Environmental Implications of Excessive Selenium: A Review

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Selenium is a naturally occurring trace element that is nutritionally required in small amounts but it can become toxic at concentrations only twice those required. The narrow margin between beneficial and harmful levels has important implications for human activities that increase the amount of selenium in the environment. Two of these activities, disposal of fossil fuel wastes and agricultural irrigation of arid, seleniferous soils, have poisoned fish and wildlife, and threatened public health at several locations in the United States. Research studies of these episodes have generated a data base that clearly illustrates the environmental hazard of excessive selenium. It is strongly bioaccumulated by aquatic organisms and even slight increases in waterborne concentrations can quickly result in toxic effects such as deformed embryos and reproductive failure in wildlife. The selenium data base has been very beneficial in developing hazard assessment procedures and establishing environmentally sound water quality criteria. The two faces of selenium. required nutrient and potent toxin, make it a particularly important trace element in the health of both animals and man. Because of this paradox, environmental selenium in relation to agriculture, fisheries, and wildlife will continue to raise important land and water-management issues for decades to come. If these issues are dealt with using prudence and the available environmental selenium data base, adverse impacts to natural resources an&public health can be avoided.

INTRODUCTION

Selenium is a trace element that is normally present in surface waters at concentrations of about 0.1-O. $3\mu g/l$ (parts-per-billion; Lemly, 1985a). In slightly greater amounts, i. e. , $1-5\mu g/l$, it can bioaccumulate in aquatic food chains and become a concentrated dietary source of selenium that is highly toxic to fish and wildlife (Lemly and Smith, 1987; Lemly, 1993a). Dietary selenium is passed from parents to offspring in the eggs, where it can be teratogenic to developing embryos and cause complete reproductive failure (Gillespie and Baumann, 1986; Heinz *et al.* , 1987, 1989; Coyle *et al.* , 1993 ; Lemly, 1993b). This scenario of poisoning has occurred in reservoirs contaminated by selenium leached from fly-ash at coal-fired electric generating stations in the eastern U. S. (Garrett and Inmann, 1984; Lemly, 1985b), and in wetlands **used** to dispose subsurface agricultural irrigation drainage in the western U.S. (Lemly, *et al.*, 1993; Lemly 1994a,1994b). Public health may also be threatened if humans consume selenium-contaminated fish and wildlife (Fan *et al.*, 1988).

Because of these and other episodes of contamination, the role and importance of selenium as an environmental pollutant has gained widespread attention among research scientists, natural resource managers, and regulatory agencies in the U. S. during the past decade. Although the basic toxicological symptoms and paradox of selenium (nutritionally required in small amounts by both animals and humans, 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, 1980a; Braunstein *et al.*, 1981). In fact, as recently as 1970 selenium was being called the "unknown pollutant" with respect to what was known about its cycling and toxicity in the aquatic environment (Copeland, 1970). Since that time much has been learned about the environmental implications of excessive selenium. This article is a concise review that gives a current perspective on major sources of selenium, impacts and threats to wildlife and humans, ways to assess environmental hazard, and criteria for protecting environmental quality and public health.

MAJOR SOURCES OF SELENIUM CONTAMINATION

Two factors stand apart as the major human-related causes of selenium mobilization and introduction into the environment. First is the procurement, processing, and combustion of fossil fuels. Selenium is an important trace element present in coal, crude oil, oil shale, coal conversion materials (liquefaction oils and synthetic gases), and their waste by-products (Pillay *et al.*, 1969 ; American Petroleum Institute, 1978 ; Fruchter and Petersen, 1979; Schlinger and Richter, 1980; Clark *et al.*, 1980; U. S.EPA, 1980; Cowser and Richmond, 1980; Nystrom and Post, 1982). Rain can leach selenium from coal and oil-shale mining, preparation, and storage sites, where it may enter down-gradient streams and reservoirs through precipitation runoff (Davis and Boegly, 1981; Heaton *et al.*, 1982, Jones, 1990). More importantly, selenium is highly concentrated in the mineral fraction (fly-ash and bottom ash) remaining after coal is burned (Kaakinen *et al.*, 1975 ; Klein *et al.*, 1975). Over 70 million tons of coal fly-ash are produced annually in the U. S. and most of it is disposed by dumping into wet-slurry or dry ash basins (Murtha *et al.*, 1983). Selenium-laden leachate and overflow from these basins often makes its way into rivers, streams, and impoundments. Selenium concentrations can rapidly increase in fish and aquatic organisms in the receiving water, ultimately resulting in tissue damage, reproductive failure, and elimination of entire fish communities (Cumbie and Van Horn, 1978; Garrett and Inmann, 1984; Lemly, 1985a, 1985b; Sorensen, 1986).

The second major factor is the irrigation of seleniferous soils for crop production in arid and semi-arid regions. For example, deposits of Cretaceous marine shales have weathered to produce high selenium soils in many areas of the western U. S. , notably the San Joaquin Valley of California and certain parts of Wyoming, Colorado, Nevada, North and South Dakota, Montana, New Mexico, Arizona, Utah, Nebraska, and Kansas (Kubota, 1980; Tanji *et al.*, 1986; Allen and Wilson, 1990; Presser and Ohlendorf, 1987; Presser *et al.*, 1990; Severson *et al.*, 1991) . These areas usually require substantial irrigation for agricultural crop production. Excessive

amounts of irrigation water are necessary to leach away salts and prevent salinization of soils. This can lead to the production of subsurface drainage that may be highly contaminated with dissolved selenium and other soil trace elements that have been leached from the soil along with salts (Presser and Barnes, 1985; Saiki, 1986a; Summers and Anderson, 1986; Fuji, 1988; Deverel et al., 1989). Selenium in agricultural irrigation drainwater was responsible for massive poisoning of fish and wildlife at Kesterson National Wildlife Refuge, California in the early to mid-1980s (Marshall, 1985; Hoffman et al., 1986; Saiki, 1986a, 1986b; Saiki and Lowe, 1987; Ohlendorf et al., 1986, 1988a, 1988b). Subsequent studies have shown irrigation-related selenium contamination to be a threat to aquatic systems and wildlife refuges in many western states (U. S. Fish and Wildlife Service, 1986; Summers and Anderson, 1986; Ohlendorf et al. , 1987; Sylvester et al. , 1991).

