitrate) for adult *E. fetida*. After 14 days, the LC_{50} was 643 mg/kg Cu. Spurgeon et al. (1994) kept adult *E. fetida* in contaminated OECD artificial soil for 8 weeks to test the effects of Cu (as $Cu(NO_3)_2$) on survival and growth of the earthworms. After 56 days, the calculated LC_{50} was 555 mg/kg, and the EC_{50} for cocoon production was 53.3 mg/kg. Bengtsson et al. (1986) investigated the effects of copper on *Dendrobaena rubida* at different acidities. After 4 months at pH 4.5, the number of cocoons produced per worm, hatchlings/cocoon, and total number of hatchlings were reduced 70, 64, and 74%, respectively, by 100 mg/kg Cu, the lowest concentration tested. The percent hatched cocoons was not affected. At pH 5.5, the number of cocoons produced per worm, hatchlings/cocoon, and percent hatched cocoons were reduced 96, 100, and 100%, respectively, by 500 mg/kg Cu, while 100 mg/kg had no effect. The total number of hatchlings was not affected. At pH 6.5, the number of cocoons produced per worm, hatchlings/cocoon, and percent hatched cocoons were reduced 90, 100, and 100%, respectively, by 500 mg/kg Cu, while 100 mg/kg had no effect. The total number of hatchlings was not affected.

In experiments by van Gestel et al. (1991b) using Cu ($CuCl_2$) mixed homogeneously with the OECD substrate, growth of *E. fetida* was reduced 32% by 100 mg/kg (32 mg/kg had no effect). The EC_{50} for clitella development (sexual development) was >100 mg/kg Cu. In a study examining the effects of soil factors on Cu toxicity and uptake, Ma (1982) used a sandy loam soil spiked with $CuCl_2$ to determine the effects of Cu on survival of adult *Lumbricus rubellus*. After 12 weeks, 1000 mg/kg Cu caused an 82% decrease in survival while 150 mg/kg had no effect. The effect of soil organic carbon on toxicity of Cu ($CuSO_4$) to the earthworm *Octolasium cyaneum* was evaluated by Streit and Jaggy (1983). They determined the 14-day LC_{50} in a Brown soil, a Rendzina soil, and a peat soil containing 3.2, 14, and 43% organic carbon, respectively. The LC_{50} concentrations were 180, 850, and 2500 mg/kg, respectively. The relative sensitivities of several lumbricid earthworms to Cu ($CuCl_2$) added to a sandy soil was investigated by Ma (1988). EC_{50}s for cocoon production of *L. rubellus*, *Aporrectodea caliginosa*, and *Allolobophora chlorotica* were 122, 68, and 51 mg/kg Cu. The work of Streit and Jaggy (1983) and others shows that the organic carbon content of the soil is a strong determinant of the bioavailability and toxicity of copper. From the studies cited, it appears that low pH increases Cu availability. Overall, reproduction is more sensitive an endpoint than mortality, and there is no consistent evidence that one genus of earthworm is any less tolerant to Cu under a given set of conditions than another genus. Table 14-4 summarizes the toxicity of Cu to earthworms.

The acute toxicity of Cu to the nematode *C. elegans* in four soils and in solution was evaluated by Donkin and Dusenbery (1993). A concentration of 105 mg/kg Cu in solution caused 50% mortality while at least 400 mg/kg (sandy loam soil) was required in soil. The highest LC_{50} (1061 mg/kg) was associated with the highest percentage organic matter in the loam soil. Parmalee et al. (1993) used a soil microcosm to test the effects of Cu on survival of nematodes and microarthropods feeding on native soil organic matter. There was an average reduction of approximately 70% in number of individuals of most categories of nematodes (fungivores, bactivores, herbivores, hatchlings) at 400 mg/kg total Cu, while 185 ppm had no effect. The number of individuals of the omnivores/predators category was reduced 85% by the lowest concentration of Cu tested, 72 mg/kg. Total microarthropod numbers were reduced about 50% by 400 mg/kg. Hopkin and Hames (1994) investigated the effects of Cu (as $CuNO_3$) in food on survival and reproduction of the terrestrial isopod *Porcellio scaber*. After 360 days, the number of juveniles

Table 14-4. Toxicity of copper to heterotrophic processes and soil invertebrates (Will and Suter 1995b).

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuSO_4$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	159	1590	44	Juma & Tabatabai, 1977.
$CuCl_2$	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	159	1590	32	Juma & Tabatabai, 1977.
$CuSO_4$	native soil microflora	loam	7	5.5	0.1	Alkaline phosphatase activity	159	1590	23	Juma & Tabatabai, 1977.
$CuCl_2$	native soil microflora	sandy loam	6	3	548	Phosphatase activity	–	1895 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	sandy peat	4	6.5	548	Urease activity	–	1970 ED_{50}	50	Doelman & Haanstra, 1986.
$CuCl_2$	native soil microflora	silty loam	8	1	548	Urease activity	–	1990 ED_{50}	50	Doelman & Haanstra, 1986.
$CuCl_2$	native soil microflora	sandy peat	4	6.5	548	Phosphatase activity	–	2442 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	sandy peat	4	6.5	42	Phosphatase activity	–	2639 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	clay	8	1.5	42	Phosphatase activity	–	2722 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	clay	8	1.5	42	Arylsulfatase activity	–	2722 ED_{50}	50	Haanstra & Doelman, 1991.

Table 14-4 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuCl_2$	native soil microflora	clay	8	1.5	548	Arylsulfatase activity	–	4853 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	silty loam	8	1	42	Phosphatase activity	–	6424 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	sandy peat	4	6.5	548	Arylsulfatase activity	–	6996 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	sandy peat	4	6.5	42	Arylsulfatase activity	–	8904 ED_{50}	50	Haanstra & Doelman, 1991.
$CuSO_4$	native soil microflora	sandy loam	7	2	21	Nitrification	1000	10000	75	Premi & Cornfield, 1969.
$CuCl_2$	native soil microflora	silty loam	8	1	42	Arylsulfatase activity	–	14946 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	159	1590	51	Juma & Tabatabai, 1977.
$CuSO_4$	native soil microflora	loam	7	2.9	0.1	Arylsulfatase activity	159	1590	32	Al-Khafaji & Tabatabai, 1979.
$CuSO_4$	native soil microflora	clay loam	8	3.2	0.1	Arylsulfatase activity	159	1590	26	Al-Khafaji & Tabatabai, 1979.
$CuSO_4$	native soil microflora	clay loam	8	3.7	0.1	Acid phosphatase activity	–	1590 LCT	26	Juma & Tabatabai, 1977.

