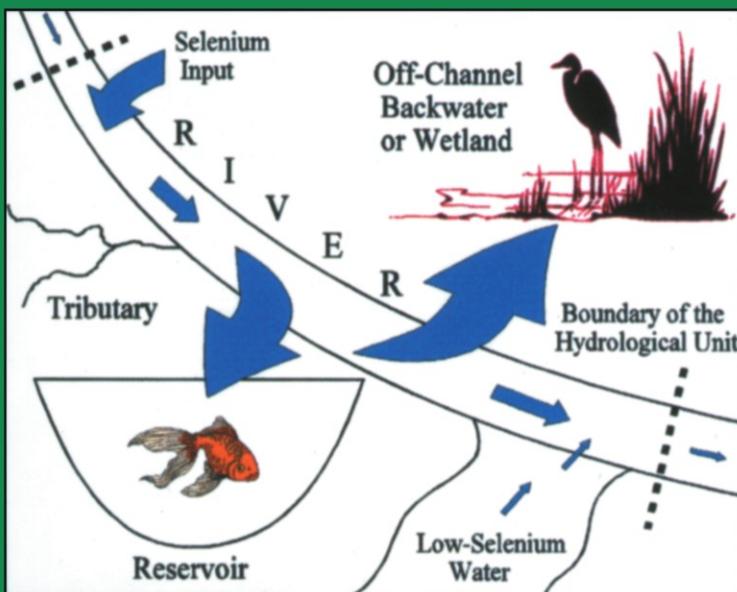


Selenium Assessment in Aquatic Ecosystems

A Guide for Hazard Evaluation and
Water Quality Criteria

A. Dennis Lemly



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A. Dennis Lemly

US Forest Service

Southern Research Station

Coldwater Fisheries Research Unit

Blacksburg, Virginia

USA

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A. Dennis Lemly
US Forest Service
Southern Research Station
Coldwater Fisheries Research Unit
1650 Rambie Road
Blacksburg, VA 24060
USA
dlemly@vt.edu

Series Editor:
David E. Alexander
Department of Geology and Geography
University of Massachusetts
Amherst, MA 01003
USA

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This book is dedicated to the environmental contaminant specialists, field biologists, scientists, and natural resource managers who, despite formidable challenges from opposing interests, have steadfastly toiled to shine the light of truth on selenium pollution issues in order to gain protection for aquatic habitats.

Series Preface

This series is concerned with humanity's stewardship of the environment, our use of natural resources, and the ways in which we can mitigate environmental hazards and reduce risks. Thus it is concerned with applied ecology in the widest sense of the term, in theory and in practice, and above all in the marriage of sound principles with pragmatic innovation. It focuses on the definition and monitoring of environmental problems and the search for solutions to them at scales that vary from the global to the local according to the scope of analysis. No particular academic discipline dominates the series, for environmental problems are interdisciplinary almost by definition. Hence a wide variety of specialties are represented, from oceanography to economics, sociology to silviculture, toxicology to policy studies.

In the modern world, increasing rates of resource use, population growth, and armed conflict have tended to magnify and complicate environmental problems that were already difficult to solve a century ago. Moreover, attempts to modify nature for the benefit of humankind have often had unintended consequences, especially in the disruption of natural equilibria. Yet, at the same time, human ingenuity has been brought to bear in developing a new range of sophisticated and powerful techniques for solving environmental problems, for example, pollution monitoring, restoration ecology, landscape planning, risk management, and impact assessment. Books in this series will shed light on the problems of the modern environment and contribute to the further development of the solutions. They will contribute to the immense effort by ecologists of all persuasions to nurture an environment that is both stable and productive.

*David E. Alexander
Amherst, Massachusetts*

Preface

Today's natural resource managers must respond to a wide array of challenges that threaten to disrupt the biological integrity and sustainability of aquatic ecosystems. Nowhere is this more evident than in the challenges brought on by environmental contaminants. Notable among these pollutants is selenium, a naturally occurring trace element that can be concentrated and released in the waste materials from certain agricultural, petrochemical, mining, and industrial activities. Once in the aquatic environment, selenium can rapidly bioaccumulate and reach levels that are toxic to fish and wildlife. Over the past 20 years this threat has become a reality at several locations across the United States. Two examples are Belews Lake, NC, where 19 species of fish were eliminated due to selenium discharged from a coal-fired power plant, and Kesterson National Wildlife Refuge, CA, where thousands of migratory waterbirds were poisoned by selenium in agricultural irrigation drainage. During the last decade, cases of selenium contamination have emerged on a global scale. Argentina, Australia, Canada, China, Egypt, Greece, India, and Russia are just a few of the locations where aquatic habitats have been impacted.

This escalation in selenium issues has prompted increased efforts to identify and characterize toxic hazards and to develop water quality criteria. The success of these efforts depends on having technically sound, selenium-specific procedures and methods that can be applied across a wide range of habitat types and environmental conditions. I have spent the past 20 years engaged in the development and testing of methods and guidelines for selenium assessment, beginning with field studies that led to new US water quality criteria for selenium in the mid-1980s, and culminating with the recent completion of a procedure for setting aquatic ecosystem loading limits. The interest in this information is growing rapidly. During the past 3 years, I have received over 500 requests for assistance on a variety of selenium issues from 64 countries around the world. Until now, I have only been able to provide assistance on a piecemeal basis, since no single publication

has covered all of my hazard assessment and water quality procedures for selenium. This book brings the information together into a comprehensive guide for characterizing hazards and deriving water quality criteria. It is intended for all those involved in the evaluation and resolution of aquatic selenium problems—from field biologists, environmental contaminant specialists, and research scientists to risk assessors, environmental planners, natural resource managers, and water quality regulators. In addition to the core material on hazard assessment and water quality, there is an opening chapter that describes the primary causes of selenium pollution and gives a sample of locations where fish and wildlife populations have been affected, and a closing chapter which discusses emerging selenium contamination issues that biologists and aquatic resource managers need to be aware of. Although some of the chapters have a context of the United States and its Environmental Protection Agency, the guidelines and methods in the book are not limited to that context. They can be applied just as well in Australia, China, and Egypt. The underlying processes that must be addressed for accurate hazard assessment and development of water quality criteria are consistent around the world. I welcome feedback from those using the book to aid in gauging its effectiveness and evaluating the need to modify a procedure or develop new techniques.

As society moves forward in the new millennium, there will be a substantial increase in activities that mobilize selenium in the environment. Protecting aquatic ecosystems from these threats will be more difficult, yet more necessary, than ever before. Equipped with the proper assessment techniques, investigators can identify, diagnose, and respond to selenium problems before they become widespread toxic episodes. I hope this book will be a useful tool in those efforts, and will provide the technical guidance necessary to make environmentally sound resource management decisions for aquatic habitats.

*A. Dennis Lemly
Blacksburg, Virginia*

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Section I

Selenium Sources, Concentrations, and Biological Effects

1

Selenium Pollution Around the World

Introduction

Selenium is a naturally occurring trace element that can be concentrated and released in the waste materials from certain mining, agricultural, petrochemical, and industrial manufacturing operations. Once in the aquatic environment, it can rapidly bioaccumulate and reach levels that are toxic to fish and wildlife. Selenium pollution is a worldwide phenomenon and is associated with a broad spectrum of human activities ranging from the most basic agricultural practices to the most high-tech industrial processes. Consequently, selenium contamination of aquatic habitats can take place in urban, suburban, and rural settings alike—from mountains to plains, from deserts to rainforests, and from the Arctic to the tropics. Few environmental contaminants have the potential to affect aquatic resources on such a broad scale, and still fewer exhibit the complex aquatic cycling pathways and range of toxic effects that are characteristic of selenium. This places additional importance on the need for techniques to identify, diagnose, and respond to aquatic selenium problems before they become widespread toxic episodes. For many years, selenium has been a largely unrecognized pollutant, particularly in developing nations, and it has been overshadowed by issues involving contaminants such as industrial chemicals, heavy metals, pesticides, and air pollutants among many others. However, during the past decade, aquatic pollution surveillance and monitoring programs have expanded markedly in terms of both the areal extent of coverage and the range of substances measured. As a result, selenium has emerged as an important environmental contaminant, and has gained the attention of natural resource managers and water quality regulators around the world. This chapter presents an overview of the wide variety of selenium sources that can lead to pollution of aquatic habitats and gives a sample of the many locations where impacts to aquatic life have occurred.

Sources and Impacts of Selenium Contamination

Coal Mining and Combustion

One of the primary human activities responsible for mobilizing selenium in the environment is the procurement, processing, and combustion of coal for electric power production (Lemly 1985). Although burning fossil fuels offers a cheaper and perhaps, seemingly, safer alternative to nuclear power production, especially in the aftermath of the Three Mile Island and Chernobyl reactor incidents, it does not necessarily constitute an environmentally clean alternative—there are numerous contaminant problems associated with both raw coal and its waste byproducts. Literally all categories of solid waste and liquid effluents from the power industry are highly enriched with selenium as compared to the Earth's crust and surface waters (Table 1.1). Enrichment factors for selenium in coal (ratios of selenium in coal to selenium in surrounding soils and mineral layers) can exceed 65, and are among the highest of all trace elements (Ensminger 1981). When coal is burned to produce electricity, the ash that remains is further enriched with selenium, perhaps by as much as 1250 times (Table 1.1, coal versus precipitator ash). Thus, the potential for enrichment of selenium in wastes arising from the power industry is compounded because the raw materials have already undergone natural mineral concentrating processes during their formation.

Selenium in freshly mined coal can be leached out of storage piles as rainwater percolates through. It can also be leached out when the coal is washed prior to being transported to power plants. Solid wastes from coal combustion (fly ash, bottom ash, scrubber ash, etc.) present an even greater risk of generating contaminated leachate because of their oxidation state and alkaline pH, which promote dissolution of selenium anions (selenate, selenite) on contact with water. Moreover, selenium can accumulate to high concentrations in process and disposal waters in a very short period of time. For example, using feed water with less than 10 µg Se/L, a power plant's flue gas desulfurization (FGD) once-through cleaning stream may acquire as much as 2700 µg Se/L during its passage through the system, and a coal ash slurry stream may pick up over 1000 µg Se/L within 15 minutes (Santhanam et al. 1979; Cumbie 1980). The power industry produces numerous waste materials that contain high concentrations of selenium. This selenium is readily mobilized during all phases of waste collection, treatment, and disposal that involve aqueous processes or that subsequently bring dry ash materials into contact with water. These two factors, along with the potential for bioaccumulation and toxic effects in aquatic life at very low waterborne concentrations (2–5 µg Se/L, see Chap-

ters 2 and 3), combine to make coal a highly hazardous source of selenium. There are serious implications for industrial waste management and environmental safety. As society's need for electric power increases, so does the volume of seleniferous coal wastes that are produced. In the United States alone, over 120 million tons of fly ash are produced annually, and the disposal of this material is creating a new selenium issue because of contaminated landfill leachate (see Chapter 9).

Episodes of selenium pollution associated with power production wastes have taken place around the world (Table 1.2, Fig. 1.1), and some of the most serious impacts to aquatic life have occurred as a result of this type of selenium contamination. In the United States, entire populations of reservoir fish have been eliminated due to selenium poisoning that resulted from power plant waste discharges (see Chapter 3). Severe reproductive effects are the hallmark of chronic selenium toxicity, and the insidious nature of these effects can make

TABLE 1.1. Concentrations of selenium present in raw material used by the electric power industry, and in various wastes produced during processing and utilization^a.

Material or waste	Selenium concentration
Earth's crust	0.2 µg/g ^{b,c}
Surface waters	0.2 µg/L ^b
Coal	0.4–24 µg/g ^c
Coal-storage-pile leachate	1–30 µg/L
Coal-cleaning-process water	15–63 µg/L
Coal-cleaning solid waste	2.3–31 µg/g ^c
Coal-cleaning solid-waste leachate	2–570 µg/L
Coal-burner ash (bottom ash)	7.7 µg/g ^c
Precipitator ash (fly ash)	0.2–500 µg/g ^c
Scrubber ash (fly ash)	73–440 µg/g ^c
Fly-ash leachate	40–610 µg/L
Flue-gas desulfurization-process water	1–2700 µg/L
Flue-gas desulfurization sludge	0.2–19 µg/g ^d
Boiler-cleaning water	5–151 µg/L
Coal-ash slurry	50–1500 µg/L
Ash-settling ponds	87–2700 µg/L
Ash-pond effluents	2–260 µg/L
Ash-disposal-pit leachate	40–950 µg/L
Coal-gasification-process water	5–460 µg/L
Coal-gasification solid wastes	0.7–17.5 µg/g ^d
Gasification solid-waste leachate	0.8–100 µg/L
Coal-liquefaction-process water	100–900 µg/L
Coal-liquefaction solid wastes	2.1–22 µg/g ^d

^aTable adapted from Lemly (1985).

^bRepresentative values.

^cExpressed on a dry weight basis.

^dExpressed on a wet-weight basis.

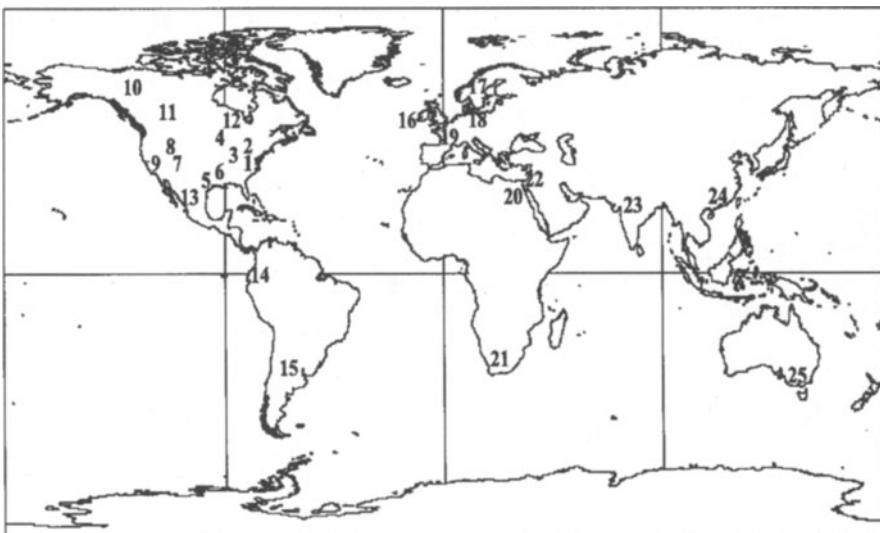


FIGURE 1.1. Selenium pollution is a worldwide phenomenon. Numbers indicate locations where fish and/or wildlife populations have been detrimentally affected. 1, North Carolina, USA; 2, Pennsylvania, USA; 3, Kentucky, USA; 4, Minnesota, USA; 5, Texas, USA; 6, Louisiana, USA; 7, Utah, USA; 8, Idaho, USA; 9, California, USA; 10, Yukon, Canada; 11, Alberta, Canada; 12, Ontario, Canada; 13, Chihuahua, Mexico; 14, Quito, Ecuador; 15, Buenos Aires, Argentina; 16, London, United Kingdom; 17, Stockholm, Sweden; 18, Torun, Poland; 19, Perrier Vittel, France; 20, Cairo, Egypt; 21, Capetown, South Africa; 22, Jerusalem, Israel; 23, New Delhi, India; 24, Wan Chai, Hong Kong; 25, New South Wales, Australia. (Refer to Table 1.1 for causes and impacts of selenium pollution at these sites.)

selenium poisoning an elusive, yet devastating, threat to aquatic life. The widespread, expanding use of coal for electric power production makes the associated risk of selenium contamination a global issue. Because it is a trace element, selenium does not biodegrade or otherwise break down into harmless materials. Rather, it moves and cycles from one environmental compartment to another, and the toxicity it causes can persist for decades (see Chapters 3 and 6). This makes state-of-the-art waste management a necessity in the development of an environmentally compatible coal industry. Although there have been major advances in reducing the particulate air pollution emanating from power plants, relatively little has been accomplished in dealing with the solid waste that is produced, other than to bury it in landfills. In fact, the same technology that improved air quality has exacerbated threats to aquatic life by producing increased volumes of seleniferous

fly ash (Clean Air Task Force [CATF 2000]). At this time, there is no treatment technology that eliminates serious environmental risks associated with disposal of this coal industry waste.

Gold, Silver, Nickel, and Phosphate Mining

Mining for precious and semiprecious metals has a long history of environmental problems, primarily associated with surface disposal of waste rock and the water used to process ore (Lemly 1994a). Increasing values for gold, silver, and nickel have pushed exploration for new deposits to the northern limits of the Canadian Arctic in North America. In addition, new technologies have emerged that make profitable the extraction of these metals, particularly gold, using ore grades that were of little or no interest just a few decades ago. One example, the heap-leach process, which percolates cyanide-laden water through ore piles and dissolves/leaches out the gold, is a widely used practice in the western United States at locations that were previously "mined out" using traditional deep-shaft and open pit methods. However, there are serious environmental risks and episodes of aquatic pollution associated with this practice. Many of the mines in North America have left a legacy of environmental damage to lakes and fish populations due to contaminants that leach from tailings and other surface residuals (Lemly 1994a). Selenium is an important elemental component of the mineral matrix of ore deposits. Although present in deceptively low concentrations relative to other constituents (low parts-per-million for selenium versus high parts-per-hundred or parts-per-thousand for the metals being mined), selenium has the potential to rapidly affect aquatic life because of its propensity to bioaccumulate and increase in concentrations as it moves up the food chain. Any mining operation that mobilizes selenium from the ore and brings it into contact with water activates this risk. Selenium has contaminated surface waters and impacted fish and wildlife near mine sites at locations ranging from the Klondike in Yukon, Canada, to the Tobe near Capetown, South Africa (see Table 1.2, Fig. 1.1). The best way to minimize the potential for selenium issues in the mining industry is to minimize surface disposal of tailings and wastewater. Practices such as backfilling of solids, recycling of process water, and *in situ* leaching can dramatically reduce risks and improve the environmental performance of mines (Lemly 1994a).

Open-pit phosphate mining is an emerging selenium issue that poses serious risks to aquatic life in the western United States (see Chapter 9). As with gold, silver, and nickel ore, selenium is associated with the mineral matrix of phosphate deposits, some of which resemble low-grade coal with respect to their carbon content. Selenium is present at

TABLE 1.2. A sample of selenium pollution episodes that have impacted fish and wildlife resources. (Refer to Figure 1.1 for map locations).

Map #	Site/ location	Cause of Se pollution	Major aquatic life affected
1	North Carolina, USA	Coal-combustion waste	Reservoir fish
2	Pennsylvania, USA	Coal-landfill waste	Stream fish
3	Kentucky, USA	Coal-mining waste	Stream fish
4	Minnesota USA	Municipal-landfill leachate	Stream fish
5	Texas, USA	Coal-combustion waste	Reservoir fish
6	Louisiana, USA	Oil-refinery waste	Aquatic birds
7	Utah, USA	Irrigation drainage	Fish, aquatic birds
8	Idaho, USA	Phosphate-mining waste	Fish, aquatic birds
9	California, USA	Irrigation drainage	Aquatic birds, fish
10	Yukon, Canada	Gold-mining waste	Stream fish
11	Alberta, Canada	Coal-mining waste	Stream fish
12	Ontario, Canada	Metal-smelting waste	Stream and lake fish
13	Chihuahua, Mexico	Irrigation drainage	Stream and river fish
14	Quito, Ecuador	Gold and silver-mining waste	Stream fish
15	Buenos Aires, Argentina	Gold-mining waste	Stream fish
16	London, United Kingdom	Municipal-landfill leachate	Stream fish
17	Stockholm, Sweden	Municipal-landfill leachate	Stream fish
18	Torun, Poland	Nickel and silver-mining waste	Stream fish
19	Perrier Vittel, France	Gold and nickel mining waste	Stream fish
20	Cairo, Egypt	Irrigation drainage	Fish, aquatic birds
21	Capetown, South Africa	Gold-mining waste	Fish, aquatic birds
22	Jerusalem, Israel	Irrigation drainage	Fish, aquatic birds
23	New Delhi, India	Oil-refinery waste	Fish, aquatic birds
24	Wan Chai, Hong Kong	Municipal-landfill leachate	Fish, aquatic birds
25	New South Wales, Australia	Coal-combustion waste	Lake and estuarine fish

concentrations of 2 to 20 µg Se/g, which is also similar to that of raw coal. Thus, many of the concerns about selenium contamination from the solid waste and surface residuals of phosphate mining parallel those of coal mining. Phosphate mining in the western United States occurs primarily in areas of low annual precipitation (<40 cm/yr). Consequently, the potential for large quantities of selenium to be leached

from surface residuals by natural rainfall/snowmelt is not as great as it would be in regions of high precipitation. For many years, these conditions prevented major selenium runoff from occurring. However, the immense size of mine tailings piles (up to 50 million m³) has changed subsurface hydrology in such a way that groundwater has risen within the tailing piles and created miniature aquifers that seep laterally and exit the mine spoils as streams of selenium-laden leachwater containing up to 1500 µg Se/L (Desborough et al. 1999; Herring et al. 1999). Local fish, wildlife, and livestock have been contaminated, and a major recreational fishery is threatened (Lemly 1999). Although there are presently no other well-documented cases of this type of selenium pollution, natural resource managers around the world should understand that phosphate mining has the potential to impact aquatic life wherever the practice occurs. It is also important to recognize that local climate and hydrology will determine the rate and type of selenium leaching that takes place from surface residuals.

Metal Smelting

Metal ores contain some amount of selenium, and the physical/chemical treatment of this ore to extract the desired metal releases selenium and other constituents into the process water or solid waste that is left. These wastes often contaminate local aquatic habitats (Lemly 1994a). However, some ores are also heated (smelted) in order to mobilize and separate the desired metal, in particular, copper, nickel, and zinc. When heating occurs, selenium is readily volatilized and can be emitted into the air as a vapor. Once released, this selenium cools and can coalesce or adhere to atmospheric dust particles (some of which are produced by the smelters themselves), subsequently reaching terrestrial and aquatic systems by either dry or wet deposition (Germani et al. 1981; Small et al. 1981). In some situations, these processes can be a substantial factor in the cycling of selenium near smelting facilities. One such example occurred near Sudbury, Ontario, Canada (Table 1.2, Fig. 1.1). Selenium is quite common in copper ore and may actually be more concentrated in copper ore than in coal (20–82 µg Se/g for copper ore versus 0.4–24 µg Se/g for coal) (Table 1.1) (Nriagu and Wong 1983). On a total-mass basis, the Sudbury ore deposits north of Lake Huron in Canada are the largest source of selenium in North America (Shamberger 1981). Large-scale copper smelting at Sudbury began in the early 1900s, and sampling conducted in the late 1970s showed that selenium discharges had contaminated freshwater lakes for a distance of at least 30 km downwind of the smelter (Nriagu and Wong 1983). The aerial plume was also implicated as the primary cause of elevated concentrations of selenium in fish and other biota of Georgian Bay in

Lake Huron, some 100 to 200 km away (Hodson et al. 1984). It is important to recognize this source of selenium because, in addition to contaminating local habitats, smelting can contribute to selenium inputs in distant aquatic systems due to the mechanism by which it is transported in the atmospheric vapor/particle phase (Small et al. 1981). This aspect of selenium pollution operates on the same principle as the acid rain phenomenon—emission of gas-phase pollutants that eventually reach aquatic systems and threaten aquatic life, and that form deposition/pollution corridors downwind from major sources. Large-scale metal smelting operations should be viewed as an important contributor to this phenomenon for selenium.

Municipal Landfills

Although not currently recognized as a widespread cause of selenium pollution, municipal landfills can generate leachwater that contains elevated concentrations of selenium (5–50 µg Se/L) if seleniferous materials have been disposed there. Noteworthy examples have occurred in the United States, United Kingdom, Sweden, and Hong Kong (see Table 1.2, Figure 1.1). The source materials for selenium in these landfills seem to be similar: large amounts of photoelectronic components (rectifiers, capacitors, photocopy printer/toner products, etc., which contain/require selenium to operate properly) that were disposed from local computer and electronics manufacturing facilities (Minnesota Pollution Control Agency [MPCA] 2000; Stockholm Water Authority [SWA] 2000). Selenium is widely used in electronics because of its photosensitive and semiconducting properties (Sharma and Singh 1983). The disposal histories of these landfills suggest that selenium-laden leachate could have been released for a number of years but was only detected recently due to expanded pollution screening programs mandated by local water quality authorities. The global distribution of electronics and computer/copier industries, coupled with the practice of landfill disposal of their solid wastes, causes this source of selenium to pose an important localized threat of selenium contamination. The lesson to be heeded from these examples is that it is important for contaminant surveillance programs to monitor landfill leachate for possible selenium contamination. This can be done by simply including selenium in the list of constituents designated for chemical analysis.

Oil Transport, Refining, and Utilization

As in the coal industry, procurement and refining of oil produce a variety of selenium-laden wastes (Table 1.3). The source of selenium

TABLE 1.3. Concentrations of selenium present in oil shale, crude oil, and various products and wastes^a.

Material or waste	Selenium concentration
Oil shale	1.3–5.2 µg/g ^b
Crude shale oils	92–540 µg/L
Shale oil retort water	3–100 µg/L
Retort solid waste leachate	10–30 µg/L
Crude oil	500–2200 µg/L
Refined oils	5–258 µg/L
Refinery wastewater	15–75 µg/L
Oil burner ash (fly ash)	3–10 µg/g ^b

^aTable compiled from American Petroleum Institute (API 1978), Lemly (1985), and Ohlendorf and Gala (2000).

^bExpressed on a dry-weight basis.

in oil is also the same as for coal: natural selenium contained in the fossil raw material that formed over a geologic time scale. However, crude oil contains much higher concentrations of selenium than coal (500–2200 µg/L versus 0.4–24 µg/g), thus the potential for hazardous amounts to be released in process waters and effluents is relatively high. Once in the aquatic environment, selenium in these wastes can rapidly bioaccumulate and cause reproductive failure and other toxic effects in fish (Rowe et al. 1983) and aquatic birds (Ohlendorf and Gala 2000), and I list two examples where impacts to aquatic life have occurred (Table 1.2, Figure 1.1). In most cases, selenium pollution from refinery wastes is overlooked because of concerns over other constituents that receive higher priority: total suspended solids, polycyclic aromatic hydrocarbon compounds (PAHs), oil and grease, heavy metals, etc. (United States Environmental Protection Agency [EPA] 1974; Woodward et al. 1981, 1983; Ridlington et al. 1982; Rowe et al. 1983; Hawkins et al. 1997; Ohlendorf and Gala 2000). These constituents also get most of the attention when accidental spills of crude oil occur in marine or freshwater habitats. Notable examples include the 1978 Amoco Cadiz spill off the coast of France and the 1989 Exxon Valdez spill near the south shore of Alaska in the United States. Extensive, long-term studies of these pollution episodes show that primary concerns are for heavy oil coating of wildlife and beaches and the aquatic toxicity of the hydrocarbon fraction (Haensly et al. 1982; Mielke 1990; Schmitt 1999). However, the unrecognized dangers of trace elements such as selenium may, in fact, pose a substantial long-term risk because of bioaccumulation and persistence in the environment. The lack of attention to selenium associated with oil transport and refining

is widespread and even pervades university research conducted to develop treatment methods for reducing ecological risks from refinery effluents (eg, Hawkins et al. 1997). The oil industry transports and disposes huge volumes of selenium-laden materials on a global scale. At any point in this process it can become a major contributor to elevated selenium concentrations in aquatic ecosystems.

Agricultural Irrigation

In the early 1980s, a new selenium threat to fish and wildlife emerged: subsurface irrigation drainage. This drainage water, usually containing elevated concentrations of soil trace elements and other constituents, has poisoned fish and aquatic birds at several locations in the United States and the Middle East (Table 1.2, Figure 1.1). The mechanism underlying this phenomenon is simple, yet almost insidious. Agricultural irrigation practices in arid and semi-arid regions typically use water applications in the 60 to 80 cm/yr range. The amount of water applied is far in excess of what is needed to support crops, but the excess is used to flush away salts that tend to accumulate in crop root zones as evaporation occurs and inhibit plant growth. Subsurface irrigation drainage is produced due to a specific set of soil conditions. Shallow subsurface (3–10 m) layers of clay impede the vertical movement of irrigation water as it percolates downward. If the irrigation water is not removed, this results in waterlogging of the crop root zone and subsequent buildup of salts as excess water evaporates from the soil surface, exactly the same problem that irrigation is intended to solve in the first place (Moore et al. 1990).

Several methods of removing excess shallow groundwater can be employed, including the use of wells and surface canals to forcefully pump and drain the water away. The method of choice in the western United States is to install rows of permeable clay tile or perforated plastic pipe 3 to 7 m below the surface of agricultural fields (Letey et al. 1986). Once these drains are installed, irrigation water can be applied liberally, thus satisfying the water needs of crops while also flushing away excess salts. The resultant subsurface wastewater is pumped or allowed to drain into ponds for evaporative disposal, or into creeks and sloughs that are tributaries to major wetlands, streams, and rivers (Moore et al. 1990). Subsurface irrigation drainage is characterized by alkaline pH, elevated concentrations of salts, trace elements, and nitrogenous compounds, and low concentrations of pesticides. The natural biological and chemical filter provided by the soil effectively degrades and removes most pesticides as irrigation water percolates downward to form subsurface drainage. At the same time, naturally occurring trace elements in the soil, such as selenium (up to 1400 µg/L), are leached out under the alkaline, oxidizing

conditions prevalent in arid climates and are carried in solution in the drainwater (Presser and Ohlendorf 1987).

When subsurface irrigation drainage is discharged into surface waters, a variety of serious biological effects can take place. The immediate impact is degradation of surface and groundwater quality through salinization and contamination with toxic, or potentially toxic, trace elements (selenium, arsenic, boron, molybdenum, chromium, etc.). Long-term impacts can occur if selenium enters aquatic food chains. A landmark case of this type of impact occurred in 1985 at Kesterson National Wildlife Refuge, CA, in the United States (Table 1.2, Figure 1.1), where thousands of fish and waterfowl were poisoned. Selenium and other trace elements were leached from soils on the west side of the San Joaquin Valley and carried to the refuge in irrigation return flows that were used for wetland management (Zahm 1986). Selenium bioaccumulated in aquatic food chains and contaminated 500 ha of shallow marshes. Elevated selenium was found in every animal group inhabiting these wetlands, from fish and birds to insects, frogs, snakes, and mammals (Saiki and Lowe 1987; Clark 1987; Ohlendorf et al. 1988). Selenium-induced reproductive impairment was documented in a variety of species, and teratogenic deformities (a biomarker of chronic selenosis) were evident as well (see Chapter 3). Congenital malformations in young waterbirds were severe and consisted of missing eyes and feet, protruding brains, and grossly deformed beaks, legs, and wings (Ohlendorf et al. 1986a, 1986b, 1988; Hoffman et al. 1988). Several species of fish were eliminated, and a high frequency (30%) of stillbirths occurred in the single remaining species (Saiki and Ogle 1995). Laboratory studies conducted by the US Fish and Wildlife Service confirmed the field assessment that irrigation drainage was the source of elevated selenium and toxic effects (Lemly et al. 1993).

The biogeochemical conditions leading to the production of seleniferous subsurface irrigation drainage, culminating in death and deformities in wildlife, have been termed the "Kesterson Effect" and are prevalent throughout the western United States (Presser 1994; Presser et al. 1994; Seiler et al. 1999). These conditions include: (1) a marine sedimentary basin that contains soils formed during the Cretaceous period, which have relatively high natural concentrations of selenium; (2) alkaline, oxidized soils that promote the formation of water-soluble forms of selenium (especially selenate); (3) a dry climate, in which evaporation greatly exceeds precipitation, leading to salt buildup in soils; (4) subsurface layers of clay that impede downward movement of irrigation water and cause waterlogging of the crop root zone; and (5) subsurface drainage into wetlands and aquatic habitats by natural gradient or buried tile/pipe drainage networks. In view of the key factors that contribute to hazardous subsurface drainage, it is important

that existing and planned agricultural irrigation projects be reviewed for possible selenium contamination. The anticipation and evaluation of potential problem areas will allow changes to be made in irrigation practices for the benefit of both agriculture and wildlife (Lemly 1994b, 1994c; Lemly et al. 2000).

Conclusions

When the phenomenon of selenium pollution on a global scale is examined closely, it becomes clear that the more we look, the more examples we find. Important sources of selenium contamination in aquatic habitats are often overlooked by environmental biologists and ecological risk assessors due to preoccupation with other, higher priority pollutants. These oversights occur in experimental research as well as in field management operations. Failure to include selenium in the list of constituents measured in contaminant screening/monitoring programs is a major mistake, from the aspects of both hazard assessment and pollution control. Once selenium contamination begins, a cascade of events is set into motion that can result in major ecosystem disruption. Early detection and action is key. Environmentally sound hazard assessment and water quality goals, coupled with prudent risk management, can prevent significant biological impacts. The methods and techniques set out in the following chapters of this book are intended specifically for that purpose.