AQUATIC CYCLING AND BIOACCUMULATION

The Selenium Cycle

Four primary pathways exist for selenium in aquatic systems: (i) it can be absorbed or ingested by organisms, (ii) it can bind or complex with particulate matter, (iii) it can remain free in solution, or (iv) it can be released to the atmosphere through volatilization. Over time, most selenium is either taken up by organisms or bound to particulate matter. Through deposition of biologically incorporated selenium and settling of particulate matter (sedimentation), most of it usually accumulates in the top layer of sediment and detritus. However, sediments are only a temporary repository for selenium because there are numerous biological, chemical, and physical processes that can move it out of sediments as well. Aquatic systems are very dynamic and selenium can be cycled from sediments into biota and remain at elevated levels for years after waterborne inputs of selenium are stopped.

Immobilization and Removal 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 absorption (binding and complexation) onto clay and the organic carbon phase of particulates, reaction with iron species, and co-precipitation and settling. 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 detritus and, over time, isolated through the process of sedimentation. In total, immobilization processes effectively remove selenium from the soluble pool, especially in slow moving or still-water habitats and wetlands. Ninety percent 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 selenium

into food chains and thereby cause long-term dietary exposure of fish and wildlife. Selenium is made available for biological uptake by four chemical and/or biological processes. The first is the oxidation and methylation of inorganic and organic selenium by plant roots and microorganisms (oxidation is the conversion of selenium in the reduced organic, elemental, or selenite forms to the selenite or selenate forms; methylation is the conversion of selenium to an organic form containing one or more methyl groups, which usually results in a volatile form). The second process is the biological mixing and associated oxidation of sediments that results from the burrowing of benthic invertebrates and feeding activities of fish and wildlife. The third process consists of physical perturbation and chemical oxidation associated with water circulation and mixing (current, wind, stratification, precipitation, and upwelling). Finally, sediments may be oxidized 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 uptake of selenium by rooted plants and uptake by bottom-dwelling invertebrates and detrital-feeding fish and wildlife. These two pathways may be the most important in the 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 low (Lemly and Smith, 1987) Some of the sedimentary and waterborne selenium may be converted to volatile organic forms (e. g. , dimethyl selenide) by microbial activity in the water and sediments, and subsequently released (degassed) into the atmosphere through the water-air interface or through direct release by plants. Volatilization is an important mechanism by which selenium can be removed from aquatic systems and, thereby, help alleviate the threat of selenium toxicosis to fish and wildlife (Karlson and Frankenberger, 1989, 1990).

Bioaccumulation

The major environmental implications of excessive selenium are associated with its propensity to bioaccumulate in aquatic food chains and, thereby, contaminate the diet of fish and wildlife and, in some cases, humans. Aquatic organisms can accumulate this element to concentrations one or more orders of magnitude greater than the concentrations in their water or food (Fig. 1) . The reason for this bioaccumulation may be that selenium is chemically similar to sulfur, and it sometimes is an essential micronutrient for animals. Over evolutionary time, aquatic animals may have evolved mechanisms to retain and accumulate selenium under conditions of scarcity. Whatever the reason, bioaccumulation has important implications for toxic effects. For example, where fish have experienced chronic toxicity, selenium in the water has been concentrated from 100 to more than 30,000 times, depending on the species and tissue sampled. Selenium accumulation in the organisms eaten by fish and wildlife is usually the major pathway leading to toxicity. Biomagnification of selenium (the accumulation of progressively greater concentrations by successive trophic levels of a food chain) usually ranges from 2 to 6 times between the primary producers (algae and plants) and the lower consumers (invertebrates and forage fish). For example, fish that eat contaminated plankton or benthic invertebrates may accumulate selenium to concentrations that are 4 times those of their food, which in turn, could contain 500 times the selenium concentration in the water. The food chain biomagnification

factor would then be 4 and the total bioconcentration factor for the fish would be 2,000 (i. e. ,4 × 500). These relationships are important in natural systems because they can cause top-level consumers, such as predatory fish, birds, and mammals, to receive toxic selenium levels in the diet even though the concentration in water is low ($<10\mu g$/liter) Moreover, the risk of toxicity through the detrital food pathway will continue despite a loss of selenium from the water column, as long as contaminated sediments are present (Lemly and Smith, 1987).
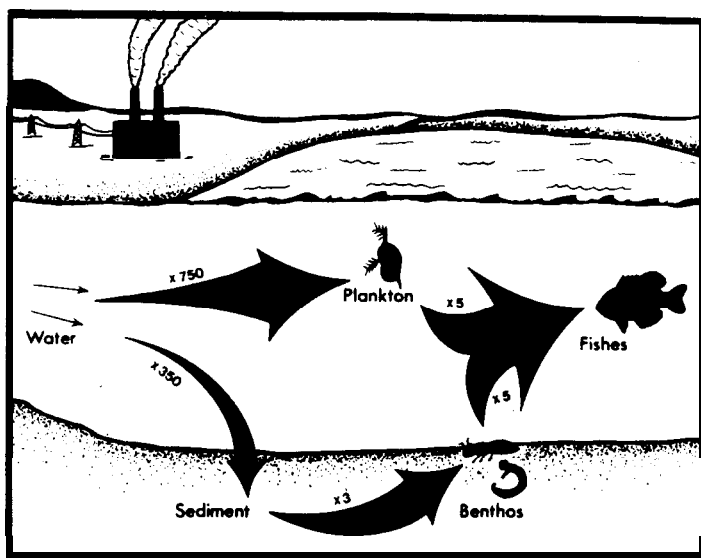

FIG. 1. A typical scenario for bioaccumulation of selenium in aquatic food chains. In this example the bioaccumulation factor for fish relative to water is 3,750 for the planktonic food pathway (750 × 5), and 5,250 for the benthic food pathway (350 × 3 × 5). The food-chain biomagnification factor for the invertebrate-to-fish trophic step is 5 for both pathways.