Table 14-4 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuCl_2$	native soil microflora	loam	7	5.5	0.1	Acid phosphatase activity	–	1590 LCT	43	Juma & Tabatabai, 1977.
$CuSO_4$	native soil microflora	clay loam	6	2.7	0.1	Arylsulfatase activity	–	1590 LCT	22	Al-Khafaji & Tabatabai. 1979,
$CuCl_2$	native soil microflora	clay	8	1.5	42	Urease activity	–	1370 ED_{50}	50	Doelman & Haanstra, 1986.
$CuCl_2$	native soil microflora	clay	8	1.5	548	Urease activity	–	1080 ED_{50}	50	Doelman & Haanstra, 1986.
$CuSO_4$	native soil microflora	sandy loam	7	2	21	N mineralization	100	1000	39	Quraishi & Cornfield, 1973.
$CuSO_4$	native soil microflora	sandy loam	5	2	21	N mineralization	100	1000	100	Quraishi & Cornfield, 1973.
$CuSO_4$	native soil microflora	sandy loam	6	2	21	N mineralization	100	1000	75	Quraishi & Cornfield, 1973.
$CuSO_4$	native soil microflora	sandy loam	8	2	56	Nitrification	100	1000	38	Premi & Cornfield, 1969/1970.
CuO	native soil microflora	sandy soil	6	2	84	C mineralization	–	1000 LCT	33	Bhuiya & Cornfield, 1972.
$CuCl_2$	native soil microflora	sandy loam	6	3	42	Arylsulfatase activity	–	967 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	silty loam	8	1	548	Arylsulfatase activity	–	763 ED_{50}	50	Haanstra & Doelman, 1991.

Table 14-4 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuCl_2$	native soil microflora	sandy loam	6	3	42	Urease activity	–	570 ED_{50}	50	Doelman & Haanstra, 1986.
$CuCl_2$	native soil microflora	sandy loam	6	3	548	Arylsulfatase activity	–	548 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	silt loam	5	–	–	Nitrification	–	0	>2	Suter and Sharples, 1984.
$CuCl_2$	native soil microflora	sandy soil	7	1	42	Arylsulfatase activity	–	391 ED_{50}	50	Haanstra & Doelman, 1991.
$CuSO_4$	native soil microflora	silty clay loam	7	6	20	N mineralization	–	320 LCT	82	Liang & Tabatabai, 1977.
$CuCl_2$	native soil microflora	sandy soil	7	1	548	Arylsulfatase activity	–	287 ED_{50}	50	Haanstra & Doelman, 1991.
$CuCl_2$	native soil microflora	sandy soil	7	1	42	Urease activity	–	260 ED_{50}	50	Doelman & Haanstra, 1986.
$Cu(NO_3)_2$	Pseudomonas denitrificans	silt loam	7	2	4	Denitrification	100	2	22	Bollag & Barabasz, 1979.
$Cu(NO_3)_2$	native soil microflora	silt loam	7	2	21	Denitrification	100	2	44	Bollag & Barabasz, 1979.
$CuCl_2$	native soil microflora	sandy soil	7	1	548	Phosphatase activity	–	170 ED_{50}	50	Doelman & Haanstra, 1989.
$CuCl_2$	native soil microflora	sandy soil	7	1	42	Phosphatase activity	–	140 ED_{50}	50	Doelman & Haanstra, 1989.

Table 14-4 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuSO_4$	native soil microflora	surface soil	–	1.3	1	Dehydrogenase activity	–	30 LCT	28	Rogers & Li, 1985.
$Cu(NO_3)_2$	Pseudomonas sp.	silt loam	7	2	4	Denitrification	–	10 LCT	53	Bollag & Barabasz, 1979.
	Dendrobaena rubida	soil & dung	7	5.7	120	cocoons/worm; hatchlings/cocoon; % hatching success	100	0_{50}	90, 100, 100	Bengtsson et al., 1986.
$C_4H_6CuO_4$	Eisenia fetida	horse manure			56	cocoon production	300	0	24	Malecki et al., 1982
$CuNO_3$	Eisenia fetida	OECD soil	6	5	14	survival LC	–	643		Neuhauser et al_{50} 1985.
$CuSO_4$	Octolasium cyaneum	Rendzina soil	–	14	14	survival LC	–	850		Streit & $Jaggy_{,50}$ 1983.
$C_4H_6CuO_4$	Eisenia fetida	horse manure	–	–	140	cocoon production	500	1000	24	Malecki et al., 1982.
$CuSO_4$	Lumbricus rubellus	loamy sand	5	2.9	18	cocoon production	83	148	26	Ma., 1984.
$CuCl_2$	Lumbricus rubellus	sandy loam	7	4	84	survival	150	1000	82	Ma., 1982.
$CuCl_2$	Lumbricus rubellus	loamy sand	5	2.9	42	cocoon production	54	131	42	Ma., 1984.
Soluble forms	Eisenia fetida	horse manure	–	–	42	growth; cocoon production	1000	2000	27, 85	Neuhauser et al., 1984.
$CuSO_4$	Octolasium cyaneum	Peat soil	–	43	14	survival LC	–	2500	50	Streit & Jaggy, 1983.
	Dendrobaena rubida	soil & dung	6	5.7	120	cocoons/worm; hatchlings/cocoon; hatching success	100	500	96, 100, 100	Bengtsson et al., 1986.

Table 14-4 (continued)

CHEMICAL FORM	ORGANISMS	GROWTH MEDIUM	pH	% OC	EXP (D)	RESPONSE PARAMETER	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	REFERENCE
$CuSO_4$	Octolasium cyaneum	Brown soil	–	3	14	survival LC	–	180	50	Streit & Jaggy, 1983.
	Dendrobaena rubida	soil & dung	5	5.7	120	cocoons/worm; hatchlings/cocoon	–	100LCT	70, 64	Bengtsson et al., 1986.
						total hatchlings			74	
$CuCl_2$	Allolobophora chlorotica	sandy loam	5	2.5		cocoon production	–	51	50	Ma, 1988.
$CuCl_2$	Lumbricus rubellus	sandy loam	7	1.7	42	cocoon production	13	63	41	Ma, 1984.
	Allolobophora caliginosa	polder soil	–	–	60	cocoon production	–	110LCT	27	van Rhee, 1975.
$CuCl_2$	Eisenia andrei	OECD soil	6	5	84	growth	32	100	32	van Gestel et al., 1991.
$Cu(NO_3)_2$	Eisenia fetida	OECD soil	6	–	56	cocoon production EC_{50}	–	53.3	50	Spurgeon et al., 1994.
$CuCl_2$	Lumbricus rubellus	sandy loam	5	2.5	–	cocoon production	–	122	50	Ma, 1988.
$CuCl_2$	Apporectodea caliginosa	sandy loam	5	2.5	–	cocoon production	–	68	50	Ma, 1988.

Note: Chemical concentrations are g of element/kg of growth medium
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls
EXP (D) = exposure in days
Growth Medium:OECD soil (% dry weight): sphagnum peat, 10; kaolin clay, 20; fine sand, 69; $CaCO_3$, 1; pH 6.0
% OC = % organic carbon
CEC = cation exchange capacity of growth medium (milliequivalents/100 g)
NEOC = no observed effect concentration

produced was decreased 53% by 50 mg/kg Cu while 100 mg/kg of Cu was required to reduce total survival. The slug, *Arion ater*, was used as the test organism by Marigomez et al. (1986) to determine the effects of several pollutants on terrestrial mollusks. After 27 days, the animals experienced a 55% decrease in growth at 1000 mg/kg Cu, while 300 mg/kg had no effect. The studies of Donkin and Dusenbery (1993) and Parmalee et al. (1993) taken together show a higher concentration in soil than in solution is required to affect the survival of nematodes. Differences among groups of nematodes in sensitivity to Cu is shown by Parmalee et al. (1993). The application of soluble form of Cu to food material by Hopkin and Hames (1994) and Marigomez et al. (1986) show very distinct sensitivities of woodlice and slugs to Cu.

