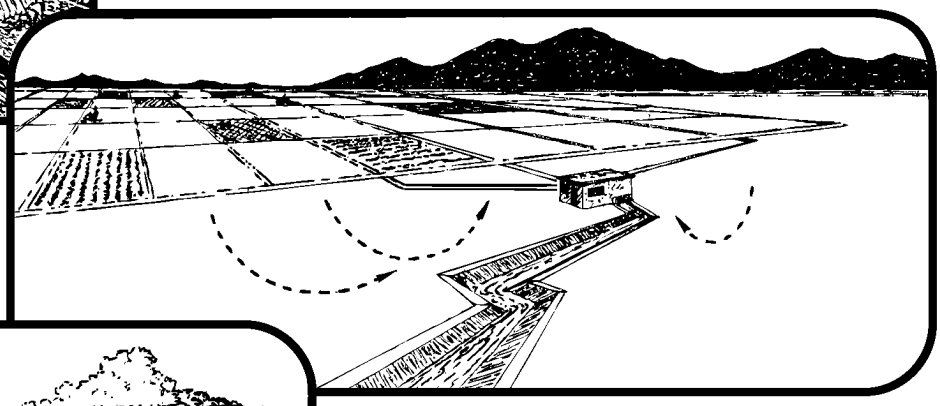
Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment



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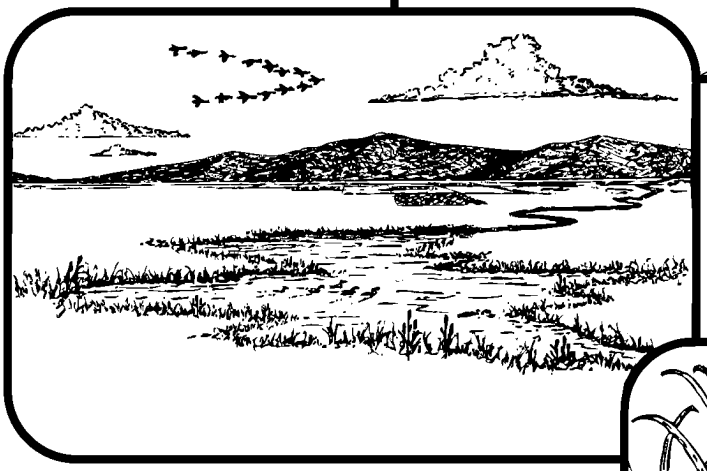
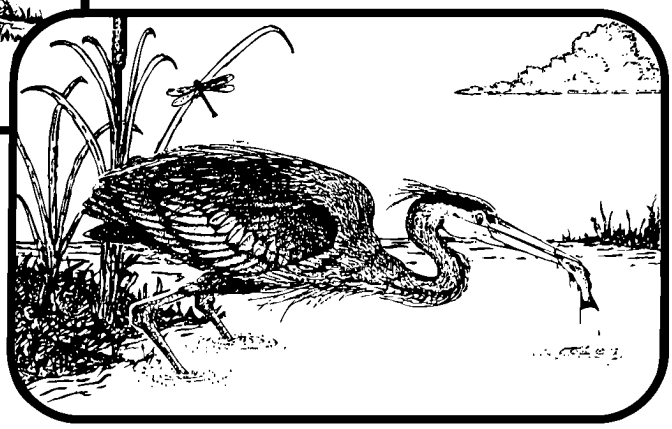


National Irrigation Water Quality Program Information Report No. 3



United States Department of the Interior
Bureau of Reclamation
Fish and Wildlife Service
Geological Survey
Bureau of Indian Affairs

UNITED STATES DEPARTMENT OF THE INTERIOR

NATIONAL IRRIGATION WATER QUALITY PROGRAM INFORMATION REPORT NO. 3

Guidelines for Interpretation of the Biological Effects of Selected Constituents in Biota, Water, and Sediment

Participating Agencies:

Bureau of Reclamation
U.S. Fish and Wildlife Service
U.S. Geological Survey
Bureau of Indian Affairs

November 1998

UNITED STATES DEPARTMENT OF THE INTERIOR

BRUCE BABBITT, Secretary

To request copies of this report or additional information, contact:

Manager, NIWQP (D-5010)
Bureau of Reclamation
P.O. Box 25007
Denver, CO 80225-0007

Or visit the NIWQP web site at:
http://www.usbr.gov/niwqp

Introduction

The guidelines, criteria, and other information in this volume were originally compiled for use by personnel conducting studies for the Department of the Interior's National Irrigation Water Quality Program (NIWQP). The purpose of these studies is to identify and address irrigation-induced water quality and contamination problems associated with any of the Department's water projects in the Western States. When NIWQP scientists submit samples of water, soil, sediment, eggs, or animal tissue for chemical analysis, they face a challenge in determining the sig-nificance of the analytical results. How much of a given chemical constituent is "normal" in the tested medium? How much is unusually high? What adverse effects— if any— may result from the reported concentration? Studies that address these questions are myriad: they are widely scattered in the literature, they use many different approaches and testing protocols, and they yield greatly varying— and sometimes contradictory— results. The chapters in this volume are intended to: (1) identify the most important, most relevant studies for several "constituents of concern" that are commonly encountered in environments affected by irrigation drainage; (2) present a sampling of notable results from these studies in tables organized according to tested medium; (3) explain further, in the accompanying text, the significance of these results; and (4) give full and accurate references to the original studies, for those who desire more detailed information.

Although this volume is targeted for scientific specialists, it may also be of interest to government officials, farmers, ranchers, conservationists, reporters, and anyone else interested in the environmental health of freshwater ecosystems. These readers may find the glossary in Appendix II especially helpful.

The Limitations of This Volume

It is important to note five limitations on the material presented here:

(1) Out of the hundreds of substances known to affect wetlands and water bodies, this volume focuses on only nine constituents or properties commonly identified during NIWQP studies in the Western United States— salinity, DDT, and the trace elements arsenic, boron, copper, mercury, molybdenum, selenium, and zinc. Financial and time restraints do not allow consideration of other contaminants at this time.

(2) For the most part, these are only guide-lines, merely reports of toxic effects that were noted for certain concentrations in particular circumstances. Individual constituents may be more or less toxic at other sites or for other species, depending on many factors. Some of these com-plicating factors are described in the following section on data interpretation, which readers are urged to review before attempting to apply these guidelines.

(3) Caution is particularly appropriate in using the summary tables (the first numbered table in each of the chapters). These are designed to give only a general indication of concentrations that may be troublesome in various types of media. In some cases the "no effect" and "threshold" values for a class of organisms have been distilled from hundreds of individual studies of the diverse species that make up the class. In other cases, we have had to rely on only a handful of studies to set *tentative* values for the entire class. Readers should make no final, formal decisions regarding the toxicity of

a particular compound to a particular species without consulting the more detailed information presented later in each chapter and, when possible, the original studies.

(4) Results from many recent studies could not be included here. Most of the research for these chapters was completed by mid-1996, and only the literature pub-lished prior to that time was system-atically surveyed. During subsequent review and preparation of this volume, more recent results that came to our attention were added opportunistically, not systematically.

(5) Legally enforceable standards are not presented here, with two exceptions. The U.S. Environmental Protection Agency has established "maximum contaminant levels," applicable only to drinking water, for most of these constituents, and the U.S. Food and Drug Administration has "action levels for human consumption" for two of them (DDT and mercury). These legal standards are noted near the end of each chapter, in the section "Regulatory standards." Note, however, that even in those sections, values identified as "goals" or "criteria" do not have the force of law.

Individual States may set legal standards that are stricter than those of the Federal Government, and many have chosen to do so. State standards are too variable and voluminous to be listed here; however, Appendix I lists addresses and phone numbers for the offices responsible for water quality standards in each of the 17 Western States.

The Need for Caution in Interpreting Toxicological Data

The contents of this report are described as *guide lines*, rather than rules or standards, because toxicological effects vary greatly in natural ecosystems. Many variables can cause individual constituents to be more or less toxic at other sites or for other species. This section describes some of the better known factors that may complicate the interpretation of toxicity data.

Unnatural Laboratory Settings

Most laboratory studies test toxicity under completely unnatural conditions: they test the effect of a single compound on a single species, delivered by only one pathway under carefully controlled conditions. In the wild, organisms are exposed to many different chemical and physical agents simultaneously. (See "Interactions," below.)

Generally, laboratory specimens in an experimentally contaminated environment are given food from outside, uncontaminated sources, whereas wild creatures must eat food that has grown in the same environment and that may have accumulated, through bioconcentration, lethal levels of whatever toxins are present. Thus, for instance, fish or waterfowl could end up dying in areas where waterborne toxin concentrations are at levels that caused no harm to laboratory specimens.

On the other hand, most laboratory specimens are taken from uncontaminated populations, which have no previous history of exposure to the toxin being tested. In the wild, organisms living in a contaminated environment may have acclimated or adapted to the toxin, especially if the contamination developed gradually. In this case, one might find fish and waterfowl thriving in areas where waterborne concentrations are at levels experimentally determined to be lethal.

Laboratory specimens are rarely threatened by predators or challenged by others of their own kind in mating competitions, whereas their undomesticated cousins deal with both conflicts. These conflicts can add to the overall stress on the organisms, making them more susceptible to toxic effects. Conversely, the higher metabolic

rates of creatures in conflict may help them dispose of toxins more readily.

These differences between natural and laboratory environments mean that measure-ments collected in natural settings are generally preferable to laboratory measure-ments for predicting toxic effects in natural systems. In cases where natural studies are lacking, though, the laboratory studies may provide the only useful guidance to possible toxic effects. Moreover, only in controlled laboratory studies can the effects of individual variables be studied, by holding all other factors constant.

Interactions

The toxicity of an element or compound may be either reinforced or weakened through its interaction with other substances. In toxi-cology studies, such interactions are generally classified as being adversely additive, syner-gistic (greater than additive), or antagonistic (less than additive or even acting as antidotes to one another). For instance, various chapters in this volume describe synergistic relationships between boron and selenium, between copper and zinc, and between DDE and Arochlor, meaning that when both agents are present, their toxic effect is greater than would be expected just from adding together their individual effects. Elsewhere, these chapters describe antagonistic relationships between arsenic and selenium and between cadmium and copper: tests show these combinations of elements to be *less* toxic than either one would be by itself. In the case of selenium and mercury, however, the selenium chapter cites a study (Heinz and Hoffman 1996) showing that these two elements are antagonistic to each other in their effect on adult mallards but synergistic in their effect on mallard reproduction.

In some cases, two substances that interact antagonistically at first may eventually become synergistic with increasing concentrations. For instance, some interactions may transform a toxic compound to a less toxic, but also less soluble, form. These low-solubility compounds may then accumulate in the liver, the kidneys, or other bodily organs, eventually overtaxing the capacity of these storage sites. Physical damage may occur to organs storing too many solids.

However, our understanding of biogeo-chemical interactions is still rudimentary. The potential combinations of trace elements are essentially infinite, and research thus far has defined the additive, antagonistic, and synergistic effects of only a few simple com-binations. Some compounds cause toxic effects by interfering in essential chemical metabolic pathways, yet different chemical species of the same two elements may interact on different metabolic pathways and produce a completely different result. Under present conditions it takes years of research— perhaps an entire career— to positively define just one or two complex metabolic chemical pathways. Many apparent discrepancies appear in the literature.

Temperature

All organisms have optimal temperature ranges in which they function most efficiently. Outside of these ranges they will be more susceptible to toxins. The DDT chapter, for instance, cites studies showing that both high and low temperatures increase the toxicity of DDT to the water flea *Daphnia*. Temperature fluctuations affect the rate of chemical reactions, the solubility of chemical species, and the metabolic rates of organisms. High temperatures generally increase the chemical reaction rate and the solubility of most solid substances. Oxygen and other gases, however, are more soluble in cold water than in warm. The effect of temperature on metabolism depends on whether organisms are exothermic

("cold blooded") or endo-thermic ("warm blooded"). Among exo-therms, such as fish and invertebrates, higher temperatures cause metabolic rates to rise. Endotherms, such as birds and mammals, increase their metabolic rate at lower temper-atures in order to maintain a constant body temperature. An elevated metabolism increases the intake of a toxin and distributes it more rapidly to sensitive organs within the body.

Water Chemistry

The effect of any toxin may be altered by variations in water hardness, pH (acidity/ alkalinity), and dissolved oxygen content. Water hardness, for instance, causes such great variation in the toxicity of copper and zinc that the Environmental Protection Agency, rather than setting fixed values as the freshwater criteria for these elements, has instead established formulas that make the criteria relative to hardness. (See tables at end of copper and zinc chapters.)

Disease

It seems likely that populations weakened by disease would be more susceptible to toxins and vice versa. According to Sprague (1985), though, the empirical evidence for this relationship is scanty. At the very least, the presence of disease in a population can complicate the task of interpreting which deaths and other adverse effects are attributable to toxins and which are due to the disease.

Nutrition

A species' susceptibility to toxins may be affected not only by a shortage of food but also by variations in the quality of the food. Organisms obliged to deviate from their customary diets may lack crucial vitamins, minerals, or proteins that play a role in detoxifying harmful compounds.

Sampling Biases

Interpretation of field data for plants and animals can be confounded by a sampling bias that favors "survivors." Most biological sampling techniques are designed to sample live biota. In contaminated environments, live biota represent "survivors" and, hence, these are likely to be the organisms that either were less sensitive to the toxin or had less exposure to it. Bird eggs are probably less affected by this bias than other media because they are sampled without regard for the status of the embryo inside the egg. So long as the egg is intact, live and dead embryos have equal probabilities of being sampled.

Off-Site Exposure

Some organisms travel considerable distances and may be exposed to toxins at places other than the site where they are collected. Many birds, for example, may feed several kilo-meters away from their nesting sites. Hence, responses such as teratogenesis among their offspring may not be attributable to contami-nation in the immediate vicinity. Although this complication is obviously most pro-nounced in the case of birds, many mammals, fish, and even insects also travel widely.

Confusion About Measurements

Chemical concentrations in plants, animals, soil, sediment, and water are measured in various ways, and there is even greater variety in the ways these measurements are expressed. Although all contributors to this volume have endeavored to clarify both the type of measurement and the units of measure for every value presented, some may remain unclear. Concentrations in any solid medium (such as

organic tissues, sediment, or animal feed) may be measured on either a dry-weight (dw) basis or a wet-weight (ww) basis. The resulting values are markedly different, and the dw value is invariably higher. In fish and animal tissues, the dw concentration is generally in the range of 3 to 5 times the ww value, but there is no set conversion factor. The ratio between dw and ww depends on the water content of the tissue, which varies between species and between organs, and even varies within individual organs over time. Criteria based on wet-weight measurements should not be used to assess the toxicity of dry-weight concentrations, and vice versa.

"Fresh weight" describes a wet-weight measurement that is made either in the field or within a few hours after collection. Media such as eggs and animal tissue may begin losing water as soon as they are collected, which results in higher wet-weight concentrations of most other constituents if they are not analyzed promptly.

Many chemical elements have two or three different valences or oxidation states that are common in the environment, and the toxicity of these varying forms can differ greatly. Arsenic (III), for instance, is much more toxic than arsenic (V), yet some tests do not differentiate between these forms and report only "total arsenic." A criterion established using arsenic (III) would be misleadingly low in most natural settings, for arsenic (V) is usually more abundant.

Even where the valence state doesn't vary, the various compounds an element makes with other elements can greatly affect toxicity. Dimethyl mercury (C_2H_6Hg), for instance, is far more poisonous than mercuric sulfide (HgS), even though both of them are based on mercury (II). It is common for organic (carbon-based) compounds to be more toxic than others because they are more readily taken up in the metabolism of living organisms.

Concentrations of elements or compounds in water may be measured in two different ways. Under one method, water samples are filtered before analysis to remove all microorganisms and other suspended particles. The resulting measurement is called a *total dissolved* concentration. In the other method, no filtering is done, and the resulting measurement is a *total recoverable* concentration. The difference between these figures can be strongly influenced by the overall biotic productivity of a water body. In highly productive waters, both nutrients and toxins are quickly taken up by microorganisms, leaving only small amounts of these dissolved in the water column. Thus, a measurement showing only dissolved constituents may miss significant amounts of toxins that are nonetheless present in the water column and available through the food chain. Where productivity is low, the dissolved concentration will be very close to the total recoverable concentration.

Many reports give chemical concentrations in either parts per million (ppm) or parts per billion (ppb). A few use the ambiguous abbreviation "ppt," which may stand for either parts per thousand or parts per trillion. Obviously, in reading such reports, it is important to know which meaning of "ppt" was intended. In accordance with principals of the International System of Units, most concentrations in this volume are expressed in units of either weight per weight (for solid media) or weight per volume (for liquids). Here is a brief list of equivalents that clarify how these units relate to one another:

Parts per thousand (ppt *or* per mil *or* ‰)	=	g/kg *or* g/L
Parts per million (ppm)	=	mg/kg *or* mg/L
Parts per billion (ppb)	=	µg/kg *or* µg/L
Parts per trillion (ppt)	=	ng/kg *or* ng/L

The relationship shown here between weight/weight measurements and weight/volume measurements comes about because 1 liter of water weighs almost exactly 1 kilogram.

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Jeff Lucero (Chairman)
Bureau of Reclamation

Jan Hren (succeeded by Herman Feltz)
U.S. Geological Survey

Steve Smith (succeeded by Tim Hall)
U.S. Fish and Wildlife Service

Frank Khattat (succeeded by Lewis Fry)
Bureau of Indian Affairs

James W. Yahnke
Bureau of Reclamation

Joe Skorupa
U.S. Fish and Wildlife Service

The nine chapters were prepared both by NIWQP participants and by an outside consultant, using the extensive library of the University of California at Davis, California. The principal individual responsible for each chapter is listed below:

Arsenic, copper, DDT, salinity, and zinc
Harry Ohlendorf
CH2M Hill, Sacramento, California

Boron and molybdenum
Judy Sefchick-Edwards
U.S. Fish and Wildlife Service, Fergus Falls, Minnesota

Mercury
Steve Schwarzbach
U.S. Fish and Wildlife Service, Sacramento, California

Selenium
Joe Skorupa
U.S. Fish and Wildlife Service, Sacramento, California

The chapters were edited by Peter L. Martin of the Bureau of Reclamation, with guidance and advice from Denise E. Larsen. Martin and Larsen also reorganized the chapters to give them a consistent organization and added some supplementary material.

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CH2M Hill
Harry Ohlendorf

U.S. Bureau of Indian Affairs
Lewis Fry

U.S. Bureau of Reclamation
Michael Armbruster
N. John Harb
Scott Irvine
John Johnson
Jerry Miller
James Setmire
Rodney TeKrony
James Yahnke

U.S. Fish and Wildlife Service
Larry Gamble
Tim Hall
Joel Lusk
Joseph Skorupa

U.S. Geological Survey
Charles Demas
Herman Feltz
Steven Hamilton
Stephen B. Smith
Doyle Stephens

References Cited

Heinz, G.H. and D.J. Hoffman. 1996. Combined effects of mercury and selenium on mallard reproduction. In: *Abstracts for 17th Annual Meeting, Society for Environmental Toxicology and Chemistry, Washington, DC, November 17–21, 1996*. p. 58.

Sprague, J.B. 1985. Factors that modify toxicity. Chapter 6 of: G.M. Rand and S.R. Petrocelli, eds. *Fundamentals of aquatic toxicology—Methods and applications*. Hemisphere Publishing Corp., Washington. p. 124–163.

Arsenic

Description

Arsenic (As) is a metalloid, with properties intermediate between those of a metal and a nonmetal. In its pure state, it generally takes the form of a dense, gray metal, although a much lighter, yellowish powder may be formed through sublimation of the vapor. In nature, arsenic exists in four oxidation states (As^{-3}, As^{0}, As^{+3} [referred to as "As (III)"], and As^{+5} ["As (V)"]), and it may be in either organic or inorganic forms. Its common ores include the minerals arsenopyrite (FeAsS) and realgar (As_2S_2). Arsenopyrite is a white to gray orthorhombic mineral resembling pyrite, commonly found in lead and silver veins. Realgar forms red to reddish-orange nodules in ore veins and similarly colored coatings around some hot springs.

Occurrence

Arsenic is ubiquitous—present in air, water, soil, plants, and other living organisms. In water, common forms of arsenic are As (III), As (V), methanearsonic acid, and dimethyl-arsinic acid (EPA 1985). Inorganic As (V) is the most common species in water. As (III) in water converts readily to As (V) under aerobic conditions (Clement and Faust 1973), but some As (III) may persist depending on microorganisms, temperature, and other factors.

Background Concentrations.—The arsenic concentration in soil normally ranges from 1 to 50 mg/kg, though it does not generally exceed 10 mg/kg (Brown et al. 1983), and in water it is normally <10 μg/L (Eisler 1988). Terrestrial flora and fauna, birds, and freshwater biota usually contain <1 mg As/kg by wet weight (ww). Arsenic at 0.27 mg/kg ww (≈1 mg/kg dry weight [dw]) is reported to be the 85th percentile concentration for freshwater fish (Schmitt and Brumbaugh 1990), and background concentrations in terrestrial plants range from 0.01 to 1.7 mg/kg dw (Bodek et al. 1988). Concentrations of arsenic in livers of adult amphibians collected in an apparently uncontaminated area averaged 0.164 mg/kg ww (Byrne et al. 1975). These levels are sometimes much higher in biota collected near areas with high geo-thermal activity and near manufacturers of arsenical defoliants and pesticides (Eisler 1988).

Each year, as a result of agricultural and industrial activities, large quantities of arsenicals that may be hazardous to fish and wildlife are released into the environment (Eisler 1988). Agricultural applications provide the largest artificial source of arsenic in the environment (Eisler 1988). It is contained in wastes from the production of certain herbicides, fungicides, insecticides, algicides, and wood preservatives (Brown et al. 1983); in particular, sodium arsenite was commonly used as an aquatic herbicide between 1940 and the 1970's, especially in the United States (Tanner and Clayton 1990). Arsenic is also present in large amounts in water contaminated by mine tailings, smelter wastes, and natural mineralization (Eisler 1988). EPA also states that sources of arsenic in drinking water include glass, electronic wastes, and orchards (EPA 1994).

Summary of Effects

Arsenic is not normally considered an essential element to most species, and it has been shown to be both teratogenic and carcinogenic in many

mammal species (Eisler 1988, 1994). However, beneficial effects have been reported in tadpoles, silkworm, rats, goats, and pigs at low dietary concentrations (Eisler 1988). Mammals with arsenic deficiencies display poor growth, reduced survival, and inhibited reproduction, whereas low doses of arsenic actually stimulate growth in plants and animals (Eisler 1994).

Arsenic's toxicity and bioavailability may vary significantly, depending on the chemical forms and routes of exposure. In general, inorganic arsenic compounds are more toxic than organic compounds, and As (III) is more toxic than As (V) (Eisler 1988, 1994). Hence, the natural conversion of As (III) to As (V), which is favored in most aquatic environ-ments (Manahan 1989), somewhat reduces the overall hazard of this element. It should be noted, though, that most dietary studies rely on only a single species of arsenic—generally inorganic—and that such studies thus do not reflect the diversity of arsenic species present in the environment. The varying effects of different arsenic compounds should be considered before using experimental data to assess the toxicity of arsenic in the environment.

In the aquatic environment, adverse effects of arsenic have been reported at a wide range of concentrations in water, sediment, and diets. Suter and Mabrey (1994) evaluated a series of toxicological benchmarks for screening various contaminants for their potential effects on aquatic biota. In addition to the national ambient water quality (NAWQ) criteria, they provided secondary acute and chronic values, lowest chronic values (including those for fish, daphnids, nondaphnid invertebrates, aquatic plants, and all organisms), test EC20s (concentrations that cause observable ill effects in 20 percent of specimens), sensitive species test EC20s, and population EC20s. These data were used to establish the general biotic effect levels presented in table 1. As listed there, "No effect" is the lowest chronic value for all organisms; "Toxicity threshold" is the NAWQ chronic criterion (if established) or the secondary chronic value; and "Level of concern" is the range between the two other values.

Field Cases

Though arsenic is ubiquitous in the environment, the incidence of wildlife poisoning by arsenic is relatively rare (Eisler 1988). Sandhu (1977) reported an intensive fish kill in a reservoir at Orangeburg, South Carolina, after aerial spraying of arsenic defoliants in a nearby cotton field. The arsenic concentration in the water was elevated to 2,500 μg/L, and catfish in the reservoir were reported to contain 5 and 12 mg As/kg in skeletal muscle after 5-hour and 7-week exposures (weight basis not specified).

Arsenic is also relatively persistent in the aquatic environment. Tanner and Clayton (1990) reported elevated concentrations of arsenic in macrophytes (193–1,200 mg/kg dw) and surficial sediments (540–780 mg/kg dw) in Lake Rotoroa, New Zealand, 24 years after an application of sodium arsenite herbicide; arsenic levels in a nearby reference lake (Lake Rotokauri) were <20 mg/kg dw in macro-phytes and 16.5–40 mg/kg dw in sediments. (Note, however, that the "reference lake" had arsenic concentrations in the sediments that are in the middle of the levels of concern in table 1, and the detection limit for the macro-phyte datum was four times the toxicity threshold for plants in table 1. Alternatively, the "living" macrophytes had arsenic con-centrations of between 39 and 240 times the toxicity threshold and are obviously tolerant species.)

Natural sources, such as hot springs and volcanic activity, also contribute to elevated levels of arsenic in the environment. Lacayo et al. (1992) determined arsenic levels in water, fish, and sediments from Xolotlán, Managua, Nicaragua, a lake which contained high levels of arsenic from such sources.

Table 1.—Summary of comprehensive biotic effects of arsenic
[See Appendix II for explanation of abbreviations and technical terms]

Medium	No effect	Level of concern	Toxicity threshold	Comments/Explanation
Water (µg/L)	48	48–190	190	48 µg/L is lowest chronic value for As (V) in aquatic plants; 190 µg/L is NAWQ chronic criterion for As (III). See Suter and Mabrey (1994).
Sediment (mg/kg dw)	8.2	8.2–70	70	"ERL" and "ERM" values of Long et al. 1995.
Plants (mg/kg dw)	1–1.7	2–5	5	Levels in plants (Kabata-Pendias and Pendias 1992) and invertebrates (see table 2) not well established, but at least some show no effects below these tissue concentrations.
Invertebrates (mg/kg dw)	30	30–50	50	
Fish (mg/kg dw)	1.0	1–12	12	No-effect level is 85th percentile concentration from Schmitt and Brumbaugh (1990). Toxicity threshold from Sandhu (1977).
Bird eggs (mg/kg dw)	1.3	1.3–2.8	<2.8	J.P. Skorupa, unpub. data, 1996.
Amphibians/reptiles	—	—	—	Diagnostic effect levels not available. Mammals, in particular are poor biomonitors for As (Talmage and Walton 1991).
Mammals	—	—	—	

Note: Although diagnostic levels for biota concentrations are generally not well defined, arsenic concentrations in biota are usually <1 mg/kg fresh weight except near sources of arsenic pollution (Eisler 1988, 1994). (Dry-weight concentrations, such as those shown above, are generally several times higher than fresh-weight concentrations, although no reliable conversion factor can be defined.)

In Texas, Clark et al. (in press) reported what they believed to be the highest concentrations of arsenic found in tadpoles (6.87 mg As/kg ww). Their report provides a good review of information concerning arsenic (as well as chromium and zinc) in amphibians and reptiles. Tadpoles were collected in 1994 from areas immediately downstream from Finfeather Lake, which had been directly contaminated during 53 years of industrial production of arsenic-based cotton defoliants. No tadpoles were found in Finfeather Lake, probably because arsenic, chromium, or zinc concentrations there were still toxic, even though contaminated sediments had been removed about 10 years earlier.

Dead and blind turtles (red-eared slider, *Trachemys scripta*, and common snapper, *Chelydra serpentina*) were found at Finfeather Lake in 1973, when waterborne arsenic concentrations in the lake averaged 7.9 milligrams per liter (mg/L) (Cearley 1973). The turtles showed symptoms similar to those of arsenic-poisoned domestic mammals. These included keratinization (leathery appearance) of the eyelids, nasal areas, and roof of the mouth. The nasal passages of one turtle were completely occluded with the keratinized tissue, forcing the turtle to breathe through its mouth. Clark et al. (in press) observed no turtles or snakes in Finfeather Lake in 1994 or 1995, leading them to speculate that few or none were present.

Fish populations in Finfeather Lake also were affected (Cearley 1973, Sorensen et al. 1985). Green sunfish (*Lepomis cyanellus*) in the system exhibited liver pathology related to arsenic. In 1991, Cantu et al. (1991) found that

large-mouth bass (*Micropterus salmoides*) from Finfeather Lake had deformed fins, jaws, heads, and eyes; waterborne arsenic concen-trations at the time were 0.54 mg/L.

Abiotic Factors Affecting Bioavailability

Water

Many factors influence arsenic toxicity in water, including water temperature, pH, organic content, phosphate concentration, suspended soils, the presence of other substances and oxidants, and arsenic speciation. A study by McGeachy and Dixon (1990) confirmed that more arsenic is taken up as the water temperature increases.

Sediment

Higher levels of arsenic in sediment were correlated with levels in macrophytes in a study done by Tanner and Clayton (1990), but other studies (Cain et al. 1992, Smith et al. 1992) reported low bioavailability and little partitioning of arsenic from contaminated sediments. Long and Morgan (1990) and Long et al. (1995) made a comprehensive evaluation of chemical concentrations in sediments that were associated with adverse biological effects. They concluded that arsenic concentrations of 8.2 mg/kg dw or less do not usually produce adverse effects, but concen-trations of 70 mg/kg or higher usually do. Although many of the data evaluated were for estuarine and marine sediments, Hull and Suter (1994) concluded that those screening levels also were appropriate for freshwater sediments until more specific guidelines become available. However, it is also recommended that these concentrations be compared to local background levels when possible.

Biotic Effects

Tables 2, 3, and 4 at the end of this chapter list the reported biotic effects of arsenic in water, sediment, and diet, respectively.

Plants

Arsenic is not an essential element in plants (Kabata-Pendias and Pendias 1992), although small increases in yield have been observed for several species at low levels of soil arsenic (Woolson 1975). Some forms of arsenic, such as sodium arsenate and arsenic trioxide, are extremely toxic to plants. Arsenic uptake seems to be passive (Bodek et al. 1988) from terrestrial soil to plants. The major symptoms of arsenic toxicity in plants are red-brown necrotic spots on old leaves, yellowing or browning of the roots, wilting of new leaves, and depressed tillering (Kabata-Pendias and Pendias 1992). Sensitive species such as spinach (*Spinacia oleracea*) showed 40-percent reduction in growth when exposed to As (V) at 10 mg/kg in soil (table 3). Low concen-trations of As (V) in water (1–15.2 µg/L) have been reported to inhibit certain aquatic plants, resulting in noticeable changes throughout the ecosystem. Sanders and Cibik (1985) have reported consequent changes in the composi-tion and succession of species and in predator-prey relations in chronic studies.

Amphibians/Reptiles

Very few studies have investigated the effects of arsenic on amphibians and reptiles. Khangarot et al. (1985) determined the acute toxicity of As (III) to tadpoles (*Rana hexadactyla*). Under the conditions of pH 6.1, temperature 15 °C, and total hardness 20 mg/L (calcium carbonate), they found that a concentration of 249 µg As/L

would kill 50 percent of specimens in 4 days (96-h LC50). Average arsenic concentrations in the livers of adult frogs and toads were 0.164 mg/kg ww at an uncontaminated area (Hall and Mulhern 1984). This value was considerably lower than the levels of arsenic in many other freshwater animals (Wagemann et al. 1978).

Birds

There are great differences in tolerance to arsenic among bird species. As shown in table 4, female mallard (*Anas platyrhynchos*) ducklings showed a reduced growth rate when they were fed 30 mg As (V)/kg dw over 10 weeks (Camardese et al. 1990). In adult mallards, arsenic toxicity from sodium arsenate in the diet was significant at 400 mg/kg dw (Stanley et al. 1994). Other sensitive species, such as the brown-headed cowbird (*Molothrus ater*), showed 50-percent mortality in 11 days when fed copper acetoarsenite at 99.8 mg/kg dw (table 4). Opresko et al. (1994) estimated the no-observed-adverse-effect levels (NOAEL) for dietary concentrations of arsenic in several species of aquatic and terrestrial birds. The belted kingfisher (*Ceryle alcyon*) and great blue heron (*Ardea herodias*) are the most relevant species for aquatic habitats. For those two species, the dietary NOAELs were 19 to 22 mg/kg ww when based on sodium arsenite in the diet and 3.4 to 3.9 mg/kg ww when based on copper acetoarsenite (Paris green).

Stanley et al. (1994) found that adult mallards fed arsenic as sodium arsenate showed reduced weight gain, reduced liver weight, delayed egg laying, reduced egg weight, and eggshell thinning. Adult mallards exposed to dietary concentrations of 300 mg As/kg (dw) as sodium arsenate rapidly accumulated the compound but also rapidly eliminated it; the compound had a half-life of 1 to 3 days after removal from the diet and reached equili-brium levels in 10 to 30 days (Pendleton et al. 1995). The greatest accumulation of arsenic was in the liver, and lower levels were found in the blood and brain. Arsenic also reduced the growth and the body and liver weights in mallard ducklings (Stanley et al. 1994).

Some studies indicate that arsenic is extremely toxic to avian eggs when injected (Birge and Roberts 1976, Gilani and Alibhai 1990). However, elevated levels of arsenic rarely occur naturally in eggs, even in those collected at agricultural drainwater evaporation ponds where arsenic was present at high concentra-tions. Among 81 eggs collected during 1987–89 in the San Joaquin Valley of California, only one contained arsenic above the detection limit of 0.4 mg/kg dw (Ohlendorf et al. 1993). Libby et al. (1953) found that domestic poultry fed a diet containing high levels of arsenic (arsanilic acid at 180 mg/kg dw) nevertheless produced eggs that contained an average of only 1.3 mg As/kg and showed normal embryo viability. Many studies have shown that arsenic actually stimulates growth and egg pro-ductivity in poultry. Stute and Vogt (1968) fed 3-nitro-4-hydroxyphenylarsonic acid to hens at 50 mg/kg dw and observed a 4-per-cent increase in egg production.

Mammals

Although arsenic is officially classified as a human carcinogen (EPA 1995), there is little evidence that it is carcinogenic to other mammals (Eisler 1988). It does, however, cause teratogenic effects in many species. Mammals are exposed to arsenic mainly by the ingestion of contaminated vegetation and water. Adverse effects were noted in rats at dietary levels of 20 mg/kg dw (table 4). Acute or subacute arsenic poisoning is much more common than chronic poisoning in mammals (National Academy of Sciences 1977). The probability of chronic arsenic exposure is rare because detoxification and excretion are rapid (Woolson 1975). As various studies have noted (see review by Talmage and Walton 1991), mammals normally are not good biomonitors for arsenic in the

environment. Sharma and Shupe (1977), for instance, observed no relationship between arsenic concentrations in soil and vegetation and those in the liver of ground squirrels.

Bioaccumulation

Waterborne arsenic is known to accumulate to high concentrations in some species (table 2). The accumulated arsenic concentrations in stoneflies, snails, and Daphnia were as much as 131, 99, and 219 times, respectively, the water concentration according to a study by Spehar et al. (1980), whereas rainbow trout and amphipods showed no sign of bioaccumulation. Though the bioaccumulation of arsenic from the water has been well documented, there is no evidence of magnification along the aquatic food chain (Eisler 1988).

Arsenic has been found to accumulate in the lipid fractions of marine plants, invertebrates, and higher organisms (Eisler 1994). Marine biota, in particular, contain unusually high levels of arsenic in their lipids because of their ability to accumulate the element from both seawater and food sources. For mallards, Stanley et al. (1994) found that arsenic accumulated in both adult and duckling livers and in whole eggs (table 4). Pendleton et al. (1995) found that arsenic (as sodium arsenate) accumulated in all tissues but was also rapidly eliminated when birds were switched to an uncontaminated diet.

In order to evaluate the cumulative toxicity of arsenic and various metals (Cd, Cu, Hg, Pb) along the food chain, Yannai et al. (1979) raised a large quantity of algae (*Micractinium* and *Chlorella*) on metal-rich waste water, fed the algae to chickens and carp, and then fed the meat of these chickens and carp to rats. They found that bioaccumulation did not increase the levels of any of these metals in chickens or carp except for chickens' livers (which contained higher arsenic than the livers of control chickens), and they observed no change in the general appearance, behavior, and survival of the rats that ate the chicken and carp meat. They concluded that such meat would pose no hazard to consumers.

Interactions

An antagonistic interaction between arsenic and selenium is found in several animal species, including rats, dogs, swine, cattle, and poultry, and it is best documented for non-domestic birds in a study done by Stanley et al. (1994). According to the study, "As reduced Se accumulation in liver and egg, and alleviated the effects of Se on hatching success and embryo deformities" in mallards. However, exposure to As and Se at contaminated sites may not be in the chemical forms administered in that study, and exposure levels, especially for As, may be lower than those administered. Thus, the interactions observed may not occur under natural conditions and, therefore, may not be an important consideration in the management of contaminated sites.

Regulatory Standards

Standards and criteria established by the U.S. Environmental Protection agency are listed in table 5. For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments

[dw, dry weight; ww, wet weight. See Appendix II for explanation of other abbreviations and technical terms]

Species	As compound	Concentration in water (µg/L)	Effects	Reference
Aquatic plants				
Algae, various species	As (V)	75	Decreased growth	Eisler 1988
Alga (*Ankistrodesmus falcatus*)	As (V)	260	14-day EC50; inhibited growth	
Alga (*Scenedesmus obliquus*)	As (V)	48	14-day EC50; inhibited growth	
Alga (*Selenastrum capricornutum*)	As (V)	690	4-day EC50; inhibited growth	
Stoneworts (*Chara corallina*)	Total As	<10	As in biomass 340–400 mg/kg (dw)	Tanner and Clayton 1990
Aquatic invertebrates				
Amphipod (*Gammarus pseudolimnaeus*)	As (III)	88	28-day LC20	Eisler 1988
		1,000	Significant reduction in survival after 7 days	Spehar et al. 1980
	Disodium methyl-arsenate	85	28-day LC10	Eisler 1988
	Sodium dimethyl-arsenate	850	28-day LC0	
Cladoceran (*Daphnia magna*)	As (III)	600–1,320	MATC[1]	Eisler 1988
		960	28-day LC5	
	As (V)	520	Reproductive impairment of 16% in 3 weeks (at pH 7.74)	Eisler 1988, SJVDP 1990
		930	28-day LC5. Maximum BCF of 219	Eisler 1988
	Total As	1,000	18% decrease in body weight in 3 weeks	Eisler 1988
		1,400	50% reproductive impairment in 3 weeks	
		2,800	21-day LC50	
Cladoceran (*Daphnia pulex*)	As (III)	1,300	96-h LC50	Eisler 1988
Midge larvae (*Chironomus tentans*)	As (III)	680	48-h LC50	Khangarot and Ray 1989
		1,310	24-h LC50	

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments—Continued

Species	As compound	Concentration in water (µg/L)	Effects	Reference
Aquatic invertebrates—Continued				
Snail (*Helisoma campanulata*)	As (III)	960	28-day LC10. As in biomass 80 mg/kg dw. Maximum BCF 83	Eisler 1988, Spehar et al. 1980
	As (V)	970	28-day LC0. Maximum BCF 99	
Stonefly (*Pteronarcys california*)	As (III)	960	28-day LC0	Eisler 1988
Stonefly (*Pteronarcys dorsata*)	Total As	1,000	No effect. As in biomass 29–44 mg/kg (dw); BCF 33–45 in 28 d	Spehar et al. 1980
	As (V)	89	No effect. As in biomass 12 mg/kg (dw); BCF 131 in 28 d	
Zooplankton	As (III)	400	No effect	Eisler 1988
Fish				
Arctic grayling (*Thymallus arcticus*)	As (III)	13,700	96-h LC50 for juvenile	Buhl and Hamilton 1991
		27,700	96-h LC50 for alevin	
Black crappie (*Pomoxis nigromaculatus*)	Total As	22,400–114,800 (mean=49,000)	As 0.14–2.04 mg/kg (ww) 2.9–41.6 bioaccumulation ratio	Foley et al. 1978
Brown bullhead (*Ameiurus nebulosus*)	Total As	<10	As = 0.9 mg/kg (ww) in flesh	Tanner and Clayton 1990
Chinook salmon fry (*Oncorhynchus tshawytscha*)	As (III)	21,400	96-h LC50. Mean weight 1.99 g	Hamilton and Buhl 1990
		25,100	96-h LC50. Mean weight 0.5 g	
		56,500	24-h LC50. Mean weight 1.99 g	
		59,600	24-h LC50. Mean weight 0.5 g	
	As (V)	66,500	96-h LC50. Mean weight 1.99 g	
		78,000	24-h LC50. Mean weight 1.99 g	
		90,000	96-h LC50. Mean weight 1.99 g	
		167,000	24-h LC50. Mean weight 0.5 g	
Midas cichlid (*Cichlasoma citrinellum*)	Total As	10–30	No effect. As in fish muscle <0.01–0.37 mg/kg (ww)	Lacayo et al. 1992
Jaguar guapote (*Cichlasoma managuense*)			No effect. As in fish muscle <0.01–0.24 mg/kg (ww)	
Coho salmon (*Oncorhynchus kisutch*)	As (III)	18,500	96-h LC50 for juveniles	Buhl and Hamilton 1991
		49,400	96-h LC50 for alevins	
Eel (*Angulia australis*)	Total As	<10	As = 0.4 mg/kg (ww) in flesh	Tanner and Clayton 1990

Table 2.—Biological effects of various waterborne arsenicals on selected species in aquatic environments—Continued

Species	As compound	Concentration in water (µg/L)	Effects		Reference
Fish—Continued					
Pallas (*Notopterus notopterus*)	As (III)	30,930	96-h LC50		Gosh and Chakrabarti 1990
		40,000	50% mortality in 43 h		
Perch (*Perca fluviatilis*)	Total As	<10	As = 0.3–0.5 in flesh; 0.2 in scales (mg/kg, ww)		Tanner and Clayton 1990
Rainbow trout (*Oncorhynchus mykiss*)	As (III)	960	28-day LC0; no bioaccumulation		Buhl and Hamilton 1991, Spehar et al. 1980
		16,000	96-h LC50 for juveniles		
		91,000	96-h LC50 for alevins		
	Sodium arsenate	18,000	8% mortality, whole-body As 2–3 mg/kg ww after 11 weeks at 15°C		McGeachy and Dixon 1990
		36,000	34% mortality, whole-body As 2–3 mg/kg ww after 11 weeks at 5°C		
Rainbow trout larvae	As (III)	42	1% mortality (in moderately hard water of pH 6.9–7.8)		SJVDP 1990
Rudd (*Scardinius erythrophthalmus*)	Total As	<10	As (mg/kg, ww) <0.2 in flesh; 0.3 in scales; 5.5 in gut contents		Tanner and Clayton 1990
Birds					
Shag (*Phalacrocorax* sp.)	Total As	<10	As <0.2 mg/kg (ww) in flesh, liver, and brain		Tanner and Clayton 1990
Amphibians					
Frog (*Rana hexadactyla*) tadpoles	As (III)	249	96-h LC50	Conditions: 15°C, pH 6.1, hardness 20 mg/kg (as $CaCO_3$)	SJVDP 1990
		270	48-h LC50		
		368	24-h LC50		

[1] Maximum acceptable toxicant concentration. Lower value in the range shown indicates highest concentration tested producing no measurable effect on growth, survival, reproduction, or metabolism during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

Table 3.—Biological effects of concentrations of various arsenicals in sediment

[Concentrations in milligrams per kilogram; dw, dry weight; ww, wet weight]

Species	As compound	Concentration in sediment	Concentration in biomass and other effects	Reference
Plants				
Mixed submerged macrophytes	Total As	3.6–5.0 (ww)	2.3–26 (ww)	Tanner and Clayton 1990
		19–38 (ww)	5.7–7.9 (ww)	
		20–105 (ww)	66–80 (ww)	
Stoneworts (*Nitella hookeri*)	Total As	100–780 (dw)	2,400–1,128 (dw)	
Stoneworts (*Chara corallina*)		<0.01 (dw)	200–240 (dw)	
		100–780 (dw)	235–300 (dw)	
Mixed submerged macrophytes (contaminated by mine and industrial effluent)	Total As	40–3,500 (ww)	250–920 (ww)	
		6.3–3,300 (ww)	150–3,700 (ww)	
Spinach plants (*Spinacia oleracea*)	As (V)	10 (in soil)	40% growth reduction	Woolson 1973
Fish				
Midas cichlid (*Cichlasoma citrinellum*)	Total As	5.37–8.65 (dw)	<0.01–0.37 (ww) in muscle. No effect	Lacayo et al. 1992
Jaguar guapote (*Cichlasoma managuense*)			<0.01–0.12 (ww) in muscle. No effect	
Rudd (*Scardinius erythrophthalmus*)	Total As	100–780 (dw)	<0.2 in flesh; 0.3 in scales; 5.5 in gut contents (ww)	Tanner and Clayton 1990
Perch (*Perca fluviatilis*)			0.3–0.5 in flesh; 0.2 in scales (ww)	
Catfish (*Ameiurus nebulosus*)			0.9 in flesh (ww)	
Eel (*Angulia australis*)			0.4 in flesh (ww)	
Birds				
Shag (*Phalacrocorax* sp.)	Total As	100–780 (dw)	<0.2 in flesh; <0.2 in liver; <0.2 in brain (ww)	Tanner and Clayton 1990

Table 4.—Biological effects of arsenicals in the diet on selected species

[LC50, median lethal concentration—50% mortality after a stated time interval. Similarly, LC100 denotes 100% mortality; dw, dry weight]

Species	As compound	Concentration in diet (mg/kg dw)	As concentration in biomass and other effects	Reference
Fish				
Rainbow trout (*Oncorhynchus mykiss*)	As (V)	10	No effect	Eisler 1988
		90	Some adaptation to dietary As observed, as initial negative growth gave way to slow positive growth over time	
	Sodium arsenite	30	Reduced weight gains after 8 weeks	SJVDP 1990
Birds				
Brown-headed cowbird (*Molothrus ater*)	Copper aceto-arsenite	11	1.7 mg/kg dw (maximum whole-body concentration). All survived after 6 months	Eisler 1988
		33	6.6 mg/kg dw (whole body). All survived after 6 months	
		99.8	11-day LC50	
		100	3-month LC100. Brain 6.1 mg/kg dw; liver 40.6 mg/kg dw	
Mallards (*Anas platyrhynchos*)	Sodium arsenate	25	Adult liver 0.49, duckling liver 0.65 mg/kg dw. No significant differences in body weight or growth rate in ducklings, compared to controls fed 0.26 mg/kg dw	Stanley et al. 1994
		100	Adult liver 2.4, duckling liver 4.5 mg/kg dw. Reduced body weight and lower growth rate at 14 days in ducklings from parents fed As. Antagonistic interactions observed between As and Se	Stanley et al. 1994
		200	Duckling liver 5.1 mg/kg ww. Increased mortality, decreased growth, and liver histopathology in ducklings fed a low-protein (7%) diet. As reduced effects of Se when fed together in a diet with adequate protein (22%)	Hoffman et al. 1992
		400	Adult liver 6.6, duckling liver 33 mg/kg dw. Arsenic accumulated in adult liver and egg, reduced adult weight gain and liver weight, delayed onset of egg laying, decreased whole egg weight, and caused eggshell thinning. Reduced body weight, growth, and liver weight in ducklings. Antagonistic interactions observed between As and Se	Stanley et al. 1994
One-day-old mallard ducklings (female)	As (V)	300	Brain 0.8 mg/kg dw; liver 1.3 mg/kg dw. Reduced growth rate, and standing and bathing time; increased resting time over 10 weeks	Camardese et al. 1990
		30	Reduced growth rate over 10 weeks; no significant bioaccumulation in brain or liver when compared to controls	

Table 4.—Biological effects of arsenicals in the diet on selected species—Continued

Species	As compound	Concentration in diet (mg/kg)	Concentration in biomass and other effects	Reference
Birds—Continued				
Chicken	Arsanilic acid	180	1.3 mg/kg in eggs; embryo viability normal	Libby et al. 1953
		>1,000	Depressed growth	Abbott et al. 1959
		2,000	Mortality increased 33%	
		2,250	Mortality increased 40%	
Mammals				
Domestic sheep	Total As	58	No outwardly visible effect. Tissue As increased after 3-week exposure, then declined rapidly after return to low-As diet	Eisler 1988
Mice	As (III)	0.46	No significant difference in growth and survival	Schroeder and Balassa 1967
Rats	As (III)+(V)	5	No significant differences in growth and survival	Sharpless and Metzger 1940
		20	Growth decreased by 50%	

Table 5.—U.S. Environmental Protection Agency standards and criteria for arsenic

[See Appendix II for explanation of terms. Sources: EPA, 1985, 1995]

Status	Known carcinogen; EPA priority pollutant
Drinking water MCL[1]	50 µg/L
Freshwater criteria (AS-III)	360 µg/L for acute exposure 190 µg/L for chronic exposure
Freshwater LOAEL[2] (As-V)	850 µg/L for acute exposure
1/10,000 cancer risk	2 µg/L
1/1,000,000 cancer risk	0.018 µg/L (water and organisms) 0.14 µg/L (organisms only)

[1] Maximum contaminant level
[2] Lowest-observed-adverse effect level.

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Boron

Description

Boron (B) is a metalloid, with properties intermediate between those of carbon and aluminum. Like aluminum, it has an oxidation state of +3 in all of its chemical compounds, and it is an electrical conductor in its pure form. Like carbon, though, it can sometimes form complex chains and rings, and its crystalline form is nearly as hard as diamond. Boron has an atomic number of 5 and an atomic weight of 10.81. It melts at 2,180°C. Boron is found as a hard black solid and as an amorphous blackish-brown powder, although the more common boron salts are generally white or pale shades of yellow, blue, green, or gray. (Pais and Jones 1997.)

Occurrence

Boron is widespread in the environment but generally occurs in low concentrations; it constitutes only 3 mg/kg of the Earth's crust and occurs naturally only in combined form, usually as borax ($Na_2B_4O_7 \cdot 10H_2O$), colemanite ($Ca_2B_6O_{11} \cdot 5H_2O$), boronatrocalcite ($CaB_4O_7NaBO_2 \cdot 8H_2O$), or boracite ($Mg_7Cl_2B_{16}O_{30}$) (EPA 1975; NAS 1980). Areas with the highest natural inputs of boron to the environment are the Mojave Desert, California, the plateau of the Alpine-Himalayan system, and the high plateau of the Andes (Butterwick et al. 1989). The brines of Borax and Mono Lakes in California are rich in boron due to arid conditions and high evaporative concentration (Livingstone 1963). Boron compounds usually are degraded or transformed to boric acid and borates, which are the main boron compounds of ecological significance (Sprague 1972).

In natural freshwater ecosystems, surface water concentrations of boron rarely exceed 1 mg/L and are usually less than 0.1 mg/L; however, in systems where boron has been mobilized by human activities, the concentrations may be much higher (Maier and Knight 1991). In a survey of 1,546 river- and lake-water samples from throughout the United States, the mean concentration of boron was 0.1 mg/L, with 5.0 mg/L being the maximum (Powell et al. 1997). Groundwater boron concentrations are usually <0.5 mg/L worldwide; in the United States, concentra-tions can be as high as 5 mg/L in ground-water. Aquatic fauna can usually tolerate up to 10 mg B/L in water for extended periods of time without adverse effects (Eisler 1990). Recently, South Africa has developed a water-quality criterion of 1 mg B/L to protect aquatic ecosystems (including terrestrial animals that use them). Recognizing that boron sensitivity of plants is greater than that of animals, South Africa's water-quality criterion was based on calculation of a "final plant value" (Roux et al. 1996).

Boron concentrations in U.S. irrigation water typically range from <0.1 to 0.3 mg/L (Adriano 1986). Some irrigation water (especially pumped groundwater) used in the western San Joaquin Valley, California, contain far greater concentrations (Shelton and Miller 1988); boron concentrations in the San Luis Drain and Kesterson Reservoir were 11–18 and 13–65 mg/L, respectively (USBR 1986). Agricultural drain water contaminated with boron is considered potentially harmful to waterfowl and other wildlife populations throughout areas of the Western United States (Smith and Anders 1989).

Boron compounds enter the North American environment at an estimated rate of 32,000 tons annually, primarily from laundry products, irrigation drain water, fertilizers and other agricultural chemicals, coal combustion, and mining and processing (Eisler 1990). Boron compounds also are used as fire retardants and leather-tanning compounds and have even been used in rocket fuels. Elemental boron is frequently used for neutron absorption in nuclear reactors, and sodium borohydride is used by the pulp and paper industry in the production of the whitening agent sodium dithionite (Thurston et al. 1979). The United States supplies about 70 percent of the global boron demand.

Summary of Effects

Plants in general are far more sensitive than animals to boron toxicity, and there is a large literature base documenting boron's effects on plants, especially crop plants. The exact mechanism of boron toxicity in animals is not fully understood. It is not known whether boric acid, the borate ion, or some other boron complex is the toxic boron compound (Maier and Knight 1991). Boric acid and the borate ion exhibit remarkable stability in natural aquatic systems, and any boron that is not taken up by plants and/or animals will tend to accumulate and remain bioavailable over extended periods of time (Perry et al. 1994).

Considering the paucity of data on boron toxicity, effect levels can be predicted only tentatively at this time. These tentative predictions are listed in table 6.

Study Approaches

The majority of papers reviewed for this report were laboratory studies dealing with boron effects on plants and birds. Most of the plant literature concerned toxicity or

Table 6.—Predicted boron effect levels

Medium	No effect	Level of concern	Toxicity threshold	Explanation
Water (mg/L)	0.5	0.5–10	10	For crops and aquatic plants (Perry et al. 1994)
	6	6–13	13	For aquatic invertebrates (NOAEL and LOAEL for *Daphnia magna*)
	5	5–25	25	For fish (*viz*., catfish and trout embryos; Birge and Black 1977; Perry et al. 1994)
			<200	For amphibians (LC100 for leopard frog embryos)
Bird eggs (mg/kg fw)	13	13–20	20	Smith and Anders (1989), Stanley et al. (1996); 20 = EC10 for viability of mallard eggs
Waterfowl diet (mg/kg)		>30		LOAEL for mallards; impaired growth of ducklings
Mammal diet (mg/kg bw/day)		>80		LOAEL for rodents; decreased fetal body weight

deficiency in crops and is not comprehen-sively summarized in this report. The crop literature has been summarized compre-hensively by Eaton (1935) and most recently by Perry et al. (1994). Avian literature consisted mostly of studies done on mallards from the late 1980s to early 1990s. Some poultry literature was reviewed, but the bulk of this literature may have been missed since electronic literature retrievals do not date back further than the 1960s. Mammalian studies consisted mostly of laboratory studies done on rats, although some information was available for mice, rabbits, and other species. Available aquatic toxicity data for boron are limited. For aquatic species, the literature was composed primarily of freshwater laboratory studies. Fish, herptile, and invertebrate information was limited or lacking. The published scientific "white" literature was reviewed adequately, but the scientific "gray" literature, which includes government reports and unpublished data, was not.

Abiotic Factors Affecting Bioavailability

Water

The predominant species of boron in most freshwater systems (pH<9) is undissociated boric acid (Hem 1970; Maier and Knight 1991); the chemical form of boron found in water is dictated by pH and other constituents (Sprague 1972). Boron compounds are water soluble and tend to accumulate in aquatic ecosystems (EPA 1975).

Soil

In the United States, soil usually contains around 30 mg B/kg, dry weight (dw) (range 10–300 mg/kg). The precipitation:evaporation ratio of an area is a key factor in determining the degree to which boron can concentrate in soils and reach toxic levels (Butterwick et al. 1989). The total boron content of soil is of little value for diagnosing boron status; experi-mental work by Gupta (1968) suggests that less than 5 percent of the soil boron is avail-able for plant uptake (Butterwick et al. 1989).