Role of Habitat Variability

The processes regulating selenium cycling are very similar in all aquatic habitats and wetlands. However, the relative importance of each process may vary from one location to another depending on hydrologic factors that affect food chain bioaccumulation and exposure of wildlife. For example, 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 out of the system. In such waters there is little opportunity for a contaminated surface layer of sediment to develop. Moreover, rooted plants and benthic invertebrates are often scarce in this habitat type. The benthic-detrital component of the system and the associated food pathways thus play a smaller role in the selenium cycle in lotic (flowing) waters than in lentic (slow water) habitats such as reservoirs and wetlands. Systems that tend to accumulate selenium most efficiently are shallow wetlands and marshes,

and reservoirs with low flushing rates. In these systems biological productivity is often high and selenium may be readily trapped through immobilization processes or through direct uptake by organisms. Sediments often build up a high selenium concentration that is remobilized gradually and continually through detrital and planktonic organisms. These habitats are also typically some of the most important feeding and breeding habitats for fish and wildlife, especially waterfowl and shorebirds. Several habitat types often occur together in one aquatic system or wetland complex. For example, rivers may have fast flowing waters, slow moving pools, and shallow backwater marshes all within a few hundred meters of each other. The degree of fish and wildlife exposure to selenium varies among habitats according to the intensity of use, type of use (**e. g .,** feeding vs. resting), and the relative contributions of the various processes that regulate selenium cycling and bioaccumulation in food chains (Lemly and Smith, 1987).

THREATS TO ECOSYSTEMS

Case Example 1. *Toxic Impacts to Fish*

Belews Lake, North Carolina was contaminated by selenium in wastewater released from a coal-fired electric generating facility. From 1974 through 1985, 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 hectare ash basin, where suspended solids were collected by gravitational settling. Runoff water from the coal storage area and power plant site was collected by sump units and also pumped into the ash basin. Selenium-laden (150-$200 \mu g/l$) return flows from the ash basin entered the west side of Belews Lake through an ash sluice water canal.

Selenium bioaccumulated in aquatic food chains and caused severe reproductive failure and teratogenic deformities in fish (Cumbie and Van Horn, 1978; Lemly, 1985a, 1985b, 1993b). Congenital malformations consisted of missing fins, protruding eyes, and grossly deformed spines and heads (Fig. 2). Concentrations of selenium in the lake water averaged only $10 \mu g/l$, but were accumulated from 519 times (periphyton) to 3,975 times (visceral tissue of fish) in the biota. The pattern and degree of accumulation was essentially complete within 2 years after the initial operation of the power plant, and persisted throughout the period of selenium discharge into the lake (1974-1985). Highest concentrations of selenium were found in fish, followed by benthic macroinvertebrates, plankton, and periphyton. The planktonic and detrital food pathways exposed fishes to potential dietary concentrations of selenium that were some 770 and 510-1, 395 times the waterborne exposure, respectively. Of the 20 species of fish originally present in the reservoir, 16 were eliminated (which included all of the important gamefish species) through a combination of dietary toxicity and reproductive failure (Fig. 3). Two species were rendered sterile but persisted as adults and one additional species was eliminated but managed to partially recolonize from a relatively uncontaminated headwater area. Only one of the original resident species, the selenium-tolerant mosquitofish (*Gambusia affinis*) survived, along with two introduced cyprinids. The severe toxic impacts in Belews Lake occurred even though concentrations of waterborne selenium were only 10-20 times those in nearby uncontaminated reservoirs; the flora and fauna contained only about 10-15 times as
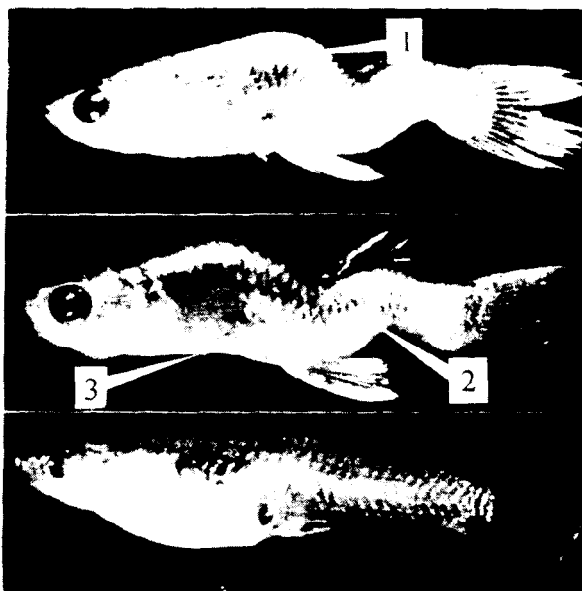
FIG. 2. An example of selenium-induced teratogenic deformities in fish. These are mosquitofish (*Gambusia affinis*) collected from Belews Lake, North Carolina during the period of selenium contamination. The top two individuals are deformed and have severe kyphosis (convex curvature of the thoracic region of the spine, Arrow 1) and lordosis (concave curvature of the lumbar region of the spine, Arrow 2). They also are missing or have vestigial pelvic fins (Arrow 3) The bottom individual is normal.

much selenium.