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2

Interpreting Selenium Concentrations

Introduction

The importance of selenium as an environmental contaminant has gained widespread attention among field biologists, scientists, and natural resource managers during the past two decades. Although the basic toxicological symptoms and the nutritional paradox of selenium (nutritionally required in small amounts but highly toxic in slightly greater amounts) have been known for many years (Draize and Beath 1935; Ellis et al. 1937; Rosenfeld and Beath 1946; Hartley and Grant 1961), it was not until the late 1970s and early 1980s that the potential for widespread contamination of aquatic ecosystems due to human activities was recognized (Andren et al. 1975; Cherry and Guthrie 1977; Evans et al. 1980; National Research Council 1980; Braunstein et al. 1981). As recently as 1970, selenium was being called the “unknown pollutant” in the context of what was known about its cycling and toxicity in the aquatic environment (Copeland 1970). Yet, within a few years, several major cases of selenium contamination would take place and reveal the need to interpret selenium concentrations in a variety of aquatic habitats. Two examples were the pollution event at Belews Lake, NC, in which an entire fish community (19 species) was eliminated due to selenium in wastewater from a coal-fired power plant (Cumbie and Van Horn 1978; Garrett and Inman 1984; Sorensen et al. 1984; Lemly 1985a, 1993, 1997; Sorensen 1986), and the episode at Kesterson National Wildlife Refuge, CA, in which thousands of fish and waterbirds were poisoned by selenium in agricultural irrigation drainage (Marshall 1985; Hoffman et al. 1986; Ohlendorf et al. 1986, 1988; Saiki 1986a, 1986b; Saiki and Lowe 1987).

The research studies that have resulted from these and other cases of selenium contamination provide a foundation for interpreting selenium residues in the various environmental components that influence bioaccumulation and toxicity. This chapter reviews key literature describing toxic effect levels, and gives guidelines for evaluating sele-

nium in water, sediments, food-chain organisms, and fish and aquatic bird tissues.

Toxic Thresholds

All values for tissue selenium residues in this chapter are given on a dry-weight basis. Data from references that only reported wet weights were converted to dry weight assuming 75% moisture, that is, by multiplying the wet-weight concentration by 4. It is important to understand that the values given as thresholds are levels at which toxic effects begin to occur in sensitive species of fish and aquatic birds; for example, centrarchid and salmonid fishes (eg, genus *Lepomis*, *Micropterus*, *Oncorhynchus*, *Salvelinus*), ducks (genus *Anas*), and wading birds (genus *Recurvirostra*). They are not levels that signify the point at which everything dies from selenium poisoning, that is, the point at which total reproductive failure or massive mortality of juveniles and adults occurs. At the threshold level, selenium-tolerant species will be unaffected.

Water

Selenium is strongly bioaccumulated in aquatic habitats; this results in a marked elevation of residues in food-chain organisms as compared to waterborne concentrations (Lemly 1985b; Maier et al. 1988; Ogle et al. 1988; Ohlendorf 1989). It is critical to know how much bioaccumulation can be expected for a given aqueous level of selenium in order to evaluate the potential for dietary toxicity and reproductive effects in predatory species of fish and wildlife. Laboratory studies show that organoselenium compounds (seleno-L-methionine) can be bioconcentrated over 200 000 times by zooplankton when water concentrations are in the 0.5 to 0.8 µg Se/L (parts per billion) range (Besser et al. 1989, 1993). Resultant selenium residues were over 100 µg Se/g, a concentration that far exceeds the dietary toxicity threshold for fish (3 µg Se/g). Organoselenium compounds can comprise a substantial portion of the total waterborne selenium concentration in aquatic environments (Chau et al. 1976; Cutter 1982, 1986, 1991; Cooke and Bruland 1987), although the complete range of chemical species is poorly described. The potential for bioaccumulation and toxicity due to organic selenium is very high.

Inorganic selenium (selenate, selenite) bioaccumulates more readily in phytoplankton than zooplankton, and residues of up to 18 µg Se/g can occur when waterborne concentrations are in the 7 to 10 µg Se/L range (Besser et al. 1993), resulting in bioaccumulation factors of about 3000. It is at the primary producer and primary consumer levels of the food chain (phytoplankton and zooplankton) that most of the

bioaccumulation occurs. Biomagnification of selenium (progressively higher concentrations in successive trophic levels of the food chain) is not clearly indicated in laboratory studies (Bennett et al. 1986, 1989, 1993). However, some field studies have found that the levels continue to rise from 2 to 6 times through the food chain in a pattern suggestive of biomagnification (Woock and Summers 1984; Lemly 1985a, 1986; Saiki 1986a; Lemly and Smith 1987; Saiki and Lowe 1987; Barnum and Gilmer 1988; Hothem and Ohlendorf 1989).

Field studies have documented selenium bioaccumulation factors of 500 to 35 000 in contaminated aquatic habitats where levels of waterborne selenium were in the 2 to 16 µg Se/L range (Sager and Cofield 1984; Woock 1984; Woock and Summers 1984; Lemly 1985a, 1985b; Baumann and Gillespie 1986; Barnum and Gilmer 1988). These waterborne concentrations resulted in food-chain residues of 10 to 60 µg Se/g which, again, far exceed the dietary toxicity threshold for fish and wildlife. Until about 1986, most of the published literature on selenium residues in aquatic food chains and associated impacts to predatory species pertained to fisheries in power plant cooling reservoirs in the southeastern United States (eg, Duke Power Company 1980; Garrett and Inman 1984; Woock and Summers 1984; Lemly 1985a, 1985b; Gillespie and Baumann 1986). In these systems, it appeared that the threshold for significant bioaccumulation was in the range of 2 to 5 µg Se/L (Lemly 1985a, 1985b, 1986; Lemly and Smith 1987).

Several studies of selenium in agricultural irrigation drainwater in the western United States show that selenium residues may accumulate to toxic levels (10–20 µg Se/g) in the food chain when waterborne concentrations are in the 0.5 to 3 µg Se/L range (Barnum and Gilmer 1988; Schroeder et al. 1988; Stevens et al. 1988; Hoffman et al. 1990; Saiki 1990; Skorupa and Ohlendorf 1991; Hallock et al. 1992). This may be due to the presence of ultra-trace amounts (<1 µg Se/L) of organoselenium compounds that bioaccumulate similar to free seleno-amino acids and produce disproportionately high tissue residues as compared to inorganic selenate and selenite (Besser et al. 1989, 1993). In some cases, it may be the recycling of organic selenium contained in decaying plant and animal detrital material that is responsible for unexpectedly high residues in the food chain and in consumer species of fish and wildlife (Saiki and Lowe 1987; Parker and Knight 1989; Hallock et al. 1992; Sanders et al. 1992).

The environmental speciation of selenium is complex, and several chemical forms are likely to be present in solution at a given location and time (Cutter 1982, 1986, 1991; McKeown and Marinas 1986; Cooke and Bruland 1987). However, the patterns and magnitude of bioaccumulation are similar enough between aquatic systems impacted by power production wastes, by agricultural irrigation drainwater, and

by other selenium sources that a common number can be given as the threshold for detrimental effects. I recommend that waterborne selenium concentrations of 2 µg Se/L or greater (total recoverable basis in 0.45 µm filtered samples) be considered highly hazardous to the health and long-term survival of sensitive fish and aquatic birds. This threat is due to food-chain bioaccumulation and resultant dietary exposure and reproductive effects, not direct waterborne toxicity. Some species will be relatively unaffected at the 2 µg Se/L level, but sensitive species, many of which are the most important in terms of ecological integrity and public recreational value, can be seriously impacted. It should also be recognized that, under certain environmental conditions, waterborne concentrations of 1 µg Se/L or less have the potential to bioaccumulate to levels in the food chain that are toxic to predatory species.

Sediments

In order to identify an accurate and reliable threshold for hazardous amounts of selenium in sediments, it is necessary to have long-term measurements of selenium concentrations and associated detrital food-chain bioaccumulation across a gradient from little or no accumulation/effects to detrimental levels. One of the most useful data sets for this purpose is from Belews Lake, NC. Various investigators monitored environmental conditions and selenium concentrations in water, sediments, and associated biota from the precontamination stage (1970–1973) through the time of selenium input (1974–1986) and for a 10-year period of natural recovery after selenium inputs stopped (1987–1996) (Cumbie 1978; Cumbie and Van Horn 1978; Weiss and Anderson 1978; Holland 1979; Lemly 1985a, 1993, 1997).

Selenium in sediments typically exhibits wide spatial and temporal variability, and benthic organisms also undergo seasonal cycles of growth, movement, emergence, and recolonization. These factors make it difficult to sort out the variability and come up with a reliable toxic threshold. The extensive data set from Belews Lake makes it possible to reduce the variability and link a narrow range of selenium concentrations in sediments to a narrow range of concentrations in benthic macroinvertebrates. The threshold value given here is the approximate selenium concentration in sediments that elevates residues in benthos to levels that are toxic to fish and aquatic birds that eat them. Benthic organisms can tolerate body burdens of selenium far greater than the dietary toxic level for fish (3 µg Se/g dry weight) and aquatic birds (7 µg Se/g dry weight) without suffering ill effects (see next section of this chapter). Thus, the most important aspect of selenium residues in sediments is not direct toxicity to benthic organisms themselves,

TABLE 2.1. Concentrations of selenium in sediments and associated benthic macroinvertebrates of Belews Lake, NC, during precontamination (1970–1973), selenium discharge (1974–86), and recovery (1987–1996) periods.

Site and year	Selenium in sediment ^a	Selenium in benthos ^a
Littoral zone (<1 m depth)		
1970–1973	<1	<2
1974–1986	8–12	25–57
1987–1996	1–2	1–3
Bays (1–5 m depth)		
1970–1973	<1	<2
1974–1986	4–12	15–51
1987–1996	1–4	2–10
Open lake (>5 m depth)		
1970–1973	<1	<2
1974–1986	6–11	9–63
1987–1996	1–5	3–18

^aParts per million ($\mu\text{g Se/g}$), dry weight.

but rather the dietary source of selenium they provide to fish and wildlife species that feed on them.

Table 2.1 lists selenium concentrations in sediments and associated benthic macroinvertebrates from a variety of shallow- and deep-water habitats in Belews Lake over the period of 1970 to 1996. From these data, a value of 2 $\mu\text{g Se/g}$ in sediment emerges as the threshold beyond which bioaccumulation exceeds 3 $\mu\text{g Se/g}$ in benthos—that is, the dietary toxic level for fish. At 4 $\mu\text{g Se/g}$ in sediment, concentrations in benthos can reach 10 $\mu\text{g Se/g}$, and when sediments contain 5 $\mu\text{g Se/g}$, concentrations in benthos may exceed 15 $\mu\text{g Se/g}$. I recommend that 2 $\mu\text{g Se/g}$ be used as the toxic threshold value for selenium in sediments. Data from many other locations in the United States (for example, selenium studies at various sites impacted by agricultural irrigation drainwater (eg, Hallock et al. 1992, National Irrigation Water Quality Program [NIWQP] 1998) support this value. Some investigators have noted that flowing water habitats may not have sediment–biota selenium relationships equivalent to those for lentic systems because relatively little organic detrital material is present (Canton and Van Derveer 1997; Van Derveer and Canton 1997). This might raise questions about the applicability of the 2 $\mu\text{g Se/g}$ sediment threshold in lotic habitats. However, isolated parts of streams or rivers should not be considered separately when evaluating selenium hazards, because hydrological connections can cause high bioaccumulation in adjacent or downstream habitats (see Chapter 6) (Hamilton and Lemly 1999). Therefore, the threshold sediment value of 2 $\mu\text{g Se/g}$

should be applied uniformly to all aquatic systems when interpreting selenium concentrations.

Aquatic Food Organisms of Fish and Wildlife

Although many laboratory and field studies have determined either the toxicity or bioaccumulation of selenium in invertebrates and other aquatic food organisms of fish and wildlife (see reviews by Eisler 1985; Maier et al. 1988; Ogle et al. 1988; Ohlendorf 1989), very few have done both and also report tissue residues associated with toxic effects. Foe and Knight (1986) cultured the green alga *Selenastrum capricornutum* in solutions of sodium selenite and found that Chlorophyll-a concentrations, dry weight, and cell replication were reduced when tissue residues reached about 20 µg Se/g (Table 2.2). Cell division was completely stopped when residues reached the 100 to 500 µg Se/g range. Kiffney and Knight (1990) exposed the cyanobacterium *Anabaena flos-aquae* to solutions of sodium selenate, sodium selenite, and seleno-L-methionine and found that Chlorophyll-a concentrations were unaffected until tissue residues reached 700 µg Se/g regardless of the chemical form of selenium used. Ingersoll et al. (1990) exposed the cladoceran *Daphnia magna* to a 6:1 ratio of waterborne sodium selenate and sodium selenite, and observed that residues of 20 µg Se/g or greater were associated with reduced weight of adults. The production of young was significantly reduced when tissue residues reached about 30 µg Se/g (Table 2.2).

Field studies show that benthic invertebrates and certain forage fishes (mosquitofish *Gambusia affinis*, red shiners *Notropis lutrensis*, fathead minnows *Pimephales promelas*) can accumulate 20 to 370 µg Se/g and still maintain stable, reproducing populations (Woock and Summers 1984; Lemly 1985a, 1985b; Saiki 1986a, 1986b; Saiki and Lowe 1987; Barnum and Gilmer 1988; Roth and Horne 1988; Schuler 1989; Schuler et al. 1990). Plankton and aquatic plants appear to be largely unaffected with residues of 30 µg Se/g or more (Woock and Summers 1984; Lemly 1985a; Saiki 1986a, 1986b; Roth and Horne 1988; Schuler et al. 1990).

The most important aspect of selenium residues in aquatic food chains is not direct toxicity to the organisms themselves, but rather the dietary source of selenium they provide to fish and wildlife species that feed on them. The consensus of research studies is that most of the selenium in fish tissues results from selenium in the diet rather than the water (Cumbie and Van Horn 1978; Lemly 1982, 1985a; Finley 1985; Hamilton et al. 1986, 1990; Woock et al. 1987; Besser et al. 1993; Coyle et al. 1993). The environmental selenium cycle includes strong bioaccumulation steps in the aquatic food chain, and these bioaccumulation steps greatly increase the dietary levels of selenium available to fish and birds that consume

TABLE 2.2. Tissue selenium concentrations associated with toxic effects in fish, birds, and aquatic organisms.

Species	Tissue	Selenium concentration ^a	Effect	Reference
Rainbow trout (<i>Oncorhynchus mykiss</i>)	Whole-body	2 µg/g	Blood changes	Hodson, Spry, and Blunt 1980
	Liver	51 µg/g	Blood changes	Hodson, Spry, and Blunt 1980
	Whole-body	5 µg/g	Mortality	Hilton, Hodson, and Slinger 1980
	Whole-body	1 µg/g	Mortality	Hunn, Hamilton, and Buckler 1987
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	Whole-body	20 µg/g	Reduced smolting	Hamilton et al. 1986
	Whole-body	2 µg/g	Reduced growth	Hamilton et al. 1990
	Whole-body	5 µg/g	Mortality	Hamilton et al. 1990
Fathead minnow (<i>Pimephales promelas</i>)	Whole-body	5 µg/g	Reduced growth	Ogle and Knight 1989
	Ovaries	24 µg/g	Reproductive failure	Schultz and Hermanutz 1990
	Whole-body	16 µg/g	Reproductive failure	Schultz and Hermanutz 1990
Striped bass (<i>Morone saxatilis</i>)	Muscle	14 µg/g	Mortality	Coughlan and Velte 1989
	Whole-body	2 µg/g	Mortality	Saiki, Jennings, and Wiedmeyer 1992
Bluegill sunfish (<i>Lepomis macrochirus</i>)	Muscle	20 µg/g	Mortality	Finley 1985
	Liver	34 µg/g	Mortality	Finley 1985
	Carcass	24 µg/g	Reproductive failure	Gillespie and Baumann 1986
	Ovaries	23 µg/g	Reproductive failure	Gillespie and Baumann 1986

(Continued on next page)

aquatic organisms (Lemly 1985a, 1989; Saiki 1986a, 1986b; Lemly and Smith 1987; Saiki and Lowe 1987; Hothem and Ohlendorf 1989). Thus, a small increase in waterborne selenium will result in a disproportionately large elevation of selenium residues in fish and wildlife tissues. Moreover, selenium is efficiently transferred from parents to offspring through the eggs (Gillespie and Baumann 1986; Heinz et al. 1987, 1989; Schultz and Hermanutz 1990; Coyle et al. 1993). A contaminated aquatic food chain can leave a legacy of selenium poisoning in fish and wildlife populations for many generations (Duke Power Company 1980; Lemly 1985a, 1997; Sorensen 1988; Coughlan and Velte 1989; Ohlendorf 1989).

TABLE 2.2. (Continued)

Species	Tissue	Selenium concentration	Effect	Reference
Aquatic birds (<i>Anas</i> sp.)	Whole-body	5 µg/g	Mortality	USFWS 1990
	Whole-body	19 µg/g	Reproductive failure	Coyle et al. 1993
	Ovaries	34 µg/g	Reproductive failure	Coyle et al. 1993
	Eggs	42 µg/g	Reproductive failure	Coyle et al. 1993
	Ovaries	18 µg/g	Reproductive failure	Hermanutz et al. 1992
	Muscle	16 µg/g	Reproductive failure	Hermanutz et al. 1992
	Liver	29 µg/g	Reproductive failure	Hermanutz et al. 1992
	Whole-body	18 µg/g	Reproductive failure	Hermanutz et al. 1992
	Liver	10 µg/g	Reproductive failure	Skorupa, Mormon, and Sefchick-Edwards 1996
	Eggs	7 µg/g	Reproductive failure	Skorupa, Mormon, and Sefchick-Edwards 1996
Green alga (<i>Selenastrum capricornutum</i>)	Whole organism	20 µg/g	Reduced cell replication	Foe and Knight 1986
Cyanobacterium (<i>Anabaena flos-aquae</i>)	Whole organism	394 µg/g	Reduced Chlorophyll-a	Kiffney and Knight 1990
Cladoceran (<i>Daphnia magna</i>)	Whole organism	15 µg/g	Reduced weight	Ingersoll, Dwyer, and May 1990
	Whole organism	32 µg/g	Reproductive failure	Ingersoll, Dwyer, and May 1990

^a Selenium concentrations in parts per million on a dry weight basis.

The toxic effect thresholds for selenium impacts on food-chain organisms (20 to 700 µg Se/g) are much higher than the dietary effect levels for fish and wildlife. Dietary concentrations of 6.5 µg Se/g or greater (as selenomethionine) have been shown to cause mortality and reproductive failure of centrarchids (Woock et al. 1987; United States Fish and Wildlife Service [USFWS] 1990; Coyle et al. 1993), while dietary levels of greater than 3 µg Se/g reduce survival of juvenile salmonids (Goettl and Davies 1978; Hilton et al. 1980; Hilton and Hodson 1983; Hamilton et al. 1986, 1989, 1990) (Table 2.3). The uptake kinetics and effect levels for natural field-source selenium diets and seleno-L-methionine spiked commercial

diets are nearly identical (Woock et al. 1985, 1987; Hamilton et al. 1986, 1989), indicating that the results of dietary toxicity studies with seleno-L-methionine in the laboratory can be used to accurately evaluate the hazard of food-chain residues in the field. Hilton, Hodson and Slinger (1980) concluded that diets containing in excess of 3 µg Se/g may ultimately be toxic to rainbow trout. Similar dietary effect levels have been found for aquatic birds. Studies by Heinz et al. (1987, 1989) showed that reproduction of mallard ducks, *Anas platyrhynchos*, was impaired at dietary levels of 7 µg Se/g (Table 2.3). These values contrast sharply with those for aquatic invertebrates, which can tolerate up to 300 µg Se/g in the diet without effects on reproduction, growth, or survival (Foe and Knight 1986). Food-chain organisms can thus build up tissue concentrations of selenium that are toxic to predators while remaining unaffected themselves. I recommend that 3 µg Se/g be used as the toxic threshold value for dietary

TABLE 2.3. Dietary selenium concentrations known to be toxic to fish and aquatic birds.

Species	Dietary selenium concentration ^a	Effect	Reference
Rainbow trout (<i>Oncorhynchus mykiss</i>)	9 µg/g	Mortality	Goettl and Davies 1978
	>3 µg/g	Mortality	Hilton, Hodson, and Slinger 1980
	10 µg/g	Kidney damage	Hilton and Hodson 1983
Chinook salmon (<i>Oncorhynchus tshawytscha</i>)	6.5 µg/g	Mortality	Hamilton, Buhl, and Faerber 1989
	5 µg/g	Reduced growth	Hamilton et al. 1990
Fathead minnow (<i>Pimephales promelas</i>)	20 µg/g	Reduced growth	Ogle and Knight 1989
Striped bass (<i>Morone saxatilis</i>)	35 µg/g	Mortality	Coughlan and Velte 1989
Bluegill (<i>Lepomis macrochirus</i>)	50 µg/g	Mortality	Finley 1985
	6.5 µg/g	Mortality	USFWS 1990
	13 µg/g	Reproductive failure	Woock et al. 1987
	16 µg/g	Reproductive failure	Coyle et al. 1993
Mallard duck (<i>Anas platyrhynchos</i>)	11 µg/g	Reproductive failure	Heinz et al. 1987
	7 µg/g	Reproductive failure	Heinz, Hoffman and Gold 1989

^a Selenium concentrations in parts per million on a dry weight basis.

selenium transferred to fish through aquatic food chains, and that 7 µg Se/g be used as the threshold value for aquatic birds.

Fish Tissues

The salmonids are very sensitive to selenium contamination, and they exhibit toxic symptoms even when tissue residues are quite low. In laboratory studies, Hunn et al. (1987) exposed rainbow trout fry, *Oncorhynchus mykiss*, to waterborne sodium selenite and found that significant mortality occurred when whole-body residues exceeded 4 µg Se/g (parts-per-million). Hodson et al. (1980) and Hilton et al., (1980) exposed juvenile rainbow trout to waterborne and dietary sodium selenite and found that significant changes in blood chemistry occurred when whole-body tissue residues reached about 3 µg Se/g (liver tissues contained 12 µg Se/g). Survival was reduced when whole-body residues exceeded 5 µg Se/g. Hamilton et al. (1986, 1989, 1990) exposed juvenile chinook salmon, *Oncorhynchus tshawytscha*, to combinations of waterborne and dietary selenium (6:1 ratio of waterborne sodium selenate and sodium selenite; field-source selenium diet; and seleno-DL-methionine spiked commercial diet) and observed that smoltification and seaward migration were impaired when whole-body tissue residues reached about 9.5 µg Se/g (Table 2.2). Mortality occurred when concentrations exceeded 10 µg Se/g. However, growth was impaired at whole-body tissue levels of only 2 to 3 µg Se/g; these levels were only 2 to 3 times those of the controls (0.8–1.0 µg Se/g).

In studies of juvenile and adult fathead minnows, *Pimephales promelas* (a cyprinid), Bennett et al. (1986) and Ogle and Knight (1989) reported that growth was inhibited at whole-body tissue levels of 6 to 8 µg Se/g or greater (selenium administered as waterborne sodium selenate and a dietary mixture of 25% sodium selenate, 50% sodium selenite, and 25% seleno-L-methionine). Schultz and Hermanutz (1990) dosed outdoor experimental streams with sodium selenite and observed the effects on reproduction in fathead minnows. Reproductive success (survival of fry to swim-up) was impaired when the ovarian tissue of spawning females contained about 15 µg Se/g and resultant fry contained about 8 µg Se/g on a whole-body basis (Table 2.2).

Coughlan and Velte (1989) fed selenium-laden red shiners, *Notropis lutrensis*, collected from a contaminated power plant reservoir to juvenile striped bass, *Morone saxatilis* (a percichthyid), and found that the fish accumulated 14 to 16 µg Se/g in skeletal muscle tissue, did not gain weight, and died within 78 days. There was also extensive tissue damage in the liver and trunk kidney of these fish. Poor survival of juvenile striped bass has been associated with whole-body tissue lev-

els of about 5 to 8 µg Se/g in fish exposed to agricultural irrigation drainwater (Greenberg and Kopec 1986; Saiki and Palawski 1990; Saiki et al. 1992).

Several field and laboratory studies conducted in the United States describe the effects of selenium on bluegill sunfish, *Lepomis macrochirus*, and other centrarchids (Table 2.2). Cumbie and Van Horn (1978) and Lemly (1985a, 1985b) found that selenium levels of 12 to 16 µg Se/g in skeletal muscle and 40 to 60 µg Se/g in ovaries were associated with reproductive failure and mortality of all 9 species of centrarchids present in a power plant cooling reservoir in North Carolina. Similar effects were reported for centrarchids in a selenium-contaminated reservoir in Texas; the skeletal muscle residues for the centrarchids were in the 8 to 36 µg Se/g range (Garrett and Inman 1984). Several physiologically important changes in blood parameters, in tissue structure in major organs (ovary, kidney, liver, heart, gills), and in organ weight/body weight relationships have also been described for centrarchids in these contaminated reservoirs (Sorensen et al. 1984; Sorensen 1986, 1988). Selenium residues of 20 to 80 µg Se/g were associated with the various pathological conditions.

Finley (1985) fed selenium-laden invertebrates from a contaminated reservoir to juvenile bluegill and found that mortality occurred when skeletal muscle tissues contained 20 to 32 µg Se/g and liver tissue contained 32 to 86 µg Se/g. Whole-body concentrations of only 4 to 6 µg Se/g were associated with mortality when juvenile bluegill were fed selenomethionine-spiked commercial diets in the laboratory (USFWS 1990).

Gillespie and Baumann (1986) brought selenium-laden adult bluegill from a contaminated power plant reservoir into the laboratory and spawned them artificially to produce crosses between clean and contaminated parents. The results showed that the contaminated females (selenium levels of 8 to 36 µg Se/g in carcasses, 12 to 55 µg Se/g in ovaries) did not produce viable offspring. Fertility and hatchability of the eggs were not affected, but the high level of selenium (12 to 55 µg Se/g) transferred from the eggs to the developing embryos during yolk-sac absorption resulted in edema, morphological deformities, and death prior to the swim-up stage. Similar findings were reported by Woock et al. (1987).

In a laboratory study, Coyle et al. (1993) evaluated the effects of waterborne and dietary selenium (6:1 ratio of waterborne sodium selenate and sodium selenite; seleno-L-methionine-spiked commercial diet) on the reproductive success of bluegill. Offspring from females that contained whole-body selenium residues of 16 to 18 µg Se/g (30–38 µg Se/g in ovaries, 40–45 µg Se/g in eggs) failed to survive beyond the swim-up stage (5–7 days posthatch). No effect was observed on adult fish, spawning frequency, eggs per spawn, or hatchability of eggs.

The authors recommended that gravid ovarian tissue be used to monitor selenium residues and potential effects on bluegill populations, since this tissue delivers the toxic "dose" to the developing fry. They concluded that ovarian selenium levels in excess of 13 µg Se/g may result in reproductive impairment. This number agrees very well with the findings of Gillespie and Baumann (1986), who observed that feral bluegill with ovarian selenium levels of 12 µg Se/g or greater failed to produce viable offspring. It also agrees with Hermanutz et al. (1992), who dosed outdoor experimental streams with sodium selenite, allowed natural cycling and bioaccumulation to occur in the food chain and in adult bluegill, and then measured spawning success. Ovarian selenium residues of 10 to 28 µg Se/g (skeletal muscle , 10–24 µg Se/g; liver , 22–85 µg Se/g; whole body , 12–35 µg Se/g) were associated with a 15% decrease in survival of adult fish and almost complete reproductive failure, with larvae exhibiting edema, lordosis (spinal deformities), and hemorrhaging.

Lemly (1993) studied the teratogenic effects of selenium in natural populations of centrarchids and other warm-water fish species. He determined the prevalence of abnormalities and associated tissue selenium concentrations in a contaminated lake and 2 reference lakes over a period of 17 years. Whole-body selenium concentrations of 15 µg Se/g were associated with a 10-fold higher incidence of teratogenic defects in centrarchid populations in the contaminated lake (tissue selenium was 1–3 µg Se/g in the reference lakes). The relationship between tissue selenium residues and the prevalence of malformations approximated an exponential function over the range of 1 to 80 µg Se/g and 0 to 70% deformities ($R^2 = 0.881$, $P < 0.01$). Lemly concluded that this relationship could be used to predict the impact of teratogenic defects in warm-water fish populations suspected of having selenium-related reproductive failure (see Chapter 5).

Depending on the specific tissue (skeletal muscle, ovary, liver, whole body, etc.), concentrations of selenium in fish from control test groups or habitats with low ambient selenium levels usually range from about 1 to 8 µg Se/g (Baumann and May 1984; Lemly 1985a; Gillespie and Baumann 1986; Hermanutz et al. 1992; Coyle et al. 1993). However, tissue damage in major organs, reproductive impairment, and mortality begin to occur when levels reach 4 to 16 µg Se/g (Table 2.2). This extremely narrow margin between "normal" and toxic levels in tissues, along with the propensity of selenium to bioaccumulate in aquatic food chains, underscores the biological importance of even slight increases in environmental selenium.

I recommend that the following tissue residues be used as toxic effects thresholds for the overall health and reproductive vigor of freshwater and anadromous fish: whole body , 4 µg Se/g; skeletal muscle , 8 µg Se/g; liver , 12 µg Se/g; ovary and eggs , 10 µg Se/g. Laboratory

and field studies show that the most sensitive indicator of selenium impacts on centrarchid populations is reproductive success (Cumbie and Van Horn 1978; Lemly 1985a; Gillespie and Baumann 1986; Woock et al. 1987; Hermanutz et al. 1992; Coyle et al. 1993). This is also true for fathead minnows (Pyron and Beitinger 1989; Schultz and Hermanutz 1990), and probably for salmonids and percichthyids as well, since the residue levels at which tissue damage and mortality of fry and juveniles occurs is almost identical to that for centrarchids (Lemly 1985a; Hamilton et al. 1986, 1989, 1990; Coughlan and Velte 1989). The most precise way to assess selenium status and potential reproductive impairment of adult fish is to measure selenium levels in gravid ovaries. This single measure integrates waterborne and dietary exposure and allows evaluation of the most sensitive biological endpoint. Biologists and natural resource managers should consider this when designing aquatic monitoring studies to assess selenium contamination.

Aquatic Bird Tissues

Several publications provide detailed discussions of the interpretation of tissue concentrations of selenium in waterfowl and other aquatic birds (eg, Ohlendorf 1989; Skorupa and Ohlendorf 1991; Heinz 1996; Skorupa et al. 1996). A brief summary of the conclusions of these reports is presented here.

Field and laboratory studies have shown that the most sensitive indicator of selenium toxicity in aquatic birds is reproductive failure. Embryo mortality and teratogenic effects in hatchlings are markers for selenium toxicity, and impacts begin to occur when tissue concentrations reach 7 µg Se/g in eggs and 10 µg Se/g in adult bird livers. However, not all species are affected at these concentrations, and follow-up studies of reproductive performance are recommended to provide conclusive evidence of adverse effects. The levels of mortality and deformities increase markedly as concentrations rise, and 50% or more of all birds may be affected when residues reach 10 µg Se/g in eggs and 30 µg Se/g in adult livers. Logistic response curves show that the pattern of selenium-induced teratogenesis is consistent between locations for same-species exposures (Skorupa and Ohlendorf 1991), lending support for the premise that uniform environmental quality guidelines can be applied across a range of habitat types and environmental conditions. Moreover, studies of aquatic birds show that there is agreement among thresholds at which waterborne selenium begins to become a toxic hazard via food-chain bioaccumulation. For both fish and wildlife, this threshold falls within the range of 2 to 5 µg Se/L (Skorupa 1998). Based on these findings, I recommend that 7 µg Se/g in bird eggs and 10 µg Se/g in bird liver tissue be used as the thresholds for toxic effects for selenium impacts on avian reproduction. The simplest and most accurate way to assess selenium status and potential

reproductive impairment in aquatic birds is to sample freshly laid eggs. As with gravid fish ovaries, this single measure integrates dietary and waterborne exposure and allows an evaluation based on the most sensitive biological endpoint.

Conclusions

Assessing selenium hazards to aquatic life is a two-step process. The first step is to measure selenium concentrations in several environmental components and then interpret the potential toxicity of these residues using biological effects guidelines. Key data and recommendations for toxic threshold values for selenium in water, sediments, food-chain organisms, and fish and aquatic bird tissues (Table 2.4) have been presented in this chapter. These values can be used as guidelines

TABLE 2.4. Toxic effect thresholds^a for selenium in aquatic ecosystems.^b

Selenium source	Selenium concentration ^c	Effect
Water		
Inorganic selenium	2 µg/L	Food-chain bioaccumulation and reproductive failure in fish and aquatic birds
Organic selenium	< 1 µg/L	Food-chain bioaccumulation and reproductive failure in fish and aquatic birds
Sediments		
	2 µg/g	Food-chain bioaccumulation and reproductive failure in fish and aquatic birds
Food-chain organisms		
	3 µg/g	Reproductive failure in fish
	7 µg/g	Reproductive failure in aquatic birds
Fish tissues		
Whole-body	4 µg/g	Mortality of juveniles and reproductive failure
Skeletal muscle (skinless fillets)	8 µg/g	Reproductive failure
Liver	12 µg/g	Reproductive failure
Ovary and eggs	10 µg/g	Reproductive failure
Aquatic Birds		
Liver	10 µg/g	Reproductive failure
Eggs	7 µg/g	Reproductive failure

^aThese are levels at which toxic effects begin to occur in sensitive species of fish and aquatic birds. They are not levels that signify the point at which all species die from selenium poisoning.