In soils, boron may be found in four forms: organically bound, water-soluble, adsorbed, and fixed in clay and mineral lattices (Adriano 1986). Arid, saline soils generally contain the highest boron concentrations. In sandy soils, boron is leached more readily than in clay soils and is thus less likely to accumulate to toxic concentrations (Adriano 1986). Boron can react and bind with clays, suspended matter, and sediments of aquatic systems. Boron adsorbed onto clays accounts for a major proportion of the boron in many aquatic systems (Maier and Knight 1991).

Biotic Effects

Plants

The environmental effects of boron are most noticeable in plants (Sprague 1972). Boron is an essential trace element for the growth and development of higher plants, for it plays important roles in the calcium cycle and in respiratory processes and the utilization of carbohydrates (Browning 1969). However, the range between insufficiency and excess is usually narrow. Gupta et al. (1985), for instance, found that some plants show signs of deficiency when boron concentrations in soil solution are <2 mg/L and show toxic effects at concentrations >5 mg/L. Other researchers report similarly narrow ranges of boron tolerance (Sprague 1972; Weir and Fisher 1972; Birge and Black 1977; Goldbach and Amberger 1986). The waterweed *Elodea canadensis* is sensitive to even very low ambient concentra-tions of boron; Perry et al. (1994) reported that it showed a reduced rate of photosynthesis in water containing 1 mg B/L (28-day exposure). In addition, *Hydrocotyle umbellata*, commonly found in the Southeastern United States, exhibited reduced growth and yellowing of the

leaves when exposed to <1 mg B/L (Powell et al. 1997). A recent ecological risk assessment for a natural community of aquatic plants concluded that, at median spring and fall concentrations of 5.9 and 3.6 mg B/L, patterns of leaf tissue discoloration (yellowing) may indicate adverse ecological impacts on the vegetation (Powell et al. 1997).

Several factors affect plant uptake of boron, including soil texture, pH, macronutrients, temperature, light, evapotranspiration rate, and plant growth stage (Butterwick et al. 1989; Glandon and McNabb 1978). Frick (1985), for instance, found that a concentration of 20 mg B/L was sufficient to inhibit the growth of duckweed at pH 7.0 but that 100 mg B/L was required to produce the same effect at pH 5.0. Once boron is incorporated into plant tissues, it becomes relatively immobile. Leaves generally accumulate the greatest concentra-tions of boron in plants (Gupta et al. 1985).

Plant species do not all draw on the same boron supplies. Emergent plants absorb most of their boron from the hydrosoil; floating-leaf species absorb a large proportion of boron from sediments and water. Submerged plants, which lack or have greatly reduced root systems, obtain most of their boron from the water (Hutchinson 1975). Generally, floating-leaf species contain more boron than submerged or emergent plants, and dicotyledons usually contain more boron than monocotyledons (Boyd and Walley 1972; Cowgill 1974). In aquatic macrophytes, boron concentrations are usually less than 20 mg/kg dw. Based on samples from 22 species of aquatic macrophytes collected from natural environments, the mean tissue level of boron was 11.3 mg/kg dw (Powell et al. 1997). In green algae (Maeso et al. 1985) and blue-green algae (Martinez et al. 1986), adverse sublethal effects are apparent at boron concentrations of 50 mg/kg and higher.

Boron-contaminated irrigation water is one of the main causes of boron toxicity to plants. Evapotranspiration from irrigated fields concentrates boron in the soil and leads, eventually, to toxicity (Gupta et al. 1985). At some places in the Southwestern United States, naturally elevated boron concentrations in surface water used for irrigation are high enough to be toxic to plants of commercial importance (Benson et al. 1984). High con-centrations of boron were found in aquatic plants growing in irrigation drain water at Kesterson Reservoir in the San Joaquin Valley of California. Widgeon grass contained 120–780 mg B/kg dw, and in one pond, Hothem and Ohlendorf (1989) found concentrations (1,630 mg/kg) high enough to impair avian reproduction if widgeon grass from that pond were a sole-source food supply. Widgeon grass seeds contained 430–3,500 mg B/kg dw (Schuler 1987) and algae contained 390–790 mg/kg (Hoffman et al. 1991) at Kesterson Reservoir. Levels of boron in plant tissues were elevated compared to mean concentrations found in water (20 mg/L) and sediment (20 mg/kg), indicating that boron was bioconcentrating in aquatic plants. Toxic effects of boron to various plant species, as reported in the literature, are summarized in table 7 at the end of this chapter.

Macroinvertebrates

Little information is available on the toxicity of boron to aquatic invertebrates (table 7). In tests with *Daphnia magna*, the no-observed-adverse-effect level (NOAEL) and the lowest-observed-adverse-effect level (LOAEL) were found to be about 6 and 13 mg B/L, respectively (Lewis and Valentine 1981; Gersich 1984). Hothem and Ohlendorf (1989) found that the boron concentration in adult damselflies was 27 percent lower than in nymphs. This result suggests that a greater proportion of boron in nymphs may be incorporated in the exoskeleton. Maier and Knight (1991) found a significantly decreased growth rate by *Chironomus decorus* larvae at boron concentrations of 20 mg B/L and

greater. The concentrations of boron eliciting chronic sublethal responses in *C. decorus* are close to those reported in severely contaminated systems in the Central Valley of California (15–29 mg B/L).

Fish

The boron toxicity database for fish is relatively extensive, and several compre-hensive summaries have been compiled recently (e.g., SJVDP 1990; Perry et al. 1994). This literature, however, is mostly limited to evaluations of waterborne exposures to boron (i.e., without dietary exposure) and also does not include any definitive data relating boron levels in fish tissues to toxic effects. Con-sequently, although the database is extensive, its interpretive value is hampered by the critical gaps in "field-relevant" toxicity data (i.e., dietary exposures and tissue-based toxicity thresholds). Another confounding feature is the fact that threshold-level effects are commonly seen at water concentrations of boron much lower than the EC50 (see Appendix II for definition of terms), but EC50s and LC50s are the only standardized measures of toxicity consistently used in most bioassay-type toxicity studies. For sake of comparison, table 7 is largely restricted to summarizing EC50 and LC50 estimates of various studies. Toxicity measures based on various other endpoints are reported in SJVDP (1990) and Perry et al. (1994). The general concentrations of boron associated with threshold-level (e.g., EC1 to EC10) measures of toxicity will, however, be briefly summarized in discussions to follow.

The available literature indicates that boron levels of 0.001–0.1 mg/L could reduce the reproductive potential of sensitive fish species, and concentrations exceeding 0.2 mg/L could impair the survival of developmental stages for other species, under conditions providing continuous exposure from fertilization through 4 days posthatching (Birge and Black 1977). Birge and Black also found that boron compounds were more toxic to developmental and early posthatched stages than to adult fish. However, Hamilton and Buhl (1990) found no difference in the sensitivity of various life stages of fish exposed to boron for 96 hours. Both studies indicated that water hardness did not seem to affect boron toxicity (Birge and Black 1977; Hamilton and Buhl 1990).

The early life stages of rainbow trout appear to be the most sensitive to boron, with a consistent dose-response-related lowest observable effect concentration (LOEC) of 0.1 mg B/L (Birge and Black 1977). High boron concentrations (25–200 mg/L) were required to consistently produce substantial impairment to trout embryos and alevins.

High frequencies of both embryonic and postembryonic mortality in trout eggs were recorded only at boron concentrations of 50 mg/L or more. Embryonic mortality and teratogenesis were the principal boron-induced responses at 50 mg/L or less. Percent hatchability of trout eggs generally was inversely proportional to exposure level from 1 to 200 mg B/L (Birge and Black 1977). Borax at or below 0.5 mg B/L did not reduce hatching frequency; at 200 mg/L, hatchability dropped to zero. A high incidence of terato-genesis was observed over the range of exposure levels from 1.0 to 200 mg B/L. Borax and boric acid are unusual in that they exert low-level embryopathic effects on trout over a wide span of exposure levels (0.001–1.0 mg/L) (Birge and Black 1977).

In channel catfish, at a concentration of 200 mg B/L, normal survival at 4 days posthatching was only 0–2 percent; at 300 mg B/L, many of the eggs did not hatch, and those that did produced deformed hatchlings. Normal survival was 100 percent at and below 1.0 mg B/L. In both channel catfish and rainbow trout, embryonic mortality and teratogenesis increased in hard water, and boric acid produced higher frequencies than borax (Birge and Black 1977).

The low-level effects observed in reconstituted laboratory water, however, may not predict the much higher first effect levels under natural water conditions. Studies conducted for and by Procter and Gamble found that natural waters

containing 0.75 mg B/L did not affect rainbow trout early life stages (Butterwick et al. 1989). Bingham (1982) was able to find at least some wild healthy trout in surface waters containing as much as 13 mg B/L, although it was not known how long those trout had been exposed nor whether they constituted a demographically healthy population. In demographically open populations, as was the case in Bingham's study, upstream and downstream movements can continually maintain the presence of fish even in a habitat where a closed population could not sustain itself. Thus, in such cases, data on the presence or absence of fish are of questionable value for delineating acceptable water quality characteristics.

Based on a limited number of field surveys, Saiki and May (1988) suggested that whole freshwater fish typically contain <4 mg B/kg. Results from laboratory and field studies suggest that boron bioaccumulation is common in fish, but bioconcentration is not (Perry et al. 1994; Ohlendorf et al. 1986; Saiki and May 1988; Hamilton and Wiedmeyer 1990; and Thompson et al. 1976).

Amphibians/Reptiles

Birge and Black (1977) found that leopard frog embryos suffered 100 percent lethality or teratogenesis in water treated with borax or boric acid at exposure levels of 200 or 300 mg B/L, respectively. Boron compounds are more toxic to embryos and larvae than to adult amphibians, and amphibians are more tolerant of boron than fish, particularly at low concentrations (Birge and Black 1977).

Birds

Toxic effects of boron in birds, as reported in the literature, are summarized in table 7 at the end of this chapter.

In mallards, adverse reproductive effects have been reported at dietary concentrations of 1,000 mg B/kg; hatching success of fertile eggs, body weights of ducklings at hatch, and survival of ducklings from hatching to day 7 were all substantially reduced when breeding adults and their offspring were maintained on a diet supplemented with 1,000 mg B/kg. Although the mallards had markedly impaired embryo survival, the teratogenic effects described in boron egg-injection studies were not observed in this study. Mallard embryo mortality was greatest during the second half of incubation, when energy demands for embryonic growth were great. Because no adults died as the result of dietary boron treatment, it appears that embryos and hatchlings are the most sensitive mallard life stages to boron toxicosis (Smith and Anders 1989).

Stanley et al. (1996) also found statistically significant adverse reproductive effects in mallards fed 900 mg B (as boric acid) per kilogram of dry feed. Hatching success was reduced to only 58 percent of controls, suggesting that this level of dietary exposure is close to the EC50 value. At a dietary exposure of 450 mg B/kg, hatching success was reduced to 88 percent of controls, suggesting an approximate EC10 value. Concentrations of boron in mallard eggs associated with these approximate EC50 and EC10 dietary exposures of hens were, respectively, 38 and 22 mg/kg dw.

Boric acid in the diet of ducklings hatched from untreated eggs proved to be less toxic than reported for ducklings hatched from boron-contaminated eggs. Hoffman et al. (1990) found 10 percent mortality at 10 weeks in ducklings from uncontaminated eggs that received 1,600 mg/kg dietary boron. Smith and Anders (1989) reported 21 percent mortality during the first week and 12 percent mortality during the second week in duck-lings that received 1,000 mg B/kg both from the adult hen mallard and in their own diet. In a natural setting, the ducklings would

probably encounter both types of boron exposure, during embryogenesis and posthatching development, and so these higher mortality figures are probably more relevant.

Smith and Anders found that diets containing as little as 30 mg B/kg fresh weight (fw) fed to mallard adults adversely affected the growth rate of their ducklings. In a study by Hoffman et al. (1990), dietary levels of 100 mg B/kg fw resulted in reduced growth of female mallard ducklings. These findings indicate that concentrations greater than 30–100 mg B/kg in natural diets of ducklings could adversely affect their development.

Mallards fed concentrations up to 2,000 mg B/kg did not exhibit any histological pathologies. Therefore, histology may not prove to be an adequate means of assessing boron exposure or toxicosis in mallard ducks. Boron levels in egg, liver, and brain tissues increased in proportion to dietary concentrations of boron; however, these tissues contained residues that were at least one order of magnitude lower than the dietary concentration administered. Hoffman et al. (1990) and Smith and Anders (1989) found that boron accumulation in the brain and liver was substantially greater in all boron-supplemented groups than in controls, with a greater accumulation in the brain.

Pendleton et al. (1995) reported extremely rapid accumulation and elimination of boron in mallard tissues. Adult male mallards fed a diet containing 1,600 mg B/kg accumulated equilibrium levels of boron in liver tissue and blood within 2–15 days. After boron was removed from the diet of these mallards, it was completely cleansed from the liver and blood within 1 day. These findings are consistent with early research on cows and rats which revealed that the boron concen-tration of cow's milk could increase tenfold within the first 24 hours of dietary boron supplementation and that boric acid fed to rats is eliminated with extreme rapidity (Hove et al. 1939).

Mammals

The reported toxic effects of boron on mammals are summarized in table 7, at the end of this chapter. In general, excessive boron consumption by mammals results in a reduced growth rate and in some cases loss of body weight. Growth retardation has been reported in cattle given 150 mg B/L drinking water, in dogs consuming diets containing 1,750 mg B/kg, in rabbits eating rations equivalent to >140 mg B/kg bw daily, and in rats given 150 mg B/L in drinking water or 1,060 mg B/kg in food (Eisler 1990). In some instances, animals avoid boron-contaminated drinking water; rats reject drinking water containing as little as 1.0 mg B/L (Dixon et al. 1976), and cattle avoid water containing >29 mg B/L (Green and Weeth 1977).

Adverse effects on the reproduction of laboratory mammals have been reported in sensitive species fed diets containing more than 1,000 mg B/kg or given drinking water containing 1.0 mg B/L (Eisler 1990). Boric acid caused decreased fetal body weight and increased malformations in rats, mice, and rabbits with doses in the range of 80–400 mg/kg/day, given either throughout gestation or only during major organogenesis (Heindel et al. 1994).

Boron is readily transmitted into milk and eggs, as well as through the placenta (Hove et al. 1939). Boron compounds, especially boric acid, can accumulate in animal tissues and produce a reduction in fertility, an increase in developmental abnormalities, and death (Weir and Fisher 1972; Lee et al. 1978; Landolph 1985). Boron is found at concentra-tions ranging from 0.05–0.6 mg/kg fw in most animal tissues but may be several times higher in bones (Nielsen 1986). Mule deer metacarpals have been found to contain 0.8–3.6 mg B/kg dw, with younger animals having much higher bone boron concentra-tions than adults (Stetler 1980). Boron from boric acid has been shown to concentrate in the brain, spinal cord, and liver following ingestion (Beyer et al. 1983). Nontoxic concentrations of dietary boron (sodium borate or boric acid) are rapidly and

almost completely absorbed from the gastrointestinal tract, do not seem to accumulate in healthy tissues, and are excreted in urine, usually within hours (NAS 1980; Benson et al. 1984; Nielsen 1986; Siegel and Wason 1986).

Bioaccumulation

Boron can be bioconcentrated to varying degrees by aquatic organisms (Ohlendorf et al. 1986). Green algae *(Chlorella pyrenoidosa*) had a bioconcentration factor of 5 (boron concentration five times the level in the surrounding medium) after being exposed to a 50–100 mg B/L boric acid solution for 7 days (Fernandez et al. 1984). In the San Joaquin Valley, filamentous algae accumulated 390–787 mg B/kg when exposed to brackish tile drainage containing 12–41 mg B/L, and they accumu-lated 64–140 mg/kg when exposed to fresher water containing 1.4–2.2 mg/L (Schuler 1987). Aquatic insects living in the tile drainage contained 22–340 mg B/kg, but those living in fresh water untainted by agricultural tile drainage contained 6–47 mg/ kg (Ohlendorf et al. 1986; Schuler 1987; Hothem and Ohlendorf 1989).

Lemna species are outstanding boron bioaccumulators. Proficiency in boron stripping coupled with a high growth rate distinguishes *Lemna minor* as an important species with respect to boron cycling in a freshwater macrophyte community. The effectiveness of this species in consuming boron may be a potent force in lowering the concentrations of this essential element in aquatic systems (Glandon and McNabb 1978). Frick (1985) determined that pH affected the bioaccumulation of boron in *Lemna minor*, indicating that chemical speciation of boron may affect bioaccumulation and toxicity.

Interactions

Hoffman et al. (1991) examined boron-selenium interaction effects in mallard ducklings under two very different conditions: (1) a protein-adequate diet and (2) an iso-caloric protein-deficient diet. Unquestionable interaction effects were noted only under conditions of protein deficiency. However, Hoffman et al.'s protein-deficient diet was unlike any likely to be encountered by ducklings in the wild, so the results for part 2 of their experiments are essentially irrelevant to these guidelines. The results of part 1 are more relevant to the real world and failed to reveal any substantive interaction effects (despite unrealistically high dosing levels). More recently, Stanley et al. (1996) experimentally studied the effects of boron-selenium interactions on mallard reproductive performance, duckling growth, and duckling survival. Their experiments also found little evidence of interaction between these elements.

Regulatory Standards

U.S. Environmental Protection Agency standards and criteria
[See Appendix II for explanation of terms. Source: EPA 1995]

Status	Listed for regulation; carcinogenicity unknown	
Drinking water MCL	Not established	
Drinking water health advisories for 10-kg child	1-day HA: 10-day HA: Long-term HA:	4 mg/L 0.9 mg/L 0.9 mg/L
Drinking water health advisories for 70-kg adult	Reference dose: Long-term HA: Lifetime HA: DWEL:	0.09 mg/kg/d 3 mg/L 0.6 mg/L 3 mg/L

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

Table 7.—Summary of literature for boron ecotoxicology

[LC50, median lethal concentration; LD50, median lethal dose; both indicate 50 percent mortality after a stated time interval. Similarly, LC100 denotes 100 percent mortality. dw, dry weight; bw, body weight; conc., concentration]

Species	Boron compound	Concentration	Test conditions	Effect	Reference
Plants					
Blue-green algae (*Anacystis nidulans*)	Boric acid	75–100 mg B/L	72 hours	Photosynthetic pigments depleted	Martinez et al. 1986; Mateo et al. 1987
Duckweed (*Lemna minor*)	Boric acid	100 mg B/L	pH 5.0	Growth inhibited	Frick 1985
		20 mg B/L	pH 7.0	Growth inhibited	
Waterweed (*Elodea canadensis*)	Boric acid	1 mg B/L	28 d	Reduced photosynthesis	Perry et al. 1994
Invertebrates					
Mosquito larvae (3 spp.)	Boric acid	700–2,797 mg B/L	Freshly hatched to pupae stages	LC100 (48 hr)	EPA 1975
Midge (*Chironomus decorus*)	Borax	1,376 mg B/L	Fourth instar	LC50 (48 hr)	Maier and Knight 1991
		20 mg B/L	Decrease in growth (96 hr)	Significant decrease in growth rate	
Water flea *(Daphnia magna)*	Boric acid	420 mg/L	Neonates	LC100 (48 hr)	Lewis and Valentine 1981; Gersich 1984
		115-246 mg/L	LC50 (48 hr)	LC50 (48 hr)	
		13–53 mg/L	Hard water	LC50 (21 d); reduced mean brood size and body length	
		13.6 mg/L	21 d	LOAEL, reproductive effects	
		6.4 mg/L	21 d	NOAEL	
Fish					
Bluegill (*Lepomis macrochirus*)	Boron trifluoride	15,000 mg B/L		LC50 (24 hr)	Birge and Black 1977
Chinook salmon	Boric acid	>1,000 mg/L	Eyed eggs, alevins and fry; soft water	LC50 (24 hr)	Hamilton and Buhl 1990
		566–725 mg/L	Fry; very hard and soft fresh water	LC50 (96 hr)	
Chinook and Coho salmon	Boric acid	>1,000 mg/L	Fry; very hard fresh and brackish water	LC50 (24 hr)	
Coho salmon	Boric acid	447 mg/L	Very hard fresh water	LC50 (96 hr)	

Table 7.—Summary of literature for boron ecotoxicology—Continued

Species	Boron compound	Concentration	Test conditions	Effect	Reference
Fish—Continued					
Channel catfish	Borax and boric acid	155 mg/L	Embryos and fry	LC50 (9 d)	Birge and Black 1977
Goldfish	Boric acid	75 mg/L	Embryos and fry; hard water	LC50 (7 d)	Birge and Black 1977
	Borax	59 mg/L			
Minnows	Boric acid	18,000–19,000 mg/L	Distilled water	Minimum lethal dose	EPA 1986
		19,000–19,500 mg/L	Hard water		
	Borax	19,000–19,500 mg/L	Distilled and hard water	Minimum lethal dose	Sprague 1972
	Anhydrous borax	3,000–7,000 mg/L			
	Boric acid	1,600–3,700 mg/L			
Mosquitofish (*Gambusia affinis*)	Boric acid	979 mg B/L	Adults	LC50 (96 hr)	Birge and Black 1977
Rainbow trout		339 mg/L	Adults	LC50 (48 hr)	Sprague 1972; Birge and Black 1977; Lewis and Valentine 1981
Amphibians					
Toad (*Bufo vulgaris*)	Boric acid	874 mg B/L	Embryos, 24-hr exposure	Edema, microcephalia, short tail, suppressed forebrain development	EPA 1975
Fowler's toad	Boric acid	145 mg/L	Embryos and tadpoles; soft water	LC50 (7.5 d)	Birge and Black 1977
		25–123 mg/L	Embryos and tadpoles; hard and soft water		
Leopard frog	Boric acid	130 mg/L	Embryos and tadpoles; soft water	LC50 (7.5 d)	Birge and Black 1977
	Borax	47–54 mg/L	Embryos and tadpoles; hard and soft water		

Table 7.—Summary of literature for boron ecotoxicology—Continued

Species	Boron compound	Concentration	Other conditions	Effect	Reference
Birds					
Domestic chicken	Boric acid (in food)	875 mg B/kg	Adult; 6-day exposure	Egg production ceased	Birge and Black 1977
Mallard duck (*Anas platy-rhynchos*)	Boric acid (in food)	1,600 mg/kg dw	Ducklings; 10-week exposure	Reduced growth; increased resting time; duckling brain 51 mg/kg; liver 29 mg/kg dw	Hoffman et al. 1990; Stanley et al. 1996
		400 mg/kg dw		Delayed and reduced rate of growth among females; increased resting time in ducklings; adult brain 5 mg/kg dw, liver 3 mg/kg dw	
		100 mg/kg dw		Delayed and reduced rate of growth among females; reduced bathing time in ducklings; adult brain, 4 mg/kg dw, liver, 3 mg/kg dw	
		1,000 mg/kg dw	Hen dosed beginning 3 weeks prior to mating; ducklings dosed for 21 days after hatching	48% reduction in hatching success; reduced weight and survival of ducklings. Resulting B conc. (mg/kg dw): adult brain 41, liver 33; egg 49; duckling brain 66, liver 51	Smith and Anders 1989
		300 mg/kg dw		Reduced weight gain rate in ducklings. Resulting B conc. (mg/kg dw): adult brain 14, liver 15; egg 13; duckling brain 19, liver 17	
		30 mg/kg dw		Reduced weight and weight gain in ducklings through 21 days; egg, 3 mg/kg dw; duckling brain, 4 mg/kg dw	
Mallard duck (*Anas platy-rhynchos*)	Boric acid (in food)	900 mg/kg dw		Reduced hen weight gain; reduced egg size, weight, and hatching success (by ~50% compared to controls)	Stanley et al. 1996
		450 mg/kg dw		Reduced egg hatching success (~10% compared to controls)	

Table 7.—Summary of literature for boron ecotoxicology—Continued

Species	Boron compound	Concentration	Test conditions	Effect	Reference
Mammals					
Dog (*Canis familiaris*)		1,170 mg B/kg	38 weeks exposure	Testicular degeneration; spermatogenesis cessation	Nielsen 1986; Weir and Fisher 1972
Rabbit (*Oryctolagus* sp.)	Borates (in food)	800–1,000 mg/kg bw	4-day exposure	Growth retardation	Anonymous 1983
Rat (*Rattus* sp.)	Boric acid (in food)	1,750 mg B/kg	25 days exposure	50% reduction in growth rate	Seal and Weeth 1980
	Boric acid (in food)	1,170 mg B/kg	2 years exposure	Sterility in males and females	Sprague 1972; Weir and Fisher 1972
	Sodium borate (in food)	1,060 mg/kg	Chronic exposure	Growth retardation; testicular atrophy	Anonymous 1983
	Boric acid	710–550 mg B/kg bw	Oral, single dose	LD50	Weir and Fisher 1972; EPA 1975; Dani et al. 1971
	Boric acid (in drinking water)	0.05 mg B/ kg bw	Daily for 6 months	Decreased spermatozoid count and activity	Krasovskii et al. 1976
	Boric acid (in food)	0.015–0.3 mg B/kg bw	Daily for 6 months	Adverse changes in testes	Anonymous 1983

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Copper

Description

Copper (Cu) is one of the most familiar metals, having been used for thousands of years to make tools, ornaments, utensils, and coins. The earliest metal workers found copper very easy to work with, owing to its malleability, ductility, and moderate melting point (1,083 °C). Copper is also one of the best conductors of both heat and electricity, which makes it useful in cookware and invaluable in electrical circuits. Native copper is shiny and brown to reddish-brown, but its many salts and minerals take on a variety of hues ranging from bright green to purple to indigo blue to yellowish-brown. Chemically it has two oxidation states, forming either cuprous (Cu^{+}) or cupric (Cu^{+2}) compounds. The cuprous compounds are not common in natural waters, though, as they oxidize readily to the bivalent form.

Occurrence

Copper is widespread in the environment, having an overall crustal abundance of about 50 mg/kg. It is present in both seawater and fresh water at concentrations generally in the range of 1–20 µg/L (Irwin 1996). Its most common ores include the sulfides chalcopyrite, bornite, chalcocite, and covellite, and the carbonates azurite and malachite. In the Western United States, large copper mining operations are found in Arizona, New Mexico, Utah, and Montana. Most of these are exploiting low-grade porphyry deposits (<1% Cu, mostly as chalcopyrite). Veins of native copper are rare in the Western States.

Copper is one of the most common contaminants found in urban runoff: it is present in the leachate from municipal landfills and in sludges generated by sewage treatment plants, and it is commonly leached from drinking water pipes, particularly in areas where the drinking water is somewhat acidic (Irwin 1996). Significant amounts of copper are produced in wastes from textile mills and cosmetics plants, and in sludge from hardboard production, mining, smelting, and the burning of coal in powerplants (Brown et al. 1983, Furness and Rainbow 1990, Kabata-Pendias and Pendias 1992).

Background Concentrations.—Background copper levels in soil range from 13 to 24 mg/kg in uncontaminated areas (Kabata-Pendias and Pendias 1992). The average concentration for water in lakes and streams is reported to be 2 µg/L (Nriagu 1979), and average freshwater fish concentration is 0.65 mg/kg wet weight (ww) (or about 2.60 mg/kg dry weight [dw]); the 85th percentile concentration in fish was 1 mg/kg ww (4 mg/kg dw) (Schmitt and Brumbaugh 1990). Copper concentrations in plants are generally in the range of 2 to 20 mg/kg dw (Thompson et al. 1991). Whole-body concentrations in small mammals collected from various uncontaminated sites ranged from 8.3 to 13.4 mg/kg dw (Talmage and Walton 1991). Whole-body concentrations of copper in amphibians vary from 8 to 845 mg/kg dw, but extremely high copper concentrations (up to 2,091 mg/kg dw) have been observed in livers of some amphibians from uncontaminated areas (Hall and Mulhern 1984).

Summary of Effects

Copper is an essential element for all living organisms, but elevated levels of copper in

the environment may be harmful at or near copper-contaminated sites. Both deficient and excess amounts of copper cause adverse effects in all species. Copper is generally more toxic to aquatic organisms than to birds or mammals. This is reflected by the relatively low ambient water quality criteria for copper and by the rarity of toxic effects through excess dietary exposure in birds and mammals under field conditions. However, some ungulates, such as sheep (NAS 1980; Puls 1988), are more sensitive to copper than other mammals. Nevertheless, copper concentra-tions in the bodies of aquatic birds and mammals are generally well regulated (Furness and Rainbow 1990), and copper toxicity is more likely to affect aquatic plants, invertebrates, and fish. A summary of biotic effect levels is presented in table 8.

Suter and Mabrey (1994) evaluated a series of toxicological benchmarks for screening various contaminants for their potential effects on aquatic biota. In addition to the national ambient water quality (NAWQ) criteria, they provided secondary acute and chronic values, lowest chronic values (including those for fish, daphnids, nondaphnid invertebrates, aquatic plants, and all organisms), test EC20s, sensitive species test EC20s, and population EC20s. The values for water in table 8 are as follows: "No effect" is the lowest chronic value for all organisms; "Toxicity threshold" is the NAWQ chronic criterion (if established) or the secondary chronic value; and "Level of concern" is the range between the two other values.

Field Cases

Three examples illustrate the potential impacts of copper-contaminated mine drainage on the aquatic environment:

(1) Acid mine drainage from the Iron Mountain Mine near Redding, California, containing high concentrations of copper and zinc caused numerous fish kills in the upper Sacramento River (Finlayson and Ashuckian 1979, Finlayson and Verrue 1980). Some of these occurred as far back as the early 1900's, but they became more frequent and more serious following the construction of Shasta Dam in 1944 and Keswick Dam in 1950. Finlayson and Ashuckian (1979) hypothesized that these dams had effectively diminished the "dilution effect" in the Sacramento River. Yet, the outflows from these dams have also been used to purposely dilute elevated concentrations that are detected at downstream monitoring stations.

(2) Similarly, toxic concentrations of copper and zinc from the Penn Mine area in the Sierra Nevada of California caused sizable fish kills in the lower Mokelumne River Basin (Finlayson and Rectenwald 1978). During a fish kill in the Mokelumne River in 1958, copper was elevated to 3.8 mg/L a short distance downstream from the mine.

(3) In Canada, the effects of mixed mining wastes on fish were examined through integrated field sampling of water, sediment, invertebrates, and fish (Munkittrick et al. 1991, Miller et al. 1992). Miller et al. (1992), in particular, made an extensive study of the relationships between concentrations of zinc and copper in all these media in the Manitouwadge chain of lakes in northern Ontario. They found a correlation between zinc concentrations in invertebrates and in sediment but observed no such relationship with water concentrations. Neither did they find any relationship between zinc concentrations in fish tissue and those in invertebrates, although several lab studies had suggested that food and particulates are much more important sources of zinc than water (Patrick and Loutit 1976, Dallinger and Kautzky 1985, as cited in Miller et al. 1992). For both zinc and

Table 8.—Summary of comprehensive biotic effects of copper

["—" indicates that no data are available]

Medium	No effect	Level of concern	Toxicity threshold	Explanation
Water (µg/L)	0.23	0.23–12	12	Hardness-dependent criteria: 0.23 µg/L is lowest chronic value for aquatic organisms; 12 µg/L is NAWQ chronic criterion at hardness of 100 mg/L (as $CaCO_3$). (See Suter and Mabrey 1994.) Sensitive species may be affected in the "level of concern" range (depending partly on effects of pH, temperature, and dissolved oxygen).
Sediment (mg/kg, dw)	34	34–270	270	"ERL" and "ERM" values of Long et al. (1995). However, sulfides in the sediment may reduce copper toxicity (see text).
Plants (mg/kg dw)	3–30	—	>20	Kabata-Pendias and Pendias (1992). Toxicity threshold varies depending on species of Cu present.
Invertebrates	—	—	—	Diagnostic levels not established because Cu generally is homeostatically regulated. Some invertebrates (e.g., crustaceans and mollusks) require Cu for hemocyanin and may normally have higher levels than other species (Furness and Rainbow 1990).
Fish, whole body (mg/kg, dw)	9.8	9.8–13.3	13.3	Diagnostic levels not established because Cu generally is homeostatically regulated. After 9-week dietary exposure, rainbow trout showed decreased weight gain at 13.3 mg/kg but no significant effects at 9.8 (Julshamn et al. 1988).
Birds, liver (mg/kg dw)	<60	25–300	>540	Data for ducks from Puls (1988); toxic concentrations in waterfowl diets are >200 mg/kg dw.
Eggs (mg/kg dw)	5.5	—	—	Egg data from J.P. Skorupa (unpub. data, 1996).
Amphibians/reptiles	—	—	—	Diagnostic levels not established; even at uncontaminated sites, some amphibian tissues have 800–2,000 mg/kg dw (Hall and Mulhern 1984).
Mammals	—	—	—	Diagnostic levels for wild mammals not established; for sheep, liver concentrations >250 mg/kg dw may be toxic (Puls 1988).

copper, the water concentration was a better indicator of metal concentration in fish tissue than the sediment or invertebrate concentrations in this field study. Miller et al. (1992) also reported reduced growth in females of white sucker after sexual maturation, decreased egg size and fecundity, no sig-nificant increase in fecundity with age, and an increased incidence of spawning failure at a water-borne zinc concentration of 156 mg/L and a sediment concentration of 6,397 mg/kg. In addition, they found kidney and liver

concentrations to be better indicators of chronic zinc and copper exposure than muscle concentrations.

Many more recent field studies have investigated the toxicity of copper and copper-zinc mixtures in effluents. Finlayson and Verrue (1980) and Finlayson and Ashuckian (1979) conducted long-term and short-term toxicity studies on Chinook salmon and steelhead trout, respectively, in order to estimate "safe" levels of copper and zinc for those species. Harrison and Klaverkamp (1990) also studied the bioaccumulation of copper, zinc, and other metals in northern pike and white suckers from lakes near a smelter.

Abiotic Factors Affecting Bioavailability

Water

In natural waters, dissolved copper occurs in several different chemical forms and in various inorganic and organic complexes. Copper is present as Cu^{+2} in acidic waters and $CuOH^{+}$ in soft waters, and a dissolved fraction of copper is believed to be toxic in fish (Davies et al. 1979). Factors that affect the speciation of copper in water are pH, temperature, hardness, and dissolved organic carbon (Bodek et al. 1988). Low pH, soft water, and higher temperature are known to increase the copper toxicity.

Bottom Sediment

Harrison and Klaverkamp (1990) found no consistent relationship between copper concentrations in bottom sediment and those in tissues of white sucker and northern pike (table 9). Long and Morgan (1990) concluded that copper in bottom sediment at concen-trations of about 20 mg/kg dw may induce sublethal behavioral effects in clams if it is not tightly chelated or bound to sedi-ments. However, Long et al. (1995) found that copper concentrations of 34 mg/kg rarely impair the survival or reproduction of benthic invertebrates but that concentrations of 270 mg/kg or higher usually do. Although many of the data that were evaluated were for estuarine and marine sediments, Hull and Suter (1994) concluded that those screening levels also were appropriate for freshwater sediments until more specific guidelines become available. However, they also recommend that these concentrations be compared to local background levels when possible, and that concentrations within the background range should not be considered a problem.

Acid-volatile sulfide (AVS) in the sediment may bind a certain portion of some metals (Cd, Cu, Ni, Pb, and Zn) and render that portion unavailable and nontoxic to biota (Di Toro et al. 1992). In order to assess the effects of acid-volatile sulfide on metal toxicity, the AVS is extracted from sediment with hydrochloric acid, and the metal concentration that comes with it is called the simultaneously extracted metal (SEM). All SEMs that would contribute appreciably to the total SEM are measured and totaled (Di Toro et al. 1992). *If* the sediments are not fully oxidized (Adams et al. 1992), then an SEM:AVS ratio <1 indicates that acute toxicity is unlikely. The method has not yet been adapted for chronic toxicity.

Soil

Copper is able to form complexes with various soil constituents. Copper in soils can precipitate with hydroxide, phosphate, carbonate, and silicate to become a component of the amorphous fraction of soil. It can be adsorbed on the negatively charged sorption sites of silicate clay and it can form both soluble and insoluble complexes with components of soil organic matter (Baker and Amacher 1982).

Table 9.—Biological effects of copper concentrations in sediment

Species	Cu in sediment (mg/kg)	Cu in biomass (mg/kg ww, except as noted) and other effects	Location/Comments	Reference
Food chain				
Invertebrates	102	Complete absence of Plecoptera, Ephemer-optera, Odonata, Trichoptera, Amphipoda, and Unionidae	Manitouwadge Lake, Ontario, Canada (Zn = 1,149 mg/L)	Munkittrick et al. 1991
Fish				
White sucker (*Catostomus commersoni*)	11.4	Liver 50 (dw), muscle 7 (dw), stomach contents 7 (dw)	Loken Lake, Ontario, Canada (Zn = 43 mg/L)	Munkittrick et al. 1991
	102	Liver 83 (dw), muscle 6 (dw), stomach contents 155 (dw). Lowered growth rate.	Manitouwadge Lake, Ontario, Canada (Zn = 1,149 mg/L)	
	34	Liver 0.21, muscle 0.25	Top soil of Lake Nekik, Manitoba, Canada	Harrison and Klaverkamp 1990
	49	Liver 0.01, muscle 0.28	Top soil of Lake Naosap Mud, Manitoba, Canada	
	76	Liver 0.01, muscle 0.28	Top soil of Lake Kotyk, Manitoba, Canada	
	2,775	Liver 13.4, muscle 0.16	Top soil of Lake Hamell, Flin Flon, Canada	
	2,858	Liver 24.3, muscle 0.25	Top soil of Lake Meridian, Flin Flon, Canada	
	5,950	Liver 19.8, muscle 0.24	Top soil of Lake Cliff, Flin Flon, Canada	
	6,988	Liver 14.3, muscle 0.26	Top soil of Lake Phantom, Flin Flon, Canada	
	12,625	Liver 29.2, muscle 0.18	Top soil of Lake Douglas, Flin Flon, Canada	
Northern pike (*Esox lucius*)	34	Liver 15.2, muscle 0.11	Top soil of Lake Nekik, Manitoba, Canada	Harrison and Klaverkamp 1990
	49	Liver 11.2, muscle 0.12	Top soil of Lake Naosap Mud, Manitoba, Canada	
	76	Liver 16.8, muscle 0.11	Top soil of Lake Kotyk, Manitoba, Canada	
	198	Liver 11.2, muscle 0.2	Top soil of Lake Cleaver, Manitoba, Canada	
	2,775	Liver 19.5, muscle 0.17	Top soil of Lake Hamell, Flin Flon, Canada	
	2,858	Liver 17.1, muscle 0.16	Top soil of Lake Meridian, Flin Flon, Canada	
	5,950	Liver 11.9, muscle 0.18	Top soil of Lake Cliff, Flin Flon, Canada	
	6,988	Liver 7.6, muscle 0.13	Top soil of Lake Phantom, Flin Flon, Canada	
	12,625	Liver 28.5, muscle 0.14	Top soil of Lake Douglas, Flin Flon, Canada	

The strength of copper sorption by soil constituents occurs in the following relative order: manganese oxides < organic matter < iron oxides < clay minerals. Other soil components that may play a less significant role in copper sorption include free phosphates, iron salts, and clay-size aluminosilicate minerals. Copper retention in soils increases with increasing soil pH. The lack of adsorption of copper at low pH may be due to competition for sorption sites from other soil cations (Mn^{+2}, Fe^{+2}, H^{+}, and Al^{+3}) (Brown et al. 1983).

Biotic Effects

Plants

Copper sulfate has been used for more than 80 years as an algicide, typically at a concentration of 1 mg/L for the upper 0.5 meter of water (Mackenthun and Ingram 1967). In the soil, copper is toxic to sensitive plants at concentra-tions of 25 to 50 mg/kg (Demayo et al. 1982). Cereals, legumes, spinach, citrus seedlings, and gladiolus are known to be most sensitive to copper. The general symptoms of copper toxicity to plants are dark green leaves fol-lowed by induced iron chlorosis (yellowing); thick, short, or barbed-wire roots; and depressed tillering (Kabata-Pendias and Pendias 1992).

Macroinvertebrates

Most aquatic organisms are relatively sensitive to copper, even at low concentra-tions. The effects of low concentrations of copper on various invertebrates are noted in table 10. In a field study, Munkittrick et al. (1991) observed concentrations of 9.7 µg Cu/L and 232 µg Zn/L in Manitouwadge Lake, Ontario, Canada. Under these conditions, they noted the complete absence of Unionidae and several families of arthropods (table 10). Miller et al. (1992) found a correlation between copper concentrations in invertebrates and water concentrations but observed no such relationship with sediment concentrations.

Sediments in the Upper Clark Fork River and Milltown Reservoir in Montana have been contaminated with mine-related wastes (As, Cd, Cu, Pb, Mn, and Zn). In soft sediment depositional areas, taxa of Oligochaeta and Chironomidae generally accounted for more than 90 percent of the benthic invertebrate communities. Canfield et al. (1994) observed higher numbers of Chironomidae genera in areas where sediments had been identified as toxic using 28-day laboratory tests with the amphipod *Hyalella azteca*. Frequency of Chironomidae mouthpart deformities and total abundance of organisms did not correspond to concentrations of metals in sediment. In areas where benthic communi-ties were affected, sediment and surface water Cu concentrations ranged from 364 to 7,820 micrograms per gram and 274 to 11,080 µg/L, respectively.

Some aquatic organisms can tolerate rela-tively high levels of copper in their diets. Hatakeyama (1989) compared the toxicity of copper through water and diets in mayfly larvae and concluded that the high mortality at 100 µg/L (table 10) was principally attributable to copper in the water because, at this level, algae bioaccumulated only 450 mg/kg (dw), and that much copper in the diet was not enough to kill mayfly larvae (table 11).

Fish

Miller et al. (1992) extensively studied the relationship between concentrations of copper and zinc in water, sediment, invertebrates, and fish. For both copper and zinc, the water concentration was a better indicator of metal concentration in fish tissue than the sediment or invertebrate concentrations. In addition, they found kidney and liver concentrations to be better indicators of chronic copper and zinc

Table 10.—Biological effects of copper on aquatic species

Species	Cu concentration in water (µg/L)	Effect	Comments	Reference
Invertebrates				
Cladoceran (*Daphnia magna*) egg	10,000	Significant effect in development	46-h exposure; eggs more tolerant than adult	Bodar et al. 1989
	22	16% reproduction impairment	Chronic exposure	Nriagu 1979
	44	3 week LC50		
Cladocerans (*Daphnia ambigua, D. parvula, D. pulex*)	60	Significant drop in instantaneous rate of population growth	Chronic exposure	Nriagu 1979
Amphipod (*Gammarus pseudolimnaeus*)	4.6	No effect	Chronic exposure	Nriagu 1979
	8	Second-generation growth affected, but no effect in first generation		
Crayfish (*Orconectes rusticus*)	15	15% growth retarded	Chronic exposure	Nriagu 1979
Mayfly larvae (*Epeorus latifolium*)	5–10	No significant effect in growth rate	Temp. 12.5°C	Hatakeyama 1989
	15	Growth rate decreased during first 3 weeks but restored gradually after 4 weeks		
	20–25	Growth rate <7% of the control. 100% mortality by 10 weeks		
	100	83% mortality in 1 week	Temp. 11.5°C	
Midge larvae (*Chironomus tentans*)	327	48-h EC50; immobilization	Temp. 14°C; pH 6.3	Khangarot and Ray 1989
Invertebrates, general	9.7	Complete absence of Plecoptera, Ephemeroptera, Odonata, Trichoptera, Amphipoda, and Unionidae	Manitouwadge Lake, Ontario, Canada. Zn concentration 232 µg/L	Munkittrick et al. 1991
Fish				
Brown bullhead (*Ameiurus nebulosus*)	19	Increased mortality, reduced growth	Water hardness 187 and 38 mg/L	Nriagu 1979
	27	Cu in biomass (mg/kg dw): gill 6.9, liver 11, kidney 10	20-month exposure	
		Cu in biomass (ppm dw): gill 9.4, liver 33, kidney 10	30-d exposure; pH 7.2–8.2,	
Brook trout (*Salvelinus fontinalis*)	5	Reduced growth	Water hardness 38 mg/L	Nriagu 1979
	8	Reduced growth	Water hardness 187 mg/L	

Table 10.—Biological effects of copper on aquatic species—Continued

Species	Cu concentration in water (µg/L)	Effect	Comments	Reference
Fish—Continued				
Bluegill (*Lepomis macrochirus*)	400	96-h LC50 (if Zn present)	pH 6.8–7.5; temp. 22°± 1 °C; Zn = 1400 µg/L	Thompson et al. 1980
	1,000	96-h LC50 (if Zn absent)	pH 6.8–7.5; temp. 16.5°–23 °C; Zn = 0	
Chinook salmon fry (*Oncorhynchus tshawytscha*)	54	96-h LC50	In fresh water. Mean weight 0.87 g	Hamilton and Buhl 1990
	58	96-h LC50	In fresh water. Mean weight 0.66 g	
	60	96-h LC50	In brackish water. Mean weight 1.6 g	
	78	24-h LC50	In fresh water. Mean weight 1.60 g	
	81	24-h LC50	In brackish water. Mean weight 0.87 g	
	145	24-h LC50	In brackish water. Mean weight 0.66 g	
Chinook salmon (*Oncorhynchus tshawytscha*) eggs (to hatching)	26	28-d LC10s, based on various mixed solutions of Cu and Zn	Zn = dissolved Cu	Finlayson and Verrue 1980
	29		Zn = 6× dissolved Cu	
	40		Zn = 3× dissolved Cu	
	49		Zn = 11× total Cu	
	50		Zn = 6× total Cu	
	70		Zn = 3× total Cu	
Chinook salmon (*Oncorhynchus tshawytscha*) hatchlings to swim-up fry	14	28-d LC50s, based on various mixed solutions of Cu and Zn	Zn = 11× dissolved Cu	Finlayson and Verrue 1980
	20		Zn = 6× dissolved Cu	
	27		Zn = 11× total Cu	
	32		Zn = 3× dissolved Cu	
	37		Zn = 6× total Cu	
	56		Zn = 3× total Cu	
Coho salmon (*Oncorhynchus kisutch*)	60	96-h LC50	Smolts in May; temp. 10–12 °C; hardness 68–78 *or* 89–99 mg/L (as $CaCO_3$)	Lorz and McPherson 1976
	74	96-h LC50	Yearlings in November; other conditions as above	
Fathead minnow (*Pimephales promelas*)	18	Reduced spawning and egg production, increased mortality	Water hardness 31 mg/L	Nriagu 1979
	33	Reduced egg production	Water hardness 198 mg/L	
	37	Reduced egg production	Water hardness 200 mg/L	

Table 10.—Biological effects of copper on aquatic species—Continued

Species	Cu concentration in water (µg/L)	Effect	Comments	Reference
Fish—Continued				
Neomacheilus barbatulus	760	Cu in biomass (mg/kg dw): gill 164, liver 115, muscle 8.5	64-d exposure; pH 8.6	Nriagu 1979
Rainbow trout (*Oncorhynchus mykiss*)	19	Reduced percent hatch and increased mortality	Water hardness: 100 mg/L	Nriagu 1979
Steelhead trout (*Oncorhynchus mykiss*) eggs (to hatching)	<10	60-d LC10s, based on various mixed solutions of Cu, Zn, and Al	Cu:Zn:Al = 1:12:18, dissolved copper	Finlayson and Ashuckian 1979
	19		Cu:Zn:Al = 1:12:18, total copper	
	19		Cu:Zn:Al = 1:4:6, dissolved copper	
	43		Cu:Zn:Al = 1:4:6, total copper	
Steelhead trout (*Oncorhynchus mykiss*) hatchlings to swim-up fry	<10	60-d LC10s, based on various mixed solutions of Cu, Zn, and Al	Cu:Zn:Al = 1:12:18, for both total and dissolved copper	Finlayson and Ashuckian 1979
	14		Cu:Zn:Al = 1:4:6, dissolved copper	
	36		Cu:Zn:Al = 1:4:6, total copper	
White sucker (*Catostomus commersoni*)	2.1	Control group; Cu in biomass (mg/kg dw): liver 50, muscle 7, stomach contents 7	Loken Lake, Ontario, Canada; Zn conc. 10 µg/L	Munkittrick et al. 1991
	9.7	Lowered growth rate; Cu in biomass (mg/kg dw): liver 83, muscle 6, stomach contents 155	Manitouwadge Lake, Ontario, Canada; Zn conc. 232 µg/L	
Amphibians				
Narrow-mouthed toad (*Gastrophryne carolinensis*)	40	17-d LC50	Adult	Birge and Black 1979
	50	3-d LC50	Tadpole	
Southern gray tree frog (*Hyla chrysoscelis*)	40	9-d LC50	Adult	Birge and Black 1979
	60	3-d LC50	Tadpole	
Leopard frog *(Rana pipiens*)	40	8-d LC50	Adult	Birge and Black 1979
	60	4-d LC50	Tadpole	
Marbled salamander (*Ambystoma opacum*)	70	8-d LC50	Adult	Birge and Black 1979
	359	4-d LC50	Tadpole	
Birds				
Mallard (*Anas platyrhynchos*)	10,000	No adverse effect	Juveniles	Foster and Ramsdell 1997

Table 11.—Summary of exposure-response or exposure-bioaccumulation of copper

Species	Cu concentration in diet (mg/kg dw)	Exposure duration	Cu concentration in biomass (mg/kg ww)	Effects	Reference
Food chain					
Woodlouse (*Porcellio scaber*)	200,000			No effect on survival	Beyer et al. 1984
Mayfly (*Epeorus latifolium*) adult	<590 (in algae)	8 weeks		No change in mortality or growth rate	Hatakeyama 1989
Mayfly larvae	1,140 (in algae)	1 week		50% decrease in growth rate. Slight increase in mortality.	
Fish					
Rainbow trout (*Oncorhynchus mykiss*)	3.5	9 weeks	Whole fish 1.8; liver 7.9		Julshamn et al. 1988
	102		Whole fish 3.0; liver 18		
	194		Whole fish 4.7; liver 35		
	405		Whole fish 6.2; liver 35		
	603		Whole fish 7.8; liver 91		
	810		Whole fish 9.8; liver 112	Slight increase in mortality rate	
	990		Whole fish 13.3; liver 149	Significant decrease in weight gain; significant increase in mortality	
	178	20 weeks	Liver 159	No effect. (Diet also had 683 mg Zn/kg dw)	Knox et al. 1982
Birds					
Chicken	>500			Growth retardation	Melring et al. 1959
Turkey	>50			Purified diet; reduced growth and survival	Waibel et al. 1963
	>800			Natural diet; no effects	
Mammals					
Rat	500	4 weeks		Reduced growth	Boyden et al. 1937
Domestic sheep (*Ovis aries*)	30–60	10 weeks		Decreased food consumption, weight loss, hemolytic crisis, increased mortality	Zervas et al. 1990

exposure than muscle. Nriagu (1979) reported that brook trout are especially sensitive to copper concentrations in water (table 10), and yet Knox et al. (1982) found that rainbow trout can tolerate relatively high dietary concentrations of copper (table 11). The different tolerance levels are likely due in part to varying toxicities of the copper species used in the two studies.

As shown in table 10, Nriagu (1979) reported reduced egg production in fathead minnows

at relatively low copper concentra-tions in water. In addition, Miller et al. (1992) reported reduced growth in white sucker (*Catostomus commersoni*) females after sexual maturation, decreased egg size and fecundity, no significant increase in fecundity with age, and an increased incidence of spawning failure at waterborne copper concentrations of 15 µg/L and sediment concentrations of 93 mg/kg.

Amphibians

Amphibians are relatively sensitive to water-borne copper concentrations during the developmental stages (table 10) but are relatively tolerant to high copper burdens as adults and accumulate large amounts of copper in their livers (Hall and Mulhern 1984).

Birds

There are few studies available on the toxicity of copper to birds, but it appears that they tolerate copper better than most aquatic organisms do. NAS (1980) reported that 300 mg Cu/kg in the diet is the maximum tolerable level for poultry. This value may be used to estimate the safety levels for avian wildlife with the use of safety factors. Puls (1988) considered the toxic dietary level for ducks to be >200 mg/kg and noted that ducks accumulate more copper in the liver than do chickens or turkeys fed the same dietary levels. The maximum safe dietary levels of copper for growing chicks and turkeys were estimated to be 250 and 500 mg/kg, respectively, in the diet (Neathery and Miller 1977). Juvenile mallards could tolerate a copper concentration up to 10 mg/L in drinking water with pH greater than 4.0 without adverse health effects (Foster and Ramsdell 1997).

Mammals

Mammals, too, are relatively tolerant to copper compared to aquatic organisms. For most domestic mammals, the maximum recommended tolerable level of copper in the diet ranges from 100 to 800 mg/kg (NAS 1980). The level for sheep, however, was only 25 mg/kg. Zervas et al. (1990) found that 30 mg Cu/kg in the diet was toxic to sheep (table 11). General signs of copper toxicity in mammals are inhibition of growth, muscular dystrophy, anemia, impaired reproduction, and decreased longevity. Symptoms of copper poisoning are especially apparent when molybdenum content is low (Demayo et al. 1982). Small mammals such as shrews, mice, and voles may be useful biomonitors for copper, as shown in table 12 (Talmage and Walton 1991). In shrews, copper concen-trated in individual tissues in the order hair > liver > kidney > whole body (Hunter and Johnson 1982).

Bioaccumulation

In order to evaluate the cumulative toxicity of copper and other metals (As, Cd, Hg, Pb) along the food chain, Yannai et al. (1979) raised a large quantity of algae (*Micractinium* and *Chlorella*) on metal-rich waste water, fed the algae to chickens and carp, and then fed the meat of these chickens and carp to rats. They found that bioaccumulation did not increase the levels of any of these metals in chickens or carp except for chickens' livers (which contained higher copper than the livers of control chickens), and they observed no change in the general appearance, behavior, and survival of the rats that ate the chicken and carp meat. They concluded that such meat would pose no hazard to consumers.

Table 12.—Accumulation of copper in small mammals compared to copper concentrations in soils
[Data from Hunter and Johnson (1982)]

	Copper concentration (mg/kg, dry weight)	
Medium	**Uncontaminated site**	**Copper refinery site**
Soil	9.3	2,480
Wood mouse *(Apodemus sylvaticus*)		
Kidney	10.8	11.9
Liver	13.4	23.7
Hair	6.5	14.7
Muscle	8.5	6.9
Field Vole *(Microtus agrestis*)		
Kidney	10.8	22.6
Liver	13.4	13.5
Hair	6.5	24.2
Muscle	8.5	9.3
Shrew (*Sorex araneus*)		
Kidney	22.8	38.5
Liver	31.1	56.1
Muscle	10.9	17.4

Interactions

Mixtures of copper and zinc are known to be additive or synergistic in toxicity to many aquatic organisms. Finlayson and Verrue (1980) conducted long-term and short-term toxicity studies on Chinook salmon (*Oncorhynchus tshawytscha*) using various water concentrations of copper and zinc mixture. They estimated that safe levels of copper and zinc for Chinook salmon would be below 11 and 83 μg/L, respectively. For most animals, the severity of copper toxicity changes greatly depending on the copper: molybdenum ratios in their diet. Many studies have reported that when animals consume low levels of molybdenum in their diet, copper accumulates much faster and causes copper poisoning at lower con-centrations. High concentrations of molybdenum, on the other hand, are known to induce copper deficiencies. A low-molecular-weight protein, metallothionein, also plays an important role in the transport, storage, and detoxification of copper (Hamilton and Mehrle 1986). Metallothionein synthesis is induced in most vertebrates and some plants when they are chronically or acutely exposed to copper and other heavy metals. It provides protection against copper by sequestering copper more efficiently.