The findings from Belews Lake serve as a clear illustration of how selenium can rapidly impact fish populations. Moreover, this case example highlights the fact that selenium can accumulate and be biologically magnified to toxic levels even though waterborne concentrations are in the low microgram per liter range. Were it not for bioaccumulation, these waterborne concentrations would pose little threat to aquatic life. Information from the field studies of selenium toxicity in Belews Lake was instrumental in the U. S. Environmental Protection Agency's decision to lower the national water quality criterion for selenium from $35\mu g/l$ to $5\mu g/l$ (U. S. EPA, 1987).

In response to concerns about the fishery problems in Belews Lake the electric utility company changed its ash disposal practices. This involved switching to a dry-ash handling system that disposed the waste in a landfill rather than a wet-basin. By mid 1986, selenium-laden wastewater no longer entered the lake (North Carolina Department of Natural Resources and Community Development, 1986).

Follow-up studies were conducted in 1996 to assess recovery of the ecosystem in Belews Lake (Lemly, 1997a). Selenium concentrations and associated impacts to fish were measured and compared to pre-1986 conditions to determine how much change occurred during the decade since selenium inputs stopped. Findings were also examined using a hazard assessment protocol (Lemly, 1995) to determine if ecosystem-lev-
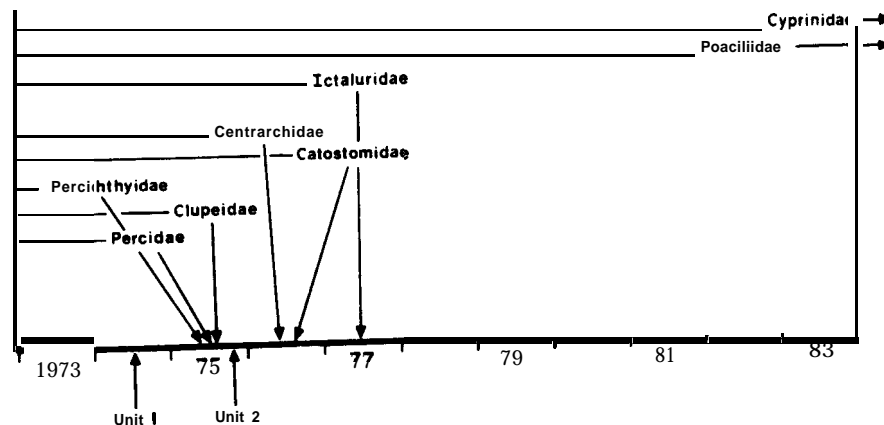
FIG. **3.** The **pattern** of poisoning of fish in Belews Lake, North Carolina due to selenium contamination. Selenium-laden wastewater (150-200µg/l) was discharged into the lake begining in 1974 and within three years most of the fish (16 species) had been eliminated due to dietary toxicity and reproductive failure.

el hazards to fish and aquatic birds had changed as well. Results showed that waterborne selenium fell from a peak of $20\mu g/l$ before 1986, to $<1\mu g/l$ in 1996; concentrations in biota were 85-95 % lower in 1996. Hazard ratings indicated that high hazard existed prior to 1986 and that moderate hazard was still present in 1996, primarily due to selenium in the sediment-detrital food pathway. Concentrations of selenium in sediments fell by about 65-75 % during the period but remained sufficiently elevated ($1-4\mu g/g$) to contaminate benthic food organisms of fish and aquatic birds. Field evidence confirmed the validity of the high hazard ratings. Developmental abnormalities in young fish persisted in 1996. This indicated that selenium-induced teratogenesis and reproductive impairment were still occurring. Moreover, the concentrations of selenium in benthic food organisms were sufficient to cause mortality in young bluegill and other centrarchids because of Winter Stress Syndrome (Lemly, 1993c, 1996a). At the ecosystem level, recovery in Belews Lake has been slow. Toxic effects were still evident ten years after selenium inputs were stopped. The sediment-associated selenium will likely continue to be a significant hazard to fish and aquatic birds for years to come in this aquatic system.

Case *Example* 2. *Toxic Impacts to Wildlife*

In 1985, subsurface irrigation drainage was implicated as the cause of death and deformities in thousands of waterfowl and shorebirds at Kesterson National Wildlife Refuge in California (Ohlendorf *et al.*, 1986). Naturally occurring trace elements and salts 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). One of the trace elements, selenium, bioaccumulated in aquatic food chains and contaminated 500 hectares of shallow marshes. Elevated selenium was found in every animal group coming into contact with these wetlands-from fish and birds to insects, frogs, snakes, and mammals (Saiki and Lowe, 1987; Clark, 1987; Ohlendorf *et al.*, 1988a). Congenital malformations in young waterbirds were

severe, and included missing eyes and feet, protruding brains, and grossly deformed beaks, legs, and wings (Ohlendorf et al., 1986, 1988b; Hoffman et al., 1988). Several species of fish were eliminated due to the combined effects of high salinity, elevated selenium, and other contaminants (Saiki et al., 1992). A high frequency (30%) of stillbirths occurred in the single remaining species, mosquitofish *Gambusia affinis* (Saiki et al., 1991). Laboratory studies conducted by the U. S. Fish and Wildlife Service confirmed the field assessment that selenium in irrigation drainage was the cause of the fish and wildlife problem (Lemly et al., 1993). The "poisoned" refuge became highly publicized and sparked a great deal of political and scientific controversy (Marshall, 1985; Popkin, 1986; Harris, 1991).