^bRecommendation by author based on synthesis and interpretation of literature cited in this chapter.

^cSelenium concentrations in parts per billion for water; parts per million on a dry weight basis for sediments, food-chain organisms, and fish and bird tissues.

for assessing the degree of contamination, the relative toxic threat to aquatic life, and the extent to which aquatic ecosystems have recovered following management actions to reduce selenium inputs. Importantly, these measures provide the data needed for the second step: comprehensive hazard assessment using procedures that integrate all 5 ecosystem components (see Chapter 4). The overall selenium status and health of aquatic ecosystems can be determined by combining these two steps.

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3

Toxic Effects of Selenium in Fish

Introduction

One of the most extensive and prolonged cases of selenium poisoning in freshwater fish occurred at Belews Lake, NC in the United States (Fig. 3.1). Belews Lake was contaminated by selenium in wastewater released from a coal-fired electric generating facility. From 1974 to 1986, water was withdrawn from the lake and mixed with bottom ash from the coal burners and fly ash collected by electrostatic precipitators. This slurry was pumped from the power plant and discharged into a 142 ha ash basin, where suspended solids were collected by gravitational settling. Selenium-laden (150–200 µg Se/L) return flows from the ash basin entered the west side of Belews Lake through an ash sluice water canal (Lemly 1985).

Selenium bioaccumulated in aquatic food chains and caused severe tissue pathology and reproductive impairment in the resident fish community, leading to the elimination of 19 of the 20 species originally present in the reservoir (Lemly 1985; Sorensen 1986). In late 1986, the power plant stopped discharging selenium-laden water into the lake, and a period of natural recovery began, augmented with artificial stocking of fish. However, monitoring studies revealed that the rate of recovery was slow; elevated selenium residues and associated biological effects in fish were still present a decade later (Lemly 1997).

The Belews Lake pollution event provides a wealth of information on aquatic cycling, long-term persistence, and hazard of selenium to freshwater biota. It is an excellent case for examining the biological symptoms and ecological consequences of selenium toxicity. This chapter utilizes that database to present a concise review of the effects of selenium poisoning in fish and discuss the environmental lessons learned from Belews Lake.

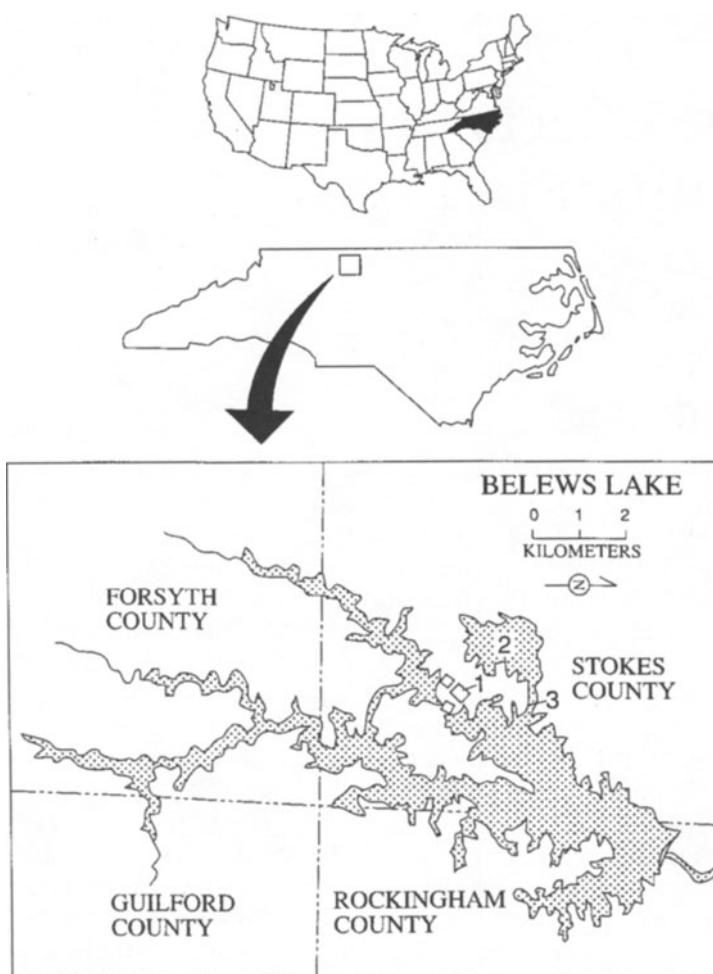


FIGURE 3.1. Geographic location of Belews Lake, North Carolina, USA. Numbers indicate: (1) Belews Creek Steam Station (coal-fired power plant); (2) coal-ash settling basin; (3) ash basin return-water canal.

Biochemical Basis of Selenium Toxicity

The primary manifestations of selenium toxicity are due to a simple but important selenium-induced error in the process of protein synthesis. Sulfur is a key component of proteins, and sulfur-to-sulfur linkages (ionic disulfide bonds) between strands of amino acids are necessary for protein molecules to coil into their tertiary (helix) structure. In turn, this tertiary structure is necessary for proteins to function prop-

erly, either as components of cellular structure (tissue synthesis) or as enzymes in cellular metabolism. Selenium is similar to sulfur in its basic chemical and physical properties (has same valence states and forms analogs of hydrogen sulfide, thiosulfate, sulfite, and sulfate), and mammalian studies show that cells do not discriminate well between the two as proteins are being synthesized (it is assumed that the mechanistic features underlying toxicity are essentially the same for fish, since the resulting pathology and teratogenic features are the same). When present in excessive amounts, selenium is erroneously substituted for sulfur, resulting in the formation of a triselenium linkage (Se–Se–Se) or a selenotrisulfide linkage (S–Se–S), either of which prevent the formation of the necessary disulfide chemical bonds (S–S). Distorted, dysfunctional enzymes and protein molecules, which impair normal cellular biochemistry, are the end result (Ganther 1974; Stadtman 1974; Diplock and Hoekstra 1976; Reddy and Massaro 1983; Sunde 1984). Thus, while selenium is a sulfur analog in some respects, its biochemistry and potential toxicity are quite different.

Selenium-induced errors in protein biosynthesis can have several outcomes. The most well documented overt toxic symptom in fish is reproductive teratogenesis. Selenium consumed in the diet of adult fish is deposited in the eggs, where it is metabolized by larval fish after hatching. A variety of lethal or sublethal deformities can occur in the developing fish, affecting both hard and soft tissues (Lemly 1993a). Substitution of selenium for sulfur can also impair proper formation of proteins in juvenile and adult fish, and many internal organs and tissues can develop pathological alterations that are symptomatic of chronic selenosis (Sorensen 1986). Studies in mammals and waterfowl show that acute toxic responses may also involve tissue damage from bioreactive superoxides produced in response to high concentrations of selenium (O'Toole and Raisbeck 1998). At the subcellular level, selenium poisoning disrupts enzyme-mediated biochemical reactions thus impairing metabolism. This impairment can result in the death of cells and the entire organism (Stadtman 1974, Diplock and Hoeckstra 1976).

Pathological Effects

Gills

The primary structure of adult teleost gills is the semicircular gill arch; there are usually 4 gill arches on each side of the head. Each arch contains a double row of filaments, and each filament has a row of microscopic lamellae projecting from each side (Fig. 3.2). The lamellae contain the blood sinusoids and capillary beds and are covered by a thin epithelial cell layer, typically 2-cells thick, underlain by support-

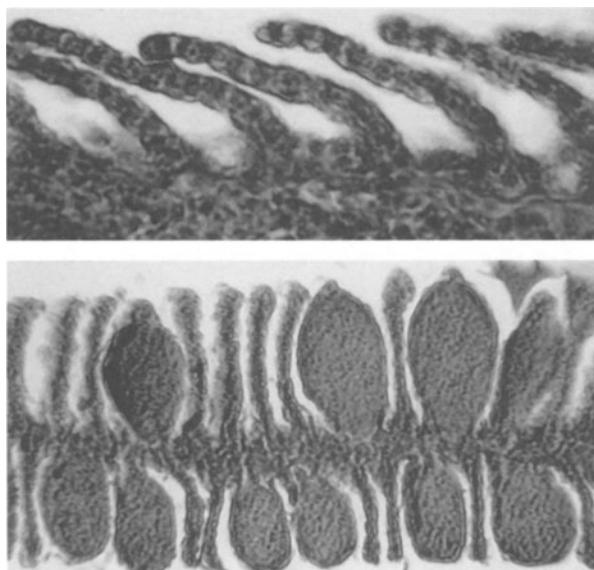


FIGURE 3.2. Section of gill tissue from a normal green sunfish (*Lepomis cyanellus*, top, original magnification 240 \times), and a green sunfish exposed to selenium in Belews Lake (bottom, original magnification 220 \times). Secondary lamellae are uniform and well defined under normal conditions, but exposure to high selenium can cause the blood sinuses to undergo extensive swelling (telangiectasia) and become packed with erythrocytes. This reduces respiratory capacity and can lead to metabolic stress and death.

ing pillar cells, which maintain the patency of vascular lumina. The normally thin, delicate structures of gill lamellae (Fig. 3.2a) are necessary for effective gas exchange in respiration. Gills from green sunfish (*Lepomis cyanellus*) exposed to selenium contamination in Belews Lake exhibited extensively dilated blood sinusoids and swollen lamellae (telangiectasia) packed with erythrocytes (Fig. 3.2b) (Sorensen et al. 1984). Hemorrhaging of the gill tissue often occurred in association with this condition. Selenium-induced dilation of gill lamellae causes impaired blood flow, ineffective gas exchange (reduced respiratory capacity), and a metabolic stress response (increased respiratory demand and oxygen consumption) that can lead to death (Lemly 1993b).

Blood

Green sunfish from Belews Lake exhibited significantly reduced hematocrit values (packed erythrocyte volumes), as compared to fish from an uncontaminated reference lake (33% versus 39%), but had significantly elevated numbers of lymphocytes. Thrombocytes constituted a

higher percentage of total leucocytes in Belew's Lake fish, but hemoblasts were less numerous than in reference fish (Sorensen et al. 1984). These shifts in hematological parameters reflect important changes in the overall health of fish. Reductions in hematocrit values are associated with anemia and lowered mean corpuscular hemoglobin concentrations (MCHC) (Lemly 1993b). Reduced MCHC causes impaired respiratory capacity because selenium can bind to hemoglobin, rendering it incapable of carrying oxygen. A decrease in respiratory capacity can quickly lead to metabolic stress because the fish must expend more energy to meet respiratory demands (Lemly 1993b). Lower numbers of hemoblasts reflect reduced erythropoiesis and delayed replacement of aging red cells in circulation, which also contribute to reduced respiratory capacity and metabolic stress (Lemly 1993b). An elevation in lymphocytes signals a generalized immune response, triggered by physiological stress, and a reduced state of health.

Liver

The structural features of liver tissue from normal green sunfish consist of bilaminar arrays of hepatocytes (liver plates) separated by small blood sinusoids. Blood enters the liver from the hepatic artery and hepatic portal vein, moves between the liver plates in the sinusoids, and ultimately collects in central veins which empty into the hepatic vein. Parenchymal hepatocytes typically contain numerous mitochondria, abundant rough endoplasmic reticulum, well developed nucleoli, and both central and peripheral chromatin islands (Sorensen 1986). Kupffer cells, (phagocytic tissue histocytes) are rarely present in healthy individuals, and lymphocytes are not numerous. Green sunfish from Belew's Lake exhibited several histopathological differences from normal liver tissue. Lymphocyte infiltration was apparent along with extensive vacuolization of parenchymal hepatocytes around central veins. Increased numbers of Kupffer cells were present, and the central veins were distended and swollen due to loss of surrounding parenchymal cells. Cell nuclei were often deformed and pleomorphic, and numerous perisinusoidal lipid droplets (unmetabolized residues) were present (Sorensen et al. 1984). Collectively, these ultrastructural changes reflect a degeneration of tissue structure that is sufficient to significantly alter liver function. This liver pathology syndrome is characteristic of chronic selenosis in fish and other vertebrates (Sorensen 1986).

Kidney

At the ultrastructure level, the kidney of a normal fish is quite similar to that of a human. It is made up of glomeruli, mesangial cells,

podocytes, endothelial and tubular cells, and both capillary and central veins (which collect and transport urine). The livers of Belews Lake green sunfish that had accumulated high levels of selenium showed focal intracapillary proliferative glomerulonephritis (Sorensen et al. 1984). In this condition, excessive numbers of mesangial cells are present along with an abnormally abundant matrix and periglomerular fibrosis (which can lead to a hardening of the tissue). Numerous tubular casts were present, and tubular epithelium was desquamated, vacuolated, and often destroyed (which can render the tubular system of the mesonephros incapable of functioning properly). These renal changes in Belews Lake fish were consistent with symptoms of chronic selenium poisoning in other vertebrates (Sorensen et al. 1984).

Heart

A clear pathological pattern occurred in the hearts of fish from Belews Lake. The pericardial spaces surrounding the heart were filled with inflammatory cells which were not present in fish from reference locations. This condition was diagnosed as severe pericarditis. Numerous inflammatory cells were also present within the ventricular myocardial tissue, a condition known as myocarditis. The occurrence of pericarditis and myocarditis was attributed to the direct action of selenium on heart tissue, coupled with indirect effects of selenium on the kidney (ie, selenium-induced glomerulonephritis and associated uremia) (Sorensen et al. 1984).

Ovary

Ovaries of fish from Belews Lake contained numerous swollen, necrotic, and ruptured mature egg follicles, especially in gravid individuals. No such pathology was observed in fish from reference locations (Sorensen et al. 1984). These toxic symptoms were primary factors contributing to reproductive failure of fish in Belews Lake. Nineteen species were affected, and the aquatic ecosystem was severely altered for over a decade (Lemly 1985, 1997).

Eyes

One of the lesser known symptoms of selenium poisoning in fish is the occurrence of selenium-induced cataracts. This condition, which can affect both the lens and cornea, has been induced experimentally in mammals by dietary exposure to selenite (Shearer et al. 1987). Numerous fish from Belews Lake had corneal cataracts on their eyes

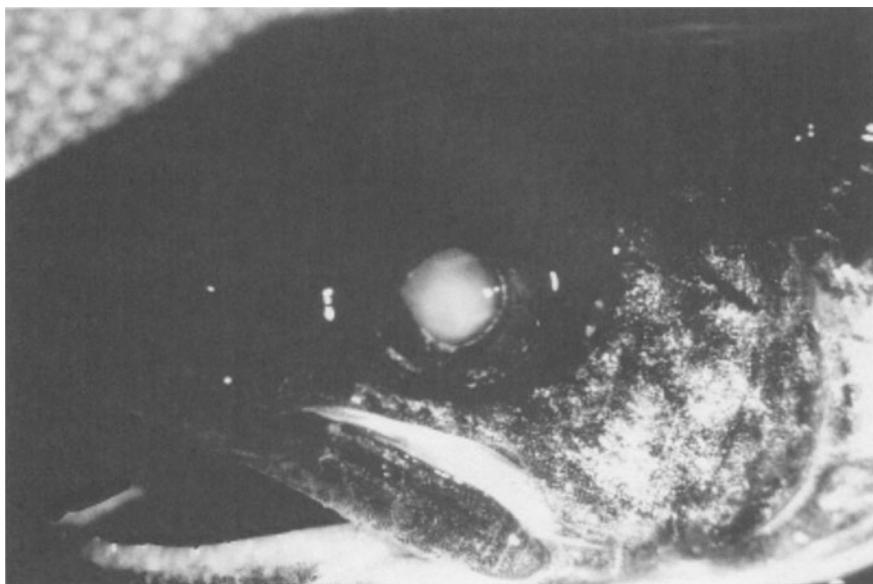


FIGURE 3.3. Corneal cataract in the eye of a juvenile largemouth bass (*Micropterus salmoides*) from Belews Lake. During the peak period of selenium contamination (1975–1985), up to 8.1% of the fish community was afflicted with cataracts.

(Fig. 3.3); none were found in fish from reference lakes. Cataracts were present in up to 8.1% of fish examined in surveys conducted from 1975 to 1982, which was the period of maximum selenium concentrations in fish. By 1992, selenium residues had fallen in fish, concurrent with reduced selenium inputs to Belews Lake, and the prevalence of cataracts had also fallen, to about 1% (Lemly 1993a).

Another abnormality of the eyes associated with selenium poisoning in fish is a condition known as edema-induced exophthalmus, or protruding eyeballs. One of the general physiological responses of fish to high levels of selenium is edema, which is the accumulation of fluid in the body cavity and head (Ellis et al. 1937). The fluid results from tissue damage, specifically an abnormal increase in cell permeability as a consequence of distorted selenoproteins in the membrane structure, that causes internal organs to become “leaky”. The excess fluid can create pressure sufficient to swell the abdomen and force the eyes to protrude from their sockets (Fig. 3.4). Blood may be present in the fluid, which can result in noticeable hemorrhaging around the eyes. Up to 21% of some fish species in Belews Lake exhibited exophthalmus, with the greatest prevalence occurring in crappie, *Pomoxis* sp. (Lemly 1993a).



FIGURE 3.4. Exposure to high levels of selenium can cause fluid to build up and create pressure inside the head, leading to exophthalmus (protruding eyeballs), shown here in this juvenile white crappie (*Pomoxis annularis*) from Belew's Lake (*top*). (*Bottom* individual is normal.)

Teratogenic Deformities

Developmental malformations are among the most conspicuous and diagnostic symptoms of chronic selenium poisoning in fish. Terata are permanent biomarkers of toxicity which can be used to reliably identify and evaluate impacts of selenium on fish populations (see Chapter 5). Deformities that affect feeding or respiration can be lethal shortly after hatching (Fig. 3.5). Few individuals bearing this type of terata will survive to join the juvenile population. Terata that are not directly lethal but which distort the spine and fins can reduce the swimming ability of fish and lead to increased susceptibility to predation, an important indirect cause of mortality. These two factors generally prevent most deformed individuals from surviving to adulthood. In Belew's Lake, the reproductive impacts on piscivorous species eliminated much of the predation pressure and allowed many of the deformed individuals of nonpiscivorous species to persist into the juvenile and adult life stages (Lemly 1985).

Several types of teratogenic deformities were evident in Belew's Lake fish, and many individuals exhibited multiple malformations. The most overt terata were spinal deformities consisting of kyphosis, lordosis, and

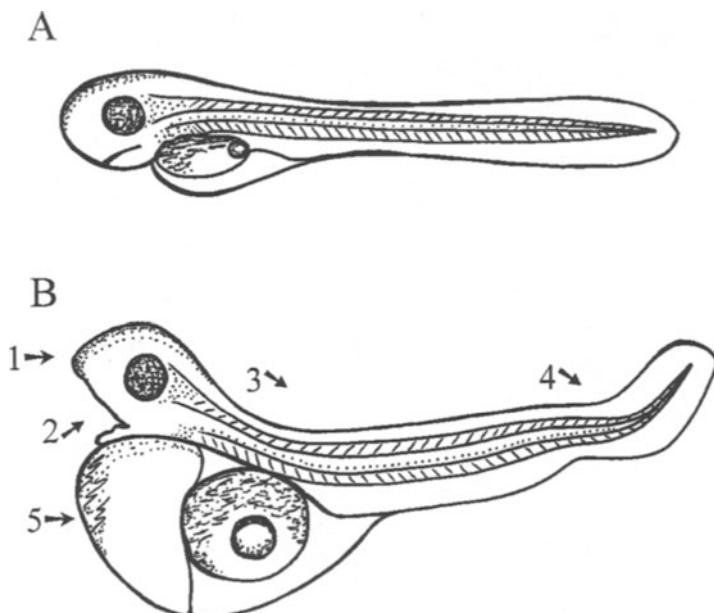


FIGURE 3.5. Typical appearance of larval fish at about 2 to 4 days after hatching. A Normal larvae with yolk absorption nearing completion and straight, developing spine, B Abnormal development due to selenium-induced terata: 1 deformed, pointed head; 2 gaping mouth and deformed lower jaw; 3 kyphosis (curvature of the thoracic region of the spine); 4 lordosis (concave curvature of the lumbar and/or caudal region of the spine). Other symptoms of selenium poisoning that usually accompany terata include 5 edema (swollen, fluid-filled abdomen) and delayed yolk absorption.

scoliosis (Figs. 3.6 through 3.8). Less obvious, but no less common, were terata involving the mouth and fins (Fig. 3.9) (Lemly 1993a). The prevalence of deformities varied among species and between years, reaching a high of 70% in green sunfish during 1982. There was a close parallel between levels of selenium in fish tissues and frequency of deformities. Terata became more common as selenium increased from 1975 to 1982, peaked in 1982, and decreased in frequency following the cessation of selenium inputs to the lake in 1986 (Lemly 1993a). By 1996, selenium residues had fallen by 85 to 95% from their 1982 high, and the prevalence of deformities was 6% or less (Lemly 1997). An overall relationship between tissue selenium burdens and incidence of deformities in the Belews Lake fish community is shown in Chapter 5, Figure 5.1 (Lemly 1993a, 1997). Belews Lake was the first site to provide conclusive evidence that exposure to elevated selenium causes teratogenic deformities in natural populations of freshwater fish.

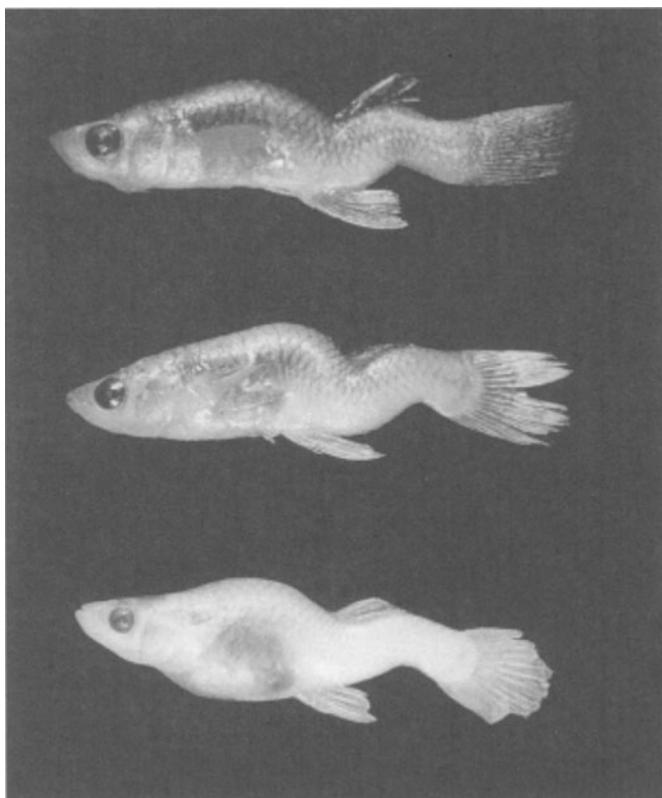


FIGURE 3.6. One of the most common and outwardly visible teratogenic effects of selenium in Belews Lake fish was dorso-ventral spinal deformities (kyphosis and lordosis), shown here in mosquitofish (*Gambusia affinis*).

Ecological Implications

Insidious Mode of Toxicity

Selenium poisoning in fish can be “invisible” because the primary point of impact is the egg, which receives selenium from the female’s diet and stores it until hatching, whereupon teratogenic deformity and death may occur. Adult fish can survive and appear healthy despite the fact that massive reproductive failure is occurring (Lemly 1985; Coyle et al. 1993). Consequently, fish populations can decline or even disappear over the course of a few years for no apparent reason. In Belews Lake, fish populations disappeared over a 4-year period (1974–1977), and by the time biologists documented changes in population struc-

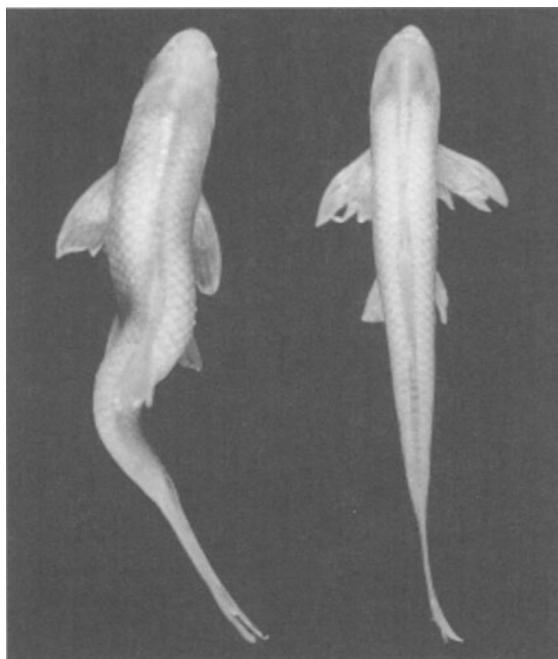


FIGURE 3.7. Lateral curvature of the spine (scoliosis, left individual) in a red shiner (*Notropis lutrensis*), caused by exposure to elevated selenium in Belews Lake. Individual on right is normal.

ture associated with elevated selenium (Cumbie and Van Horn 1978), it was far too late to prevent the fishery from collapsing.

Another factor contributing to selenium's insidious mode of toxicity is Winter Stress Syndrome (WSS, see next section in this chapter), which is metabolic distress brought on by cold water temperature in combination with selenium exposure. WSS can greatly increase the toxicity of selenium to young centrarchids. Dietary and waterborne concentrations that are tolerated during warm weather become lethal in winter. The end result is a somewhat obscure seasonal poisoning of young fish, which can offset any successful reproduction. The implication of WSS is especially serious for aquatic habitats that are only slightly contaminated and may not exhibit overt symptoms of selenium toxicity (eg, teratogenic deformities in fish, high food-chain bioaccumulation).

Bioaccumulation and Reproductive Failure

Selenium bioaccumulated in the aquatic food chains of Belews Lake and caused severe reproductive failure in fish (Cumbie and Van Horn

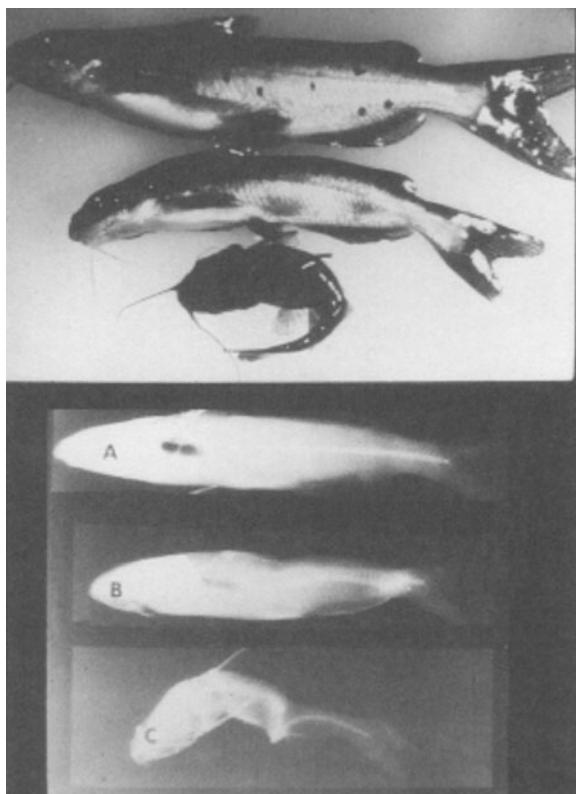


FIGURE 3.8. Outward appearance and x-ray image of selenium-induced terata in channel catfish (*Ictalurus punctatus*) from Belews Lake. (A , Normal; B , mild spinal deformity (kyphosis and lordosis); C , severe deformities (kyphosis and scoliosis)).

1978; Lemly 1985). This reproductive impairment occurred because of two factors: (1) reduced production of viable eggs due to ovarian pathology in spawning females (necrotic and ruptured mature egg follicles), and (2) posthatch mortality due to metabolism of egg selenium by developing larval fish (teratogenic deformities and biochemical dysfunction). Concentrations of selenium in the lake water averaged 10 µg Se/L (uncontaminated reference locations had selenium concentrations <1 µg Se/L), but were accumulated from 519 times (periphyton) to 3975 times (visceral tissue of fish) in the biota. The pattern of accumulation and degree of accumulation were essentially complete within 2 years of the initiation of power plant operation, and persisted throughout the period of selenium discharge into the lake (1974–1985). Fish had the highest concentrations of selenium, followed

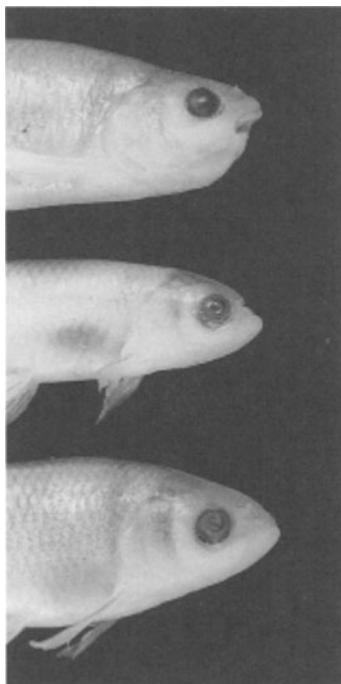


FIGURE 3.9. Other teratogenic effects of selenium in Belews Lake fish, shown here in red shiners (*Notropis lutrensis*), included deformed mouth and jaws (top), and deformed upper head and vestigial pectoral fins (middle). Individual on bottom is normal.

by benthic macroinvertebrates, plankton, and periphyton. The planktonic and detrital food pathways exposed fish to dietary concentrations of selenium that were some 770 and 510 to 1395 times the waterborne exposure, respectively.

Of the 20 species of fish originally present in Belews Lake, 19 were effectively rendered sterile because of reproductive failure. Some persisted as adults for a few years, but eventually all 19 were eliminated. Only one of the original resident species, the selenium-tolerant mosquitofish (*Gambusia affinis*) survived relatively unaffected, along with one introduced cyprinid, red shiner (*Notropis lutrensis*). The fishery was decimated without massive fish kills because of the insidious, yet lethal, mechanisms by which selenium poisoning occurs. The severe toxic impacts in Belews Lake took place even though concentrations of waterborne selenium were only 10 to 20 times those in nearby uncontaminated reservoirs; the flora and fauna contained 10 to 50 times as much selenium.

Persistence of Impacts

In response to concerns about the fishery problems in Belews Lake, the electric utility company switched to a dry-ash handling system that disposed the waste in a landfill rather than a wet basin. By late 1986, selenium-laden wastewater no longer entered the lake (North Carolina Department of Natural Resources and Community Development [NCDNRCD] 1986), and in subsequent years a stocking program was successful in re-establishing adult populations of sport fish (eg, centrarchids such as largemouth bass, *Micropterus salmoides*, and bluegill, *Lepomis macrochirus*). Follow-up studies were conducted in 1996 to assess recovery of the ecosystem in Belews Lake (Lemly 1997). Tissue selenium concentrations and associated impacts to fish were measured and compared to pre-1986 conditions to determine how much change had occurred since selenium inputs stopped. Findings were also examined using a hazard assessment protocol (see Chapter 4) to determine if ecosystem-level hazards to fish and aquatic birds had changed as well.

Results showed that waterborne selenium fell from a peak of 20 µg Se/L before 1986, to less than 1 µg Se/L in 1996; concentrations in biota were 85 to 95% lower in 1996. Hazard ratings indicated that high hazard existed prior to 1986, and moderate hazard was still present in 1996, primarily due to selenium in the sediment-detrital food pathway. Concentrations of selenium in sediments fell by 65 to 75% during the period but remained sufficiently elevated (1–4 µg Se/g) to contaminate benthic food organisms of fish and aquatic birds. Field evidence confirmed the validity of the hazard ratings. Developmental abnormalities in young fish persisted in 1996, indicating that selenium-induced teratogenesis and reproductive impairment were still occurring a decade after selenium inputs stopped. Moreover, the residual levels of selenium were sufficient to cause seasonal mortality in young bluegill and other centrarchids because of Winter Stress Syndrome (see next section in this chapter), further impeding the recovery of fish populations. The latent effects occurred because of selenium present in sediments, which was mobilized gradually, yet continually, through the detrital food chain and accumulated to toxic levels in fish tissues. Impacts to reproduction and overwinter survival of young fish persisted, even though adult populations were re-established through a stocking program. Projections indicate that several more decades may be necessary for the Belews Lake ecosystem to fully recover (Lemly 1997). The low inflow of water and long retention time (volume replacement time about 1500 days), combined with low productivity (oligotrophic), tend to reduce natural flushing and enhance recycling of selenium within the reservoir.