Regulatory Standards

Standards and criteria established by the U.S. Environmental Protection agency are listed in table 13. For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

Table 13.—U.S. Environmental Protection Agency standards and criteria for copper

(See Appendix II for explanation of terms. Source: EPA, 1985, 1995)

Status	EPA priority pollutant; carcinogencity unknown
Drinking water MCL	1,300 µg/L (may vary with treatment technique)
Freshwater criteria (hardness dependent)[1]	
At hardness of 50 mg/L $CaCO_3$	9.2 µg/L for acute exposure 6.5 µg/L for chronic exposure
At hardness of 100 mg/L $CaCO_3$	18 µg/L for acute exposure 12 µg/L for chronic exposure
At hardness of 200 mg/L $CaCO_3$	34 µg/L for acute exposure 21 µg/L for chronic exposure

[1] Official criteria are given as hardness-dependent equations; values listed here are examples that result from these equations at the stated hardness levels. The criterion for acute exposure is equal to $e^{[0.9422\{\ln(\text{hardness})\}-1.464]}$; that for chronic exposure equals $e^{[0.8545\{\ln(\text{hardness})\}-1.465]}$.

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DDT

Description

DDT is a synthetic organochlorine compound, which has been used extensively for insect control throughout the world. Its technical name is dichlorodiphenyltrichloroethane or, more precisely, 2,2-*bis*(*p*-chlorophenyl)-1,1,1-trichloroethane. It has a molecular formula of $C_{14}H_9Cl_5$, a molecular weight of 354.5, and a melting point of 108 °C. It is insoluble in water but very soluble in ethanol and acetone. Technical grade DDT consists of a mixture of isomers, especially *p,p'*-DDT and *o,p'*-DDT (see figure 1); it is a cream-colored to gray powder with a faint fruitlike odor.

DDD and DDE (figure 1) are metabolites of DDT (Klaassen et al. 1986). These two breakdown products and DDT are often found together in the environment and are referred to collectively as total DDT.

Occurrence

DDT, DDD, and DDE are synthetic compounds and have no natural sources. DDT was synthesized as early as 1874, but its insecticidal properties were not discovered until 1939. DDT was patented for use in 1942 and was used during World War II to control lice and other insects on humans (Klaassen et al. 1986, EPA 1975). It was used most extensively during the 1950's and '60s, mainly to control insects on crops and to check vector-borne diseases, such as malaria, in humans. The domestic use of DDT peaked in 1959, but production did not peak until 1963. Since that time, exports of DDT have exceeded domestic use. DDT was banned in the United States in 1972, primarily due to its environmental effects (EPA 1975), but is very persistent in the environment and is still detected in many biochemical and geochemical surveys. In

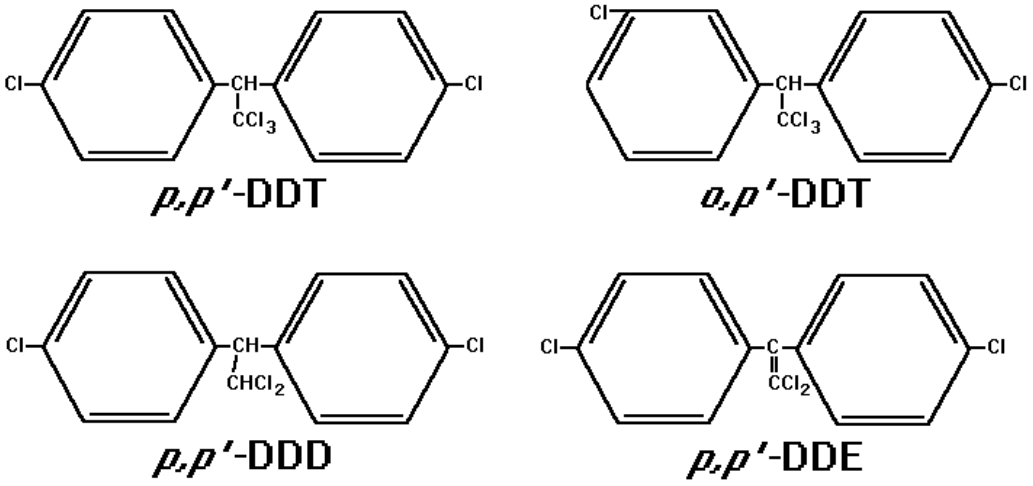

Figure 1.—Chemical structure of the two most common isomers of DDT and of the metabolites DDD and DDE.

addition, DDT is still in use in many parts of the world and is transported into the United States through animal migration and air and water movement.

Some DDT residues are derived from dicofol, an organochlorine pesticide with a structure similar to DDT, that has been used historically to treat mites in citrus orchards and cotton fields (Clark 1990). Studies conducted in 1982 found that dicofol products contained up to 15 percent total DDT. Although toxicity testing indicated that dicofol products were predominantly less toxic than DDT to fish, crustaceans, insects, birds, and mammals, EPA restricted the amount of total DDT contamination in dicofol to 2.5 percent, effective in 1986, and then reduced it further—to 0.1 percent—in 1989.

Summary of Effects

Background concentrations, effect levels, and criteria for the protection of fish and wildlife for DDD, DDE, and DDT are presented below for both physical media (surface water, sediment, and soil) and biotic media (aquatic organisms, terrestrial invertebrates, amphib-ians and reptiles, birds, and mammals). A summary of effect levels of DDT and its metabolites is presented in table 14. This summary is based on the available data, and ranges of effects were established using the following guidelines:

- No effect—studies reporting no observed effect levels or concentrations (NOELs and NOECs) and no observed adverse effect levels or concentrations (NOAELs and NOAECs)

- Level of concern—studies reporting some effect, including lowest observed effect and lowest observed adverse effect levels and concentrations (LOELs, LOECs, LOAELs, LOAECs), effective concentrations at which a certain percent of the test species showed an effect (e.g., EC25 or EC50), median effect concentrations where mortality was not the endpoint (e.g., MD25)

- Toxicity threshold—studies reporting mortality as the endpoint, such as lethal concentrations and doses (LC50s and LD50s)

In some cases, there is a gap or an overlap between levels of concern and toxicity thresholds. This may be the result of interspecies differences or of a lack of data reporting effects between the two ranges.

Field Cases

The persistence of organochlorine pesticides in the environment can be measured in different ways, including sampling and analysis of plants and wildlife (e.g., food items, predator species, or eggs) or abiotic media (e.g., surface water, sediment, or soil). The field studies described in this section include examples of these different measurements.

The persistence of DDT in surface water, sediment, and fish from the Yakima River drainage system in Washington was evaluated periodically from 1968 to 1982 by the U.S. Geological Survey and in recent studies by the State of Washington, Department of Ecology (Johnson et al. 1988). The primary source of DDT in the Yakima River was irrigation water runoff. At certain times of the year, irrigation water accounts for up to 80 percent of the water in the Yakima River.

The predominant compounds found during 1985 sampling of the Yakima River were as follows:

Table 14.— Summary of comprehensive effects: DDD, DDE, and DDT

["—" indicates that endpoints are not reported in available literature; dw = dry weight; ww = wet weight; bw, body weight. See Appendix II for explanation of other abbreviations and technical terms]

Medium	Compound	No effect	Level of concern	Toxicity threshold	Explanation
Water (µg/L)	Total DDT	—	—	0.013	For freshwater organisms (EPA 1996, ecotoxicity threshold).
		<0.3	0.3–800,000	—	For aquatic plants (ORNL 1996, lowest chronic value; EPA 1980, reduced growth).
	DDT	—	0.016	0.36	For *Daphnia* (see table 15).
	DDD	—	1.69–3.99	—	For fish (see table 15).[1]
	DDE	—	—	4,400	
	DDT	—	0.008–0.2	0.2	
Sediment (µg/kg dw)	DDD	—	8–110	6,000	Persaud et al. (1993), lowest (8) and severe (6,000) effect levels; ORNL 1996 screening criterion (110).
	DDE	—	2.2–27	19,000	Long et al. (1995) ERLs and ERMs; Persaud et al. (1993) severe effect levels.
	Total DDT	—	1.5–46	12,000	
Soil (mg/kg dw)	DDE	—	1.5	—	Toxic to *Lumbricus* (Cathey 1982).
Terrestrial inverte-brates (mg/kg dw)	Total DDT	—	32	—	Minimum hazardous level for birds (Beyer and Gish 1980).
Reptiles/amphibians	—	—	—	—	
Terrestrial birds, diet (mg/kg dw)	DDT	—	5–25	311	White-throated sparrow delayed development; LC50 for ring-necked pheasant (table 16).
	DDE	—	4	825	Bengalese finch impaired fledging; bobwhite quail LC 50 (table 16)
Raptor eggs (mg/kg ww)	DDE	—	3–16	—	Bald eagle eggshell thinning, reduced productivity (table 16).
Waterfowl diet (mg/kg dw, insectivores)	DDT	—	—	200	Mallard 95–100% lethality (Davison and Sell, 1974).
	DDE	—	10–30	3,572	Black duck eggshell thinning; mallard LC50 (table 16).
Waterfowl eggs (mg/kg ww, insectivores)	DDE	—	46–144	—	Black duck eggshell thinning (Longcore et al. 1971).
Waterfowl eggs (mg/kg ww, omnivores)	DDE	—	0.25–20	—	White-face ibis eggshell thinning (table 16).
Waterfowl eggs (mg/kg ww, piscivores)	DDE	—	0.62–66	—	Eggshell thinning, hooded merganser and brown pelican (table 16).
Mammals, diet (mg/kg bw/day)[1]	DDT	—	0.26–32.5	—	Mouse LOAELs (table 17).
		—	—	40	Bat LD50 (Clark and Stafford 1981)
		40	40–83	—	Hamster NOAEL & LOAEL (table 17)
		16	16–80	—	Dog NOAEL & LOAEL (Lehman 1965)
	DDD	107	—	—	Mouse NOAEL (NCI 1978).
Mammals, tissue (mg/kg dw)	DDT	—	—	210	LC50 for shrews (Blus 1978).

[1] Fish and mammalian effect levels vary widely depending on the endpoint and species. See tables 15 and 17 for species-specific toxicity information.

- Surface water
  River main stem: *p,p'*-DDE
  Tributaries: *p,p'*-DDE and *p,p'*-DDT

- Sediment: *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, and *p,p'*-DDD

- Fish: *p,p'*-DDE

The predominant metabolite found in surface water and fish collected from the main stem of the Yakima River was *p,p'*-DDE. In fish, it accounted for approximately 88 percent of the total DDT. These findings in fish and surface water from the main stem of the Yakima River indicate that DDT from historical releases has remained in the ecosystem for many years. In surface water collected from tributaries to the river and in sediment collected from both the main stem and from the tributaries, the concentrations of *p,p'*-DDT were equal to or greater than those of *o,p'*-DDT. In addition, *p,p'*-DDT to *o,p'*-DDT ratios in sediment were similar to those in technical-grade DDT (5:1). These ratios indicate that most of the DDT found in the Yakima River basin is under-graded material and suggest that DDT has a long half-life in these areas—perhaps near the upper end of the 4–30 year range proposed by Johnson et al. (1988).

Studies of other river drainages have also shown that levels of *p,p'*-DDT can remain high years after the last known usage. These include sediment from Puget Sound; surface water and sediments in Wisconsin streams; soils in New Mexico and Texas; and sediment, fish, and soil in California agricultural areas (Johnson et al. 1988). In addition, the occur-rence of *o,p'*-DDT and *p,p'*-DDT in ratios resembling technical-grade DDT indicates that *o,p'*-DDT is much more persistent than originally expected. Early studies of *o,p'*-DDT did not show it to be such a persistent com-pound because of difficulties in distinguishing it from other interfering compounds in the packed columns that were used in older analytical procedures (Johnson et al. 1988, Pham et al. 1993).

The occurrence and distribution of DDT in the St. Lawrence River in Quebec, Canada, also follows the trends observed in watersheds in the United States. Agricultural use of DDT in Canada peaked in 1969 but was banned during the 1970's. Restricted uses were still allowed until 1990 (Pham et al. 1993). Observed levels of *o,p'*-DDT in the St. Lawrence River indicated that *o,p'*-DDT was more persistent than *p,p'*-DDT. In addition, *p,p'*-DDT was found in the water column but not in the sediment. This may have been a result of dilution of suspended particulates carrying the *p,p'*-DDT as they settled through the water column or a result of the more rapid degradation of *p,p'*-DDT in sediment than in surface water. The pre-dominant degradation products of *p,p'*-DDT in sediment tend to be *p,p'*-DDE in the top aerobic layers and *p,p'*-DDD in the bottom anaerobic layers. It was hypothesized that *o,p'*-DDT would follow the same degradation processes as *p,p'*-DDT but at a slower rate (Pham et al. 1993).

The persistence of DDT in soil and earth-worms was studied for a 20-year period at the Patuxent Wildlife Research Center in Maryland (Beyer and Gish 1980, Beyer and Krynitsky 1989). DDT was sprayed on two replicate hay fields at a concentration of 9.0 kilograms per hectare. Concentrations in earthworms were measured several times throughout the 20-year period and half-lives (amount of time for the concentrations to decrease by 50 percent) were calculated for DDT and its metabolites in soil and earth-worms. Earthworms of various species and ages were collected from the test plots because presumably predators would not be selective in the type or age of the earthworms they fed on. Species collected included *Aporrectodea turgida*, *Aporrectodea trapezoides*, *Allolobophora chlorotica*, and *Lumbricus terrestris*. DDT metabolites found in soil 11 years after application included *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, and trace amounts of *p,p'*-DDD (Beyer and Gish 1980). DDT metabolites

found in earthworms 11 years after applica-tion included *p,p'*-DDT, *p,p'*-DDD, and *p,p'*-DDE. The half-life for total DDT in both soil and earthworms was 3.2 years (Beyer and Gish 1980). DDE was the most persistent DDT metabolite in earthworms (Beyer and Krynitsky 1989). DDE concentrations in earthworms increased during the first 3 years of the experiment to 13 mg/kg dry weight (dw) and then decreased through the remaining 17 years to 1.2 mg/kg dw, which was 9 percent of the peak concentration.

Abiotic Factors Affecting Bioavailability

Water

DDT and its metabolites are found in most surface water bodies, especially those that are downstream from tributaries draining urban and agricultural areas (EPA 1975). DDT can be transported to previously unimpacted water bodies through soil erosion, movement of suspended particulates, accumulated residues in free-swimming aquatic organisms, and rainfall carrying volatilized DDT.

Because of the low water solubility of DDT (1.2 µg/L), the water column primarily serves as a transfer mechanism between con-taminated sediments and aquatic organisms (EPA 1975). DDT present in the water column is directly available for uptake by aquatic plants and animals at all levels of the food web. However, for animals in the higher trophic levels, transfer of DDT through the water column is probably less important than food-chain transfer, which is discussed in later sections.

The toxicity of DDT in water can be affected by several factors, including temperature and pH. Studies using *Daphnia pulex* indicate that *p,p'*-DDT was significantly more toxic at 20 °C than at 17 °C (Smith et al. 1988). Lower temperatures may also increase toxicity. DDT was seven times more toxic to the scud and twice as toxic to *Daphnia* sp. at 5 °C than at 21 °C (EPA 1975).

Studies along the Yakima River in Washington and the St. Lawrence River in Quebec evaluated the persistence of DDT and its derivatives in water and in other media, as described in the "Field Cases" section, above.

Sediment

Sediments function as the primary sink for DDT and its metabolites (EPA 1975). In general, waterborne DDT concentrations in excess of 1.2 µg/L (water solubility) will either adsorb to or precipitate onto the bottom sediments. The DDT in the sediments is then available for direct contact or ingestion by bottom-dwelling organisms. The DDT can also be redissolved back into the water column whenever the water concentration falls below the saturation point. The per-sistence and degradation of DDT in sediment can be affected by several factors, including pH, organic carbon content, turbidity, and oxygen content.

Sediment quality guidelines developed by Long et al. (1995) were based on comparisons of effects levels for various organisms exposed to DDT-contaminated sediments and sedi-ment characteristics. These guidelines were developed for estuarine and marine sediments but are considered useful as screening levels for freshwater sediments. The distributions of effects data were evaluated to develop two guidelines: (1) an effects-range-low (ERL) guideline, the lower 10th percentile of the effects data for each chemical, and (2) an effects-range-median (ERM) guideline, the 50th percentile of the effects data. These two guidelines can be used to predict three levels of potential toxicity for aquatic organisms exposed to contaminated sediments. Adverse effects should be rare at concentrations below the ERL but may occur between the ERL and the ERM and are very likely above the ERM.

Concentrations below the ERL typically represent conditions where adverse effects would rarely occur. Concentrations between the ERL and the ERM represent conditions in which adverse effects may occur, and concentrations above the ERM represent conditions in which adverse effects are likely to occur. The ERL and ERM values for DDE and DDT are as follows:

Compound	ERL (µg/kg dw)	ERM (µg/kg dw)
p,p'-DDE	2.2	27
Total DDT	1.58	46.1

Oak Ridge National Laboratory in Tennessee developed sediment-screening criteria based on its own research (ORNL 1996). Its sedi-ment quality benchmarks are calculated using equilibrium partitioning with either secondary chronic water quality criteria or the lowest reported chronic values for fish or daphnids and assumes 1 percent organic carbon. The available values are as follows:

Compound	Secondary chronic value (µg/kg dw)	Lowest chronic values (µg/kg dw)	
		Fish	Daphnids
p,p'-DDD	110	16,865	—
DDT	343	19,280	422

The EPA's Office of Solid Waste and Emergency Response has selected benchmark values for use in screening contaminated sediments at sites being managed under the Comprehensive Environmental Response, Compensation, and Liability Act (EPA 1996). The value for DDT is 1.6 µg/kg dw, based on the ERL developed by Long et al. (1995).

Soil

The persistence of DDT in soil can be affected by several factors, including method of application, soil type, soil fertility, type of formulation, topography, climatic conditions, farming practices, soil pH, and organic carbon content (EPA 1975). The amount of time for concentrations of DDT to be reduced by 95 percent in soils ranged from 4 to 30 years, with an average time of 10 years (Edwards 1966).

DDT applied to the surface may be subject to volatilization, but as it becomes bound to the soil, its volatility decreases (Beyer and Gish 1980). DDT mixed with the soil is even less volatile. In addition, because of the low water solubility of DDT, it does not tend to leach through the soil.

Davis (1971) found that the accumulation of DDE and DDT in earthworms tends to decrease as the organic carbon content of the soil increases. The earthworm *Allolobophora caliginosa* accumulated the highest concentrations of DDT in soils with 2 percent organic carbon and showed low accumulation in soils with 21 percent organic carbon. The accumulation of DDE, however, increased as organic carbon increased up to 3.6 percent and then decreased with increasing organic carbon. The cause of the increased DDE accumulation up to 3.6 percent organic carbon was not determined, but it may have been due to other factors such as pH, soil density, and soil moisture, which can influence the feeding activity of the worms. The differences in accumulation between soils with low organic carbon (2 percent to 3.6 per-cent) and those with higher organic carbon (6.5 percent to 21 percent) were statistically significant for both DDE and DDT.

A 20-year study at the Patuxent Wildlife Research Center in Maryland, described above in the "Field Cases" section, examined the persistence of DDT in soil and earthworms.

Biotic Effects

Plants

Aquatic Plants.—Little toxicity information was found concerning toxicity of DDT to aquatic

plants. ORNL (1996) reported a lowest chronic value of 0.3 µg/L for aquatic plants but listed no specific species. EPA (1980, based on the work of Sodergren 1968) also reported a chronic value (for reduced growth and unusual morphology) of 0.3 µg/L in the green alga *Chlorella* sp. Other subacute effects of DDT reported by EPA (1980) include reduced growth in *Anacystis nidulans* at 800 µg/L and in *Scenedesmus quadricauda* at 100 µg/L, and inhibited photosynthesis in *Selenastrum capricornutum* at 3.6 µg/L.

Terrestrial Plants.—No toxicity information was found for effects of DDD, DDE, or DDT on terrestrial plants.

Aquatic Invertebrates

DDT is toxic to most aquatic organisms, and arthropods have been shown to be very sensitive to low levels of DDT. Studies using static and flow-through tests have established median lethal concentrations (48-h) for several freshwater arthropods, as shown in table 15.

Smith et al. (1988) conducted 48-hour static acute toxicity tests for several chemicals found in the Great Lakes. Chemical toxicity was ranked in comparison to that of *p,p'*-DDT, which had been found in fish tissues collected from the Great Lakes. The endpoint selected was an EC50 of immobilization or no movement when prodded. The EC50 for *Daphnia pulex* was 1.1 µg/L.

The marine crustacean *Artemia salina* has gained popularity for use in short-term toxicity testing. The toxicity of various chemicals to this species has been used to predict toxicity to *Daphnia magna* and marine copepods. Sanchez-Fortun et al. (1995) found that the relative order of toxicity of DDE, DDT, and other organochlorine pesticides (i.e., dieldrin and lindane) was the same for *A. salina* as for other crustaceans, but *A. salina* was much more tolerant to DDE and DDT than most other aquatic organisms. This tolerance can range from 7 to 27 times that of other marine decapods. Sanchez-Fortun et al. also found that sensitivity to DDE and DDT increased with increasing age of the *A. salina* larvae. Three-day-old larvae were twice as sensitive as 1-day-old larvae (table 15).

EPA's data base for *p,p'*-DDT contains more than 40 acute toxicity values for various aquatic organisms (EPA 1980). These range from 0.36 µg/L for *Daphnia pulex* to 1,230 µg/L for the planarian *Polycellis felina*. The AQUIRE data base for *p,p'*-DDD and *p,p'*-DDE has several acute and one chronic toxicity value for aquatic organisms (EPA 1984). For *p,p'*-DDD the acute toxicity values range from 1 µg/L for *Bosmina longirostris* to 2,360 µg/L for a flatworm. The chronic toxicity value reported for *p,p'*-DDD is 0.3 µg/L for *Nitocra spinipes*. For *p,p'*-DDE, the acute values range from 0.68 µg/L for the scud (*Gammarus* sp.) to 4,400 µg/L for the fathead minnow (*Pimephales promelas*).

Swartz et al. (1994) evaluated the sediment toxicity and abundance of amphipods at several sites along the Lauritzen Channel and in parts of Richmond Harbor in California. The property adjacent to these portions of the channel was historically used for the formulation and grinding of DDT. Although much of the contaminated sediment was removed, a concentration gradient still exists. Swartz et al. measured toxicity to the amphipod *Eohaustorius estuarius* and the field abundance of amphipods at several sites, and their results are shown in table 15. For comparison purposes, Swartz et al. also tested the toxicity of sediments collected at other sites in the United States. Table 15 includes LC50s for *Hyalella azteca* exposed to sediments from a freshwater stream in Alabama and for *Rhepoxynius abronius* exposed to sediments from the Palos Verdes Shelf, California.

Table 15.—DDD, DDE, and DDT effects on aquatic organisms

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical	Concentration (µg/L in surface water, except as noted)	Effects	Reference
Aquatic plants				
Mixed macrophytes	DDT	0.3	Lowest chronic value	ORNL 1996
Aquatic invertebrates				
Daphnia pulex	DDT	0.36	48-h LC50	EPA 1975
	p,p'-DDT	1.1	48-h EC50 - immobilized	Smith et. al. 1988
Daphnia magna	DDT	4.4	48-h LC50	EPA 1975
	DDT-tech	1.1	48-h LC50	Randall et al. 1979
Daphnia sp.	DDT	0.016	Estimated lowest chronic value	ORNL 1996
Bosmina longirostris	*p,p'*-DDD	1	Acute LC50	EPA 1984
Marine crustacean *Artemia salina*	*p,p'*-DDE	159,000	24-h LC50 (1-day-old larvae)	Sanchez-Fortun et al. 1995 (DDE and DDT test solutions prepared by dissolving in DMSO and diluting in water)
		116,000	24-h LC50 (2-day-old larvae)	
		94,270	24-h LC50 (3-day-old larvae)	
	p,p'-DDT	43,010	24-h LC50 (1-day-old larvae)	
		17,040	24-h LC50 (2-day-old larvae)	
		16,400	24-h LC50 (3-day-old larvae)	
Scud (*Gammarus* sp.)	DDT	2.1	48-h LC50	EPA 1975
	p,p'-DDE	0.68	Acute LC50	EPA 1984
Caddisfly	DDT	3.4	48-h LC50	EPA 1975
Mayfly	DDT	0.3	48-h LC50	EPA 1975
Oyster	DDT	7	Reduced shell growth by 50%	EPA 1975
Amphipod *Hyalella azteca*	*p,p'*-DDD	0.19	10-d LC50	Phipps et al. 1995; Hoke et al. 1994
	p,p'-DDE	1.66	10-d LC50	
	p,p'-DDT	0.07	10-d LC50	
	p,p'-DDD	1.08 in pore water	10-d LC50	Hoke et al. 1994
	Total DDT	2,580 mg/kg C in sed.[1]	10-d LC50	Swartz et al. 1994
Amphipod *Eohaustorius estuarius*	Total DDT	2,500 mg/kg C in sed.[1] 300 mg/kg C in sed.[1] 100 mg/kg C in sed.[1]	10-d LC50 Toxic threshold Reduced abundance	Swartz et al. 1994
Amphipod *Rhepoxynius abronius*	Total DDT	1,040 mg/kg C in sed.[1]	10-d LC50	Swartz et al. 1994
Midge *Chironomus tentans*	*p,p'*-DDD	0.18	10-d LC50	Phipps et al. 1995
	p,p'-DDE	3		
	p,p'-DDT	1.23		
Oligochaete *Lumbriculus variegatus*	*p,p'*-DDE	>3.27	10-d LC50	Phipps et al. 1995
Planarian *Polycellis felina*	*p,p'*-DDT	1,230	Acute LC50	EPA 1980
Flatworm	*p,p'*-DDD	2,360	Acute LC50	EPA 1984
Nitocra spinipes	*p,p'*-DDD	0.3	Chronic toxicity	EPA 1984

[1] Milligrams of DDT per kilogram of organic carbon in sediment.

Table 15.—DDD, DDE, and DDT effects on aquatic organisms—Continued

Species	Chemical	Concentration (µg/L in surface water, except as noted)	Effects	Reference
Fish				
Mixed species	DDD	1.69	Lowest chronic value	ORNL 1996
		3.99	Estimated EC20	
		0.61	Population EC20	
	DDT	0.73	Lowest chronic value	
		0.35	Estimated EC20	
		0.008	Sensitive species EC20	
Bluegill sunfish (*Lepomis macrochirus*)	DDT-tech	3.4	96-h LC50	Randall et al. 1979
	DDT	0.2–1.0	96-h LC50	Ellgaard et al. 1977
		0.008	Increased locomotor activity	
	DDT	5.8	96-h LC50 at 7°C	Mayer and Ellersieck 1988
		1.6	96-h LC50 at 29°C	
Carp (*Catla catla*)	DDT	6,800	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Carp (*Cirrhinus mrigala*)	DDT	6,300	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Carp (*Labeo rohita*)	DDT	6,400	96-h LC50	Kulshrestha et al. 1986
		3,000–3,500	MATC	
Catfish *Heteropneustes fossilis*	DDT-tech	500	Decrease in white blood cell count	Mustafa and Murad 1984 (DDT dissolved in acetone and diluted with water)
		3,020	72-hr LC50	
		2,950	96-hr LC50	
		3,000	Erratic swimming, jerky movement	
		3,020	72-hr LC50	
		3,550	48-hr LC50	
Cichlid - Tilapia *Oreodhromis spilurus*	DDT	80	96-hr LC50,	Parkinson and Agius 1988
		190	72-hr LC50	
		250	48-hr LC50	
Fathead minnow *(Pimephales promelas)*	*p,p'*-DDE	4,400	Acute LC50	EPA 1984
	Total DDT	1.5 (water only)	Chronic exposure LC50	Jarvinen et al. 1977
		0.9 (+46 µg/g in food)	Chronic exposure LC50	
		0.9	MATC (DDT in water only)	
		0.4	MATC (DDT in water and food)	
		2	Reduced embryo hatchability	
Flounder (*Platichthys flesus*)	DDT	12.5 mg/kg bw in food	Hyperactivity; 20-fold increase in activity	Bengtsson and Larsson 1981
		1 mg/kg (in brain)	Hyperactivity	
Humpback salmon (*Oncorhynchus gorbuscha*)	DDT	1.32	Increased enzymatic activity	Andryushchenko and Khokhryakov 1982
Loach (*Misgurnus anguillicaudatus*)	DDT	350	24-hr LC50	Yang and Sun 1977
Sarotherodon mossambicus	DDT	1	Change in thyroid follicle organization and structure	Shukla and Pandey 1986

Effluent discharges from a DDT manufacturing company have affected both surface water and sediment in the Huntsville Spring Branch of the Indian Creek stream system in Alabama. Hoke et al. (1994) measured the toxicity of DDT and its metabolites to *Hyalella azteca* exposed to surface water.

Terrestrial Invertebrates

Historical studies of terrestrial invertebrates have found that earthworms are much more tolerant of organochlorine pesticides than arthropods (Davis 1971). Cathey (1982) estimated an effective toxic soil concentration of DDE to *Lumbricus*, at 1,500 μg/kg.

Laboratory studies on the uptake and accumulation of DDE and DDT were conducted on two earthworms (*Lumbricus terrestris* and *Allolobophora caliginosa*) that have different feeding patterns (Davis 1971). *L. terrestris* feeds on the soil surface and on vegetation, whereas *A. caliginosa* feeds primarily on soils below the surface. The uptake of DDT from soil by *L. terrestris* indicated that DDT concentrations in worms rose from 0.15 mg/kg (at 1 mg/kg soil residue) to 45 mg/kg (at 64 mg/kg soil residue). The accumulation of DDT increased with increasing soil concentration, and bioconcentration factors ranged from 0.15 to 0.7. DDE was the primary metabolite formed. The proportion of DDE to DDT was approximately 20 percent. The uptake and accumulation of DDT were also measured using apple leaves that had been sprayed with technical grade DDT. Results indicated that accumulation from treated leaves was significantly higher in *L. terrestris* than in *A. caliginosa*. Accumulation from treated leaves was also less than that from treated soil for both species. The differences in accumulation between *L. terrestris* and *A. caliginosa* for different types of DDT application were due to the differences in feeding activity and intake between the two species. When DDT was applied to the soil and cultivated in, A. caliginosa accumulated much higher levels than *L. terrestris* because it ingests relatively more soil and is more active below the surface; but, when DDT was applied to leaves at the soil surface, then *L. terrestris* accumulated higher levels because it is more active at the surface.

The accumulation of DDT in earthworms exposed to contaminated soil in the Rhine delta flood plains indicated that ratios of concentrations in earthworm fat and dry organic matter were independent of octanol-water partition coefficients (K_{ow}) (Hendriks et al. 1995). Earthworms collected from loca-tions along the Rhine delta accumulated DDT at 0.6 times and DDE at 2.3 times the soil concentrations measured at the same loca-tions. Previous studies had indicated that dry weight concentrations of DDE and DDT in field earthworms ranged from 1.8 to 9.2 times the soil concentrations (Ma 1985). When converted to a fat weight basis, this range increased to 2.9 to 15 times the soil concentration. The lower accumulation factors observed in the Rhine delta may have been the result of reduced bioavailability, non-equilibrium conditions, or biotransformation in local populations (Hendriks et al. 1995).

Beyer and Gish (1980) studied the accumulation of DDT in earthworms exposed to treated soils for 11 years. The storage ratios of total DDT in earthworms were calculated for DDE (6), DDD (0.27), *p,p'*-DDT (0.56), and total DDT (5.1). The storage of total DDT in earthworms can lead to harmful effects in higher trophic-level organisms, including birds and mammals. (See "Bioaccumulation".)

Fish

The toxicity and accumulation of DDT in fish are correlated with age, fat content, and body length. Signs of toxicity are similar to those exhibited by insects (Ellgaard et al. 1977). Exposure to lethal concentrations of DDT results in increasing levels of irritability or

excitability followed by muscular spasms, complete loss of equilibrium, convulsions, and eventually death. Toxic effect levels for various species of fish are presented in table 15.

Several studies have evaluated the toxicity and sublethal effects of DDT and its meta-bolites to various fish species (table 15). Ellgaard et al. (1977) found that exposure to DDT concentrations as low as 0.008 µg/L can affect locomotor rates of bluegill sunfish (*Lepomis macrochirus*). Behavioral changes, including erratic swimming, fast jerky movement, and convulsions, were observed in catfish (*Heteropneustes fossilis*) exposed to technical-grade DDT at concentrations greater than 3,000 µg/L (Mustafa and Murad 1984). Changes in thyroid follicle organization and structure were observed at DDT concentra-tions as low as 1.0 µg/L in *Sarotherodon mossambicus* (Shukla and Pandey 1986).

Hyperactivity and abnormal diurnal activity were observed in flounders (*Platichthys flesus*) that were force-fed DDT (Bengtsson and Larsson 1981). Dosages of DDT at 12.5 mg/kg bw resulted in a 20 percent increase in swimming activity, and extractable fat resi-dues in the brain greater than 1 mg/kg resulted in hyperactivity. Andryushchenko and Khokhryakov (1982) evaluated enzymatic changes in humpback (pink) salmon (*Oncorhynchus gorbuscha*) and found that concentrations of DDT at 1.32 µg/L resulted in increases of cytochrome P-450 by 30 percent and cytochrome b5 by 21 percent. In addition, benzpyrenhydroxylase activity was increased threefold.

Mayer and Ellersieck (1988) found a negative correlation between temperature and toxicity for DDD, DDT, dimethrin, methoxychlor, pyrethrins, and pyrethroids. This is opposite of the relationship observed for most other organic chemicals. The differences in toxicity due to temperature have been attributed to changes in respiration rates and chemical absorption, detoxification, and excretion.

Yang and Sun (1977) studied toxicity and rates of absorption of DDT in leaches (*Misgurnus anguillicaudatus*) and found that 96.5 percent of a predetermined concentration of DDT present in water was absorbed within 24 hours. The high rate of absorption is primarily due to the lipophilicity of DDT.

Jarvinen et al. (1977) tested the partial chronic toxicity of DDT to fathead minnows (*Pimephales promelas*). In separate tests, they administered the DDT in diet alone, in water alone, and in both diet and water. Fish fed DDT in the diet had lower survival rates than those fed clean food (table 15). High mortality was observed at two stages: (1) juveniles 45 to 73 days old and (2) spawning male fish. In addition, embryo residue levels and larval mortality rates for offspring of parent fish that were exposed to DDT in both water and food were two times higher than those for offspring of fish that were exposed to DDT only in the water. Tissue residues of DDT in adults were also higher for fish exposed to DDT in water alone than for fish exposed to DDT in diet alone, but results for fish exposed to DDT in both diet and water indicated that residue levels were additive based on the single-exposure studies. An equilibrium between tissue residues and concentrations in food was reached within 56 days. Bioconcentration factors were calculated for diet (1.2) and water (100,000).

In lake trout (*Salvelinus namaycush*) stocked in Cayuga Lake, New York, tissue residues of *p,p'*-DDE increased significantly with both the age of the fish and the fat content (Gutenmann et al. 1992). Fish collected in 1978 averaged 3.06 mg/kg dw for 6-year-old fish and 10 mg/kg dw for 12-year-old fish. Similarly, fish collected in 1991 averaged 0.89 mg/kg dw for 6-year old fish and 1.9 mg/kg dw for 12-year-old fish. In addition, the DDT concentrations in fish tissue showed a marked decrease in both the 6- and 12-year-old fish in the intervening 13 years.

Similar studies at another lake in New York found that residues of *p,p'*-DDE in lake trout were positively correlated to the length of fish (Young et al. 1994). Length accounted for 81 percent of the variation in residue levels. Length can also be used as an indicator of age and, hence, these results agree with those of Gutenmann et al. (1992).

Contrary to the results of the lake trout studies, Larsson et al. (1993) found that total DDT residue levels in northern pike (*Esox lucius*) taken from a eutrophic lake in southern Scandinavia were negatively correlated with age and with muscle or fat content. The decrease with increasing age was most pro-nounced in female pike, and this result was attributed to the seasonal use and elimination of lipids and, hence, of lipophilic pollutants during reproduction and release of eggs. In eggs and ovaries (roe), both the fat content and the contaminant levels were 10 times higher than in muscle. Male pike contained higher levels of contaminants than females
but have lower elimination via gonadal products. Germinal tissue can account for up to 15 percent of the body weight in females but only accounts for approximately 2 percent in males. Male germinal tissue also contains less fat than the ovaries in females. In addition, the largest fat deposits in pike are found in the germinal tissues; their muscle tissue has lower levels of fat (0.6 to 0.8 per-cent) than most other fish, including salmonids. These results differ from those
of the trout studies primarily because trout and other salmonids deposit more fat than pike do in the muscle, adipose, and visceral tissues, and their fat content increases with age. Other fish also tend to use fat deposits as an energy reserve. Pike are more lean, their percent fat content in muscle and germinal tissue is relatively constant throughout life, and they do not use fat deposits as energy reserves. Instead, they catabolize ordinary tissue when fasting. Overall, the uptake and elimination of persistent pollutants such as DDT can vary not only within a species because of sex, age, and size, but also between species because of differences in fat deposition.

Cullen and Connell (1992) studied whole-body residues of total DDT in fish collected from three rivers in New South Wales, Australia. The rivers selected had large plantations and a history of heavy use of chlorohydrocarbon pesticides. Fish species collected included whiting (*Sillago ciliata*), bream (*Acanthopagrus australis*), mullet (*Mugil cephalus*), and carp (*Cyprinus carpo*). At some point during their life history, whiting, bream, and mullet migrate from upstream impacted areas to downstream estuarine or open-sea areas with lower levels of contamination. The tissue levels in these fish increased with age in upstream juveniles but decreased after maturation and migration downstream. The levels of total DDT increased linearly with age for fish (carp) that did not migrate. The effects of migration included both exposure to lower environmental concentrations and the metabolism of fat reserves during migration and spawning. In addition, Cullen and Connell found that fish with higher fat content (mullet) contained significantly higher total DDT residues than fish with a lower fat content (bream and whiting), but variations occurred based on differences in movement patterns and exposure history.

Along the Yakima River of Washington, Johnson et al. (1988) studied the persistence of DDT and its metabolites in fish and in water and sediment, as described above in the "Field Cases" section.

Amphibians/Reptiles

Few studies were found that describe DDT's effects on amphibians and reptiles. Toxic effects can include uncoordinated behavior, loss of equilibrium, restricted development, weight loss, and death (Russell et al. 1995). Spring peepers (*Pseudacris crucifer*) collected from an area of historic DDT application were analyzed

for DDD, DDE, and DDT. All had elevated tissue concentrations (mean concen-trations in μg/kg ww: *p,p'*-DDD, 26.5; *p,p'*-DDE, 1,001; *p,p'*-DDT, 161). The high concentrations indicated that DDT application in the area may have accounted for local extinctions of three other species of frogs and toads (Russell et al. 1995).

Bishop et al. (1994) studied concentrations of DDE in the eggs of the common snapping turtle (*Chelydra serpentina serpentina*) to determine if higher concentrations of DDE were found in eggs produced by larger, older turtles or those with the largest clutch size or mass. The results of this study showed no correlation between the age or size of the female turtle and the amount of DDE found in the eggs. Relationships were previously found between body size of turtles and the concentrations of lipophilic compounds in fat or liver. The differences of DDE found in different clutches may simply reflect individual preferences in food and feeding locations. In addition, the lipids used for egg production may be derived from daily dietary intake shortly before egg production rather than from stored lipids in the female.

Bishop et al. (1995a) also found that concentrations of DDE varied widely in common snapping turtle eggs collected from the same clutch. The first five eggs laid in the nest contained the highest concentrations of DDE on both a wet-weight and a lipid-weight basis. The first-laid eggs of the snapping turtles tended to have greater lipid content and consequently greater organochlorine content than later eggs. This trend is opposite from what has been observed in some birds (e.g., herring gulls), in which eggs with the highest lipid and organochlorine content were those laid later.

Birds

The toxicity and accumulation of DDT and its metabolites are of primary concern in birds. During the early 1970's, DDT and its metabolites were probably present in the tissues of essentially all wild birds in the world (Fleming et al. 1983). Since then, however, many studies have shown that DDE residue levels in birds have been decreasing in many parts of the United States (Mora 1995). These chemicals can accumulate in fat after even brief, low-level exposures. In general, birds that feed on fish or other birds have greater tissue residues than those that feed on vegetation or seeds, and DDE is more common than either DDT or DDD in bird tissues (Stickel 1973, Blus 1996). Other adverse effects associated with DDT poisoning in birds include reproductive impairment, reduced fledging success, and eggshell thinning. Toxic effect levels for various types of birds are presented in table 16.

The storage of DDT in various tissues can be a function of the exposure concentrations (Stickel 1973). Continuous exposure to sublethal concentrations tends to result in tissue residues that are directly correlated to each other. The balance of tissue residues is broken when the exposure is to lethal concentrations or when stored tissue residues are metabolized and released back into the system at lethal concentrations. The residue level in the brain has been shown to be the best criterion for establishing that total DDT was the cause of death, and lethal levels of brain residues are similar for many different species of birds. Residue levels in the liver are a better indication of recent exposure and can be correlated either to an environmental dose or to metabolism of stored residues. Whole-body residue levels indicate the storage reserve and can be used to estimate the potential for adverse effects from metabolism to lethal levels or from normal metabolism and excretion (Stickel 1973).

Through their review of earlier studies, Noble and Elliot (1990) derived critical levels of DDE, resulting in acute toxicity, in the brains, livers, and eggs of several raptors. Critical levels in the brain and liver were 250 and 100 mg/kg

Table 16.—DDD, DDE, and DDT impacts to birds

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Raptors					
American kestrel	DDE	10 ww	Egg	Minimum critical levels (lowest levels at which productivity is affected), determined by review of available literature	Noble and Elliot 1990
Bald eagle		6 ww			
Golden eagle		10 ww			
Falcons[2]		10 ww			
Hawks[3]		10 ww			
Merlin		5 ww			
Northern harrier		10 ww			
Owls[4]		10 ww			
Osprey		4 ww			
Prairie falcon		1.2 ww			
Mixed species		250 ww	Brain		
		100 ww	Liver		
American kestrel	DDE	3 dw	Diet	13% eggshell thinning; reduced pipping	Lincer 1992
Bald eagle (*Haliaeetus leucocephalus*)	DDE	3.3 ww	Egg	8.8% thinner than pre-1947 eggs from Southern CA and Baja	Grubb et al. 1990
		3–5 ww		Depressed productivity	Wiemeyer et al. 1984
		5 ww		10% eggshell thinning	
		15 ww		No productivity	
		3.6–6.3 ww		50% reduction in productivity	Wiemeyer et al. 1993
		>6.3 ww		75% reduction in productivity	
		16 ww		15% eggshell thinning	
Osprey (*Pandion haliaetus*)	DDE	4 ww	Egg	15% eggshell thinning	Noble and Elliot 1990
		2 ww		10% eggshell thinning	Wiemeyer et al. 1988
		4.2 ww		15% eggshell thinning	
		8.7 ww		20% eggshell thinning	
Peregrine falcon (*Falco peregrinus*)	DDE	15 ww	Egg	Depressed productivity	Peakall et al. 1975
Terrestrial birds					
Bengalese finch (*Lonchrua striata*)	DDE	4 ww	Diet	Reduced fledging success	Jeffries 1971
	DDT	8 dw			
Bobwhite quai (*Colinus virginianus*)	DDE	825 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	611 dw		5-d LC50	
	Total DDT	25–200 dw	Diet	3.1 mg/kg in brain; no effect	Hill et al. 1971
		400 dw		Weight loss	
		800 dw		7.5 mg/kg in brain; tail tremors, irregular head carriage	
		1,600 dw		Stumbling gait, tail tremors, head bobbing, loss of equilibrium, death	
		1,170-1,610 dw		5-d LC50	

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Terrestrial birds—Continued					
Blue jay (*Cyanocitta cristata*)	Total DDT	415 dw	Diet	5-d LC50	Hill et al. 1971
	DDT	611 dw	Diet	5-d LC50	Heath et al. 1972
Brown-headed cowbird (*Molothrus ater*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
California quail (*Callipepla californica*)	DDT	595 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Cardinal (*Richmondena cardinalis*)	Total DDT	535 dw	Diet	5-d LC50	Hill et al. 1971
Common grackle (*Quiscalus quiscula*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
Coturnix quail (*Coturnix japonica*)	DDE	1,355 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	416 dw		5-d LC50	Hill and Camardese 1986
		568 dw		5-d LC50	Hill et al. 1975; Heath et al. 1972
		841 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
House sparrow (*Passer domesticus*)	Total DDT	415 dw	Diet	5-d LC50	Hill et al. 1971
Red-winged blackbird (*Agelaius phoeniceus*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
Ring-necked pheasant (*Phasianus colchicus*)	DDE	829 dw	Diet	5-d LC50	Hill et al. 1975
	DDT	311 dw		5-d LC50	
		1,334 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Rock dove (*Columba livia*)	DDT	>4,000 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
Starling (*Sturnus vulgaris*)	DDE	1,500 dw	Diet	300–400 mg/kg in brain residue; increased likelihood of death	Stickel et al. 1984
White-throated sparrow (*Zonotrichia albicollis*)	DDE	5–25 ww	Diet(?)	Delayed development of migratory condition	Mahoney 1975
Waterfowl—Insectivores					
Black duck (*Anas rubripes*)	DDE	10 dw	Diet	Increased egg residues; 20% eggshell thinning (over 2 years)	Longcore and Stendell 1977
				Egg residues 46.3 mg/kg; 10% shell cracking	Longcore et al. 1971
		30 dw		Egg residues 144 mg/kg; 21% shell cracking	
		46.3 ww	Egg	Eggshell thinning of 18–29%	
		144 ww		Eggshell thinning of 24–38%	
Clapper rail (*Rallus longirostris*)	*p,p'*-DDT	1,612 dw	Diet	5-d LC50 (male)	Van Velzen and Kreitzer 1975
		1,896 dw		5-d LC50 (female)	
		30 ww	Brain	Lower lethal limit diagnostic of DDT-related death	

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Insectivores—Continued					
Common goldeneye (*Bucephala clangula*)	DDE	0.52 ww	Egg	Egg breakage; 15.4% eggshell thinning	Zicus et al. 1988
Mallard (*Anas platyrhynchos*)	DDT-tech	200 dw	Diet (12-week exposure)	95% lethality; 20% eggshell thinning	Davison and Sell 1974
	p,p'-DDT	200 dw		100% lethality after 343 d	
	p,p'-DDT	1,202 dw	Diet	20-d LC50 (5-d-old ducklings)	Friend and Trainer 1971
		1,622 dw		20-d LC50 (30-d-old ducklings)	
		1,419 dw		20-d LC50 (adults)	
	DDT	>2,240 bw	Oral dose	LD50 (single dose)	Hudson et al. 1984
	DDT	1,869 dw	Diet	5-d LC50	Hill et al. 1975
	DDE	3,572 dw	Diet	5-d LC50	
Waterfowl—Omnivores					
Black-crowned night-heron (*Nycticorax nycticorax*)	DDE	<1 ww	Egg	6.5% eggshell thinning	Findholt and Trost 1985
		1.01–4 ww		5.1% eggshell thinning	
		4.01–8 ww		10.2% eggshell thinning	
		>8 ww		15.6% eggshell thinning	
		8 ww		Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1984, 1985
		8.62 ww		8–13% thinner than pre-1947 eggs	Ohlendorf and Marois 1990
		11–12 ww		36–39% hatching success; 14–17% eggshell thinning	Price 1977
		8–12 ww		27–58% decrease in nesting success	Blus 1984
		12 ww		Critical level for reproductive success based on field studies	
		25–50 ww		Total reproductive failure	
		36 ww		18% thinning based on regression analysis	
		54 ww		20% thinning; critical level for reproductive success based on regression analysis	
Great egret (*Casmerodius aalbus*)	DDE	24 ww	Egg	8–13% thinner than pre-1947 eggs	Ohlendorf and Marois 1990
Green-backed heron (*Butorides striatus*)	DDE	5–10 ww	Egg	Reduced hatching success	White et al. 1988
Red-necked grebe (*Podiceps grisegena*)	DDE	6.68 ww	Egg	Low egg viability; 6.5% eggshell thinning; reduced fledging success	De Smet 1987
Sandhill crane (*Grus canadensis*)	DDT	>1,200 bw	Single oral dose	LD50	Hudson et al. 1984
Snowy egret (*Egretta thula*)	DDE	5 ww	Egg	Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1985

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Omnivores—Continued					
Western grebe (*Aechmophorus occidentalis*)	DDE	1 ww	Egg	1% thinning	Boellstorff et al. 1985
		5.4 ww		2.3% eggshell thinning; reduced productivity	Lindvall and Low 1980
White-face ibis (*Plegadis chihi*)	DDE	3 ww	Egg	Reduced clutch size, decreased productivity, egg breakage	Henny et al. 1985
		0.94 ww		3.2% eggshell thinning	King et al. 1980
		0.25 ww		4.5% eggshell thinning	
		4–8 ww		15% eggshell thinning	
		8–16 ww		17.4% eggshell thinning	Henny and Herron 1989
		16–20 ww		27.8% eggshell thinning	
Waterfowl—Piscivores					
American white pelican (*Pelicanus erythrorhynchos*)	DDE	2 ww	Egg	10–15% thinning in eggs from CA	Boellstorff et al. 1985
Black skimmer (*Rhyncops niger*)	DDE	3.2 ww	Egg	Decreased hatching and fledging success	Custer and Mitchell 1987
		3.4 ww		5% thinning, but no adverse effect on reproductive success	King et al. 1991
Brown pelican (*Pelicanus occidentalis*)	DDE	1 ww	Egg	5–10% shell thinning (FL, SC)	Blus et al. 1974, 1979
		2 ww		11% eggshell thinning (FL)	
		3 ww		16% eggshell thinning (SC)	
		5 ww		17% eggshell thinning (SC)	
		3 ww		Reduced productivity	King et al. 1985
		3.2 ww		11% thinner than normal	King et al. 1977
		2.6–3.0 ww		29–40% decrease in nesting success	Blus 1984
		3 ww		Critical level for reproductive success based on field studies	
		>3.7 ww		Total reproductive failure	
		5 ww		18% thinning based on regression analysis	
		8 ww		20% eggshell thinning	
		3 ww		18% eggshell thinning (BC)	Jehl 1973
		8 ww		26% eggshell thinning (BC)	
		25 ww		47% eggshell thinning (BC)	
		66 ww		46% eggshell thinning (BC)	
		59 ww		44% eggshell thinning (CA)	Risebrough 1972
Caspian tern (*Sterna caspia*)	DDE	9.3 ww	Egg	22% hatching failure; 4.6% died in hatching	Ohlendorf et al. 1985
Common tern (*Sterna hirundo*)	DDE	6.67 ww	Egg	17% thinning; hatching failure; embryo mortality	Fox 1976
Double crested cormorant	DDE	10 ww	Egg	20% eggshell thinning	Pearce et al. 1979

Table 16.—DDD, DDE, and DDT impacts to birds—Continued

Species	Chemical species	Concentration (mg/kg)[1]	Where measured	Effects	Reference
Waterfowl—Piscivores—Continued					
Elegant tern (*Sterna elegans*)	DDE	3.79 ww	Egg	Chick mortality during hatching	Ohlendorf et al. 1985
Forster's tern	DDE	1.6 ww	Egg	7% thinning, but no adverse effect on reproductive success	King et al. 1991
Hooded merganser (*Lophodytes cucullatus*)	DDE	0.62 ww	Egg	9.6% eggshell thinning; egg breakage	Zicus et al. 1988
Leach's storm petrel	DDE	12 ww	Egg	12% eggshell thinning	Noble and Elliot 1990
Northern gannet (*Sula bassanus*)	DDE	18.5 ww	Egg	17% eggshell thinning; low reproductive success	Elliott et al. 1988

[1] Weight basis: dw, dry weight; ww, wet weight; bw, dosage relative to body weight.
[2] Falcons include: peregrine falcon and gyrfalcon.
[3] Hawks include: Cooper's hawk, ferruginous hawk, northern goshawk, red-shouldered hawk, red-tailed hawk, rough-legged hawk, sharp-shinned hawk, and Swainson's hawk.
[4] Owls include: burrowing owl, great grey owl, great horned owl, long-eared owl, short-eared owl, and snowy owl.

ww, respectively. Critical levels of DDE in eggs were generally 10 mg/kg, but lower concentrations were found for prairie falcons (1.2 mg/kg), osprey (4 mg/kg), merlin (5 mg/ kg), and bald eagle (6 mg/kg).

Tissue concentrations in terrestrial birds have resulted in a number of adverse effects. Many workers have studied the toxicity of DDT to terrestrial birds. Table 16 shows a wide range of dietary LC50s and dose LD50s from Hill et al. (1975), Hill and Camardese (1986), and Hudson et al. (1984), among others.

Hill et al. (1971) studied the correlation between dietary exposure to DDT, brain residues of total DDT, and lethality for several species of birds; in particular, they correlated brain residues in bobwhite quail (*Colinus virginianus*) to dietary concentrations of DDT and to various toxic effects. Dietary concentrations of 400 mg/kg resulted in weight loss in the majority of the test birds
but no other signs of toxicity. Dietary concentrations of 800 mg/kg resulted in tail tremors and irregularities in head carriage. The brain residues of total DDT at this dose ranged from 7.5 to 30 mg/kg. Birds fed 1,600 mg/kg DDT had tail tremors, stumbling gait, head bobbing, loss of equilibrium, and death within 100 hours of dosing. Although other research had suggested a critical brain residue level of 30 mg/kg DDD+DDT, indicative of serious danger or death (Stickel et al. 1966), the results of this study indicate that 20 mg/kg in the brain would be a more appropriate critical level, especially as signs of intoxication were observed at levels as low as 7.5 mg/kg.

Mahoney (1975) conducted studies on the effects of DDT on migratory behavior. He found that DDT tissue concentrations of 5 to 25 mg/kg ww delayed the development of the migratory condition in white-throated sparrows (*Zonotrichia albicollis*).

Stickel et al. (1984) measured lethal brain residues in four wild birds exposed to DDE in the diet. In all species, brain residues of 300 to 400 mg/kg resulted in an increased likelihood of death. Stickel et al. (1984) also measured the loss rate of DDE in grackles (*Quiscalus quiscula*). Test animals were fed 1,500 mg/kg DDE in the diet for 7 days then given untreated food. A loss rate of 0.3 percent per

day was calculated at the end of 112 days. The estimated half-life for DDE was 229 days.

Mallards released to three experimental stations in Canada were evaluated for uptake of DDT as compared to controls to determine the feasibility of using the mallard as a sentinel species (Gebauer and Weseloh 1993). Two of the sites—a confined waste disposal facility and a sewage lagoon—were known to have sediment contaminant levels that exceeded guidelines of the Ontario Ministry of Environment (Persaud et al. 1993). The third location was a relatively clean natural marsh. All three sites were important resting and feeding areas for migratory and resident waterfowl. Mallards from each location were collected at specified intervals and were analyzed for DDE residues in breast muscle. Concentrations of DDE in muscle tissue prior to release were 3.3 μg/kg ww. For the birds released at the confined waste disposal facility, residues averaged 8.5 μg/kg ww after 10 days and 27.9 μg/kg ww after 115 days. Residues in birds released at the sewage lagoon had increased to 13.8 μg/kg ww after 10 days, 58.4 after 70-days, and 216.9 after 112 days. The tissue residues in birds collected from the sewage lagoon were significantly higher than the levels at the time of release at both 70 and 112 days. It was estimated from these results that the mean rate of accumulation of DDE at 30 days ranged from 0.09 micrograms per kilogram per day (μg/kg/d) at the waste disposal facility to 0.99 μg/kg/d at the sewage lagoon. The accumulation rate at the sewage lagoon after 112 days was 1.9 μg/kg/d and had not reached equilibrium.