The findings at Kesterson National Wildlife Refuge led to a new awareness of the environmental hazards posed by selenium in agricultural irrigation drainage. In 1986, the U.S. Department of the Interior, the Federal steward of more than 400 irrigation-drainage facilities and 200 wildlife refuges in the western states (U. S. Bureau of Reclamation, 1981), established a multi-agency program to investigate irrigation-related drainwater problems. This program conducted screening-level assessments in 13 states, including 20 national wildlife refuges (Table 1). The western San Joaquin Valley and Kesterson National Wildlife Refuge were used as models for identifying and prioritizing potential study areas based on the occurrence of conditions known to contribute to drainwater problems. Samples of water, sediment, and biota (invertebrates, whole-fish, bird liver, bird eggs) were analyzed for a variety of trace elements, heavy metals, and pesticides, and the results were compared to concentrations known to be toxic to fish and wildlife in experimental studies. Geological and hydrological studies were' conducted and, where possible, observations were made to document the occurrence of deformed embryos and hatchlings, which is a biomarker for selenium poisoning in birds (Hoffman and Heinz, 1988).

By 1992 it was known that eleven of the sixteen study areas where biological samples had been taken were seriously contaminated by selenium. The concentrations present at these eleven sites exceeded toxicity thresholds for fish and wildlife (Presser et al., 1994). These study areas are spread across nine states (Fig. 4) Overt selenium toxicosis -i. e., deformities in bird embryos and hatchlings-was found in five states; California, Utah, Wyoming, Nevada, and Montana (Fig. 4, Table 1). In some cases, these teratogenic effects occurred even though the waterborne concentrations of selenium were below those recommended by the U. S EPA for the protection of aquatic life (Lemly et al., 1993).

The biogeochemical conditions leading to the production of subsurface irrigation drainage, culminating in death and deformities in wildlife, have been termed the "Kesterson Effect" (Presser, 1994). The Kesterson Effect is prevalent throughout the western United States and consists of these key conditions: (i) a marine sedimentary basin that contains Cretaceous soils, which usually have relatively high concentrations of selenium; (ii) alkaline, oxidized soils that promote the formation of water-soluble forms of selenium and other trace elements; (iii) a dry climate in which evaporation greatly exceeds precipitation, leading to salt buildup in soils; (iv) subsurface layers of clay that impede downward movement of irrigation water and cause waterlogging of the crop root zone; and (v) subsurface drainage, by natural gradient or buried tile drainage networks, into migratory bird refuges or other wetlands.

TABLE 1

Study Areas and National Wildlife Refuges Investigated in the U.S. Department of the Interior's
Irrigation Drainage Program (Lemly *et* al. , 1993)

State and Study Area	National Wildife Refuge (NWR)
-Oregon	
Malheur[b]	Malheur NWR
Oregon/California	
Klamath Basin	Lower Klamath NWR
California	
Sacramento Complex	Sacramento NWR
	Delevan NWR
	Colusa NWR
	Sutter NWR
Tulare Lake Bed'	Kern NWR
	Pixley NWR
	Tule Lake NWR
Salton Sea[b]	Salton Sea NWR
California/Arizona	
Lower Colorado River	Havasu NWR
	Cibola NWR
	Imperial NWR
Nevada	
Stillwater'	Stillwater NWR
Utah	
Middle Green River'	Ouray NWR
Montana	
Sun River'	Benton Lake NWR
Milk River Basin	—
Colorado	
Gunnison River Basin[b]	—
Pine River	—
Wyoming	
Kendrick Project'	Bowdoin NWR
Riverton Project[b]	—
South Dakota	
Belle Fourche Project[b]	—
Angostura Project	—
Kansas	
Middle Arkansas River[b]	—
Texas	
Lower Rio Grande Valley	Laguna Atascosa NWR
New Mexico	
Middle Rio Grande Valley	Bosque del Apache NWR
Idaho	
American Falls Reservoir	Minidaka NWR

[a] Study areas where overt symptoms of selenium toxicosis (deformities) were found in young migratory birds.
[b] Study areas where toxicity is predicted based on concentrations of selenium found in fish and bird tissues.
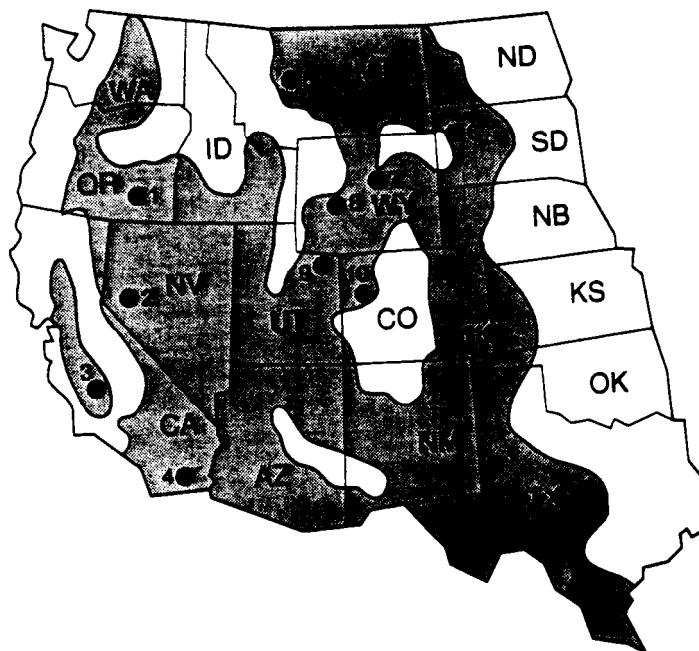
FIG. 4. Locations in the western United States where selenium in subsurface agricultural irrigation drainage has poisoned fish and wildlife. 1 = Malheur National Wildlife Refuge (NWR); 2 = Stillwater NWR; 3 = Tulare Lake Bed Area: 4 = Salton Sea Area; 5 = Benton Lake NWR; 6 = Belle Fourche Reclamation Project; 7 = Bowdoin NWR; 8 = Riverton Reclamation Project; 9 = Ouray NWR; 10 = Gunnison River Basin: 11 = Middle Arkansas River. Teratogenic deformities associated with selenium bioaccu-. mulation in young birds were found at locations 2, 3. 5. 7, and 9.