Winter Stress Syndrome and Selenium Toxicity

Characteristics and Symptoms

Winter Stress Syndrome (WSS) is a term I coined to describe a condition of metabolic distress in warm-water fish (Lemly 1993b). The syndrome develops when external stressors that cause increased metabolic demands are present when water temperature decreases in the autumn. Cold weather, and the associated short photoperiod of winter, environmentally "programs" these fish for reduced physical activity and food intake, and they do not respond to the stressors with increased feeding. If the elevated metabolic demands persist, stored body lipid necessary for overwintering is depleted, body condition drops, and the fish may die. The key element in WSS is the influence of environment on behavior, that is, reduced feeding as a result of cold water temperature and short photoperiod.

Many species of fish in temperate regions of North America undergo a normal seasonal period of reduced feeding, decreased body condition, and lowered nutritional status during winter (Oliver et al. 1979; Toney et al. 1980; Thompson et al. 1991). This, in turn, results in an annual cycle of ability to resist metabolic stressors. WSS is a serious threat to these fish because it occurs when their capability to compensate is at its lowest point. Stressors that are normally tolerated during warm weather and active feeding can become lethal as water temperatures and food intake drop in late autumn (Lemly and Esch 1984; Lemly 1993b).

Three conditions must be met concurrently in order for WSS to develop: (1) a significant metabolic stressor must be present; (2) cold water temperature must be present ($<10^{\circ}\text{C}$); and (3) fish must respond to cold water temperature by reducing activity and feeding. The stressor alone is usually not sufficient to cause problems, but when combined with reduced feeding as water temperatures drop, it can cause the syndrome to develop within 60 days (Lemly 1993b). Fish in the later stages of WSS are somewhat thin and appear to be undernourished. The stressor increases the nutritional requirements of fish by increasing metabolism and oxygen consumption. If food intake is adequate to offset these demands, that is, if water temperature remains warm enough to promote active feeding, no change in condition or apparent health occurs. However, cold water temperature causes reduced feeding, and a nutritional deficit can soon develop (Lemly 1993b).

Several physiological factors contribute to the nutritional deficit and metabolic distress. Pathological conditions may develop in direct

response to the stressor, leading to increased respiration and oxygen consumption, and depletion of stored body lipids (Lemly and Esch 1984). If the stressor is a chemical contaminant, for example, selenium, a variety of sublethal tissue and organ responses can occur and increase energy requirements (Ribelin and Migaki 1975; Hocutt and Stauffer 1980; Rand and Petrocelli 1985; Lemly 1993b). These factors impinge on warm-water fish at a time when they are forced to be inactive by cold temperature. They do not respond behaviorally or physiologically to offset the metabolic effects of the stressor.

The end result of WSS is often death. As water temperature drops, the continued presence of a metabolic stressor greatly accelerates the normal seasonal cycle of lipid depletion. Without this source of energy to draw upon, the limited feeding that takes place is insufficient to supply adequate nutrition. The lipid content of young bluegill (*Lepomis macrochirus*), for example, does not normally fall below about 10% (dry weight) during winter. Bluegill experiencing WSS cannot maintain as much stored lipid, and levels may fall below 5%, which is the threshold for significant mortality in this species (Lemly 1993b).

Increased Selenium Toxicity

As many as one third of young centrarchids exposed to combined dietary and waterborne selenium at concentrations normally tolerated in warm conditions may die within one month of the onset of WSS (Lemly 1993b) (Fig. 3.10). The critical point for survival is reached when body lipid falls to about 5%, which occurs within 30 to 60 days once water temperature drops below 10°C. Individuals that have sufficient lipid to live for another 60 days with limited feeding will likely survive indefinitely, but those with inadequate lipid will not. WSS causes a 5-fold or greater increase in the sensitivity of young centrarchids to selenium. In warm conditions (25°C), greater than 25 µg Se/g in the diet (dry weight) and greater than 330 µg Se/L in water are necessary to cause significant mortality (Cleveland et al. 1993), but only 5 µg Se/g in the diet and 5 µg Se/L in water can be lethal to fish experiencing WSS (Lemly 1993b).

In North America, centrarchids (family Centrarchidae) appear to be at high risk of developing WSS, even in habitats where selenium concentrations are only slightly elevated above background levels. In Belew's Lake, for example, residual selenium concentrations of 1 to 4 µg Se/g in sediment and 1 µg Se/L or less in water were sufficient to cause WSS. The greatest threat is to young-of-the-year and yearling fish, because they reduce activity and feeding during winter to a greater extent than older age classes. The population and community-level implications are serious, because a large percentage (a third or more)

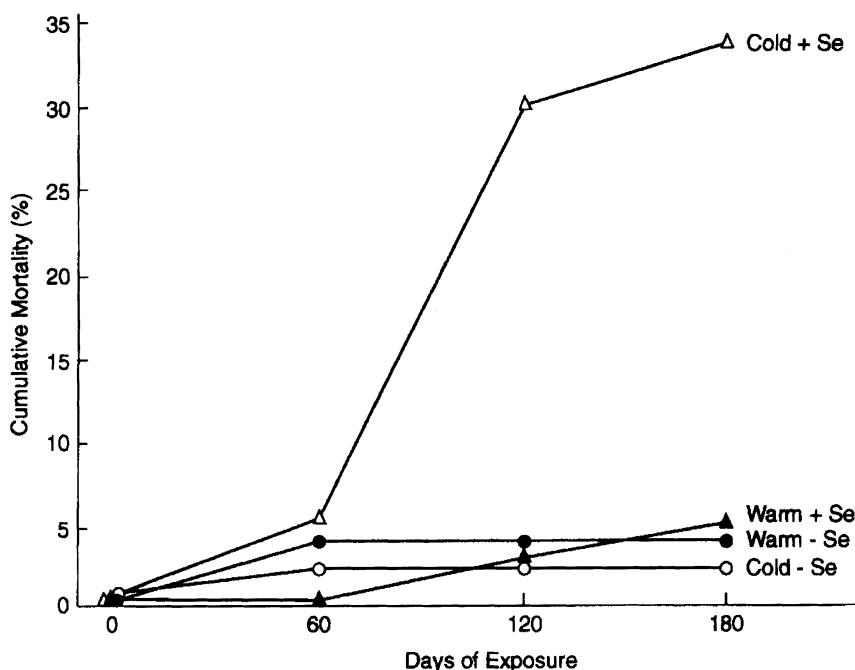


FIGURE 3.10. Increased toxicity of selenium in fish due to Winter Stress Syndrome. These data are for bluegill (*Lepomis macrochirus*) exposed to selenium in water and diet simultaneously. Treatments subjected bluegill to selenium while concurrently decreasing water temperature and photoperiod, mimicking the onset of winter conditions. Water temperature was held constant at 20°C throughout the study for exposures designated as "Warm"; temperature was decreased 2°C per week for 8 weeks and then held constant at 4°C in exposures denoted "Cold". The exposures for "Cold" treatments began with a 16:10 hr light:dark photoperiod which was gradually reversed to a 10:16 hr light:dark cycle by day 60 of the tests. +Se indicates fish which received 5.1 µg/g dry weight selenium in the diet (as seleno-L-methionine) and 4.8 µg/L in water (as a 1:1 ratio of selenate to selenite); -Se indicates fish that served as controls with regards to selenium. They received 0.8 µg/g in the diet and 0.16 µg/L in water (Lemly 1993b).

may die each winter from selenium levels that are nonlethal during other times of the year. In the short term, this seasonal mortality can offset reproduction and substantially alter year class strength. The cumulative effect over several years could shift fish community composition, causing centrarchids to be replaced by cool-water species, in the northern parts of their range, or by competing warm-water species that may not be as susceptible to WSS.

Fishes other than centrarchids are likely to experience WSS if they reduce feeding and activity during cold weather. In North America, field and laboratory observations suggest that young cyprinids (family Cyprinidae) and percids (family Percidae) would also be susceptible to selenium-induced WSS (Toneys and Coble 1980, Thompson et al. 1991). Basic knowledge of life history characteristics and feeding ecology, particularly for young-of-the-year, would allow identification of potentially vulnerable fish species in other temperate regions of the world.

Conclusions

The findings from Belews Lake serve as clear evidence of how selenium can rapidly, yet insidiously, impact fish populations. Several key symptoms of selenium poisoning were documented for the first time in the field at this location: for example, damage to gills and internal organs, cataracts and exophthalmus, teratogenic deformities, and reproductive failure. This case also demonstrated that selenium can accumulate and be biologically magnified to toxic levels when waterborne concentrations are only 5 to 10 µg Se/L. This has important implications for hazard assessment and water quality criteria. Lessons learned from Belews Lake, regarding the mode, rate, and persistence of selenium toxicity, have provided a foundation for protecting aquatic ecosystems in the United States and elsewhere for many years. For example, this information was instrumental in the US Environmental Protection Agency's decision to lower the United States national water quality criterion for selenium from 35 µg Se/L to 5 µg Se/L in the 1980s (United States Environmental Protection Agency [EPA] 1987). The Belews Lake event has also had a major influence on the development of the methods and guidelines presented in this book.

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Section II

Techniques for Evaluating Hazard

4

Protocol for Aquatic Hazard Assessment

Introduction

Agencies that manage natural resources and regulate water quality are becoming aware of the toxic threat posed by selenium, and much time and effort is being expended to monitor selenium concentrations in aquatic habitats. Once these data are collected, it is essential to conduct an overall evaluation and determine the degree of hazard present in order to identify appropriate management actions and develop local water quality criteria to protect aquatic life. However, few comprehensive hazard assessments have been completed. There are two likely reasons for this. First, it has been difficult for those conducting the monitoring programs to determine the toxicological significance of selenium residues in aquatic organisms. Locating, obtaining, and interpreting the results of selenium toxicity tests for a variety of aquatic species is a difficult task because of the time required. Fortunately, this type of information synthesis has been done. Guidelines are now available for evaluating selenium in food-chain organisms and fish and aquatic bird tissues based on an extensive amount of toxicological information (see Chapter 2). Interpreting selenium residues is no longer a problem.

The second reason is the lack of a broadly applicable procedure for conducting an aquatic hazard assessment of selenium. This has left investigators on their own, struggling with selenium monitoring data and trying to come up with a reasonable approach for characterizing hazard. Several approaches have been tried. Early attempts relied on comparisons between waterborne concentrations measured in the field and concentrations that were toxic to aquatic organisms in the laboratory (Cumbie and Van Horn 1978; Adams and Johnson 1981; Lemly 1982). However, the laboratory data did not adequately explain the greater toxic effects observed for fish in the field. Subsequent research on selenium indicated that food-chain bioaccumulation, dietary intake, and reproductive effects

should be given high priority in hazard assessment (Garrett and Inman 1984; Sorensen et al. 1984; Woock and Summers 1984; Lemly 1985a, 1985b; Baumann and Gillespie 1986; Gillespie and Baumann 1986; Heinz et al. 1987). Utilizing this and other information, some investigators have developed mathematical aquatic toxicity models (ATMs) to describe and predict selenium cycling and toxicity (eg, Bowie and Grieb 1991; Porcella et al. 1991; Peterson and Nebeker 1992; Bowie 1995). However, ATMs have a major drawback. They are complex and require considerably more information than is available from a typical contaminant monitoring program. For example, speciation of waterborne selenium, rates of selenium movement to and from sediments, biological uptake and excretion rates may be required. Thus, the potential for models to be routinely used in hazard assessment of selenium is quite limited. Another approach utilizes the United States national water quality criterion for selenium to derive a Hazard Quotient (EPA 1992), but this method is flawed because it uses geometric mean waterborne concentrations and a criterion value that is too high (see section on Evaluation, this chapter).

Despite the need to evaluate selenium hazards at many locations across the United States (Peterson and Nebeker 1992; Presser et al. 1994; Lemly 1999; Seiler et al. 1999), and elsewhere (see Chapter 1), there is no common assessment method in use. This has resulted in confusion and frustration for those involved in hazard assessment of both site-specific and regional selenium contamination problems (Sylvester et al. 1991; Presser et al. 1994). I developed the selenium Protocol described in this chapter to provide a simple, yet scientifically credible, technique for conducting aquatic hazard evaluations of this trace element. It includes the key parameters that are implicit in basic toxicological risk assessment, such as concentration and exposure, but it also integrates biotic and abiotic cycling components that are essential for site-specific hazard evaluation of selenium. The method generates numerical scores that can be compared between years and across sites and locations, thereby providing a consistent approach for evaluating hazard. The assessment focuses on food-chain bioaccumulation and associated reproductive impairment in fish and aquatic birds, which is the most sensitive biological endpoint for determining ecosystem-level hazards of selenium (Lemly 1993a, 1997a).

Definition of Aquatic Hazard

In the context of the Protocol, aquatic hazard is an expression of the toxic threat to fish and aquatic birds that use a specific habitat known or suspected of being contaminated with selenium. Hazard is characterized from two types of information: (1) the degree of selenium con-

tamination present, which is used as an estimate of the expected environmental concentration; and (2) the extent of fish and wildlife exposure to potentially toxic selenium concentrations. Exposure can be difficult to characterize precisely. However, the data set required for the Protocol helps with this problem because it specifies the use of egg selenium concentrations. This focuses the assessment on the reproductive cycle, which is the critical period of exposure necessary for toxic impacts. Hazard is maximized when the degree of contamination is high and exposure occurs during the breeding season.

Five categories of hazard are recognized in the Protocol based on the potential for reproductive impairment in fish and aquatic birds. High hazard denotes a toxic threat sufficient to cause complete or nearly complete reproductive failure in sensitive species of fish and aquatic birds (eg, sunfish, family Centrarchidae; trout, family Salmonidae; ducks, family Anatidae; stilts, family Recurvirostridae). Moderate hazard indicates a toxic threat of sufficient magnitude to substantially impair but not eliminate reproductive success; some species will be severely affected while others will be relatively unaffected. Low hazard denotes a toxic threat that could marginally affect the reproductive success of some sensitive species but leave most species unaffected. Minimal hazard indicates that no imminent toxic threat is identified, but concentrations of selenium are slightly elevated in one or more ecosystem components (water, sediment, benthic invertebrates, fish, birds) as compared to uncontaminated reference sites; continued comprehensive environmental monitoring is recommended. No hazard indicates that no toxic threat is identified, and selenium concentrations are not elevated in any ecosystem component; periodic baseline reconnaissance monitoring is recommended.

Data Required for Assessment

The Protocol requires a set of data for selenium concentrations measured in 5 ecosystem components: water ($0.45\text{ }\mu\text{m}$ filtered samples), sediments, benthic macroinvertebrates, fish eggs, and aquatic bird eggs. Incomplete data sets, that is, sets in which one ecosystem component is missing, will weaken the predictive power of the assessment, but it can still be performed. Depending on species and time of year, bird eggs and gravid fish ovaries may be very difficult or even impossible to obtain. In that case, selenium can be measured in bird livers and whole-body samples of fish, and the results converted to approximate egg concentrations using the following conversion factors: bird egg selenium = bird liver selenium $\times 0.33$; fish egg selenium = fish whole-body selenium $\times 3.3$ (Lemly and Smith 1987; Skorupa et al. 1996).

When the eggs of nesting birds are sampled, it is desirable to collect eggs of species that use a localized feeding area which represents the study site or Hydrological Unit (see Chapter 6) and will thus reflect the local conditions of exposure to dietary selenium. Species such as American coots (*Fulica americana*), grebes (*Podilymbus* sp.), and dabbling ducks (*Anas* sp.) are good choices in this regard. With fish, species that are resident in the area will suffice. Depending on location, minnows (Cyprinidae), sunfish (Centrarchidae), suckers (Catostomidae), catfish (Ictaluridae), and trout (Salmonidae) should be readily available and easy to sample. For some species, particularly cyprinids in streams, it will probably be necessary to select large individuals in order to obtain sufficient egg mass for analysis. Migratory species of birds and fish that could have recently arrived (within 2 weeks) at the study site from distant locations should be avoided because they may not have had time to accumulate tissue residues of selenium that reflect local exposure conditions (Heinz et al. 1990).

Data for the Protocol can be from a one-time sampling effort or from an ongoing monitoring program of considerable duration. If the latter case exists, I recommend that the most recent data (1–2 years) be used, since it would yield the best estimate of current hazard. There is no minimum or maximum number of measurements as long as the specified ecosystem components are sampled (see next section). However, spatially complete sampling efforts will result in hazard ratings that reflect the range of habitat types and environmental conditions that are present. Moreover, the Protocol assesses hazard based on the highest concentrations of selenium found; thus, it is important to take a sufficient number of samples to detect these upper levels. Hazard estimates generated from robust data sets will also lend credibility to ensuing management actions or follow-up studies, should they be needed.

Eggs are a key component in the assessment because change in reproductive success is the most sensitive biological response for determining selenium toxicity to fish and aquatic birds (Lemly 1997a). Selenium ingested in the diet is readily transmitted from the parent to developing eggs, where it can cause teratogenic deformities and embryomortality. Complete reproductive failure can occur in the absence of observable toxic effects on the adults (Gillespie and Baumann 1986; Heinz et al. 1987, 1989; Woock et al. 1987; Coyle et al. 1993). The Protocol thus incorporates a sensitive indicator that reflects the mode of toxicity responsible for the most serious community and ecosystem-level effects.

In order for the assessment to be reliable, the data for selenium concentrations must also be reliable. Analytical methods used to determine selenium should be sensitive and under strict, documented quality control. Methods for water and tissues should have limits of detection in the sub- μg Se/L range and sub- μg Se/g range, respectively. This is important because an error of only 1 μg Se/L or 1 μg

Se/g in measured concentrations could cause the hazard rating for an environmental component to fall in the wrong category. Propogation of this error throughout the 5 environmental components used in the Protocol could result in an inappropriate final hazard characterization. Acceptably sensitive analytical methods include neutron activation, hydride-generation atomic absorption spectrophotometry, and cathodic stripping voltammetry (McKown and Morris 1978; Jarzabek and Kublik 1982; Brooks et al. 1983). Tissue and sediment preparation techniques appropriate for use with these methods are available (eg, Grant 1981; May 1982; Krynnitsky 1987; Fio and Fujii 1988; Brumbaugh and Walther 1989). Inductively coupled argon plasma spectroscopy (ICAP) should be avoided if possible. Although ICAP scans are good for reconnaissance-level monitoring to identify field sites of concern, the detection limits are generally too high and the precision too low for determining concentrations to be used in the hazard assessment. Establishing a contract for selenium measurements with a reputable environmental laboratory is a desirable cost-effective alternative to developing in-house analytical chemistry capabilities.

Description of the Protocol

Selenium concentrations measured in samples collected from a study site, preferably across a Hydrological Unit (see Chapter 6), are compared to hazard profiles (Figs. 4.1–4.5). These profiles are based on the results of extensive field and laboratory studies that investigated selenium bioaccumulation and toxic thresholds across a wide range of habitat types and environmental conditions. Waterborne concentrations are compared to the profile for water, sediment concentrations are compared to the profile for sediment, and so forth. A hazard rating is determined for each of the 5 ecosystem components based on where the highest concentrations of selenium in the samples fall on the hazard scale (high, moderate, low, minimal, none). Individual hazard ratings are then given a numerical score: high hazard = 5, moderate hazard = 4, low hazard = 3, minimal hazard = 2, and no identifiable hazard = 1. A final hazard characterization is determined by adding together the 5 individual scores and comparing the total to the following evaluation criteria:

No hazard = 5

Minimal hazard = 6–8

Low hazard = 9–11

Moderate hazard = 12–15

High hazard = 16–25

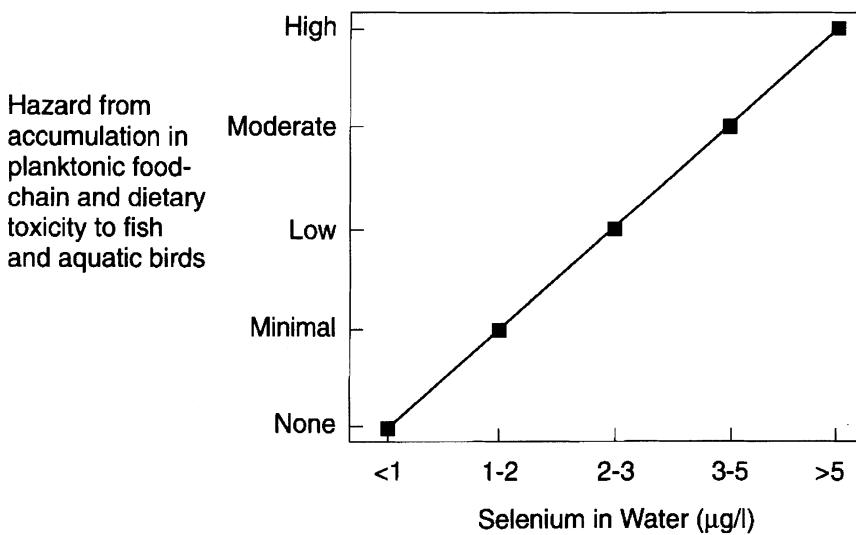


FIGURE 4.1. Hazard profile for selenium accumulation from water into the planktonic food chain and resultant dietary toxicity to fish and aquatic birds. Water-borne selenium concentrations are for $0.45 \mu\text{m}$ filtered samples. This and subsequent hazard profiles (Figs. 4.2–4.5) are based on a synthesis of toxicity, bioaccumulation, and sediment data from the references listed at the end of this chapter.

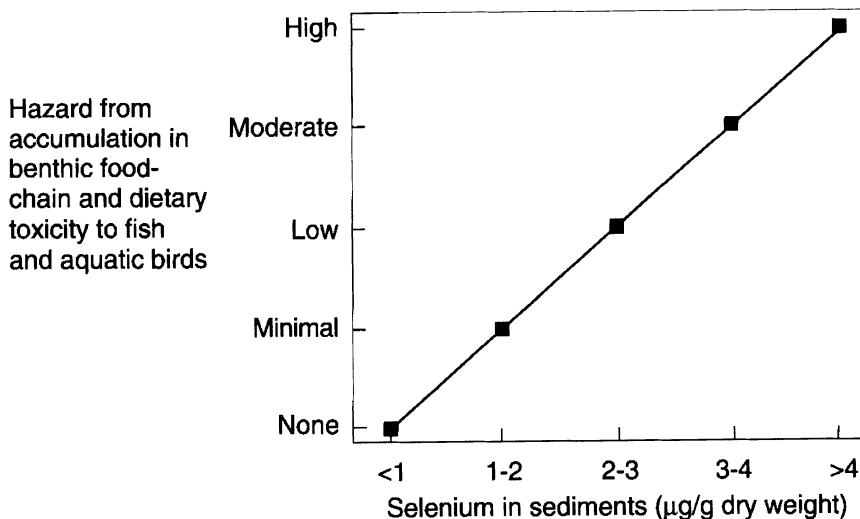


FIGURE 4.2. Hazard profile for selenium accumulation from sediments into the benthic food chain and resultant dietary toxicity to fish and aquatic birds.

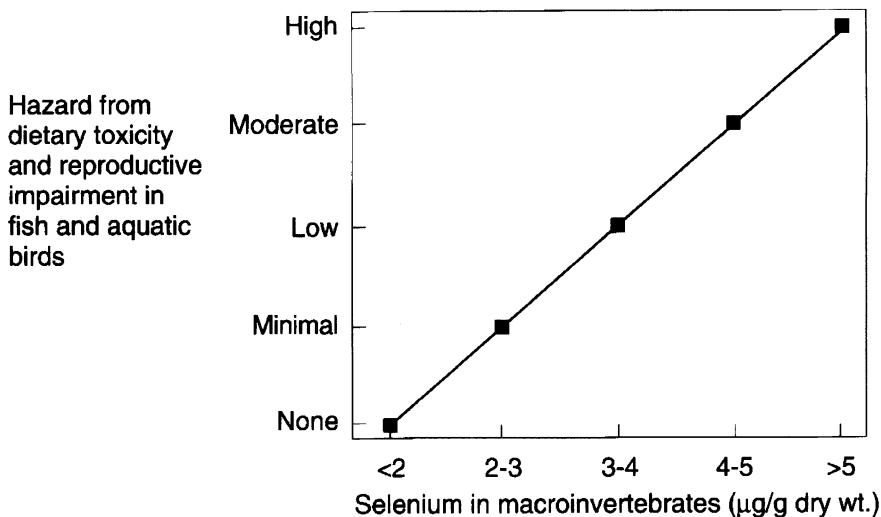


FIGURE 4.3. Hazard profile for dietary toxicity and reproductive failure in fish and aquatic birds from ingestion of selenium-contaminated macroinvertebrates.

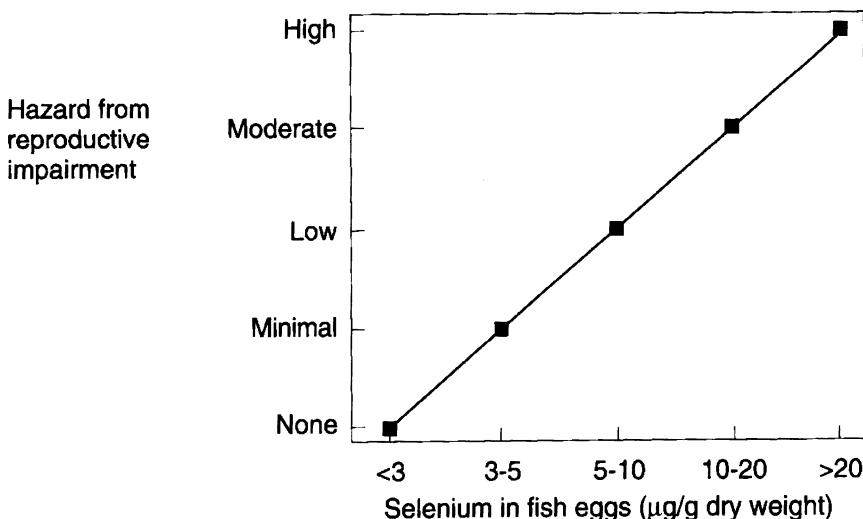


FIGURE 4.4. Hazard profile for selenium-induced reproductive impairment in fish.

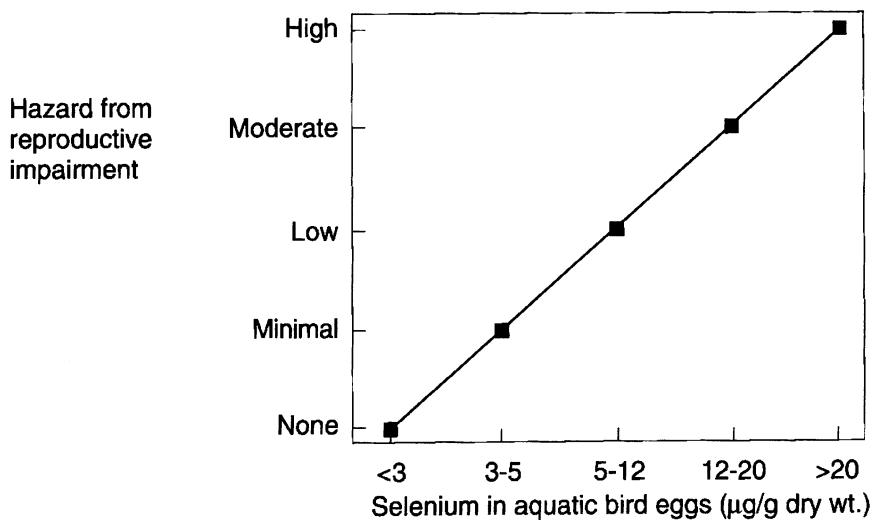


FIGURE 4.5. Hazard profile for selenium-induced reproductive impairment in aquatic birds.

The assessment thus integrates hazard component scores and provides an ecosystem-level evaluation.

In some situations it may be impossible to obtain data for all 5 ecosystem components, particularly when the method is applied to monitoring data that have already been collected. When selenium data are missing for one ecosystem component, the following evaluation criteria should be used:

No hazard = 4

Minimal hazard = 5–7

Low hazard = 8–10

Moderate hazard = 11–14

High hazard = 15–20

This latter set of criteria was not derived by prorating the numbers in the other set used for 5-component evaluation (eg, subtracting 5 from high hazard, 4 from moderate hazard, 3 from low hazard, 2 from minimal hazard, and 1 from no hazard), but rather, by considering multiple routes of exposure and the resultant aggregate hazard. For both sets of final hazard numbers, the intervals are based on the scores for the individual ecosystem components but they are not a simple average or midpoint. For example, a final hazard rating of "high" can

occur even though each of the individual components was rated "moderate". The rationale for this "weighting" is that 3 distinct routes of exposure are possible for selenium: water, planktonic food chain, and detrital food chain. Based on field evidence of bioaccumulation and toxic effects, the hazard of all 3 together should be greater than if each is present separately. Thus, the aggregate hazard for the ecosystem as a whole can be greater than the hazard for individual components. This is analogous to synergism, that is, the parts (the hazard scores for the components) are not simply additive.

The Protocol should not be used if data are missing for more than 1 ecosystem component. Accepting only 3 components could mean that data for both fish and birds were absent. The objective of the Protocol is to estimate ecosystem-level hazard by focusing on bioaccumulation in fish and aquatic birds. If one of these components is missing, the predictive power of the assessment will not be as strong as if both are included, but the assessment will still have validity. However, if both are missing, the procedure does not meet its objective and, therefore, is invalid. One other precaution is necessary. An extraneous ecosystem component should never be substituted for one that was specified for use in the Protocol (for example, zooplankton should not be substituted for benthic macroinvertebrates), because the hazard profiles were constructed for use with the indicated ecosystem components only. Substitutions will invalidate the results.

Depending on the needs of the investigator, the Protocol can be applied to data collected for a single sampling site or to pooled data from a monitoring effort covering a large area. The Protocol can thus address several levels of landscape scale and provide site-specific, as well as regional, assessment of selenium hazard. Comparisons can be made between locations and years, and follow-up assessments can be made to evaluate the success of management actions and cleanup activities at selenium-contaminated sites.

Accuracy of Hazard Ratings

Comparison to Other Methods

The Protocol is an important new method for assessing selenium hazards to aquatic life. It is designed for application across a wide range of habitat types and environmental conditions. In order for this objective to be met, it must provide hazard ratings that are verified in the field and are more accurate than those generated by other methods. Prior to its development, the other major technique available was the USEPA's Hazard Quotient (HQ) procedure (EPA 1992). The HQ technique is generally considered to be a good method to use when there is a USEPA

water quality criterion for the substance under evaluation, and it has been widely applied for aquatic risk assessment in the United States for many years. However, there are substantial differences between the Protocol and HQ. The Protocol focuses on food-chain bioaccumulation and reproductive impairment in fish and aquatic birds whereas HQ primarily uses information on waterborne selenium. Comparison of the 2 methods gives additional insight into the validity of the Protocol for hazard assessment.

The HQ for selenium is calculated by dividing the geometric mean measured or expected environmental waterborne concentration by the United States national water quality criterion for chronic exposure (5 µg Se/L). The rationale of this technique is that, if waterborne concentrations of selenium exceed the criterion, substantial hazard exists. If concentrations are below the criterion, then minimal hazard is expected. This method provides a quantitative estimate of the hazard associated with single chemicals or elements, primarily from a waterborne route of exposure. However, the USEPA process for deriving national water quality criteria does allow for inclusion of data that would account for bioaccumulation. In the case of selenium, field data on food-chain transfer and reproductive toxicity to fish were included in the derivation, and were largely responsible for the criterion being lowered to its current value of 5 µg Se/L (EPA 1987). Thus, even though HQ uses only waterborne values, it reflects the extent to which the United States national criterion is protective of dietary and reproductive hazards. Guidelines for interpreting HQ calculations are:

HQ < 0.1, No hazard exists

HQ = 0.1–1.0, Hazard is low

HQ = 1.1–10, Hazard is moderate

HQ > 10, Hazard is high

Field Sites and Hazard Ratings

Selenium data from three ecologically distinct systems were used in the comparison: wetlands (Stephens et al. 1988, 1992; Hoffman et al. 1990; Pelz and Waddell 1991; Waddell and Stranger 1992), rivers (Finger 1995), and reservoirs (Lemly 1997b). Selenium concentrations in whole-body fish samples and aquatic bird livers were converted to equivalent egg concentrations for use in the Protocol method (Lemly and Smith 1987: whole-body values × 3.3 for fish and liver values × 0.33 for birds).

Hazard ratings generated by the two methods are given in Table 4.1. The Protocol indicated greater hazard than HQ for all locations except Leota Bottom and High Rock Lake, which are considered to be uncon-

taminated references sites (Stephens et al. 1992; Lemly 1997b). In 4 cases, no hazard was indicated by HQ, whereas moderate to high hazard was indicated by the Protocol. At these 4 locations, concentrations of selenium in water were very low, but selenium was elevated in one or more of the other ecosystem components used in the Protocol. The Protocol method was compared with HQ and evaluated for 8 sites for which full data sets were available (all 5 ecosystem components), as well as 3 sites for which one component was missing (aquatic bird eggs). The results were similar in all cases; the Protocol consistently indicted greater hazard than HQ.