Friend and Trainer (1971) found the toxicity of *p,p'*-DDT to be age dependent in mallards (*Anas platyrhynchos*), with 30-day-old ducklings having a higher LC50 than either adult birds or younger ducklings (table 16). In addition, the onset of mortality and the mean elapsed time until death were age related and were earlier for younger birds, indicating a dose-dependent mortality relationship. Moreover, the body weights of surviving birds were less than those of controls for both groups of ducklings, although the difference was not statistically significant. Brain residues of DDT were also measured in birds dying within each test group, and no correlation was found between the residue levels at time of death and the initial DDT dose. In addition, adult birds that died of DDT contained brain residues of DDD, DDE, and DDT that were 6 to 17 times greater than those in adult birds that survived the test. The average ratio of DDE, DDD, and DDT in the surviving birds was 2:5:3 (DDE: DDD: DDT).

Van Velzen and Kreitzer (1975) found that the toxicity of *p,p'*-DDT to clapper rails (*Rallus longirostris*) varies by sex. As shown in table 16, the 5-day LC50 was notably higher for females than for males. In addition, brain residues of DDD, DDE, DDT, and DDD+DDT were significantly higher in birds that died than in those that survived. Van Velzen and Kreitzer established a lower lethal limit of 30 mg/kg of DDD+DDT in the brain for diagnosing DDT as the cause of death. Research by Stickel et al. (1966) and Stickel and Stickel (1970) has shown that the lower limit of the lethal range for DDE is 250 mg/kg in the brain, but for the combined residues of DDE and DDT, 30 mg/kg in the brain is the practical separation point between birds that live and those that die in laboratory studies. Similarly, the combined residues of DDD and DDT in the brain are lethal at 20–30 mg/kg (Bernard 1963, Stickel et al. 1966, Hill et al. 1971).

Several investigators have studied DDT toxicity and accumulation in herons (Ohlendorf et al. 1981, Henny et al. 1984, 1985; Findholt and Trost 1985; Henny and Blus 1986; White et al. 1988; Custer and Custer 1995; Hothem et al. 1995). These studies indicate that herons are sensitive to DDT and that high DDT levels can be found in the tissues of birds throughout the United States.

Custer and Custer (1995) found that black-crowned night-heron chicks exposed to environmental concentrations of DDT have varying accumulation rates for DDT and its metabolites. Mean accumulation rates of p,p'-DDD, *p,p'*-DDE, and *p,p'*-DDT were 0.48, 42.9, and 0.2 mg/ kg/d, respectively. Ohlendorf et al. (1981) found that the level of DDE residues in the brain of a black-crowned night-heron from Nevada (230 mg/kg ww) was high enough to cause severe impairment, but death was probably not due to poisoning. Similarly, Call et al. (1976) found brain residues of 246 mg/kg and liver residues of 570 mg/kg ww of DDE in a great blue heron found dead in South Dakota. A great blue heron from North Carolina, which had 20 mg/kg ww of DDT in its brain, almost certainly died of DDT poisoning (Ohlendorf et al. 1981).

Adverse reproductive effects resulting from DDT poisoning in birds include reproductive impairment, reduced fledging success, and eggshell thinning. Through review of laboratory studies conducted with DDD, DDE, and DDT, Stickel (1973) observed that DDD did not produce significant eggshell thinning in mallards, but DDE produced significant eggshell thinning in three major groups of birds: the orders Strigiformes (screech owls, *Otus asio*), Falconiformes (American kestrels, *Falco sparverius*), and Anseriformes (mallards and black ducks, *Anas rubripes*). DDT resulted in eggshell thinning only after longer expo-sure duration, by which time some of the DDT may have been metabolized into DDE.

Several researchers have shown that while eggshell thinning of 5–7 percent is statistically significant, it is probably not biologically significant, and field studies have shown that an average thinning of 10 percent is seldom associated with egg breakage or population decline (King et al. 1980, 1991; Anderson et al. 1969; Blus 1970, 1982). In addition, the amount of thinning resulting from each incremental increase in DDE is greater at lower residue levels (Blus 1996). For example, 1 mg DDE/kg may result in 5–10 percent thinning, whereas 59 mg/kg results in 44 percent thinning in brown pelicans.

Most studies report eggshell thinning as the indicator of reproductive problems, but other factors are also important indicators, such as egg residue levels compared to the percentage of chick survival, the number of young pro-duced per nest, and eggshell strength (Blus 1996). Egg residue levels of DDD, DDE, and DDT and associated thinning or other repro-ductive effects are summarized in table 16.

Wiemeyer et al. (1984, 1993) studied repro-ductive activity in bald eagles (*Haliaeetus leucocephalus*) exposed to DDT. Eggshells were thinned by 10 percent at DDT levels of 5 mg/ kg (Wiemeyer et al. 1984) and by 15 percent at 16 mg/kg (Wiemeyer et al. 1993). The production of young was normal when eggs contained less than 3.6 mg/kg DDE but was reduced by nearly 50 percent when concentrations ranged between 3.6 and 6.3 mg/kg. When concentrations exceeded 6.3 mg/kg, reproduction was reduced by another 50 percent.

Similar results were observed by Grubb et al. (1990) for bald eagles in Arizona. Samples of eggshells collected from 1977 to 1985 had a mean DDE concentration of 3.3 mg/kg and were 8.8 percent thinner than pre-1947 eggs from southern California. However, pro-ductivity over the same period increased slightly, from an average of 0.8 young per occupied territory in 1975 to 1.13 young per occupied territory from 1985 to 1986. They speculated that although many factors other than decreasing environmental contamination could be contributing to the increase in population, the levels of DDE and eggshell thinning did not seem to be adversely affecting reproductive success.

Nesting failures for bald eagles in Oregon were associated with many factors, including exposure to DDT. Anthony et al. (1994)

surveyed 89 failed nests from 1980 to 1987 to determine the probable cause of failure. Nest condition, the presence of new nesting material, prey remains, unhatched eggs, and remains of dead young were evaluated. Based on results by Wiemeyer et al. (1984), nesting failures were attributed to DDE if egg concen-trations were >10 mg/kg ww or if eggshell thinning was >15 percent. DDE was attri-buted as the major cause of nesting failure in 32 percent of the nests.

Eggshells of American kestrels exposed to 3 mg/kg DDE in the diet were 13 percent thinner than those of controls, but none of the eggs broke (Lincer 1992). Of the eggs that were artificially incubated, only 30 percent pipped, but those that pipped generally hatched.

Clark et al. (1995) collected eggs of northern harriers (*Circus cyaneus*), great-tailed grackles (*Quiscalus mexicanus*), and black-necked stilts (*Himantopus mexicanus*) from several locations throughout California and Texas. Egg residue levels of DDT averaged 2.2 mg/kg ww for the grackle, 2.79 for the stilt, and 4.07 for the harrier. Although these concentrations were as high as the concentrations that had been reported as resulting in impaired repro-duction in bald eagles, black-crowned night-herons, and white-faced ibises, the researchers did not report observing any adverse effects in the birds from which eggs were collected.

For terrestrial birds, Jefferies (1971) found that tissue concentrations of 4 mg DDE/kg ww and 8 mg DDT/kg dw resulted in reduced fledging success of Bengalese finches (*Lonchura striata*).

Among the waterfowl, mallards have been studied extensively because even low concentrations of DDT can cause eggshell thinning in this species. Davison and Sell (1974) found that mallards that survived having 200 mg/kg of technical-grade DDT in their diet for 12 weeks produced eggs that had shells 20 percent thinner than normal and produced hatchlings with tremors.

Black ducks fed dietary concentrations of 10 mg DDE/kg dw for two breeding seasons (June through November in two successive years) and then clean food for 2 years continued to exhibit eggshell thinning at the end of the 2-year cleanup period (Longcore and Stendell 1977). Eggs collected during the two breeding seasons and the first year of clean diet were significantly thinner than controls (average 20 percent thinner). Eggs collected during the second year of clean diet were 10 percent thinner than controls. Residues in eggs increased significantly during the years of treatment (up to 64.9 mg/kg ww), then decreased during years of clean feed (down to 6.2 mg/kg ww). In addition, exposed hens continued to produce significantly fewer young than did controls after the second year of clean diet.

White et al. (1988) found that green-backed herons (*Butorides striatus*) and anhingas (*Anhinga anhinga*) had decreased hatching success and eggshell thinning in areas that had not been treated with DDT for more than 13 years. Concentrations of 5.1–10 mg/kg ww in eggs of green-backed herons were the threshold for reduced hatching success.

Studies of black-crowned night-herons in Idaho (Findholt and Trost 1985) indicated that relatively low concentrations of DDE resulted in eggshell thinning (table 16). Henny et al. (1984) studied black-crowned night-heron populations in Washington, Oregon, and Nevada between 1978 and 1980 to determine contaminant patterns and eggshell thinning. Eggs with residues greater than 8 mg/kg ww correlated with decreased clutch size, lower productivity, and an increased incidence of cracked eggs. Henny et al. (1982, 1984) also found that, with the exception of two locations along the Columbia River, there was a strong north-south DDE contaminant gradient. DDE residues in southern colonies were much

higher than those in northern colonies. This trend may contribute to increased exposure at wintering grounds to migrating waterfowl.

Henny and Blus (1986) found similar trends in DDE contamination for black-crowned night-herons from Idaho and Oregon that wintered in coastal Mexico, compared to those from Nevada that wintered in southern California. On a more local basis, Hothem et al. (1995) noted the same type of trend when they compared black-crowned night-herons within San Francisco Bay to those in the San Joaquin Valley.

Mora et al. (1987) evaluated the potential importance of the uptake of DDT and its metabolites from wintering grounds for northern pintail and gadwall, which migrate from northern California to southern California or Mexico. These results confirmed those for other pintails and mallards migrating through California and for black ducks that migrate to Texas. Pintails sampled from the Salton Sea, California, had higher whole-body DDE residues than those from the Lower Klamath National Wildlife Refuge, California, again suggesting a north-south organochlorine pesticide gradient in California. However, residues decreased in resident waterfowl (black-bellied and fulvous whistling ducks) collected farther south in Mexico. The researchers hypothesized that the increased levels found at the Salton Sea may have resulted from past heavy use of DDT, from current illegal use of DDT, or from DDE and DDT impurities in dicofol. They further speculated that the lower residues found in waterfowl from Mexico may have been due to variability in uptake between the species sampled and do not necessarily indicate lower levels of organochlorine pesticides in the Mexican wintering grounds.

Eggshell thinning in various species of grebes and northern gannets has also been reported for DDE. Eggshell thinning of 2.3 percent and reduced productivity was reported in western grebes (*Aechmophorus occidentalis*) from Utah with egg DDE concentrations of 5.4 mg/kg ww (Lindvall and Low 1980). Red-necked grebes (*Podiceps grisegena*) collected from Manitoba exhibited low egg viability, egg-shell thinning of 6.5 percent, and reduced fledging success at egg DDE concentrations of 6.68 mg/kg ww (De Smet 1987). Repro-ductive impairment, low reproductive success, and eggshell thinning (17 percent) in northern gannets (*Sula bassanus*) were associated with egg DDE concentrations of 18.5 mg/kg ww (Elliot et al. 1988 as cited in Forsyth et al. 1994).

Zicus et al. (1988) collected eggs from nests of hooded merganser (*Lophodytes cucullatus*) and common goldeneye (*Bucephala clangula*) where the hens had been found dead on the nest. Their analyses indicated that while organo-chlorine pesticides probably did not con-tribute to the death of the hens, eggshell thinning and egg breakage were probably the result of DDE. The mean egg concentration of DDE for mergansers was 0.62 mg/kg ww with an associated 9.6 percent thinning of eggshells. The mean egg concentration of DDE for goldeneye was 0.52 mg/kg ww, and the associated eggshell thinning was 15.4 per-cent. Breakage of eggs in successful nests was also greater for goldeneye than for mergansers.

Mammals

Studies of DDT toxicity to mammals have been generally limited to laboratory mammals. Liver, neurological, develop-mental, reproductive, and carcinogenic effects after exposure to DDT have also been noted for mice, rats, shrews, hamsters, monkeys, dogs, and bats (table 17). Laboratory studies with wild mammals have indicated that big brown bats (*Eptesicus fuscus*) are much more sensitive to DDT than other mammals (Stickel 1973).

Table 17.—DDD, DDE, and DDT effects on mammals

[See Appendix II for explanation of abbreviations and technical terms]

Species	Chemical species	Concentration (mg/kg bw)[1]	Exposure duration	Effects	Reference
Bat (*Eptesicus* sp)	DDT	40		LD50 (oral dose)	Clark and Stafford 1981
Bat (*Myotis* sp.)	DDE	600 (in tissue)		LC50	
Dog	DDT	16	160 weeks	NOAEL	Lehman 1965
		80		LOAEL, liver alterations	
	DDT-tech	1	2 generations	NOAEL	Ottoboni et al. 1977
		5		LOAEL, premature puberty	
	p,p'-DDT	12	14 months	LOAEL, maternal and fetal death	Deichmann et al 1971
Hamster	DDT-tech	40	Lifetime	NOAEL	Cabral et al. 1982
		41.5	128 weeks	LOAEL, necrosis	Rossi et al. 1983
		83		LOAEL, tremors	
Mouse	*p,p'*-DDD	107	78 weeks	NOAEL	NCI 1978
	p,p'-DDT	0.26	Lifetime	LOAEL, liver tumors	Tomatis et al. 1972
		6	78 weeks	NOAEL	NCI 1978
	DDT-tech	6.5	Lifetime + 5 generations	NOAEL	Turusov et al. 1973
		32.5		LOAEL, increase in preweening death	
Rat	DDT	0.5	2 years	LOAEL, liver lesions	Fitzhugh and Nelson 1948
		0.8	2 years	NOAEL	ORNL 1996
		1 mg/kg diet	—	NOAEL	Worthing 1987
	p,p'-DDT	1 mg/kg diet	27 weeks	NOAEL	Laug et al. 1950
		5 mg/kg diet		LOAEL, hepatocellular hypertrophy	
	DDT-tech	1	2 generations	NOAEL	Ottoboni 1969
		10		LOAEL, increased constricting rings of the tail	
	DDT-tech	12.5	Lifetime	LOAEL, liver tumors	Cabral et al. 1982
	DDT	260		LD50 (oral dose)	Gaines and Linder 1986
Rhesus monkey (*Macaca mulatta*)	DDT	8	7.5 years	NOAEL	Durham et al. 1963
Shrew (*Blarina* sp.)	total DDT	910		LC50	Blus 1978
		210 (in tissue)		LC50	

[1] Concentration is the daily dose, as milligrams per kilogram of body weight, unless stated otherwise.

Laboratory rats exposed to DDT in their feed for 27 weeks showed no effects on growth at dietary concentrations up to 50 mg/kg (Laug et al. 1950). However, at dietary concentra-tions of 5 mg/kg and above, they showed pathologic changes, including increased hepatocellular hypertrophy and cytoplasmic oxyphilia, as well as peripheral basophilic cytoplasmic granules. This study established the 5 mg/kg dietary level as the LOAEL and 1 mg/kg as the NOAEL. Fitzhugh and Nelson (1948) studied the long-term effects of relatively high DDT concentrations on rats. They observed liver lesions in rats exposed to 10–800 mg DDT/kg in the diet for 2 years and established an LOAEL of 0.5 mg/kg dw/day.

Although less research has been conducted on wild mammals than on birds, the distribution of tissue residues in mammals seems to be similar. Brain residue levels tend to provide the best indication of toxicity, and DDE is the predominant metabolite found (Stickel 1973).

Predatory and aquatic mammals tend to accumulate the highest residues (Stickel 1973). Mink accumulate higher levels than hares living in the same area. Similarly, shrews and other species in the order Insectivora accumu-late higher levels of total DDT than do mice and voles. Many small seed-eating mammals accumulate only low levels of total DDT even in areas with high environmental levels (Stickel 1973).

Accumulation differences between shrews, mice, voles, mink, and hares were observed in specimens collected over a 9-year period from a forest that had been treated with 1.12 kg DDT per hectare (Dimond and Sherburne 1969). The following residues were measured during the first and last years of the study:

Species	Total DDT, whole body (mg/kg)	
	Year of application	Nine years later
Shrews	15.6	1.18
Mice and voles	1.06	0.03
Mink	—	1.6
Hares	—	0.02

Throughout the study, residues in shrews were 10–36 times those in mice and voles. Similarly, mink collected from the same area contained residues 10–90 times those found in hares.

Analyses of small herbivorous and omniv-orous mammals collected from agricultural areas of Alabama, Arkansas, and Mississippi from 1965 to 1967 (U.S. Department of Agriculture 1969) indicated that rabbits, rice rats, and muskrats contained whole-body residues of total DDT less than the detectable limit; fox squirrels and chipmunks contained less than 0.1 mg/kg; and white-footed mice, cotton rats, and wood rats generally contained less than 0.5 mg/kg. However, harvest mice and house mice contained up to 3.94 mg/kg, and opossums contained up to 8.76 mg/kg.

Ranges of total DDT residues in fat from aquatic mammals and big game mammals collected from several Western States are presented in Stickel (1973). Residue levels in big game mammals include:

Species	Residue levels (mg/kg)
Bear	0.34
Elk	<0.06–29
Moose	0.17
Mountain goats	<0.09–0.9
Mule deer	<1.35–43
Pronghorn antelope	<0.17–0.23
White-tailed deer	<0.4–3

Bioaccumulation

Birds and other wildlife may become exposed to DDT through ingestion of contaminated prey species. Bioaccumulation factors for several species exposed to various environ-mental media are presented in table 18.

Jarvinen et al. (1977) tested fathead minnows for accumulation of DDT present in diet, water, and both diet and water. Tissue residues were greater in fish exposed to DDT in water than in the diet, and accumulation from both water and diet was additive.
Mean bioconcentration factors are shown in table 18. In addition, residue levels and mortality rates for embryos whose parents were exposed to DDT in both water and diet were approximately twice those of embryos whose parents were exposed to DDT only in water.

Table 18.—Bioconcentration factors for biota exposed to DDD, DDE, and DDT

Species	Uptake from:	Compound	Bioconcen-tration factor	Study time	Reference
Aquatic					
Brook trout	Diet	DDT	0.6	120 days	Macek and Korn 1970
Brown bullhead	Water	DDD	125,000	Not stated	Hunt and Bischoff 1960
Fathead minnow (*Pimephales promelas*)	Water	Total DDT	100,000	266 days	Jarvinen et al. 1977
	Diet		1.2		
Golden shiner (*Notemigonus crysoleucas*)	Water	DDT	100,000	15 days	Courtney and Reed 1972
Goldfish (*Carassius auratus*)	Diet	DDT	0.8		Grzenda et al. 1970
Rainbow trout (*Oncorhynchus mykiss*)	Water	DDT	34,900–91,000 (depending on conditions)	96 hours	Muir et al. 1994
Terrestrial					
Black duck (*Anas rubripes*)	Diet	DDE	4.63–4.8	Reproductive cycle	Longcore et al. 1971
Earthworm (*Lumbricus terrestris*)	Soil	DDT	0.71	4 weeks	Davis 1971
Mixed earthworms	Soil	DDD	0.27	11 years	Beyer and Gish 1980
		DDE	6		
		p,p'-DDT	0.56		
		Total DDT	5.1		
			1.8–9.2	Not stated	Ma 1985
Red-winged blackbird (*Agelaius phoeniceus*) eggs	Sediment	DDE	12.9–582.4	Not stated	Bishop et al. 1995b
Short-tailed shrew (*Blarina brevicauda*)	Prey	DDT	1–2.5	Not stated	Blus 1978
Tree swallow (*Tachycineta bicolor*) eggs	Sediment	DDE	16.2–868.6	Not stated	Bishop et al. 1995b
Tree swallow (*Tachycineta bicolor*) nestlings	Sediment	DDE	5–48.9	Not stated	Bishop et al. 1995b

Earthworms compose a large portion of the diets of some birds and reptiles, including woodcock (*Philohela minor*), robin (*Turdus migratorius*), red-bellied snake (*Storeria occipitomaculata*), and eastern garter snake (*Thamnophis sirtalis*). Earthworms are also eaten occasionally by mammals such as insectivores (shrews and moles), rodents, and carnivores (Mustelidae), and by other cluster flies (Beyer and Gish 1980). Based on toxicity studies for birds exposed to DDT, Beyer and Gish (1980) estimated that total DDT residues of 8 mg/kg ww or 32 mg/kg dw in earth-worms would constitute the minimum hazardous level for birds.

Bishop et al. (1995b) measured the bioconcentration of *p,p'*-DDE in red-winged blackbird eggs and in tree swallow (*Tachycineta bicolor*) eggs and nestlings as a function of lipid-normalized concentrations in biota and organic-carbon-normalized concentrations in sediment. In each case, they reported wide ranges of bioconcentration factors (table 18).

Interactions

Interactions between organochlorine pesticides were studied in the earthworm *Lumbricus terrestris* by Davis (1971). Measurements of the uptake of DDT and dieldrin indicated that while dieldrin accumulated more than DDT, the accumulation of either chemical did not affect the accumulation of the other.

Lincer (1992) examined the reproductive success of American kestrels exposed to DDE and Aroclor 1254 in the diet. Birds fed DDE alone and those fed both DDE (3 mg/kg) and Aroclor 1254 (10 mg/kg) had eggs with shells that were significantly thinner than controls. The thinning was 13 percent for birds fed DDE alone and 16 percent for birds fed both DDE and Aroclor 1254. Unexpectedly, birds fed Aroclor 1254 alone exhibited shells that were significantly thicker (6 percent) than controls. In addition, there was no egg breakage in the nests of birds fed either DDE or Aroclor 1254, but those that were fed a combination of DDE and Aroclor 1254 experienced some egg breakage in the all of the nests, and none of their eggs pipped. This study indicates that effects of DDE and Aroclor 1254 are synergistic.

Regulatory Standards

Federal

Ambient Water Quality.—EPA ambient water quality criteria (AWQC) for DDT and its metabolites have been developed for both freshwater and saltwater plants and animals (*Federal Register* 1980, 1992). These criteria are based on levels of DDT that would exceed Food and Drug Administration action levels for human consumption of fish (5 mg/kg; EPA 1996). In addition, screening ecotoxicity thresholds, based strictly on toxicity to ecological species, have been developed using methodology presented in the Great Lakes Water Quality Initiative—Tier II (40 CFR 9, 122 etc., 1995). These screening values represent concentrations above which adverse ecological effects could occur. The AWQCs and the Great Lakes screening ecotoxicity thresholds are listed in table 19.

State

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

International

Quality standards for DDT have been established in the Netherlands and in Ontario, Canada. The Dutch quality standards for total

Table 19.—U.S. Environmental Protection Agency standards and criteria for DDT, DDD, and DDE

[See Appendix II for explanation of terms. Sources: EPA 1986, 1996; *Federal Register* 1980, 1982]

Status	Known carcinogen; EPA priority pollutant
Drinking water MCL	None established
Freshwater AWQC (DDT)	1.1 µg/L for acute exposure 0.001 µg/L for chronic exposure
Saltwater AWQC (DDT)	0.13 µg/L for acute exposure 0.001 µg/L for chronic exposure
Great Lakes Water Quality Initiative (DDT)	0.013 µg/L for acute exposure
Freshwater LOAEL (DDD)	0.6 µg/L for acute exposure
Freshwater LOAEL (DDE)	1,050 µg/L for acute exposure
1/1,000,000 cancer risk (water and organisms *or* organisms only)	DDT: 0.59 ng/L DDD: 0.83 ng/L DDE: 0.59 ng/L

DDT include limits of 10 µg/kg dw in sediment, 3 µg/kg dw in soil, and 500 µg/kg ww in wildlife food (Hendriks et al. 1995). The Ontario sediment quality guidelines (Persaud et al. 1993) are as follows:

Compound	Lowest effect level (mg/kg dw)	Severe effect level (mg/kg organic carbon)[1]
p,p'-DDD	0.008	6
p,p'-DDE	0.005	19
o,p'+*p,p'*-DDT	0.008	71
Total DDT	0.007	12

[1] Multiply times the total organic carbon content of a sample to find the bulk-sediment severe effect level for that sample.

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Mercury

Description

Mercury is the only metallic element that is liquid at normal environmental temperatures. It freezes at –39 °C and boils at 357 °C. Owing to its bright silvery color, the Romans called it *hydrargyrum* ("liquid silver"), which is why it is designated by the chemical symbol Hg. It has an atomic number of 80, an atomic weight of 200.6, and a specific gravity of 13.5. Ele-mental mercury has a low solubility in water, but some of its salts are highly soluble. Unlike many trace elements, mercury has no known biological function.

Mercury has three stable valence states—Hg(0) the native element, Hg(I) [Hg_2^{2+}], and Hg(II) [Hg^{2+}]—and it forms a variety of organic and inorganic compounds. The formation of methylmercury (CH_3Hg^+) is the most significant transformation because methyl-mercury is far more toxic and bioavailable than any other form of mercury. Methylation may be accomplished via bacteria in both sediments and water (Compeau and Bartha 1985). In some organisms and tissues, nearly all mercury is methylmercury.

Cinnabar (HgS), the most common ore of mercury, occurs either as long, slender, brilliant red crystals or as irregular red to gray or brownish masses. In powdered form, it is used as a pigment called "Chinese red." At some sites, minute globules of liquid ele-mental mercury are disseminated through the cinnabar.

Occurrence

Primary sources of natural mercury emissions include volcanic eruptions and volatilization or solubilization of mercury from rocks, soils, and sediment. In rocks and soils, mercury most commonly occurs as mercuric sulfide (cinnabar). Mercury-enriched deposits are known in the Franciscan Formation of the coastal mountains of California, in the Green River Formation of the western Colorado Plateau, and in the vicinity of hot springs in many parts of the world. Mercury concen-trations in and around deposits of the Franciscan Formation may be in the 10 to 100 mg/kg range, whereas concentrations in the Green River Formation have been reported as high as 10 mg/kg (USGS 1970). The U.S. Geological Survey (USGS) reports mean background concentrations of mercury in surficial materials of the United States as 0.065 mg/kg (dw) (Schacklette and Boerngen 1984). Mercury occurs in coal at concentra-tions ranging from <0.01 to 8.0 μg/g depending on the geographic region and type of coal (Malani and Modetz 1981).

Atmospheric sources also contribute mercury to the environment (Haines 1991), although their concentrations are orders of magnitude lower than those of some geological sources. Atmospheric deposition is particularly important in environments where subsequent methylation is enhanced, as in the Everglades and other nutrient-enriched wetlands.

In the 20th century, mercury releases from artificial sources have been almost 10 times higher than calculated releases due to natural weathering (Moore and Ramamoorthy 1984). Mercury mining is a common source of mercury in the West; the mine wastes can be classified in five groups in order of increasing bioavailable mercury: (1) Waste rock, (2) low-grade unprocessed ore, (3) efflorescent salts, (4) processed ore tailings (calcines), and (5) soot or ash from the condenser system (J. Rytuba, USGS, pers. comm.). Other human activities that enhance mercury releases include the use of mercury as an amalgam in gold

mining; use of mercurials in seed dressings, fungicides, paints, and slimicides; fossil-fuel combustion; the industrial production of chlorine; and spills from field instruments (such as manometers) used to measure pressure at wellheads in gas fields. One study estimated that, among the popula-tion of Sweden, the digestion and excretion of mercury from dental amalgams contribute about 100 kg of mercury to the environment each year (Skare 1995).

Summary of Effects

Table 20 summarizes the predicted effects of environmental exposures to mercury, based on the information currently available.

Field Case

Clear Lake, in Lake County, California, is a very large freshwater lake contaminated with more than 100 tons of mercury from the Sulphur Bank Mine, most of which is still present in the bed sediments. The waste mercury is only a small percentage of more than 5000 tons of mercury that the mine had produced. The lake and the mine are now included in an EPA Superfund cleanup site. The ecological assessment of this site included an excellent survey of mercury bioaccumu-lation factors from sediment in a contaminated freshwater lake. In sediment, total mercury concentrations range from 0.27 to 183 mg/kg (dw), and methylmercury concentrations range from 0.18 to 15.9 mg/kg (Suchanek et al. 1995). Table 21 lists observed bioaccu-mulation factors for total mercury and methylmercury from sediment to oligo-chaetes, chironomids, and carp observed in this study.

The bioaccumulation factors in these results are consistently higher for methylmercury than for total mercury: five orders of magnitude greater for fish and one to two orders of magnitude greater for benthic infauna. Clearly, the parameters affecting net methylation are controlling bioaccumulation of mercury. For fish, trophic position was found to be the most influential factor in bioaccumulation at Clear Lake, a result supported by many other studies (Sorensen 1991). BAFs are presented for sediment to carp because carp are directly grubbing about within the sediment. However, carp had less mercury than other fish species.

Ecological effects on populations and communities were shown to be related to mercury concentrations in the sediment at Clear Lake. Leech biomass was inversely correlated to both total mercury and methyl- mercury sediment concentrations (Suchanek and Richerson 1994). Population numbers of *Chironomus* species exhibited an exponential decline as a function of sediment total mercury concentrations. Benthic infaunal community diversity (as measured by both Shannon's index and Simpson's index) showed linear declines inverse to sediment mercury concentrations (Suchanek et al. 1995).

Abiotic Factors Affecting Bioavailability

Water

Analytical methods to detect mercury in water have dramatically improved in the last 5–10 years, resulting in lower detection limits and reduced interference from contamination. Because of the earlier analytical limitations, caution must be exercised when evaluating older mercury studies and reviews—especially studies that measured mercury concentrations in water. Background estimates of mercury in water conducted before the early 1980s likely report concentrations that are artificially high and bioconcentration factors that are too low. Older studies that elucidated tissue residues and soil concentrations may still generally be relied upon. The role of global atmospheric transport, until recently, was underestimated, and the net flux of mercury from various aquatic and sediment compartments was inaccurately measured prior to the mid-1980s.

Table 20.—Summary table for predicted mercury effect levels

[All matrix values expressed as total mercury (includes organic and inorganic forms). All criteria relate mercury risk to populations, not individuals.]

Matrix	No effect[1]	Level of concern[2]	Toxicity threshold[3]	Explanation
Water (µg/L)	- - -	- - -	>30	Sublethal effects to fish (Eisler 1987)
Sediment (mg/kg dw)	<0.065	>0.15	0.2	0.065, surficial materials background (Shacklette and Boerngen 1984); 0.15, ERL of Long et al. (1990); 0.2, threshold to protect clapper rail (Schwarzbach et al. 1993)
			0.24	Toxic to guppies (Gillespie and Scott 1961)
Fish, whole body (mg/kg ww):				
Warm-water sp.	0.11	- - -	- - -	Background in bluegill (table 23).
Cold-water sp.	- - -	- - -	1.0	FDA action level
Birds, diet (mg/kg bw/day)	- - -	- - -	0.064	Effects in mallards (Heinz 1979)
Birds, diet (mg/kg ww)	- - -	- - -	0.3	Loon reproductive and behavioral effects (Barr 1986)
			0.1	Mallard reproductive and behavioral effects (Heinz 1979)
Bird eggs (mg/kg fww)	0.1	0.2–1.0	0.5–1.5	0.1, no effects in osprey; 0.5–1.5, low hatchability for pheasant (table 24)
			0.86	Mallard reproductive and behavioral effects (Heinz 1979)
			5.0	Mallard brain lesions (Heinz 1975)
Bird brain (mg/kg ww)	0.13	0.13–1		0.13 = mean in controlled, nonexposed population (Finley and Stendell 1978)
			1	Obvious signs of intoxication (Scheuhammer 1988)
			4	Lethal in embryos (Finley and Stendell 1978)
			15	Lethal in adults (Scheuhammer 1988)
Bird feathers (mg/kg dw)	5	5–40	40	Effects highly variable; sample other tissues. 5, upper end of background range; 20, reflects >1 mg/kg in diet (Scheuhammer 1991). Reproduction impaired over range of 5–40 (Eisler 1987)
Bird kidney (mg/kg ww)	<2	- - -	20	Varies depending on species, sex, form of Hg, and Hg:Se ratio. Toxicity likely whenever kidney conc. > liver conc. See Littrel (1991), Heinz (1996).
Bird liver (mg/kg ww)	<1	1–2	3	1–2, behavioral effects (Zillioux et al. 1993); 3, reproductive harm (Barr 1986)
			5	Threshold for adult waterbirds (Zililoux et al. 1993)
			25	Kidney disease, gout in herons (Spalding et al. 1994)

[1] Concentrations below this level are close to background and are not known to cause adverse effects.

[2] Concentrations at this level are above background but rarely appear to cause any adverse effects.

[3] Concentrations exceeding this level seem to cause some adverse effects, including reproductive impairment and sublethal impacts.

Table 21.—Mercury bioaccumulation factors from sediment to oligochaetes, chironomids, and carp in Clear Lake, California
[Compiled from data in Suchanek et al. 1993. ND, not determined]

	Hg in sediment (mg/kg dw)		Hg bioaccumulation factors					
			Oligochaetes		Chironomids		Carp	
Site	Total	Methyl	Total	Methyl	Total	Methyl	Total	Methyl
WB–1	77.80	10.36	0.14	1.15	0.07	2.0	0.01	124
WB–2	27.16	ND	.17	ND	.1	ND	ND	ND
WB–3	16.68	ND	.09	ND	.11	ND	ND	ND
WB–4	4.13	7.39	.2	1.0	.2	1.4	ND	ND
WB–5	2.61	4.16	.26	1.6	.23	2.5	.16	185
WB–6	8.85	7.1	.16	2.8	.05	1.1	.0797	ND
WB–7	1.40	2.43	.24	2.6	.16	23.6	.39	ND

Water concentrations are typically used to assess mercury hazards to fish and aquatic life. Gill and Bruland (1990) have shown that total dissolved mercury concentrations are not as useful in predicting concentrations in fish as are the dissolved concentrations of organic mercury compounds. Concentrations are typically measured in picomolar (p*M*) quantities (5 p*M* Hg is roughly equivalent to 1 ng of Hg). Estimates of background total Hg concentrations in freshwater, prior to 1980, were incorrectly measured in the range of 50 to 250 p*M* (10 to 50 ng/L). Freshwater background concentrations are now thought to be less than 50 p*M* (10 ng/L). Some exceptionally pristine areas have concen-trations less than 1 ng/L (Gill and Bruland, 1990). The concept of "background" concen-tration is somewhat complicated by the global distribution of mercury through atmospheric water and may not be a very useful concept. Concentrations greater than "background" are routinely found in continental rainwater, and levels of 120 ng/L or more have been documented (Dvonch et al. 1995).

Much of the research on mercury in freshwater systems has been conducted in poorly buffered systems, in mesic environ-ments dominated by atmospheric mercury sources rather than geologic sources. The low pH of these systems (Canada, Sweden), due to acid rain, appears to promote mercury bioaccumulation. In the Western United States, lakes in areas of mercuriferous rocks and soils tend to be more alkaline; the climate in these areas tends to be arid to semiarid. Clear Lake, California, and the Carson River in Nevada are two such areas in the west, and intense research is currently underway in both areas. Results of these studies should produce new insights into mercury cycling in aquatic ecosystems typical of the Western United States.

Table 22 shows an extraordinary range of variability of estimated and measured effects of mercury in water. Differences in mercury toxicity between taxa are greater than dif-ferences between the organic and inorganic forms of mercury. Fish toxicity concentrations (96-h LC50), vary by two orders of magnitude, from 11 to 1800 µg/L (Sorensen 1991). At 10 °C, methylmercury is about seven times more toxic than Hg^{+2} to fingerling rainbow trout (Macleod and Pessah 1973). Eisler (1987) concluded that total mercury concentrations in water of 100 to 2000 µg/L were fatal to sensitive aquatic species, and concentrations between 30 and 100 µg/L caused significant sublethal effects in fish.

Table 22.—Mercury concentrations in water and associated effects on wildlife

Concentration (µg/L)[1]	Species	Comments/Effects	Reference
0.00092–0.0036 (4.6–18 p*M*)	—	Background in Great Lakes water (50 times lower than pre-1980 measures)	Gill and Bruland 1990
0.0025	—	Background concentration in freshwater lakes with atmospheric source of Hg only	Sorensen et al. 1990
0.012	Aquatic life	EPA freshwater chronic criterion for aquatic life protection (current)	EPA 1986
0.18	Birds	Great Lakes Initiative proposed criterion	EPA 1993
0.77	Aquatic life	Proposed EPA freshwater chronic criterion for aquatic life protection	EPA 1997a
1.4	Aquatic life	Proposed EPA freshwater acute criterion for aquatic life protection	EPA 1997a
1.6	Mammals	Great Lakes Initiative proposed criterion	EPA 1993
2.4	Aquatic life	EPA freshwater acute criterion for aquatic life protection (current)	EPA 1986
240	Fathead minnow	No effect (MeHg, 48 mo)	Olson et al. 1975
290–930	Brook trout	MATC, maximum acceptable toxicant concentration (≈0.4 to 1.3% of 96-h LC50)	McKim et al. 1976
900	Brook trout	LOEC, lowest observed effect concentration, behavioral effect	McKim et al. 1976
1000	*Rana pipiens*	Arrested metamorphosis (MeHg, 4 mo)	EPA 1980
2930	Brook trout	Early embryo death (3-generation exposure)	McKim et al. 1976

[1] Total Hg, unless otherwise noted.

The mercury concentrations proposed for the Great Lakes Initiative (table 22) are below the concentrations found in continental rain and, presumably, below background levels for freshwater lakes. The proposed mammalian value is an order of magnitude higher than the avian value, principally because an arbitrary species sensitivity factor of 0.1 was applied due to differences between observed test animals and the target birds. These proposed wildlife numbers probably represent safe concentrations but may be ultraconservative and unachievable in many environments. Controlling the processes and factors affecting bioaccumulation and methylation of mercury may ultimately be more important than maintaining low total mercury concentrations. Controlling bioaccumulation and methylation will require a good deal more understanding than we now have.

Bottom Sediment

Sediment may be both a sink for mercury and a source of it, with changing physical and biological conditions. The effects of mercury in sediment were extensively investigated during the assessment of the contamination at Clear Lake, California, as described above. Equally high natural concentrations are sometimes noted in geothermal areas: measurements at Yellowstone National Park show contents as

high as 500 mg/kg (dw) in sediments from springs and pools and
150 mg/kg in fine-grained muds from mudpots and mud volcanoes.

Transport of mercury from watersheds depends strongly on the content of organic matter, which is usually greatest downstream from wetlands (Zillioux et al. 1993). Dis-turbance of wetland sediments may facilitate mercury transport by changing oxidation states, lowering pH, and resuspending sediment-bound mercury complexes in the water column. Limited measurements of methylmercury (2–14 percent of total Hg) show that disturbed wetlands produce more of it than undisturbed wetlands. Freshwater systems show strong correlations between dissolved organic carbon and filtered total mercury (Zillioux et al. 1993).

Sediment is definitely a source of methyl-mercury to biota and the water column. Even relatively low concentrations may result in bioaccumulation. Guppies (*Poecilia reticulata*) exposed to "control level" sediment (0.24 mg Hg/kg dw) at 21–23 °C achieved whole-body mercury concentrations of 1 mg/kg ww in only 60 days (Gillespie and Scott 1971). Schwarzbach (1993) proposed a sediment toxicity threshold of 0.2 mg/kg (dw) total mercury in sediment to protect the clapper rail (*Rallus longirostrus obsoletus*), a benthic forager, in San Francisco Bay. This sediment toxicity threshold, proposed for guiding sediment criteria in new wetlands created with dredge spoils, was based upon the ratio of the LOAEL for bird eggs (500 mg Hg/kg fww) to the observed bioaccumulation factors for mercury in sediment to mercury in rail eggs in four independent marshes within the bay (Schwarzbach 1993).

Criteria have not been established by the EPA for either total or methylmercury in sediment. Long and Morgan (1991) evaluated a wide variety of *marine* sediment toxicity studies in lab and field for the effects of sediment concentrations on benthic organisms. They established Effects Range-Low (ERL) and Effects Range-Median (ERM) concentrations for each constituent evaluated. The ERL is the lower 10 percentile toxicity value in the database, and the ERM is the median toxicity value. For total mercury the ERL is 0.15 mg/kg (dw) and the ERM is 1.3 mg/kg. Fresh-water sediment criteria for mercury have been proposed by Canada (Smith et al. 1996). These show a threshold effect level of 0.174 mg/kg (dw) and a probable effect level of 0.486 mg/kg. These draft values were calculated based on information compiled in BEDS (biological effects database for sediments) as of January 1994. This is the same data base described by Long et al. (1995). These values are based on toxicity to benthic organisms and not on biological transfer coefficients to benthic predators.

Biotic Effects

The biokinetics and toxicology of organo-mercurials, particularly methylmercury, have been more extensively studied than those of the inorganic form. This is because the methylated form has both greater toxicity and greater bioaccumulation than the inorganic forms. Intestinal absorption of inorganic mercury is limited to a few percent, whereas absorption of methylmercury is nearly
100 percent (Scheuhammer 1987). The ability to demethylate mercury almost certainly confers some relative resistance to mercury toxicity and, together with excretion mechanisms, may account for the high variability in sensitivity to mercury observed between taxa. The half-life of mercury in seabirds has been estimated to be about
60 days (Monteiro and Furness 1995). Inorganic mercury appears to have the greatest effect upon the kidneys, whereas methylmercury is highly toxic to embryos and the nervous system. MeHg readily penetrates the blood-brain barrier, produces brain lesions, spinal cord degeneration, and central nervous system dysfunctions.

Fish

Bioconcentration of mercury in fish (table 23) is influenced by many water quality variables, including temperature, pH, hardness, and mercury speciation. Mercury concentrations in water are reflected, in a dose-dependent manner, in residue levels in fish. The primary focus for most monitoring has been to evaluate the hazard of mercury in fish flesh to human consumption. This focus has utilized either fillets or, less often, whole-body concentrations. The old U.S. EPA aquatic life criterion for mercury (EPA 1980) was not based upon the hazard to fish but rather the hazard to human consumption. The criterion regulated that concentration in water which can be expected to result in a concentration of 1 mg/kg ww in fish—the FDA action level for mercury in the United States. As of 1992, fish mercury levels high enough to warrant consumption advisories had been observed in portions of 26 States (Clean Water Fund 1992). (In recent years, though, most such advisories have been directed at "sensitive subpopulations," such as children and pregnant women, rather than at the general population.) It should also be noted that many State and national governments (Canada, Germany, Florida, etc.) have adopted a more restrictive human health advisory standard of 0.5 mg/kg, consistent with the National Academy of Sciences recommendations (NAS 1978). In a survey of 370 surface water bodies, the Environmental Protection Agency found that fish from15 percent of the water bodies had mercury concentrations above the 0.5 mg/kg level (EPA 1997a).

Table 23.—Effects of mercury residues in fish and herptile tissues

Species	Hg concentration (mg/kg ww)	Tissue	Effect or interpretation	Reference
Fish				
Redbreast sunfish (*Lepomis auritus*)	0.08	Skinless fillet	Background mean	Southworth et al. 1994
Bluegill (*Lepomis machrochirus*)	0.11	Skinless fillet	Background mean	Southworth et al. 1994
Brook trout	2.7	Whole body	Mercury intoxication	McKim et al. 1976
Rainbow trout (*Oncorhyncus mykiss*)	1–5	Whole body	Chronic effects estimate	Niimi and Kissoon 1994
	10–20	Whole body	Lethal estimate	
	13	Based on 25 mg/kg in diet for 189 d	Minimata disease[1]	Southworth et al. 1994
Amphibians/Reptiles				
Amphibian	0.04–0.49	Muscle	"Uncontaminated"	Byrne et al. 1975
American alligator (*Alligator mississippiensis*)	0.08	Brain	Background	Heaton-Jones et al. 1994
	1.37		Irreversible visual impairment suspected	

[1] Symptoms include a rolling swim, inability to stop in front of obstacles, and visual disturbances (Matida et al. 1972).

In addition to human health, another focus of mercury monitoring has been to examine the status of mercury contamination or the trophic transfer of mercury in various aquatic sys-tems. Unfortunately, only a small number of studies have examined mercury in fish as a hazard to the fish themselves. Whole-body concentrations appear most useful for evaluating both the bioaccumulation of mercury and its biological or toxicological hazard to fish. Niimi and Kissoon (1994) strongly advocate the use of whole-body mercury concentrations for evaluating mercury risk to fish, rather than water or any specific tissue concentrations. Whole-body fish residues were specifically recommended over individual organs or fillet tissue con-centrations because of the large degree of uncertainty in identifying the tissues critical
to fish health.

Most mercury in fish is methylmercury (Sorensen 1991). The measurement of total mercury alone in fish is therefore entirely sufficient for evaluating mercury risk. The half-life of methylmercury in fish muscle is estimated at 2–3 years (Sorensen 1991).

There is both field and laboratory evidence that diet is the most important route of fish exposure to mercury; it contributes more than 90 percent of the methylmercury accumulated. The assimilation efficiency for uptake of dietary methylmercury in fish is probably
65 to 80 percent or greater. To a lesser extent, fish may obtain mercury from water passed over the gills, and fish may also methylate inorganic mercury in the gut (Wiener and Spry 1996). However, trout have been shown to be about seven times more efficient at extracting methylmercury from dietary sources than from water via the gills (Phillips and Buhler 1978), and Hall et al. (1994) experimentally confirmed the dietary route of exposure as the most important one for fish.

As is the case for top avian, reptilian, and mammalian predators in aquatic systems, piscivorous fish, particularly long-lived species, may be at risk from mercury bioconcentration and biomagnification. As noted above, measurements of mercury in water prior to the mid-1980s were unreal-istically high. Tissue measurements, however, were reasonably accurate, and hence the bioconcentration factors (BCFs) estimated at that time were unrealistically low (the result of dividing fairly accurate tissue concentrations by overstated water concentrations). Zillioux et al. (1993) point out that BCFs of 23,000 and 81,700 for methylmercuric chloride exposure of brook trout and fathead minnows were used to calculate freshwater final residue values, whereas recent BCF estimates derived from sampling and analyses using clean techniques generally exceed 1 million. The Mercury Study Report to Congress (EPA 1997b) listed median BCFs (called "bio-accumulation factors" in that report) for methylmercury in fish at two different trophic levels. For those at trophic level 3 (fish that feed on plants and plankton), this factor is 1,600,000; for those at trophic level 4 (fish that feed on other fish), it's 6,800,000.

The Mercury Study Report (EPA 1997b) also expressed these BCFs in terms of total dissolved mercury (concentration of all mercury species remaining in the water after filtering), based on the assumptions that
(1) essentially all of the mercury measured in fish tissue is methylmercury, and (2) methyl-mercury averages 7.8 percent of the total dissolved mercury in water. Therefore, the BCFs for total mercury are 124,800 for trophic level 3 and 530,400 for trophic level 4.

These BCFs may be applied to tissue concen-trations associated with harmful effects in order to derive values for possibly harmful total mercury concentrations in water. For instance, McKim et al. (1976) reported reproductive impairment in brook trout that had whole-body mercury concentrations of
2.7 mg/kg (=2,700,000 ng/kg). Applying the total-mercury BCF for trophic level 4 fish gives:

$$\frac{2{,}700{,}000\ \text{ng/kg}}{530{,}400} = 5.0\ \text{ng/kg} \quad \text{OR} \quad 5.0\ \text{ng/L}$$

as the total dissolved mercury concentration at which salmonid reproduction is impaired.

Although the Mercury Study Report to Congress (EPA 1997b) generated data on a range of national BCFs, that report emphasized the value of applying site-specific and field-derived BCFs when developing criteria for specific regions. Factors which affect these site-specific BCFs are many and varied. These include the number of trophic levels present and food web structure, the abundance of sulfur-reducing bacteria, and the concentration of sulfates, dissolved oxygen, temperature, organic carbon availability, pH, the nature of the mercury source, and a number of other parameters (Porcella et al, 1995).

Developing embryos are the most vulnerable life stage to mercury exposure. In all vertebrates, including fish, the transfer of methylmercury to the embryo represents the greatest hazard. According to Wiener (1995), "methylmercury derived from the adult female probably poses greater risk than waterborne mercury for embryos in natural waters." Sublethal and lethal effects on fish embryos are associated with mercury residues in eggs that are perhaps 1 to 10 percent of the residues associated with toxicity in adult fish. Mercury-intoxicated rainbow trout have between 4 and 30 mg/kg in whole bodies, while intoxicated embryos contain 0.07 to 0.1 mg/kg (Weiner 1995).

Both size and species of fish are important variables in mercury sensitivity. Smaller fish tend to accumulate mercury at greater rates than larger fish due to higher metabolic rates (Reinert et al. 1974).

In an extensive evaluation of mercury bio-accumulation in aquatic and benthic life in the contaminated waters of Clear Lake, California, Suchanek et al. (1995) showed that mercury concentrations in fish increased with increasing body size, and they noted the following species-specific differences: carp < silversides < channel catfish < largemouth bass (Suchanek et al. 1993). Bioconcentration factors for mercury from water to silversides in Clear Lake were in the range of 10^4 to 10^5 for total mercury and from 10^6 to 10^7 for methylmercury. Concentrations in large-mouth bass were further increased by as much as 26 times over silversides.

In fish as in other groups, taxonomic differences can also influence mercury susceptibility. Bluegills are capable of demethylation of mercury in the liver (Burrows and Krenkel 1973), but it is doubtful whether rainbow trout have the same ability (Olson et al. 1978). Methylation can occur in the livers of some fish (e.g., albacore and yellowfin tuna) but not rainbow trout or mackerel (Sorensen 1991).

Mercury causes histopathological effects in nearly every fish tissue evaluated (gill, kidney, exocrine pancreas, bile ducts, liver, and erythrocytes). It appears that inorganic mercury is trapped in mucus of the gills, whereas methylmercury traverses this boundary readily.

Amphibians/Reptiles

In a review of metal accumulations in amphibians, Hall and Mulhern (1984) reported that adult amphibians from areas uncontaminated with mercury had mercury concentrations in muscle ranging from 0.04 to 0.49 mg/kg ww; muscle of amphibians from contaminated areas ranged from 1.39 to 2.85 mg/kg ww (Byrne et al. 1975). Hall and Mulhern concluded that amphibians do not seem to accumulate mercury as much as some other species (i.e., predatory birds and some fish). Zoll et al. (1988) examined genotoxicity and bioaccumulation in laboratory studies of the newt *Pleurodeles waltl*. They observed cells with broken chromosomes and others with improper numbers of

chromosomes in blood smears from larvae exposed to both mercuric chloride ($HgCl_2$) and methyl mercuric chloride (CH_3HgCl). Bioaccumulation ratios after 12 days were 600 for mercuric chloride and 1,200 for methyl mercuric chloride.

Birds

Symptoms of acute methylmercury poisoning in birds include reduced food intake leading to weight loss; progressive weakness in wings and legs; difficulty flying, walking, and standing; and an inability to coordinate muscle movements (Scheuhammer 1987). For acute mercury poisoning, brain residues are most diagnostic. Kidney disease and gout also seem to be strongly associated with elevated mercury in the liver (>25 mg/kg ww) (Spalding et al. 1994). If birds have been found dead, and mercury poisoning is suspected, the birds' brain, liver, and kidney concentrations of mercury should be determined in order to confirm the cause.

Birds may show significant adverse effects even at relatively low tissue concentrations if these concentrations result from chronic mercury exposures (table 24). In great white herons, liver mercury contamination >6 mg/kg ww correlated with mortality from chronic diseases (Sundlof et al. 1994). Reproduction is one of the most sensitive physiological processes and may show toxic effects even at very low dietary concentra-tions. Concentrations in the egg are typically most predictive of mercury risk to avian reproduction, but concentrations in the liver have also been evaluated for predicting reproductive risk. The documented effects of mercury on reproduction range from embryo lethality to sublethal behavioral changes in juveniles at low dietary levels. Effects of mercury include reduced hatchability due to increases in early mortality of embryos; some amount of eggshell thinning; reduced clutch size; increased numbers of eggs laid outside the nest; aberrant behavior of juveniles; and potentially may include impaired hearing of juveniles.

The dietary concentrations of methylmercury required to produce significant reproductive impairment are about one-fifth those required to produce overt toxicity in adult birds of the same species (Scheuhammer 1991). In some cases, overall reproductive success in birds has decreased as much as 35–50 percent due to dietary methylmercury exposure insufficient to cause obvious signs of intoxication in adults. Heinz (1979) fed methylmercury to three generations of mallards at the level of 0.5 mg/kg dw (0.1 mg/kg ww). Females laid fewer eggs and produced fewer ducklings. Moreover, the ducklings that survived were less responsive to taped maternal warning calls and were hypersensitive to fright stimulus. Barr (1986) made similar observa-tions in a field study of the common loon in northwestern Ontario. Egg laying and territorial fidelity were both reduced where the mean mercury concentra-tion in loon prey was 0.3–0.4 mg/kg fresh weight; loons established few territories, and none laid any more than a single egg. The eggs contained mercury residues as high as 1.39 mg/kg ww. Around waters where the mean mercury concentrations of prey exceeded 0.4 mg/kg fresh weight, the loons raised no progeny.

The kidney is the major reservoir of inorganic mercury in birds as well as mammals. In renal tissue, mercury binds to metallothionein. Not surprisingly, the major toxic effect of inorganic mercury is kidney damage—specifically, necrosis of the proximal tubular cells (Ware et al. 1975). Spalding et al. (1994) found kidney disease and gout were present in great white herons that had >25 mg/kg ww liver mercury. In the same field study of great white herons, liver mercury contamination >6 mg/kg correlated with mortality from chronic diseases. However, the authors urged caution in interpreting these results because they examined only birds that had been found

Table 24.—Observed effects of mercury residues in bird eggs and tissues

[dw, dry weight; ww, wet weight]

Species	Hg concentration (mg/kg)	Tissue	Effects	Reference
Black duck	4–6 dw	Brain	Eggs failed to hatch	Finley and Stendell 1978
Black-footed albatross	37.4 dw (tot. Hg) 6.2 dw (MeHg)	Kidney	No adverse effect observed	Kim et al. 1996
	306 dw (tot. Hg) 20.4 dw (MeHg)	Liver		
Common loon	>2 ww	Brain	Reduced egg laying; reduced nest-site and territory fidelity	Barr 1986
	29.73 ww	Liver	Reduced nesting success	Barr 1986
	51.9 ww		Reduced hatching success	Fimreite 1974
Common tern	1.0 ww	Egg	Successful reproduction	Fimreite 1974
	3.65 ww		27% hatching success; 10–12% fledging rate	
	1.06 ww	Liver	No effect	Gochfeld 1980
	2.22 ww		Abnormal feather loss—juveniles	
	9.08 ww		Successful nesting	Fimreite 1974
	20.7 ww		27% hatching success; 10–12% fledging rate	
	27.5 ww		10–12% fledge rate	
Gannet	97.7 dw	Liver	Death	Parslow et al. 1973
Grackle	40.4 ww	Kidney	LD33	Finley et al. 1979
	54.5 ww	Liver		
Great white heron	>6 ww	Liver	Correlated mortality from chronic disease	Spalding et al. 1994
	7.2 ww		Increased disease and emaciation	Spalding and Forrester 1991
	>25 ww		Kidney disease, articular gout	Spalding et al. 1994
Grebe	23.3 ww	Liver	Death	Littrel 1991
Grey heron	58.4 dw (11.7 ww)	Kidney	Death	Van der Molen et al. 1982
	95.5 dw	Liver		
Herring gull	2–16 ww	Egg	No decrease in hatchability	Fimreite 1974
Mallard	0.86 ww	Egg	Aberrant nesting behavior	Heinz 1979
Merlin	1–5 dw (0.2–1.0 ww)	Egg	Reduced productivity in ½ of populations	Newton and Haas 1988

Table 24.—Observed effects of mercury residues in bird eggs and tissues—Continued

[dw, dry weight; ww, wet weight]

Species	Hg concentration (mg/kg)	Tissue	Effects	Reference
Osprey	0.05–0.11 ww	Egg	No adverse reproductive effects	Audet et al. 1992
	1.5–3.0 dw (0.3–0.6 ww)	Egg	Decrease in number of young fledged	Odsjö 1982
	35 ww	Liver	Death	Wiemeyer et al. 1987
Pheasant	0.5–1.5 ww	Egg	Decrease in hatchability	Fimreite 1971
Red-winged blackbird	74.3 ww	Kidney	LD33	Finley et al. 1979
	126.5 ww	Liver		
Starling	86.4 ww	Kidney	LD33	Finley et al. 1979
	103.6 ww	Liver		
Various species	30. ww	Liver	Neurologic effects	Scheuhammer 1991
Water birds, generally	1.0–3.6 ww	Egg	Residue threshold for significant toxic effects	Zillioux et al. 1993
	5. ww	Liver	Conservative residue threshold for major toxic effects	
White tailed eagle	33. ww	Liver	Death	Falandysz et al. 1988
	56. ww	Kidney		
Zebra finch	20. ww	Brain	25% mortality	Scheuhammer 1988

dead. Zillioux et al. (1993) found in their review of the literature that concentrations in the liver between 1 and 2 mg Hg/kg ww may result in behavioral effects, whereas liver-mercury concentrations of about 11 mg/kg ww and above lead to high embryo and duckling mortality and to brain lesions. Spalding and Forrester (1991) suggested neurological effects may be associated with liver-mercury levels in birds as low as 5 mg/ kg ww. Zillioux et al. (1993) concluded that a conservative residue threshold for major toxic effects in waterbirds would be 5 mg/kg ww in the liver.