The field studies conducted by U. S. Department of the Interior indicate that the toxic threat of selenium-laden irrigation drainage to wetlands, fish, and wildlife is not restricted to Kesterson National Wildlife Refuge, the San Joaquin Valley, or the State of California. Contamination has proven to be pervasive throughout the western states (Fig. 4) and threatens waterfowl populations in the Central and Pacific flyways (Presser *et al.* , 1994; Skorupa *et al.,* in press). Managers of wetlands in the western U. S. and elsewhere must recognize selenium-laden irrigation drainage as a threat with the potential to affect wildlife populations on an international scale.

THREATS TO HUMAN HEALTH

Excessive selenium in the environment can threaten public health in the same way it threatens fish and wildlife, i.e. , by accumulating in potential food items. In some locations soils that are naturally high in selenium may cause selenium concentrations in agricultural crops to be excessive, leading to chronic poisoning in humans

(Yang *et al.*, 1983). For situations in which human activities have led to contaminated aquatic systems, public health risks are associated with consumption of selenium-contaminated fish and wildlife. The U. S. National Academy of Sciences and the U.S. Environmental Protection Agency recommend that a daily intake of $50\text{-}200\mu g$ selenium should be adequate for nutritional requirements in adults (National Research 'Council, 1980b; U.S. EPA, 1984). The dose necessary to cause chronic selenosis in humans is not well defined but the threshold for toxicity appears to lie somewhere in the range of $850\text{-}900\mu g$ per day (Yang *et al.*, 1989a, 1989b; Longnecker *et al.*, 1991). Thus, the margin of safety for selenium, i. e., the difference between the dietary levels that are beneficial and those that are possibly harmful, is small-a factor ranging from about 5 to 18.

In Case Example 2, public health agencies recognized the possibility that selenium poisoning could occur in humans that ate fish and wild game taken from contaminated sites. Federal land management authorities erected warning signs at these locations to alert the public to the health risks associated with selenium contamination and guard against consumption of contaminated food (Fig. 5). Dietary guidelines formulated by the California Department of Health stipulated that adults should eat no more than one meal (4 ounces) of fish or wild game per 2 weeks (Moore *et al.*, 1990). Children less than 15 years of age and women of childbearing age were advised not to eat any fish or wild game due to concerns about potential reproductive and developmental effects. Advisories were issued when the selenium concentrations reached or exceeded 2 $\mu g/g$ wet weight (Fan *et al.*, 1988). The rationale for this threshold concentration was as follows: By simple calculation, fish or game containing 1 $\mu g/g$ selenium contributes 113 μg per 4 oz serving. At 2 $\mu g/g$ the single serving would contribute 226 μg, which exceeds the upper limit of the recommended daily intake

FIG. 5. An example of the signs used to alert the public of the health risks from selenium contamination at Federally managed wetlands in the western United States.

(200 μg) This exceedance would be due to a single meal, not including other daily intake. Therefore, a concentration of 2 μg/g was designated as the action level for issuing consumption advisories. A complete ban on consumption was issued and posted at sites where concentrations of selenium in fish or bird tissues exceeded 5 μg/g wet weight (a concentration of 5 μg/g would contribute 565 μg per serving, which is in excess of the 500 μg/day chronic toxicity threshold suggested by Levander, 1987).

The risks to public health in Case Example 2 prompted the first widely publicized consumption advisories and bans due to excessive environmental selenium in the United States. These advisories were issued in 1984-85 (Fan et al., 1988). Since that time, several other advisories have been issued for selenium contaminated reservoirs and wetlands. For example, the fishery of Belews Lake (Case Example 1) began to recover after 1986 as selenium levels fell, but the concentrations in fish tissues were still great enough (3-8μg/g) to pose a threat to human health. This threat was recognized and advisories were issued begining in 1989 (Lemly, 1997a). The stipulations of those advisories paralleled the ones given above. Warnings have also been posted at several wildlife refuges contaminated by seleniferous irrigation drainage in the western U. S (e.g., Stephens et al., 1992; Butler et al., 1994). It is now widely recognized in the U. S. that excessive selenium in the environment can, and does, pose a threat to human health as well as fish and wildife. In regard to the two case examples discussed in this paper, timely intervention by public health agencies prevented what could have otherwise developed into episodes of human poisoning. Other countries would be well advised to take note of these examples.

HAZARD ASSESSMENT AND ENVIRONMENTAL QUALITY CRITERIA

It is important to monitor and evaluate environmental conditions as a way of predicting, detecting, and, hopefully, avoiding potential selenium problems before they occur. A hazard assessment protocol (Protocol) for selenium has recently been developed and is now available for use in this regard (Lemly, 1995, 1997b). The Protocol characterizes hazard in terms of the potential for food-chain bioaccumulation and *re*productive impairment in fish and aquatic birds, which are the most sensitive biological responses for estimating ecosystem-level impacts of selenium contamination. Five degrees of hazard are possible depending on where the highest concentrations of selenium measured in environmental samples fall on the corresponding hazard profile given in the Protocol (Fig. 6). The degree of hazard is given a numerical score: 5 = high hazard, 4 = moderate hazard, 3 = low hazard, 2 = minimal hazard, and 1 = no identifiable hazard. A separate hazard score is given to each of five ecosystem components; water, sediments, benthic macroinvertebrates, fish eggs, and aquatic bird eggs. A final hazard characterization is determined by adding individual scores and comparing the total to the following evaluation criteria: 5 = no hazard, 6-8 = minimal hazard, 9-11 = low hazard, 12-15 = moderate hazard, 16-25 = high hazard (Fig. 6, Table 2 ; Lemly, 1995, 1997b).