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15. CYANIDE

15.1 BACKGROUND

Cyanide (CN) may occur in the form of hydrocyanic acid (HCN), the cyanide ion (CN^-), simple cyanides, metallocyanide complexes, and as simple chain and complex ring organic compounds. Free cyanide (CN), defined as the sum of the cyanides present as HCN and as CN^-, is the primary toxic agent of concern. HCN has the higher toxicity of the two forms, and CN^- may readily convert to HCN (Eisler 1991). Various cyanides found in the aquatic environment can be modified by water pH, temperature, and oxygen content. For example, when pH is <8 and the temperature is <25°C, at least 94% of the free cyanide exists as HCN. When pH and/or temperature are higher, a greater percentage of free cyanide exist as CN^- (EPA 1985). Anthropogenic sources of cyanide include electroplating plants, steel mills, petroleum refineries, and gas works, all which discharge effluent containing cyanide (Lind et al. 1977).

15.2 TOXICITY IN WATER TO AQUATIC LIFE

15.2.1 Acute Toxicity

Acute toxicity values for aquatic organisms range between 0.0447 and 2.490 mg/L as cyanide (EPA 1985) (Table 15-1). Fish were found to be the most sensitive aquatic organisms and tolerance was life stage, temperature, and species dependent. Embryos and sac fry were more resistant than more mature fish and fathead minnows were particularly resistant. Concentrations between 0.005 and 0.0072 mg/L of cyanide showed adverse effects on swimming and reproduction, with lethal effects on the sensitive fish occurring between 0.020 and 0.076 mg/L (EPA 1985).

15.2.2 Chronic Toxicity

Chronic toxicity values (CVs) for aquatic animals range between 0.00785 and 0.0341 mg/L (EPA 1985) (Table 15-1). Fathead minnow's reproductive cycle was highly sensitive to the presence of cyanide. 0.01 mg/L CN impaired swimming capabilities of salmonid fishes (Lind et al. 1977). Severe necrosis of the liver was discovered in rainbow trout exposed for 18 days at 0.01mg/L CN (Dixon 1975, as cited in Leduc 1978). Chronically toxic concentrations show deleterious effects in juvenile fish at 0.01 mg/L CN (Leduc 1977, as cited in Leduc 1978).

A number of studies have been done investigating the effects of cyanide on two generations of fish chronically exposed to cyanide. Egg production of the fathead minnow was down at 0.02 mg/L CN when exposed for 256 days (Lind et al. 1977). Kimball et al. (1978) found that overall, cyanide had little effects on the survival of adult bluegill (0–0.08 mg/L CN). Spawning, however, was completely inhibited at concentrations higher than 0.005 mg/L CN. After 144 days of exposure, there was no decrease in growth of brown trout adults at 0.005–0.077 mg/L. Number of eggs spawned decreased at concentrations above 0.0112 mg/L and the egg viability was down at 0.054 mg/L and nonexistent at 0.065 mg/L CN. Growth of the subsequent generation was affected at concentrations over 0.033 mg/L up to 90 days post-hatch. Judged from the spawning data, the lowest chronic value (LCV) for brook trout lies between 0.0057 and 0.011 mg/L CN (Koenst et al. 1977, as cited in Kimball et al. 1978). Leduc (1978) found an LCV around 0.005 mg/L for the Atlantic salmon. Adult salmon were exposed to concentrations between 0.01 and 0.1 mg/L CN for 103 days. The hatching success decreased from 80 to 54% at the lowest to highest concentrations tested. Those adults exposed to 0.04 mg/L CN and above produced fry that were 10% shorter than the controls, and the percentage of abnormal fry climbed from 5.8% of the control group to 18.5% at the highest concentration tested (Leduc 1978).

Table 15-1. Toxicity of cyanide to aquatic organisms (EPA 1985)

Conc. (mg/L)[1]	Species	Effect
2.49	Midge (*Tanytarsus dissimilis*)	LC_{50}
2.326	Isopod (*Asellus communis*)	LC_{50}
0.432	Snail (*Physa heterostropha*)	LC_{50}
0.426	Stonefly (*Pteronarcys dorsata*)	LC_{50}
0.318	Goldfish (*Carassius auratus*)	LC_{50}
0.167	Scud (*Gammarus pseudolimnaeus*)	LC_{50}
0.16	Water flea (*Daphnia magna*)	EC_{50}
0.147	Guppy (*Poecilia reticulata*)	LC_{50}
0.1251	Fathead minnow (*Pimephales promelas*)	LC_{50}
0.102	Largemouth bass (*Micropterus salmoides*)	LC_{50}
0.102	Black crappie (*Pomoxis nigromaculatus*)	LC_{50}
0.09928	Bluegill (*Lepomis macrochirus*)	LC_{50}
0.11	Water flea (*Daphnia pulex*)	EC_{50}
0.09264	Yellow perch (*Perca flavescens*)	LC_{50}
0.09	Atlantic salmon (*Salmo salar*)	LC_{50}
0.0858	Book trout (*Salvlinus fontinalis*)	LC_{50}
0.04473	Rainbow trout (*Onchorynchus mykiss*)	LC_{50}
0.03406	Isopod (*Asellus communis*)	CV
0.01833	Scud (*Gammarus pseudolimnaeus*)	CV
0.01639	Fathead minnow (*Pimephales promelas*)	CV
0.01357	Bluegill (*Lepomis macrochirus*)	CV
0.007849	Brook trout (*Salvelinus fontinalis*)	CV

[1]Concentrations are given as CN, not the compound.

15.2.3 Toxicity to Aquatic Plants

Adverse effects of cyanide on plants are unlikely at concentrations that were found to cause acute effects in aquatic animals. The natural sources of cyanide include various bacteria, algae, fungi, and higher plants. Higher plants may be adversely affected by cyanide through cytochrome oxidase inhibition (Eisler 1991). EC_{50}s for Eurasian watermilfoil *(Myriophyllum spicatum)* were reported at

20.0 mg/L for shoot weight, 22.4 mg/L for root weight, 27.3 for shoot length, and 28.6 for root length (Stanley 1974).

15.2.4 Bioaccumulation

Studies of bioaccumulation and biomagnification of cyanide have not been reported. Although cyanide is taken up by aquatic organisms, bioaccumulation cannot be demonstrated due to its high rate of metabolism (EPA 1985).

15.2.5 Aquatic Mode of Action

Cyanides are absorbed by aquatic organisms through respiratory surfaces, ingestion, or skin contact and then distributed throughout the body by the blood. Upon exposure, toxic reactions may occur in seconds, and, depending on the amount absorbed, may lead to death in a matter of minutes. The toxic effect of cyanide occurs with the inhibition of cytochrome oxidase. Respiratory arrest and death occur after blockage of aerobic ATP synthesis.