However, apparently normal seabirds have been found with mercury concentrations many times this level in the liver, but analysis has shown these concentrations to be pri-marily inorganic mercury (Kim et al. 1996). In some species, especially Procellariformes, it appears that demethylation of mercury is an important detoxification strategy. Therefore, characterizing the different forms of mercury in tissues is increasingly recognized as important to meaningful interpretation of residue data.

In the majority of wild birds sampled, liver concentrations of mercury are higher than kidney concentrations. However, in some cases of mercury poisoning, kidney con-centrations are found to be nearly the same as the liver concentration (Lewis and Furness 1991). Kidney concentrations of 20 mg/kg ww have been noted in birds found dead in mercury-contaminated environments (Littrel 1991).

Brain mercury as low as 3–7 mg/kg ww can be lethal to ducklings. Concentrations four times this high are required to cause direct mortality in adults. The lowest concentration of mercury in the brain to produce obvious signs of

intoxication in adults is 5 mg/kg dw or 1 to 1.6 mg/kg ww (Scheuhammer 1991). Heinz and Locke (1975) found that the brains of mallard ducklings found dead with brain lesions contained an average of 6.17 and 5.19 mg Hg/kg ww in two successive years.

The toxic effects of mercury in bird eggs have been documented by many investigators in both laboratory and field studies (Barr 1986; Birge et al. 1976; Fimreite 1971, 1974; Heinz 1974, 1979; Heinz and Locke 1975; Hoffman and Moore 1979; Finley and Stendell 1978; Tejning 1967; etc.). Mercury is an extremely potent embryo toxicant, and dietary mercury is transferred to avian eggs in a dose-dependent manner. Reproductive impair-ment is one of the most sensitive endpoints of mercury toxicity. Mercury accumulates particularly in the egg-white proteins, which derive from serum proteins. Egg concentrations, therefore, more closely reflect mercury from recent dietary uptake than from accumulated tissue stores. There is also evidence that the ovalbumin fraction of egg white has a specific affinity for dietary mercury, while the ovoglobulin fraction tends to accumulate low levels of "nondietary" mercury. Because of the strong dietary connection, Walsh (1990) suggested that eggs provide a particularly good indicator of mercury exposure in the vicinity of the nesting site in the immediate pre-laying season. One can expect methylmercury to predominate in eggs, particularly within the albumen fraction. Because mercury is predominantly deposited in albumen, more intra-clutch variation in mercury content is also to be expected than in contaminants preferentially distributed to yolk. Becker (1992) reports that, among the Charadriiformes, the last egg of a clutch commonly has lower mercury content than the first egg. The first egg laid contained as much as 39 percent more mercury than the second or third egg. Becker et al. (1994) predict that the toxic effects of mercury would be more pronounced in *a*-chicks (the chick from the first laid egg). In elevated mercury environments, this will result in abnormally high losses of *a*-chicks, a reversal of the normal situation. Barr (1986) documented adverse effects on loons associated with egg concentrations of 1.39 mg/kg ww.

Hoffman and Moore (1979) treated mallard eggs with externally applied methylmercury chloride. Effects were dose related and included decreased embryo weights, developmental abnormalities, and embryonic death. The lowest dose applied which affected survival was 27 micrograms. Given an average mallard egg weight of 55 grams, this dose corresponds to about 0.5 mg/kg. With increasing concentrations, abnormalities progressed in severity from mostly minor skeletal deformities to gross external ones such as micromelia, gastroschisis, and eye defects as well as internal deformities such as brain defects and a reduction in liver size. Such laboratory work is useful because it may efficiently elucidate the types of effects that can be produced, but these results should not be literally extrapolated to the field. External mercury exposures by Hoffman and Moore had more pronounced effects at lower doses than organic mercury incorporated into the egg through the hen's metabolism (Heinz 1974) presumably because the applied mercury was not completely bound to the ovalbumin and ovoglobulin.

Reproductive effects may extend beyond the embryo and may reduce the rate of juvenile survival. Mercury in the eggs of mallards has caused brain lesions in hatched ducklings. Heinz and Locke (1975) reported on mallards that were fed 3.0 mg/kg methylmercury dicyandiamide (equivalent to 0.6 mg Hg/kg in a natural succulent duck diet) over two successive years. Mercury accumulated in the eggs to an average of 7.18 and 5.46 mg/kg ww in the two years. Lesions included demyelination, neuron shrinkage, necrosis, and hemorrhage in the meninges overlying the cerebellum.

In a field study of total mercury in eggs of common terns, Fimreite (1974) estimated the threshold level for toxic effects to be between 1.0 and 3.6 mg/kg ww. Heinz (1979) was able

to relate egg concentrations to subtle behavioral effects in mallard ducklings. As described earlier, he fed ducks a diet including 0.5 mg Hg/kg dw over three generations and found decreased reproductive success and altered behavior of ducklings. The mean mercury concentration in eggs in this study was 0.86 mg/kg (fww). In a study of ring-necked pheasants, Fimreite (1971) found a significant reduction in hatchability associated with dietary mercury levels between 0.5 and 1.5 mg/kg ww. The low end of this effect range continues to be the lowest observed adverse effect level (LOAEL) for mercury in bird eggs.

Establishing effect levels based on mercury concentrations in feathers must be considered with caution. Feathers represent a route of excretion and not a target organ. Mercury is deposited in feathers at the time of molt, when there is active feather growth and a corresponding blood supply to the growing feather (Goede and deBruin 1984; Furness et al. 1986; Braune 1987). Once mercury is in feathers, it is bound to the sulfide bonds of feather keratin and is not physiologically available for redistribution to target organs. Mercury content of feathers will vary with time to last molt, feather type, and age and species of the bird (Monteiro and Furness 1995). Feathers have the advantage of being a nondestructive exposure-assessment matrix which may be resampled in the same individual and which may also be compared with museum specimens (Applequist et al. 1984). The concentration of mercury in tissues may actually decrease during molting as mercury is mobilized from tissues into feathers (Furness et al. 1986). In sequential feather-loss patterns, the first primary feather to be grown back has the greatest mercury concentration, and the concentrations decrease in subsequent feathers (Lewis and Furness 1991; Braune 1987; Braune and Gaskin 1987). Becker et al. (1994) found results in three species of larids which implied that mercury in the first down of chicks was a consequence of mercury levels in the egg, whereas levels in feathers of chicks were largely due to mercury ingested in food. Lewis and Furness (1991) found that in laboratory-reared black-headed gulls, 49 per-cent of the administered mercury was accumulated in the plumage independent of the dose administered. The percentage of the mercury body burden found in plumage varies from species to species.

Almost all feather mercury is the organic form (Thompson and Furness 1989). Species that are effective in demethylating mercury, such as the Procellariformes, will tend to have a lower percentage of their total mercury body burden partitioned into the feathers than other species do (Kim et al. 1996). This character-istic has been interpreted as an adaptation to the slow molt of feathers in Procellariformes; inasmuch as they do not shed feathers as quickly as other species, the feathers are less useful as a medium for the sequestration and ultimate excretion of methylmercury (Kim et al. 1996).

The molt pattern of any given species will also have a large influence on the variation of mercury content between different feathers within an individual bird (Applequist et al. 1984). Greater variation in mercury with feather type should also be expected in more contaminated environments (Becker et al. 1994). The timing of feather growth may also influence mercury accumulation in other tissues if the levels of mercury exposure differ greatly between the birds' wintering and breeding grounds. For meaningful quantitative monitoring of mercury using feathers, the feather/mercury pattern for a species should be established and similarly sampled among those individuals or populations which are to be compared. For historic comparisons using older museum specimens, especially if preservation methods are vaguely recorded, it may be prudent to determine both total mercury and methyl-mercury in feathers to evaluate the relative contribution of mercurial used in preservation of the avian skins.

In a review of effects related to mercury concentrations in feathers, Eisler (1987)

reported that concentrations between 5 and 40 mg/kg in feathers are linked to impaired reproduction. Sterility was observed in the Finnish sparrow hawk (*Accipiter nisus*) at feather mercury concentrations of 40 mg/kg. Bowerman et al. (1994) found that feathers of bald eagles in the Great Lakes region had mercury concentrations of 13 to 21 mg/kg, but they could make no association between mercury concentrations and bald eagle reproduction. Scheuhammer (1991) suggests that feather mercury concentrations >20 mg/kg can result from diets containing >1 mg Hg/kg and that these concentrations probably indicate a wetland that poses a mercury risk to birds. He estimates the normal background of mercury in raptor feathers to be 1–5 mg/kg.

Mammals

Though far fewer studies have been conducted assessing mercury toxicity in mammals than in birds, many of the general mechanisms are similar. Like birds, mammals accumulate mercury from various environ-mental matrices, but those living in or near water tend to accumulate the most. Kucera (1983) reports that mink and river otter, in drainage areas supporting 16 pulp and paper mills and a chlor-alkali plant, accumulated 10 times more mercury than predatory fish from the same drainage areas. Generally, carnivorous or piscivorous animals tend to have the highest body burdens, omnivores have intermediate body burdens, and herbivores tend to have the lowest body burdens. There is also an age-related effect: older animals tend to have higher body burdens than younger ones. This is probably due to a combination of factors including the length of time the older animals have had to bioaccumulate mercury, the younger animals' higher metabolic rate, and the older animals' slower rate of mercury excretion.

In mammals, the highest mercury concentrations are generally found in hair or in liver or kidney tissue, depending upon the species (table 25). Muscle and brain tissue also tend to accumulate mercury. The primary route of excretion is through the hair. Animals that grow heavy winter coats and shed them in summer tend to excrete more mercury than those that don't. Mercury excreted through the hair is bound to proteins, just as in bird feathers.

Manifestations of mercury poisoning in mammals include loss of muscle coordination, loss of appetite, and sensory impairment. Sensory impairment has been described in humans and monkeys as constriction of the visual field and loss of hearing. Organic mercury is more toxic to mammals than inorganic mercury; methylmercury irreversi-bly destroys the neurons of the central nervous system. Animals exposed to sub-lethal mercury concentrations, or suffering from chronic low-level exposure, may appear normal. When stressed, though, they may not be able to perform adequately to survive the rigors of living in the wild.

Mercury crosses the placental barrier and reaches the developing fetus. A great deal of research has been done studying the impacts of occupational and accidental mercury exposures to human fetuses. From this research, the fetus is known to be much more sensitive than the adult to the ill-effects of mercury (Girard and Dumont 1995). For all organisms tested, early developmental stages have been shown to be the most sensitive to mercury poisoning (Eisler 1987). Methyl-mercury is furthermore a known teratogen and mutagen.

Bioaccumulation

Mercury strongly bioaccumulates and even biomagnifies through trophic levels in aquatic systems. Biomagnification of mercury has been documented in birds (Fimreite 1974), fish, and even zooplankton communities (Watras and Bloom 1992). Within aquatic

Table 25.—Mammalian mercury exposures and associated effects

Species	Hg concentration (mg/kg dw)	Tissue	Effects	Reference
Cat (*Felis domesticus*)	121–392 (fw)	Hair	Death	Jenkins 1980
Mink (*Mustela vison*)	30–40	Liver	Death; also suffering from cold stress	Wren et al. 1987
	4–18	Brain		
	7.8	Muscle	Nervous system pathology	Burton et al. 1977
	25.4	Liver	Nervous system pathology	Hallbrook et al. 1994
	58.2	Liver	Death	Wobeser et al. 1976
	34.9	Fur		
	31.9	Kidney		
	15.2	Muscle		
	13.4	Brain		
Monkeys	1.2–4	Blood	Visual disturbance	Suzuki 1979
	6–9	Brain	Visual disturbances	Burton et al. 1977
Mountain lion	110	Liver	Death	Roelke 1990
River otter (*Lutra canadensis*)	13.5	Muscle	Death	Hallbrook et al. 1994
	30	Liver	Death	
White-footed mouse (*Peromyscus maniculatus*)	0.31	Hair	No effect	Burton et al. 1977
	10.8		Normal appearance; poor performance under stress	

systems, the net rate of methylation/ demethylation processes in water and sediments ultimately governs the bio-availability of mercury. Both processes are biological, and demethylation is better understood than methylation (Zillioux et al. 1993). Several factors have been shown to influence the net rate of methylmercury production. Those that increase production include high dissolved organic carbon content, low pH, the presence of sulfides, and high temperatures.

Within a watershed, wetlands enhance the rate of methylmercury production (Lee and Hultberg 1990), and so do reservoirs, particularly new reservoirs. The "new reservoir effect," whereby a pulse of methylmercury is produced in the first few years of reservoir filling, is usually credited to the initial nutrient input from decaying terrestrial vegetation killed by the rising waters. It should be noted that this effect is not limited to those regions that have local geological or industrial sources; atmospheric input alone may be sufficient to produce a detectable pulse of methylmercury. Although mercury bioconcentration factors (BCFs) from water to fish of over a million are not uncommon (Watras et al. 1994; Bloom et al. 1991; Porcella 1994; Suchanek et al. 1993), Porcella has cautioned that these BCFs may not be applicable over a wide range of water qualities because so many factors influence mercury bioavailability.

Interactions

Mercury is known to interact toxicologically with other elements in additive, synergistic, and antagonistic ways. Its interactions with selenium are of particular interest. Both mercury and selenium bioaccumulate, both bind to organo-thiol groups, and both have their greatest toxic effect through dietary exposure to the organic forms. Interactions between selenium and mercury have been extensively documented (Cuvin-Aralar and Furness 1991; Sorensen 1991), although many conflicting results have been reported. These interactions vary greatly in character and in strength, depending on whether the forms of the two elements are organic or inorganic. The exact chemical speciation, and other factors, can cause the toxicity of selenium to be increased, reduced, or unaffected by the presence of mercury. Selenite has been shown to protect against kidney poisoning caused by inorganic mercury salts. El-Begearmi et al. (1977) demonstrated that sodium selenite reduced the toxicity of methylmercury and increased the survival of Japanese quail. However, Heinz and Hoffman (1998) found conflicting results when they studied the interactions of selenomethionine and methylmercury in the diets of captive mallards: the selenium and mercury together were less likely to poison adult birds than either element separately but more likely to impair reproduction. The presence of methylmercury in the diet also greatly enhanced the storage of selenium in both eggs and livers, and, similarly, the presence of selenium enhanced the storage of mercury. Teratogenesis was most severe in the eggs of mallards that had been fed both methyl-mercury and selenomethionine.

Regulatory Standards

U.S. Environmental Protection Agency Standards and Criteria
[See Appendix II for explanation of terms. Source: EPA 1986, 1995, 1997a, 1997b]

Status	EPA priority pollutant
Drinking water MCL	2 µg/L
Drinking-water health advisories for 70-kg adult	Reference dose: 0.3 µg/kg/day Long-term HA: 2 µg/L Lifetime HA: 2 µg/L DWEL: 10 µg/L
Freshwater criteria (for dissolved Hg)	2.4 µg/L for acute exposure 0.012 µg/L for chronic exposure
1/1,000,000 cancer risk	0.144 µg/L (water and organisms) 0.146 µg/L (organisms only)
Human health criterion	0.05 µg/L total Hg (water; based on bioconcentration in fish)
Wildlife criterion for protection of bald eagles	0.000082 µg/L methylmercury (based on biomagnification through multiple trophic levels)
Wildlife criterion for protection of piscivorous species	0.00005 µg/L methylmercury (≈0.00064 µg/L total mercury)

The U.S. Food and Drug Administration action level for methylmercury in the edible portions of fish, shellfish, crustaceans, and other aquatic animals is 1.0 mg/kg ww (FDA 1992).

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

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Molybdenum

Description

Molybdenum (Mo) is a silver-white metallic element of the second transition series; its atomic number is 42, and its atomic weight is 95.94 (Pais and Jones 1997). It has chemical properties similar to those of chromium. It is commonly used in steel alloys because it imparts hardness, strength, heat resistance (melting point 2,617°C), and corrosion resistance to these alloys. Molybdenum is present in all plant and animal tissues and is considered an essential micronutrient for most life forms (Schroeder et al. 1970; Underwood 1971; Chappell and Peterson 1976; Chappell et al. 1979; Goyer 1986; Eisler 1989).

Occurrence

Molybdenum does not occur free in nature and is only found in combination with sulfur, oxygen, tungsten, lead, uranium, iron, magnesium, cobalt, vanadium, bismuth, or calcium. Its principal ore is molybdenite (MoS_2), "a lead-gray hexagonal mineral . . . [which] resembles graphite in appearance and to the touch, but has a bluer color" (Bates and Jackson 1987). Less important sources include wulfenite ($PbMoO_4$), powellite ($CaMoO_4$), and molybdophyllite ($PbMoSiO_4$). Molybdenum is widely disseminated in the environment: its abundance in the Earth's crust is estimated at 1–1.5 mg/kg (Budaveri 1996); and back-ground concentrations in the United States are 1.2–4.1 µg/L for rivers, <1 µg/L for ground-water, 5–57 mg/kg dw for river sediments, and 1.2 (0.1–40) mg/kg dw for soils (Friberg et al. 1975; Chappell et al. 1979). Molybdenum concentrations in animal tissues are generally highest in liver, followed by kidney, spleen, lung, brain, and muscle (Berman 1980). Total body molybdenum is present to the largest degree in skeletal tissue (Underwood 1977).

Molybdenum is used primarily in the manufacture of steel alloys for aircraft and weapons. It is also used as an electrode material and as a catalyst in petroleum refining. Most of the recent global production of about 100,000 tons annually comes from the United States. Three mines in Colorado account for nearly 70 percent of domestic pro-duction. Human activities that contribute to molybdenum contamination include the combustion of fossil fuels, and smelting, mining, and milling operations for steel, copper, and uranium.

Summary of Effects

Table 26 summarizes the predicted effects of environmental exposures to molybdenum, based on the limited information currently available.

Study Approaches

The majority of papers reviewed for this report were laboratory studies dealing with molybdenum effects on mammals and poultry. Mammalian literature consisted of studies done on domestic animals, primarily rats, and was published from the late 1940's to early 1960's. Avian literature consisted exclusively of poultry studies published in the late 1950's to early 1960's. For aquatic species, the literature was composed primarily of freshwater laboratory studies and offered little information about fish, invertebrates, amphibians, or reptiles. The review adequately addressed the formally published scientific

Table 26.—Predicted molybdenum effect levels

Media	No effect	Level of concern	Toxicity threshold	Explanation
Water (mg/L)	0.02	0.02–0.12	0.12	For fish. 0.02, upper limit of natural background (Eisler 1989); 0.12, LC10 for larval trout (Birge et al. 1980)
	0.02	0.02–0.96	0.96	For amphibians. 0.02, upper limit of natural background (Eisler 1989); 0.96, LC50 for larval toads (Birge 1978)
	- - -	- - -	>50	For plants. Reduced growth of green algae; 96-h exposure
Domestic chickens (mg/kg in feed)	- - -	>500	>6,000	Adverse effects on reproduction and on survival, respectively
Bird eggs (mg/kg dw)	23	23–33	<33	Lepore and Miller (1965)
Mammals (Cu:Mo ratio in feed)	6:1–10:1	<2:1 *or* >10:1	- - -	Ratios found to lead to either Cu deficiency or Cu toxicosis

literature, but not the scientific "gray literature," which includes government reports and unpublished studies.

Abiotic Factors Affecting Bioavailability

Water

Natural molybdenum concentrations in ground and surface waters rarely exceed 20 µg/L; higher concentrations probably indicate industrial contamination (Eisler 1989). Concentrations in surface waters range from 0.4 µg/L in uncontaminated North American rivers to as much as 100,000 µg/L in mining wastewater. In the United States, ground-water molybdenum concentrations are usually <1 µg/L but have been reported as high as 50,000 µg/L near uranium mills in Colorado (Eisler 1989). Molybdenum concentrations in saline water appear to be directly related to salinity (Prange and Kremling 1985; Sloot et al. 1985).

Aquatic organisms are relatively resistant to molybdenum and generally show no adverse effects on growth or survival at water concentrations lower than 50 mg/L (Eisler 1989); however, there are large differences between species in their ability to bioconcentrate molybdenum. Blue-green algae (*Anabaena oscillaroides*) had a bioconcentration factor (BCF) of 3,300 after 1 hour of exposure at a concentration of 0.005 µg Mo/L (Steeg et al. 1986). The freshwater alga *Nitella flexilis* and some lake periphyton had BCFs of 628 and 3,570, respectively, in 25 days when placed in a 0.014-µg Mo/L concentration (Short et al. 1971). In a 3.3-mg Mo/L con-centration, crayfish (*Pacifastacus leniusculus*) had BCFs of 5.7 in muscle and 9.8 in the carapace. High bioconcentration of molyb-denum by certain species of aquatic algae and invertebrates has been recorded without apparent harm to the organism; however, the hazard potential to organisms that feed on the bioconcentrators is not clear (Eisler 1989).

Soil

Soils average 1–2 mg/kg molybdenum, although they range from trace concentrations to 40 mg/kg or greater. In the United States,

molybdenum concentrations in soils generally increase from east to west (Adriano 1986; Kubota 1977).

The largest concentrations of molybdenum are found in the 30 cm of soil nearest the surface. Its uptake into certain legumes and other plants may correlate with the soluble molybdenum concentrations in the soil, but this relationship does not occur with all types of plants. Molybdenum uptake by plants can vary dramatically even between different varieties of the same species (Barshad 1948).

Biota

Plants

Molybdenum is considered essential for aquatic plant growth, but the concentrations required are not known. Aquatic plants are relatively resistant to molybdenum toxicity. Concentrations observed to cause adverse effects in sensitive species were 50 mg/L for growth and 108 mg/L for development (table 27).

The molybdenum content of some plants has been shown to vary by stage of development, as evidenced by a twofold to threefold increase from spring to fall in leaf and stem concentrations of alfalfa and some grasses. Although older plants may contain more molybdenum, younger plants appear to cause more molybdenosis in animals. This dif-ference may arise because animals consume different parts of young, succulent plants.

Fish

Acute toxicity values for molybdenum in the literature (table 27) indicate that it is relatively nontoxic to fish. The one exception was newly fertilized eggs of rainbow trout exposed for 28 days through 4 days posthatch; these had an LC50 of 0.79 mg/L and an LC10 of 0.12 mg/L. In general, molybdenum was more toxic to younger fish than to older fish, although a study by Hamilton and Buhl (1990) found that the 96-hr LC50 values for all Chinook and coho salmon exceeded 1,000 mg/L regardless of the quality of the dilution water (soft, fresh, or brackish) or the life stage tested (eyed egg, alevin, or fry). Moreover, the addition of molybdenum to test mixtures of boron, selenite, and selenate seemed to increase the acute toxicity of these mixtures to Chinook and coho salmon (Hamilton and Buhl 1990).

Few studies have compared the molybdenum concentrations in fish tissues to ambient concentrations, and the toxicological effects of molybdenum in fish tissues are unknown (Eisler 1989; Saiki et al. 1993). As shown in table 27, Ward (1973) found that, in nature, tissue molybdenum concentrations in rainbow trout increased only slightly with increasing water concentrations. Saiki et al. (1992) confirmed Ward's observations in controlled experiments. An eightfold range of waterborne molybdenum caused very little variation in the concentration of molybdenum in tissues of juvenile Chinook salmon or striped bass.

Birds

There are no data showing molybdenum's effects on wild birds. In domestic birds, adverse effects have been reported for growth at dietary molybdenum levels >200 mg/kg, for reproduction at 500 mg/kg, and for survival at 6,000 mg/kg (Eisler 1989). Poor growth was the only symptom of molyb-denum toxicity noted by Miller and Denton (1959) even at 2,250 mg Mo/kg added to the diet. In all groups in which the level of added molybdenum inhibited growth, inorganic sulfate alleviated part of the growth inhibition. The addition of inorganic sulfate caused a considerable decrease in the molybdenum content of liver tissues. Liver molybdenum

Table 27.—Effects of molybdenum on living organisms as reported in published studies

Species	Mo concentration (mg/L *or* mg/kg)	Where measured	Effects	Reference
Plants				
Euglena gracilis	108	Water	Abnormal development	Colmano 1973
	>960		No growth	
Green algae, *Chlorella vulgaris*	50	Water	Reduced growth after 96 h	Sakaguchi et al. 1981
Invertebrates				
Amphipod, *Crangonyx pseudogracilis*	2,650	Soft water	96-h LC50	Martin and Holdich 1986
	3,618		48-h LC50	
Hermit crab, *Eupagurua bernhardus*	222	Water	48-h LC50	Abbott 1977
Fish				
Chinook and coho salmon (eyed eggs, alevins, and fry)	>1,000	Fresh, brackish and soft water	96-h LC50	Hamilton and Buhl 1990
Fathead minnow, *Pimephales promelas*	70	Soft water	96-h LC50	McConnell 1977
	360	Hard water		
Rainbow trout, *Oncorhynchus mykiss*	Trace	Water	Mo in tissue 5–118 µg/kg ww	Ward 1973
	6		Mo in tissue 10–146 µg/kg ww	
	300		Mo in tissue 13–332 µg/kg ww	
	800		96-h LC50 (20-mm size class)	McConnell 1977
	1,320		96-h LC50 (55-mm size class)	
Rainbow trout (fertilization through 4 days post-hatch)	0.12	Moderately hard water	28-d LC10	Birge et al. 1980
	0.79		28-d LC50	
Sheepshead minnow, *Cyprindon variegatus*	3,057	Water	96-h LC50	Knothe and Van Riper 1988
Amphibians				
Frogs	2,000	Water	Zone of toxic action	Venchikov and Kaprielov 1976

Table 27.—Effects of molybdenum on living organisms as reported in published studies—Continued

Species	Mo concentration (mg/L *or* mg/kg)	Where measured	Effects	Reference
Birds				
Chicken	2,250 Mo	Dietary; basal diet 0.25% sulfur, 3.3 ppm Mo, and 13 ppm Cu	Body weight 37% that of control chicks	Miller and Denton 1959
	2,250 Mo + 13,200 sulfate		Body weight 72% that of control chicks	
	750 Mo + 2,200 sulfate		Decrease in Mo content in liver tissues of chicks	
Chicken (chicks)	200 (sodium molybdate)	Diet	Reduced growth after 56 d	Arthur et al. 1958
	300 (sodium molybdate dihydrate)	Diet	25% reduced growth after 24 d	Kratzer 1952
	500 (sodium molybdate dihydrate)	High-sulfate purified diet	Minimum toxic dose after 28 d; lowest concentration depressing growth	Davies et al. 1960
Turkey (poults)	300 (sodium molybdate dihydrate)	Diet	25% reduced growth after 24 d	Kratzer 1952
Mammals				
Cattle, *Bos* spp.	60	Diet	Low Cu in liver; intestinal disturbances; brittle bones prone to fracture	Penumarthy and Oehme 1978
Cattle (lactating cows)	40 Mo, 6 Cu	Diet	30% reduction in milk yield after 63 d; rapid decline in plasma copper; milk Mo levels 1.6 ppm; growth reduction in nursing calves	Wittenberg and Devlin 1987
Mouse (*Mus* sp.)	10	Drinking water	Decrease in survival of 2d and 3d generations	Earl and Vish 1979
Mule deer	2,500 (sodium molybdate)	Diet	Weight loss after 27 d	Ward and Nagy 1976
	5,000 (sodium molybdate)		Reduced feeding after 14 d	
Rat	80	Cu-deficient diet	Inhibited growth and reduced survival	Underwood 1971, 1979
	5000	Diet	Lethal in 2 weeks	Chappell et al. 1979; Friberg et al. 1975

Table 27.—Effects of molybdenum on living organisms as reported in published studies—Continued

Species	Mo concentration (mg/L *or* mg/kg)	Where measured	Effects	Reference
Mammals—Continued				
Rabbit (*Oryctolagus* sp.)	100	Diet	Reduced growth, hair loss, dermatosis, anemia, skeletal and joint deformities (lifetime exposure)	Chappell et al. 1979
	1000	Diet	Weight and hair loss, leg deformities, dermatosis, anemia, death (28 d)	Arrington and Davis 1953
	2,000–4,000	Diet	Many deaths of weanlings in 37 d, adults in 53 d; survivors were anorexic, diarrhetic, anemic, and had front leg abnormalities	Friberg et al. 1975; Arrington and Davis 1953
Sheep, *Ovis* spp.	~5.5–12.5	Grazing pastures treated with 420 g Mo/ha at start, week 45, and week 72	Lameness, connective tissue lesions in most sheep; Mo concentrations (mg/kg fw): plasma 1.7, liver 6.0–6.4; kidney 6.9–8.1	Pitt et al. 1980
Sheep (lambs)	Cu:Mo < 0.4	Soil	15–39% swayback	Friberg and Lener 1986

concentrations of 22-36 mg/kg dw (6–10 mg/kg ww) have been correlated with toxic effects in domestic birds (Puls 1988). When copper was added to the diet, in addition to molybdenum and inorganic sulfate, a further reduction in molybdenum liver tissue concentrations was observed. These studies show that the amount of molybdenum stored by the liver tissues is dependent upon the amount in the diet and upon the ratios of molybdenum, copper, and sulfate in the diet. Increasing the molybdenum content of the diet increased the copper storage of the liver.

Avian eggs normally contain <1 mg Mo/kg (dry weight basis), averaging about 0.25 mg Mo/kg (Romanoff and Romanoff 1949). Lepore and Miller (1965) studied the effects of maternally deposited molybdenum content on the viability (i.e., hatchability) of eggs laid by White Rock chickens. They observed normal egg viability up to about 23 mg Mo/kg in the egg (dry weight basis). At 33 mg/kg, about 50 percent of the eggs were inviable (i.e., the approximate EC50). Thus, the threshold for avian embryotoxicity occurs between 23 and 33 mg Mo/kg egg (dry weight basis). The EC100 concentration was approximately 60 mg/kg. Based on transfer rates of molybdenum from the maternal diet to the eggs, as documented by Lepore and Miller (1965) and Motzok et al. (1957), the dietary threshold for reproductive impairment lies somewhere between 100 and 500 mg Mo/kg (dry feed basis). This suggests that, in the absence of significant interaction effects, molybdenum-induced avian embryotoxicity in the field may be very rare. Lynch et al. (1988) reported that even downstream from spills of molybdenum mill tailings in the Red River of New Mexico,

benthic invertebrates averaged only 29 mg Mo/kg (dry weight basis). Evaporation ponds for subsurface agricultural drainage water in California's Tulare Lake Basin were found to contain as much as 40,000 μg Mo/L in the water (Westcot et al. 1988), yet the maximum concentration of molybdenum in aquatic invertebrates was about 80 mg/kg (dry weight basis; Moore et al. 1989), and the maximum level in avian eggs was 16 mg/kg (Ohlendorf et al. 1993). Presumably, cases of environmental contamination with molybdenum more severe than these examples would be extremely rare.

Mammals

Currently available data for molybdenum's effects on wild mammals are inadequate (table 27). The toxicological properties of molybdenum in mammals are governed by its interaction with copper and sulfur; residues of molybdenum alone are not sufficient to diagnose molybdenum poisoning (Eisler 1989). The optimum dietary copper: molybdenum ratio (Cu:Mo) is between 6:1 and 10:1 (assuming that concentrations of both elements are above minimum requirements). A Cu:Mo ratio less than 2:1 will result in a copper deficiency, whereas a Cu:Mo above 10:1 increases the risk of developing copper toxicosis, particularly in sheep (Osweiler et al. 1985).

Molybdenosis is a copper-deficiency disease that is caused by the depressing effect of molybdenum on the physiological availability of copper (Clawson et al. 1972; Dollahite et al. 1972; Alloway 1973; Erdman et al. 1978; and others cited in Eisler 1989). Because of the unique environment of the rumen, cattle and other ruminants are far more susceptible to the toxic effects of molybdenum than other species. Toxicity generally occurs when cattle graze pastures where the forage contains 20–100 mg Mo/kg dw (Underwood 1979). Younger animals and lactating cows appear more susceptible. Molybdenosis can be controlled by oral or intravenous administration of copper sulfate.

Where ruminant diets contained copper at 8–11 mg/kg dw, cattle were poisoned at molybdenum levels of 5–6 mg/kg and sheep at 10–12 mg/kg. Where dietary copper was low (<8 mg/kg) or the sulfate-ion level was high, molybdenum at 1–2 mg/kg ration was toxic to some cattle (Buck 1978).

Generally in monogastric animals, sulfate protects against molybdenum toxicity, whereas in ruminants it enhances the toxicity. Sulfate alleviated molybdenum-induced symptoms in rats, chicks, and rabbits. In ruminants, molybdenum toxicosis was induced by feeding diets supplemented with both molybdenum and sulfate to sheep and cattle. Sulfate greatly increased the severity of molybdenum toxicosis in cattle. Sulfate intensified molybdenum toxicity in copper-deficient rats but prevented molybdenum toxicity in copper-sufficient rats. Where sulfate was used to alleviate molybdenum toxicity in monogastric animals, the dietary level of sulfate was in the range 1,500– 8,000 mg/kg. In sheep, molybdenum toxicosis was produced by feeding diets containing molybdenum at levels of 2–50 mg/ kg and sulfate at 4,000–10,000 mg/kg (Pitt 1976).

Some animals may be able to adapt to excess molybdenum over successive generations. When compared to rats on a control diet, second- and third-generation rats exposed to excess dietary molybdenum did not show physiological alterations like those seen in first-generation rats (such as reduced stress response) (Winston et al. 1976).

Interactions

Molybdenum toxicological properties are governed to a large extent by interactions with copper and sulfur, but interactions with other metals and compounds may confound this interrelation. For molybdenum, interactions are so dominant that a particular level of intake in an animal's diet can lead to either molybdenum deficiency or toxicity, depending on the relative intakes of copper and inorganic sulfur (Schroeder et al. 1970; Underwood 1971, 1979; Clawson et al. 1972; Suttle 1973, 1983; Friberg et al. 1975; Buck 1978; Friberg and Lener 1986; Goyer 1986; Kincaid et al. 1986; and others cited in Eisler 1989). A low copper-to-molybdenum ratio (<2), rather than the absolute dietary concen-tration of molybdenum, is the primary determinant of an organism's susceptibility to molybdenum poisoning. The first indications of the interaction between copper and molybdenum came from England more than 40 years ago, when cattle grazing on herbage rich in molybdenum developed molybdenosis. Molybdenosis is not expected to occur in animals when the copper-to-molybdenum ratio is near 5 (Buck 1978; Ward 1978; Mills and Bremner 1980).

On the other hand, studies of molybdenum metabolism are of limited value unless the status of inorganic sulfate in the diet is known (Underwood 1971, 1979); inorganic sulfate alleviates molybdenum toxicity by increasing molybdenum excretion. Molybdenum levels in animal tissues give little indication of the dietary molybdenum status and are of little value for diagnosing molybdenum toxicity unless the sulfate, protein, and copper status of the diet are also known (Eisler 1989).

Regulatory Standards

U.S. Environmental Protection Agency standards and criteria
[See Appendix II for explanation of terms. Source: EPA 1995]

Status	Listed for regulation; carcinogenicity unknown.
Drinking water MCL	None established
Drinking-water health advisories for 70-kg adult	Reference dose: 5 µg/kg/day Long-term HA: 50 µg/L Lifetime HA: 40 µg/L DWEL: 200 µg/L

No regulatory standards currently exist for the protection of fish and wildlife from dietary exposure to molybdenum. Molybdenum is not an EPA priority pollutant, and no national water-quality criteria for the protection of freshwater aquatic life have been developed. For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

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Salinity

Description

Salinity is a measure of the mass of dissolved salts in a given mass of solution. The term "salinity" includes many different types of salts. Salinity is not precisely equivalent to total dissolved solids content (TDS), but the two terms are closely related. For most purposes, they can be considered equivalent ([EPA 1986).

Measurement Techniques and Units

Salinity is usually assessed by measuring some related physical property, such as conductivity, density, sound speed, or refractive index. The conductivity and density methods are recommended for their high sensitivity and precision, and conductivity can be measured easily with field meters. The conductivity of a water sample is determined by measuring its electrical resistance between two electrodes and comparing this resistance with that of a standard solution of potassium chloride at 25 °C. Conductivity is the reciprocal of the measured resistance (Hem 1985).

Salinity is usually expressed in parts per thousand (ppt) for marine or other highly saline waters (e.g., those in the Salton Sea). (In contrast, "ppt" is used to denote parts per trillion for freshwater studies of trace elements and organics; however, ppt as used in this section refers to parts per thousand.) TDS is usually expressed in units of milligrams per liter (mg/L). For fresh water, the terms salinity and TDS are often used interchange-ably, and irrigation and drainage engineers and soil scientists typically express salinity in parts per million (ppm). One mg/L is numerically equivalent to one ppm by weight.

Conductivity (or specific conductance [SC]), in the International System of measurements (SI), is expressed in microsiemens per centimeter (μS/cm), although many reports still use the non-SI measurement micromhos per centi-meter (1 μmho/cm = 1 μS/cm). Specific conductance is defined as the conductivity of a conductor 1 centimeter long and 1 square centimeter in cross-sectional area.

An approximate value for TDS can be derived from conductivity results, although the relationship between TDS and conductivity varies on the basis of ionic composition of the salts present in solution. For specific con-ductance less than 5,000 μS/cm at 25°C,

$$TDS = 0.584 \times SC + 22.1$$

where TDS = total dissolved solids in mg/L and SC = specific conductance in μS/cm.
For a specific conductance from 5,000 to 9,000 μS/cm,

$$TDS = 0.682 \times SC - 269$$

Roughly, for most waters, the concentration of dissolved solids in mg/L is in the range of 0.55 to 0.7 times the conductivity in μS/cm (Linsley and Franzini 1979). Another "rule of thumb" for converting conductivity measure-ments to TDS is to multiply SC by 0.64 (U.S. Bureau of Reclamation 1993). According to Hem (1985), the range of the slopes of the regression lines is 0.54–0.96 for dilute waters, and 0.67 is often used to derive TDS values from measured conductivity (pers. comms. from J. Yahnke, U.S. Bureau of Reclamation; and M.K. Saiki, U.S. Fish and Wildlife Service). Values in the upper part of this range (>0.7) tend to have higher sulfate concentrations, which are pertinent to National Irrigation Water

Quality Program work. Many of the sites being investigated are in Cretaceous marine shales, which tend to be high in gypsum ($CaSO_4$).

The conductivity of surface and ground waters varies widely (Hem 1985). In areas where the rainwater is relatively pure and the rocks are resistant to erosion, conductivity may be as low as 50 µS/cm. In other areas, conductivities may exceed 50,000 µS/cm; this is about the same as the conductivity of seawater. Surface waters of enclosed basins (where mineral accumulation occurs in ponded water) may have salinities equal to
or greater than that of seawater.

Summary of Effects

Table 28 summarizes the predicted effects of salinity in the environment, based on the limited information currently available.

Biotic Effects

Salinity is a critical factor influencing the distribution and maintenance of aquatic life in estuaries and other brackish areas. Estuaries are characterized by high densities of a few species, with species richness increasing along the salinity gradient (Hall and Anderson 1995). Nonmarine saline water bodies tend to have fewer species than freshwater bodies, but many of them host large populations of those few species. Examples of such water bodies include Mono Lake and the Salton Sea (Setmire et al. 1993, CH2M Hill 1994), both in California. In the San Joaquin Valley of California, evaporation basins are the disposal receptors for much of the subsurface agri-cultural drainwater. These evaporation basins are highly saline and contain a high concen-tration of nutrients. Like other saline water bodies, evaporation basins have high invertebrate populations and high wildlife use but relatively low species diversity (Parker and Knight 1989, CH2M Hill et al. 1993).

Plants

Maas (1990) listed salinity sensitivities for many types of grasses and forage crops. Some of these are shown in table 29 (expressed in terms of conductivity [µS/cm] of a saturated soil extract [EC_e]). Some of the cultivated grasses shown there also grow wild, so their tolerances could be used as an indicator for naturally occurring grasses. These thresholds represent the maximum soil salinities that do not reduce crop yield below that achieved under nonsaline conditions.

Invertebrates

Studies conducted at the Stillwater Wildlife Management Area (SWMA), Nevada, provide data for evaluating toxic effects of salinity and contaminants in irrigation drainwater effluent. Ingersoll et al. (1992) conducted static acute effluent tests with water collected from the SWMA. The test animals for this study consisted of amphipods (*Hyalella azteca*), daphnia (*Daphnia magna*), and two species of fish. In reconstituted water representative of one of the sample sites, salinity was acutely toxic to amphipods at a concentration of
22 ppt and to daphnia at 8 to 10 ppt. Dwyer et al. (1992) found a similar toxicity level for daphnia.

A study conducted by Galat et al. (1988) produced similar results for salinity effects in benthic invertebrates (table 30). The 96-h LC50 for *H. azteca* was 19.5 ppt, short-term mortality for *Chironomus utahensis* was
100 percent at a salinity of 13.3 ppt, and mortality of *Heterocypris* sp. was 50 percent at a salinity of 18.6 ppt.

Fish

Fish species represented in the Ingersoll et al. (1992) study at SWMA were fathead minnows (*Pimephales promelas*) and striped bass

Table 28.—Summary of comprehensive biotic effects of salinity

[All values are aqueous concentrations in parts per thousand (=g/L)]

Affected organisms	Effect level (ppt)			Reference/Explanation
	No effect	Level of concern	Toxicity threshold	
Plants				
Freshwater marsh grass	—	—	10–12	Pezeshki et al. 1987
Clover, various grasses	—	—	0.9	Maas 1990, salt tolerance threshold, 1.5 dS/m (=1,500 µS/cm)
Invertebrates				
Amphipods	—	—	22	Ingersoll et al. 1992, acute toxicity
Daphnia magna	—	0.3–6	6–10	Level of concern from Dwyer et al. 1992. 6-10 ppt is acute toxicity level from Ingersoll et al. 1992
Hyalella azteca	—	8.0–11	16–19.5	Galat et al. 1988. After 100 days at 8–11 ppt, sample populations were 80–90% less than control group. 16 ppt = 96-h LC10; 19.5 ppt = 96-h LC50
Chironomus utahensis	—	5.5–8.9	13.3	Galat et al. 1988. Cumulative mortality of 30–50% after 17 days at 5.5–8.9 ppt. 13.3 ppt = 96-h LC100
Heterocypris spp.	—	9.0–11	13–18.6	Galat et al. 1988. Approximate 96-h mortality 5% at 9–11 ppt, 7% at 13 ppt, 50% at 18.6 ppt
Fish				
Fathead minnow	—	—	6–10	Ingersoll et al. 1992, acute toxicity
Striped bass	—	—	14–34	Ingersoll et al. 1992; Dwyer et al. 1992, acute toxicity
Striped bass 2-day larvae	—	6	12	Winger and Lasier 1994. Mortality after 6 days: 20% at 6 ppt, 36% at 12 ppt
Birds				
Mottled duck	—	—	9–18	Moorman et al. 1991. "Threshold level" 9 ppt; 100% mortality at 18 ppt
Mallard	9–12	10–15	—	Nystrom and Pehrsson 1988; Swanson et al. 1984
Black duck	—	—	20	Swanson et al. 1984
Peking duck	—	20	—	Nystrom and Pehrsson 1988
Amphibians	—	—	—	—
Mammals	—	—	—	—

Table 29.—Salt tolerance thresholds of herbaceous crops

[Data from Maas (1990)]

Common name	Botanical name	Threshold (µS/cm)	Common name	Botanical name	Threshold (µS/cm)
Alfalfa	*Medicago sativa*	2,000	Orchardgrass	*Dactylis glomerata*	1,500
Barley (forage)	*Hordeum vulgare*	6,000	Ryegrass, perennial	*Lolium perenne*	5,600
Bermudagrass	*Cynodon dactylon*	6,900	Sesbania	*Sesbania exaltata*	2,300
Clover, ladino	*Trifolium repens*	1,500	Sphaerophysa	*Sphaerophysa salsula*	2,200
Clover, Berseem	*Trifolium alexandrinum*	1,500	Sudangrass	*Sorghum sudanense*	2,800
Clover, alsike	*Trifolium hybridum*	1,500	Trefoil, big	*Lotus uliginosus*	2,300
Clover, red	*Trifolium pratense*	1,500	Trefoil, narrowleaf birdsfoot	*Lotus corniculatus tenuifolium*	5,000
Clover, strawberry	*Trifolium fragiferum*	1,500	Vetch, common	*Vicia angustifolia*	3,000
Corn (forage)	*Zea mays*	1,800	Wheat (forage)	*Triticum aestivum*	4,500
Cowpea (forage)	*Vigna unguiculata*	2,500	Wheat, Durum (forage)	*Triticum turgidum*	2,100
Fescue, tall	*Festuca elatior*	3,900	Wheatgrass, fairway crested	*Agropyron cristatum*	7,500
Foxtail, meadow	*Alopecurus pratensis*	1,500	Wheatgrass, standard crested	*Agropyron sibiricum*	3,500
Hardinggrass	*Phalaris tuberosa*	4,600	Wheatgrass, tall	*Agropyron elongatum*	7,500
Lovegrass	*Eragrostis* sp.	2,000	Wildrye, beardless	*Elymus triticoides*	2,700

(*Morone saxatilis*). In their results, the reconstituted water samples from SWMA were acutely toxic to striped bass at a salinity of 22 ppt and to fathead minnows at 8 to 10 ppt. Dwyer et al.'s (1992) results for striped bass were similar. These cited results do not, however, apply to all striped bass. Like salmon, striped bass have both anadromous (seagoing) and landlocked varieties. The marine striped bass readily tolerate salinities of 33–37 ppt (J. Yahnke, U.S. Bureau of Reclamation, pers. comm.).

Nelson and Flickinger (1992) conducted 96-hour acute toxicity tests to determine the salinity tolerance of the Colorado squawfish (*Ptychocheilus lucius*). The tests yielded a 96-hour LC50 of 13.1 ppt using saline water diluted with fresh wellwater. This result is fairly consistent with the salinity tolerances of other freshwater fishes, especially those of the family Cyprinidae (minnows and carps).

According to EPA Ambient Water Quality Criteria (1986), the goldfish (*Carassius auratus*) has a 96-hour LC50 of 16.1 ppt, and the fat-head minnow has a 96-hour LC50 of 11.9 ppt.

In a study by Saiki et al. (1992), juvenile Chinook salmon (*Oncorhynchos tshawytscha*) and striped bass were exposed to serial dilutions of agricultural subsurface drainwater, reconstituted drainwater, and reconstituted seawater. The researchers found that after 28 days of exposure, the survival of chinook salmon averaged 28 percent in the full-strength agricultural subsurface drainwater and was 100 percent in all other

Table 30.—Biological effects of various concentrations of salinity on selected species

Species	Salinity concentration in water (ppt)	Effects/Comments	Reference
Plants			
Freshwater marsh grass	10–12	Toxicity threshold	Pezeshki et al. 1987
Ruppia maritima	46	No growth	McMillan and Moseley 1967
Typha sp.	26	Growth ceased after 9 days	McNaughton 1966
	4.5–12.5	Reduction in height (salinity increased over time)	Shekov 1974
	12	Stunted growth	U.S.Department of Agriculture 1972
Invertebrates			
Amphipods	22	Acute toxicity	Ingersoll et al. 1992
Daphnia magna	0.3–6	Level of concern	Dwyer et al. 1992
	6 - 10	Acute toxicity	Dwyer et al. 1992; Ingersoll et al. 1992
Chironomid	89	LC50	Kokkinn 1986
	13.25	100% mortality at 96h	Galat et al. 1988
Hyalella azteca	19.5	LC50	Galat et al. 1988
Heterocypris sp.	18.57	LC50	
Fish			
Striped bass (*Morone saxatilis*) eggs	18	71% mortality at 72 h	Winger and Lasier 1994
	24	100% mortality at 72 h	
Striped bass (*Morone saxatilis*) 2-day-old larvae	0	100% mortality at 6 d	
	6	20% mortality at 6 d	
	12	36% mortality at 6 d	
	18	75% mortality at 6 d	
	24	100% mortality at 6 d	
Striped bass	14–34	Acute toxicity	Ingersoll et al. 1992, Lal et al. 1977, Dwyer et al. 1992
	15.6	100% mortality at 23 d	Saiki et al. 1992
	14.7	95% mortality at 28 d	
Fathead minnow	6–10	Acutely lethal	Ingersoll et al. 1992, Adelman and Smith 1976
Chinook salmon	20.5	75% mortality at 28 d	Saiki et al. 1992
	14.3–20.5	Reduced growth at 28 d	
Birds			
Mallard	~11	Reduced growth; fatal to young ducklings	Swanson et al. 1984
	8.8–12	100 percent mortality	Mitcham and Wobeser 1988b
	9–12	No effect	Nystrom and Pehrsson 1988, Swanson et al. 1984
	10–15	Level of concern	
	15	100 percent mortality (7-day-old ducklings)	Barnes and Nudds 1991
Mottled duck	9	Threshold level for adverse effects	Moorman et al. 1991
	12	Reduced growth, 10% mortality	
	15	90% mortality	
	18	100% mortality	
Peking duck	20	Level of concern	Nystrom and Pehrsson 1988

water types and dilutions. An important conclusion of this study was that salinity itself may not be as important as the ionic composition of the salts present in the water. The tile drainwater used in the study was toxic because fish were unable to tolerate atypical ratios of major cations and anions constituting the dissolved salts, the high concentrations of sulfate, or both.

Birds

Excess salinity in the drinking water of some birds can adversely affect health and reduce their survival (table 30). During the winter of 1985, a die-off of waterfowl was reported at White Lake, a highly saline lake in Mountrail County, North Dakota. Windingstad et al. (1987) studied this die-off and found that about 150 waterfowl died and another 250 became weak and lethargic, apparently as a result of salt poisoning. Frigid temperatures made fresh water unavailable, forcing the birds to ingest the saline waters, with resultant toxic effects. Although salinity was not measured at the time of the die-off, sodium concentrations of more than 17,000 mg/L were measured in July 1986.

Swanson et al. (1984) found that ducklings on saline lakes in North Dakota were closely associated with fresh inflow from spring seepage or from adjacent wetlands. Young ducklings died when water conductivity was 16,000 µS/cm (about 10.7 ppt salt) and could not tolerate water in prairie lakes that exceeded 20,000 µS/cm (13 ppt) unless fresh water was also available. Duckling growth was significantly reduced on water that measured 17,000 µS/cm (11.3 ppt).

Mitcham and Wobeser (1988a) tested the effects of saline water on ducklings ranging from 1 day old up to 28 days. The tested water contained sodium (up to 3.1 ppt) and magnesium (up to 3.0 ppt) as sulfates added to tap water at concentrations similar to those found in natural saline wetlands of Saskatchewan. Much of the ingested salt was excreted by passage of large volumes of fluid excreta. This effect occurred in birds given water with as little as 0.5 ppt magnesium or 1.0 ppt sodium. The supraorbital salt gland was actively excreting salt within 4 days in ducklings drinking water containing ≥1.5 ppt of sodium. Ducklings drinking water with 3.0 ppt of either ion, or 1.5 ppt of each, grew more slowly than control birds. Ducklings drinking water with 3.0 ppt of either sodium or magnesium had reduced thymus size and bone strength. Ducklings reared on fresh or slightly saline water adapted very poorly to an abrupt change to more saline water (SC = 15,250 µS/cm) at 14 days of age. These birds stopped eating, became inactive, and some died within 3 days; survivors had many tissue and biochemical alterations at 20 days of age. Many of the sublethal effects were subtle and nonspecific manifestations of stress and would be difficult to detect in wild ducklings on saline wetlands.

In a second study by Mitcham and Wobeser (1988b), 1-day-old mallard ducklings received drinking water from 10 naturally saline wetlands in Saskatchewan. Table 31 sum-marizes their results.

A study of the effects of saline water on the growth and survival of mottled ducks in Louisiana indicated a threshold level at 9 ppt. Reduced growth rate and negative effects on body mass and carcass components at 12 ppt suggested a range of tolerable salinity between 9 and 12 ppt (Moorman et al. 1991). Duckling mortality was 100 percent at 18 ppt salinity, 90 percent at 15 ppt, and 10 percent at 12 ppt.

Ducklings appear to be sensitive to increases in salinity; however, a study conducted on American black ducks, mallards, and their hybrids demonstrated that duckling mortality decreased with age and that acclimation to salt water is age-dependent (Barnes and Nudds 1991).

Table 31.—Effects of naturally saline drinking water on 1-day-old mallard ducklings

[From Mitcham and Wobeser (1988b)]

Water conductivity (µS/cm)	Salt concentrations (ppt)		Length of exposure	Effects
	Sodium	Magnesium		
3,750–7,490	0.512–0.911	0.195–0.639	14 d	No apparent effect
4,000	0.821	0.56	28 d	Poor growth in last 2 weeks
7,720	1.98	0.062	14 d	Poor growth
20,000	2.55	1.31	14 d	6 of 10 died. Survivors had poor growth, other effects
21,500	3.86	1.3	14 d	7 of 9 died
35,000	8.79	1.31	60 h	100% mortality
67,000	12.3	5.26	30 h	100% mortality

Interactions

The bioavailability of chemicals—and hence their toxicity—may be altered by physic-ochemical factors such as salinity and tempera-ture (Brecken-Folse et al. 1994). Estuarine organisms may face particular difficulties in coping with toxic substances introduced primarily from industrial and agricultural sources on land because (1) fluctuating salinity may impose a stress of its own and (2) the stress may be com-pounded by tidal variations in toxicant concentrations. In addition, salinity may control the speciation and hence the toxicity of certain heavy metals and other substances (Forbes 1991). Similarly, variations in the salinity of nonmarine wetlands may have the same sort of effect on the toxicity of chemicals that are present.

Hall and Anderson (1995) compared the effects of salinity on the toxicity of various classes of inorganic and organic chemicals. Their results indicate that the toxicity of most metals (e.g., cadmium, chromium, copper, mercury, nickel, zinc) increases with decreasing salinity. One possible explanation for this finding is the greater bioavailability of the free metal ion (toxic form) at lower salinity conditions. Another possibility is that at higher salt concentrations, physiological osmotic effects cause a greater flow of liquid through the kidneys, thus eliminating all salts, including toxic ions, out of the system. Hall and Anderson found no consistent trend for the toxicity of most organic chemicals with salinity. The one exception to this was the class of organophosphate insecticides, which appeared to gain toxicity with increasing salinity.