Verification of Results

The data sets used in this analysis represent a wide variety of habitat types and ecological conditions, ranging from shallow freshwater marshes to large riverine and reservoir systems. They also represent levels of selenium contamination ranging from clean reference sites to heavily contaminated wetlands. This range of conditions should cover the spectrum of pathways by which selenium cycles, accumulates, and causes toxicity in aquatic habitats. It is important to examine different conditions because a dominant pathway in one system may be relatively insignificant in another (Lemly and Smith 1987). Hazard assessment methods that are overly sensitive to one particular pathway will give inaccurate estimates. The data sets examined provide a good perspective from which to judge the performances of the two techniques.

Hazard estimates from HQ analysis were consistently lower, usually much lower, than those generated from the Protocol. Information on the toxic effects of selenium on fish and aquatic birds at the field sites can be used to check the results and determine which method produced the most valid estimates of hazard.

Example 1.

The contaminated sites in Utah (Roadside Ponds and Sheppard Bottom) have produced acute and chronic selenium toxicosis and reproductive impairment in fish and aquatic birds (Waddell and Stanger 1992; Hamilton et al. 1996). The severity of these biological effects, as evidenced by the presence of both acute and chronic toxicity, shows that the hazard from selenium was high. The Protocol indicated high hazard at both sites, whereas HQ indicated low to moderate hazard.

Example 2.

Decreased survival of juvenile ducks due to selenium in the diet and associated decreases in waterfowl production were identified at Fernley,

TABLE 4.1. Hazard assessment of selenium using Protocol and Hazard Quotient (HQ) methods.

Site and ecosystem component	Selenium concentration ^a	Evaluation by component		Totals for the site		HQ Method	
		Hazard	Score	Score	Hazard	HQ	Hazard
Wetlands							
Leota Bottom, UT	<1-3	Low	3	-	Low	0.23	
Water	0.7-1.0	None	1	11	Low		Low
Sediments	1.0-3.0	Minimal	2				
Invertebrates	2.0-4.0	Minimal	2				
Fish eggs	2.0-7.0	Low	3				
Bird eggs							
Roadside Ponds, UT							
Water	9-93	High	5	25	High	5.6	Moderate
Sediments	7-41	High	5				
Invertebrates	12-72	High	5				
Fish eggs	75-120	High	5				
Bird eggs	12-120	High	5				
Sheppard Bottom, UT							
Water	3-4	Moderate	4	21	High	0.61	Low
Sediments	0.6-3.0	Low	3				
Invertebrates	3.0-33	High	5				
Fish eggs	8-27	High	5				
Bird eggs	1-17	Moderate	4				
Stillwater WMA ^b , NV							
Water	<1	None	1	14	Moderate	0.05	None
Sediments	0.2-0.8	None	1				
Invertebrates	0.3-7.0	High	5				

Continued

	Fish eggs	2.9–12.2	Moderate	4		
	Bird eggs	0.9–10.6	Low	3		
Fernley, WMA, NV						
Water	<1	None	1	16	High	0.04
Sediments	0.1–0.6	None	1	16	High	None
Invertebrates	3.5–13.0	High	5			
Fish eggs	10.8–36.3	High	5			
Bird eggs	1.9–13.0	Moderate	4			
Humboldt WMA, NV						
Water	<1	None	1	16	High	0.04
Sediments	0.3–1.2	Minimal	2			
Invertebrates	2.5–5.1	High	5			
Fish eggs	6.3–12.9	Moderate	4			
Bird eggs	2.3–15.8	Moderate	4			
Rivers						
Animas River, CO, NM						
Water	1–20	High	5			
Sediments	0.1–2.3	Low	3	14	Moderate	0.28
Invertebrates	1.8–2.9	Minimal	2			
Fish eggs	3.0–15.8	Moderate	4			
La Plata River, CO, NM						
Water	1–12	High	5			
Sediments	0.1–0.95	None	1	13	Moderate	0.22
Invertebrates	1.1–2.2	Minimal	2			
Fish eggs	2.6–39.6	High	5			
Mancos River, CO, NM						
Water	2–29	High	5			
Sediments	0.2–0.8	None	1	16	High	2.1
Invertebrates	1.8–11.2	High	5			Moderate
Fish eggs	5.6–46.2	High	5			

Continued

TABLE 4.1. (Continued). Hazard assessment of selenium using Protocol and Hazard Quotient (HQ) methods.

Site and ecosystem component	Selenium concentration ^a	Protocol Method			HQ Method		
		Evaluation by component		Totals for the site	Score	HQ	Hazard
		Hazard	Score				
Reservoirs							
Belews Lake, NC							
Water	<1	None	1	18	High	0.08	None
Sediments	0.1–4.3	High	5				
Invertebrates	2.0–5.9	High	5				
Fish eggs	3.6–12.8	Moderate	4				
Bird eggs	2.5–11.0	Low	3				
High Rock Lake, NC							
Water	<1	None	1	5	None	0.03	None
Sediments	0.1–0.3	None	1				
Invertebrates	0.9–1.5	None	1				
Fish eggs	1.4–2.9	None	1				
Bird eggs	1.7–2.9	None	1				

^aSelenium concentrations in µg/L (parts per billion) for water, µg/g (parts per million) dry weight for sediments, invertebrates, and eggs.

^bWMA denotes wildlife management area.

Humboldt, and Stillwater wildlife management areas in Nevada. Irrigation drainage containing elevated concentrations of selenium was determined to be toxic to young fish and other aquatic organisms, and chronic selenosis was diagnosed in some waterbirds (Hallock and Hallock 1993). The toxic effect of selenium at these sites shows that a substantial biological hazard existed. The Protocol rating indicated high hazard for 2 sites (Fernley and Humboldt) and moderate hazard for the other (Stillwater). The HQ analysis indicated that no hazard was present at any of the sites.

Example 3.

Belews Lake has a well documented history of selenium-induced teratogenesis and reproductive impairment in fish (Lemly 1985a, 1993a, 1997b). Selenium concentrations were somewhat elevated in bird eggs but did not reach levels that substantially impair reproduction. However, the toxic effect of selenium on fish was devastating (19 species were eliminated) and persistent, indicating high hazard for the site. The Protocol gave a high hazard rating for Belews Lake, whereas HQ analysis indicated that no hazard was present.

Evaluation

Hazard estimates from HQ analysis were incorrect for two reasons: (1) shortcomings in the USEPA water quality criterion, and (2) a basic flaw in the HQ approach as it applies to selenium. Although the USEPA criterion ($5 \mu\text{g Se/L}$) was developed in the mid-1980s using field data that reflected current (pre-1988) information on bioaccumulation, more recent information indicates that $5 \mu\text{g Se/L}$ is too high to protect sensitive species of fish and aquatic birds from reproductive toxicity and the effects of Winter Stress Syndrome (Skorupa and Ohlendorf 1991; Lemly 1993a, 1993b, 1996; Skorupa et al. 1996; NIWQP 1998; Hamilton and Lemly 1999). The USEPA criterion is now well over 10 years old, and several research studies and reviews published since then (eg, Skorupa and Ohlendorf 1991; Peterson and Nebeker 1992; Lemly 1993a, 1997a; Skorupa 1998) indicate that the value should probably be somewhere in the $1\text{--}2 \mu\text{g Se/L}$ range, especially for wetlands and impoundments where bioaccumulation is maximized. Had a value of $2 \mu\text{g Se/L}$, for example, been used instead of $5 \mu\text{g Se/L}$, the HQ numbers would be quite different, and the hazard estimates would more closely match those indicated by the Protocol for sites in Utah and New Mexico. Thus, as it is currently practiced, HQ analysis will consistently underestimate selenium hazard because it relies on an invalid USEPA water quality criterion.

A basic flaw with the HQ method is that it uses geometric mean waterborne concentrations of selenium. A large part of the selenium bioaccumulation and associated toxic impact to fish and wildlife in an aquatic system can result from one-time or periodic waterborne inputs that have high concentrations relative to the long-term mean (Garrett and Inman 1984; Sorensen 1988; Presser et al. 1994). On a mass-balance basis, these spikes in concentration deliver a disproportionately large amount of selenium that can load sediments and be cycled into detrital food chains long after the waterborne spike is gone (Lemly and Smith 1987; Lemly 1997b). Once in the aquatic system, this “pulse” of selenium may be all that is necessary to rapidly escalate bioaccumulation and cause a cascade of impacts. The toxicity threshold for selenium is very steep. An increase of only a few $\mu\text{g Se/L}$, which may represent the difference between mean and maximum concentrations in HQ analysis, can cause a totally different, much more serious impact on fish and aquatic birds (Lemly 1985b, 1993a; Skorupa and Ohlendorf 1991). It is critical to capture this underlying principle of selenium ecotoxicology when conducting hazard analyses and risk assessments. Relying on mean waterborne concentrations diminishes and masks the importance of the peaks in terms of selenium loading, bioaccumulation, and toxic threat. Results are biased, and hazard is seriously underestimated. This could lead to risk management decisions that would not protect fish and wildlife from selenium toxicity. In order to make the best decisions, resource managers need to know what the maximum hazard is, not the mean hazard. Thus, it is essential to estimate hazard using the highest concentrations of selenium measured in the samples. The USEPA’s HQ approach does not do this.

Hazard profiles used in the Protocol method integrate updated information on selenium bioaccumulation and reproductive toxicity to fish and wildlife. Hazard is characterized in terms of the potential for food-chain bioaccumulation and reproductive impairment in fish and aquatic birds, which are the most sensitive biological responses for estimating ecosystem-level impacts of selenium contamination (Lemly 1993a). Hazard estimates generated from the Protocol are not driven by a single concentration value or water criterion for effect/no effect evaluations, as is the case for the HQ method. There is no implicit assumption or evaluation of toxicity related to direct waterborne exposure. Rather, water concentrations are used to estimate hazard associated with bioaccumulation in the planktonic food chain. Each of the ecosystem components used in the Protocol is given equal weight; thus, spatial or temporal variation in the relative dominance of one selenium cycling pathway will be accounted for in the assessment. This is a key point that differentiates the two methods. The Protocol utilizes multiple parameters that determine selenium bioaccumulation and exposure on a

site-specific basis, whereas the HQ method is more generic and is tied to a single criterion that is assumed to be protective under all exposure conditions.

The Protocol's hazard estimates closely matched the field evidence of selenium toxicity to both fish and aquatic birds; the HQ estimates did not. The inability of HQ to correctly assess hazard reflects the outdated USEPA water quality criterion. Data from the field sites show, quite clearly, that bioaccumulation of selenium in aquatic food chains and resultant toxicity to fish and wildlife can occur when concentrations of waterborne selenium are well below the current United States water quality criterion. This indicates that a revision (lowering) of the USEPA criterion is necessary, either at a national level, or at a local site-specific level (see Chapter 7). Even then, there will likely be many locations where HQ will perform poorly because of the potential for high selenium bioaccumulation with little detectable elevation in waterborne concentrations (eg, the sites in Nevada). This situation can develop because of the presence of ultra-trace amounts (<1 µg Se/L) of dissolved organic selenium that cause disproportionately high bioaccumulation relative to the inorganic forms of selenium (Besser et al. 1989, 1993), or because of a very strong sediment-detrital buildup of selenium that is possible under certain limnological conditions (Lemly and Smith 1987; Lemly 1997b). Accounting for this scenario in selenium cycling is essential for accurate hazard assessment. **The Protocol is sensitive to all of the bioaccumulation pathways for selenium, and its predictions are accurate regardless of waterborne concentrations.**

Another problem with HQ analysis, due to its dependence on waterborne selenium, is that it does not accurately evaluate the success of remediation at contaminated sites. It is possible for waterborne selenium to fall substantially with little or no accompanying reduction in the overall ecosystem-level hazard to fish and wildlife. For example, hazard to fish remained high at Belews Lake, NC, because of selenium-laden sediments, even though waterborne concentrations dropped to near background levels because of site management actions that eliminated selenium inputs (Lemly 1997b). The HQ ratings suggest that remediation has been successful and no hazard remains, because waterborne selenium is well below the USEPA criterion. However, this is clearly not the case. The Protocol correctly identified and rated the hazard that persists at this site; HQ analysis did not. Thus, there are several important limitations in the application of HQ analysis to aquatic hazard assessment of selenium.

A possible criticism of the Protocol is that it requires considerably more data than HQ (ie, water, sediments, invertebrates, fish, and birds for the Protocol; just water for HQ). However, given the serious impacts of selenium on fish and wildlife that have resulted because of environmental management actions that were based on inadequate

risk assessment (eg, Lemly 1985b, 1993b, 1994), the effort and expense of collecting and analyzing additional samples is well justified from an ecological perspective. Moreover, the components specified for use in the Protocol are typically sampled in the course of routine environmental monitoring. To adequately address environmental safety issues it is necessary to use hazard assessment procedures that yield the most ecologically sound information. For selenium, the most reliable method is the Protocol.

Conclusions

The Protocol provides a straightforward consistent technique for evaluating the aquatic hazard of selenium. Although the method is simple and easy to carry out, it is based on a large body of field and laboratory research data. Hazard is characterized from sensitive biological endpoints linked to important ecosystem-level effects. The integrative nature of the method (ie, its use of information from 5 ecosystem components) makes it responsive to site-specific differences in selenium cycling, bioaccumulation, and toxicity. The Protocol can be applied to all aquatic habitats, and it produces accurate hazard ratings. It is an excellent tool for screening a wide variety of sites and identifying those that need further attention. It is important to understand that the Protocol does not determine toxic impacts to biota. Rather, it characterizes the potential for toxic impacts to occur and reveals the relative magnitude of threats across different habitats and seasons. Field verification is necessary to determine actual impacts, which are influenced by the sensitivity of local species to selenium and site characteristics that regulate food-chain bioaccumulation. A good technique to use in this verification process is the Teratogenic Deformity Index, which evaluates impacts on fish populations (see next chapter).

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5

Teratogenic Deformity Index for Fish

Introduction

Excessive selenium can cause a wide variety of toxic effects in fish (see Chapter 3). The most prominent outwardly visible symptom of selenium poisoning is teratogenic deformities. When observed in the field concurrently with elevated tissue selenium residues, terata can be used as an easily recognized, reliable indicator of selenium toxicity. Because of this characteristic, they are a useful tool for evaluating selenium hazards in aquatic ecosystems. However, a simple notation of the presence of deformities is not an assessment of mortality and potential long-term impacts on fish populations. Such an assessment is needed to fully understand the level of ecological damage that has taken place. To make such an assessment, it is necessary to link the prevalence of terata with corresponding degrees of mortality. This linkage can be derived by examining laboratory and field studies that document teratogenic mortality in response to selenium exposure. I have reviewed that information and used it to develop the Teratogenic Deformity Index (TDI) presented in this chapter. The TDI provides field verification of reproductive failure and assesses the magnitude of toxic impacts to current and future generations of fish. The examples presented illustrate how TDI is applied to fish population sampling data.

Occurrence and Persistence of Teratogenic Effects

Teratogenic deformities are a permanent pathological marker of selenium poisoning. They are congenital malformations that occur due to excessive selenium in eggs. The process begins with the diet of the parent fish. Excess dietary selenium ($>3 \mu\text{g Se/g}$) causes elevated concentrations of selenium to be deposited in developing eggs, particu-

larly in the yolk. When the eggs hatch, larval fish rapidly utilize the selenium-contaminated yolk as both an energy supply and a source of protein for building new body tissues. Hard and soft tissues may be deformed if the molecular structure of the protein building blocks has been distorted due to substitution of selenium for sulfur. Some tissues may not be generated at all, resulting in missing body parts.

The prevalence of teratogenic deformities increases rapidly once selenium concentrations in eggs exceed 10 µg Se/g (Woock et al. 1987). Hatchability of eggs is not affected by elevated selenium, even though there may be a high incidence of deformities in resultant larvae and fry, and many may fail to survive (Gillespie and Baumann 1986; Coyle et al. 1993). The time of induction of teratogenesis is when larval fish are relying on their attached yolk sac for nourishment and development. Once external feeding begins, the potential for teratogenic effects declines and soon disappears. Feeding excessive selenium (up to lethal levels) to fry or juvenile fish as they are growing will not cause teratogenic malformations to occur (Hamilton et al. 1990; Cleveland et al. 1993). Moreover, dietary selenium levels sufficient to load eggs beyond teratogenic thresholds (a diet including 5–20 µg Se/g) do not cause teratogenesis in parent fish or otherwise generally affect their health or survival (Coyle et al. 1993). Thus, the teratogenic process is strictly an egg-larvae phenomenon. Because of this, teratogenesis can be a very subtle, but important, cause of reproductive failure in fish. Entire populations may disappear with little evidence of "toxicity", since major impacts to early life stages can be taking place at the same time that adult fish appear healthy (Cumbie and Van Horn 1978; Lemly 1985).

Mortality of larval fish can be high if the teratogenic defects are severe enough to impair critical body functions (Woock et al. 1987). However, in some cases the abnormalities may not be life threatening, and the malformations can persist into juvenile and adult life stages (Lemly 1993). This is likely restricted to locations where there is little threat from predators, since all but the most subtle deformities would probably compromise a fish's ability to feed and avoid predators. Thus, in assessing the prevalence of teratogenic defects, it is important to focus on the earliest life stages, that is, newly emerging larvae and young fry.

Identifying Teratogenic Deformities

Teratogenic deformities can occur in most, if not all, hard or soft tissues of the body. However, some of the most conspicuous (consequently, the most diagnostic) are found in the skeleton, fins, head, and mouth. These typically involve: (1) lordosis, concave curvature of the lumbar region of the spine; (2) scoliosis, lateral curvature of the spine; (3) kyphosis, convex curvature of the thoracic region of the spine result-

ing in "humpback" condition; (4) missing or deformed fins; (5) missing or deformed gills or gill covers (opercle); (6) abnormally shaped head; (7) missing or deformed eyes; (8) deformed mouth. (See Figs. 3.5–3.9, Chapter 3, for a description of deformities.)

In general, a careful fish-in-hand inspection is sufficient to diagnose any of the major teratogenic deformities. However, to make the diagnosis for larvae and fry or small species (eg, small cyprinids, poeciliids), or in situations when it is necessary to tabulate all of the subtle, less overt symptoms (eg, slightly deformed fins, opercles, etc.) the fish must be carefully examined with the aid of a dissection microscope. This is particularly true for larval fish. Some of their undeveloped features could erroneously be considered a defect, when in fact, they are a consequence of a premature life stage, not selenium teratogenesis. However, this is not a serious concern because larval fish have distinctive patterns of development that quickly become apparent to the investigator looking for teratogenesis. With a bit of hands-on experience, the true teratogenic defects are easily distinguishable even in young fish (see Fig. 3.5, Chapter 3).

There are some other symptoms of selenium poisoning that may be confused with teratogenic effects. These are generally thought to represent acute toxic responses to high doses or tissue concentrations of selenium rather than true teratogenic effects. The most common of these symptoms are: (1) edema, swollen and distended abdomen due to accumulation of fluid in the visceral cavity; (2) exophthalmus (bulging or protruding eyes) due to accumulation of fluid in the eye sockets; and (3) cataracts, which appear as a white coating on the eyes (see Figs. 3.3–3.5, Chapter 3). All of these symptoms may be present concurrently, along with the true teratogenic effects. Particular care must be exercised when examining larval fish. Edematous larvae with distended abdomens are common (eg, Bryson et al. 1984; Gillespie and Baumann 1986; Pyron and Beitinger 1989). This condition may progress to, or be associated with, the expression of terata, but the edema itself does not constitute a teratogenic defect. However, severe edema is usually accompanied by deformity of the spine (most often lordosis) or soft tissues in the abdomen. The prevalence of edema and terata can be virtually the same (eg, Schultz and Hermanutz 1990) or quite different (eg, Hermanutz et al. 1992). Thus, one should not assume a 1:1 relationship. Reasonable caution, that is, close inspection and comparison with normal larvae, will prevent inaccurate diagnoses.

In order to draw a conclusion of selenium-induced teratogenesis, the visual indicators and symptoms (deformities) must be corroborated with the presence of elevated concentrations of selenium in tissues. Concentrations in the range of 10 to 20 µg Se/g or greater (whole-body homogenate) would be sufficient to confirm the diagnosis. This corresponds to concentrations of about 6 to 12 µg Se/g in muscle (fillets) or 20 to 40 µg Se/g in visceral tissues, including the liver. Although mea-

surement of tissue concentrations is essential, it is not necessary to conduct extensive surveys on hundreds of fish. Analysis of 6 samples per fish species (eg, 6 adults or juveniles with teratogenic deformities or 6 composites for teratogenic larvae/fry) is sufficient to reveal the range of concentrations likely to be encountered (Lemly 1993, 1997).

Terata-Mortality Relationships

A considerable amount of data is available for assessing or predicting the impact of selenium-induced teratogenesis on fish populations. Laboratory studies provide important information on the relationships among egg concentrations of selenium, prevalence of teratogenic deformities in larvae, and associated mortality. These studies are of three types; (1) those in which captive adult fish were fed selenium-laden diets or exposed to high-selenium water and then allowed to spawn in indoor tanks (Bryson et al. 1984, 1985a, 1985b; Wock et al. 1987; Pyron and Beiting 1989); (2) those in which outdoor artificial streams were dosed with waterborne selenium, providing for exposure of adult fish to natural food-chain selenium prior to spawning (Schultz and Hermanutz 1990; Hermanutz 1992; Hermanutz et al. 1992); and (3) those in which adult fish were taken from selenium-contaminated aquatic habitats and spawned artificially (ie, eggs and milt were removed and mixed) and the resultant hatch was monitored (Gillespie and Baumann 1986).

The field data come from Belews Lake, NC, in the United States. This lake was impounded in the early 1970s to serve as a cooling reservoir for a large coal-fired electric generating station (2250 megawatt generating capacity). Fly ash produced by the power plant was disposed in a settling basin from which effluent containing 100 to 200 µg Se/L (about 80% selenite) was released in return flows to the lake. This selenium bioaccumulated in aquatic food chains, and within two years, the fishery of Belews Lake began to decline. Of the 20 species of fish originally present in the reservoir, 19 were eliminated due to dietary toxicity and reproductive failure. This pattern of selenium contamination from the power plant and resultant poisoning of fish persisted from 1974 to 1986 (Lemly 1985, 1997). In late 1986, the power company changed operations for fly-ash disposal, and selenium-laden effluent no longer entered the lake. In the following years, selenium levels fell substantially, and the fishery began to recover (augmented by artificial stocking), but sediments and associated benthic-detrital food chains remained moderately contaminated, and there were residual effects on the fishery, including persistent teratogenic deformities.

Teratogenic assessment was used to evaluate the reproductive success of fish and determine the degree of impact in Belews Lake in

1975, 1978, 1982, 1992 and 1996 (Lemly 1993, 1997). The 1975 survey was conducted during the period of initial selenium contamination of the reservoir, before the fishery experienced serious decline. This was the only survey made when the original assemblage of fish species was still present in the lake. This data set is quite informative because it documents levels of teratogenesis in a wide range of species that represent various feeding modes and trophic positions. Selenium concentrations in whole-body fish samples (juveniles and adults) were high ($40\text{--}65 \mu\text{g Se/g}$ dry weight), as was the prevalence of teratogenic deformities (up to 55%). Terata were present in all of the 19 species examined. Surveys conducted in 1978 and 1982 yielded similar results, although only 4 fish species remained in 1978, and 6 in 1982. Selenium concentrations were very high (up to $130 \mu\text{g Se/g}$ dry weight), and were closely paralleled by the prevalence of teratogenic deformities, which ranged up to 70% (juvenile and adult fish). The survey in 1992 indicated that gradual recovery was taking place, but there were still only 9 of the original 20 species present, and numerical abundance was quite low. Concentrations of selenium in fish had fallen to 11 to $20 \mu\text{g Se/g}$ and the incidence of terata did not exceed 11% (juveniles and adults). Fish were successfully reproducing and it was soon possible to collect larval fish for examination. Further recovery of the fishery was evident in 1996. All of the major sport fish had reestablished and were successfully reproducing. Tissue concentrations of selenium had fallen to 5 to $10 \mu\text{g Se/g}$, and the frequency of teratogenic deformities was 6% or lower (larvae, fry, juveniles, and adults).

Relationships between the amount of selenium in fish tissues, the prevalence of terata, and associated mortality are shown in Figures 5.1 and 5.2. These figures represent a compilation of all field and laboratory data on teratogenic effects (eg, field studies such as Lemly 1993, laboratory studies such as Woock et al. 1987). The prevalence of teratogenic deformities is dependent on tissue concentrations of selenium; more selenium results in more frequent terata. However, the association follows an exponential function rather than a linear relationship. In natural populations of juvenile and adult centrarchids (ie, not laboratory studies), the inflection point for the function occurs in the range of 40 to $50 \mu\text{g Se/g}$ dry weight. At these concentrations, about one fourth of the fish exhibit terata (Fig. 5.1). Beyond the inflection point, relatively small increases in selenium cause substantial increases in terata. The maximum observed frequency is 70% for individuals with body burdens of selenium in the 70 to $90 \mu\text{g Se/g}$ range. The exponential function holds for larval centrarchids as well, but the selenium concentrations for the inflection point and for the maximum are much lower. For example, at tissue concentrations of only about 30 to $40 \mu\text{g Se/g}$, the frequency of deformities is up to 80%.

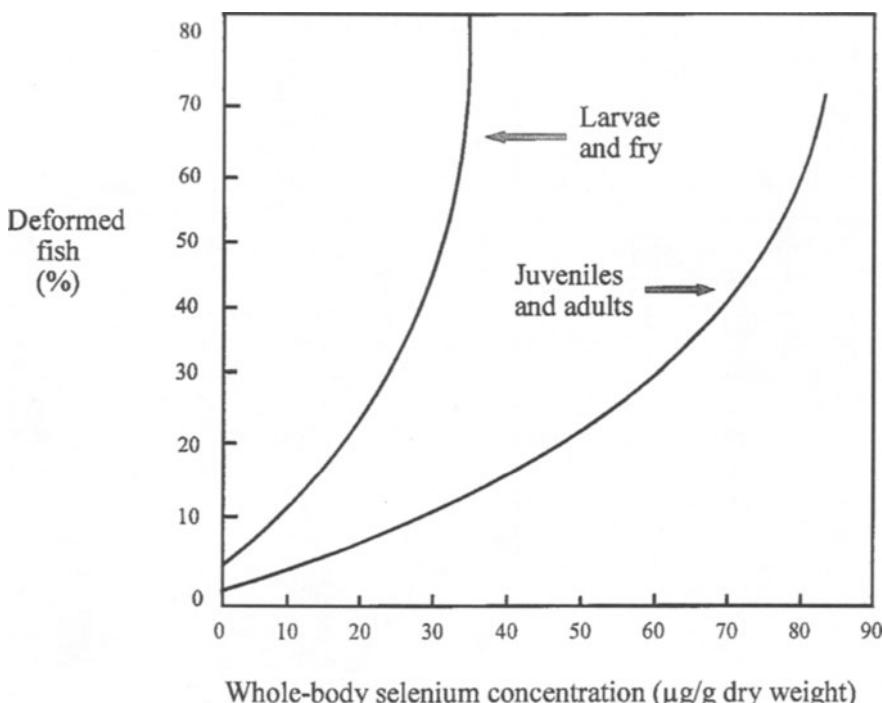


FIGURE 5.1. Relationship between whole-body concentrations of selenium and prevalence of teratogenic deformities in fish.

The relationship between teratogenesis and mortality is of primary importance in developing an assessment index. Whereas the prevalence of terata is influenced by tissue concentrations of selenium, the degree of mortality from terata is not. About 80% of teratogenic larval fish die regardless of their body burden of selenium (Fig. 5.2). This suggests that there is a maximum body burden for generation of lethal terata. Saturation beyond this maximum by additional selenium has little impact. Mortality is nearly constant for juvenile and adult fish as well, but the magnitude is not nearly as great as for larvae. Only about 25% of teratogenic juvenile and adult fish die in any given year. This is probably a reflection of simple mathematics (elimination of dead individuals) and to some extent, the severity of the terata; that is, the 20% or so of teratogenic larvae that survive will make up the teratogenic fraction of the juvenile and adult populations. Although terata persist, they may no longer be as life threatening as in younger fish.

The difference in mortality between life stages indicates that the priority for assessing or predicting population-level impacts of selenium should be the larval fish, because it is more likely that teratoge-

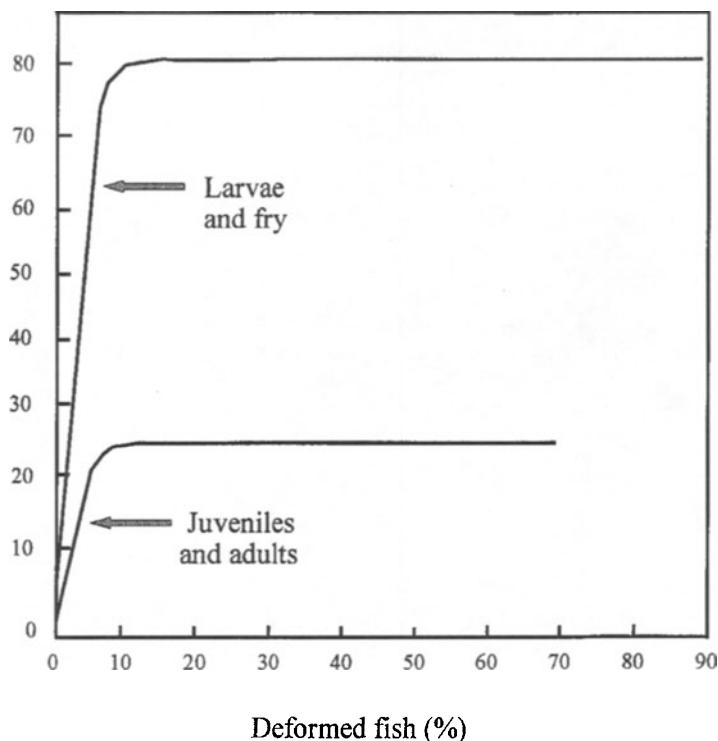


FIGURE 5.2. Relationship between prevalence of deformities and amount of teratogenic mortality in fish.

nic mortality will be expressed in this life stage. Moreover, persistence of deformities into the juvenile and adult life stages may occur only under special circumstances when natural predation has been sharply reduced or eliminated (Lemly 1993). Ideally, all life stages should be examined, with the focus placed on larval fish.

An Index for Terata-Based Assessment

Both the laboratory and field data indicate a close parallel among selenium concentrations, incidence of teratogenic deformities, and magnitude of reproductive failure in fish. Using these relationships, I developed the Teratogenic Deformity Index (TDI) to assess impacts on fish populations (Table 5.1). The TDI is composed of 3 ratings that signify increasing levels of terata-induced population mortality:

1 = Negligible Impact (<5% population mortality)

2 = Slight to Moderate Impact (5-20% population mortality)

3 = Major Impact (>20% population mortality)

TABLE 5.1. Teratogenic Deformity Index (TDI) ratings for evaluating impacts of selenium on fish populations.

Fish life stage	Percent with terata	Population mortality ^a	TDI rating	Anticipated impact
Larvae or fry	<6	<5 %	1	Negligible
	6–25	5–20 %	2	Slight to moderate
	>25	>20 %	3	Major
Juveniles or adults	<20	<5 %	1	Negligible
	20–80	5–20 %	2	Slight to moderate
	>80	>20 %	3	Major

^aMortality, expressed as a percentage of the total fish population, for the life-stage sampled (not teratogenic mortality). For example, 20% larvae with terata translates to 16% population mortality, because up to 20% of those with terata would be expected to survive to adulthood (ie, only about 80% of teratogenic larvae die).

Each rating is based on the anticipated population-level impact of the corresponding degree of mortality, that is, little effect is expected with <5% mortality, but substantial effects may occur with >20% mortality. Population mortality is calculated in 4 simple steps:

- (1) Determine the percentage of teratogenic fish and the percentage of normal fish in the total sample.
- (2) Multiply the percentage of teratogenic fish times the expected mortality rate (80% for larvae, 25% for juveniles and adults) to estimate the percentage of fish that will die and the percentage that will survive.
- (3) Add the percentage of normal fish and the percentage of surviving teratogenic fish.
- (4) Subtract this sum from 100%.

The result is population mortality, which will be less than teratogenic mortality. For example, 20% teratogenic larvae with 80% mortality translates to 16% population mortality; 20% teratogenic juveniles/adults with 25% mortality translates to 5% population mortality. Because of the differences in teratogenic mortality between larval and juvenile/adult fish, age-specific indices were developed. As discussed previously, persistence of terata in older life stages (and thus accurate evaluation) can be heavily influenced by predation. Thus, the index for juveniles and adults may have limited application.

The terata–mortality relationships are based on data for 2 fish families: Centrarchidae (bass, sunfish) and Cyprinidae (minnows). The resultant TDI for impacts may or may not be directly applicable to cold- or cool-water families such as Salmonidae (eg, trout, salmon) or

Esocidae (eg, pike and muskellunge). However, the close similarity of the relationships for centrarchids and cyprinids (virtual mirror images) suggests that impacts of terata may be generally consistent, regardless of fish family. Moreover, extrapolation to other families of fish is not necessary for many locations in North America, since centrarchids and cyprinids have characteristics that make them a good indicator or sentinel for other species: they are sensitive to selenium and are widely distributed (Lee et al. 1980; Lemly 1993).