Leduc (1978) investigated the adverse reactions of cyanide on the early life stages of the Atlantic salmon *(Salmo salar)*. Newly fertilized eggs became bleached in appearance when exposed to 0.1 mg/L CN, which may be an indication of the substitution of carotenoid pigments for oxygen as hydrogen receptors in the process of oxidation. This aerobic pathway is responsible for the normal development of embryonic stages, including early morphogenesis and cell differentiation, and was shown to be highly sensitive to the presence of cyanide.

15.2.6 Water Quality Criteria

The chronic National Ambient Water Quality criterion is 0.0052 mg/L, and the acute criterion is 0.022mg/L.

15.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on the toxicity of cyanide to benthic invertebrates.

15.4 TOXICITY TO PLANTS

15.4.1 Toxicity to Plants in Soil

Biological processes dependent on ATP, such as respiration, ion uptake, and phloem translocation, are inhibited at high concentrations of cyanide. While cyanide may enhance seed germination in tolerant species at lower concentrations, its toxic effects include germination and growth inhibition (Towill et al. 1978, as cited in Eisler 1991). Quantitative information on toxicity is not available.

15.4.2 Toxicity to Plants in Solution

No information was obtained on the toxicity of cyanide to plants in solution.

15.4.3 Phytotoxic Mode of Action

No information was obtained on the phytotoxic mode of action of cyanide.

15.5 TOXICITY TO WILDLIFE

15.5.1 Toxicity to Mammals

Cyanide has not proved to be mutagenic, teratogenic, or carcinogenic in animals, and despite its rapid lethal action at high doses, cyanide can be tolerated at sublethal, sporatic doses for long periods (Eisler 1991). Adult rats feed 100 and 300 mg/kg CN for two years, showed no signs of cyanide toxicosis (Howard and Hanzal 1955).

Most research on the toxicity of cyanide to mammals involves livestock as they are prone to forage sorgums and other cyanogenic plants. Symptoms of acute poisoning in livestock are dose dependent and include initial excitability, salivation, lacrimation, defecation, urination, labored breathing, muscular incoordination, gasping, and convulsions (Towill et al. 1978 and Cade and Rubira 1982, as cited in Eisler 1991). Horses feeding on Sudan grass and sorghums developed posterior muscle incoordination, urinary incontinence, and spinal malformations (Towill et al. 1978, as cited in Eisler 1991).

Symptoms of acute cyanide poisoning in small mammals include rapid and labored breathing, ataxia, cardiac irregularities, dialated pupils, convulsions, coma, respiratory failure, and eventual death (Egekeze and Oheme 1980; Ballantyne 1983, both as cited in Eisler 1991). Systemic arterial pressure in dogs is disrupted with exposure to cyanide, and cerebral flow increases in both cats and rabbits (Robinson et al. 1985, as cited in Eisler 1991). Motor functions are severely altered, including muscle incoordination, disrupted swimming, and conditioned avoidance response (D'Mello 1987, as cited in Eisler 1991).

The mode of action remains unknown, however, cyanide intoxication in mice appears to involve the inhibition of hepatic rhodanese activity, due to either the depletion of the sulfane-sulfur pool or to blockage by excess binding to the active site (Buzaleh et al. 1989, as cited in Eisler 1991).

Tewe and Maner (1981) fed rats a diet containing 500 mg/kg CN as KCN during gestation and lactation. While offspring growth and food consumption were both significantly reduced, values for treated individuals were only marginally less than for controls (reductions were 7% or less). Sample et al. (1996) did not consider the effects to be biologically significant, therefore, the 500 mg/kg diet (68.7 mg/kg/d) was considered to represent a chronic no-observed-adverse-effect level (NOAEL) for reproductive effects in rats.

15.5.2 Toxicity to Birds

Initial symptoms of cyanide toxicosis to intolerant species include panting, eye blinking, salivation, and lethargy, all appearing between 0.5 and 5 minutes post-exposure. At higher concentrations, breathing became increasingly deep and labored, followed by gasping and sporatic breathing. Tolerant species, such as the domestic chicken, may recover if they survive 60 minutes post-exposure (Wiemeyer et al. 1986, as cited in Eisler 1991) (Table 15-2).

Table 15-2. Acute oral toxicity of cyanide to birds (Wiemeyer et al. 1986, as cited in Eisler 1991)

Conc. (mg/kg bw)	Species	Effect
11.10	Domestic chicken	LD_{50}
9.00	European starling *(Sturnus vulgaris)*	LD_{50}
5.50	Japanese quail *(Coturnix japonica)* (male)	LD_{50}
4.60	Eastern screech-owl *(Otus asio)*	LD_{50}
4.50	Japanese quail *(Coturnix japonica)* (female)	LD_{50}
3.70	Black vulture *(Coragyps atratus)*	LD_{100}
2.54	Black vulture *(Coragyps atratus)*	LD_{50}
2.12	American kestrel *(Falco sparverius)*	LD_{50}
0.12	Rock dove *(Columba livia)*	LD_{100}
0.12	Canary *(Serinus canarius)*	LD_{100}

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16. FLUORINE

16.1 BACKGROUND

The class of chemicals known as fluorides consist of compounds derived from hydrofluoric acid [Agency for Toxic Substances and Disease Registry (ATSDR) 1993] and contain the most electronegative of all elements, fluorine (F) (Camargo et al. 1992a). Fluorides are widely, but variably, distributed in the environment, present in water, soils, and living tissues (Camargo et al. 1992a), and form about 0.06–0.09% of the upper layers of the earth's crust [National Academy of Sciences (NAS) 1980]. Free elemental fluorine rarely exists in nature and only at a valence of -1 (Camargo et al. 1992b). Fluorides enter the environment naturally due to the weathering of fluorite and other minerals, and as a result of volcanic activity. The greatest concentrations, however, are encountered near point sources related to human activity (ATDSR 1993). Fluoride compounds are used in a wide range of fields, including the glass and ceramics industries (Windholz 1976) and present in the effluent of phosphate processing and aluminum smelting operations (Wright 1977). Some fluorides such as oxygen difluoride are highly toxic and very reactive, but due to their reactivity would not migrate unchanged from a hazardous waste site (ATSDR 1993). Fluoride salts such as sodium fluoride and calcium fluoride are much less toxic, however, seepage from hazardous waste sites and subsequent exposure to biota present a significant danger.

16.2 TOXICITY IN WATER TO AQUATIC LIFE

16.2.1 Acute Toxicity

Table 16-1 summarizes acute toxicity data for fluorine. Fish tolerance of fluorine toxicity is increased by lower temperatures, lower concentration of the chlorine ion, and greater water hardness. The 96h LC_{50} for Rainbow trout rose from 51.0 to 193.0 mg/L fluorine when the hardness was increased from 17 mg/L to 385 mg/L Ca. The influence of calcium on the toxicity of fluorine is linked to the formation and precipitation of CaF_2 rendering it unavailable for uptake (Pimentel and Bulkley 1983). 25.0 mg/L fluorine did not prove to be acutely toxic to *Cyprinus carpio* (Prochnow 1978, as cited in Camargo and Tarazona 1990).