In a study using grass shrimp (*Palaemonetes* spp.) and sheepshead minnows (*Cyprinodon variegatus*), toxicity decreased as salinity increased for 4-nitrophenol (Brecken-Folse et al. 1994). However, the toxicity of 2,4-dinitrophenol decreased for sheepshead minnows but increased for grass shrimp as salinity increased.

Regulatory Standards

EPA has established no ambient water-quality criteria for salinity or dissolved solids. Secondary maximum contaminant levels for drinking water are 250 mg/L for chlorides, 250 mg/L for sulfates, and 500 mg/L for total dissolved solids (EPA 1995).

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

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Selenium

Description

Selenium (Se) is a semi-metallic trace element which has biochemical properties similar to those of sulfur. The pure element most often appears as lustrous trigonal crystals of gray selenium. Other common forms include a dark-red powder; the glassy, dark-brown vitreous selenium; and dense monoclinic crystals of red selenium, but these are all less stable than the gray variety and tend to convert to it over time. The most common selenium compounds in natural waters are selenious acid (H_2SeO_3) and selenic acid (H_2SeO_4), which correspond, respectively, to the salts selenite (Se^{+4}) and selenate (Se^{+6}). Certain metal and organic selenides (Se^{-2}) are also common in some environments, such as bottom sediments.

Occurrence

Selenium is widely distributed in rocks, soils, water, and living organisms. In the Western United States, it is most common in Upper Cretaceous and Tertiary marine sedimentary rocks (Seiler 1997). Figure 2 shows the distribution of these formations in the Western United States. Many geologic formations are seleniferous and capable of contributing to the mobile forms of selenium in soils. Selenium is highly mobile and biologically available in arid regions having alkaline soils—conditions typical of the Western United States. A number of plants, such as *Astragalus* (loco weed and milkvetch), can concentrate selenium extracted from the soil into a biologically available form, which is toxic to livestock when eaten (Hedlund 1993).

The concentration of selenium in rivers, streams, lakes, and wetland areas is greatly increased by irrigation drainage return flow in certain areas of the West. Upstream from irrigated areas in the Colorado River basin, waters generally have selenium concentra-tions of less than 1 µg/L, but downstream from irrigated areas, the concentration exceeds 30 µg/L in places (mainly backwater areas). Drainage from the Westlands Irrigation Project in California averaged 300 µg Se/L and ranged from 160 to 1,400 µg Se/L. Selenium was further concentrated in the collector drains by evaporation and bio-accumulation. Levels in plants and animals were high enough to kill some aquatic birds and fish and impair reproduction of others. (Hedlund 1993.)

Selenium can also be mobilized or released from the soil by a crop-fallow management system. Saline seeps developed in wheat-fallow areas of the plains from Texas into Canada may have high concentrations of selenium and may contaminate both ground water and surface runoff (Hedlund 1993).

Abnormally high mass-loading of selenium into aquatic environments most typically results from the disposal of coal fly ash, irrigation wastewater, or oil refinery waste-water. Mining of sulfide ores is also a common source of artificially mobilized selenium. In particular, selenium is a common waste product from uranium, bentonite, and coal mining. Soils, surface waters, and ground waters around these mining operations can become contaminated. Concentrations as high as 4,500 mg/kg have been reported in the overburden from the Powder River district in Wyoming. Bentonite mines in Wyoming, Montana, and SouthDakota are additional selenium sources, and it also may be concen-trated in coal deposits and carbonaceous

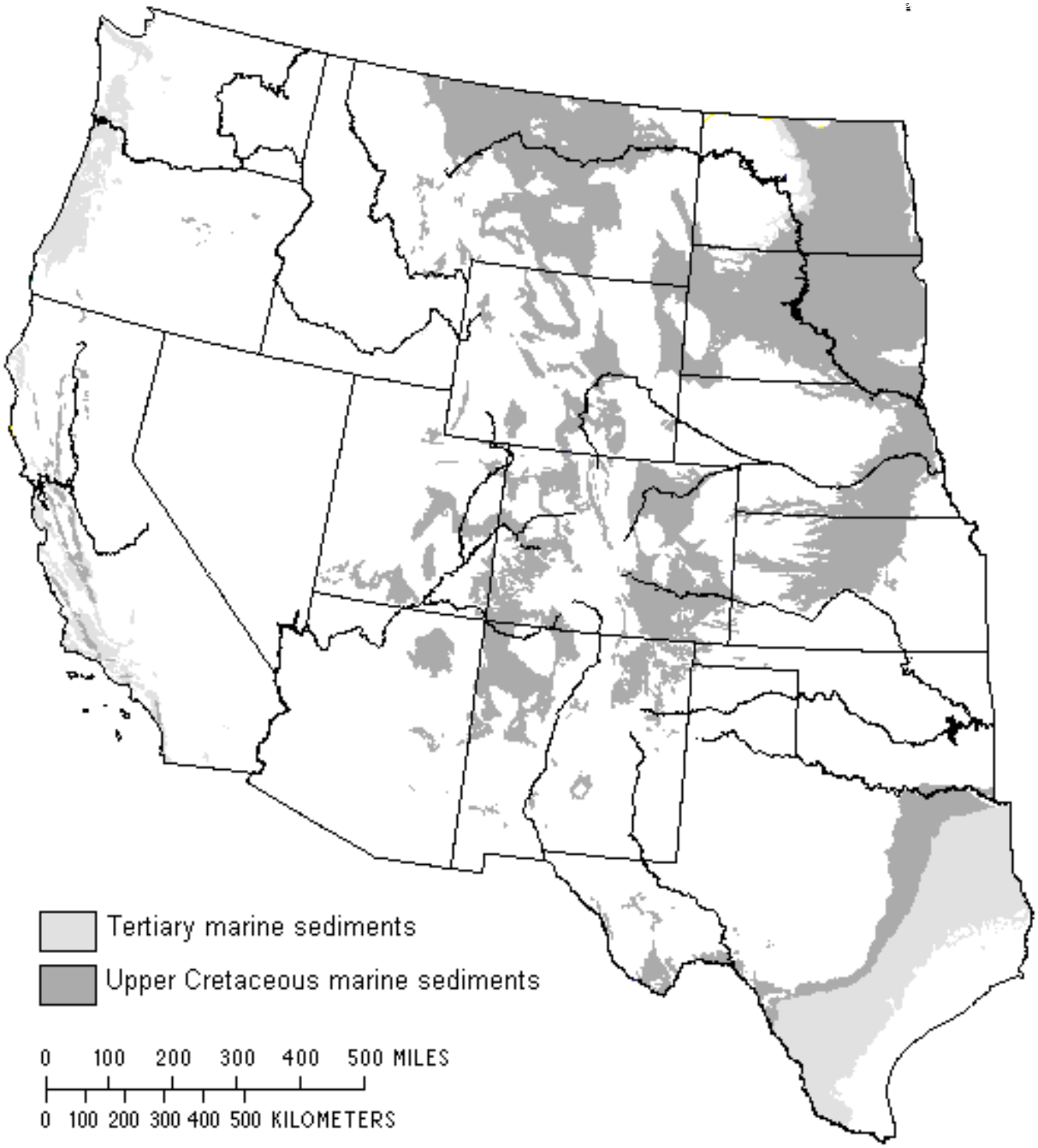

Figure 2. - Distribution of potentially seleniferous bedrock in the Western United States (adapted from Seiler 1997)

shales. Mining operations commonly increase the element's mobility and solubility. (Hedlund 1993.)

Background Concentrations.—Selenium has an average crustal abundance of 0.05 mg/kg and the following approximate background levels in various media:

Medium	Background (mg/kg dw, except as noted)
Freshwater	0.1–0.4 µg/L
Freshwater sediments	0.2–2.0
Plants:	
Freshwater algae	0.1–1.5
Freshwater macrophytes	0.1–2.0
Terrestrial plants	0.01–0.6
Invertebrates:	
Aquatic	0.4–4.5
Terrestrial	0.1–2.5
Fish:	
Liver	2–8
Other tissues	1–4
Reptiles/Amphibians:	
Liver	2.9–3.6
Other tissues	1–3
Birds: Whole body	<2
Muscle	1–3
Eggs	<5
Liver	<10
Feathers	1–4
Whole blood	0.1–0.4 mg/L
Mammals: Whole body	<1–4
Muscle	<1
Liver	1–10
Hair	<1–3
Milk	<0.05 mg/L
Whole blood	0.1–0.5 mg/L

See the separate sections for each of these media, below, for further discussion of these background levels.

Summary of Effects

Based on the known margins of safety between normal and toxic dietary exposures, selenium is more poisonous than either arsenic or mercury (Sorensen 1991). However, selenium is also an essential trace nutrient for animals, and it serves beneficial metabolic functions (Arthur and Beckett 1994). Thus, selenium deficiency as well as toxicity can cause adverse effects for fish and wildlife (Oldfield 1990; CAST 1994). Interestingly, both deficiency and toxicity cause similar effects: e.g., reproductive depression, anemia, weight loss, and immune dysfunction (Koller and Exon 1986). The known effects of selenium exposure to various classes of organisms are summarized in table 32.

One of the most important features of selenium ecotoxicology is the very narrow margin between nutritionally optimal and potentially toxic dietary exposures for vertebrate animals (Wilber 1980; NRC 1989). Nutritionally optimal dietary selenium expo-sure is generally reported as 0.1–0.3 mg/kg. Thresholds for dietary toxicity in animals are generally reported as 2–5 mg Se/kg—only 7 to 50 times the nutritionally optimal levels (Girling 1984; NRC 1989; Sorensen 1991; Eisenberg 1993). Thus, relatively small perturbations in the dietary exposure of vertebrate animals are potentially harmful. According to Spallholz (1994), extensive studies with rats have found the toxicity threshold for dietary selenite to be only 2.5 times above the nutritionally optimal dose. However, only the studies of rats have included controlled lifetime exposures and evaluated such sensitive response variables as longevity.

Table 32.—Summary of comprehensive biotic effects of selenium

Medium	No effect[1]	Level of concern[2]	Toxicity threshold[3]	Comments/Explanation
Water (µg/L, total recoverable Se)[4]	<1	1–2	>2	Peterson and Nebeker (1992)
Sediment (mg/kg dw)	<1	1–4	>4	Van Derveer and Canton (1997), SJVDP (1990), Lemly and Smith (1987)
Diet (mg/kg, dw)	<2	2–3	>3	Lemly (1996a)
Waterbird eggs (mg/kg dw)	<3	3–6	>6	No-effect level from Skorupa and Ohlendorf (1991). Toxicity threshold from Skorupa (1998a)
Fish, whole-body (mg/kg dw):				
Warm-water species	<3	3–4	>4	Lemly (1996a)
Cold-water species	<2	2–4	>4	

[1]Concentrations lower than this value produce no discernible adverse effects on fish or wildlife and are typical of background concentrations in uncontaminated environments.

[2]Concentrations in this range rarely produce discernible adverse effects but are elevated above typical background concentrations.

[3]Concentrations above this value appear to produce adverse effects on some fish and wildlife species.

[4]See the "Water" section of this chapter for a discussion of the difference between total recoverable Se and dissolved Se.

Selenium is much less toxic to most plants and invertebrate animals than to vertebrate animals. Among vertebrates, reproductive toxicity is one of the most sensitive endpoints; however, egg-laying vertebrates such as birds and fish seem to have substantially lower thresholds for reproductive toxicity than placental vertebrates (mammals).

A general ecotoxicological rule of thumb for selenium is that thresholds for adverse effects in vertebrate animals begin at concentrations less than one order of magnitude above normal (Lemly 1985b; Eisenberg 1993; Ohlendorf et al. 1993; Salyi et al. 1993). When environmental, dietary, or tissue concen-trations of selenium are 10 times normal background levels or higher, toxic effects are likely. Immunotoxic effects have been conclusively documented for birds and mammals at tissue concentrations of selenium less than five times normal background (Whiteley 1989; Schamber et al. 1995). How-ever, there are no well-documented case studies of selenium-mediated immunotoxicity and associated consequences among animals in nature.

The threshold of ecotoxicity for selenium is remarkably similar for fish and birds (Lemly 1995, 1996b), the two classes of animals most likely to be adversely affected by contamina-tion from agriculture or industry. With the exception of hepatic tissues, all fish and wildlife tissues normally average about 2 mg Se/kg or less. A concentration of 10–20 mg Se/kg in wildlife tissues or diets is above the threshold of toxicity for sensitive and moderately sensitive taxa, and at the level of 50–100 mg Se/kg, catastrophic impacts are highly likely.

Metabolic stress caused by winter weather can increase the susceptibility of birds (Heinz and

Fitzgerald 1993b), mammals (Ghosh et al. 1993), and fish (Sorensen 1991; Lemly 1993c, 1996b) to selenium poisoning. Toxicity data collected under benign climatic conditions may therefore underestimate sensitivity to selenium poisoning.

Selenium accumulates in and disperses from animal tissues fairly rapidly. Significant changes in tissue selenium status can occur within days, weeks, or months depending on the response criterion of interest and the target tissue being monitored (Wilber 1980; Bennett et al. 1986; USFWS 1990a; Heinz et al. 1990; Heinz and Fitzgerald 1993a; Heinz 1993). Furthermore, the overt symptoms of even near-fatal selenium poisoning in adult birds and mammals can be reversed quickly if the source of selenium exposure is eliminated (Ruta and Haider 1989; Heinz and Fitzgerald 1993b). By contrast, embryonic deformities caused by selenium poisoning are not reversible (Lemly 1993b), nor are some types of tissue damage in adult animals (Sorensen 1991).

Study Approaches

Selenium toxicology has been the subject of an extensive body of scientific literature. More than 5,500 titles were screened for this guidance document. Titles that were most directly relevant to evaluating *in situ* eco-toxicology of selenium were retrieved and reviewed. Finally, the retrieved literature was further screened to produce a "short list" of particularly useful reports for evaluating NIWQP measurements of environmental selenium.

Publications reporting field measures of exposure and response were afforded the greatest weight in formulating the guidelines presented in this chapter. Macrocosm and mesocosm studies were given the next highest priority. Among the remaining experimental studies of captive biota, those that utilized selenomethionine (a good surrogate for natural food-chain selenium) and that tested the effects of dietary exposure were given highest priority. Experimental studies that exposed vertebrate biota (other than eggs or larvae) to contaminated water, but provided no food or only clean food (i.e., standard aquatic toxicity tests), are not transferable to field conditions and therefore were not reviewed for this document (see EPA 1987; Maier et al. 1987; and SJVDP 1990 for reviews of studies of this type). Also, toxicity studies of marine organisms were not reviewed for this document.

Field Cases

To the extent possible, interpretive guidelines should be based either on field data or on experimental data that have been field validated. At least 11 incidents of fish or wildlife poisoning by selenium, studied in the field, have been documented in the technical literature. These incidents occurred in North Carolina, Texas, Colorado, California, Wyoming, Utah, and Sweden. Brief sum-maries of each incident are presented below.

(1) Belews Lake, North Carolina—This power-plant cooling reservoir received return flow from a fly-ash settling basin from 1974 to 1985. The ash-basin effluent contained about 150–200 μg Se/L. For the main reservoir, water-borne selenium was elevated to about 10 μg/L on average. Sediments averaged 14 mg/kg, benthic food-chain fauna averaged 20–50 mg/kg, plankton averaged 30 mg/kg, fish muscle averaged 20–40 mg/kg, and whole-body selenium concentrations averaged 40–125 mg/kg (approximate dry weights). Gonads of female fish contained about 20–170 mg Se/kg. Thus, the selenium concentrations were about 25–100× background in the water, 10–15× background in food-chain flora and fauna, as much as 130× normal in fish whole bodies, and about 5–85× normal in female fish gonads. Of 20 species of fish exposed to these contaminant conditions, 16 were extirpated, 2 had ceased reproducing, 1 was temporarily extirpated,

and 1 was unaffected. Only carp, black bullheads, and mosquitofish were present throughout the study. For most species, teratogenic effects and other overt abnormalities were observed in about 10–70 percent of the sampled fish.

Follow-up sampling of fish in 1992, 7 years after selenium loading to Belews Lake was substantially reduced, found whole-body selenium concentrations averaging 10–20 mg/kg (≈5–15× normal) associated with slightly elevated frequencies of abnormalities (5–10 percent versus a reference range of 1–3 percent). For centrarchids, a strong exposure-response relationship was evident between whole-body selenium and frequency of overt abnormalities. The EC25 was roughly 45 mg/kg whole-body Se (Lemly 1985a,b; 1993b). In an arm of Belews Lake that is semi-isolated from the main reservoir, known as the Highway 158 arm, selenium content averaged 3–4 µg/L in the water (10–20× normal), 0.7–3.0 mg/kg in sediments (1–10× normal), 4–8 mg/kg in benthic invertebrates (2–5× normal), 25–30 mg/kg in fish liver (4–6× normal), and 7–9 mg/kg in fish muscle (2–5× normal) (Sorensen et al. 1984; Cumbie pers. comm. cited in GLSAB 1981). Some fish in the Highway 158 arm showed sublethal toxic effects such as generalized edema and abnormal ovarian tissue damage (Sorensen et al. 1984). Nonetheless, an overtly normal fish fauna persisted (Lemly 1985b).

(2) Hyco Reservoir, North Carolina—This was another powerplant cooling reservoir, which received effluent from two fly-ash ponds that contained about 50–200 µg Se/L in their water. As a result, the waterborne concentra-tion of selenium in Hyco Reservoir was elevated to about 10 µg/L on average. In other media, selenium averaged approxi-mately 3–5 mg/kg (dw) in sediments, 10–30 mg/kg in benthic food-chain fauna, 2–20 mg/kg in plankton, 35–50 mg/kg in fish muscle, and 30–50 mg/kg in gonads of female fish. Compared to local reference populations, fish muscle contained about 40× normal selenium concentrations, and female gonads contained about 15–25× normal. As a consequence of this exposure, adult fish densities declined by 38–75 percent, and larval fish densities declined by 98.6 percent (Woock and Summers 1984; Gillespie and Baumann 1986).

(3) Martin Lake, Texas—This reservoir received discharges during 1978–79 from fly-ash ponds that contained selenium concentrations in excess of 2,000 µg/L. The water in Martin Lake was elevated to about 2.6 µg Se/L on average (≈5 µg/L in the impact area?). Other selenium concentrations (dw) averaged about 5 mg/kg in sediments, 30 mg/kg in fish muscle, and 17 mg/kg in gonads of female sunfish (≈8× the "normal" level of Hamilton and Waddell 1994). This level of exposure was implicated in a series of fish kills soon after Martin Lake received the ash-pond effluent (Garrett and Inman 1984; Lemly 1985a; Sorensen 1988; Texas Parks and Wildlife Department 1990). By 1986, fish muscle tissue was down to 5–10 mg Se/kg, but selenium in red-winged blackbird eggs was still elevated to 11 mg/kg (4× that of a local reference population). Two blackbird foods—grass-hoppers and mayflies—averaged 1.1 and 15 mg Se/kg, respectively. The hatching success of Martin Lake blackbird eggs was less than 50 percent that of a local reference population. Barn swallow eggs at Martin Lake averaged 3.3 mg Se/kg and showed normal hatchability com-pared to a local reference population (King 1988; King et al. 1994).

(4) Sweitzer Lake, Colorado—This lake, also known as Garnet Mesa Reservoir, was built in 1954 for recreational purposes. Sweitzer Lake is situated in an area of naturally seleniferous geological formations. It is unclear, however, how much of the cumulative selenium loading into Sweitzer Lake came from natural inputs and how much was artificial. Initial (1950's) water sampling revealed more than 100 µg Se/L. Biotic selenium contamination reached

as high as about 20 mg/kg (dw) in benthic food-chain fauna and 40 mg/kg in fish livers. This level of exposure caused progressive mortality of stocked game fishes. The Colorado Division of Wildlife decided to stop stocking the lake in 1974 but later did restock it with catfish in 1984. In the late 1980's, water samples from the lake contained about 10–25 µg Se/L. Fish muscle (channel catfish) averaged about 30 mg/kg, and bird eggs averaged about 9 mg Se/kg. At this level of exposure, there was no evidence of successful reproduction among the channel catfish, and the reproductive performance of birds at the lake was unknown; however, large popula-tions of green sunfish and carp, including various age classes, were present (Lemly 1985a; Butler et al. 1991).

(5) Kesterson Reservoir, California—This evaporation and seepage basin for irrigation drainage water in California's Central Valley received water containing about 330 µg Se/L over several years. Kesterson was operated as a series of 12 separate cells that contained 15–350 µg Se/L and averaged about 150 µg/L. Other average selenium contents were about 12 mg/kg in sediments, 20–110 mg/kg in benthic and water-column food-chain fauna, 170 mg/kg in mosquitofish (whole body), and about 10–70 mg/kg in bird eggs. These con-centrations were about 12–130× local reference values in food-chain fauna and 5–35× local reference values in bird eggs. At this level of exposure, it is suspected that several species of fish were extirpated before any systematic field studies began (USBR 1986: 4G-2). The brood size of mosquitofish in the San Luis Drain, the source of Kesterson's drainwater, was only 12.4 fry/brood, versus a local reference value of 25.7 fry/brood. The incidence of stillborn fry was about 20–30 per-cent compared to a local reference value of about 1–3 percent. Mosquitofish are extremely tolerant of selenium exposure.

The mosquitofish from the San Luis Drain contained whole-body concentrations of about 120 mg Se/kg or about 80× local reference levels. About 40 percent of 578 nests of ducks and other waterbirds at Kesterson contained one or more dead or deformed embryos. About 20 percent of all nests contained one or more overtly deformed embryos. Four species of waterbirds (American avocet, black-necked stilt, eared grebe, and American coot) experienced complete reproductive failure. Exposure-response data for black-necked stilt eggs revealed an eggwise threshold for embryo-toxicity in the vicinity of 10 mg/kg. Some adult birds also died, and many of these showed alopecia (loss of feathers), a classic symptom of acute selenium poisoning (Ohlendorf et al. 1986; Zahm 1986; Presser and Ohlendorf 1987; Ohlendorf et al. 1988a; Ohlendorf 1989; SJVDP 1990; Saiki and Ogle 1995).

(6) Tulare Lake Basin, California—About 25 evaporation and seepage ponds for irrigation drainage water in this basin received water containing from <1 to >1,000 µg Se/L. The "ponds" vary in size from 10 to 1,800 acres. Impounded water in these ponds averaged 0.5–1,014 µg Se/L. Sediments averaged 0.1–16 mg Se/kg, benthic and water-column food-chain fauna averaged 1–250 mg/kg, and bird eggs contained about 1–150 mg Se/kg. Except for intermittent introductions of mosquitofish, the Tulare evaporation ponds were never inhabited by fish. Impairment of avian reproduction was documented at about half of the evaporation ponds. Highly elevated rates of embryo teratogenesis (20 percent vs. 0.1 percent background) were documented for ponds with as little as 15 µg/L waterborne Se and 0.9 mg/kg sediment Se. At an individual level of analysis, embryos of black-necked stilts that were exposed *in ovo* to about 55–65 mg Se/kg (≈25× normal) and survived at least 8 days into incubation had about a 50 percent probability of overt teratogenesis. Data at the population level of analysis suggested that the threshold for hatchability depression in stilt eggs (only a portion of which is caused by overt terato-genesis) corresponds to a geometric mean value of >8 mg Se/kg (3–4× normal). Predictive regression equations for the ponds revealed that ponds averaging >2.7 µg Se/L in

water or >2.9 mg Se/kg in the food chain would be sufficiently contaminated for average bioaccumulation of >8 mg Se/kg in eared grebe eggs (the most proficient avian bioaccumulators of selenium). A statistical risk analysis of 354 nests revealed that black-necked stilt hens that had laid a sample egg containing as little as 4.2–9.7 mg Se/kg (the first quartile above background) had nearly a fourfold greater risk than normal of producing an inviable sibling egg. Based on a water-to-egg regression equation for stilts at the Tulare ponds (r=0.901), only 2.6–18 µg Se/L in water would result in 4.2–9.7 mg Se/kg in eggs. More recent analyses of the stilt data revealed that the embryotoxicity threshold occurs at 6 mg Se/kg in eggs and that 3-4 µg Se/L in water is sufficient to create such a concentration in eggs (Skorupa 1998a). Exposure-response data for avian terato-genesis in the Tulare Basin suggest that stilts are not the most sensitive avian species for Se-caused reproductive impairment. Ducks are nearly twice as sensitive as stilts to embryonic selenium exposure (≈50 percent probability of overt teratogenesis at 30 mg Se/kg in duck eggs, compared to 58 mg Se/kg in stilt eggs; Skorupa 1998b), but small sample sizes of duck nests in the Tulare Basin precluded any statistical risk analysis of egg viability (i.e., overall embryotoxicity). Among adult stilts, exposure-dependent loss of body weight was documented; however, no fatal poisoning of adults or alopecia was documented for any species of bird (Fujii 1988; SJVDP 1990; Skorupa and Ohlendorf 1991; CH2M Hill et al. 1993; Ohlendorf et al. 1993; CH2M Hill 1994).

(7) Chevron Oil Company Refinery Near Richmond, California—The refinery discharges process wastewater to a flow-through marsh for pretreatment prior to ultimate discharge into San Francisco Bay. The wastewater effluent contained about 10–30 µg Se/L, and water in the marsh averaged 7.5–17.5 µg Se/L. Sediment data are not available for the marsh. Food-chain organisms contained about 10–45 mg Se/kg, or about 10× normal levels. Randomly sampled eggs of black-necked stilts nesting at the marsh averaged 20–30 mg Se/kg (8–12× normal). About 18 percent of stilt nests contained at least one inviable egg, versus 9 percent in San Joaquin Valley nests confirmed to have had normal background exposure to selenium. That difference was statistically significant. Nonrandomly sampled inviable eggs of stilts, avocets, mallards, and coots contained about 15–60 mg Se/kg. Embryo teratogenesis was docu-mented for mallards, coots, and possibly stilts (CH2M Hill 1994, 1995; Medlin 1994).

(8) Rasmus Lee Lake and Goose Lake, Wyoming— These lakes, within the Kendrick Reclamation Project near Casper, received seleniferous irrigation drainage water. The median dis-solved selenium concentrations were 38 µg/ L in Rasmus Lee Lake and 54 µg/L in Goose Lake. At Rasmus Lee Lake, average selenium contents were about 4–9 mg/kg in sediments, 38 mg/kg in pondweed, 95–160 mg/kg in benthic and water-column food-chain fauna, and 5–85 mg Se/kg in bird eggs. At Goose Lake, the averages were 20–40 mg/kg in sediments, 14 mg/kg in pondweed, 45 mg/kg in benthic and water-column food-chain fauna, and 50–120 mg/kg in bird eggs. Selenium in the food chain at these lakes was elevated to about 3–16× local reference samples and about 2–50× in bird eggs. At these levels of exposure, rates of embryo teratogenesis in geese, avocets, and grebes were 4–23 percent. Nearly 40 percent of 126 monitored nests contained at least one inviable egg, and 7 percent of 120 nests contained at least one deformed embryo. Egg viability (hatchability) ranged from 45.1 to 86.2 percent in all species monitored over all years of study (compared to normal hatchability for these species of >90 percent). Poor post-hatch survivorship of avian hatchlings was also suspected (See et al. 1992).

(9) Ouray National Wildlife Refuge (NWR), Utah—Waterfowl ponds at the refuge received seleniferous irrigation drainage water via seepage of shallow groundwater from upgradient

agricultural fields and from flooding on natural drainages. The shallow ground-water near Ouray NWR showed much spatial variation in selenium content but generally averaged 10–700 µg/L. Water in each of the affected ponds, North and South Roadside Ponds, averaged about 40 µg Se/L. These two ponds are hydrologically connected. Sedi-ment from North Roadside Pond averaged 17 mg Se/kg. Sediment was not sampled from South Roadside Pond. Based on pooled data for both ponds, selenium averaged 10–30 mg/kg in aquatic plants, about 25 mg/kg in benthic and water-column food-chain fauna, about 40–80 mg/kg in fish (whole body), and about 8–90 mg/kg in bird eggs. These concentrations were about 20–60× normal in plants, 10× normal in food-chain fauna, 15–30× normal in fish, and 3–35× normal in bird eggs. At these exposure levels, more than 85 percent of all coot eggs were inviable, and deformed embryos were found in about 10 percent of the nests. Reproductive performance was very poor in small samples of grebe and duck nests, and deformed embryos were found in nests of mallards and redhead ducks. A randomly collected egg from a redhead nest that contained all dead or deformed embryos contained about 20 mg Se/kg (12× normal).

Forty-four wing-clipped game-farm mallards were released to the roadside ponds to monitor the dynamics of tissue selenium and adult survivorship. At the time of release, these birds averaged 2.8 mg Se/kg in their livers. After 1 week, liver selenium averaged 27 mg/kg (10× normal); after 4 weeks the last surviving mallard died, and its liver was found to contain 106 mg Se/kg. Breast muscle averaged about 1 mg Se/kg at release and increased to 37 mg/kg in the last surviving bird. Most of the released mallards had died by the end of the second week, at which time their livers averaged about 40–50 mg Se/kg (≈15× normal) and breast muscle had 4–8 mg Se/kg (3–10× normal). Within 2 weeks, the released mallards had lost about 20 percent of their body mass (Stephens et al. 1992), a finding consistent with selenium-induced cachexia (Albers et al. 1996; Green and Albers 1997).

(10) Imperial Valley of Southern California—Colorado River water averaging 1–2 µg Se/L is diverted into the valley for irrigation, and irrigation wastewater averaging 2–10 µg Se/L (evaporative concentration) is discharged to the Salton Sea (a 230,000-acre terminal sink). Impounded water at the Salton Sea averages about 1.5 µg Se/L (3× normal for saline sinks), and sediments contain 0.2–3.3 mg Se/kg. Algae averaged 0.9 mg Se/kg (3× normal), other food-chain organisms averaged 2–13 mg Se/kg (1–7× normal), and avian eggs averaged 4–7 mg Se/kg (2–4× normal). A monitored population of black-necked stilts, which averaged 6 mg Se/kg in their eggs, exhibited a slight depression (5.6 percent) in reproductive performance. That degree of impairment was consistent with known exposure-response curves for stilts in nature and has tentatively been attributed to selenium toxicity. At such a small effect level, however, larger sample sizes of monitored nests will ultimately be required to conclusively evaluate preliminary findings (Setmire et al. 1990; Westcot et al. 1990a; Setmire et al. 1993; Bennett 1997).

Because stilt eggs collected during Bennett's (1997) study came from numerous locations, only a few of which constituted Salton Sea "shoreline" sites, it is not precisely known what concentration of selenium in water can be associated with this case study. It is, however, highly probable that the birds in this study were predominantly using wetlands with selenium concentrations in water in the range of 2-10 µg/L.

(11) Selenite-Treated Lakes in Sweden—Eleven lakes were treated with selenite in an attempt to mitigate high levels of mercury in edible fish (Lindqvist et al. 1991; Paulsson and Lundbergh 1991). In fact, the selenite was highly success-ful in lowering the bioaccu-mulation of mercury in the fish, but it also had some detrimental effects of its own (Lindqvist et al. 1991; Meili 1996). Treatments consisted of a leachable

rubber matrix containing sodium selenite suspended in a sack 1–2 m below the lake surface for 2 years. The selenium-free rubber skeletons, remaining after continuous leaching of sodium selenite, were removed (and replaced?) at intervals of several months. During the first year of treatments (beginning in September 1987), the doses were calibrated for a target lake concentration of 3–5 μg Se/L (lakes initially contained about 0.1 μg Se/L). On average, the target concentration was achieved at most of the lakes, although selenium levels as high as 25–35 μg/L were measured within 100 m of the leach sacks. Four lakes never exceeded about 2.6 μg/L average waterborne Se. Because mitigation of mercury residues was as pronounced in the four lower selenium lakes as in the target concentration (3–5 μg/L) lakes, the dosing was adjusted in the second year of treatment for a target lake concen-tration of 1–2 μg Se/L.

Prior to treatment, pike muscle concentrations of selenium averaged 0.7–2.4 mg/kg (1.3 mg/kg grand mean) in the 11 lakes. After the first year of treatment, muscle concentrations averaged 0.9–2.3 mg Se/kg (1.6 mg/kg grand mean), and after 2 years treatment, they averaged 2.8–7.4 mg Se/kg (4.6 mg/kg grand mean). There was no evidence of catastrophic declines of pike populations in any of the lakes. Muscle concentrations of selenium in perch fry averaged 0.8–2.0 mg/kg prior to treatment and 6–36 mg/kg after 1 year. By the end of the second year of treatment, researchers were unable to find any perch fry in four of the lakes and had a substantially reduced catch from a fifth lake. Among these five lakes, concentrations in perch muscle had averaged 6.9–36 mg Se/kg (23 mg/kg grand mean) at the 1-year sampling point; by comparison, among the other six lakes, concentrations in perch muscle had averaged only 6–18 mg Se/kg (12 mg/kg grand mean). At the end of 2 years, perch muscle con-centrations in the six lakes that still had reproductively viable perch populations averaged 6.9–26 mg Se/kg (15 mg/kg grand mean).

Paulsson and Lundbergh (1991, p. 837) concluded that, "There seems to be a dependence between the selenium concentration in fish tissue and a maximum concentration in lake water being less tank [sic] 2 μg/L." Although the language of the report was confusing, the authors seemed to be suggesting that 2 μg/L waterborne Se was a threshold point for avoidance of excessive tissue selenium in perch. Lindqvist et al. (1991, p. 214) more clearly stated, "It is important not to dose so that Se concentrations in water rise above about 1 to 2 μg Se/L." They also concluded that the observed reproductive failures in perch were due to ". . . the selenium treatment and not to any of the other factors . . .." More recently, Meili (1996), as well as Nuutinen and Kukkonen (1998), concluded that the selenium treatments caused adverse effects on fish populations. For example, Meili (1996) noted the correlation between selenium dosing, elevated tissue selenium, and disappearance of perch fry and concluded, ". . . high Se levels can be linked to high doses, and furthermore to fatal ecological consequences. The results suggest that a selenium concentration of only 3 μg/L can seriously damage fish populations."

Table 33 represents a collection of the *minimum* estimates for real-world (*in situ) toxic exposures* that have been documented for natural populations of fishes and birds. These values do not necessarily correspond with true threshold points (e.g., EC10's) for toxicity because, in some cases, exposure levels are clearly above threshold regions. They do, however, provide field-validated ceilings for the exposure intervals within which true threshold points occur. Over time, the pro-gressive accumulation of data from field cases has lowered these ceilings so that most of the values are now only 5–10× normal back-ground concentrations for selenium. Data from field cases also reveal that selenium exposures in the range of 30–50× normal levels are almost certain to cause widespread *severe* adverse biological effects.

Table 33.—Summary from field cases of minimum selenium contamination having adverse effects on natural fish and wildlife populations

Matrix	Minimum toxic concentrations	Response variable	Study site
Water, µg/L	2–10	Avian reproduction	Salton Sea, CA
	2.0	Fish reproduction	Sweden (five different lakes)
	2.6–5	Fish population collapse	Martin Lake, TX
	2.6–18	Avian reproduction	Tulare Basin, CA
	3–4	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	7.5–17.5	Avian reproduction	Chevron Marsh, CA
Sediment, mg/kg dw	0.9	Avian reproduction	Tulare Basin, CA
	1–3	Avian reproduction	Salton Sea, CA
	<3	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	3–5	Fish survival and reproduction	Hyco Res., NC
Food chain fauna, mg/kg dw	2.9	Avian reproduction	Tulare Basin, CA
	4–8	Fish sublethal effects	Belews Lake, NC, Hwy 158 Arm
	3.1 (pileworms)	Avian reproduction	Salton Sea, CA
Fish muscle, mg/kg dw	7–9	Sublethal effects	Belews Lake, NC, Hwy 158 Arm
	<(10–20)	Teratogenesis	Belews Lake, NC
	6.9–36 (means), 23 grand mean	Reproductive failure	Sweden (five different lakes)
Fish whole body, mg/kg dw	10–20	Teratogenesis	Belews Lake, NC
Fish gonads, mg/kg dw	17	Population collapse	Martin Lake, TX
Fish liver, mg/kg dw	25–30	Sublethal effects	Belews Lake, NC, Hwy 158 Arm
Bird liver, mg/kg dw	40–50	Adult mortality	Ouray NWR, UT
Bird muscle, mg/kg dw	4–8	Adult mortality	Ouray NWR, UT
Bird egg, mg/kg dw	4.2–9.7	Hatchability	Tulare Basin, CA
	6 (mean)	Hatchability	Salton Sea, CA
	10	Hatchability	Kesterson, CA
	11 (mean)	Hatchability	Martin Lake, TX

Abiotic Factors Affecting Bioavailability

Water

Selenium commonly occurs as a mixture of several chemical species in natural waters, although two inorganic chemical species, selenite and selenate, are usually the predominant forms (Masscheleyn and Patrick 1993).

Normal background concentrations of selenium in uncontaminated freshwater ecosystems have been estimated (in µg/L) as 0.25 (Wilber 1980), 0.1–0.3 (Lemly 1985b), 0.2 (Lillebo et al. 1988), and 0.1–0.4 (average <0.2, Maier and Knight 1994). Even in California, a State heavily influenced by agricultural mobili-zation of selenium, the median concentration in 226 streams was 0.4 µg/L (Westcot et al. 1990b). A survey of inland saline lakes in Oregon, California, Nevada, and Utah yielded

a geometric mean concentration for selenium of 0.6 µg/L (Westcot et al. 1990a). Inland saline lakes of the western United States are subject to pronounced evaporative concen-tration of dissolved constituents and thus are likely to have the highest natural concentra-tions of selenium. Behra et al. (1993) considered 1 µg/L selenium in running waters in Switzerland to be "higher than concentrations in moderately polluted waters."

An important factor confounding interpre-tation of field data for waterborne selenium is the differential partitioning of selenium mass loads between the water column and other compartments of an aquatic ecosystem. Partitioning ratios can be strongly influenced by the overall biotic productivity of a water body. In highly productive waters, less dissolved selenium is left in the water column even though food-chain exposure of fish and wildlife may be substantial. Therefore, low waterborne selenium concentrations can indicate either low mass loading (low risk) or high biotic uptake (high risk). This inter-pretive problem can be partially ameliorated by measuring total recoverable selenium (i.e., unfiltered samples) rather than dissolved selenium (filtered samples) (Skorupa and Ohlendorf 1991). Total recoverable selenium includes suspended detrital particulate matter, a function of biotic uptake, and thus more accurately reflects the total mass load of selenium fluxing through a water column.

Estimates of normal *background* concentrations for waterborne selenium are unlikely to differ significantly regardless of whether one measures dissolved or total recoverable selenium. For contaminated waters, however, the differences between these two measures increase with the degree of eutrophication. For eutrophic, shallow waters, such as evaporation ponds, the differences can be pronounced. For one evaporation pond in California, a split sample yielded 7 µg/L dissolved Se but 25 µg/L total recoverable Se (Fujii 1988). The higher value was much more consistent with the >20 mg Se/kg found in bird eggs at the pond (Skorupa and Ohlendorf 1991). Threshold values presented below based on toxicity tests or drinking-water exposure refer to dissolved selenium, whereas those based on field sampling for bioaccumu-lative risk refer to total recoverable selenium; these two types of values should not be equated to each other. Many of the total recoverable selenium values would be lower if expressed on a dissolved basis (e.g., Peterson and Nebeker 1992). To assess biotic risk for selenium toxicity, unfiltered samples of water should be analyzed for both particulate and dissolved selenium (ERG 1998); this is referred to as "total recoverable" selenium in this document and is roughly equivalent to EPA's "acid soluble" selenium.

Waterborne selenium, ***per se,*** is not very toxic to fish and wildlife. When water is the only exposure route (e.g., standard aquatic toxicity test), toxic thresholds for selenium are generally >1,000 µg/L (≈10,000× normal) for adult fish. SJVDP (1990), Maier et al. (1987), and EPA (1987) provide good reviews of aquatic toxicity test results for fish and wildlife populations. Chronic toxicity in experimental animals and livestock has been observed when drinking water exceeds 2,000 µg Se/L (NRC 1980). Drinking water containing 2,200 µg/L selenomethionine appeared to suppress certain aspects of the mallard immune response (Fairbrother and Fowles 1990). A family of Ute Indians in Colorado suffered hair loss, nausea, and fatigue after drinking well water that contained 9,000 µg Se/L (Anonymous 1962). Eggs and larvae of both fish and amphibians may be the most sensitive vertebrate life stages to waterborne selenium *per se*. Toxicity testing revealed that the LC50 for eggs of the narrow-mouthed toad was only 90 µg Se/L. Rainbow trout sac fry are adversely affected by about 50–100 µg/L waterborne Se (Birge et al. 1979). Hamilton and Wiedmeyer (1990) reported that 70 µg/L waterborne Se (inorganic mixture)

was sufficient to reduce the 90-day survival of larval chinook salmon (exposed as eyed eggs and as larvae).

Much lower concentrations of selenium in water can be bioaccumulated to toxic levels in fish and wildlife via dietary exposure to the aquatic food chain. Field cases of selenium poisoning in fish and birds have been docu-mented for waters averaging as little as 1–10 µg Se/L (see "Field Cases" section). NIWQP data from 23 study areas in 13 western States illustrate the effect of bioaccumulation on bird eggs. Out of 10 study areas where the 75th percentile value for selenium in surface waters was 2 µg/L or less, none (0 percent) had a 75th percentile value for selenium in bird eggs that exceeded the embryotoxic threshold. Out of nine study areas where the 75th percentile for surface waters was 3–10 µg Se/L, three (33 percent) had 75th percentile values for bird eggs above the embryotoxic threshold. Of the four study areas where the 75th percentile for surface waters was >10 µg Se/L, all (100 percent) had 75th percentile values for bird eggs above the embryotoxic threshold (Seiler and Skorupa 1995).

Bluegill fish residing for 319 days in outdoor experimental streams supplemented with 2.5 µg Se/L produced larvae with elevated frequencies of edema, lordosis, and hemorrhaging (Hermanutz et al. 1990). With reference to larval edema, the 2.5-µg/L concentration was about the EC10, and a 10-µg/L treatment was about the EC95. Seven other recent reports that estimate waterborne thresholds for food-chain-mediated toxicity to fish and wildlife are summarized in Maier and Knight (1994). All the reports conclude that the threshold is about 3 µg/L or less, even though no two of them had used the same combination of field data, clinical data, and predictive modeling.

In California, human health advisories have been issued for localities where edible tissues in fish or wildlife contained >2.0 mg Se/kg on a wet weight basis (Fan et al. 1988). Those advisories recommended zero consumption of such tissues by pregnant woman and children (<15 years old). In 1975, Kaiser et al. (1979) collected trout that had been stocked 2 years earlier in a Wyoming lake containing about 2 µg/L waterborne Se. The fillets (with skin) that Kaiser et al. (1979) harvested contained about 2 mg Se/kg (wet weight).

The speciation of waterborne selenium can substantially affect the potential for bioaccumulation in fish and wildlife tissues, at least at relatively high waterborne concentrations. Waterborne selenite (coal fly-ash effluent and

Summary: Effects of selenium in water

Interpretive guidance	Waterborne selenium concentration (µg/L)
True background, freshwater environments	0.1–0.4 (typically <0.2)
Approximate background, California streams (maximum probably not natural)	<0.2–73 (median = 0.4)
Approximate background, Western inland saline lakes (maximum probably not natural)	<0.2–490 (geometric mean = 0.6)
Validated LOAEL's for fish and wildlife via bioaccumulation	1–3
10× normal background averages	<2–6
Toxicity-test LOAEL for fish and amphibian eggs/larvae (waterborne exposure only)	50–100
Experimental LOAEL for drinking water (mallards)	2,000 (seleno-methionine)
Validated LOAEL for drinking water (humans)	9,000 (inorganic)

oil refinery wastewater) is more readily bio-accumulated than waterborne selenate (irrigation wastewater) (e.g., Besser et al. 1993). For example, in California, oil refinery wastewater containing 10–30 µg Se/L produced the same tissue concentrations of selenium as irrigation wastewater containing 330 µg Se/L. Both sources of water produced water boatmen (a type of insect) averaging about 20 mg Se/kg (10× normal) and black-necked stilt eggs averaging about 25 mg Se/kg (10× normal) (SJVDP 1990; CH2M Hill 1994, 1995). Thresholds for toxicity, however, have been observed in the 1–3 µg/L range for both selenate- and selenite-dominated waters. Possibly, at these lower concentrations, selenate reduces readily to the more rapidly metabolized selenite, but higher concentra-tions of selenate may overwhelm the reduction pathways.

Bottom Sediment

Currently, there is little empirical basis for assessing fish and wildlife risk as a function of sediment concentrations of selenium. Only one study has matched sediment concentra-tions of selenium with concentrations in a benthic invertebrate from the same sediment— an obvious first step for developing empirically based risk thresholds (ERG 1998). One post hoc comparison of means for unmatched samples showed a poor correlation between selenium concentrations in sediments and benthic invertebrates
(Van Derveer 1995).

Sediments present formidable methodological and statistical obstacles for data interpretation. Methodologically, the depth to which a sediment sample is collected will strongly influence the results. Even when sampling depth is standardized between studies, results will vary depending on whether the analysis is performed on whole-bed samples or some size-fraction subsample (particle fractions <0.062 mm and <2.0 mm are frequently used). Precisely which sampling method a particular study employed is not always clear in the literature (SJVDP 1990). Furthermore, there is an immense amount of spatial variability in measures of sediment selenium. Samples collected only a few meters apart can yield substantially different results (e.g., Chilcott et al. 1990; Setmire et al. 1993; Wu et al. 1995). Thus, in comparison to water or biotic tissues, many more samples of sediment are required to adequately characterize selenium content. This statistical obstacle requires careful consideration when designing a plan for sediment sampling. Rarely will a simple random sampling design be adequate.

Because of the high variance and generally inadequate sampling designs of many existing studies, the maximum sediment values reported for selenium may have more inter-pretive value than the means. As a general rule, anytime the maximum selenium con-centration in sediments exceeds 5 mg/kg, further investigation is strongly warranted (GLSAB 1981). To the extent possible, the guidelines provided here are based on samples no deeper than the upper 3 inches of sediment and on chemical analyses of whole-bed samples.

In a selenium-deficient landscape (Finland), sediments of lakes usually contained <0.1 mg Se/kg, typically 0.04–0.08 mg/kg (Makela et al. 1995). The average concentrations of selenium for sediments in selenium-normal environ-ments are usually <1.0 mg/kg (Maier and Knight 1994). The 90th percentile value for freshwater sediments in Texas was 1.9 mg/kg (Davis 1987), but the proportion of contami-nated sites included in this statewide survey is unknown. SJVDP (1990) estimated normal background values to average as much as about 0.5 mg Se/kg in the sediments of a naturally seleniferous region of California. Martin and Hartman (1984) reported mean sediment concentrations for selenium of
0.89 and 0.52 mg/kg in pothole and riverine wetlands located primarily in North and South Dakota (two relatively seleniferous States). At 25 U.S. Department of the Interior study sites in the Western United States, median selenium

concentrations were 0.5 and 0.3 mg/kg, respectively, in fine and coarse sediment fractions (Presser 1995).

Based on a review of 27 eclectic studies, Van Derveer and Canton (1997) concluded that sedimentary selenium is a reliable predictor of adverse biological effects and that a preliminary toxic threshold existed at about 2.5 mg Se/kg (the 10th percentile for effects). They also noted that, in the literature they reviewed, adverse effects were always observed at selenium concentrations greater than 4.0 mg/ kg in sediments. For a set of 13 independent evaporation ponds in the Tulare Lake Basin of California, Skorupa et al. (unpub. data) found that mean concentrations of selenium in pond sediments correlated reasonably well with mean concentrations of selenium in the eggs of black-necked stilts nesting at the ponds (r =0.777, N=25, p<0.01). All ponds that averaged ≥1 mg Se/kg in sediments yielded stilt eggs averaging >6 mg Se/kg (the embryo-toxicity threshold). All ponds that averaged <0.4 mg Se/kg in sediments yielded stilt eggs averaging <6 mg Se/kg. Based on the relationship between sediments and stilt eggs, eggs would be expected to exceed an average of 20 mg Se/kg (≈85 percent risk level for avian populations, i.e., high risk) when sediments exceeded 2.7 mg Se/kg. A survey of fish in California's San Joaquin River system found elevated concentrations of whole-body selenium (>4 mg/kg) in most mosquitofish at sites containing 0.2–1.9 mg Se/kg in sediments. At sites having <0.13 mg Se/kg in sediments, selenium concentrations in mosquitofish were generally at background levels (<2 mg/kg) (USFWS 1990b).

Summary: Effects of selenium in sediment

Interpretive guidance	Sediment Se concentration (mg/kg)
Selenium-deficient environments	<0.1
Approximate background, Se-normal freshwater environments	0.2–2.0 (typically <1.0)
Approximate background, Texas freshwater environments	<1.9 (90th percentile)
Maximum zero-response (NOAEL) boundary for birds nesting at shallow terminal ponds (population basis)	0.4
Minimum total-response (EC100 boundary for birds nesting at shallow terminal ponds (population basis)	1.0
EC10 for fish and birds in a variety of freshwater aquatic systems (population basis)	2.5
EC100 for fish and birds in a variety of freshwater aquatic systems (population basis)	>4.0
10× normal background averages	3–5
Various regulatory clean-up criteria for soils	3–10

Soil

The presence of selenium in geologic formations does not mean it is present in toxic amounts in the soils derived from these strata. The solubility of selenium in soils depends on pH, moisture, oxidation-reduction conditions, and the degree of aeration. In areas where annual precipitation exceeds 20 inches and there is deep percolation, selenium is slowly leached and does not become concentrated in the soil. However, ground water in such areas may be contaminated from leached selenium. Where rainfall is less than 20 inches and where the soil is alkaline and generally well aerated (as in much of the Western United States), selenium in the soil may be mobilized in its oxidized, readily soluble, selenate form and may be concentrated near the surface (Hedlund 1993).

The amount of selenium in the soil is not a reliable indicator of how much is available to plants or how much can be leached by deep percolation. In moist, acidic, and reducing soils, selenium is present as insoluble selenides, elemental selenium, or insoluble pyritic selenides and not available to plants. Under dry, alkaline conditions in seleniferous soils, microorganisms produce selenates and organic selenium complexes which are very soluble. Excess precipitation or irrigation water draining these soils would leach soluble selenites and selenates and also carry dissolved and suspended organic forms of selenium (Hedlund 1993).

Various regulatory clean-up criteria for selenium in soils typically range from about 3 to 10 mg/kg (Beyer 1990).

Biotic Effects

Interpretation of field data for biota can be confounded by a sampling bias that favors "survivors." Most biological sampling techniques are designed to sample live biota. In contaminated environments, live biota represent "survivors" and are potentially biased subsets of the study populations with regard to selenium exposure and/or sensitivity.

Plants

Selenium-Concentrating Vegetation.—Different plant species have widely varying abilities to take selenium from the soil, accumulate it, and tolerate it. Common types of selenium-concentrating vegetation include *Astragalus* (loco weed and milkvetch—24 species), *Machaeranthera* (thistle), *Haplopappus* (goldenweed), and *Stanleya* (mustard). These plant species have an extraordinary ability to accumulate selenium and can achieve selenium concentrations of hundreds or even thousands of milligrams per kilogram, dry weight.

Primary producers are the foundation for most food chains supporting fish and wildlife populations. In aquatic ecosystems, algae serve as the primary source of energy assimilation and as the base of most aquatic food chains (Ogle et al. 1988). Aquatic macrophytes are important in chemical cycling and as a major input source for detrital food chains.

Filamentous algae in California's San Joaquin River system contained 0.1–0.4 mg Se/kg at sites with <1.0 µg/L waterborne Se (USFWS 1990b; Saiki et al. 1993). Algae in an uncon-taminated San Joaquin Valley freshwater marsh averaged <0.5 mg Se/kg (Saiki and Lowe 1987; Schuler et al. 1990). Background concentrations of selenium in aquatic macro-phytes usually average <1.5 mg/kg (Maier and Knight 1994). Aquatic macrophytes in an uncontaminated San Joaquin Valley freshwater marsh averaged <1.0 mg Se/kg (Saiki and Lowe 1987; Hothem and Ohlendorf 1989; Schuler et al. 1990). Terrestrial plants on nonseleniferous soils usually average <0.25 mg Se/kg (Girling 1984). The two dominant herbaceous plants collected from a normal-selenium (0.25 mg Se/kg soil) reference site in the San Joaquin Valley averaged 0.3 and 1.4 mg Se/kg (Wu et al. 1995). On seleniferous soils, non-accumulator plants may contain 1–200 mg Se/kg, and selenium-accumulator plants contain even higher concentrations (Girling 1984).

No studies in the literature report selenium toxicity thresholds for plants based on exposure in nature (i.e., based on field data). In standard toxicity tests, sublethal effects of selenium exposure are initially observed for green algae at waterborne concentrations of 10 µg/L (selenate) and 75 µg/L (selenite) (Vocke et al. 1980; Foe and Knight 1986). Growth of the green alga *Selenastrum capricornutum* was significantly reduced at tissue concentrations of 4 mg Se/kg (Williams et al. 1994). Bioaccumulation of selenate (but not selenite or selenomethionine) by algae is strongly influenced by waterborne sulfate concentrations (e.g., Kiffney and Knight 1990; Williams et al. 1994). The 10-µg/L threshold for sublethal effects of waterborne selenate occurred in both high-

sulfate and low-sulfate waters**:** algal tissue accumulated 4 mg Se/kg in high-sulfate waters and 17 mg Se/kg in low-sulfate waters (Williams et al. 1994). Blue-green algae are generally less sensitive than green algae to selenium exposure (Maier et al. 1987; Kiffney and Knight 1990), and even among green algae there are extreme species differences in sensitivity. Sublethal thresholds in green algae have ranged from 10 to 300 µg/L waterborne Se (Vocke et al. 1980; EPA 1987). Toxicity profiles for aquatic macrophytes are not known, but the sublethal toxicity thresholds for aqua-cultured lettuce were 200 µg/L waterborne selenate and 3,000 µg/L waterborne selenite. For both selenite and selenate exposure, the threshold tissue concentration for lethal effects was 800 mg Se/kg (Berry and Savage 1986). Apparently, irrigation water with ≤50 µg/L total Se is considered by agronomists to be protective of all crop plants (e.g., Eisler 1985).

The quality of habitat for fish and wildlife is closely linked to particular plant communities. Therefore, selenium contamination could impact fish and wildlife populations indirectly if plant communities are altered by its toxic effects. However, there are no documented field cases of this type of indirect effect on fish and wildlife populations. Additionally, because of the absence of threshold data for ecologically important endpoints in nature, no regulatory values have been established for aquatic toxicity of selenium to plants (EPA 1987). For all practical purposes regarding protection of fish and wildlife populations, the direct toxic effects of consuming selenium-contaminated plants are apparently more important than indirect ecological effects from changes in plant communities. Fish and wildlife risk thresholds for dietary exposure to selenium are addressed in the summaries for vertebrate animals presented below.

Summary: Effects of selenium on plants

Interpretive guidance	Plant Se concentration (mg/kg)
Background, freshwater algae	0.1–1.5 (typically <0.5)
Background, freshwater macrophytes	0.1–2.0 (typically <1.5)
Background, terrestrial plants on nonseleniferous soil	<0.01–0.6 (typically <0.25)
Experimental LOAEL for sublethal effects (growth), algal tissue	4.0
Experimental LOAEL for sublethal effects (growth), macrophyte (lettuce) tissue	250
Experimental LOAEL for lethal effects, macrophyte (lettuce) tissue	800
	Waterborne Se exposure (µg/L)
Toxicity-test LOAEL for sub-lethal effects on green algae	10–300 selenate 75 selenite
Toxicity-test LOAEL for sub-lethal effects on blue-green algae	100 seleno-methionine 3,000 selenate 3,000 selenite
Toxicity-test LOAEL for sublethal effects on a macrophyte (lettuce)	200 selenate 3,000 selenite
Irrigation water standard to protect crop plants	≤50 total

Invertebrates

Invertebrate populations are important sources of food for most species of fish and wildlife. Many species of fish and wildlife require high-protein diets for optimal reproduction, and the invertebrate component of the diet is often the

principal source of protein. Consequently, selenium-induced alterations of invertebrate density or community structure could have indirect ecological impacts on fish and wildlife populations.