The TDI can be applied to literally any aquatic habitat because it consists of impact-based assessment. Impacts (terata) are a function of selenium concentrations in fish eggs. The events responsible for the transfer of selenium to fish eggs, that is, bioaccumulation in aquatic food chains and consumption of contaminated diets by parent fish, can be highly variable from location to location. These events are influenced by such things as hydrology and landform (the amount and timing of precipitation; stream, lake, or wetland), the chemical form of selenium (selenate, selenite, organoselenium), and the timing and amount of selenium inputs relative to spawning periods (Lemly and Smith 1987). Consequently, the hazard (likelihood of toxic impacts) of selenium to fish and wildlife is also highly variable. However, the TDI is based on a measure of existing impact (terata), not potential hazard. As such, terata are an expression of the sum total of parental exposure, regardless of the temporal, spatial, or chemical variations that may exist from site to site. Thus, the applicability of the TDI is not influenced by local environmental conditions that affect selenium dynamics and biological uptake. It makes no difference whether the system is a fast-flowing stream, in which selenate predominates and bioaccumulation is low, or a terminal wetland in which high bioaccumulation from selenite is occurring; population-level impacts are indicated only if a sufficient amount of terata exists.

Example Assessments

- (1) Collect and examine 500 larval fish of the same species using ichthyoplankton sampling techniques. Assess the prevalence of teratogenic deformities and measure selenium concentrations in 6 composite samples of teratogenic individuals. The investigation reveals that 15% have terata, and the associated selenium concentrations are 10 to 15 µg Se/g. The expected population-level mortality is 12% (85% normal + 3% surviving teratogenic = 88% total survival), resulting in a TDI rating of 2. Conclusion: slight to moderate impact on the population due to teratogenic effects of selenium. A shift in the relative dominance of fish populations is possible, with selenium-sensitive species replaced by

tolerant species. Resultant long-term changes in fish community ecology are anticipated.

- (2) Collect and examine 300 juvenile and 200 adult fish of the same species. Assess the prevalence of teratogenic deformities and measure selenium concentrations in individuals with terata (whole-body samples; 6 juveniles and 6 adults). The investigation reveals that 8% have terata, and the associated selenium concentrations are 20 to 30 µg Se/g. The expected population-level mortality is 2% (92% normal + 6% surviving teratogenic = 98% total survival), resulting in a TDI rating of 1. Conclusion: negligible impact on the population due to teratogenic effects of selenium. No shift in fish populations or community-level ecology is expected.
- (3) Sample 1000 larval, 200 juvenile, and 100 adult fish of the same species. Determine the prevalence of teratogenic deformities and measure selenium concentrations in teratogenic individuals (6 composite samples for larvae; 6 each, whole-body samples for juveniles and adults). The investigation reveals that 35% of larvae have terata, and 3% of juveniles and adults have terata; selenium concentrations are 10 to 30 µg Se/g. The expected population-level mortality is 28% for larvae (65% normal + 7% surviving teratogenic = 72% total survival) and 0.6% for juveniles and adults (97% normal + 2.4% surviving teratogenic = 99.4% total survival). Resulting TDI ratings are 3 for larvae and 1 for juveniles/adults. Conclusion: major impact on the population due to teratogenic effects of selenium on larvae. Extirpation of selenium-sensitive species is likely. Population and community-level ecology will be substantially altered; collapse of the fishery is possible.

Conclusions

There are several reasons to use the TDI for impact assessment of selenium. First, the sensitivity of TDI is unaffected by variations in the environmental mixture of selenium species. Terata are produced by a rather uniform process regardless of what chemical form(s) of selenium the parent fish were exposed to. Thus, the TDI can be applied with confidence to any aquatic habitat. Second, terata-based assessment provides a conclusive cause-effect linkage between the contaminant and the fish. Because of this linkage, TDI is particularly useful for verifying selenium-induced impacts on reproductive success, since poor reproduction can be caused by many other things as well: for example, fluctuating water levels, nest predation, food shortages, poor recruitment. Third, the TDI estimates impacts of selenium on fish at the popu-

lation level, which is typically a very difficult parameter to determine for contaminants in a field setting. Fourth, it is a technique that goes hand-in-hand with the Protocol (see Chapter 4) to provide a comprehensive approach for investigating selenium contamination issues. The Protocol is a predictive tool that determines the degree of hazard present, and the TDI provides site-specific verification of selenium poisoning and assesses the magnitude of toxic impacts to current and future generations of fish. Fifth, the TDI can be used to evaluate and predict impacts to fish populations without the expenditure of large amounts of time and money on contaminant monitoring that, in itself, leaves important questions unanswered. Application of this technique will facilitate efficient use of personnel and funds early in the assessment process and allow for rapid transition to the next phase (ie, developing site-specific water quality criteria [see Chapter 7]) in resolving selenium issues.

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Section III

Applying Hazard Assessment to Water Quality Criteria

6

Hydrological Units and Selenium Criteria

Introduction

The hydrological pathways that transport selenium across the landscape, as well as the presence of different habitat types (wetlands, streams, rivers, lakes, reservoirs) within many watershed basins, have important implications for setting water quality criteria for this trace element. In the time since the USEPA established the current United States national criteria (EPA 1987), there have been several field-documented cases of toxic effects to wetland biota occurring while water-borne concentrations of selenium were below the criterion value for chronic exposure ($5 \text{ } \mu\text{g Se/L}$) (eg, Skorupa and Ohlendorf 1991; Stephens et al. 1992; Hallock and Hallock 1993; Lemly 1995; Hamilton et al. 1996; Skorupa 1998). There have also been observations which suggest that the criterion is too restrictive for some rivers and streams (Canton and Van Derveer 1997; Van Derveer and Canton 1997). This has led several states, the USFWS, and local municipalities to pursue development and implementation of criteria on a site-specific basis (eg, Guglielmino 1995). Although this is a logical course of action, the approaches used to derive criteria are often seriously flawed, because certain components of the selenium cycle are overlooked or underestimated, especially the potential for bioaccumulation and toxic impacts downstream of the "site" under consideration (Hamilton and Lemly 1999).

Because of selenium's propensity to bioaccumulate in aquatic food chains, it is important to carefully assess the *entire* selenium cycle—including down-gradient transport, transformation, and bioaccumulation—at locations where site-specific criteria are being considered. This is essential because it is possible for 2 adjacent aquatic systems (eg, a river and an off-channel wetland) to have largely different selenium cycling dynamics (Lemly and Smith 1987). A water-quality criterion that is appropriate for one system may not be sufficient for another, particularly if the two are hydrologically connected. The

mechanism of toxicity also makes it imperative to closely scrutinize the pattern of bioaccumulation that is taking place. For example, deposition of dietary selenium in eggs can cause reproductive failure in fish even though there is little or no outward evidence of toxicity to the spawning fish themselves (Lemly 1985a, 1985b). Therefore, factors contributing to food-chain transfer of selenium to adult fish need to be identified and accounted for as water quality criteria are being developed.

The degree of mobility (inter-habitat transport), transformation (from inorganic selenium to organic selenium and vice versa), and bioaccumulation of selenium all influence the toxic threat to biota and, consequently, the need for site-specific criteria. This chapter presents a brief overview of the selenium cycle and discusses why site-to-site differences in bioaccumulation and threats to down-gradient aquatic habitats favor the use of a Hydrological Unit (HU) approach for deriving water quality criteria.

The Selenium Cycle

Three things can happen to dissolved selenium when it enters an aquatic ecosystem: (1) it can be absorbed or ingested by organisms; (2) it can bind or complex with particulate matter or surficial sediments; or (3) it can remain free in solution. Over time, most of the selenium is either taken up by organisms or bound to particulate matter (Fig. 6.1). Through deposition of biologically incorporated selenium and settling of particulate matter (sedimentation), most of the selenium usually accumulates in the top layer of sediment and detritus. However, because biological, chemical, and physical processes move selenium out of, as well as into, sediments, this top layer is only a temporary repository for selenium. Aquatic systems are dynamic, and selenium can be cycled back into the biota and remain at elevated levels for years after water-borne inputs of selenium are stopped (Lemly 1997).

Immobilization Processes

Selenium can be removed from solution and sequestered in sediments through the natural processes of chemical and microbial reduction of the selenate form (Se VI) to the selenite form (Se IV), followed by adsorption (binding and complexation) onto clay and the organic carbon phase of particulates, reaction with iron species, and coprecipitation or settling (Fig. 6.2). Regardless of the route, once selenium is in the sediments, further chemical and microbial reduction may occur resulting in insoluble organic, mineral, elemental, or adsorbed selenium. Most selenium in animal and plant tissues is eventually deposited as

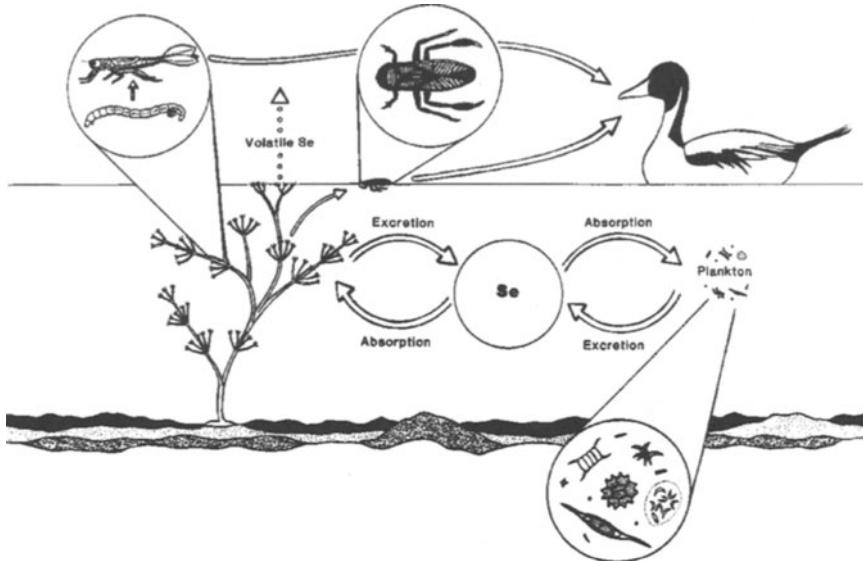


FIGURE 6.1. A highly dynamic system: biological, chemical, and physical processes cycle selenium into and out of the water and biota.

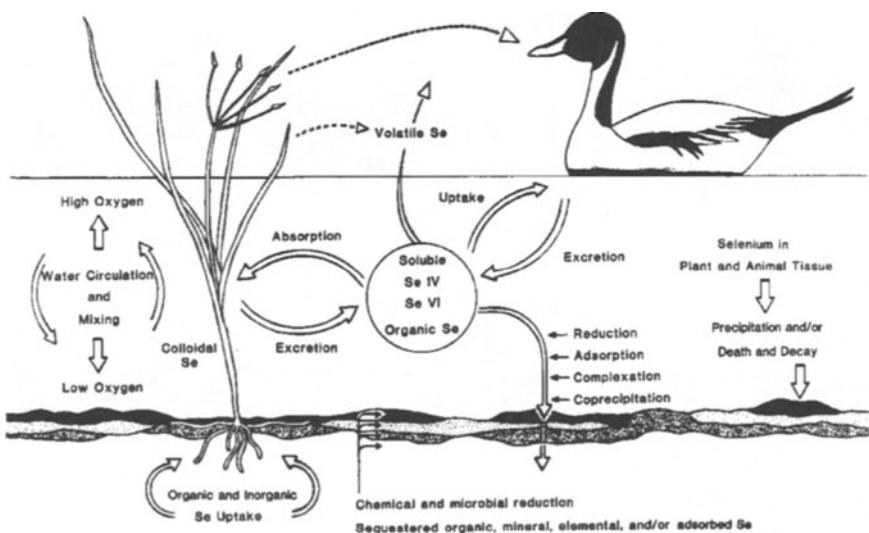


FIGURE 6.2. Processes that immobilize selenium include chemical and microbial reduction, adsorption, coprecipitation, and deposition of plant and animal tissue. Mobilization processes include uptake of selenium by rooted aquatic plants and sediment oxidation due to water circulation and mixing. Additional mobilization processes include direct uptake of selenium by benthic invertebrates and oxidation of sediments resulting from plant roots, microorganisms, and the burrowing activity of benthos.

detritus, and over time, isolated through the process of sedimentation. Some selenium, particularly certain organic forms, may be released into the atmosphere through volatilization by chemical or microbial activity in the water and sediments or through direct release by plants.

In total, immobilization processes effectively remove selenium from the soluble pool, especially in slow moving or still-water habitats and wetlands. As much as 90% of the total selenium in an aquatic system may be in the upper few centimeters of sediment and overlying detritus (Lemly and Smith 1987).

Mobilization Processes

Selenium in sediments is particularly important to long-term habitat quality, because mechanisms present in most aquatic systems effectively mobilize this pool of selenium into food chains and thereby cause long-term dietary exposure of fish and wildlife (Lemly 1993, 1997). Sediment-associated selenium is made available for biological uptake by 4 oxidation and methylation processes (Figs. 6.2 and 6.3) (oxidation

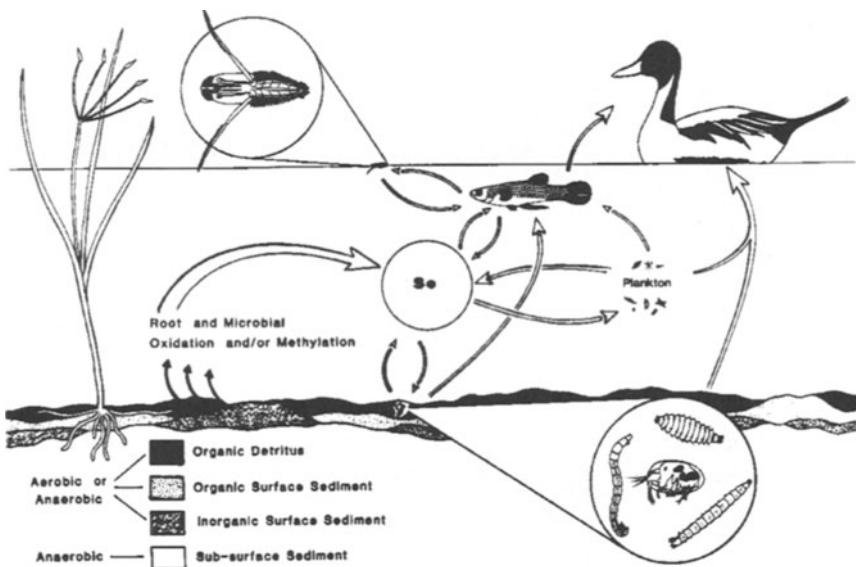


FIGURE 6.3. Dissolved selenium, whether introduced from wastewater discharge, natural geologic sources, or mobilized from sediments, is readily taken up by aquatic organisms and concentrated in food chains, particularly in wetlands, ponds, and reservoirs. These food pathways converge on top consumer species of fish and wildlife. The effects may be severe even when the concentration of waterborne selenium is low. (*Solid arrows* indicate pathways by which selenium is remobilized from sediments into the food chain).

refers to the conversion of inorganic selenium in the reduced organic, elemental, or selenite forms to the selenite or selenate forms; methylation is the conversion of inorganic or organic selenium to an organic form containing one or more methyl groups, which usually results in a volatile form): (1) oxidation and methylation of inorganic and organic selenium by plant roots and microorganisms; (2) biological mixing and associated oxidation of sediments that results from the burrowing of benthic invertebrates and feeding activities of fish and wildlife; (3) physical perturbation and chemical oxidation associated with water circulation and mixing (current, wind, stratification, precipitation, and upwelling); and (4) sediment oxidation by plant photosynthesis.

Two additional pathways provide for direct movement of selenium from sediments into food chains, even when the surface water does not contain elevated concentrations of the element. These pathways are (1) uptake of selenium by rooted plants, and (2) uptake by bottom-dwelling invertebrates and detrital-feeding fish and wildlife. These 2 pathways may be the most important factors in long-term cycling of potentially toxic concentrations of selenium. Thus, rooted plants and the detrital food pathway can continue to be highly contaminated and expose fish and wildlife through dietary routes, even though concentrations of selenium in water are very low (Lemly and Smith 1987).

Role of Habitat Variability

The processes regulating selenium cycling are similar in all aquatic habitats, but the relative contribution of each process may vary from habitat to habitat. In fast-flowing waters, fine organic sediments, such as those produced by the deposition and decay of particulate matter and plant and animal tissue, may be rare because they are continually flushed from the system. In these waters, there is little opportunity for a contaminated surface layer of sediment to develop, and rooted plants are often scarce. The benthic-detrital components of the system and the associated food pathways thus play a smaller role in the selenium cycle in flowing waters than in slow-water habitats such as wetlands or reservoirs.

The aquatic systems that accumulate selenium most efficiently are shallow, slow-moving waters that have low flushing rates. In these systems, biological productivity is often high, and selenium may be trapped through immobilization processes or through direct uptake by organisms. Sediments tend to build up a selenium load that can be remobilized gradually, yet continually, through detrital and planktonic food pathways. These habitats are also some of the most important feeding and breeding habitats for fish and wildlife, especially waterfowl and shorebirds, reservoir and lake fish, and riverine fish that utilize off-channel areas during early life.

Several habitat types often occur together in one aquatic system. For example, rivers may have fast-flowing waters, slow-moving pools, and standing backwater areas, all within a few hundred meters. The degree of fish and wildlife exposure to selenium varies among habitats according to intensity of use, type of use, and the relative contributions of the various processes that regulate selenium cycling.

Water quality criteria for selenium should be based upon an assessment of the degree of contamination and risk of toxic effects, which in turn, will depend upon the spatial and temporal variation of the selenium cycle at the site under consideration. In addition to protecting resident biota at the "site", it is important that criteria protect against possible impacts to down-gradient aquatic habitats as well. For example, a criterion that is appropriate for a stream or river where low bioaccumulation occurs may result in *seemingly* harmless concentrations of selenium becoming a problem in downstream reservoirs or in off-channel bays and wetlands where bioaccumulation is greater (Fig. 6.4).

Importance of Bioaccumulation

In developing water quality criteria for selenium, it is critical to incorporate the bioaccumulation phenomenon into the derivation process. The major principle to remember is that reproductive effects in fish and aquatic birds are the most sensitive biological indicators of aquatic ecosystem-level impacts of selenium. Selenium in water can be concentrated from 100 to over 30,000 times in the food organisms eaten by fish and wildlife, exposing them to a highly concentrated dietary source of contamination. Biomagnification may also occur, resulting in a 2- to 6-fold increase in selenium from primary producers to forage fish. Moreover, if the ecosystem is allowed to reach an equilibrium that recycles selenium from sediment, the detrital food pathway can deliver toxic doses of selenium for many years even if waterborne sources are eliminated (Lemly 1982, 1985a, 1997).

A significant portion of the selenium consumed by fish and wildlife is passed to their offspring in eggs, where it can kill developing embryos outright or induce a variety of lethal or sublethal teratogenic deformities (Lemly 1993). However, parents can consume a selenium-laden diet and experience partial or complete reproductive failure without exhibiting symptoms of selenium toxicosis themselves. Moreover, aquatic food organisms of wildlife strongly bioaccumulate selenium—to a level that is hundreds to thousands of times the waterborne concentration—but are unaffected by tissue residues that are high enough to cause reproductive failure when consumed by fish and aquatic birds. Thus, bioaccumulation in aquatic food chains and dietary transfer to eggs cause otherwise innocuous concentrations of waterborne sele-

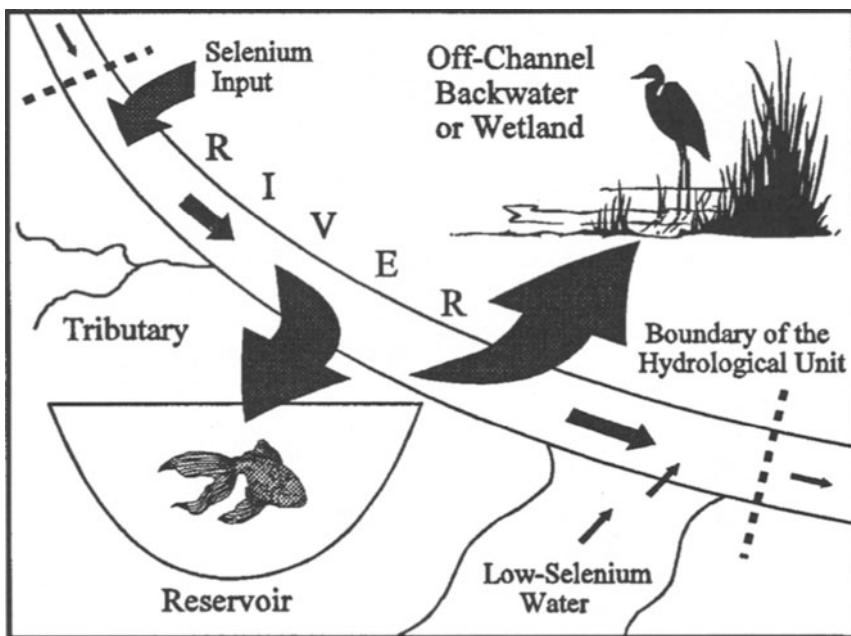


FIGURE 6.4. An illustration of the “Hydrological Unit” (HU) principle. Arrows indicate the relative concentrations of selenium. The interconnected parts of a HU may include several types of aquatic habitats; for example, a main-stem river, a wetland, and a reservoir. Given equal waterborne concentrations of selenium, the degree of bioaccumulation and toxicity to fish and wildlife can be substantially greater in off-channel lentic systems than in a river or stream. It is necessary to guard against downstream transport of hazardous concentrations of selenium that could result if a liberal criterion was set for the river. Establishing a single criterion for the entire HU is the best way to ensure protection of aquatic life.

nium to become toxic. Establishing water-quality criteria that prevent this amount of bioaccumulation in aquatic food chains should be the goal of site-specific derivations.

The degree of selenium accumulation in major ecosystem components (sediments, benthic invertebrates, fish, birds) can be used to precisely evaluate local conditions and determine if existing or proposed criteria are appropriate, too conservative, or too liberal (see next chapter). This process can be applied consistently and uniformly, regardless of location or habitat type—that is, in wetlands (seasonal or permanent), streams, rivers, lakes, reservoirs, and other impoundments. Criteria that prevent bioaccumulation from reaching levels sufficient to cause dietary toxicity and reproductive effects in fish and aquatic birds will afford protection to other aquatic life as well.

Criteria Based on Hydrological Units

The physical area from which measurements are taken to evaluate selenium residues and biological effects—that is, the database for setting site-specific criteria—must encompass more than a limited area such as an isolated segment of river or a tributary stream. The overarching principle for establishing environmentally sound water-quality criteria for selenium is that bioaccumulation needs to be kept below levels that pose significant threats to biota. In order for this to be accomplished, hazards to down-gradient aquatic habitats, most or all of which may fall outside the immediate “site” under consideration, must be assessed and taken into account.

Differences in selenium cycling among aquatic habitats is one of the main reasons that USEPA national criteria are being questioned more frequently. One size doesn’t fit all. However, the reasoning that individual sites need specific criteria can be taken too far. For example, isolating a segment of a river and setting a criterion for it that will not prevent harm to biota in the receiving waters downstream is not a prudent approach. Because of hydrological connections between the various aquatic habitats that may be present in a watershed basin (wetlands, rivers, streams, lakes, reservoirs, and other impoundments), the risk of toxic impacts from selenium contamination is also connected.

The hydrologically connected parts of a basin down-gradient of a selenium discharge or other input (any natural or anthropogenic selenium source), extending to the point at which new sources of low-selenium water dominate the hydrology (eg, confluence with a larger tributary or river, a spring, or groundwater inflow, resulting in concentrations that do not exceed background levels for the area), should be the area evaluated and given a specific criterion, not isolated components of that larger area (Fig. 6.4). Thus, a Hydrological Unit (HU) should be identified and used as the “site” for the purpose of setting criteria. A simple screening-level assessment of waterborne selenium concentrations is all that is necessary to delineate the boundaries of the HU.

A designated HU is a major departure from the traditional concept of what constitutes a “site”. Consequently, it may be useful to refer to criteria derived with the HU approach as being watershed-specific or basin-specific rather than site-specific. While still providing for focused local refinement of USEPA national criteria, this method ensures an integrated assessment of ecosystems within a watershed and allows basinwide protection of aquatic life. Importantly, criteria derived in such a manner will reflect the transport and cycling of selenium within the entire HU rather than simply focusing on a small, artificially designated segment of the system—that is, a section of river, a tributary stream, or a similar limited area.

Failure to use a HU approach can set the stage for significant biological and legal problems. Consider, for example, the following scenario. A chronic exposure criterion of 20 µg Se/L is derived and adopted for a 10 km segment of river immediately downstream of a municipal wastewater treatment plant, based on the finding that there are no toxic impacts to fish, and little bioaccumulation is taking place. However, a few kilometers downstream of this river segment, there is a 250 ha off-channel wetland that is used by wildlife for feeding, spawning, and nesting. Part of the wetland is a public wildlife management area, and part is under private ownership. The 20 µg Se/L that is permitted in the river flows into the wetland, bioaccumulates in aquatic food chains, and causes toxic impacts to fish and bird embryos. Several questions arise. What is the appropriate criterion for the wetland? Should it be imposed on the river where no problems are occurring? Is it prudent to set 2 criteria—one for the river and one for the wetland—when the two are hydrologically connected? Who is liable for toxicity to wildlife, and what recourse is possible for the landowners? Will litigation be necessary to resolve the dilemma and, if so, what parties will be involved?

This hypothetical case is becoming a reality in several western states of the United States as natural resource management agencies, local municipalities, industry, and private landowners become aware of the threat selenium poses to fish and wildlife and understand the liabilities that ensue if poisoning occurs (eg, Margolin 1979; Guglielmone 1995). The task of settling "site-specific" criteria issues may involve complex questions of land and water jurisdiction if the HU affected by selenium includes several municipalities and counties or crosses state boundaries. Nevertheless, it is important for natural resource managers to stay focused on the objective of water quality criteria—protection of aquatic life—and not be swayed by those with other interests whose goals may be totally different. Using the HU method will help maintain that focus, because it provides an accurate profile of the extent and severity of selenium contamination.

Conclusions

Pollution of aquatic ecosystems by selenium is a growing concern in the United States and elsewhere around the world (see Chapter 1). Questions about the appropriateness of using national standards to protect aquatic life at a local level are being raised more and more frequently. Those seeking to develop site-specific water quality criteria for selenium need to have a clear understanding of how to go about it. A 2 part process should be followed. This chapter describes the first

part, which is to gain a working knowledge of selenium transport and bioaccumulation and apply that knowledge to delineate Hydrological Units (HUs). Using HUs is critical to developing criteria that provide watershed-level protection of aquatic life. The second part in the process is interpreting basinwide information on selenium residues and biological effects and following a step-by-step procedure for setting local criteria. That part is presented in the next chapter.

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Developing Site-Specific Water Quality Criteria

Introduction

The United States Environmental Protection Agency (USEPA) conducted a comprehensive review of information on selenium ecotoxicology and issued revised national freshwater criteria for selenium in 1987 (EPA 1987). Since that time, two divergent lines of evidence and thought have emerged regarding whether the criterion for chronic exposure ($5 \mu\text{g Se/L}$) is adequate to protect aquatic life. One line supports the position that the criterion is too high and should be lowered. For example, Winter Stress Syndrome in fish—a disruption of the normal annual cycle of metabolic and physiological changes in cold weather—can substantially increase fish sensitivity to selenium and cause concentrations approaching the criterion value to become toxic (Lemly 1993a, 1996). Moreover, in the western United States, agricultural irrigation has caused selenium to bioaccumulate to toxic levels in food chains and to poison fish and aquatic birds when waterborne concentrations were well below $5 \mu\text{g Se/L}$ (eg, Skorupa and Ohlendorf 1991; Stephens et al. 1992; Hallock and Hallock 1993; Lemly 1995; Hamilton et al. 1996). The other line of evidence supports the position that the criterion is too low and should be raised. For example, faunal surveys in Colorado streams showed abundant fish populations where waterborne selenium consistently exceeded the criterion. These observations have led some to conclude that the national criterion is overly conservative, and that the tolerated level should be raised to as much as $31 \mu\text{g Se/L}$ in some locations (eg, Guglielmino 1995; Canton and Van Derveer 1997; Van Derveer and Canton 1997). The principal disagreements between these two schools of thought were recently discussed by Hamilton and Lemly (1999). They revolve around assumed versus demonstrated biological effects, and the need to use a Hydrological Unit (HU) as the basis for setting criteria (see Chapter 6).

The on-going controversy over whether the United States national criterion is too high or too low illustrates a very important point. Regardless

of what the criterion is, there will likely be a concerted effort by some to raise or lower it. USEPA policy allows the national criterion to be modified by states on a site-specific basis when biological evidence warrants it (Stephan et al. 1985; EPA 1985). Such modifications have been pursued; local criteria—below 5 µg Se/L in some instances and above 5 µg Se/L in others—are being considered or have been adopted in several western states, including Arizona, California, Colorado, and New Mexico (Arizona 1992; CEPA 1992; CSWRCB 1987; Guglielmino 1995; New Mexico 1995). However, there is no widely accepted or published method for site-specific criterion revision, developed by the USEPA or from other sources, that is targeted specifically at selenium. Consequently, criteria are being developed with little pertinent direction or guidance. This has caused some to use insufficient data and make assumptions about fish and wildlife exposure/effects that could introduce significant error into the derivation process (Hamilton and Lemly 1999). This type of error has important implications. A derivation might indicate that changing a criterion is appropriate when, in fact, raising the criterion is not environmentally acceptable or lowering the criterion is not biologically necessary. A scientifically credible method is needed that can be broadly and uniformly applied yet is sensitive to selenium's site-specific variations in environmental cycling and biological effects. I developed the procedure described in this chapter to provide that method. Some examples are given to illustrate cases that justify raising or lowering an existing criterion, leaving a criterion unmodified, or examining a proposed criterion.

Rationale

There are two primary considerations in developing site-specific water quality criteria for selenium: (1) local conditions must be evaluated, and the resultant information used to decide if a modified criterion is necessary; and (2) if changes are warranted, the existing or proposed criterion must be modified by an appropriate amount. A simple two-step process can be used to derive criteria for chronic exposure.

The major principle underlying this process is that reproductive effects in fish and aquatic birds are the most sensitive biological indicators of aquatic ecosystem-level impacts of selenium (Ohlendorf 1989; Lemly 1993b, 1997; Skorupa et al. 1996). Selenium is passed from parents to their offspring in eggs, where it can kill developing embryos outright or induce a variety of lethal or sublethal teratogenic deformities (eg, Hoffman et al. 1988; Lemly 1993b). However, parents can consume a selenium-laden diet and experience partial or complete reproductive failure without exhibiting symptoms of selenium toxicosis themselves (Lemly 1985a, 1997; Ohlendorf 1989). Moreover, aquatic organisms that are the food of wildlife strongly bioaccumulate sele-

nium, perhaps to thousands of times the waterborne concentration, but are unaffected by tissue residues that are high enough to cause reproductive failure when consumed by fish and aquatic birds (Lemly 1985a). Thus, bioaccumulation in aquatic food chains, and dietary transfer to eggs causes otherwise harmless concentrations of waterborne selenium to become toxic. Establishing water quality criteria that prevent this degree of bioaccumulation in aquatic food chains is the goal of the procedure given in this chapter.

Information concerning the presence or absence of reproductive effects, coupled with information on the degree of accumulation in major ecosystem components, can be used to precisely evaluate local conditions and derive an appropriate site-specific water quality criterion for selenium. This process can be applied consistently and uniformly, regardless of location or habitat type—that is, in wetlands (seasonal or permanent), streams, rivers, lakes, reservoirs, and other impoundments.