Selenium hazards to fish can vary seasonally because of a condition known as Winter Stress Syndrome (WSS; Lemly, 1993c,1996a,1997c). WSS is severe lipid depletion brought on by external stressors in combination with normal reductions in feeding and activity during cold weather. Fish can develop this syndrome in response to chemical stressors such as water pollutants, or biological stressors such as parasites. Substantial mortality can result, potentially changing year-class strength and

population structure of the affected species, and altering community-level ecological interactions. Selenium hazards should be evaluated in the context of seasonal metabolic changes that normally occur in test organisms. WSS could be an important cause of mortality in many circumstances. Wastewater discharges containing selenium may pose a greater toxic threat to fish during winter than at other times of the year (Lemly, 1997d). A comprehensive protocol for aquatic hazard assessment should include testing for WSS. Because of the possibility of WSS, conservative estimates of hazard are necessary and the role of season in aquatic hazard assessment must be accounted for.

TABLE 2

Example Data Sets for Aquatic Hazard Assessment of Selenium Following the Protocol Method (Lemly, 1995)

Site and Environmental Component	Selenium Concentration"	Evaluation by Component		Totals for the Site	
		Hazard[b]	Score	Score	Hazard
Wetland X					
Water	< 1-3	Low	3		
Sediments	0.7-1	None	1		
Invertebrates	1-3	Minimal	2		
Fish eggs	2-4	Minimal	2		
Bird eggs	2-7	Low	3		
			11	11	Low
Reservoir X					
Water	9-93	High	5		
Sediments	7-41	High	5		
Invertebrates	12-72	High	5		
Fish eggs	75-120	High	5		
Bird eggs	12-120	High	5		
			25	25	High
River X					
Water	3-4	Moderate	4		
Sediments	0.6-3	Low	3		
Invertebrates	3-33	High	5		
Fish eggs	8-27	High	5		
Bird eggs	1-17	Moderate	4		
			21	21	High

"Selenium concentrations in $\mu g/l$ (parts per billion) for water; $\mu g/g$ (parts per million) dry weight for sediments, invertebrates, and eggs.
[b] Hazard ratings were determined by comparing selenium concentrations to hazard profiles given in Fig. 6.

In addition to the Protocol, which predicts environmental hazard, there are also recent guidelines for evaluating the toxicological significance of selenium residues in

aquatic organisms and recommendations for water quality criteria to protect fish and wildlife (Lemly, 1993a, 1996b). Diagnostic selenium concentrations are available for water, food-chain, predatory fish (consuming fish or invertebrate prey), and aquatic birds. Waterborne selenium concentrations of 2 $\mu g/l$ or greater (parts per billion; total recoverable basis in 0.45μ filtered samples) should be considered hazardous to the health and long-term survival of fish and wildlife populations due to the high potential for food-chain bioaccumulation, dietary toxicity, and reproductive effects. In some cases, ultratrace amounts of dissolved and particulate organic selenium may lead to bioaccumulation and toxicity even when total waterborne concentrations are less than $1\mu g/l$.

Food-chain organisms such as zooplankton, benthic invertebrates, and certain forage fishes can accumulate up to 30 $\mu g/g$ dry weight selenium (some taxa up to 370 $\mu g/g$) with no apparent effect on survival or reproduction. However, the dietary toxicity threshold for fish and wildlife is only 3 $\mu g/g$; these food organisms would supply a toxic dose of selenium while being unaffected themselves. Because of this, food-chain organisms containing 3 $\mu g/g$ (parts per million) dry weight or more should be viewed as potentially lethal to fish and aquatic birds that consume them.

Biological effects thresholds (dry weight) for the health and reproductive success of freshwater and anadromous fish are: whole-body $= 4\mu g/g$; skeletal muscle $= 8\mu g/g$; liver $= 12\mu g/g$; ovaries and eggs $= 10\mu g/g$. Effects thresholds for aquatic birds are: liver $= 10 \mu g/g$; eggs $= 3\mu g/g$. The most precise way to evaluate potential reproductive impacts to adult fish and aquatic bird populations is to measure selenium concentrations in gravid ovaries and eggs. This single measure integrates waterborne and dietary exposure, and allows an evaluation based on the most sensitive biological endpoint. If natural resource managers obtain measurements of selenium in water, food-chain organisms, and fish and wildlife tissues, and apply the preceeding guidelines and assessment protocol, they can accurately determine the overall selenium status and health of aquatic ecosystems.

CONCLUSIONS

There have been several cases of selenium contamination in the United States that have resulted in severe poisoning of fish and wildlife. Research studies of these episodes have generated a data base that clearly illustrates the environmental implications of excessive selenium and the associated threats to public health. Moreover, the data base has been, and continues to be, used to benefit future generations of wildlife and humans by providing guidance on what the acceptable and unacceptable concentrations of selenium are from waterborne and dietary routes of exposure. It is hoped that this information will be useful for developing countries and nations around the world because they too may soon have to deal with many of the same problems. The two faces of selenium-required nutrient and potent toxin-make it a paricularly important trace element in the health of both animals and man. Because of this paradox, environmental selenium in relation to agriculture, fisheries, and wildlife will continue to raise important land and water management issues for decades to come. If these issues are dealt with in a timely, prudent manner guided by the environmental selenium data base, adverse impacts to natural resources and public health can be avoided.