For aquatic invertebrates, Maier and Knight (1994) report a range of 0.5–2.0 mg Se/kg as the national background concentrations. In an Se-normal aquatic ecosystem in Colorado, Birkner (1978) reported average invertebrate concentrations of 2.3–4.2 mg Se/kg. At an uncontaminated San Joaquin Valley wetland, invertebrates averaged 0.9–3.0 mg/kg (Saiki and Lowe 1987; Hothem and Ohlendorf 1989; Schuler et al. 1990). Small experimental freshwater ponds serving as control macro-cosms (waterborne Se <1.0 µg/L) contained invertebrate taxa with selenium concentra-tions averaging 1.4–3.8 mg/kg (Crane et al. 1992). In the San Joaquin River system, midges and amphipods averaged 0.4–1.5 mg Se/kg, and crayfish averaged 0.5–0.9 mg Se/kg at sites with ≤1.0 µg/L waterborne Se (Saiki et al. 1993). At Se-normal sites of the lower Colorado River system, crayfish contained 0.6–2.5 mg Se/kg and usually averaged ≤1.5 mg/kg (Welsh and Maughan 1994).

For terrestrial invertebrates (grasshoppers and mantids) at two reference sites near the former Kesterson Reservoir, Wu et al. (1995) reported average concentrations of <1.0 mg Se/kg. Grasshoppers and beetles collected at ref-erence agroforestry plantations in the San Joaquin Valley contained 1.3–2.5 mg Se/kg (SJVDP 1990).

No studies were found in the literature that report selenium toxicity thresholds for invertebrates based on exposure in nature (i.e., based on field data). It has been established that tissue concentrations of selenium in field-collected aquatic invertebrates are strongly related to waterborne concentrations of selenium (Birkner 1978; Wilber 1980; Lillebo et al. 1988). Crayfish caged in the ash-pit drain of a Wisconsin power plant bioaccumulated 30 mg Se/kg in the hepato-pancreas and had significantly altered respiration rates; the ash pit effluent also had high concentrations of chromium, iron, and zinc (Magnuson et al. 1980). No major effects on benthic macroinvertebrate communities were detected in experimental freshwater ponds treated with 2, 10, and 25 µg/L inorganic Se (60:40 ratio selenate to selenite) (Crane et al. 1992). Abundance data for midge larvae, however, are suggestive enough to warrant further study at doses above 2 µg/L waterborne Se. In standard laboratory toxicity tests, 4 µg/L waterborne selenomethionine was acutely toxic to an amphipod. Many factors influence toxicity-test results, including water chemistry, species tested, and life-stage tested, but the lowest waterborne thresholds for acute toxicity are approximately 200 µg/L selenite and 500 µg/L selenate (EPA 1987; Maier et al. 1987; Ingersoll et al. 1990). Lowest thresholds for chronic toxicity occur at approximately 25–100 µg/L for selenite or selenate and perhaps at <0.5 µg/L waterborne selenomethionine (Johnston 1987; EPA 1987; Boyum and Brooks 1988; Ingersoll et al. 1990).

Experimental studies of dietary toxicity are rare. Amphipods showed no adverse effects from dietary exposures to algae containing as much as 300 mg Se/kg (Foe and Knight 1986). Dietary exposure of larval midges to algae containing ≥2.1 mg Se/kg significantly inhibited growth. The inhibited larvae contained ≥2.5 mg Se/kg in their tissues (Malchow et al. 1995). Alaimo et al. (1994) exposed midge larvae to diets based on naturally contaminated widgeon grass (*Ruppia*) detrital substrates from evaporation ponds in the Tulare Basin, California. Alaimo et al.'s 14-day egg-to-prepupation feeding study produced results very similar to those of Malchow et al. (1995). Detrital substrates containing as little as about 2 mg Se/kg significantly inhibited the growth of midge larvae even though the associated selenium concentrations in larval tissue were in some cases <4 mg/kg (Alaimo et al. 1994). Tissue concentrations of 15 and 32 mg Se/kg in

amphipods were associated with reduced growth and reproduction, respectively (Ingersoll et al. 1990).

There is almost no selenium toxicity data for terrestrial invertebrates. Aphids are reportedly sensitive to selenium, and spiders and mites have been controlled by commercial selenium insecticides (Trelease 1945). Simmons et al. (1988) reported that selenite in the drinking water of house flies (4,000 µg/L) caused 28 percent mortality after 12 days.

Among microinvertebrates, such as protozoans, an extremely wide range of toxicity-test results has been reported (Sanders and Gilmour 1994). Toxic thresholds in some taxa are as high as 10,000 µg/L selenite and in other taxa are as low as 3 µg/L selenite (inhibited growth of a heterotrophic flagellate). In microcosm studies, protozoan diversity (but not total biomass) was affected by 21-day exposures to 20–160 µg/L selenite.

The field applicability of guidelines generated by toxicity tests or controlled feeding trials is highly uncertain. Populations of brine shrimp in nature exhibit substantial variability in resistance to selenium toxicity (Freeman et al. 1987). Such findings suggest a potential in nature for rapid selection of populations that are more resistant than those used in tests. On the other hand, tests that replicate drainwater ionic chemistry and the presence of multiple trace elements suggest that toxicity thresholds in nature may be lower than in standard freshwater single-element toxicity tests (Dwyer et al. 1992; Naddy et al. 1995).

There are no documented field cases of fish and wildlife populations being affected adversely by selenium-induced alterations of invertebrate density or community structure. As noted above for plants, the direct toxic effects of consuming selenium-contaminated invertebrates are apparently more important than any indirect ecological effects. Fish and wildlife risk thresholds for dietary exposure to selenium are addressed in the summaries for vertebrate animals presented below.

Summary: Effects of selenium on invertebrates

Interpretive guidance	Invertebrate Se concentration (mg/kg)
Background, aquatic invertebrates	0.4–4.5 (typically <2.0)
Background, terrestrial invertebrates	<0.1–2.5 (typically <1.5)
Experimental LOAEL for sublethal effects (growth), midge larvae and amphipod tissue concentrations	2.5–15
Experimental LOAEL for sublethal effects (respiration rate) in crayfish	30 (hepato-pancreas)
Experimental LOAEL for reproductive effects, amphipod tissue concentration	32
	Dietary Se exposure (mg/kg)
Experimental LOAEL for sublethal effects (growth) in midge larvae	2.1
Experimental NOAEL for acute toxicity in amphipods	300
	Waterborne Se exposure (µg/L)
No clear community-level effects on benthic macroinvertebrates, outdoor macrocosm studies	25 (inorganic mixture)
Altered protozoan species diversity	20–160 (selenite)
Toxicity-test LOAEL's for acute toxicity, in midge larvae and amphipods	4.0 (seleno-methionine) 200 (selenite) 500 (selenate)
Toxicity-test LOAEL for sublethal (growth) effects on protozoans	3.0 (selenite)
Toxicity-test LOAEL's for chronic toxicity in midge larvae and amphipods	<0.5(?) (seleno-methionine) 25–100 (selenite) 25–100 (selenate)
Experimental LOAEL for drinking water toxicity in house flies	4,000 (selenite)

Fish

National and global monitoring programs have revealed that most species of fish average less than 4 mg Se/kg on a whole-body basis (e.g., Walsh et al. 1977; Schmitt and Brumbaugh 1990; Jenkins 1980). These surveys include data from both contaminated and uncontaminated sites and therefore include maximum values that do not repre-sent true "background." For comparison, Lemly (1993b) recently reported whole-body concentrations of selenium for more than
20 species of fish sampled at two confirmed Se-normal (waterborne Se ≤1 µg/L; Lemly 1985b) lakes in North Carolina. With few exceptions, the species averages were below
2 mg Se/kg. At several verified Se-normal sites in the San Joaquin River system of California, Saiki (1989) and Saiki et al. (1993) also found that mosquitofish, carp, bluegill, and largemouth bass averaged <2 mg/kg whole-body Se. Sunfish sampled at confirmed Se-normal sites in the lower Colorado River system averaged 1.6–2.4 mg/kg whole-body Se (Welsh and Maughan 1994). Controlled dietary exposures as high as 2 mg Se/kg in experimental studies consistently yield whole-body selenium concentrations <2.0 mg/kg (e.g., Ogle and Knight 1989; Hamilton et al. 1990; USFWS 1990b; Cleveland et al. 1993; Coyle et al. 1993; Lorentzen et al. 1994). Dietary exposures in this range would be typical of Se-normal environments (see summary tables for plants and invertebrates above). Background concentrations of selenium in skeletal muscle, gonads, and eggs also tend to average 2–4 mg/kg or less (e.g., Baumann and Gillespie 1986; Coughlan and Velte 1989; Crane et al. 1992; Hermanutz et al. 1992; Hamilton and Waddell 1994). Back-ground concentrations for hepatic selenium have been reported to range from 2 to 8 mg/ kg but are usually <5 mg/kg (Sorensen 1988; USFWS 1990b; Hermanutz et al. 1992; Lorentzen et al. 1994).

Lemly (1993a, 1996a) provided excellent reviews of selenium toxicity thresholds for fish. Based on that review, Lemly concluded that the most precise way to assess risks associated with exposure of fish to selenium is to measure selenium levels in gravid ovaries. More recently, Hamilton and Waddell (1994) reviewed the "gonad/egg" literature and concluded that fish gonads and eggs normally average 2–3 mg Se/kg and that 16–18 mg/kg was the current LOAEL for warm-water fish. This LOAEL, however, does not indicate the *true* threshold for adverse effects because it was associated with at least 50–80 percent reproductive impairment (Hermanutz et al. 1992). The lowest concentration of selenium in gonads and eggs reported for a case of *total* reproductive failure in fish (IC100) is 25–
30 mg/kg (Crane et al. 1992). Considering selenium concentrations in bird eggs, Heinz et al. (1989) found that the ratio between the threshold of total reproductive failure (IC100) and the threshold for reproductive impairment (≈IC10) was about 3–3.5 to 1. (See terato-genesis response table for black-necked stilts presented below.) Since background levels of selenium in eggs are similar for birds and fish (Hamilton and Waddell 1994; Skorupa and Ohlendorf 1991), and exposure-response curves for embryo teratogenesis are broadly similar (Lemly 1993b, p. 201: "It therefore appears that the teratogenic effects of selenium in natural populations of fish and aquatic birds are essentially the same."), it seems likely that the IC100 to IC10 ratio (3–3.5×) is also similar and that the threshold region for reproductive failure in *sensitive* species of fish extends as low as 7–10 mg Se/kg in gonads and eggs. Crane et al.'s (1992) study is consistent with this con-clusion because perch averaging about 8 mg Se/kg in gonads had slightly lower repro-ductive output than controls. As is often the case, though, Crane et al.'s study did not have the statistical power to conclusively test for the presence of a true threshold effect.

For bluegill, a sensitive species, Coyle et al. (1993) didn't find statistically significant reproductive impairment until the gonads and eggs had accumulated about 40 mg Se/kg. Because controls in Coyle et al.'s study exhibited

only 20 percent reproductive success, the study did not have the statistical power to detect anything but catastrophic reproductive impairment. If it is assumed that Coyle et al.'s treatment group of bluegill that had 40 mg Se/kg in gonads and eggs was relatively close to the IC100 threshold point, then the projected IC10 (based on a 3–3.5 ratio) would be 11–13 mg Se/kg.

At least two studies have related levels of 30–35 mg/kg dietary selenium (organic) to total reproductive failure (Woock et al. 1987; Coyle et al. 1993). Since selenium concentra-tions in gonad and egg tissues are propor-tional to dietary exposure, this suggests that the threshold dietary exposure to organic selenium (predominantly selenomethionine and selenocysteine) for reproductive impair-ment (assuming only parental exposure) is no higher than about 10 mg/kg (30–35 divided by 3–3.5). Results from Woock et al. (1987) are consistent with this estimate. In Woock et al.'s study, a diet of 13 mg Se/kg (organic) resulted in an "apparent" 8 percent reproductive depression. Thus, when adult female fish of sensitive species are exposed to dietary Se ≥ approximately 10 mg/kg, they are likely to produce eggs that contain enough selenium to impair survival of at least some offspring, even if the offspring themselves are never exposed to elevated dietary selenium. Conversely, studies of offspring from uncontaminated eggs suggest that larval fish are very sensitive to direct dietary selenium. Dietary exposures as low as 3–8 mg Se/kg (organic) have been demonstrated to impair normal juvenile survival and/or development in salmonids (Hamilton et al. 1990) and centrarchids (Cleveland et al. 1993; Lemly 1993c). In a more recent study, larval razorback suckers fed field-collected seleniferous invertebrates from the Green River system in Utah accumulated whole-body selenium concentrations >8 mg/kg (i.e., >2× the toxicity threshold) in two out of four trials, although the invertebrate food supply contained total Se of only 2.3–3.5 mg/kg (Hamilton et al. 1996).

In the study by Coyle et al. (1993), whole-body selenium values of about 15–20 mg/kg were associated with complete reproductive failure in bluegill. Crane et al. (1992) did not report whole-body residues but did report muscle concentrations associated with total repro-ductive failure in perch. Based on the muscle-to-whole-body conversion regression for bluegill provided by Saiki et al. (1991), whole-body selenium in the perch presumably was also about 15–20 mg/kg. Again, using a ratio of about 3–3.5 to back-calculate an estimate of the threshold region, the reproductive effects threshold for sensitive species would be expected at about 4–6 mg/kg whole-body Se. That is roughly the same as the threshold range of whole-body values associated with impaired survival and/or development of larval fish (Hamilton and Wiedmeyer 1990; Hamilton et al. 1990; Cleveland et al. 1993; Hamilton 1996).

Data on hepatic concentrations associated with exposures of fish to organic forms of selenium are insufficient for estimating a sensitive-species threshold. Lemly (1993a) suggested a risk threshold of 12 mg Se/kg based on experimental results that linked selenite exposure to perturbations of blood chemistry. However, the fish component of a hazard assessment protocol for selenium presented by Lemly (1995, 1996b) relies on data for fish eggs or whole-body residues (as a surrogate for eggs) but not on data for hepatic tissues. As recognized by Lemly, only a weak basis exists for assessing ecological risk in nature based on hepatic selenium concentrations and, therefore, they have little interpretive value.

No sublethal effects have been reported for adult or juvenile fish at levels lower than the thresholds estimated above for reproductive effects (e.g., Lemly 1993a, 1995, 1996a, b). Mortality of adult fish, even in sensitive species, occurs at exposure and tissue thresholds much higher than those that produce reproductive impairment. For example, even among fingerling bluegill, some

specimens survived a 44-day dietary exposure of 130 mg Se/kg (organic) (Finley 1985; Coyle et al. 1993).

Consumption advisories to protect human health were issued in California when edible fish tissue was known to exceed 2 mg Se/kg on a wet weight basis (Fan et al. 1988; Saiki et al. 1991). When edible tissues exceed 5 mg Se/kg on a wet weight basis, health professionals advise against any human consumption (A. Fan, pers. comm., cited in Texas Parks and Wildlife Department 1990).

Summary: Effects of selenium on fish

Interpretive guidance	Fish Se concentration (mg/kg)
Background, whole body	<1–4 (typically <2)
Background, skeletal muscle	<1–4 (typically <2)
Background, gonads/eggs	<1–4 (typically 2–3)
Background, hepatic	2–8 (typically <5)
Lowest validated concentration in edible tissue (trout fillet) warranting human health advisory	2.0
Outdoor macrocosm LOAEL for reproductive impairment (bluegill)	16–18 (gonad/egg tissue)
Estimated true threshold range (≈IC10) for reproductive impairment in sensitive species (perch, bluegill)	7–13 (gonad/egg tissue)
Experimental LOAEL for total reproductive failure (bluegill)	15–20 (whole body, parental)
Estimated true threshold range (≈IC10) for reproductive impairment in sensitive species (perch, bluegill, salmon)	4–6 (whole body, parental or offspring)
Complete reproductive failure (IC100) in sensitive species (bluegill)	30–35 (food-chain Se or selenomethionine)
Estimated true threshold range (≈IC10) for reproductive failure in sensitive species (bluegill), parental exposure only	10 (food-chain Se or selenomethionine)
Experimental LOAEL's for reproductive impairment via lethal larval dietary exposure (salmon, bluegill, razorback suckers)	3–8 (food-chain Se or selenomethionine)
	Edible tissue Se (mg/kg)
Health advisories recommend limited fish consumption by healthy adults and no consumption by children and pregnant women	2 (wet weight basis)
Complete ban on human consumption of fish recommended	5 (wet weight basis)

Amphibians and Reptiles

Bullfrogs collected from reference agroforestry plantations in the San Joaquin Valley con-tained 1.0–1.9 mg/kg whole-body Se (CDFG 1993). Frog and toad livers from reference sites in the same area contained 2.9–3.6 mg Se/kg (Bryne et al. 1975; Ohlendorf et al. 1988b). In lizards and snakes from these areas, whole-body selenium averaged 0.7–2.0 mg/kg, and these values probably repre-sent normal background concentrations even though some of the snakes were collected at sites where selenium-contaminated water is used to irrigate trees (CDFG 1993). Water snakes from Florida also contained about 1–2 mg/kg whole-body Se (Winger et al. 1984). Livers of gopher snakes from reference sites near Kesterson Reservoir contained about 1–4 mg Se/kg (Ohlendorf et al. 1988b). Pine snake hatchlings from the New Jersey pine barrens

region averaged 2.6 mg Se/kg in skinless whole-body samples (skins averaged 1.6 mg/kg) (Burger 1992). These hatchlings are probably good indicators of normal selenium levels in snake eggs. American alligator eggs from Florida contained about 1.0–2.1 mg Se/kg (Heinz et al. 1991). Both the pine snake hatchlings and the alligator eggs suggest that normal background concentra-tions of Se in amphibian and reptile eggs are the same as in fish and bird eggs (i.e., typically averaging 1–3 mg/kg).

Apparently, no field data are available for assessing toxic thresholds in amphibians and reptiles. Experimental data are limited to toxicity tests that found *Xenopus* (frog) larvae sensitive to >1,000 µg/L waterborne selenite (Browne and Dumont 1979) and found an LC50 of 90 µg/L waterborne selenite for eggs and larvae of the narrow-mouthed toad (Birge et al. 1979).

Based on how similar the toxic threshold values are for fish and bird eggs (e.g., Lemly 1995, 1996b), two other classes of egg-laying vertebrates, it is probably safe to assume for amphibians and reptiles that (1) reproductive impairment is among the most sensitive response variables and (2) populations producing eggs with ≥10 mg Se/kg are reproductively impaired. Another provisional interpretive guideline that seems justified, based on existing knowledge for all other taxa of vertebrates, is that whole-body concentra-tions at or above 10× normal background (or ≥20 mg/kg) are probably toxic to populations of sensitive species.

Birds

Birds are rarely analyzed for contaminant concentrations on a whole-body or carcass basis. One national monitoring program, however, analyzed selenium concentrations in starling carcasses (feathers, legs/feet, beaks removed). Starling carcasses usually averaged less than 2 mg Se/kg (White et al., 1988).

Summary: Effects of selenium on amphibians and reptiles

Interpretive guidance	Biomass Se concentration (mg/kg)
Background, whole body	0.7–3 (typically <2)
Background, eggs	1–3
Background, hepatic	2.9–3.6
Presumptive reproductive impairment threshold	≥10 (eggs)
Presumptive adverse effects threshold on a whole body basis (10× normal)	≥20 (whole body)
	Waterborne Se concentration (µg/L)
Lowest toxicity-test LC50 for amphibian eggs/larvae	90

Selenium exposure in birds is more typically measured in specific tissues: muscle, liver/ kidney, eggs, feathers, or blood/plasma. Normal concentrations of selenium in muscle are about 1–3 mg/kg (e.g., White et al., 1987; Ohlendorf et al. 1990; Barnum 1994). Liver and kidney tissue usually contain comparable concentrations of selenium (Ohlendorf et al. 1988a; Heinz 1996) and will be referred to in aggregate as hepatic tissue. Reference inter-quartile ranges for selenium in avian hep-
atic tissue (in mg/kg) vary from 2.0–4.3 (3.3 median) in rallids (mostly American coots), to 5.2–9.5 (7.5 median) in anatids (dabbling ducks), to 6.0–9.9 (8.2 median) in recurviro-strids (stilts and avocets) (Skorupa et al., 1992). These three taxa represent herbivorous, omnivorous, and carnivorous (invertebrate prey) groups of birds and therefore should represent the full range of normal background concentrations. Bird eggs collected from Se-normal study areas usually average ≤3 mg Se/kg (grand median across all taxa of 1.9 mg/ kg), and the maximums are usually <5 mg Se/kg (Skorupa and Ohlendorf 1991; Ohlendorf et al. 1993). The selenium content
of feathers is usually 1–2 mg/kg (Parrish et al.

1983; Burger et al. 1992a; Burger and Gochfeld 1992a,b; Burger and Gochfeld 1993; Burger et al. 1994) but may be <1 mg/kg in areas of selenium-poor soil (Burger et al. 1993). However, the presence of mercury has the effect of directing more selenium content into the feathers, and so feathers may reach concentrations of 2–4 mg Se/kg in environments containing elevated mercury levels (GLSAB 1981; Burger et al. 1992b). Avian whole blood normally contains about 0.1–0.4 mg Se/kg on a wet weight basis (e.g., Ihnat 1989; Heinz et al. 1990; USBR 1995).

Heinz (1996) provides an excellent recent review of selenium toxicity thresholds for birds. He found that reproductive impairment is one of the most sensitive response variables and eggs are the most reliable tissues for interpretive purposes. Unlike most sampling techniques, egg studies easily avoid a sampling bias that favors survivors. Bird eggs are sampled without regard for the status of the embryo inside the egg. Live and dead embryos have equal probabilities of being sampled. Therefore, accurate population-level exposure assessments and unbiased exposure-response curves can be obtained from field samples of eggs. Reproductive impairment is generally a more sensitive response variable than adult mortality, *in ovo* exposure to selenium is discrete and easily measured, and birds' sensitivity to selenium is equal to or greater than that of other taxa. Consequently, bird eggs constitute one of the best biotic matrices for risk/impact interpretation (Ohlendorf et al. 1986; Heinz et al. 1987, 1989; Skorupa and Ohlendorf 1991; CH2M Hill et al. 1993; Ohlendorf et al. 1993; Skorupa 1994; Seiler and Skorupa 1995; Heinz 1996; Skorupa 1998a).

Based on a review of experimental and field data, Heinz (1996) estimated that the embryotoxic threshold for selenium in bird eggs is about 10 mg/kg. Several taxa-specific exposure-response profiles for avian embryos have been derived from field sampling during the last decade (Skorupa et al., unpub. data; Skorupa et al. 1993; Skorupa 1998a). Based on those profiles (presented below), bird species differ substantially in embryo sensitivity to selenium exposure. The variation seems to have more to do with salinity tolerance than with phylogeny. Species of birds that prefer athalassohaline (nonmarine saline) wetlands produce embryos that are more tolerant of selenium than closely related species that favor freshwater wetlands. For example, embryos of American avocets tolerate selenium better than do those of black-necked stilts, and snowy plover embryos are more tolerant than those of killdeer. A possibility that deserves some attention is that differences in embryonic selenium tolerance may be a consequence of natural selection for tolerance to sulfate salinity. Whatever is causing the interspecific variation, once identified, it would have tremendous interpretive implications.

With the possible exception of cinnamon teal, dabbling ducks appear to be among the most sensitive species of waterbirds. A recent statistical analysis of the teratogenesis data for ducks revealed that the IC10 was 23 mg Se/kg egg (Skorupa 1998b). Field-collected teratogenesis response data for duck embryos are distributed as follows:

Ducks

Egg Se range: ducks (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–10	0.0
11–20	3.2
21–30	8.7
31–40	40.0
41–50	Insufficient data
51–60	Insufficient data
61–70	Insufficient data
71–80	100.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal duck populations in Montana (i.e., representative eggs contained <5 mg Se/kg) was approximately 0.3 percent based on a monitored sample of >3,000 eggs (calculated from data in Dubois 1988). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=138 eggs, mostly from the San Joaquin Valley of California.

For individual duck eggs containing as little as 11–20 mg/kg, the observed probability of teratogenesis is low in absolute terms but is nevertheless 10× background. The above numbers are individual-level data, but they support the population-level analysis of Skorupa and Ohlendorf (1991). For inter-pretive purposes, however, it is very impor-tant to distinguish between individual-level and population-level guidelines. Population-level thresholds are usually lower than individual-level thresholds because they are based on population averages even though it is actually the maximum values that deter-mine when a population crosses the toxicity threshold (i.e., the point at which just a few hens in the population show a toxic response). Quite often, only population levels of selenium exposure (means and standard errors) are reported in the literature, and that is why Skorupa and Ohlendorf (1991) were limited to a population-level analysis of published field data. The response profiles provided here are guides for individual-level risk interpretation (the level that is most useful but requires far more effort to con-struct field-validated profiles). Teratogenesis is not as sensitive a response variable as egg hatchability, but due to high rates of nest parasitism among the duck populations sampled, it was not possible to construct a response profile based on hatchability.

Black-necked stilts are not as sensitive to selenium poisoning as ducks but are still moderately sensitive. Field-collected teratogenesis response data for stilt embryos are distributed as follows:

Stilts—Teratogenesis

Egg Se range: stilts (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–10	0.4
11–20	1.3
21–40	5.0
41–60	24.4
61–80	71.4
81–100	100.0
101–120	100.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal stilt and avocet populations (recurvirostrids) in the San Joaquin Valley of California was approximately 0.15 percent based on a monitored sample of >3,000 eggs (Skorupa et al., unpub. data). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=547 eggs, mostly from the San Joaquin Valley of California.

The response profile above is not directly comparable to the embryotoxicity curve for stilts presented by Ohlendorf et al. (1986, figure 2) because their curve was not restricted to true teratogenesis (irreversible structural deformities) but included all forms of embryo impairment (including reversible pathologies). More importantly, Ohlendorf et al.'s data set included some samples that were non- randomly selected for chemical analysis specifically because the samples contained abnormal embryos (H.M. Ohlendorf and R.L. Hothem, pers. comm.). Inclusion of some nonrandom data points statistically biases the response curve upward. Within each exposure category, eggs with abnormal embryos had a higher probability of being sampled (i.e., selected for chemical analysis) than unim-paired eggs and, therefore, the frequency of response at each exposure interval is overestimated. Comparing the duck data to that for stilts, notice that the incidence of teratogenesis in ducks shows a substantive increase at a distinctly lower threshold (≈35 mg/kg versus ≈50 mg/kg).

Field-collected clutch viability data for stilts are distributed as follows:

Stilts—clutch viability

Egg Se range: stilts (mg/kg)	Observed probability of impaired clutch (%)
0–5	8.7 (background)
6-15	18.9
16-30	26.9
31-50	33.7
51-70	65.4
71-90	100.0
91-110	100.0

Notes: These data represent the percentage fo hens within each exposure interval that were reproductively impaired; a henwise response rate based on whole-clutch viability (4-egg clutches). The background rate is a function of normal infertility. Total *N*-410 full-term, monitored, and chemically characterized clutches, mostly from the San Joaquin Valley of California.

Avocets—teratogenesis

Egg Se range: avocets (mg/kg)	Observed probability of overt embryo teratogenesis (%)
00–40	0.0
41–60	3.8
61–80	7.1
81–100	9.1
101–120	50.0

Notes: The field-measured background rate of overt embryo teratogenesis for Se-normal stilt and avocet populations (recurvirostrids) in the San Joaquin Valley of California was approximately 0.15 percent based a monitored sample of >3,000 eggs (Skorupa et al., unpub. data). Only eggs that were both randomly sampled in the field and randomly selected for chemical analysis are included above. Observed probabilities are for true teratogenesis only, not for all types of abnormalities. Total *N*=542 eggs, mostly from the San Joaquin Valley of California.

The henwise (=clutchwise) response profile presented above provides individual-level interpretive guidance that incorporates all forms of embryo impairment, not just terato-genesis. Ohlendorf et al. (1986, figure 3) also presented a clutchwise response profile for stilts, but it is biased upward due to the inclusion of some samples nonrandomly selected for chemical analysis. For example, Ohlendorf et al.'s clutchwise response curve suggests that 60 percent of stilt hens in the 31–50 mg Se/kg egg exposure category will be reproductively impaired as opposed to an estimate of 33 percent (which is below Ohlendorf et al.'s lower 95 percent confidence boundary) based on strictly random samples. Skorupa (1998a) has recently provided a detailed statistical analysis of the Tulare Basin stilt data and shown that the IC10 for teratogenesis is 37 mg Se/kg egg and that the threshold point for hatchability effects is 6–7 mg Se/kg egg.

As a final species-specific example, American avocets are very tolerant to selenium poisoning, even compared to the closely related stilts. Field teratogenesis response data for avocet embryos break down as follows:

The IC50 for embryo teratogenesis in avocets is 105 mg Se/kg egg, or about 40–45× normal background (Skorupa 1998a). That is roughly four times the value for ducks and about twice the value for stilts. Even the IC10 for avocets, 74 mg Se/kg egg, is very high (Skorupa 1998a). Interestingly, Goodsell (1990), studying Australian varieties of avocets and stilts, found a similar degree of differences in these birds' tolerance for salinity. Red-necked avocet chicks were far more salinity tolerant than black-winged stilt chicks in the same areas. As noted in Skorupa (1996), selenium tolerance and salinity tolerance are closely related. The predominant salt at many saline-sink wetlands is sodium sulfate, and the biochemistry of selenium is very similar to that of sulfur. Thus, any mechanism that has evolved to cope with the effects of sulfate salinity is likely to be equally effective against selenium. The Australian avocets and stilts are close ecological equivalents to the North American species. (Johnsgard [1981] considers black-winged and black-necked stilts to be the same species.)

Field-collected clutch viability data for avocets are distributed as follows:

Avocets—clutch viability

Egg Se range: avocets (mg/kg)	Observed probability of impaired clutch (%)
00–20	13.5 (background)
21–40	14.6
41–60	11.8
61–80	33.3
81–100	50.0

Notes: These data represent the percentage of hens within each exposure interval that were reproductively impaired; a henwise response rate based on whole-clutch viability (4-egg clutches). The background rate is a function of normal infertility. Total *N*=230 full-term, monitored, and chemically characterized clutches, mostly from the San Joaquin Valley of California.

Whereas 30 percent of stilt hens are reproductively impaired when eggs contain 40 mg Se/kg, avocet eggs have to contain 70 mg/kg for 30 percent of the hens to be reproductively impaired.

At Heinz's (1996) recommended threshold concentration of 10 mg Se/kg in avian eggs, threshold proportions (≈10 percent) of duck and stilt hens would indeed exhibit reproductive selenosis. Probably, though, no avocet hens would show any such effect. More limited sets of field response data for snowy plovers and killdeer (Skorupa et al., unpub. data) suggest profiles that are very similar to those for avocets and stilts, respec-tively. In addition to the differences between species in their responses to equal *in ovo* exposures, other differences are related to dietary habits. Three experimental studies of flesh-eating birds have found that less selenium was transferred from the hen's diet to the egg than is typical of plant- and invertebrate-eating species of birds (Smith et al. 1988, black-crowned night-herons; Wiemeyer and Hoffman 1996, screech owls; and USBR 1995, American kestrels). Based on Wiemeyer and Hoffman's screech-owl study, the only one with straightforward repro-ductive performance data, general interpretive guidelines based on *in ovo* exposure are probably applicable to embryos of flesh-eating birds, but greater environmental exposure of the hens is probably required for them to produce eggs that attain threshold concentra-tions. Based on experimental studies with chickens and quail (see Heinz 1996), embryos of upland gallinaceous game birds may be more sensitive than duck embryos. Studying white-faced ibis at Carson Lake, Nevada, Henny and Herron (1989) reported that nests in which the eggs contained >6 mg Se/kg were 16 percent less productive than nests that had eggs containing <4 mg Se/kg. That's about the magnitude of effect that would be expected at that exposure level (6–9 mg/kg) for a moderately sensitive species (like stilts), but the small sample of >6 mg/kg eggs and the confounding presence of high DDE and/or mercury levels in some eggs made Henny and Herron's (1989) study inconclusive with regard to selenium.

In birds, reproductive impairment can result from diets containing as little as about 3–8 mg Se/kg (Wilber 1980; Martin 1988; Heinz 1996). Nonbreeding adult birds can tolerate higher levels of selenium, but still it is recommended that their dietary exposure not exceed 10–15 mg Se/kg (Heinz 1989; Heinz and Fitzgerald 1993b). These dietary thresholds have been estimated primarily from feeding trials with selenomethionine. When selenium is added to artificial diets as purified seleno-methionine, its toxicity and bioavailability are virtually identical to those of naturally incor-porated selenium in high-selenium wheat (Heinz et al. 1996) and in contaminated small mammals that were collected on the site of the former Kesterson Reservoir and fed to captive American kestrels (USBR 1995). Skorupa and Ohlendorf (1991), however, presented data suggesting that perhaps naturally incorporated selenium in highly chitinous species or life stages of invertebrates might be less bio-available than purified selenomethionine added to artificial diets. As was found in the results for

fish, the lowest levels of dietary selenium that were fatal to either juvenile or adult birds occurred in treatments that coincided with winter metabolic stress (Tully and Franke 1935; Heinz 1996).

Determinations of selenium concentrations in avian hepatic tissues provide a very limited basis for interpretation. Although hepatic concentrations are always elevated in popu-lations of birds exposed to toxic levels of selenium (e.g., USFWS 1990a; Heinz 1996), they are sometimes very elevated in popu-lations inhabiting Se-normal environments (e.g., Rowe et al. 1991; Rinella and Schuler 1992). In the latter cases, avian eggs contain back-ground concentrations of selenium (consistent with environmental conditions) even though the hepatic concentrations are elevated. The converse situation, background hepatic concentrations combined with elevated egg concentrations, has never been recorded. Heinz and Hoffman (1998) reported that simultaneous dietary exposure to organic mercury and organic selenium increased the hepatic concentrations of selenium in captive mallards by >10× the level from selenium-only diets. Perhaps the confusing field cases in which low environmental (and egg) levels of selenium are accompanied by elevated hepatic levels would be explained by elevated exposures to mercury. In normal-mercury environments, it appears that hepatic con-centrations ≥30 mg Se/kg are highly likely to be associated with reproductive impairment (USFWS 1990a; Skorupa et al. 1992). The 30-mg Se/kg level may mark the approximate threshold for toxicity in young and adult birds (Heinz 1996). In any environment, hepatic concentrations ≤10 mg Se/kg indicate normal (safe) selenium exposure. In summary, hepatic concentrations of selenium are more reliable for delineating populations that are *not* suffering from selenium toxicity than they are for identifying poisoned populations. Elevated levels of hepatic selenium should not be interpreted as anything more than an indication that further study is warranted.

Field data have not been collected that reliably relate concentrations of selenium in avian muscle (usually breast muscle) to toxic effects. An important factor that might confound such attempts is the fact that muscle tissue accumu-lates and disposes of contaminants more slowly than other tissues do (Heinz 1996), and so samples are less likely than other tissues to show the effects of recent dietary exposure. In experimental studies, concentrations ≥20 mg Se/kg in breast muscle of adult mallards were sufficient to cause death (USFWS 1990a). Breast muscle is most frequently sampled in the field to provide interpretive data for assessing human health hazards (e.g., Barnum 1994). Concentrations that warrant health advisories and consumptive bans are the same as summarized above for edible fish tissues.

Feathers and blood are important sample tissues because they are the only ones that can be collected without sacrificing the animals being sampled. Therefore, these are the primary avian analytical matrices available for assessing the selenium status of endangered species. Despite this important role, no substantive interpretive basis has been developed for either tissue. For example, no study has yet established threshold concen-trations of selenium in feathers or blood that can be reliably associated with reproductive impairment. Experimental studies found that selenium-poisoned captive mallards had 5–14 mg Se/kg wet weight basis in their blood (or in the blood of treatment group survivors exposed to the same diet) (Heinz 1996). In American kestrels, blood containing ≥1 mg Se/kg (wet weight) resulted from a diet of ≥5 mg Se/kg (USBR 1995)—a level that could cause reproductive impairment in sensitive species of birds (e.g., Lemly 1995, 1996b). A follow-up study of captive kestrels suggested an adverse effects threshold between 1.2 and 2.1 mg Se/kg ww in blood, based on fertility and body condition as the response variables (USBR 1997; Gary Santolo, CH2M Hill, pers. comm. to J. Skorupa, USFWS). As a general interpretive guide, any selenium concentration

that exceeds about 5 mg Se/kg in feathers or 1 mg Se/kg in blood (wet weight) will mean that further study is warranted.

Summary: Effects of selenium on birds

Interpretive guidance	Bird Se concentration (mg/kg)
Background, whole body	Typically < 2
Background, muscle	1–3
Background, eggs	Mean <3 (typically 1.5–2.5) Maximum <5
Background, hepatic (median values)[1]	3.3 (herbivore) 7.5 (omnivore) 8.2 (carnivore)
Background, feathers	1–4 (typically 1–2)
Background, blood	0.1–0.4 (wet weight basis)
Embryo teratogenesis threshold (≈IC10), wild ducks (sensitive taxon)	23 (in ovo)
Embryo viability (=egg hatchability) threshold, captive mallards	10 (in ovo)
Embryo teratogenesis threshold (≈IC10), American avocets (tolerant taxon)	74 (in ovo)
Embryo viability (=egg hatchability) threshold, American avocets	61–80 (in ovo)
Hepatic threshold for juvenile and adult toxicity	30 (liver)
Muscle threshold for juvenile and adult toxicity	≈20 (breast muscle)
Provisional feather threshold warranting further study	5 (breast feathers)
Provisional blood threshold warranting further study	1 (whole blood) (wet weight basis)

Interpretive guidance	Dietary Se exposure (mg/kg)
Reproductive impairment threshold	3–8
Toxicity threshold for nonbreeding birds exposed to winter-stress	10–15
	Edible tissue Se (mg/kg)
Health advisories recommend limited consumption by healthy adults and no consumption by children and pregnant women	2 (wet weight basis)
Complete ban on human consumption recommended	5 (wet weight basis)

[1] Background hepatic concentration is typically <10 but can be much higher in Hg-contaminated environments with normal Se.

Mammals

Based on the lowest whole-body selenium values reported by Clark (1987) for Kesterson Reservoir, and the diet-to-whole-body transfer values reported by Ohlendorf and Santolo (1994), normal whole-body selenium concentrations in small mammals are probably <2 mg/kg. For example, kangaroo rats, little brown myotis bats, and Brazilian freetail bats from agroforestry plantations in the San Joaquin Valley had a median whole-body selenium concentration of about 1.2 mg/kg (CDFG 1993).

Normal concentrations of selenium in muscle are typically <1 mg/kg in bats (Schroeder et al. 1970), pronghorn antelope (Raisbeck et al. 1996), gophers, voles, deer mice, house mice, rabbits, hares (CDFG 1993), and macaque monkeys (Hawkes et al. 1994). The data for wild species of mammals agree with data from many species of domestic mammals (Jenkins 1980).

Normal concentrations of selenium in livers of mammals characteristic of aquatic habitats, such as muskrats and raccoons, range from about 1 to

10 mg/kg and typically average ≤5 mg/kg (Schroeder et al. 1970; Clark 1987; Clark et al. 1989). Similar results have been reported for terrestrial predators that may seasonally focus their hunting in aquatic habitats, such as foxes and coyotes (e.g., Paveglio and Clifton 1988), and for an assortment of small terrestrial mammals trapped in the vicinity of an Se-normal
San Joaquin Valley wetland (Clark 1987). Another group of small mammals collected near agroforestry plantations averaged <3 mg Se/kg in their livers, except at plantations using high-selenium water for irrigation (CDFG 1993).

Selenium in whole blood normally averages <0.5 mg/L in domestic mammals (Jenkins 1980) and in raccoons, pronghorn antelope, and coyotes (Paveglio and Clifton 1988; Clark et al. 1989; Schamber et al. 1995). Less than 0.1 mg/L in whole blood is considered deficient, and deficiency seems to be more common than excessive exposure among wild ungulates such as deer (e.g., Oliver et al. 1990; Hein et al. 1994). Human whole blood normally contains 0.1–0.3 mg/L (USPHS 1989).

Data for a wide range of mammalian species suggest that the normal concentration of selenium in individual samples of hair is less than 3 mg/kg. Population averages normally range from about 0.5 to 1.5 mg Se/kg, including those for human populations (Huckabee et al. 1972; Paveglio and Clifton 1988; Clark et al. 1989; USPHS 1989).

One study reported an average concentration of 0.015 mg Se/L in the milk of a control group of nursing macaque monkeys (Hawkes et al. 1994). Most herds of dairy cows also average <0.05 mg Se/L in milk (Jenkins 1980).

Clark et al. (1989) reported that the feces of raccoons averaged about 1 mg Se/kg at an Se-normal wetland in the San Joaquin Valley of California.

There have been no well-documented cases of widespread selenosis among wild mammals comparable to the multiple examples available for fish and birds (Skorupa 1998a). Poisoning in nature has been reported for mammals but has been largely restricted to free-range domestic livestock, primarily horses, cows, and sheep (e.g., Rosenfeld and Beath 1946; Olson 1986; Raisbeck et al. 1993). As is the case for fish and birds, young animals are generally more sensitive (Thompson et al. 1991).

The lowest dietary threshold for mammalian toxicity reported in the literature is 1.4 mg Se/kg (natural selenium, dry feed basis); sublethal effects were noted in rats following lifetime exposure at that level (Eisler 1985). At 3 mg/kg in the lifetime diet, longevity was reduced (Eisler 1985). Also at 3 mg/kg (high-Se wheat), Olson (1986) reported reproductive selenosis in rats. Sublethal effects were observed in dogs exposed to about 7 mg/kg (high-selenium corn) (Rhian and Moxon 1943). Most domestic animals exhibit signs of toxicity on diets containing ≥3–5 mg/kg (natural selenium) (NRC 1980; Eisler 1985; Olson 1986). Pronghorn antelope showed some symptoms of immunotoxicity on a diet of 13–16 mg Se/kg (high-selenium hay), but were overtly healthy and more tolerant to dietary selenium than would be expected based on veterinary standards developed for domestic ungulates (Schamber et al. 1995; Raisbeck et al. 1996). Macaque monkeys given intravenous doses equivalent to about 16 mg/ kg (dry feed basis) dietary selenomethionine showed obvious toxic effects (Hawkes et al. 1994). The minimum long-term dietary exposure found to produce sublethal toxic effects in humans is 1.9 mg/kg (natural selenium, *wet weight*) (USPHS 1989).

Several studies summarized by Hawkes et al. (1994) suggest that mammalian teratogenic

effects occur only when maternal dietary exposure is high enough to adversely affect the mother. Under such circumstances it is not clear whether the teratogenesis is a direct effect of fetal selenium exposure or a consequence of maternal poisoning (Ferm et al. 1990).

Others have reported nonteratogenic reproductive depression as the principal effect of chronic selenosis in mammals, even when maternal selenosis is not apparent (e.g., James et al. 1981). Clark et al. (1989) reported that, for raccoons, hair selenium was one of the best indicators of the extent of exposure to selenium, and James et al. (1981) presented guidelines for interpreting risk of selenium-induced reproductive depression based on hair selenium. Less than 5 mg/kg was considered nontoxic, 5–10 mg/kg borderline toxic, and >10 mg/kg hair selenium was considered a toxic exposure. Samples of hair with more than about 10 mg Se/kg from natural popu-lations of pronghorn antelope, coyotes, meadow voles, mule deer, shrews, and raccoons have been reported from California, Idaho, North Carolina, and Wyoming (Huckabee et al. 1972; Paveglio and Clifton 1988; Clark et al. 1989). A chronically poisoned human population averaged 32 mg Se/kg in hair (USPHS 1989).

Based on whole blood, toxicologically significant thresholds for land mammals are generally cited as about 1–5 mg Se/L for chronic selenosis (e.g., Rosenfeld and Beath 1946; Edwards et al. 1989). Raisbeck et al. (1993) documented four recent cases of overt equine selenosis in Wyoming and reported blood values of 0.86, 0.96, 1.1, and 1.3 mg Se/L. Pronghorn antelope fed high-selenium hay exhibited immune system disorders and had blood selenium values of 1.0–1.3 mg/L (≈4–5× normal) (Schamber et al. 1995). Raccoons at Kesterson Reservoir had as much as 9.4 mg Se/L in their blood (average 2.6 mg/L) without overt signs of selenosis (among "survivors"), although four of eight specimens had deformed liver cells. A blood level of 5 mg Se/L was found in captive sea lions that had died after eating high-selenium fish (Edwards et al. 1989). A chronically poisoned human population had blood selenium averaging 3.2 mg/L (USPHS 1989).

General guidelines for interpreting concentrations of liver selenium have been developed primarily through veterinary studies. For example, Edwards et al. (1989) cite a toxicological threshold range of about 45–60 mg/ kg for liver selenium in domestic livestock, based on a veterinary handbook (Osweiler et al. 1985). Raisbeck et al. (1996) cited a more conservative toxic threshold of about 20 mg Se/kg in the liver but noted that feeding trials with pronghorn antelope show that the criterion may not be reliable. Buechner (1950), however, had already reported that prong-horn antelope are notably tolerant of toxic forage. This raises the question of whether the diagnostic criterion is broadly inapplicable or whether pronghorn antelope are a particularly insensitive taxon. More recently, O'Toole and Raisbeck (1998) reported for cattle and horses that hepatic selenium concentrations exceeding 2 mg/kg wet weight (~6-8 mg/kg dry weight), combined with other typical clinical symptoms, was a sufficient basis for a firm diagnosis of selenium poisoning. Interpretive criteria for hepatic tissues are not well supported, and as suggested above for birds, the primary interpretive value of hepatic measures may be to identify Se-normal populations of mammals rather than to identify poisoned populations.

Presumably the guidelines used for human health advisories regarding consumption of fish and birds (see above) should also apply to edible tissues of mammals. These call for targeted restrictions at 2 mg/kg (ww) and a complete consumptive ban at 5 mg/kg (ww).

Summary: Effects of selenium on mammals

Interpretive guidance	Mammal Se concentration (mg/kg dw, except as noted)
Background, whole body	<1–4 (typically <2, mean)
Background, muscle	<1
Background, liver (aquatic habitat mammals)	1–10 (typically <5, mean)
Background, blood	0.1–0.5 mg/L (typically 0.2–0.3, mean)
Deficient, blood	<0.1 mg/L
Background, hair	<1–3 (typically 0.5–1.5, mean)
Background, milk	<0.05 mg/L
Background, feces	<2
Reproductive depression threshold, hair	>10
Overt equine selenosis threshold, blood	1 mg/L
Human chronic selenosis threshold, blood	3 mg/L
Acute lethal toxicity LOAEL, sea lions, blood	5 mg/L
Veterinary toxicological handbook threshold, domestic livestock, liver	45–60
	Dietary Se exposure (mg/kg dw, except as noted)
Sublethal effects threshold, lifetime exposure of rats	1.4
Chronic selenosis threshold, humans	1.9 (ww)
Reduced longevity threshold, lifetime exposure, rats	3
LOAEL for reproductive selenosis, in rats	3
Overt toxicity thresholds, domestic livestock	3–5
Sublethal effects LOAEL, dogs	7
Health advisories recommend limited consumption by healthy adults and no consumption by children or pregnant women	≥2
Complete ban on human consumption recommended	≥5

Bioaccumulation

Toxicity varies for different forms of selenium, animal species, duration of exposure, method of uptake, and other factors. In wetland areas, bioaccumulation increases the levels of selenium in the food chain. Selenium is taken up by aquatic biota, including phytoplankton, zooplankton, and insects, which contribute to the diet of higher forms of wildlife. In particular, selenium accumulation in the food chain has caused the deaths of many fish and aquatic birds, young and old, and has led to reproductive failure and deformed offspring (Skorupa 1998a).

Selenite and selenate, the most common aqueous forms of selenium, are biotrans-formed into organic chemical species after uptake by primary producers such as algae (Ogle et al. 1988). Speciation of dissolved selenium in water strongly influences how much aquatic loading is required to bio-accumulate dangerous concentrations of selenium in the food chain, but waterborne speciation does not appear to influence the unit toxicity of food chain incorporated selenium (USFWS 1990b; Besser et al. 1993). After selenium becomes incorporated in the food chain, apparently the issue of chemical speciation is not an important interpretive factor. Toxicologically, food chain selenium in nature seems to be fairly uniform, with a toxicity profile very similar to that of selenomethionine (e.g., Woock et al. 1984; Hamilton et al. 1990; Heinz 1996). This is a particularly useful interpretive consideration since dietary exposure is the primary exposure pathway for fish and wildlife populations.

Dietary plant selenium is readily absorbed by animals. Most of the selenium (70–80 percent) is quickly metabolized and eliminated, but the remaining selenium becomes bound or incorporated into blood and tissue and is only slowly eliminated (Olson, 1978). Selenium easily enters metabolic pathways and there-fore is highly bioaccumulative. The high pro-pensity for biotic uptake of selenium is at least partially explained by its biochemical similarity to sulfur.

Interactions

Interactions between selenium and mercury have been extensively documented (Cuvin-Aralar and Furness 1991; Sorensen 1991), although many conflicting results have been reported. Depending on the exact chemical speciation of the two elements, and other factors, the toxicity of selenium can be increased, reduced, or unaffected by the presence of mercury. Few Se-Hg interaction studies have examined combined dietary exposure to methyl mercury and seleno-methionine in fish or wildlife, even though these forms of mercury and selenium are the ones most likely to be combined in a natural diet. A recent study using captive mallards found conflicting antagonistic and synergistic Se-Hg interactions with reference to adult toxicity and reproductive impairment: i.e., selenium and mercury together are less likely to poison adult birds than either element separately but more likely to impair repro-duction (Heinz and Hoffman 1998).

Dietary protein, boron, arsenic, and methionine concentrations are other factors clinically demonstrated to alter the toxicity of selenium to wildlife (Hoffman et al. 1991, 1992a,b; Stanley et al. 1994). However, the dose combinations of these factors that altered selenium toxicity in the laboratory would rarely be found in nature. Stanley et al. (1996) recently reported a lack of interaction effects between selenium and boron added to the diets of captive mallards.

The strong exposure-response relationships documented for selenium in field-collected bird eggs and fish suggest that interactions with other elements rarely affect selenium toxicity in the field (Ohlendorf et al. 1986; Skorupa and Ohlendorf 1991; Lemly 1993b; Ohlendorf et al. 1993; Skorupa 1998a). A multivariate statistical analysis of chemical data for bird eggs from California's
San Joaquin Valley (Skorupa 1998b) demonstrated a strong correlation between embryo deformity and *in ovo* selenium concentration, which was unaffected by other potentially interactive chemical constituents present in the eggs. There is, however, sub-stantial individual and taxonomic variability in sensitivity to selenium poisoning (EPA 1987; SJVDP 1990; USFWS 1990a, b; Sorensen 1991; Lemly 1993b).

In some cases, levels of exposure to selenium that wouldn't be directly toxic may increase susceptibility to otherwise benign pathogens due to a selenium-induced immune dys-function (Fairbrother and Fowles 1990; Whiteley and Yuill 1991; Schamber et al. 1995).

Regulatory Standards

U.S. Environmental Protection Agency Standards and Criteria
[See Appendix II for explanation of terms. Sources: EPA 1995; Federal Register 57(246):60911]

Status	EPA priority pollutant
Drinking water MCL	50 µg/L
Freshwater criteria	20 µg/L for acute exposure 5 µg/L for chronic exposure
1/1,000,000 cancer risk	10 µg/L (water and organisms)

For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water-quality officials in the 17 Western States.

Existing Literature Reviews

Over the last two decades, many publications have surveyed the literature pertaining to the effects of selenium exposure on fish and wildlife populations. Some of these reviews are listed here:

Adams and Johnson 1981
GLSAB 1981
Brooks 1984
Eisler 1985
Lemly 1985a
Lemly and Smith 1987
EPA 1987
Lillebo et al. 1988
UC Committee 1988
DuBowy 1989
Ohlendorf 1989
Beyer 1990
Hodson 1990
SJVDP 1990
USFWS 1990a,b
Skorupa and Ohlendorf 1991
Sorensen 1991
Peterson and Nebeker 1992
CH2M Hill et al. 1993
Emans et al. 1993
Lemly 1993a
CAST 1994
Maier and Knight 1994
Lemly 1995
Albers et al. 1996
Heinz 1996
Lemly 1996a,b,c
Green and Albers 1997
O'Toole and Raisbeck 1997
Van Derveer and Canton 1997
Adams et al. 1998
Hamilton 1998
Lemly 1998a,b
O'Toole and Raisbeck 1998
Skorupa 1998a

In addition, some useful interpretive guidance is found in the more general reviews of selenium chemistry and toxicology by Wilber (1980), Maier et al. (1987), Ogle et al. (1988), USPHS (1989), and Oldfield (1990). The core scientific basis for interpretive guidance is contained in these references. Where other sources are not cited to support statements in this chapter, the papers listed above are the source(s) of information.

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Zinc

Description

Zinc (Zn) is a lustrous white to pale-bluish-gray metal when freshly cut or polished, though it more commonly appears dull gray, owing to a coating of hydrated zinc carbonate that develops after extended exposure to the air. It is a relatively soft metal, having a hardness intermediate between that of gypsum and calcite. It is brittle at normal temperatures but is more malleable under low heat (100–150 °C). It melts at 419 °C and boils at 907 °C.

Pure metallic zinc is rarely seen in nature, for the element is highly reactive and forms a variety of white to pale-colored salts. Most of its compounds are water soluble, though the metal itself is not. Because of its high reactivity, zinc is commonly used as a coating over steel (galvanizing). The steel is thus protected, as corrosive agents preferentially attack the zinc coating. Also, for many centuries, zinc has been alloyed with copper to make brass. The most common zinc ore is sphalerite (ZnS), a yellowish to dark-brown cubic mineral, though some zinc also is produced from smithsonite ($ZnCO_3$) and from hemimorphite ($Zn_4Si_2O_7(OH)_2 \cdot H_2O$).

Occurrence

Mean total zinc contents in surface soil range from 17 to 125 mg/kg, and grand mean zinc for worldwide soils is calculated to be 64 mg/kg (Kabata-Pendias and Pendias 1992). Background concentrations of zinc in soils or sediments seldom exceed 200 mg/kg (Eisler 1993). In fresh water, the concentration is normally less than 40 to 60 µg/L (Taylor et al. 1982, Eisler 1993).

The most important artificial sources of zinc in the environment include electroplaters, smelting and ore processors, mine drainage, domestic and industrial sewage, road surface runoff, corrosion of zinc alloys and galvanized surfaces, and erosion of agricultural soils (Eisler 1993).

Summary of Effects

Zinc is an essential element for all living organisms, but elevated levels of zinc in the environment may be harmful near zinc-contaminated sites. Zinc is bioaccumulated by all organisms, even in areas of low zinc concentrations. Both deficient and excessive amounts cause adverse effects in all species.

Zinc is most harmful to aquatic life during early life stages, in soft water, under conditions of low pH, low alkalinity, low dissolved oxygen, and elevated temperatures (Eisler 1993). In contrast to its toxicity to sensitive aquatic organisms, zinc is relatively nontoxic to birds and mammals, and tissue concentrations are homeostatically controlled (Furness and Rainbow 1990, Eisler 1993).