Acute exposure to fluorine by net-spinning caddisflies (trichoptera) has been linked to migration and retreat from capture nets, and the protrusion of anal papillae. The percentage of darkened, protruded anal papillae increases with fluorine concentration in the test medium (Camargo et al. 1992b). As a whole, trichoptera test larvae appear to be more sensitive to fluorine than other aquatic invertebrates. *Daphnia magna* had a no-discernible-effect concentration of 110 mg/L (LeBlanc 1980). See Table 16-1.

16.2.2 Chronic Toxicity

No standard chronic toxicity data were found for fluorine. In soft water, the 20-day LC_{50} was between 2.7 and 4.7 mg/L F^- for rainbow trout, 10–20 cm in length (Vallin 1968, as cited in Wright 1977).

16.2.3 Toxicity to Aquatic Plants

The fluorine content in aquatic plants increases with the element's concentration in the water. An increase in fluorine has been linked to damaged cell membranes and RNA structure and to a decrease

Table 16-1. Fluorine toxicity towards aquatic organisms (AQUIRE 1996)

Chemical	Conc. (mg/L)[1]	Species	Effect
BF_3	15000.0	Bluegill *(Lepomis macrochirus)*	24h LC_{50}
F	125.0	Brown trout *(Salmo trutta)*	48h LC_{50}
NaF	353600.0	Water flea *(Daphnia carinata)*	24h EC_{50}[2]
NaF	157900.0	Water flea *(Ceriodaphnia dubia)*	24h EC_{50}[2]
NaF	83200.0	Water flea *(Ceriodaphnia pulchella)*	24h EC_{50}[2]
NaF	850.0	Algae *(Scenedesmus subspicatus)*	72h EC_{50}
NaF	680.0	Water flea *(Daphnia magna)*	24h LC_{50}
NaF	560.0	Mosquitofish *(Gambusia affinis)*	24h LC_{50}
NaF	418.0	Mosquitofish *(Gambusia affinis)*	48h LC_{50}
NaF	380.0	Stickleback *(Gasterosteus aculeatus)*	96h LC_{50}
NaF	315.0	Fathead *(Pimephales promelas)*	96h LC_{50}
NaF	205.0	Water flea *(Daphnia magna)*	48h EC_{50}[2]
NaF	164.5	Brown trout *(Salmo trutta)*	96h LC_{50}
NaF	158.0	Rotifer *(Philodina acuticornis)*	48h EC_{50}[2]
NaF	128.0	Caddisfly *(Cheumatopsyche pettiti)*	48h EC_{50}[2]
NaF	53.5	Caddisfly *(Hydropsyche occidentalis)*	72h LC_{50}
NaF	52.6	Caddisfly *(Ceratopsyche bronta)*	48h EC_{50}[2]
NaF	51.0	Rainbow trout *(Oncorhynchus mykiss)*	96h LC_{50}
NaF	44.9	Caddisfly *(Chimarra marginata)*	96h LC_{50}

[1]Concentrations are given as F, not the compound.
[2]Immobilization.

in photosynthesis, phosphate metabolism, and carbonate metabolism (LeBlanc et al. 1971, as cited in Samecka-Cymerman and Kempers 1990; Kabata-Pendias and Pendias 1984). The 48-hour LC_{100} for the aquatic liverwort *(Scapania undulata)* was found to be 400.0 mg/L F^- (Samecka-Cymerman and Kempers 1990).

16.2.4 Bioaccumulation

Invertebrates and vertebrates tend to accumulate fluorine in their exoskeletons and skeletons under chronic exposure (Wright and Davidson 1975, as cited in Wright 1977; Henry and Burke 1990, as cited in Camargo et al. 1992a).

16.2.5 Aquatic Mode of Action

The accumulation of fluorine in bone and soft tissue is attributed to its formation of stable complexes with calcium in blood and bone (Sigler and Neuhold 1972, as cited in Wright 1977).

16.2.6 Water Quality Criteria

No water quality criteria exist for fluorine.

16.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

No information was found on fluorine toxicity to sediment invertebrates.

16.4 TOXICITY TO PLANTS

16.4.1 Toxicity to Plants in Soil

Fluoride present in the air as a gas (HF, SiF_4) or a particulate (NaF, $NaSiF_6$, AlF_3, Na_3AlF_6, $Na_5Al_3F_{14}$, CaF_2) is readily absorbed through the stomata of the leaf or cuticular layer and presents a significant hazard (Muramoto et al. 1991). Kloke (1979) reported unspecified reductions in plant growth in a surface soil with the addition of 200 mg/kg fluorine.

16.4.2 Toxicity to Plants in Solution

The EC_{50} for root length in lettuce *(Lactuca sativa)* was found to be 660.0 mg/L when exposed to NaF in solution (Ratsch and Johndro 1984). Scharrer (1955) reported unspecified reductions in plant growth in a solution culture with the addition of 5 mg/L fluorine.

16.4.3 Phytotoxic Mode of Action

Fluorine is not an essential plant element. Toxicity symptoms observed in plants exposed to HF gas are marginal leaf chlorosis and interveinal chlorosis (Brewer 1966).

16.5 TOXICITY TO WILDLIFE

16.5.1 Toxicity to Mammals

Acute oral exposures to extremely high concentrations of sodium fluoride resulting from accidental or intentional poisoning may cause gastrointestinal effects and death due to respiratory or cardiac arrest. Animal data provide evidence of the lethality of sodium fluoride following acute oral exposure (ATSDR 1993). Adverse reproductive effects from fluoride have been observed in dogs (Shellenberg et al. 1990).

Aulerich et al. (1987) assessed the effects of sodium fluoride on growth, reproduction, and survivorship in mink by feeding them diets containing 33, 60, 108, or 350 mg/kg supplemental fluorine for 382 days (basal diet contained 35 mg/kg F). No significant differences in growth were observed among the control and treatment mink, nor were there any measurable adverse reproductive effects. Survivorship was unaffected at dietary concentrations ≤194 mg/kg; survivorship of adult mink was reduced at 350 mg/kg, and only 14% of kits from females in the 350 mg/kg treatment group survived to 3-weeks of age (Aulerich et al. 1987). Based on the results of this study, Sample et al. (1996) estimated the no-observed-adverse-effect level (NOAEL) and lowest-observed-adverse-effect level (LOAEL) for reproductive effects in mink to be 31.37 and 52.75 mg/kg/d, respectively. Fluorides at ≥100 mg/L in drinking water impaired reproduction and retarded the growth of mice (Messer et al. 1973). Male white-tailed deer (*Odocoileus virginianus*) fed 50 mg/kg fluoride as NaF for 2 years developed dental lesions and accumulated >7000 mg/kg fluoride in bone ash (Suttie et al. 1985).