Procedure

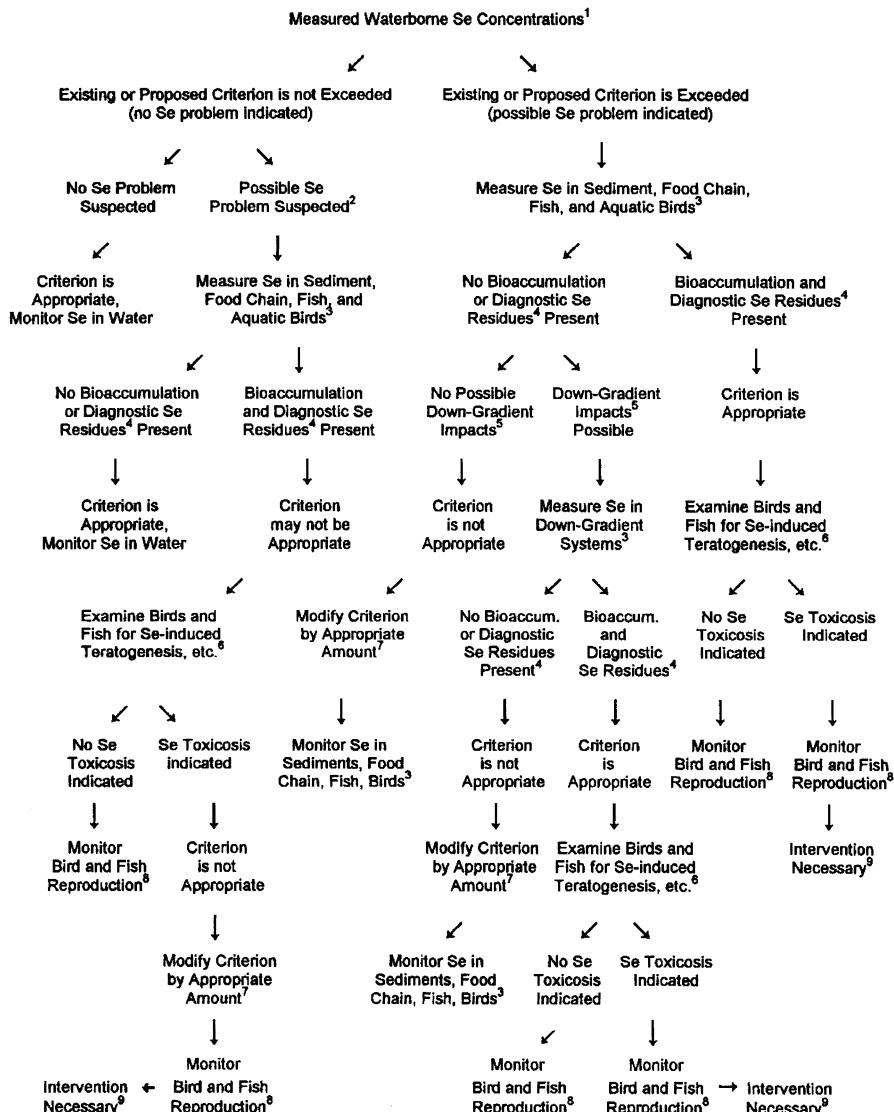
Step 1

The first step is to gather information on selenium residues and biological effects. This is done to provide an empirical foundation for evaluating a criterion, which may be national, state, or local, and which



FIGURE 7.1. A decision tree for determining if modification of a selenium water quality criterion is necessary. Numbers indicate the following: 1, a sufficient number of samples to provide some spatial and temporal integration, collected across a Hydrological Unit (see Chapter 6); 2, possible problem suspected based on observations and available information collected from the Hydrological Unit; 3, measure and interpret according to guidelines and procedures given by Skorupa and Ohlendorf (1991) and Lemly (Chapters 2 and 3); 4, residues that equal or exceed the toxicity thresholds given in Table 7.1; 5, down-gradient impacts to other aquatic systems, wetlands, or selenium sensitive species in the Hydrological Unit, including confluence with receiving waters that cross state lines or other jurisdictional boundaries; 6, teratogenic deformities and/or embryomortality, measured according to guidelines and procedures given by Ohlendorf et al. (1986a, 1986b, 1988), Hoffman et al. (1988), and Lemly (1993b) (see also Chapters 2, 3, and 5); 7, modify according to procedures given in the text; 8, monitor for evidence of teratogenic deformities and/or embryomortality, and associated selenium residues in eggs, embryos, and larvae; 9, regulatory and management actions to effectively control or modify the source in order to reduce selenium inputs.

may be currently in place or under consideration. A decision tree (Fig. 7.1) is used to guide the process. It specifies what types of information are necessary for the evaluation and also identifies conditions under which the criterion should be modified. The selenium criterion value is examined in the context of site-specific selenium concentrations in combination with potential and observed toxicity to fish and aquatic birds. The residue data set required for this process is virtually the same as for the Protocol (see Chapter 4) and consists of selenium mea-



surements for water, sediments, benthic invertebrates, and fish and aquatic bird tissues. There is no minimum number of measurements, but spatially complete sampling efforts will ensure that the range of habitat types and environmental conditions present are included in the assessment, and that inclusion of the full range results in a criterion which accurately reflects local selenium cycling and aquatic ecology. Moreover, criteria generated from robust data sets will be considered more credible by regulatory agencies that must approve their implementation (eg, USEPA) than criteria based on just a few measurements. Refer to Chapter 4 for details on sampling procedures.

It is important to designate a Hydrological Unit (HU) as the physical area to be examined in the criterion review process. The hydrological pathways that transport selenium across the landscape have important implications for setting water quality criteria for this environmental contaminant. For example, a criterion that is appropriate for a stream or river where low bioaccumulation occurs may result in *seemingly* harmless concentrations of waterborne selenium becoming a problem in downstream reservoirs or in off-channel bays and wetlands where bioaccumulation is greater. In order to develop environmentally sound criteria, hazards to down-gradient aquatic habitats, most of which may fall outside of what would typically be considered the "site", must be assessed and accounted for. The HU technique is the only method that does this effectively. Failure to use a HU approach can result in significant biological and legal problems. Consult Chapter 6 for information on the rationale and justification for using HUs in the criterion derivation process and for details on how to delineate HUs.

Reproductive assessment (teratogenic deformities and embryo/larval mortality) should be conducted on at least one selenium-sensitive species of fish and aquatic bird that resides in the HU: a centrarchid or salmonid fish (eg, genus *Lepomis*, *Micropterus*, *Oncorhynchus*, *Salvelinus*), a duck (eg, genus *Anas*), or a wading bird (eg, genus *Recurvirostra*) for example. The basic procedure is to collect larval fish using ichthyoplankton sampling techniques, to randomly select eggs from aquatic bird nests, and to examine both for terata or embryo mortality. Consult Chapter 5, and Skorupa and Ohlendorf (1991) for details on methods to evaluate impacts of selenium on reproductive success. Guidance for measuring selenium residues, interpreting tissue concentrations, and making comparisons to biological-effects thresholds is available in Table 7.1 and Chapter 2.

Several outcomes and conclusions are possible from the assessment in Step 1. The criterion may be appropriate as it is, or it may be inappropriate and need to be raised or lowered. If the empirical evidence indicates that a revised criterion is necessary (guided by the decision tree), the investigator should proceed with Step 2.

TABLE 7.1. Selenium residues that signify the thresholds for reproductive effects in fish and aquatic birds. Concentrations and biological effects in the Hydrological Unit (HU) are compared to diagnostic residues to determine if modification of a water quality criterion is necessary. The measured concentrations and indicated changes are given as examples of how to appropriately lower a criterion of 5 µg Se/L, assuming that waterborne concentrations in the HU are below or equal to 5 µg Se/L. Consult Figure 7.1 and the text for guidance on raising criteria.

Type of sample	Diagnostic residue ^{a,b} (reference ^c)	Measured concentration ^b and toxic effect	Appropriate change to a criterion of 5 µg Se/L ^d
Sediments	2–4 (<1)	1–5, no effect	None
Food chain ^e	3–7 (<2)	3–8, embryo mortality in birds	Lower by 2.5 µg/L (50% decrease)
Fish			
Whole-body	4–7 (<3)	6–9, no effect	None
Muscle (fillets)	8–10 (<3)	5–10, no effect	None
Liver	12–15 (<8)	4–7, no effect	None
Eggs	10–12 (<3)	6–17, terata	Lower by 2.5 µg/L (50%)
Larvae/fry	8–12 (<3)	5–12, terata	Lower by 1.5 µg/L (25%)
Aquatic birds			
Muscle	15–20 (<3)	7–19, no effect	None
Liver	10–25 (<10)	23–32, terata	Lower by 2.5 µg/L (50%)
Eggs	3–6 (<3)	4–9, no effect	None
Embryos	10–15 (<3)	18–25, terata	Lower by 4.0 µg/L (75%)

^aResidues that indicate ecosystem contamination is sufficient to cause reproductive impairment of fish and aquatic birds (teratogenic deformity and/or mortality of embryos and larvae/fry).

^bAll values are in µg Se/g (parts-per-million) dry weight. Values for reference and contaminated habitats were compiled from data given by Lemly (1985a, 1993b, 1997) (see also, Chapter 2), Ohlendorf et al. (1986a, 1986b, 1988), Hoffman et al. (1988), Ohlendorf (1989), Skorupa and Ohlendorf (1991), and Skorupa et al. (1996).

^cConcentrations typical for uncontaminated aquatic systems.

^dLowering a criterion is indicated only if reproductive effects are present in combination with diagnostic residues.

^eOrganisms commonly utilized as food by fish and wildlife, such as benthic macroinvertebrates, aquatic plants and seeds, and forage fish.

Step 2

A simple procedure is followed to determine what the new criterion should be. This technique is based on relationships between the de-

gree of bioaccumulation present in the HU (measured selenium residues), the known threshold concentrations for reproductive effects in fish and wildlife (diagnostic residues for teratogenesis and embryomortality), and any observed reproductive impacts in the HU (terata or embryomortality).

A more restrictive (lowered) criterion is indicated when the existing or proposed criterion value for waterborne selenium is *not* exceeded, but residues in the HU equal or exceed diagnostic toxic thresholds, and reproductive effects are observed. The criterion value should be lowered by an amount that corresponds to the magnitude of bioaccumulation present. Three sets of conditions are possible, each signifying a different level of toxic threat which, in turn, indicates a different amount of revision to the criterion: (1) if residue values fall partly below the threshold toxic ranges (see Diagnostic Residues, Table 7.1) and partly within the threshold toxic ranges, then the criterion should be lowered by 25%; (2) if residue values fall entirely within the threshold ranges or span the ranges such that some are below, some are within, and some are above, then the criterion should be lowered by 50%; (3) if all of the residue values exceed the upper end of the threshold ranges, then the criterion should be lowered by 75%. If these guidelines are followed, criteria are lowered in each instance by an amount that is proportional to the degree of biological hazard present.

No change in the criterion is necessary if: (1) the criterion is not exceeded and no selenium problem is suspected; (2) the criterion is not exceeded, a selenium problem is suspected (diagnostic residues may or may not be present), but there are no observed reproductive effects; (3) the criterion is exceeded, and residues exceed or fall within the toxic threshold ranges, or are above the ranges for uncontaminated aquatic systems (Table 7.1); or (4) the criterion is exceeded, residues are within the ranges for uncontaminated systems, but down-gradient impacts are possible in the HU (Fig. 7.1).

If the existing or proposed criterion is exceeded, yet residues fall within the range of values for reference conditions in uncontaminated systems (eg, background concentrations for the local area), which would indicate that little bioaccumulation is taking place, then it may be reasonable to consider raising the criterion. However, the appropriateness of raising a criterion is contingent upon firmly establishing that there are no reproductive impacts to any of the species in the HU, especially selenium-sensitive species such as centrarchid and salmonid fish, ducks, and wading birds. It is not sufficient to simply make an observational assessment and assert that if fish and birds are plentiful, then there must not be a selenium problem. In open aquatic systems, fish and birds can move about freely, both into and out of the HU. This can lead to the erroneous assumption that all of the observed individ-

als are long-time residents reflecting local conditions of selenium exposure in the HU. Because that may not be the case, faunal surveys alone are not adequate to determine the presence or absence of selenium toxicity. Actual reproductive assessment based on tissue selenium residues and occurrence of terata or mortality in fish larvae/fry and bird embryos (sensitive species) must be used to draw a conclusion of effect/no effect (see Chapters 2 and 5, and Skorupa and Ohlendorf, 1991, for details and guidelines on reproductive assessment). The absence of selenium-sensitive species in the HU is not a legitimate justification for raising the criterion, because their absence could be due to selenium poisoning. A comparison of species present in neighboring HUs, including supporting tissue selenium residue data, is necessary to evaluate whether sensitive species are likely absent due to selenium contamination or some other environmental attribute (eg, physical habitat quantity/quality, unique or additional competing species, other anthropogenic disturbances). A no-effect determination should be made for at least one selenium-sensitive species in the HU before raising a criterion is proposed. If this procedure is followed, and a clean bill of health can be given to the entire HU, then the criterion should be raised by 50%. In summary, criteria can be left unmodified, adjusted upward by a fixed amount (50%), or adjusted downward by one of 3 amounts (25%, 50%, or 75%).

Modified criterion values should be rounded to the nearest 0.5 µg Se/L. A criterion that falls equidistant from 2 values is rounded down if the criterion is being lowered, and up if the criterion is being raised. For example, a criterion of 5 µg Se/L that is lowered by 25% (1.25 µg Se/L) becomes 3.5 µg Se/L ($5.0 - 1.25 = 3.75$, rounded down to 3.5). Lowering the same criterion by 50% yields a value of 2.5 µg Se/L, and lowering it by 75% results in a final criterion of 1.0 µg Se/L (1.25 rounded down to 1.0). Criteria that are raised are modified by adding 50% of the criterion value and then rounding up, if necessary, to determine the final value. In theory, criteria could be set at smaller intervals, for example, 0.1 µg Se/L, but it would likely be impossible to scientifically justify or reliably monitor and enforce such criteria. For example, the range of bioaccumulation and effects displayed among fish and aquatic birds (even among the most sensitive species) would offset the perceived benefits of setting criteria at 2.2 and 2.6 µg Se/L rather than 2.0 and 2.5 µg Se/L. Rounding to 0.5 µg Se/L intervals should accomplish the goal of criterion revision—that is, to achieve the desired reduction or prevention of selenium bioaccumulation/effects in aquatic ecosystems—yet also be practical for monitoring, compliance, and treatment/remediation considerations. Diagnostic selenium residues and examples of measured concentrations/effects and appropriate modifications to criteria are given in Table 7.1.

Justification

What is an Appropriate Modification of a Criterion?

The main objective of the procedure given in this chapter is to modify a criterion (by default, the USEPA national criterion) by an *appropriate* amount. *Appropriate*, as used here, is the extent necessary to prevent bioaccumulation from reaching levels that induce toxic effects in fish and aquatic birds. A criterion that is appropriate will be both effective and reasonable. That is, it will provide the needed level of protection to biota throughout the HU without being overly conservative or too liberal. This translates to a criterion which does not require that reference conditions be achieved in every instance but also does not permit toxic impacts to biota. For example, in situations when a criterion is not exceeded, and diagnostic residues are present, but no effects are evident, the decision tree indicates that no modification is necessary (Fig. 7.1). Conversely, when diagnostic residues are present without effects but the criterion is exceeded, no change is indicated. This, in practice, sets 2 major conditions for triggering criterion modification: (1) diagnostic residues *and* toxic effects must be coupled (both must be present) in order to lower a criterion, and (2) diagnostic residues *and* toxic effects must be coupled (both must be absent) in order to raise a criterion. Coupling residues and effects makes the procedure sensitive to the natural inter- and intraspecific variation in bioaccumulation and toxic responses exhibited by fish and wildlife in aquatic ecosystems.

An additional precaution is necessary if the scenario under evaluation is one in which the criterion is not exceeded, and diagnostic residues are present, but there are no observed toxic effects. In this case, the recommended decision is to monitor reproduction but maintain the criterion at the current level. However, it is essential that all of the fish and aquatic bird species that are characteristic of the site—that is, those that are typical for the habitat type and are commonly found in nearby reference locations—be screened for possible use in the assessment of reproductive effects. Selenium-sensitive species can experience reproductive failure and be virtually eliminated while at the same time, some of the most tolerant species remain present and only exhibit elevated tissue residues (Lemly 1985a). This scenario could lead to the false impression that there are no effects and thus no need to lower the criterion, when in fact, selenium-sensitive species have been severely affected and perhaps locally extirpated. This type of error is also possible when the scenario is one in which the criterion is exceeded but no diagnostic residues or toxic effects are evident among the species that are present. It is possible that sensitive species have been eliminated, and the tolerant species that re-

main exhibit very low bioaccumulation, which may partially explain their ability to tolerate selenium. This could lead to the false assumption that increasing the criterion value is appropriate. Thus, in certain situations the biota that “should be present” must be considered, particularly if they represent known selenium sensitive species, in addition to those that are actually present (see previous section, Step 2, for additional guidance in this process). Comparing the fauna and selenium residues of adjacent HUs is one way to evaluate such a situation and gain additional insight into the need to raise a criterion.

Why Use Percentages and Ranges?

Percentages are used because they represent the simplest practical way of modifying criteria while also addressing the main objective from both an ecological and a regulatory perspective. The decision tree specifies that selenium concentrations and effects should be monitored in each case when criterion modification is indicated (Fig. 7.1). It will quickly become apparent if a criterion derived using the percentage method is too conservative or too liberal. In the decision tree, the monitoring requirements function as feed-back loops that provide an on-going field test of the appropriateness of the criterion. Monitoring of residues and biological effects in the HU is an essential part of the validation process for site-specific criteria.

There is a very narrow range covering typical reference waterborne concentrations of selenium in aquatic systems ($0.1\text{--}0.3 \mu\text{g Se/L}$), concentrations that can cause bioaccumulation and toxicity in fish and wildlife ($1\text{--}3 \mu\text{g Se/L}$), and the current national and state criteria for selenium ($2\text{--}5 \mu\text{g Se/L}$). Moreover, the values for site-specific criteria that are likely to be developed by states and approved by USEPA probably fall within the range of 1 to $20 \mu\text{g Se/L}$. Within this narrow range, the most straightforward way to address necessary changes in environmental selenium residues is by using percentages (25%, 50%, 75%) that are linked to different degrees of biological effects. This technique is appropriate because the magnitude of teratogenic deformity and/or embryomortality, and resultant ecosystem-level impacts to fish and aquatic birds are distinctly greater in cases when a 75% reduction in a criterion is indicated (all residues are greater than the upper end of the toxicity threshold ranges) than when a 25% reduction is indicated (some residues are below and some within the threshold ranges).

The use of ranges for threshold values, rather than a single number, makes the criterion-modification process sensitive to natural variation in bioaccumulation and toxic responses of biota. It prevents the process from being overly conservative in cases when a lowered criterion is indicated. Using reference values and reproductive assessment prevents the process

from being too liberal in cases when it is appropriate to raise a criterion. Increases are contingent upon finding low levels of bioaccumulation and normal reproduction in selenium-sensitive species. Thus, modification is always linked to a “no effect” range. Allowing a 50% increase in a criterion is reasonable if there is no evidence of bioaccumulation or reproductive impacts in the HU. Moreover, the feed-back loop provided by monitoring will indicate if further revisions (upward or downward) should be undertaken once a criterion is raised by this amount. This is also true for criteria that are lowered. It is possible that more than one revision will be necessary before an appropriate criterion is achieved.

Implementing Criteria

The USEPA provides states the option of developing site-specific criteria, and regulations governing their implementation, if adequate justification is given. The goal of the derivation process at the state and local levels should be to establish criteria that are sensitive to selenium’s local variations in aquatic cycling and toxic effects. The purpose of this chapter is to provide a procedure for deriving criteria at those levels. However, it is also important to consider how criteria will be implemented and enforced. Will there be provisions for averaging concentrations over several days in order to meet criterion levels? Will periodic exceedance of criteria be allowed? Will mixing zones be used to dilute selenium inputs and achieve criterion concentrations? In many cases, states may choose to follow the example set by the USEPA (eg, Stephan et al. 1985; EPA 1985, 1987), which allows averaging, exceedances, and mixing zones to factor into the implementation of criteria. However, the guidance provided by USEPA is generic and can greatly reduce the effectiveness of site-specific selenium criteria. It is essential that selenium’s profile of environmental cycling and toxicity be used to formulate implementation policies at state and local levels. There are several important precautions that should be taken by water quality regulators if they intend to rely on USEPA guidance documents to implement site-specific selenium criteria.

USEPA guidelines allow the national criterion for chronic exposure to be exceeded periodically (once every 3 years, on average) as long as the 4-day average concentration is 5 µg Se/L or less (EPA 1987). During exceedances, the permissible ambient (ecosystem-wide) water-borne concentration can be as high as 20 µg Se/L (Fig. 7.2). Stephan et al. (1985) gives the rationale for this approach: “the averaging period of 4 days was selected by the USEPA on the basis of data concerning how rapidly some aquatic species react to increases in the concentrations of some pollutants, and 3 years is the Agency’s best scientific judgement of the average amount of time aquatic ecosystems should

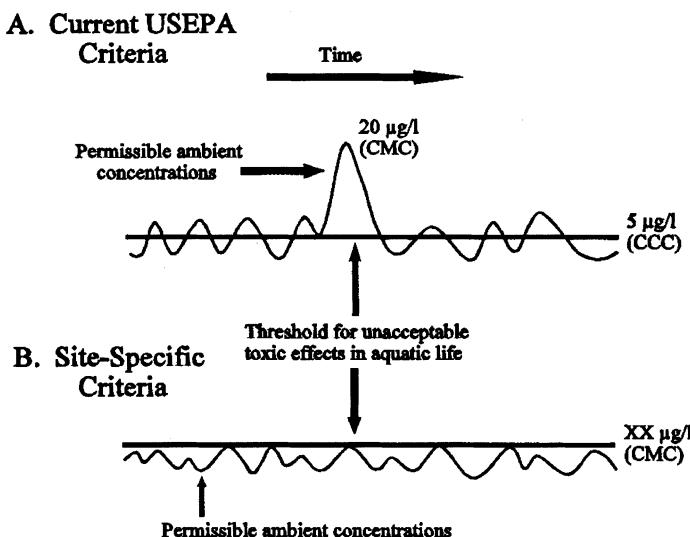


FIGURE 7.2. Contrasts between the existing USEPA national water quality criterion for selenium (chronic exposure, A) and the process for deriving site-specific criteria (this chapter, B). A principal difference is that in implementing United States national criteria, the USEPA guidelines allow 4-day averages and exceedances up to the Criterion Maximum Concentration (CMC, 20 µg Se/L). This can offset the protection to aquatic life that is afforded by the Criterion Continuous Concentration (CCC, 5 µg Se/L). To provide full protection, site-specific criteria should set biologically-based concentration limits as the CMC, and not allow averages, exceedances, or mixing zones (see text for details).

be provided between excursions". The wording of the statement (ie, the words "some aquatic species" and "some pollutants") reveals that this is a generic model for contaminant exposure-response and associated derivation of criteria. The model was developed in the early 1980s when there was relatively little field data on selenium cycling and bioaccumulation in aquatic systems, and USEPA has not tested its assumptions using selenium data that have become available since that time.

There are 4 specific flaws that invalidate the USEPA model when it is applied to selenium:

- (1) The USEPA guidance document clearly indicates that the process for the United States national criteria is molded to fit publicly owned wastewater treatment facilities (POTWs) that discharge a point source into a flowing receiving waterway (Stephan et al. 1985). However, the most widespread threats of selenium poisoning in

aquatic habitats are threats to lentic systems (reservoirs, wetlands, and off-channel bays and impoundments) and are threats from power-plant discharges, agricultural irrigation, and other sources, not from POTWs (see Chapters 1 and 9). The environmental dynamics of selenium in lentic ecosystems is quite different than the riverine conditions used for the USEPA model.

- (2) The 4-day average is based on organism responses to waterborne exposure alone. However, food-chain bioaccumulation and dietary intake are more important in causing chronic selenium toxicity to aquatic life (Lemly 1985a, 1997). This component of selenium cycling is not addressed in the USEPA model. Moreover, exposure–bioaccumulation–response times for selenium in fish and aquatic birds (whether exposure is due to waterborne or dietary selenium intake) are on the order of weeks or months rather than 4 days (eg, Lemly 1982; Heinz et al. 1988; Coyle et al. 1993; Heinz and Fitzgerald 1993). The USEPA model assumption of 4 days is not correct.
- (3) The concentrations of waterborne selenium allowed by the USEPA during exceedances (up to 20 µg Se/L) are not environmentally acceptable for lentic systems or lotic systems that will deliver selenium into off-channel bays, wetlands, reservoirs, or other down-gradient lentic systems. Studies such as those by Cumbie and Van Horn (1978), Bryson et al. (1984), Lemly (1985a), Gillespie and Baumann (1986), and Hamilton et al. (1996) show that concentrations of 10 to 20 µg Se/L can quickly cause dietary levels that are toxic to fish and aquatic birds. Consider, for example, a scenario in which an exceedance causes waterborne selenium in a reservoir or wetland to reach 15 µg Se/L, an acceptable concentration in the USEPA model (Fig. 7.2). By the time ambient locations attain this level, the entire “bioaccumulation engine” of the ecosystem will have been fueled by the influx of new selenium, which substantially escalates the toxic threat to aquatic life (Lemly 1985b).
- (4) The 3-year period between excursions (exceedances), although perhaps reflecting the best scientific judgement available for *some* pollutants in the early 1980s, is not appropriate for selenium given present knowledge of the environmental dynamics and cycling of this trace element. Once an aquatic ecosystem has captured the selenium dose delivered by an exceedance, the ecosystem can continue to cycle the selenium tightly within the components of the system for many years. For example, studies show that the recovery period for reservoirs contaminated by 10 µg Se/L could

be greater than 10 years, perhaps several decades, due to recycling of selenium from sediments into benthic-detrital food chains and associated dietary and reproductive toxicity to fish (Garrett and Inmann 1984; Lemly 1997).

Similar problems are evident with the use of dilution or mixing zones, which are areas exempt from ambient criteria. This concept was developed for application to flowing waters (Stephan et al. 1985). It has no credible basis for application to selenium in lentic/wetland systems because the "dilution zone" may constitute the entire body of open water. Even in riverine habitats, the notion of mixing zones has not been supported by data verifying that mixing zones can effectively dilute a selenium-laden effluent and also be environmentally compatible with fish and wildlife habitat uses, which is a requirement of US federal statutes such as the Migratory Bird Treaty Act and the Endangered Species Act (Margolin 1979). Selenium strongly bioaccumulates in food organisms and makes the dilution zone an area of extremely high exposure for fish and wildlife. Several case studies show that using mixing zones to dilute seleniferous water creates more biological hazards than it resolves (eg, Skorupa 1998). The apparent benefits gained by achieving target concentrations in a mixing zone may be more than offset by detrimental effects that are caused by other aspects of the selenium cycle. The threat of toxic impacts overrides the need to attempt "dilution as a solution".

Given these flaws, it is important to closely examine the rationale for, and the distinction between, national and site-specific criteria. USEPA criteria are intended to provide protection for most aquatic species most of the time, not for all species all of the time (Stephan et al. 1985). Because of this basic caveat and also because there are differences in ecosystem and aquatic species sensitivity to selenium, there may be a plausible argument for allowing some leeway in meeting the national criterion. That is, a *reasonable* averaging of concentrations over time might be permissible if reproductive assessment indicates that there are no biological effects (but not 20 µg Se/L exceedances). However, at a local level, the national criterion's intent to protect "most species" still leaves large gaps that could lead to substantial inconsistencies (toxic effects at or below the criterion level for some species, but no effects for other species even above the criterion). Site-specific water quality criteria should reflect the sensitivity of local biota and close the gaps.

Site-specific criteria for selenium should designate a biologically based concentration limit using the procedure described in this chapter (Figs. 7.1 and 7.2). If full protection of aquatic life is desired, then there should be no provision for averages, exceedances, or mixing/dilution zones in the implementation of these site-specific criteria.

Examples of Criterion Derivation

Example 1: Lowering a Criterion

The existing criterion is 5 µg Se/L, and the following information is available for the HU (µg Se/g concentrations are in dry weight):

Waterborne concentrations of selenium are 1–3 µg/L.

Concentrations of selenium in sediments are 2–4 µg/g.

Concentrations of selenium in aquatic food-chain organisms are 1–7 µg/g.

Concentrations of selenium in randomly sampled aquatic-bird eggs are 4–12 µg/g.

Teratogenic deformities occurred in 2% of randomly sampled aquatic-bird eggs.

Embryomortality occurred in 4 % of randomly sampled aquatic-bird eggs.

Concentrations of selenium in whole-body samples of fish are 6–10 µg/g.

Teratogenic deformities occurred in 5% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 8–12 µg/g.

Evaluation: The residues present in the HU span the toxic threshold ranges (some are below, some are within, and some are above), and reproductive effects are occurring.

Conclusion: These conditions warrant a 50% (2.5 µg Se/L) reduction in the criterion. The final criterion for chronic exposure is set at 2.5 µg Se/L.

Example 2: Raising a Criterion

The existing criterion is 5 µg Se/L, and the following information is available for the HU (µg Se/g concentrations are in dry weight):

Waterborne concentrations of selenium are 5–12 µg/L.

Concentrations of selenium in sediments are <1 µg/g.

Concentrations of selenium in aquatic food-chain organisms are <2 µg/g.

Concentrations of selenium in randomly sampled aquatic-bird eggs are <3 µg/g.

Teratogenic deformities occurred in 0% of randomly sampled aquatic-bird eggs.

Embryomortality occurred in <1% of randomly sampled aquatic-bird eggs.

Concentrations of selenium in whole-body samples of fish are <3 µg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are <3 µg/g.

Evaluation: The residues present in the HU were equivalent to reference levels, and no reproductive effects were evident. Samples from a variety of wetland and riverine habitats indicated the same pattern: little bioaccumulation and no reproductive effects.

Conclusion: The criterion for chronic exposure should be raised by 50%, to a final value of 7.5 µg Se/L.

Example 3: No Modification Necessary

The existing criterion is 5 µg Se/L, and the following information is available for the HU (µg Se/g concentrations are in dry weight):

Waterborne concentrations of selenium are 2–5 µg/L.

Concentrations of selenium in sediments are 1–4 µg/g.

Concentrations of selenium in aquatic food-chain organisms are 1–6 µg/g.

Concentrations of selenium in randomly sampled aquatic-bird eggs are 2–8 µg/g.

Teratogenic deformities occurred in 0% of randomly sampled aquatic-bird eggs.

Embryomortality occurred in <1% of randomly sampled aquatic-bird eggs.

Concentrations of selenium in whole-body samples of fish are 3–6 µg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 3–8 µg/g.

Evaluation: Residues of selenium are at toxicity thresholds, but no reproductive effects are evident (the fraction of a percent of embryomortality present is considered background level, not due to selenium).

Conclusion: No change to the criterion for chronic exposure is indicated, but monitoring of bird and fish reproduction (ie, sampling for evidence of teratogenesis and embryomortality) and associated selenium residues is recommended.

Example 4: Examining a Proposed Criterion

The proposed criterion for chronic exposure is 15 µg Se/L, and the following information is available for the HU (µg Se/g concentrations are in dry weight):

Waterborne concentrations of selenium are 10–20 µg/L.

Concentrations of selenium in sediments are 1–2 µg/g.

Concentrations of selenium in aquatic food-chain organisms are 1–3 µg/g.

Concentrations of selenium, terata, and embryomortality in randomly sampled aquatic bird eggs are unavailable because few aquatic birds nest at the site.

Concentrations of selenium in whole-body samples of fish are 2–5 µg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 3–6 µg/g.

Possible impacts to down-gradient systems are unknown.

Evaluation: No reproductive effects are evident in fish, and potential threats to aquatic birds are likely minimal because few of them were found to nest or feed at the site. However, the habitat usage and selenium status of some river habitats and associated wetlands have not been characterized. Moreover, concentrations of selenium in fish are at the lower end of the diagnostic toxicity range.

Conclusion: The proposed criterion of 15 µg Se/L is rejected because of possible threats to down-gradient systems in the HU as well as the presence of elevated selenium residues in fish and food-chain organisms. Monitoring of selenium residues in fish and down-gradient habitats is recommended.

Conclusions

During the past 2 decades, selenium has gained a reputation as an important aquatic contaminant, and it is currently classified as one of the USEPA's priority pollutants. The USEPA issued a revised (lowered) national freshwater criterion for selenium in 1987, but there is considerable disagreement as to the applicability of the national criterion at the state and local levels. Some believe it should be lowered; some believe it should be raised. This has led to more and more instances of states and municipalities attempting to develop site-specific criteria. However, there is no published or widely accepted technique available that gives specific guidance for selenium. The method presented in this chapter should fill that infor-

mation gap. A simple, straightforward procedure is provided for using information on the selenium status of aquatic ecosystems and resultant threats/impacts to biota as the basis for deriving criteria. The method is not limited to the United States and its EPA context. It can be applied for water quality assessment and regulation throughout North America and abroad. An important part of the procedure is the delineation of a Hydrological Unit (HU) as the physical area to be examined in the review process. The HU approach ensures that site-specific criteria will protect biota in all habitat types and environmental conditions. Once appropriate criteria are derived and implemented, it may be necessary to reduce selenium inputs to the HU in order to achieve the desired water quality. However, reducing selenium loading is not a simple task and may involve a combination of point-source reduction and changes in land use activities. A procedure that is becoming widely used in the United States to control pollutants on a watershed basis, especially when non-point sources are involved, is known as Total Maximum Daily Loads (TMDLs). When combined with the HU approach, TMDLs may become an effective tool for controlling selenium levels in order to meet water quality goals. The next chapter presents a method for setting environmentally safe TMDL limits for selenium.

