



Human and Ecological Risk Assessment: An International Journal

ISSN: 1080-7039 (Print) 1549-7860 (Online) Journal homepage: <http://www.tandfonline.com/loi/bher20>

Selenium Impacts on Fish: An Insidious Time Bomb

A. Dennis Lemly

To cite this article: A. Dennis Lemly (1999) Selenium Impacts on Fish: An Insidious Time Bomb, Human and Ecological Risk Assessment: An International Journal, 5:6, 1139-1151, DOI: 10.1080/10807039.1999.10518883

To link to this article: <https://doi.org/10.1080/10807039.1999.10518883>



Published online: 09 Oct 2012.



Submit your article to this journal [↗](#)



Article views: 76



View related articles [↗](#)



Citing articles: 30 View citing articles [↗](#)

Selenium Impacts on Fish: An Insidious Time Bomb

A. Dennis Lemly

United States Forest Service, Blacksburg, VA 24061–0321*

ABSTRACT

A selenium time bomb situation is developing in the United States and elsewhere that may result in substantial impacts on fish populations. The selenium time bomb has three components: (1) high food-chain bioaccumulation, (2) steep toxic response curve for fish, and (3) insidious mode of toxicity. If the threshold for selenium toxicity is exceeded, the time bomb explodes and a cascade of events is set into motion that will result in major ecosystem disruption. Several human-related factors are emerging that are capable of igniting the fuse of the time bomb by increasing waterborne concentrations of selenium and providing conditions favorable for bioaccumulation. Some of these factors are (1) mobilization of selenium due to open-pit phosphate mining, (2) use of constructed wetlands to treat selenium-laden wastewater from oil refineries and agricultural irrigation, (3) landfill disposal of seleniferous fly ash from coal-fired power plants, and (4) mobilization of selenium from animal feedlot wastes. Collectively, these threats may be sufficient to cause widespread, unanticipated toxic effects in fish populations. Only environmentally sound risk assessments followed by prudent management actions can defuse the selenium time bomb — once it explodes, it is too late to avoid significant impacts.

Key Words: coal-fired power plants, fly-ash landfills, phosphate mining, livestock feedlot wastes, wastewater treatment wetlands, aquatic pollution

* United States Forest Service, Southern Research Station, Coldwater Fisheries Research Unit, Department of Fisheries and Wildlife Sciences, Virginia Tech University, Blacksburg, VA 24061–0321; Tel: (540) 231–6663; Fax: (540) 231–7580; E-mail: dlemly@vt.edu

INTRODUCTION

Because of an escalation in human activities that promote the introduction and bioaccumulation of selenium in aquatic ecosystems, coupled with the insidious, almost stealthy nature of its toxic effects on fish, I regard selenium as a time bomb.

The potential for selenium to rapidly and severely affect fish populations has been recognized for over 2 decades (*e.g.*, Cumbie and Van Horn, 1978). However, selenium poisoning 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, 1985a; Coyle *et al.*, 1993). Consequently, fish populations can decline or even disappear over the course of a few years for no apparent reason — unless one is cognizant of the subtle way in which selenium operates.

An important factor in the time bomb scenario 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 over a range of only a few $\mu\text{g/L}$ (parts per billion) waterborne selenium (Figure 1). Thus, even slight increases can light the bioaccumulation fuse of the selenium time bomb and push it over the toxic threshold. There are several case histories that illustrate the propensity for selenium to bioaccumulate and poison fish. I discuss two of those cases in this paper to support the assertion that selenium is not “just another contaminant”. One case involved a long-term input of selenium (>10 years), but the other involved a much shorter input (8 months). Although there is a stark difference in the period of exposure, similarities in the magnitude and persistence of impacts on fish suggest that if the threshold for selenium toxicity is exceeded, an inevitable cascade of events is set into motion that will result in major ecosystem disruption. Once the time bomb explodes, it is too late to avoid significant impacts.