16.5.2 Toxicity to Birds

Diets containing 1000 mg/kg of fluoride as sodium fluoride fed to chicks for 28 days resulted in reduced growth (Doberenz et al. 1965). Hatching success in eastern screech owls (*Otus asio*) was adversely affected at dietary concentrations of 232 mg/kg fluoride as sodium fluoride (wet weight), but not at concentrations of ≤56.5 mg/kg (Hoffman et al. 1985; Pattee et al. 1988). Based on the results of these studies, Sample et al. (1996) estimated the NOAEL and LOAEL for reproductive effects in screech owls to be 7.8 and 32 mg/kg/d, respectively. High levels of fluoride (700–1000 mg/kg) in diet of chickens resulted in reduced egg size and increased mortality (Guenter 1979).

16.6 TOXICITY TO SOIL HETEROTROPHIC PROCESS

The effects of fluoride added as potassium fluoride on nitrogen and phosphorus mineralization by native microflora in poplar litter was investigated by van Wensem and Adema (1991). After 9 weeks, K mineralization was reduced 22% in litter treated with 32.3 mg/kg fluorine, the lowest concentration tested. Nitrogen mineralization was reduced 26% by 100.7 mg/kg, while 32.3 mg/kg had no effect. The effects of fluorine on dehydrogenase activity of the native soil microflora in soil from the Rocky Mountain Arsenal was assessed by Rogers and Li (1985). A concentration of 5000 mg/kg fluorine reduced dehydrogenase activity by 30% (3000 mg/kg had no effect). The toxic concentration of 32 mg/kg (van Wensem and Adema 1991) is the lowest of the two reported (Table 16-2).

Table 16-2. Fluorine toxicity to soil heterotrophic processes

Chemical form	Organism	Growth medium	pH	%0 C	EXP (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
Naf	native soil microflora	surface soil	-	1.3	1	Dehydrogenase activity	3000	5000	30	Roger & Li 1985
KF	native soil microflora	leaf litter	-	-	63	P mineralization	-	32 LCT	22	van Wensen & Adema 1991

Note: Chemical concentration are g of element kg of growth medium.
% DEC=% decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls.
EXP D=exposure in days.
%OC= organic carbon

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17. MANGANESE

17.1 BACKGROUND

Manganese (Mn) makes up about 0.10% of the earth's crust and is the 12th most abundant element (NAS 1980). In soil, natural levels of manganese range from 0.6 to 0.9 mg/kg and its solubility increases with decreasing pH. In surface water, manganese is present at concentrations ranging from 0.001 to 0.04 mg/L (Rouleau et al. 1995). Manganese oxides and peroxides are used in industry as oxidizers, and elemental manganese is used for manufacturing metal alloys to increase hardness and corrosion resistance. Increasingly, manganese as a manganese carbonyl compound, is being utilized as an anti-knocking agent in engines and is released into the air as Mn_3O_4 following combustion (Brault et al. 1994). Manganese is also present in the air and water discharges from mining and smelting activities (Saric 1986). In living systems, manganese is an essential element that is found most often in the +2 valence state. There is evidence that manganese occurs in surface waters both in suspension in the quadrivalent state and in the trivalent state in a relatively stable, soluble complex (APHA 1989). Manganese is one of the first metals to increase in concentration in acidified waters (Harvey 1983).

17.2 TOXICITY IN WATER TO AQUATIC LIFE

17.2.1 Acute Toxicity

Manganese is very rarely acutely toxic to fish. Table 17-1 summarizes acute toxicity data.

Acute toxicity tests using fathead minnows and *Daphnia magna* were conducted in relatively hard water. Acute toxicity values for manganese ranged from 19.4 mg/L for *D. magna* to 33.80 mg/L for fathead minnow (Kimball 1978). Manganese toxicity was increased by the presence of food during the *D. magna* acute toxicity tests. Biesinger and Christensen (1972) reported an LC_{50} of 9.80 mg/L for *D. magna* without food added (Table 17-1).

17.2.2 Chronic Toxicity

Biesinger and Christensen (1972) reported an EC_{50} value for reproductive impairment of 5.2 mg/L and a 3-week LC_{50} of 5.7 mg/L. The chronic value (CV) of 4.10 mg/L based on a 3-week toxicity test resulted in 16% reproductive impairment of *D. magna*.

After a 30-day exposure to manganese, brown trout showed a decrease in body calcium concentrations and impaired development in the two highest concentrations (0.36 and 1.08 mg/L) tested (Reader et al. 1988). The CV for this assay was calculated as 0.21 mg/L.

These results are not included in Table 17-1, because they are not much differant than from standard toxicity tests.

17.2.3 Toxicity to Aquatic Plants

No information on the toxicity of manganese to aquatic plants was found.

Table 17-1. Acute toxicity of manganese to aquatic organisms

Conc.[1] (mg/L)	Species	Effect	Reference
3230.0	tropical perch *(Colisa fasciatus)*	96h LC_{50}	Nath and Kumar 1987
1679.0	*(Oreochromis mossambicus)*	96h LC_{50}	Seymore et al. 1993
771.0	aquatic sowbug *(Asellus aquaticus)*	48h EC_{50}[2]	AQUIRE 1996
333.0	aquatic sowbug *(Asellus aquaticus)*	96h EC_{50}[2]	AQUIRE 1996
130.0	logfin dace *(Agosia chrysogaster)*	96h LC_{50}	Lewis 1978
51.0	crayfish *(Orconectes limosus)*	96h LC_{50}	AQUIRE 1996
38.7	rotifer *(Brachionus calyciflorus)*	24h LC_{50}	AQUIRE 1996
33.8	fathead minnow *(Pimephales promelas)*	96h LC_{50}	Kimball 1978
28.0	crayfish *(Austropotamobius pallipes)*	96h LC_{50}	AQUIRE 1996
19.4	water flea *(Daphnia magna)*	48h LC_{50}	Kimball 1978
16.6	frog *(Microhyla ornata)*	24h LC_{50}	AQUIRE 1996
16.0	frog *(Microhyla ornata)*	48h LC_{50}	AQUIRE 1996
9.8	water flea *(Daphnia magna)*	48h EC_{50}	Biesinger and Christensen 1972

[1]Concentrations given as Mn, not the compound.
[2]Immobilization.

17.2.4 Bioaccumulation

Body concentrations of manganese in aquatic organisms is poorly correlated with the total ambient concentration of the metal (Bendell-Young and Harvey 1986). In young trout, manganese has been known to concentrate in newly forming fibrous or cartilaginous bone (Hibiya 1982), while in older fish, manganese targets the liver and gills (Rouleau et al. 1995). Studies on the bioaccumulation of manganese have found bioconcentration factors for brown trout, fathead minnow, and yellow perch to be 17.8, 22.6, and 12.0, respectively (Rouleau et al. 1995; Kwasnik et al. 1978; Kearns and Vetter 1982).

Benthic invertebrates appear to play an important role in the transfer of manganese to fish. 70% of the whole body concentrations of manganese was present in the gut contents of *Tipula* (spp.), a detritus-feeding aquatic insect (Elwood et al. 1976).