Hazard from accumulation in planktonic food-chain and dietary toxicity to fish and aquatic birds

Selenium in Water (μg/l)
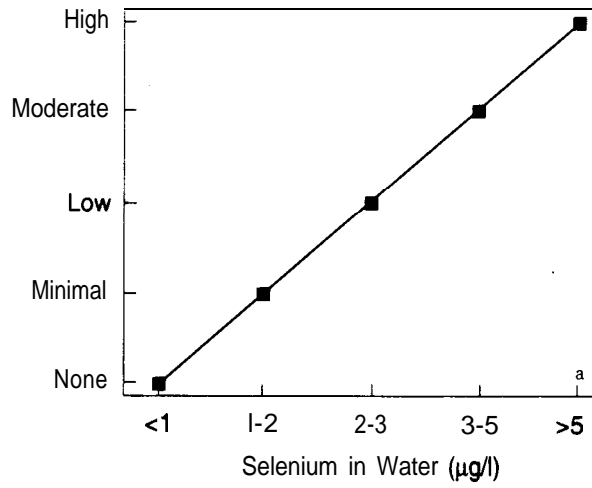
Hazard from accumulation in benthic food-chain and dietary toxicity to fish and aquatic birds

Selenium in sediments (μg/g dry weight)
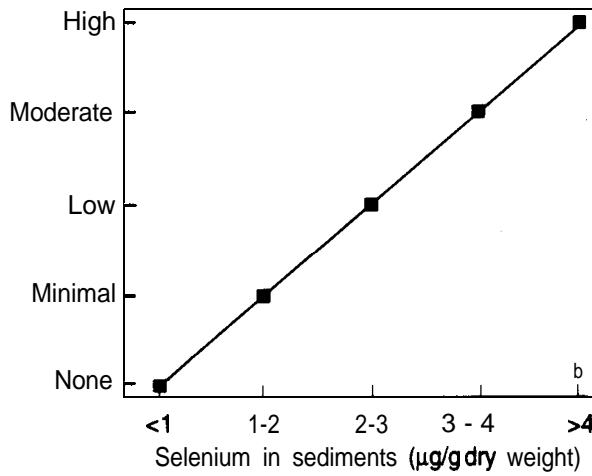
Hazard from dietary toxicity and reproductive impairment in fish and aquatic birds

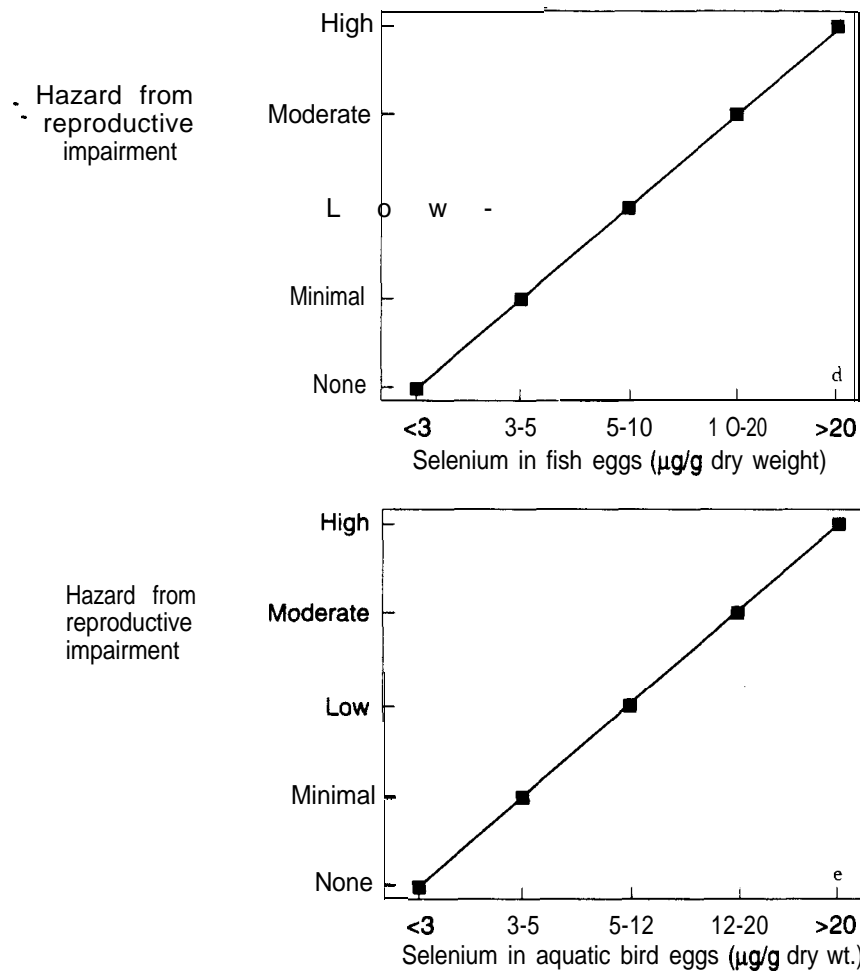Selenium in macroinvertebrates (μg/g dry wt.)
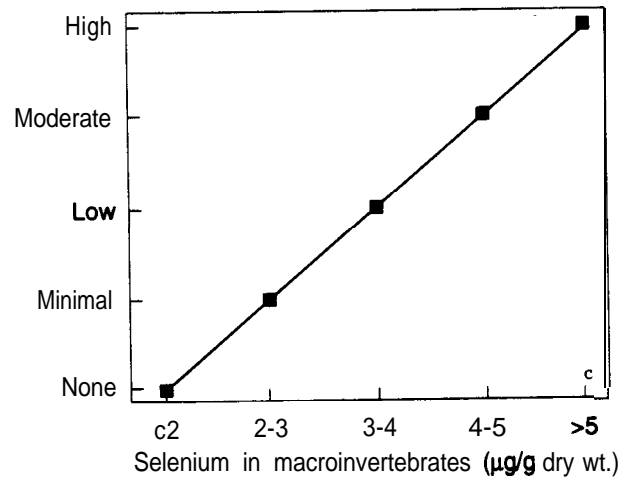
FIG. 6. Guidelines for rating hazard and predicting the toxic threat of selenium to fish and aquatic birds based on selenium concentrations. Numerical scores are assigned to each hazard rating and added to derive a final hazard estimate for the site (see text for explanation) .

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