Although tissue residues are not yet reliable indicators of zinc contamination, zinc poisoning occurs in birds when liver or kidney concentrations exceed 2,100 mg/kg dry weight (dw), and in mammals when levels exceed 274 mg/kg dw in kidney or 465 mg/kg dw in liver (Eisler 1993). In amphibians, the tissue values range from 33 to 150 mg/kg dw at uncontaminated sites (Hall and Mulhern 1984). A summary of biotic effect levels is presented in table 34.

Suter and Mabrey (1994) evaluated a series of toxicological benchmarks for screening

Table 34.—Summary of comprehensive biotic effects of zinc

[Note: Diagnostic levels for toxicity are not well established in *any* animal tissues because the zinc concentrations generally are homeostatically regulated]

Medium	No effect	Level of concern	Toxicity threshold	Comments/Explanation
Water (µg/L)	<30	30–110	110	30 µg/L is lowest chronic value for aquatic life (Suter and Mabrey 1994). Threshold value assumes chronic exposure at hardness of 100 mg/L (as $CaCO_3$).[1]
Sediment (mg/kg dw)	150	150–410	410	From Long et al. (1995); however, sulfides in sediment may reduce Zn toxicity.
Plants (mg/kg dw)	27–150	150–300	>300	Kabata-Pendias and Pendias (1992).
Invertebrates	—	—	—	
Fish (white sucker muscle tissue; mg/kg dw):	—	—	20	From Munkittrick et al. (1991), but this is lower than normal background (88) for whole fish (Schmitt and Brumbaugh 1990).
Birds (mg/kg dw): Eggs Liver/kidney	 50 <210	 — —	 — >2,100	J.P. Skorupa, unpub. data, 1996.
Reptiles/amphibians	—	—	—	
Mammals (mg/kg dw): Kidney Liver	 <210 <210	 — —	 >274 >465	Talmage and Walton (1991).

[1] Zinc toxicity in water is affected not only by hardness but also by factors such as pH, temperature, dissolved oxygen, and alkalinity. Toxic effects may occur in sensitive phytoplankton, invertebrates, or fish life stages at concentrations in the "level of concern" range. In most of the West, hardness of more than 200 mg/L is much more common, and zinc would be less toxic under those conditions.

various contaminants for their potential effects on aquatic biota. In addition to the national ambient water quality (NAWQ) criteria, they provided secondary acute and chronic values, lowest chronic values (including those for fish, daphnids, nondaphnid invertebrates, aquatic plants, and all organisms), test EC20s, sensitive species test EC20s, and population EC20s. The values for water in table 34 are as follows: "No effect" is the lowest chronic value for all organisms; "Toxicity threshold" is the NAWQ chronic criterion (if established) or the secondary chronic value; and "Level of concern" is the range between the two other values.

Field Cases

Many studies have been conducted in recent years to investigate the toxicity of zinc and zinc-copper mixtures in effluents. Finlayson and Verrue (1980) and Finlayson and Ashuckian (1979) conducted long-term and short-term toxicity studies on Chinook salmon and steelhead trout, respectively, in order to estimate "safe" levels of zinc and copper for those species. Harrison and Klaverkamp (1990) also conducted an extensive study on the bioaccumulation of zinc, copper, and other metals in northern pike and white sucker from lakes near a smelter.

Three examples illustrate the potential impacts of zinc-contaminated mine drainage on the aquatic environment:

(1) Acid mine drainage from the Iron Mountain Mine near Redding, California, containing high concentrations of zinc and copper, caused numerous fish kills in the upper Sacramento River (Finlayson and Ashuckian 1979, Finlayson and Verrue 1980). Some of these occurred as far back as the early 1900's, but they became more frequent and more serious following the construction of Shasta Dam in 1944 and Keswick Dam in 1950. Finlayson and Ashuckian (1979) hypothesized that these dams had effectively diminished the "dilution effect" in the Sacramento River. Yet, the out-flows from these dams have also been used to purposely dilute elevated concentrations that are detected at downstream monitoring stations.

(2) Similarly, toxic concentrations of zinc and copper from the Penn Mine area in the Sierra Nevada of California caused sizable fish kills in the lower Mokelumne River Basin (Finlayson and Rectenwald 1978). During a fish kill in the Mokelumne River in 1958, zinc concen-trations of 1.4 milligrams per liter (mg/L) were measured 6.4 kilometers down-stream from the mine.

(3) In Canada, the effects of mixed mining wastes on fish were examined through integrated field sampling of water, sediment, invertebrates, and fish (Munkittrick et al. 1991, Miller et al. 1992). Miller et al. (1992), in particular, made an extensive study of the relationships between concentrations of zinc and copper in all these media in the Manitouwadge chain of lakes in northern Ontario. They found a correlation between zinc concentrations in invertebrates and in sediment but observed no such relationship with water concentrations. Neither did they find any relationship between zinc concentrations in fish tissue and those in invertebrates, although several lab studies had suggested that food and particulates are much more important sources of zinc than water (Patrick and Loutit 1976, Dallinger and Kautzky 1985, as cited in Miller et al. 1992). For both zinc and copper, the water concentration was a better indicator of metal concentration in fish tissue than the sediment or invertebrate concentrations in this field study. Miller et al. (1992) also reported reduced growth in females of white sucker after sexual maturation, decreased egg size and fecundity, no significant increase in fecundity with age, and an increased inci-dence of spawning failure at a waterborne zinc concentration of 156 mg/L and a sediment concentration of 6,397 mg/kg. In addition, they found kidney and liver concentrations to be better indicators of chronic zinc and copper exposure than muscle concentrations.

Abiotic Factors Affecting Bioaccumulation

Water

In natural waters, zinc occurs both in dis-solved form and as suspended particulates. Only the dissolved fraction is believed to be toxic to fish (Finlayson and Verrue 1980). Dissolved zinc assumes several different chemical forms in various inorganic and organic complexes. Zinc is present as Zn^{2+} in acidic waters and $ZnOH^{+}$ in soft waters. According to some studies, zinc is also present as a toxic "aquo ion," $(Zn(H_2O)_6)^{2+}$, almost exclusively in fresh water (Campbell and Stokes 1985). Softer water is also known to increase the toxicity of zinc in fish, and ambient water quality criteria are based on water hardness (EPA 1991, 1992). Most of the zinc introduced into the aquatic environment is eventually deposited in sediments.

Bottom Sediment

Biological effects have not been associated with zinc concentrations of 50 mg/kg (dry weight) or less in sediments, but the available data suggest that sublethal effects may occur at zinc concentrations between 50 and 125 mg/kg (Long and Morgan 1990). Long et al. (1995) identified 150 mg/kg as a safe level for zinc and 410 mg/kg as a concentra-tion above which adverse effects are common. Although many of the data that were evaluated were for estuarine and marine sediments, Hull and Suter (1994) concluded that those screening levels also were appropriate for freshwater sediments until more specific guidelines become available. However, they also recommend that these concentrations be compared to local background levels when possible, and that concentrations within the background range should not be considered a problem.

Acid-volatile sulfides (AVS) in the sediment may combine with a portion of certain metals (Cd, Cu, Ni, Pb, and Zn) and render that portion unavailable and nontoxic to biota (Di Toro et al. 1992). In order to assess the effects of acid-volatile sulfides on metal toxicity, the AVS is extracted from sediment with hydro-chloric acid, and the metal concentration that comes with it is called the simultaneously extracted metal (SEM). All SEMs that would contribute appreciably to the total SEM are measured and totaled (Di Toro et al. 1992). *If* the sediments are not fully oxidized (Adams et al. 1992), then an SEM:AVS ratio <1 indicates that acute toxicity is unlikely. The method has not yet been adapted for chronic toxicity.

Biotic Effects

Zinc concentrations in plants and animals are extremely variable. In plants, the background concentration ranges from 8 to 150 mg/kg (Bodek et al. 1988). In fish, concentrations are normally <700 mg/kg dw (Eisler 1993); based on a nationwide survey of zinc in fish, Schmitt and Brumbaugh (1990) reported a mean concentration of 21.7 mg/kg ww (about 88 mg/kg dw) and an 85th percentile concentration of 34.2 mg/kg ww (136 mg/kg dw). For both birds and mammals, normal tissue zinc concentrations are <210 mg/kg dw (Eisler 1993).

Plants

Sensitive terrestrial plants such as oak and maple seedlings died when soil zinc levels were >100 mg/kg, as shown in table 35. In general, zinc uptake by plants is promoted by low soil pH and is restrained by high soil pH, high clay content, high cation exchange capacity, or a high phosphate level in the soil (Bodek et al. 1988). The general symptoms of zinc toxicity in terrestrial plants are chlorotic and necrotic leaf tips, interveinal chlorosis in new leaves, retarded growth of the entire plant, and injured roots resembling barbed wire (Kabata-Pendias and Pendias 1992). Cereals and spinach are the common crop plants most sensitive to zinc toxicity. The recommended maximum acceptable zinc concentration in soil for terrestrial plants is 70–400 mg/kg, depending on the form of zinc and the soil conditions.

Fish

Significant adverse effects were observed in the most sensitive fish species at a waterborne zinc concentration of 10 mg/L (table 36). When larvae and alevins of rainbow trout (*Oncorhynchus mykiss*) were exposed to 10 µg Zn/L, 54 percent of them died after a 28-day exposure (Spear 1981). Acute 96-h LC50 values for salmon were measured at >1,270 µg/L (Hamilton and Buhl 1990).

Knox et al. (1982) found that rainbow trout can tolerate relatively high dietary concen-trations of zinc. There was no effect on growth or health of rainbow trout when they

Table 35.—Biological effects of zinc in sediment or soil

Species	Zn in sediment/ soil (mg/kg dw)	Zn in biomass (mg/kg dw) and other effects	Comments	Reference
Plants				
Oak (*Quercus rubra*)	100	Lethal to seedlings	Planted in culture medium	Eisler 1993
Red maple (*Acer rubrum*)	100	Lethal to seedlings		
Invertebrates				
Aquatic invertebrates	1,149	Complete absence of Plecoptera, Ephemeroptera, Odonata, Trichoptera, Amphipoda, and Unionidae	Manitouwadge Lake, Ontario, Canada	Munkittrick et al. 1991
Earthworm (*Aporrectodea tuberculata*)	28	320		Eisler 1993
	97	810		
	110	1,300		
	190	1,100		
	320	650		
	470	No worms found		
Fish				
White sucker (*Catostomus commersoni*)	43	Liver 112; muscle 25; stomach contents 7	Loken Lake, Ontario, Canada	Munkittrick et al. 1991
	1,149	Liver 210; muscle 20; stomach contents 886. Lowered growth rate	Manitouwadge Lake, Ontario, Canada	
Mammals				
Field vole (*Microtus agrestis*)	21,000	Whole body 191.6	Pb-Zn mine site	Johnson et al. 1978
	131	Whole body 121.2	Uncontaminated site	Roberts and Johnson 1978
	59	Whole body 100; liver 113; kidney 121	Uncontaminated site	Anderson et al. 1982
Shrew (*Sorex araneus*)	21,000	Whole body 141	Pb-Zn mine site	Johnson et al. 1978
Vole (*Clethrionomys glareolus*)	21,000	Whole body 123.4	Pb-Zn mine site	

were fed a diet containing zinc at a level of 683 mg/kg dw (table 37). Other feeding studies using rainbow trout found no observed effect with dietary concentrations ranging from 440 to 1,700 mg/kg dw, although progressively higher zinc concen-trations were observed in the liver, blood, and gills (Wekell et al. 1983).

Amphibians

Amphibian embryos are known to be more sensitive to zinc than older stages. As shown in table 36, most amphibians show serious adverse effects at waterborne zinc concentra-tions >1,500 µg/L. Amphibians are reported to accumulate zinc more than other species. Compared to concentrations in fish, zinc concentrations in tadpoles were 10 times as high, and those in eviscerated tadpoles were twice as high (Jennett et al. 1977).

Birds

Birds are relatively tolerant to zinc (Puls 1988; Eisler 1993). Ducks (*Anas* spp.) had reduced survival when they consumed 2,500 to 3,000 mg Zn/kg in diets. When the same amount (3,000 mg/kg) was fed to mallards (*Anas platyrhynchos*), they developed diarrhea after 15 days, leg paralysis in 20 days, and high mortality after 30 days. The lowest con-centration of zinc in diet that caused adverse effects in birds was 178 mg/kg. When 178 mg Zn/kg was fed to domestic breeding hens for 3 weeks, it caused immunosuppression of young progeny without affecting growth (Eisler 1993).

Mammals

Mammals also are relatively tolerant to zinc (Puls 1988; Eisler 1993). Most mammals can consume much higher levels of zinc than their normal intakes without showing any deleterious effects. Most studies of zinc in mammals have focused on whole-body concentrations; zinc did not bioconcentrate in liver or kidney tissue. Among the small mammals listed in table 35, the field vole (*Microtus agrestis*) was found to accumulate the most zinc in whole-body concentrations (Talmage and Walton 1991). At a soil concentration of 21,000 mg/kg dw, it accumu-lated a zinc concentration of 191.6 mg/kg dw, significantly higher than the concentrations in control voles. Many studies show that mammals generally can tolerate dietary zinc levels up to 100 times their usual daily requirement for long periods without showing significant adverse effects (Eisler 1993).

Bioaccumulation

Bioconcentration factors (BCFs) of zinc can vary greatly between freshwater species. For insects, the BCF can vary from 107 to 1,130 and for fish from 51 to 432 (EPA 1980).

Interactions

In solution, inorganic oxides and humic substances increase the bioavailability of zinc (EPA 1991, 1992). Moreover, mixtures of zinc and copper are known to be additive or "more-than-additive" in toxicity to many aquatic organisms. Finlayson and Verrue (1980) conducted long-term and short-term toxicity studies on Chinook salmon (*Oncorhynchus tshawytscha*) using various water concentrations of copper and zinc mixture. From the results, they estimated that safe levels of copper and zinc for Chinook salmon would be below 11 and 83 mg/L, respectively. Other metals that are additive to zinc in toxicity are lead and nickel. On the other hand, cadmium is known to be antagonistic to zinc. A low-molecular-weight protein, metallothionein, also plays an important role in the transport, storage, and detoxification of zinc (Hamilton and Mehrle 1986). Metallothionein synthesis is induced when most vertebrates and some plants are

Table 36.—Biological effects of zinc on aquatic species

Species	Zn in water (µg/L)	Effect	Comments	Reference
Plants				
Brown macroalgae (*Fucas serratus*)	9.5	BCF: 10,770 in 140 days	Marine algae	Eisler 1993
Freshwater alga (*Selenastrum capricornutum*)	30	Some growth inhibition in 7 days		Eisler 1993
	40–68	95% growth inhibition in 14 days		
	100	100% growth inhibition in 14 days		
Freshwater algae, most species	>1,000	Growth inhibition		Eisler 1993
Phytoplankton	15	Primary productivity reduced in 14 days		Eisler 1993
Invertebrates				
Cladoceran (*Daphnia magna*) eggs	10,000–50,000	No effect on mortality	46-h exposure. Eggs more tolerant than adults	Bodar et al. 1989
	100,000	Increase in mortality		
Mayfly (*Epeorus latifolium*) larvae	10–30	Decreased growth rate after 2 weeks. Notable increase in mortality at 4 weeks		Hatakeyama 1989
	100	Growth rate 37% of control at 1 week, near 0% at 2 weeks. All died before emergence		
	300	Growth rate 24% of control at 1 week, near 0% at 2 weeks. All died before emergence		
Midge (*Chironomus tentans*) larvae	8,200	48-h EC50 (Effect: immobilization)	Temp. 14°C, pH 6.3	Khangarot and Ray 1989
Mosquito (*Aedes aegypti*) hatched pupae	500	20% increase in mortality	pH 6.1	Abbasi et al. 1985
Snail (*Ancylus fluviatilis*) juvenile	80	100d LC50	Shell length <2 millimeters	Eisler 1993
	130	100d LC50	Shell length >3 millimeters	
Snail (*Ancylus fluviatilis*) adult	100	No adverse effect on reproduction in 100 days		
	180	Reproduction reduced in 100 days		
Snail (*Biomphalaria glabrata*) embryos	500	Survival reduced to 50%		Eisler 1993
Snail (*Biomphalaria glabrata*) adults	500	Growth and reproduction inhibited		

Table 36.—Biological effects of zinc on aquatic species—Continued

Species	Zn in water (µg/L)	Effect	Comments	Reference
Invertebrates—Continued				
Sponge (*Ephydatia fluviatilis*) adults	6.5	No effect on growth; no tolerance developed with long-term exposure		Eisler 1993
	26	After exposure for 10 days, tissue deterioration and death during 3-week post-exposure period		
Fish				
Bluegill (*Lepomis macrochirus*)	1,400	96-h LC50 (Cu present)	Cu = 400 µg/L; temp. 22±1 °C; pH 6.8–7.5	Thompson et al. 1980
	3,200	96-h LC50 (Cu absent)	Cu = 0; other conditions as above	
Chinook salmon (*Oncorhynchus tshawytscha*)	1,270	96-h LC50	In fresh water; mean weight 1.03 g	Hamilton and Buhl 1990
	2,880		In brackish water; mean weight 2.60 g	
	5,530	24-h LC50	In fresh water; mean weight 1.03 g	
	12,600		In brackish water; mean weight 2.60 g	
Chinook salmon (*Oncorhynchus tshawytscha*) eggs to hatchlings	145	28-d LC10s, based on various mixed solutions of Cu and Zn	Zn = 3× dissolved Cu	Finlayson and Verrue 1980
	175		Zn = 3× total Cu	
	224		Zn = 6× dissolved Cu	
	254		Zn = 6× total Cu	
	396		Zn = 11× dissolved Cu	
	437		Zn = 11× total Cu	
Chinook salmon (*Oncorhynchus tshawytscha*) hatchlings to swim-up fry	119	28-d LC50s, based on various mixed solutions of Cu and Zn	Zn = 3× dissolved Cu	Finlayson and Verrue 1980
	145		Zn = 3× total Cu	
	156		Zn = 6× dissolved Cu	
	187		Zn = 6× total Cu	
	208		Zn = 11× dissolved Cu	
	234		Zn = 11× total Cu	

Table 36.—Biological effects of zinc on aquatic species—Continued

Species	Zn in water (µg/L)	Effect	Comments	Reference
Fish—Continued				
Coho salmon (*Oncorhynchus kisutch*), yearling	4,600	96-h LC50	Temp. 10–12°C; hardness 68–78 *or* 89–90 mg/kg (as $CaCO_3$)	Lorz and McPherson 1976
Rainbow trout (Oncorhynchus mykiss)	10	28-d LC54	Larvae and alevins	Spear 1981
	70-140	25-d LC50	Early life stages	
Steelhead trout (*Oncorhynchus mykiss*) eggs to hatchlings	170	60-d LC10s, based on various mixed solutions of Cu, Zn, and Al	Cu:Zn:Al = 1:4:6, dissolved Zn	Finlayson and Aschuckian 1979
	200		Cu:Zn:Al = 1:4:6, total Zn	
	280		Cu:Zn:Al = 1:12:18, dissolved Zn	
	290		Cu:Zn:Al = 1:12:18, total Zn	
Steelhead trout (*Oncorhynchus mykiss*) hatchlings to swim-up fry	140	60-d LC10s, based on various mixed solutions of Cu, Zn, and Al	Cu:Zn:Al = 1:12:18, dissolved Zn	Finlayson and Aschuckian 1979
	140		Cu:Zn:Al = 1:4:6, dissolved Zn	
	170		Cu:Zn:Al = 1:4:6, total Zn	
	180		Cu:Zn:Al = 1:12:18, total Zn	
Amphibians				
South African clawed frog (*Xenopus laevis*)	>1,500	At 96 h, some mid-gut malformations and pericardial edema		Eisler 1993
	2,700	50% malformations in 96 h		
Newt (*Triturus cristatus*)	200–3,000	Newts became lethargic, ate poorly, and had skin darkening before death. Elevated Zn concentrations in kidney, brain, liver, and intestine		Eisler 1993

Table 37.—Summary of exposure-response or exposure-bioaccumulation of zinc

Species	Zn in diet (mg/kg dw)	Zn in biomass (mg/kg dw) and effect when observed	Comments	Reference
Food chain				
Mayfly (*Epeorus latifolium*) larvae	940 (algae)	No change in growth rate; slight increase in mortality		Hatakeyama 1989
	1,380 (algae)	Growth rate decreased to 55% of control within 1 week but rebounded after 2 weeks. Significant increase in mortality; impaired emergence		
	2,170 (algae)	Impaired emergence and growth rate; significant increase in mortality		
Woodlouse (*Porcellio scaber*)	5,000	Survival reduced to 74% of control		Beyer et al. 1984
	20,000	Survival reduced to 34% of control		
Slug *(Arion ater*)	<100	No significant effect		Eisler 1993
	300–1,000	Reduced food consumption at day 27		
	470	Significantly impaired growth		
Fish				
Rainbow trout (*Oncorhynchus mykiss*)	440	Liver 106; blood 171; gill 308. No effect		Wekell et al. 1983
	860	Liver 120; blood 192; gill 522. No effect		
	1,700	Liver 151; blood 244; gill 1,120. No effect		
	683	Liver 24. No effect	20-week exposure. Diet also contained 178 mg/kg Cu	Knox et al. 1982
Birds				
Mallard (*Anas platyrhynchos*)	3,000	Leg paralysis and decreased food consumption	30-day exposure	Eisler 1993
	>3,000	Many deaths		
Peking duck (*Anas platyrhynchos*)	2,500	Progressive ultrastructural degeneration of pancreatic acinar cells evident as early as day 5	56-day exposure	Eisler 1993
Mammals				
Horse (*Equus caballus*)	1,000	2,728–3,511 in liver; caused Cu deficiency		Eisler 1993
	2,000	4,364–4,524 in liver; caused Cu deficiency		
Domestic mouse (*Mus* sp.)	682	No effects		Eisler 1993
	6,820	Reduced survival, growth, and food intake		

chronically or acutely exposed to zinc and other heavy metals. It protects against the ill effects of zinc by sequestering zinc more efficiently.

Regulatory Standards

Standards and criteria established by the U.S. Environmental Protection agency are listed in table 38. For standards and criteria set by State agencies, contact those agencies directly. See Appendix I for a listing of water quality officials in the 17 Western States.

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Table 38.—U.S. Environmental Protection Agency standards and criteria for zinc

(See Appendix II for explanation of terms. Source: EPA, 1985, 1995)

Status	EPA priority pollutant; carcinogencity unknown
Drinking water MCL/MCLG	None established
Secondary MCL	5 mg/L
Drinking-water health advisories for 10-kilogram child	1-day HA: 6 mg/L 10-day HA: 6 mg/L Long-term HA: 3 mg/L
Drinking-water health advisories for 70-kilogram child	Reference dose: 0.3 mg/kg/day Long-term HA: 10 mg/L Lifetime HA: 2 mg/L DWEL: 10 mg/L
Freshwater criteria (hardness dependent)	
At hardness of 50 mg/L $CaCO_3$	65 µg/L for acute exposure 59 µg/L for chronic exposure
At hardness of 100 mg/L $CaCO_3$	120 µg/L for acute exposure 110 µg/L for chronic exposure
At hardness of 200 mg/L $CaCO_3$	210 µg/L for acute exposure 190 µg/L for chronic exposure

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APPENDIX I

Offices Responsible for Water Quality Standards in 17 Western States

State	Clean Water Act—Standards	Safe Drinking Water Act—Standards
Arizona	Mark Charles, Manager Water Quality Compliance Section Dept. Of Environmental Quality 3033 North Central Avenue Phoenix, AZ 85012–2809 (602) 207–4567	Bob Munari, Manager Drinking Water Section Dept. Of Environmental Quality 3033 North Central Avenue Phoenix, AZ 85012–2809 (602) 207–4617
California	Gail Linck, Supervisor Division of Water Quality State Water Resource Control Board P.O. Box 994213 Sacramento, CA 94244–2130 (916) 657–2188 http://www.swrcb.ca.gov	David Spats, Division Chief Division of Drinking Water & Environmental Management State Department of Health Services 601 North 7th Street Sacramento, CA 95814 (916) 322–2308
Colorado	Dennis Anderson, Env. Engineer Water Quality Control Division Department of Public Health & Environment 4300 Cherry Creek Drive South Denver, CO 80222–1530 (303) 692–3571 Dennis.Anderson@state.co.us	Jerry Biberstine State Drinking Water Program Department of Public Health & Environment 4300 Cherry Creek Drive South Denver, CO 80222–1530 (303) 692–3546 Jerry.Biberstein@state.co.us
Idaho	Mark Shumar Water Quality Program Division of Environmental Quality 1410 North Hilton Boise, ID 83706 (208) 373–0120	Lance Nielsen Drinking Water and Wastewater Program Division of Environmental Quality 1410 North Hilton Boise, ID 83706 (208) 373–0409
Kansas	Theresa Hodges Office of Science and Support Kansas Department of Health & Environment Forbes Field, Building 283 Topeka, KS 66620 (916) 296–5572	David Waldo Bureau of Water Kansas Department of Health & Environment Forbes Field, Building 283 Topeka, KS 66620 (916) 296–5503

State	Clean Water Act—Standards	Safe Drinking Water Act—Standards
Montana	Abe Horpestad Resource Protection & Planning Division Department of Environmental Quality Metcalf Building P.O. Box 200901 Helena, MT 59620 (406) 444–2459	John Camden Public Water Supply Specialist Permitting and Compliance Division Department of Environmental Quality Metcalf Building P.O. Box 200901 Helena, MT 59620 (406) 444–4019
Nebraska	John Bender Surface Water Division Department of Environmental Quality 1200 North Street, Suite 400 P.O. Box 98922 Lincoln, NE 68509–8922 (402) 471–4201	Jack Daniel Department of Registration and Licensure Nebraska Health and Human Services P.O. Box 95007 Lincoln, NE 68509 (402) 471–2541
Nevada	Adele Basham Bureau of Water Quality Planning Division of Environmental Protection 333 West Nye Carson City, NV 89706–0851 (702) 687–4670 ext. 3102	Dr. Jonathon C. Palm Bureau of Health Protection Services Nevada State Health Division 505 East King Street, Room 103 Carson City, NV 89710 (702) 687–6615 ext. 229
New Mexico	Ed Kelley Surface Water Quality Bureau New Mexico Environment Department P. O. Box 26110 Santa Fe, NM 87502 (505) 827–0187	Robert Gallegos Drinking Water & Community Services Bureau New Mexico Environment Department P. O. Box 26110 Santa Fe, NM 87502 (505) 827–7536
North Dakota	Mike Ell Surface Water Quality Management Program Department of Health P.O. Box 5520 Bismarck, ND 58506–5520 (701) 328–5210	D. Wayne Kern Municipal Facilities Program Department of Health P.O. Box 5520 Bismarck, ND 58506–5520 (701) 328–5211
Oklahoma	John Dyer Water Quality Division Department of Environmental Quality 1000 NE 10th Street Oklahoma City, OK 73117–1212 (405) 271–5205 ext. 191	Mike Harrell Water Quality Division Department of Environmental Quality 1000 NE 10th Street Oklahoma City, OK 73117–1212 (405) 271–5205 ext. 200

State	Clean Water Act—Standards	Safe Drinking Water Act—Standards
Oregon	Lynne Kennedy, Standards Coordinator Science and Data Section Water Quality Division Department of Environmental Quality 811 SW Sixth Avenue Portland, OR 97204 (503) 229–5371	Patrick Meyer Drinking Water Program Oregon Health Division 800 NE Oregon, Room 611 Portland, OR 97232 (503) 731–4381
South Dakota	Bill Baer, Program Scientist Surface Water Quality Program Department of Environment and Natural Resources 523 East Capitol Avenue Pierre, SD 57501 (605) 773–3351	Darron Busch, Office Administrator Drinking Water Program Department of Environment and Natural Resources 523 East Capitol Avenue Pierre, SD 57501 (605) 773–3754
Texas	Jim Davenport Water Quality Division Texas Natural Resource Conservation Commission 12100 Park 35 Circle Austin, TX 78711–3087 (512) 239–4585	Tony Bennett, Manager Water Utility Division Public Drinking Water Supply Section Texas Natural Resource Conservation Commission 12100 Park 35 Circle Austin, TX 78711–3087 (512) 239–6020
Utah	Dr. William Moellmer Division of Water Quality Department of Environmental Quality 288 North 1460 West P.O. Box 144870 Salt Lake City, UT 84114–4870 (801) 538–6329	Ken Bousfield, Compliance Section Manager Division of Drinking Water Department of Environmental Quality 150 North 1950 West P.O. Box 144830 Salt Lake City, UT 84114–4830 (801) 536–4207
Washington	Mark Hicks, Standards Coordinator Water Quality Standards Department Department of Ecology P.O. Box 47600 Olympia, WA 98504–7600 (360) 407–6477	Jim Hudson Division of Drinking Water Department of Health P.O. Box 47822 Olympia, WA 98504–7822 (360) 753–9674
Wyoming[1]	US EPA Region VIII Water Program 999 18th Street, Suite 500 Denver, CO 80202–2466 (303) 312–6260	US EPA Region VIII Water Program 999 18th Street, Suite 500 Denver, CO 80202–2466 (303) 312–6120

[1] The State of Wyoming does not administer the Clean Water Act or the Safe Drinking Water Act. The Environmental Protection Agency Region VIII in Denver, Colorado, has jurisdiction.

A II

Glossary of Abbreviations and Technical Terms

A

abiotic: Not involving living organisms or life processes.

absorption: Taking in of fluids or other substances by cells or tissues (cf. *adsorption*).

acclimation: Physiological and behavioral adjustments of an organism to changes in its environment (especially changes that occur within single individuals and single generations; cf. *adaptation*).

action levels: Limits established by the U.S. Food and Drug Administration (FDA) to control levels of contaminants in human food and animal feed. The FDA will take legal action to remove products from the market if they exceed these limits.

acute exposure: A single exposure to a toxic substance which results in severe biological harm. Acute exposures are usually characterized as lasting no longer than a day.

adaptation: Changes in an organism's structure or habits that help it adjust to its surroundings (especially changes that occur through natural selection over multiple generations; cf. *acclimation*).

adipose: Fatty; of or relating to fat.

adsorption: The adherence of a gas, liquid, or dissolved material on the surface of a solid. Should not be confused with absorption.

aerobic: Living or existing in the presence of oxygen.

albumen: The white of an egg.

alevin: A young fish which has not yet absorbed its yolk sac.

algicide: A chemical that kills algae.

amalgam: An alloy of mercury.

ambient: Surrounding natural conditions (or environment) in a given place and time.

amphipod: A small crustacean of the order Amphipoda, such as a beach flea. Amphipods lack eye stalks, have little or no shell, and have laterally compressed bodies.

anadromous: Fish that migrate from salt water to freshwater to breed.

anaerobic: Living or existing in the absence of oxygen.

anatid: Any member of the Anatidae, an order of waterfowl that includes ducks, geese, mergansers, and swans.

Anseriformes: An order of waterfowl comprising the ducks, geese, swans, mergansers, and screamers.

antagonism: Interference or inhibition of the effect of one chemical by the action of another chemical.

aquo ion: An ion that incorporates water molecules. Most metallic ions, if dissolved in water, form a complex with six water molecules, resulting in an aquo ion with the same charge as the free metallic ion. Pb^{2+}, for instance, is really $[Pb(H_2O)_6]^{2+}$.

Arochlor: A clear, colorless to pale-yellow, viscous organic liquid consisting of a mixture of polychlorinated biphenyls (PCBs). It is widely used in electrical components, vacuum pumps, gas-transmission turbines, heat- exchange fluids, coatings, inks, insecticides, fillers, adhesives, paints, and duplicating papers. It is a common environmental contaminant.

arsenical: (1) Containing or relating to arsenic. (2) An arsenic compound.

arthropod: Any member of the phylum Arthropoda, which includes insects, crustaceans, arachnids, millipedes, and centipedes.

articular gout: Swelling around the joints caused by accumulation of urates (uric acid salts).

As: Chemical symbol for arsenic.

athalassohaline: Refers to a nonmarine saline environment. Nonmarine salinity is far more variable than the salinity in seawater, but it generally includes much less sodium chloride and higher proportions of other salts, such as calcium and magnesium sulfides.

avian: In, of, or related to birds.

AVS: Acid-volatile sulfide. Sulfide ions in sediment that are extractable by cold hydrochloric acid. These form insoluble and, hence, nontoxic compounds with metals. Therefore, metal toxicity is reduced, ion for ion, by the amount of AVS in the sediment.

AWQC: Ambient water quality criteria (established by U.S. Environmental Protection Agency).

B

B: Chemical symbol for boron.

basophilic granules: Bodies from a cell's cytoplasm that take up basic dyes when stained in preparation for microscopic examination. The dyes give them a blue, gray, or bluish-gray color under the microscopic.

BCF: Bioconcentration factor, the concentration of a chemical in biota (e.g., earthworm) divided by the concentration of the chemical in media (e.g., soil).

benthic organism: Plant or animal that lives on or near the bottom of a stream, lake, or sea.

bioaccumulation: General term for the uptake and storage of chemical constituents by plants and animals.

bioassay: Determination of the potency of a chemical by comparing its effect on an organism to the effect of a standard preparation on the same type of organism
(cf. *toxicity test*).

bioavailability: The extent to which a substance is capable of entering into biological metabolism.

bioconcentration: The accumulation of a chemical in tissues of an organism (such as fish) to levels that are greater than the level in the organism's food or in its environment.

biomagnification: The cumulative result of bioconcentration as a chemical passes up the food chain, becoming progressively more concentrated at each trophic level.

biomonitor: An organism sensitive to changes in water quality, which is kept under observation in order to detect such changes promptly.

biota: Plant and animal life.

biotransformation: The chemical alteration of a compound caused by enzymatic activity within an organism.

bivalent: Capable of forming single bonds with two other atoms or a double bond with one other atom.

bw: Body weight.

C

C: Chemical symbol for carbon.

Ca: Chemical symbol for calcium.

cachexia: Severe weight loss and general wasting of the body.

calcium carbonate ($CaCO_3$) equivalent: An expression of the concentration of specified constituents in water in terms of their equivalent value to calcium carbonate. For example, water hardness is usually described as calcium carbonate equivalent.

carcinogen: Any substance that tends to produce cancer in an organism.

centrarchid: Any fish from the family Centrarchidae, which includes the freshwater or black basses and several sunfishes.

cerebellum: A part of the brain lying below the cerebrum, consisting of three lobes and concerned with muscular coordination and maintenance of equilibrium.

Charadriformes: An order of birds including the auks, gulls, snipes, and other shorebirds.

chelation: Process by which a metallic ion bonds to a complex molecule at two different points, which creates a ring structure and greatly reduces the chemical reactivity of the metallic ion but greatly increases its solubility.

chironomid: Members of the family Chironomidae (the true midges), two-winged flying insects which live their larval stage underwater.

chlorohydrocarbons: A class of persistent, broad-spectrum insecticides that linger in the environment and accumulate in the food chain. Among them are DDT, aldrin, dieldrin, heptachlor, chlordane, lindane, endrin, mirex, hexachloride, and toxaphene.

chlorosis: Yellowing of leaves and stems of green plants, caused by a loss of chlorophyll and other green pigments.

chronic exposure: Long-term, low-level exposure to a toxic chemical.

Cl: Chemical symbol for chlorine.

Cladocera: An order of small, freshwater branchiopod crustaceans having a transparent bivalve shell. Also called "water fleas."

conductance: The capacity of a medium to carry an electrical current. In water, conductance is directly related to the concentration of ionized substances and, hence, a conductance test serves as a rapid method of estimating the water's dissolved solids content (salinity).

contaminant: Any physical, chemical, biological, or radiological substance or matter that has an adverse effect on air, water, soil, or living things.

criterion (plural: criteria): An estimate of the concentration of a chemical or the magnitude of a physical property that will preserve an organism or group of organisms from harm if not exceeded. Criteria are not standards but may serve as the basis for standards. Under the Clean Water Act, each State is required to set up enforceable water quality standards that are at least as protective as the Environmental Protection Agency's criteria.

crust (of Earth): Rocks and soil close to the surface of the Earth. The crust is generally considered to extend 20–50 kilometers below the continents and 5–10 kilometers below the ocean floor.

crustal abundance: Estimated average abundance of an element in the crust of the Earth.

Cu: Chemical symbol for copper.

cyanobacteria: Blue-green algae.

cytoplasm: All of the fluids and microscopic bodies (organelles) that make up the contents of a living cell, except for the nucleus.

cytoplasmic oxyphilia: A condition of living cells in which the cytoplasm contains an unusual excess of bodies that take up acidic dyes when stained in preparation for microscopic examination.

D

d: Days.

daphnid: A water flea, particularly one of *Daphnia* or a related genus. A cladoceran.

DDD: A common metabolite of DDT. Chemical name: dichlorodiphenyl-dichloroethane or 2,2-*bis*(*p*-chlorophenyl)-1, 1-dichloroethane. CAS No. 72–54–8.

DDE: A common metabolite of DDT. Chemical name: dichlorodiphenyl-dichloroethene or 2,2-*bis*(*p*-chlorophenyl)-1, 1-dichloroethene, CAS No. 72–55–9.

DDT: The first chlorinated hydrocarbon insecticide banned in the United States since 1972 because of its persistence in the environment and its accumulation in the food chain. Chemical name: dichlorodiphenyl-trichloroethane or 2,2-*bis*(*p*-chlorophenyl)-1,1,1-trichloroethane. CAS No. 50–29–3.

defoliant: An herbicide that removes leaves from trees and growing plants.

demethylation: Loss or removal of the methyl group (CH_3) from a methylated compound.

demyelination: Destruction or loss of myelin—a soft, white fatty substance that forms a sheath around certain nerve fibers.

dermatosis: General term for a disease of the skin.

dicofol: Organochlorine pesticide that often has a trace of DDT contamination. Commonly used on citrus fruits and cotton. Also known as Kelthane®. Chemical name: dichlorodiphenyltrichloroethanol.

dicotyledon: Any plant of the class Dicotyledonae (or Magnoliopsida), which includes most broad-leafed flowering plants and trees.

dry weight: Weight determined after water and other volatile liquids have been driven off by heating or by prolonged exposure to dry air.

dw: Dry weight.

DWEL: Drinking water equivalent level—a lifetime exposure concentration estimated to be safe from all toxic effects other than cancer. Calculated based on the assumption that all exposure to a contaminant is from drinking water.

E

EC50: Median effective concentration; estimated concentration at which 50 percent of exposed specimens exhibit a particular effect. Similarly, 'EC ' may be used with other percentages to indicate concentrations for which the affected population is equal to those percentages.

ecosystem: Complex system composed of a community of animals and plants as well as the chemical and physical environment.

ecotoxicity: The harmful effect a substance may inflict on any part of an ecosystem.

ED50: Median effective dose; the dose estimated to produce a particular effect in 50 percent of the test specimens. Similarly, 'ED ' may be used with other percentages to indicate doses for which the affected population is equal to those percentages.

effluent: An outflow, especially the partially or completely treated wastewater flowing out of a treatment facility, reservoir, or basin.

embryopathic: Having to do with malformations that are congenital but not necessarily hereditary.

emergent plants: Plants that are rooted underwater but rise above the water surface (e.g., cattails).

endrin: A pesticide toxic to freshwater and marine aquatic life that produces adverse health effects in domestic water supplies.

EPA: U.S. Environmental Protection Agency.

Ephemeroptera: An order of the class Insecta that includes all mayflies.

ERL: 'Effects range-low ' value of Long et al. (1995). Level at which 10 percent of test specimens show adverse effects.

ERM: 'Effects range-median ' value of Long et al. (1995). Level at which 50 percent of test specimens show adverse effects.

estuarine: In, of, or relating to an estuary.

estuary: A river mouth, particularly that portion subject to tidal action and fluctuating salinity.

eutrophication: The increase in the nutrient levels of a lake or other body of water; this usually promotes greater growth of aquatic animal and plant life.

evapotranspiration: The process by which water in the soil is drawn up through the roots of plants and then is transpired through the plants' leaves into the atmosphere.

F

Falconiformes: An order of birds which includes all raptors except owls.

fauna: Animal life.

fecundity: Fertility; the capacity for producing offspring in abundance.

fingerling: (1) A young or small fish no larger than a human finger. (2) A life stage of fish from 2 weeks after absorption of the yolk sac up to 1 year of age.

fledging: Growing feathers.

floating-leaf plants: Plants that are rooted in the sediment on the bottom of a water body but have leaves that float on the surface (e.g., water lilies).

flora: Plant life.

fresh weight: Wet weight, as determined in the field or shortly after sample collection.

fry: Life stage of fish between the egg and fingerling stages. Depending on the species of fish, fry can measure from a few millimeters to a few centimeters.

fungicide: Pesticides used to control, deter, or destroy fungi.

fw: Fresh weight.

fww: Fresh wet weight.

G

g: Grams.

gastroschisis: An opening or fissure in the ventral wall of the abdomen.

germinal tissue: Specialized reproductive tissue, such as egg or sperm cells.

H

H: Chemical symbol for hydrogen.

h: Hours.

HA: A Drinking Water Health Advisory issued by the Environmental Protection Agency: a non-regulatory health-based reference level of chemical traces in drinking water at which there are no adverse health risks when ingested over various periods of time. (See separate definitions for 1-day, 10-day, long-term, and lifetime HAs.) HAs incorporate a large margin of safety.

half-life: The length of time required for the mass, concentration, or activity of a chemical or physical agent to be reduced by one-half.

hardness, water: The concentration in water of various mineral salts, primarily carbonates, bicarbonates, sulfates, chlorides, and nitrates of calcium and magnesium. Usually expressed as *calcium carbonate equivalent*. Hardness reduces the toxicity of copper, zinc, and some other contaminants.

hectare: A measure of area in the metric system similar to an acre. One hectare is equal to 10,000 square meters or 2.4711 acres.

hepatic: In, of, or related to the liver.

hepatocellular hypertrophy: Swelling of the cells of the liver.

herbicide: A chemical pesticide designed to control or destroy plants, weeds, or grasses.

Hg: Chemical symbol for mercury.

histopathological: Pertaining to or caused by diseases of bodily tissues.

homeostatic: Regulated internally as part of an animal's metabolism.

hydrosoil: Nutrient-rich pore water within the bottom sediment of a water body.

I

IC50: Inhibitory concentration estimated to reduce the normal response of an organism by 50 percent. Only quantifiable responses, such as growth rates, are expressed as IC values. Other percentage values may be used with "IC" to indicate greater or lesser amounts of inhibition.

immunotoxicity: Harmful effect of a substance to the immune system. Immunotoxins cause increased susceptibility to diseases.

infauna: Aquatic animals that live within rather than on the bottom sediment.

inorganic: Chemical substances of mineral origin, not of basically carbon structure.

insecticide: A compound specifically used to kill or prevent the growth of insects.

insectivore: An animal that subsists primarily on a diet of insects.

instar: Life stage of an insect or other arthropod between one molt and the next.

invertebrates: All animals that lack a vertebral column (e.g. insects, crustaceans, mollusks, worms).

ion: An atom or molecule that has acquired a net electrical charge through gaining or losing electrons.

isocaloric: Providing the same number of calories.

isomers: Chemical compounds that have identical atomic compositions but differ in structure.

K

keratin: A tough, fibrous protein material, somewhat softer than bone, that makes up many semi-hard body parts, such as horns, nails, and feathers.

keratinization: Growth of keratin on skin or other soft tissues.

kg: Kilograms.

L

L: Liter, 1,000 cubic centimeters. (1 L of fresh water normally weighs almost exactly 1 kilogram, which makes it possible to relate weight-per-volume measurements, such as micrograms per liter,to weight-per-weight measurements, such as micrograms per kilogram or parts per billion.)

larid: Any bird of the family Laridae, which includes gulls and terns, and is sometimes considered to include jaegers.

LC50: Median lethal concentration; estimated concentration at which 50 percent of exposed specimens would die. Similarly, "LC " may be used with other percentages to indicate concentrations that produce mortality rates equal to those percentages.

LD50: Median lethal dose; estimated dose sufficient to cause death to 50 percent of exposed specimens. Similarly, "LD " may be used with other percentages to indicate doses that produce mortality rates equal to those percentages.

leachate: The ground-water solution that results from leaching.

leaching: The dissolution and removal of soluble constituents from soil by water percolating through the soil.

legume: Any plant of the family Legumi-nosae, which includes peas, beans, clover, alfalfa, and other plants having seeds in bilaterally symmetrical pods.

lesion: Any alteration of tissue caused by injury or disease.

level of concern: Concentration of a toxic substance above which adverse effects may result from even brief exposure.

lifetime exposure: Total amount of exposure to a substance that a human would receive in a lifetime (usually assumed to be 70 years).

lifetime HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects over a lifetime of exposure, with a margin of safety.

lipids: Fats, fatty acids, waxes, and similar long-chain organic compounds (e.g., alcohols, amines, aldehydes), which make up a large proportion of every living cell.

lipophilic: Having an affinity for fats or other lipids. Also, promoting the dissolution of lipids.

LOAEL Lowest-observed-adverse-effect level; the lowest dose in an experiment which produced an observable adverse effect.

long-term HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcino-genic effects up to approximately 7 years
(10 percent of an individual's lifetime) of exposure, with a margin of safety.

lordosis: Exaggerated forward curvature of the spine.

M

m: Meters.

macroalgae: Free-floating water plants that are large enough to be seen with the naked eye.

macroinvertebrate: An invertebrate large enough to be seen with the naked eye.

macrophyte: A plant large enough to be seen with the naked eye.

MATC: Maximum allowable toxicant concentration.

MCL: Maximum contaminant level—the maximum permissible level of a contaminant in water delivered to any user of a public water system. MCLs are enforceable standards.

MCLG: Maximum contaminant level *goal*—the maximum level of a contaminant in drinking water at which no adverse health effects would be anticipated. MCLGs are nonenforceable health goals.

MD25: Dose or concentration that results in an adverse effect other than death to 25 percent of the test population. Similarly, MD may be used with other percentages to indicate doses that produce adverse effects in larger or smaller population segments.

MeHg: Methylmercury.

meninges: Membranes that cover the brain and spinal cord.

metacarpals: Five major bones in the upper part of the forefoot or the hand. (In humans, the metacarpals diverge outward from the "heel" of the hand toward each of the digits.)

mercurial: (1) Containing or related to mercury. (2) A mercury compound.

mercuriferous: Containing mercury.

mesic: Having moderate amounts of moisture; intermediate between humid and arid.

metabolism: The sum of the chemical reactions occurring within a cell or a whole organism; includes the energy-releasing breakdown of molecules (catabolism) and the synthesis of new molecules (anabolism).

metabolite: Any product of metabolism, especially a transformed chemical.

metalloid: A nonmetallic element that has some of the chemical properties of a metal.

metallothionein: A low-molecular-weight protein that binds and detoxifies divalent heavy metals, such as mercury, copper, and zinc. Some plants and animals synthesize metallothionein when exposed to heavy metals.

methylation: A chemical or biochemical reaction that adds a methyl group (CH_3) to an element or molecule.

methylmercury: An organic ion (CH_3Hg^+) that represents the most toxic form of mercury generally found in the environment. Methylmercury enters into metabolism more readily than any of the inorganic forms.

mg: Milligrams.

mg/kg: Milligrams per kilogram, equivalent to parts per million.

mg/L: Milligrams per liter, essentially equivalent to parts per million in water.

microinvertebrate: An invertebrate too small to be seen by the naked eye.

micromelia: A condition characterized by undersized and/or deformed extremities.

micronutrients: Elements and compounds required by living organisms only in minute amounts.

mine tailings: Residue of low-grade material and waste discarded during the processing of mineral ores.

Mo: Chemical symbol for molybdenum.

molybdenosis: A disease of cattle and sheep caused by an excess of molybdenum in forage plants, which leads to copper deficiency because of molybdenum's ability to sequester copper. Common symptoms include loss of pigmentation in the hair or wool, diarrhea, anemia, poor growth, anorexia, and deformed offspring.

monocotyledon: Any plant of the class Monocotyledonae (or Liliopsida), which includes all of the grasses, lilies, orchids, and other flowering plants with narrow, parallel-veined leaves.

monoclinic: Describes mineral crystals having three axes of unequal length, two of which are perpendicular to the third but not perpendicular to each other.

monogastric: Having a single, undivided stomach (cf. *ruminant*).

mutagen: A chemical that induces genetic mutations.

N

NAWQ: National ambient water quality criteria, issued by the Environmental Protection Agency.

necrosis: Death of cells or tissue. May result in spots or small discolored areas on leaves of a plant or on the skin of an animal.

necrotic: Affected by necrosis.

neuron: A nerve or brain cell.

ng: Nanogram (one-billionth of a gram).

NOAEL: No-observed-adverse-effect level—the highest tested concentration at which no adverse effect was observed.

O

O: Chemical symbol for oxygen.

octanol-water partition coefficient: The ratio of a chemical's solubility in *n*-octanol ($C_8H_{17}OH$) to its solubility in water. Symbol: K_{ow}. The ratio indicates the chemical's propensity for bioconcentration by aquatic organisms.

Odonata: An order of the class Insecta that includes all dragonflies and damselflies.

oligochaete: Worms of the class Oligochaeta, including the common earthworms.

omnivore: Animal that eats both vegetable and animal substances.

one-day HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects for up to 5 consecutive days of exposure, with a margin of safety.

organic: In general, produced by or having to do with living organisms. In chemistry, refers specifically to carbon and its many compounds.

organochlorine: Refers to any organic compound containing one or more chlorine atoms.

organomercurial: An organic compound containing mercury.

organophosphate: An organic compound containing phosphorus and oxygen in combination. Organophosphate pesticides are short-lived, but some can be toxic when first applied.

orthorhombic: Describes mineral crystals having three mutually perpendicular axes of symmetry, all of different lengths.

ovalbumin: The white of an egg.

ovoglobulin: Non-water-soluble simple proteins found in the yolk of an egg.

oxidation state: The effective ionic charge of an atom or compound which determines, among other things, which other atoms it may combine with and in what proportions.

P

particulates: Very small solid particles suspended in water. They vary in size, shape, density, and electrical charge and can be gathered together by coagulation and flocculation.

periphyton: Organisms that live attached to underwater surfaces.

pH: A measurement of the acidity or alkalinity, based on the inverse logarithm of the concentration of hydrogen ions. On a scale from 0 to 14, 7 is considered exactly neutral, lower values are increasingly acidic, and higher values are increasingly alkaline or basic.

phytoplankton: The portion of the plankton community that consists of tiny plants, such as green algae and diatoms.

piscivore: An animal that subsists primarily on fish.

planarian: A type of flatworm common in aquatic environments.

plankton: Free-floating plants and animals that live near the surface of a body of water

Plecoptera: An order of insects commonly known as stoneflies.

p*M*: Picomoles, one-trillionth of a mole. One p*M* . 6.02×10^{11} molecules.

porphyry deposit: A low-grade mineral deposit in which the commodity of interest is concentrated in isolated crystals dispersed within a fine-grained rock.

prepupation: The stage in an insect's life cycle prior to formation of a pupa (such as a cocoon or chrysalis). Basically, the larval stage.

Procellariformes: An order of sea birds including the albatrosses, petrels, and shearwaters.

ppt: Parts per thousand.

R

rallid: Any member of the Rallidae, a family of birds that includes the rails and coots.

raptor: A bird of prey.

recurvirostrid: Any member of the Recurvirostridae, a family of birds that includes the stilts and avocets.

RfD: Reference dose—an estimate of a daily exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime.

riparian: Of, on, or pertaining to the bank of a river, pond, or lake.

riverine: Riparian; pertaining to a riverbank.

rumen: The first chamber in the stomach of a ruminant.

ruminant A mammal of the suborder Ruminantia, having a stomach subdivided into multiple (usually four) compartments, which chews a cud consisting of regurgitated, partially digested food. Ruminants are invariably hoofed, even-toed grazing animals, and most grow horns. Examples include cattle, sheep, goats, deer, camels, and giraffes.

S

salinity: A measure of the concentration of dissolved mineral substances in water.

salmonid: Any fish belonging to the family Salmonidae, which includes trout, salmon, whitefish, and grayling.

salt gland: A compound tubular gland which secretes a watery, highly saline fluid. Located near the eyes and nasal passages of certain birds, snakes, and marine turtles.

Se: Chemical symbol for selenium.

secondary maximum contaminant level: Nonenforceable Environmental Protection Ageny-recommended maximum concentra-tion of a drinking water contaminant, based on considerations of odor, taste, and appearance, rather than any finding of toxicity

seed dressing: Any of various types of chemical coatings applied to seeds.
Fungicides and insecticides are most common.

seleniferous: Containing selenium.

selenosis: Selenium poisoning.

sink: Any component in the natural cycle of an element or compound which takes that element or compound out of circulation.

slimicide: A chemical that prevents the growth of slime in paper stock.

smolt: A young salmon at the stage at which it migrates from freshwater to the sea.

solubilization: A process that makes a substance soluble or increases its solubility.

sorption: A general term referring to either absorption, adsorption, or a combination of the two.

standard: Legally enforceable limit on a constituent or condition (e.g., pH, cloudiness) in water or some other medium

Storage ratio: See BCF.

Strigiformes: An order of birds consisting of the owls.

sublethal: Having an effect less severe than death.

Superfund: Program administered by the Environmental Protection Agency that identifies high-priority pollution sites and oversees cleanup at such sites.

supraorbital: Situated or occurring above the eye socket.

swim-up fry: The stage at which young fish are able to swim up from the gravel bed in which they hatched.

synergism: An interaction of two or more chemicals which results in an effect that is greater than the sum of their effects taken independently.

T

TDS: Total dissolved solids—total amount of dissolved material contained in water. Depending on analytical method, this may include only inorganic material or both organic and inorganic material.

Technical grade DDT: A mixture of DDT isomers, especially *p,p'*-DDT and *o,p'*-DDT; it is a cream-colored to gray powder with a faint fruitlike odor.

Ten-day HA: The concentration of a chemical in drinking water that is not expected to cause any adverse noncarcinogenic effects up to 14 consecutive days of exposure, with a margin of safety.

teratogen: A chemical that causes congenital malformations.

teratogenesis: The induction of congenital malformations (birth defects) in a developing fetus by a toxin acting inside the egg or the womb; interference with normal embryonic development.

threshold: The lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

thymus: A gland in the neck or chest of all vertebrates, most prominent in the early stages of life, which plays a role in regulating the supply of white blood cells.

tile drainage: Water drained from beneath a field by a system of pipes. (Originally, nearly all the pipes were tile, though now various materials are used.)

tillering: Production of new shoots (especially in grasses).

total DDT: DDT plus all metabolites.

toxicity test: A measure of the degree of response of an organism exposed to a particular concentration of a chemical or a particular level of some other environmental variable.

toxicosis: A pathological condition caused by a toxin.

Trichoptera: An aquatic order of insects commonly known as caddis flies.

trigonal: Describes mineral crystals having threefold symmetry.

trophic position: The position of a plant or animal in the food chain.

turbidity: The cloudy appearance of water caused by suspended and colloidal matter. Technically, an optical property of the water based on the amount of scattering of polarized light by suspended particles.

U

ungulate: Generally, any hoofed mammal.

Unionidae: The freshwater mollusks; a family of bivalve mollusks.

V

valence: The capacity of an atom or molecule to combine in specific proportions with other atoms or molecules.

vector: An organism, often an insect or rodent, that carries disease.

visceral: Of or having to do with the abdominal organs.

volatile: Readily vaporizable at a relatively low temperature.

volatilization: The transformation of a substance into a volatile form.

wet weight: Weight determined through techniques that preserve and measure all the moisture in the original sample.

ww: Wet weight.

Z

Zn: Chemical symbol for zinc.

zooplankton: The portion of the plankton community that consists of tiny animals.

Greek Letter µ

µg: Microgram.

µg/L: Micrograms per liter, essentially equivalent to parts per billion in water.

µS: Microsiemens, a measure of conductivity (1 µS = 1 micromho). Conductivity is expressed as the inverse of electrical resistance; hence 1 µS corresponds to 1 million ohms.

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