17.2.5 Aquatic Mode of Action

Manganese uptake to the brain occurs via the olfactory system which by its nature enables the quick deposition of the metal in the olfactory bulb of the brain (Rouleau et al. 1995). Body deposition occurs via the circulatory system targeting the liver, kidney, muscle, and inorganic portions of the bone (Bendell-Young and Harvey 1986).

Once transfered to the organs, manganese impairs calcium transport. Fish from lakes with high levels of manganese exhibit symptoms of impaired transport of calcium during oogenesis and altered calcium deposition in the skeleton (Beamish et al. 1975; Fraser and Harvey 1982). Ca^{2+}- ATPase activity on the gill is inhibited and both net uptake and deposition of calcium were impaired by manganese (Reader et al. 1988).

17.2.6 Water Quality Criteria

There are no water quality criteria for manganese.

17.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Very little sediment toxicity information is available for manganese. The Ontario Ministry of the Environment has estimated that most benthic organisms in that region will not be adversely affected by concentrations of 460 mg/kg, which is consistent with the Great Lakes pre-colonial sediments background level of 400 mg/kg. A concentration of 1110 mg/kg Mn is estimated to be toxic to most benthic organisms (Persaud et al. 1990).

17.4 TOXICITY TO PLANTS

17.4.1 Toxicity to Plants in Soil

The availability of manganese to plants in soil is dependent on the concentration of other cations, cation exchange capacity, pH, drainage, organic matter content, temperature, compactness and bacterial activity (NRC 1973; Adriano 1986). For example, an excess of zinc has been shown to increase the uptake of manganese. It has also been shown that uptake increases as the soil becomes more acidic and water-logged, promoting the reduction of manganese to the more bioavailable Mn(II) (Brault, et al. 1994; Foy et al. 1978).

Wallace et al. (1977) evaluated the effects of manganese added as $MnSO_4$ on leaf and stem weights of bush beans grown from seed in a loam soil for 17 days. Stem weight was reduced 29% by 500 mg/L Mn, the lowest concentration tested.

17.4.2 Toxicity to Plants in Solution

The effect of manganese ($MnSO_4$) on Norway spruce seedling growth was evaluated by Langheinrich et al. (1992). In an experiment run at pH 6 for 32 days, manganese added at 44 mg/kg reduced root growth approximately 45% (11 mg/kg had no effect). In experiments run at pH 4 for 77 days, manganese added at 44 mg/kg (only concentration tested) reduced growth by approximately 50%.

Rye grass, bush beans, tomatoes, and potatoes have also been tested. After 14 days, a concentration of 0.75 mg/kg Mn in solution (pH 7) caused a 71% reduction in rye grass growth ($MnSO_4$; lowest concentration tested) (Wong and Bradshaw 1982). In a 16-day experiment, bush bean weights were reduced approximately 25% by 5.5 mg/kg Mn ($MnSO_4$; lowest concentration tested) (Wallace et al. 1977). In a 21-day experiment, weights were reduced approximately 40% by 55 mg/kg, while 5.5 mg/kg Mn had no effect. LeBot et al. (1990) evaluated the effect of manganese as $MnSO_4$ on weight of tomato plants growing in nutrient solution (pH 5.5) for 17 days. Manganese at 5.5 mg/kg reduced plant weight by 27%, while 2.8 mg/kg had no effect. A concentration of 33.5 mg/kg Mn (lowest concentration tested) caused a 23% reduction in potato shoot weight after 32-day growth (Marsh and Peterson 1990) (Table 17-2).

17.4.3 Phytotoxic Mode of Action

Manganese is essential for plant growth. It is involved in nitrogen assimilation, as a catalyst in plant metabolism, and functions with iron in the synthesis of chlorophyll (Labanauskas 1966). Toxicity symptoms include marginal chlorosis and necrosis of leaves, leaf puckering, necrotic spots on leaves, and root browning. Excess manganese interferes with enzymes, inhibits DNA replication, decreases respiration, and is involved in the destruction of auxin (Foy et al. 1978). Manganese is uniformly distributed between all parts of the plant, however, more damage is inflicted on the plant tops (Wallace and Romney 1977). The chemical elements Si, Fe, Ca, and P have been known to alleviate toxic symptoms under specific conditions (Foy et al. 1978).

17.5 TOXICITY TO WILDLIFE

17.5.1 Toxicity to Mammals

Laskey et al. (1982) fed rats diets containing 400, 1100, or 3550 mg/kg Mn (as Mn_3O_4) for 224 days. While the pregnancy percentage and fertility among rats consuming 3550 ppm Mn in their diet was significantly reduced, all other reproductive parameters (e.g., litter size, ovulations, resorptions, preimplantation death, fetal weights) were not affected. No effects were observed at lower manganese exposure levels. Sample et al. (1996) considered the 1100 mg/kg diet (88 mg/kg/d) to be a chronic NOAEL and the 3550 mg/kg diet (284 mg/kg/d) to be a chronic LOAEL for reproduction in rats.

17.5.2 Toxicity to Birds

Laskey and Edens (1985) fed Japanese quail a diet containing 5056 mg/kg Mn (as Mn_3O_4) for 75 days. While no reduction in growth was observed, aggressive behavior was 25% to 50% reduced relative to controls. Reduced aggressive behavior was not considered to be a significant adverse effect. Daily manganese consumption was reported to range from 575 mg/kg/day for adults at the end of the study and 977 mg/kg/d for 20-day-old birds. The 977 mg/kg/d dose was considered to be a chronic NOAEL (Sample et al. 1996).

17.6 TOXICITY TO SOIL HETEROTROPHIC PROCESS

Liang and Tabatabai (1977) investigated the effects of manganese on nitrogen mineralization by native soil microflora in four soils. Manganese at 275 mg/kg reduced nitrogen mineralization in one soil. Premi and Cornfield (1969) investigated the effects of manganese added to a sandy loam soil on nitrogen transformations by native soil microflora. In a 21-day experiment, nitrification was severely

Table 17-2. Phytotoxicity data for manganese derived from experiments conducted in solution (Will and Suter 1995a)

Chemical Form	Plant species	NOEC (mg/L)	LOEC (mg/L)	Growth parameter	Reference
$MnSO_4$	ryegrass	-	0.75 LCT	length longest root	Wong and Bradshaw 1982
$MnSO_4$	cotton	-	4 LCT	root & leaf weight	Foy et al. 1995
$MnSO_4$	cotton	-	4 LCT	root & leaf weight	Foy et al. 1995
$MnSO_4$	bush beans	-	5.5 LCT	root, leaf & stem weights	Wallace et al. 1977
$MnSO_4$	tomato	2.8	5.5	plant weight	Le Bot et al. 1990
$MnSO_4$	cotton	4	8	root & leaf weight	Foy et al. 1995
$MnSO_4$	cotton	8	16	root & leaf weight	Foy et al. 1995
$MnSO_4$	wheat	-	30 LCT	root weight	Burke et al. 1990
$MnSO_4$	wheat	-	30 LCT	root weight	Burke et al. 1990
$MnSO_4$	wheat	-	30 LCT	root & shoot weights	Burke et al. 1990
$MnSO_4$	wheat	-	30 LCT	root weight	Burke et al. 1990
$MnSO_4$	spruce	11	44	root length	Langeheinrich et al. 1992
$MnSO_4$	spruce	11	44	growth rate	Langheinrich et al. 1992
$MnSO_4$	potato	-	33.5 LCT	fresh shoot weight	Marsh and Peterson 1990
$MnSO_4$	spruce	-	44 LCT	height epicotyl	Langheinrich et al. 1992
$MnSO_4$	spruce	-	44 LCT	height epicotyl	Langheinrich et al. 1992
$MnSO_4$	bush beans	5.5	55	root, leaf & stem weights	Wallace et al. 1977
$MnSO_4$	wheat	30	90	root & shoot weights	Burke et al. 1990
$MnCl_2$	rice	-	100 EC_{50}	radicle weight	Wang 1994