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8

Setting Ecosystem Loading Limits

Introduction

This chapter describes a step-by-step procedure I developed to set Total Maximum Daily Load (TMDL) limits for selenium. The need for this procedure in the United States stems from actions taken by the US Environmental Protection Agency (USEPA) that require TMDL limits on impaired water bodies. For many years, water-quality regulations in the United States have been implemented through point-source control programs such as the NPDES (National Pollution Discharge Elimination System) permit system. Recently, the USEPA has placed more emphasis on characterizing and reducing non-point sources of pollution in order to provide watershed-level improvement of water quality. This approach, known as Total Maximum Daily Loads, sets a limit on the total aggregate amount of a contaminant allowed in an aquatic system. The objective of TMDLs is to identify all pollution sources and then allocate/regulate discharges to meet the water-quality needs of aquatic life within a watershed. The states are required to identify impaired water bodies in order to comply with Section 303(d) of the Clean Water Act. The USEPA is working with states and implementing the TMDL program as a way to gauge point and non-point sources of pollution, to control/reduce discharges, and to improve overall water quality pursuant to the requirements of Section 303(d). The USEPA has requested that state and federal biologists provide input for this process for substances ranging from nutrients and sediments to pesticides and trace elements such as selenium. Every substance identified as a priority must be given a separate TMDL assessment, and this must be done individually for each impaired water body. The biologist's role may range from a simple review and recommendation, based on available information, to coordination/conduct of field work to assess contaminant cycling and fate, fish and wildlife exposure, toxicity, and other factors, involving considerable time and resources.

The magnitude of this effort becomes apparent when one considers that each state may have dozens to even hundreds of impaired water

bodies that will need TMDLs, many of which could involve selenium, because of its widespread concern as a water quality issue (Lemly et al. 1993; Lemly 1999). Although the USEPA has published an overview document that explains principles underlying the development and implementation of TMDLs (EPA 1999), no procedures specific to selenium are given. This leaves biologists and environmental contaminant specialists without the assessment framework necessary to effectively address the TMDL issue for selenium. Due to selenium's propensity to bioaccumulate, to cause reproductive impairment in fish and wildlife, and to persist in the environment, selenium-specific methods are needed. It is critical to have a technically sound approach for evaluating selenium, because it is considered a priority contaminant by natural resource managers and the USEPA. This chapter provides guidance by laying out an assessment method that links the basic components of the USEPA's TMDL program to the contaminant-specific information necessary for selenium.

Procedure

This method is structured to answer 2 basic questions: (1) Is selenium impairing the water body based on biological criteria? and (2) If so, what amount of selenium load reduction is necessary to correct the problem? A 7-step procedure (Fig. 8.1) can be used to answer these questions and develop environmentally safe TMDL limits for selenium. The basic premise in this approach is that selenium concentrations should be kept below levels that threaten the reproduction of fish and aquatic birds.

Step 1: Delineate and Characterize the Hydrological Unit

TMDLs for selenium should be based on an assessment of the degree of toxicological hazard to fish and wildlife. This assessment is influenced by the hydrology of the site under consideration. The physical area from which measurements are taken to evaluate selenium concentrations and biological threats/effects, that is, the database for setting TMDLs, must encompass more than an isolated segment of a river, a tributary stream, or other limited area. Because of hydrological connections between the various aquatic habitats that may be present in a watershed basin—wetlands, rivers, streams, lakes, and impoundments—the toxic threat from selenium contamination is also connected. For example, a TMDL limit that is set for a stream or river where low bioaccumulation occurs may result in *seemingly* harmless concentrations becoming a problem in downstream impoundments or in off-channel bays and wetlands where bioaccumulation is greater.

TMDL DECISION TREE FOR SELENIUM

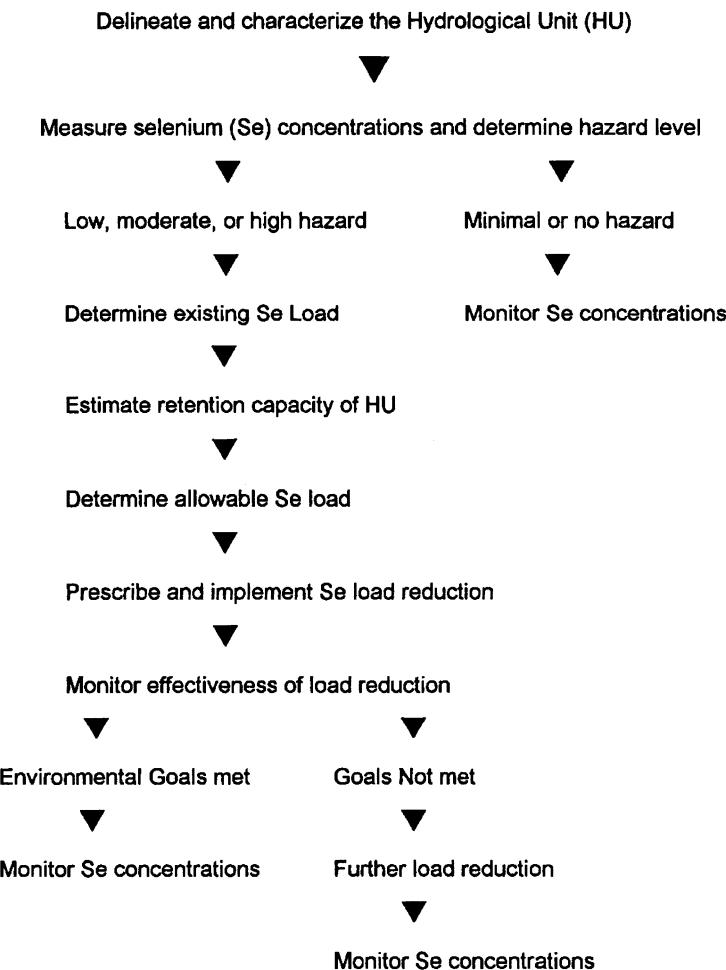


FIGURE 8.1. Steps in the process of establishing a Total Maximum Daily Load (TMDL) for selenium.

The hydrologically connected parts of a basin that are down-gradient of a selenium input (natural or anthropogenic selenium source), extending to the point at which new sources of low-selenium water dominate the hydrology and cause concentrations to fall below levels of concern, should be the area evaluated and given a specific TMDL, not isolated parts. Such a source of low-selenium water might be a confluence with a larger tributary or river, a spring, or groundwater inflow, resulting in concentrations that do not exceed background levels for the area. Thus, a Hydrological Unit (HU) should be identified and used as the “site” for the

purpose of setting TMDL limits. Importantly, TMDLs derived in this manner will reflect the transport and bioaccumulation of selenium within the entire HU, rather than simply focusing on a small, artificially designated segment of the system. Failure to use a HU approach can set the stage for significant biological problems and undermine the intended outcome of the TMDL process. Consult Chapter 6 for more information on the rationale and justification for using HUs.

In order to properly delineate the HU, some information on water-borne selenium concentrations is needed. This can be obtained from monitoring data or from new measurements taken to provide a screening-level assessment. Use the entire HU as a "mixing zone" for the purpose of evaluating potential selenium transport and bioaccumulation. Characterize and map the aquatic system of the HU using available information in combination with field reconnaissance to identify/verify hydrological connections. Identify all aquatic habitats within the HU: wetlands, streams, rivers, off-channel bays, lakes, reservoirs, other impoundments. Map their spatial and gradient/hydrological relationships, determining what flows where and what flows into/out of what. Obtain information on trophic status, prevailing sediment type (organic, inorganic, mixed), and volume replacement times or flushing rates for lakes, reservoirs, and other impoundments, bays off main-stem rivers, and wetlands. Describe the general level of primary productivity (low-oligotrophic, moderate-mesotrophic, high-eutrophic), predominant flow regime (slow, moderate, swift), and dominant sediment characteristics (depositional, erosional, particle size, organic, inorganic, mixed) of flowing-water habitats. Large rivers merit special attention in order to identify, map, and describe the variety of habitats that may be present, for example, main-stem, off-channel bays, seepage or floodplain wetlands. Characterize fish and wildlife uses (feeding, spawning, nesting, migration, etc.) and identify biota of special concern, for example, endangered or threatened species, management priorities, and selenium-sensitive species (eg, centrarchid fishes, *Lepomis* sp., *Micropterus* sp.; waterbirds such as stilts, *Recurvirostra* sp.; and ducks, *Anas* sp.). Also, identify habitats where bioaccumulation would likely be greatest (eg, wetlands, lakes, reservoirs and other impoundments, and off-channel backwater areas of rivers).

Step 2: Determine if Hazardous Levels of Selenium are Present

Gather information on selenium concentrations. If there is no recent monitoring data (within the past 2 years), or if the data do not satisfy the following requirements, then it will be necessary to collect and analyze new samples. Selenium concentrations are needed for 5 ecosystem components: water, sediments, benthic macroinvertebrates, fish

eggs, and aquatic bird eggs (convert fish/bird tissue values to egg values using a conversion factor if no eggs are available; that is, bird liver selenium $\times 0.33$ = approximate bird egg concentration; fish whole-body selenium $\times 3.3$ = approximate fish egg concentration; Lemly and Smith 1987). If bird eggs *or* fish eggs cannot be obtained (if only one of these two is available), a 4-component assessment can be done. Collect a minimum of 10 samples of each component in each major habitat type present in the HU (stream, river, off-channel bay, wetland, lake, reservoir, other impoundment). This number of samples is sufficient to reveal the range of concentrations likely to be encountered (Lemly 1993, 1997). Make sure to designate sampling sites throughout the HU to provide adequate spatial coverage. Some of the required measurements for waterborne selenium may be available from the screening-level assessment done in Step 1. Maintain high quality assurance/quality control in all sampling and analysis; document QA/QC procedures for future reference. Evaluate selenium concentrations with the Protocol (see Chapter 4) to determine the hazard rating. A rating of low, moderate, or high hazard indicates that the TMDL process should continue. A rating of either minimal or no hazard indicates that TMDL calculations are not necessary, but the HU should be monitored by applying the Protocol to selenium measurements made at 3-year intervals. If monitoring reveals that hazard has increased above the minimal level, TMDL reductions are needed.

Step 3: Determine Selenium Sources, Concentrations, and Volumes

Identify all possible sources of selenium (agricultural, industrial, petrochemical, mining, etc.) and map them in the HU, noting their proximity/discharge to specific habitat types, that is, wetlands, streams, rivers, reservoirs, and other HU habitats. Determine/verify selenium concentrations from each source, using existing data or analyzing new samples. Determine/estimate average discharge volume from each source. Calculate total existing selenium loading rate for the HU (kg Se/day).

Step 4: Estimate the Retention Capacity of the Hydrological Unit for Selenium

A key part of the TMDL process for selenium is to estimate retention capacity (RC). The RC will reflect the propensity of the HU to accumulate selenium and, thereby, serve as an indicator of how much selenium the system will likely retain and potentially cycle into bioaccumulation pathways. For the purposes of this chapter, the RC is defined as the natural ability of a system to accumulate and conserve selenium. Components of

the RC include bioaccumulation, detrital retention, physical and chemical sequestration, and recycling within the HU. The more that selenium is held within a HU—through incorporation into biota, deposition in sediments, etc.,—the higher the RC. It is necessary to know the RC in order to develop an environmentally sound TMDL because the higher the RC, the lower the TMDL has to be to prevent toxic threats to fish and wildlife.

To a large extent, the RC depends on the degree of bioaccumulation and internal recycling in the HU, which is reflected in: (1) the primary productivity, (2) the water flow regime, and (3) the sediment type. From the characterization of HU done in Step 1, information on these 3 factors should be available for each type of aquatic habitat. Use the matrix in Table 8.1 to assign each of these factors a separate RC rating; low, medium, or high. A rating should be done for each distinct habitat in the HU, that is, for each main-stem river, off-channel bay, stream, wetland, lake, reservoir, and other impoundment.

An overall RC rating for each habitat is determined by combining the 3 factor ratings as follows:

3 low ratings = low RC

2 low and 1 medium = low RC

2 low and 1 high = medium RC

2 medium and 1 low = medium RC

2 medium and 1 high = medium RC

3 medium ratings = medium RC

2 high and 1 low = medium RC

2 high and 1 medium = high RC

3 high ratings = high RC

The final RC rating for the HU should be set equal to the highest individual habitat rating. For example, if there are two habitats with low RC and one with medium RC, the final RC rating for the HU is medium.

Step 5: Calculate the Total Allowable Selenium Load

Begin by plugging the hazard rating from Step 2, and the RC from Step 4, into the matrix in Table 8.2. The table indicates the appropriate amount of load reduction needed: small, medium, or large. The total existing selenium load should be reduced by 10% if the amount designated is small, 25% if it is medium, and 50% if it is large. Subtract the indicated amount from the total existing selenium loading rate (kg/

TABLE 8.1. Retention capacity (RC) ratings for selenium in aquatic systems based on habitat type and general biological/physical characteristics.

	Habitat type		
	Stream, main-stem river	Lake, reservoir, off-channel bay, impoundment	Wetland
Productivity			
High (eutrophic)	High	High	High
Moderate (mesotrophic)	Medium	Medium	Medium
Low (oligotrophic)	Low	Low	Low
Flow			
Swift	Low	Low	Low
Moderate	Medium	Medium	Medium
Slow	High	High	High
Sediment			
Inorganic	Low	Low	Low
Mixed	Medium	Medium	Medium
Organic	High	High	High

day) calculated in Step 3. This will yield the total allowable selenium load for the HU.

Step 6: Allocate Allowable Load Among Selenium Sources

Designate allowable discharges keeping habitat types and sensitive species in mind. For example, it would be inappropriate to allow the largest loading to occur in habitats occupied by priority species (threatened or endangered, or management priority), selenium-sensitive species (eg, centrarchid fishes, *Lepomis* sp., *Micropterus* sp.; waterbirds such as stilts, *Recurvirostra* sp.; and ducks, *Anas* sp.), or where bioaccumulation would likely be greatest (eg, in a wetland, lake, reservoir, or off-channel backwater area of a river).

TABLE 8.2. Amount of selenium load reduction necessary for a Hydrological Unit (HU) based on hazard rating and retention capacity (RC)^a.

	Hazard Rating		
	Low	Moderate	High
RC of HU			
Low	Small	Medium	Large
Medium	Medium	Medium	Large
High	Medium	Large	Large

^a Necessary load reductions = 10% for small, 25% for medium, 50% for large.

Step 7: Monitor to Determine Effectiveness of Load Reduction

The objective of the TMDL process is to keep selenium concentrations below levels that are toxic to biota. Therefore, it is important to use environmental quality goals as a guide in follow-up effectiveness monitoring. For this purpose, I recommend that the following guidelines be used as maximum allowable selenium concentrations (see Chapter 2):

Water = 2 µg Se/L, filtered samples (0.45 µm pore size)

Sediment = 2 µg Se/g dry weight

Benthic invertebrates = 3 µg Se/g dry weight

Fish tissues:

whole body = 4 µg Se/g dry weight

skeletal muscle (skinless fillets) = 8 µg Se/g dry weight

liver = 12 µg Se/g dry weight

ovary and eggs = 10 µg Se/g dry weight

Aquatic bird tissues:

liver = 10 µg Se/g dry weight

eggs = 7 µg Se/g dry weight

These guideline values represent concentrations that are protective of fish and wildlife reproduction. Monitor selenium residues annually and apply the Protocol (same as for Step 2) to determine if hazard is reduced to either the minimal or no hazard level. If it is, then no further load reductions are necessary—conduct environmental monitoring every 3 years. If it is not, repeat Step 5 to determine the additional amount of selenium load reduction necessary, implement load reduction, and monitor annually. The entire TMDL process is summarized in Figure 8.1.

Conclusions

On the surface, the TMDL program may seem to be a formidable USEPA regulatory requirement that generates uncertainty, apprehension, and is not easily addressed. Without adequate technical guidance, this may be true in many cases. However, the straightforward, 7-step procedure given in this chapter takes away the uncertainty and allows environmentally safe TMDL limits to be set for selenium. This method is tailored to account for selenium's ability to bioaccumulate and cause reproductive toxicity to fish and wildlife. A key part of the method is the identification and use of a Hydrological Unit (HU) as the basis for evaluation. The HU approach provides the contaminant-specific site

characterization that is necessary for selenium. Proper use of this TMDL technique will ensure compliance with USEPA regulatory requirements and also protect fish and wildlife resources. However, application of the method is not limited to the United States and the USEPA context. It is appropriate wherever TMDLs are adopted as a means of improving water quality.

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Section IV

Outlook for the Future

9

Emerging Selenium Contamination Issues

Introduction

Because of recent increases in human activities that promote the introduction of selenium into aquatic habitats, new threats are emerging on a broad scale in the United States and elsewhere. An important factor in these threats is that food-chain bioaccumulation and resultant dietary exposure cause the response curve for selenium poisoning in fish to be very steep. For example, a transition from no effect to complete reproductive failure can occur within a range of only a few $\mu\text{g/L}$ (parts per billion) waterborne selenium (Fig. 9.1). Thus, activities that cause even slight increases can quickly push selenium bioaccumulation over the toxic threshold.

In the United States, anthropogenic disturbances have greatly increased the likelihood that aquatic ecosystems will experience elevated selenium. From the 1960s through the 1980s, two disturbances stood apart as the major human-related causes of selenium mobilization on a regional and national scale: (1) procurement, processing, and combustion of fossil fuels (Lemly 1985); and (2) irrigation of seleniferous soils for crop production in arid and semi-arid regions (Lemly et al. 1993). Since the early 1990s, other issues have emerged as potentially important factors in the mobilization and bioaccumulation of hazardous concentrations of selenium. Primary among these are: (1) phosphate mining wastes, (2) use of constructed wetlands to treat selenium-laden wastewater, (3) landfill disposal of ash from coal-fired power plants, and (4) accumulation of animal waste at livestock feedlots and intensive rearing facilities. This chapter examines these issues in the context of new, largely uninvestigated selenium threats that may be sufficient to cause widespread impacts to aquatic ecosystems.

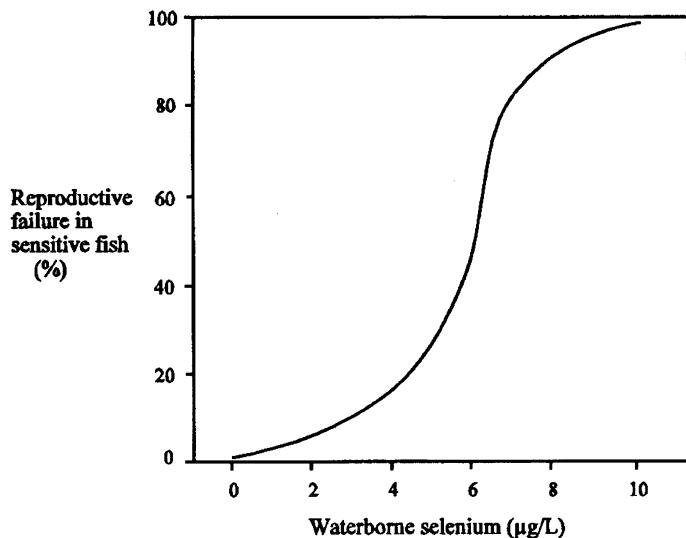


FIGURE 9.1. Relationship between the concentration of selenium in habitats favorable for bioaccumulation (eg, impoundments, off-channel bays, wetlands) and the degree of reproductive failure in sensitive fish species (eg, bluegill, *Lepomis macrochirus*). A small increase in waterborne selenium can result in catastrophic impacts on reproductive success. Recent escalation in human activities that promote the mobilization of selenium into aquatic ecosystems threatens to impact fish populations on a widespread scale in the United States and elsewhere.

Escalating Threats of Contamination

Open-Pit Phosphate Mining

In the United States, geologic formations of phosphate-bearing rock (Phosphoria) occur in Montana, Idaho, Wyoming, and Utah. This western phosphate field contains about 40% of the total phosphate reserves of the continental United States. Commercial mining began in 1906 near Montpelier, Idaho, and since that time has steadily expanded to include numerous open-pit operations in southeastern Idaho that extract phosphate for use in fertilizer or as elemental phosphorus. A large part of the phosphate mineral reserves are located on national forest lands. Mineral leases for mining are issued by the US Forest Service and administered by the US Bureau of Land Management.

During the mining process, large amounts of waste residuals are excavated and disposed on the surface in dumps and landfills. Some of

these waste beds contain over 50 million cubic meters of tailings, and most intersect or drain into streams. Water quality concerns were raised in 1996 when it was discovered that livestock grazing on pastures bordering streams near the waste dumps developed symptoms that were diagnosed as selenium poisoning. Subsequent monitoring studies revealed high concentrations of selenium in phosphate mine solid wastes (up to 1040 µg Se/g, parts per million), seepage water (up to 1500 µg Se/L), tailings ponds (up to 1500 µg Se/L), and streams (up to 474 µg Se/L) near the waste dumps (Desborough et al. 1999; Herring et al. 1999; Lemly 1999).

Most of the mining activity occurs within the Blackfoot River watershed, which contains a Class 1 fishery (highest valued) of Yellowstone cutthroat trout (*Oncorhynchus clarki bouvieri*). A preliminary hazard assessment of selenium in the watershed indicated that waterborne selenium concentrations in the Blackfoot River and in 14 of its tributaries met or exceeded toxic thresholds for fish. Concentrations of selenium in fish tissues exceeded toxic thresholds for reproduction at 1 location and approached the threshold at 2 others. It was concluded that there was a high risk of toxic impacts to Yellowstone cutthroat trout and other fish associated with the Blackfoot River, its tributary streams that receive drainage from mine spoil sites, and Blackfoot Reservoir (Lemly 1999).

This selenium problem centers around surface disposal of mine spoils. With the advent of more stringent environmental controls in the 1990s, backfilling of mine pits has been used as a way to minimize the need for surface disposal of tailings. However, because soil and rock expands when it is dug out of a mine pit, from 10 to 30% of the excavated material still requires surface disposal. Compounding this problem is the presence of historic tailings dumps, many of which are huge (>10 million cubic meters) and contain a tremendous reservoir of selenium that has the potential to be mobilized and introduced into aquatic habitats. Large mineral leases are awaiting development, both on and off national forest lands (Desborough et al. 1999; Herring et al. 1999), which suggests that continued expansion of phosphate mining is likely. Phosphate mining has the potential to elevate selenium levels in aquatic ecosystems across extensive areas of the intermountain West. The cascade of events leading to toxic impacts has already started at some locations.

Constructed Wetlands

In the mid-1980s, a new selenium issue emerged in central California. Irrigation of crop fields in the San Joaquin Valley produced seleniferous drainage water that was disposed in the San Luis Drain and

conveyed to Kesterson National Wildlife Refuge, where it caused death and deformities in thousands of migratory waterfowl and shorebirds (Lemly et al. 1993). A variety of treatment options for removing selenium from subsurface irrigation drainage and reducing hazards to fish and wildlife in downstream waters were examined. One method, tested experimentally in the late 1980s and promoted during the 1990s, is the use of constructed wetlands. In addition to treating irrigation drainage, this "phytoremediation" approach has also been advocated as a means to remove selenium from oil refinery effluents (Terry and Zayed 1998). However, it is important to recognize that serious ecological risks may accompany this treatment technology.

The major objective of treatment wetlands is to remove materials that could threaten the health and biological integrity of down-gradient receiving waters. If that goal is achieved, ecological benefits result. However, if the wastewater being treated contains selenium, the apparent benefits to downstream water quality can be more than offset by toxic hazards created within the wetlands because of bioaccumulation. Moreover, wetlands constitute attractive habitat for fish and wildlife, which are then likely to be exposed to hazardous levels of selenium. The end result can be a net loss of benefits and creation of an ecological liability that did not previously exist. Treatment wetlands may thus create selenium problems rather than solve them. That result did take place at the Chevron USA Oil Refinery in Richmond, California in the mid-1990s. A 40 ha constructed wetland, intended to provide "water enhancement" by removing conventional pollutants (biological oxygen demand, total organic carbon, total suspended solids, ammonia, etc.), was also effective in removing selenium from the waste stream. This was initially viewed as an unanticipated net benefit. The habitat feature the wetland provided attracted large numbers of migratory waterfowl and shorebirds, which was also promoted as a benefit of the wetland. However, bioaccumulation caused selenium levels to exceed toxic thresholds for wildlife, and waterbirds were poisoned. In 1995, selenium concentrations in birds were "high enough to reduce hatchability of eggs and may cause some reduction of post-hatch survival among chicks" (Ohlendorf and Gala 2000). That finding prompted the implementation of an alternative management plan designed to reduce wildlife exposure by manipulating vegetation and water levels in areas of highest selenium concentrations so as to make them less attractive to birds. After several years under this new management plan, selenium levels in bird eggs had decreased but still exceeded thresholds for reproductive toxicity (Chevron 2000). This example illustrates the difficulty of meeting water-quality treatment objectives without creating toxic hazards to wildlife. In the United States, there are also important legal issues associated with the creation of

such hazards. For example, federal statutes such as the Migratory Bird Treaty Act and the Endangered Species Act prescribe strict penalties (monetary fines and/or imprisonment) for killing wildlife by these means, whether or not the owner/operator knowingly allows this "take" to occur (eg, Margolin 1979).

The underlying problem with constructed wetlands is the failure of those who develop wetland selenium treatments to adequately evaluate risks to fish and wildlife. For example, researchers developing treatment methods typically seek to establish how effective wetlands can be in removing selenium from water, but make little or no effort to document or acknowledge ecological hazards (eg, Hansen et al. 1998; Terry and Zayed 1998). Consequently, the methods have inherent dangers that are not readily apparent to potential users, a major shortcoming that is pervasive in the wetland treatment technology field. Many wetland-treatment methods are being marketed on a national scale in the United States without full knowledge or disclosure of the risks they pose to fish and wildlife. Constructing a wetland to treat selenium-laden wastewater may result in selenium poisoning wherever this practice is used.

Disposal of Fly Ash

Treatment technologies to reduce airborne particulate emissions from coal-fired power plants have reached a high level of efficiency, sometimes achieving in excess of 99.5% removal. However, this impressive protection of air quality belies other environmental risks associated with large-scale burning of coal. Huge volumes of seleniferous fly ash (50–300 µg Se/g) and other combustion wastes are generated in the process. The current annual production of coal ash in the United States is about 120 million tons and is projected to steadily increase in the coming decades (EPA 1998). Most fly ash is disposed in landfills that are generally built on clay soils (to impede downward movement of contaminants or upward movement of groundwater), capped with a layer of clay (to impede infiltration of rainwater) and topsoil, and revegetated. Over time, landfills can become unstable, allowing the surface clay cap or the underlayment clay to crack, rainwater or groundwater to infiltrate, and leaching of selenium to occur. Selenium-laden seepage (50–200 µg Se/L) can then be transported off-site, where it may ultimately reach streams or other surface water, bioaccumulate, and threaten the health of fish populations. In fact, the design specifications for fly-ash landfills acknowledge that, even under the best conditions, some contaminated leachate will result (Murtha et al. 1983).

One example of this problem occurred in 1991 in eastern Pennsylvania, where plans were being made to construct a 65 hectare landfill

to dispose of fly ash from 5 different power plants. Concerns were voiced by local wildlife conservation groups regarding the possibility that selenium-laden leachate from the landfill would threaten populations of native brook trout (*Salvelinus fontinalis*), a highly valued sport fish in the region. This prompted an investigation by the Pennsylvania Department of Environmental Resources (PDER) into the leaching behavior of selenium in fly-ash. The initial investigation provided evidence that selenium leaching was a legitimate concern, leading the PDER to revoke the disposal permit for 2 of the 5 facilities identified in the original application (Pennsylvania Bulletin 1992) and to call for strict controls on the physical and chemical characteristics of ash materials allowed in the landfill (Commonwealth of Pennsylvania 1991). Additional investigations revealed more potential problems with selenium in leachate from the site and resulted in the landfill being repermitted to handle only construction and demolition waste—no fly ash was allowed. The events in this case represent major adjustments to Pennsylvania laws brought about by environmental concerns over selenium in fly-ash landfills. Moreover, the actions taken by the PDER set an important precedent by establishing very rigid requirements for landfills that are proposed in the future.

Coal-fired power production in the new millennium will increase the need for new fly-ash landfills which, in turn, will steadily increase the threat of selenium-laden leachate contaminating aquatic habitats and impacting fish populations. When the large number of existing waste dumps is factored in, it becomes clear that the risk of serious ecological consequences is on the rise. Every new fly-ash landfill is a potential selenium source to down-gradient aquatic habitats, expanding the threat on a regional and national scale.

Feedlot Wastes

Selenium is widely used as a nutritional supplement to livestock diets, and it is common for the liquid manure associated with swine or cattle feedlots to contain 50 to 150 µg Se/L (Oldfield 1998). A primary reason for these relatively high levels is that livestock feeds are typically supplemented with selenium to enhance growth; thus, higher intake means higher concentrations in wastes. Because excreted selenium has been physiologically processed by the animal, the chemical forms present in this matrix likely include various organic metabolites or other organic compounds. Importantly, organic selenium has a much greater bioaccumulation potential in aquatic ecosystems than the inorganic selenium that is associated with other pollution sources (eg, coal-fired power plants, subsurface agricultural irrigation drainage). For example, waterborne inorganic selenate and selenite typically bioaccumulate 100 to 4000 times in aquatic food chains, but organic selenoamino

acids can produce bioaccumulation factors in excess of 350 000 (Besser et al. 1993). This magnitude of bioaccumulation means that a water-borne concentration of only 0.1 µg Se/L organic selenium, in the right chemical form, is sufficient to elevate residues in food-chain organisms to levels that are toxic in the diet of fish and aquatic birds (5–15 µg Se/g).

The threat of selenium contamination from intensive feedlot operations becomes apparent when one considers the size and number of these operations in combination with the magnitude of pollution events that have occurred due to drainage or spills from manure pits. For example, in the Coastal Plain region of eastern North Carolina, modern swine rearing farms house 5000 to 20 000 animals each. Hog production nearly tripled during the 5-year period from 1990 to 1995, increasing from 5 million to 14 million animals. The large volume of liquid manure produced is stored in multimillion-gallon lagoons until it is removed and sprayed onto fields. Spills or overflows from these sewage lagoons or pollution from runoff of manure from excessively sprayed fields is a common occurrence (Schildgen 1996). In 1995, a 25-million gallon spill entered North Carolina's New River and killed fish for 20 miles downstream. Hog waste was also implicated in massive pollution of the state's Neuse River, which was quarantined for a 35-mile stretch in 1995 (Schildgen 1996). Hog wastes also likely contributed to outbreaks of a toxic dinoflagellate (*Pfiesteria piscicida*) in the Pamlico River Estuary (Glasgow et al. 1995).

Concerns for aquatic impacts of livestock wastes are currently focused on nutrient enrichment, bacterial contamination, and depression of dissolved oxygen. There is little or no recognition of selenium issues. Nevertheless, intensive livestock feeding operations generate a large volume of seleniferous waste that has the potential to severely impact nearby aquatic ecosystems as well as downstream habitats such as wetlands and estuaries. This is an escalating threat that could affect many areas of the United States. Two types of monitoring are needed: first, to evaluate the acute or residual selenium contamination of aquatic life in pollution events associated with feedlot operations; and second, to determine selenium movement from fields sprayed with large amounts of liquid manure.

Conclusions

The risk of widespread impacts from new selenium threats is greater than ever before. Human activities that increase waterborne concentrations of selenium and provide conditions favorable for bioaccumulation are on the rise. Overshadowed by other concerns, selenium is the "unknown contaminant" for some of these activities, yet it may

pose the most serious long-term threat to aquatic habitats and fishery resources. The cascade of events leading to toxic impacts has already started at some locations. Only environmentally sound hazard assessments followed by appropriate management actions can prevent selenium poisoning from taking place. The methods and guidelines in this book should prove useful in the assessment and resolution of new selenium issues in the United States and elsewhere.

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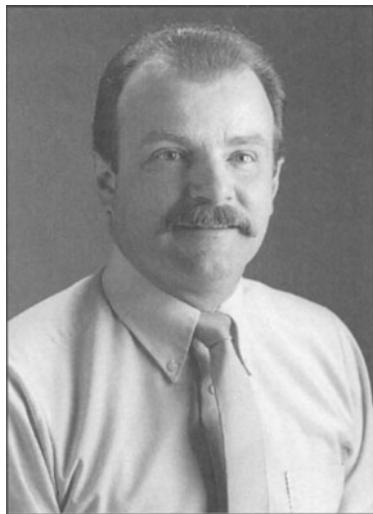
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About the Author

A. Dennis Lemly has spent over 20 years investigating the effects of selenium pollution in aquatic ecosystems. His career began in the late 1970s with studies of the landmark pollution event at Belews Lake, North Carolina, which established the fundamental principles of selenium bioaccumulation and reproductive toxicity in fish. In the 1980s, he was a research project manager for the US Fish and Wildlife Service, directing studies that determined impacts of selenium from agricultural irrigation on aquatic life in California and other western states. In the 1990s, his emphasis shifted to the development of methods and guidelines for hazard assessment and water quality criteria for selenium, which led to the publication of this book. He has consulted on selenium contamination issues around the world, including such problems as power plant discharges in Australia, gold mining effluents in the Canadian Arctic, and agricultural irrigation drainage in Egypt. He has written over 100 scientific articles, book chapters, and technical reports on selenium. Dennis has a Ph.D. in Biology from Wake Forest University, and holds appointments on the faculty at Duke University and Virginia Tech University. He is currently a Senior Research Scientist with the US Forest Service in Blacksburg, Virginia, where he is investigating new causes of selenium pollution in the USA and elsewhere.

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