inhibited at 100 mg/kg added as sulfate salt, the lowest concentration tested. Juma and Tabatabai (1977) evaluated the effect of manganese on soil acid and alkaline phosphatase activities. Acid phosphatase activity was affected at 1375 mg/kg only in the soil with the lowest pH and organic matter and clay contents. Alkaline phosphatase activity was reduced in one of the soils tested by this same concentration. The effective concentration of 100 mg/kg (Premi and Cornfield 1969) is the lowest of the four reported (Table 17-3).

Table 17-3. Toxicity of manganese to soil heterotrophic processes (Will and Suter 1995b)

Chemical form	Organisms	Growth medium	pH	%OC	Exp (D)	Response parameter	NOEC (mg/kg)	LOEC (mg/kg)	% DEC	Reference
$MnCl_2$	native soil microflora	clay loam	8	3.7	0.1	Alkaline phosphatase activity	–	1375 LCT	25	Juma & Tabatabai 1977
$MnCl_2$	native soil microflora	clay loam	8	4	20	N mineralization	–	275 LCT	26	Liang & Tabatabai 1977
$MnSO_4$	native soil microflora	sandy loam	7	2	21	Nitrification	–	100 LCT	67	Premi & Cornfield 1969
$MnCl_2$	native soil microflora	loam	6	2.6	0.1	Acid phosphatase activity	137.5	1375	62	Juma & Tabatabai 1977

Note: Chemical concentrations are expressed in grams of element per kilogram of growth medium.
% DEC = % decrease in measured parameter at lowest observed effect concentration (LOEC) as compared to controls.
EXP (D) = exposure in days.
% OC = % organic carbon.

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18. STRONTIUM

18.1 BACKGROUND

Strontium (Sr) is usually found in the basic mineral form of strontianite ($SrCO_3$) but is often tested in the form of strontium chloride ($SrCl_2$). The mean level of strontium found in the earth's crust is 450 mg/kg (Oehme 1979).

18.2 TOXICITY IN WATER TO AQUATIC LIFE

18.2.1 Acute Toxicity

The strontium 96-hour LC_{50} for striped bass (*Morone saxatilis*) was >92.8 mg/L (Dwyer et al. 1992). Survival of the striped bass increased with increased hardness in the strontium test.

According to Biesinger and Christensen (1972), an acute toxicity test (48-hour) on *Daphnia magna* had an LC_{50} of 125 mg/L. More recent studies of the toxicity of strontium on *Daphnia magna* reported by Khangarot and Ray (1989) were similar, with strontium having a 24-hour EC_{50} of 162.93 mg/L and a 48-hour EC_{50} of 94 mg/L. Acute toxicity tests on *Tubifex tubifex* worms yielded EC_{50}s of 540 mg/L for the 24-hour test, 320 mg/L for the 48-hour test, and 240.8 mg/L for the 96-hour test (Table 18-1).

18.2.2 Chronic Toxicity

A strontium chronic (3-week) toxicity test on *Daphnia magna* produced a 16% reproduction inpairment at 42.0 mg/L (Biesinger and Christensen 1972).

18.2.3 Toxicity to Aquatic Plants

Information on the toxicity of strontium to aquatic plants was not found.

18.2.4 Bioaccumulation

Specific data pertaining to the bioaccumulation of strontium were not found in the literature; however, strontium has been known to substitute for calcium in the bony tissues of fishes (Snyder et al. 1992).

18.2.5 Aquatic Mode of Action

Information on the mode of action of strontium to fish or aquatic invertebrates was not found.

18.2.6 Water Quality Criteria

Strontium was reported as having a secondary acute value of 14.5 mg/L and a secondary chronic value of 1.5 mg/L (Suter and Tsao 1996).

Table 18-1. Toxicity of strontium to aquatic organisms

Conc. (mg/L)[1]	Species	Effect	Reference
540.00	Tubificid worm *(Tubifex tubifex)*	24h EC_{50}	Khangarot 1991
320.00	Tubificid worm *(Tubifex tubifex)*	48h EC_{50}	Khangarot 1991
240.80	Tubificid worm *(Tubifex tubifex)*	96h EC_{50}	Khangarot 1991
162.93	Water flea *(Daphnia magna)*	24h EC_{50}[2]	Khangarot & Ray 1989
125.00	Water flea *(Daphnia magna)*	48h LC_{50}	Biesinger & Christensen 1972
94.00	Water flea *(Daphnia magna)*	48h EC_{50}[2]	Khangarot & Ray 1989
86.00	Water flea *(Daphnia magna)*	3w LC_{50}	Biesinger & Christensen 1972
60.00	Water flea *(Daphnia magna)*	3w EC_{50}[2]	Biesinger & Christensen 1972
42.00	Water flea *(Daphnia magna)*	3w EC_{16}[3] 16%	Biesinger & Christensen 1972

[1]Concentrations are given as Sr, not the compound.
[2]Immobilization.
[3]Reproductive impairment.

18.3 TOXICITY IN SEDIMENT TO BENTHIC INVERTEBRATES

Information on the toxicity of strontium to benthic invertebrates was not found.

18.4 TOXICITY TO PLANTS

18.4.1 Toxicity to Plants in Soil

Information on the toxicity of strontium to plants in soil was not obtained.

18.4.2 Toxicity to Plant in Solution

Information on the toxicity of strontium to plants in solution was not obtained.

18.4.3 Phytotoxic Mode of Action

Information on the phytotoxic mode of action of strontium was not obtained.

18.5 TOXICITY TO WILDLIFE

18.5.1 Toxicity to Mammals

Kroes et al. (1977) fed rats diets containing 0.75–4800 mg/kg $SrCl_2$ for 90 days and observed that behavior, growth, food intake, and food efficiency were not affected at any concentration. The only changes evident in the exposed animals were an increase in the relative thyroid weights in the males of the two highest concentration groups and a decrease in relative pituitary weights in the females of the 300- and 4800-mg/kg groups (Kroes et al. 1977). Skoryna (1981) exposed rats to 70, 147, or 263 mg Sr/kg/d in drinking water for three years. No effects were observed. Other research indicates that strontium may inhibit calcium absorption, leading to decreased calcium-binding protein production (Omdahl and de Luca 1971).

18.5.2 Toxicity to Birds

Information on the toxicity of strontium to birds was not found.

18.6 REFERENCES

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