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. These were (1) procurement, processing, and combustion of fossil fuels, and (2) irrigation of seleniferous soils for crop production in arid and semi-arid regions (Lemly, 1985b; Lemly, Finger, and Nelson, 1993). During the 1990s, other issues have emerged as potentially important factors in the mobilization and bioaccumulation of hazardous concentrations of selenium, including (1) phosphate mine 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. In this paper I examine these issues in the context of new, largely uninvestigated selenium threats that may be sufficient to cause

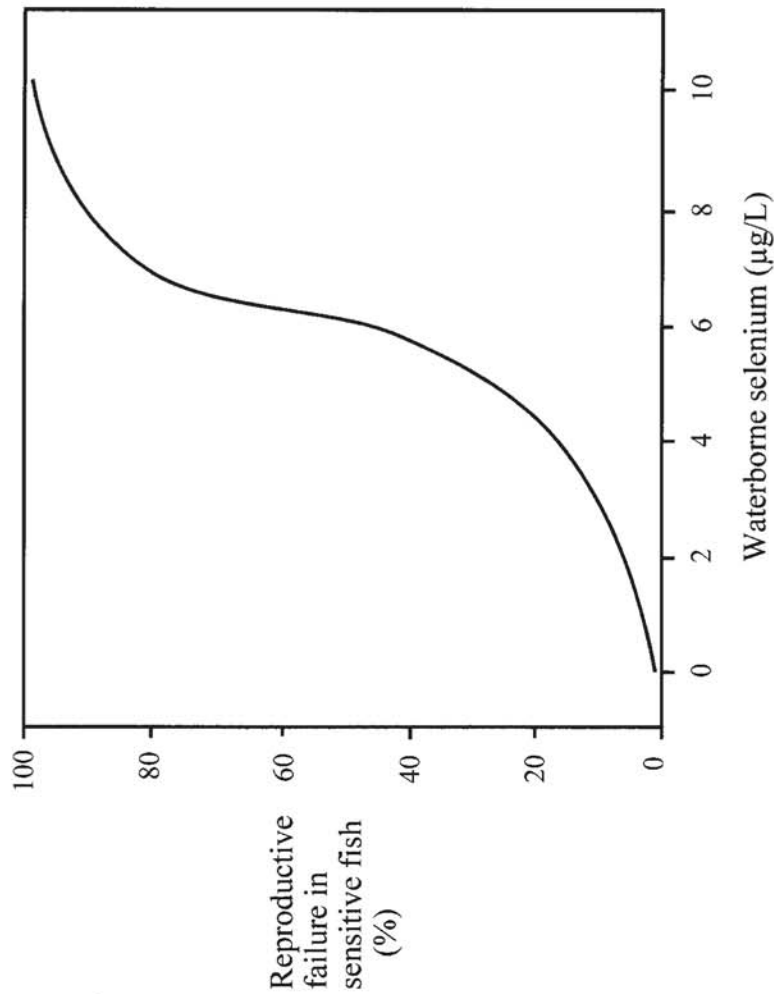


Figure 1. Relationship between the concentration of selenium in habitats favorable for bioaccumulation (e.g., lakes, wetlands) and the degree of reproductive failure in sensitive fish species (e.g., bluegill, *Lepomis macrochirus*). A small increase in waterborne selenium can result in catastrophic impacts on reproductive success. Recent escalation in human activities that promote the mobilization of selenium into aquatic ecosystems threatens to activate this time bomb on a widespread scale in the United States and elsewhere.

widespread, unforeseen effects in fish populations. Each of these threats adds credence to the time bomb aspect of selenium in the environment.

INSIDIOUS TOXIC EFFECTS

Case Example 1. Belews Lake, North Carolina

The episode of contamination at Belews Lake is the most extensively documented case of selenium poisoning in freshwater fish. It serves as an excellent example of the insidious nature of selenium toxicity, brought about by food-chain bioaccumulation and reproductive failure.

Belews Lake 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 to 200 µg Se/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, 1993a). Congenital malformations consisted of missing fins, protruding eyes, and grossly deformed spines and heads (Figure 2). Concentrations of selenium in the lake water averaged 10 µg/L (uncontaminated reference locations had selenium concentrations <1 µg/L), but were accumulated from 519 times (periphyton) to 3975 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 to 1985). Highest concentrations of selenium were found in fish, followed by benthic macroinvertebrates, plankton, and periphyton. The planktonic and detrital food pathways exposed fish to potential 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 the reservoir, 18 were effectively rendered sterile because of reproductive failure. Some persisted as adults for a few years, but eventually all 18 were eliminated. Only one of the original resident species, the selenium-tolerant mosquitofish (*Gambusia affinis* Baird and Girard) survived, along with two introduced cyprinids. The fishery was decimated without massive fish kills because of the subtle, yet lethal mechanism by which selenium impacts can occur. 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 about 10 to 50 times as much selenium.

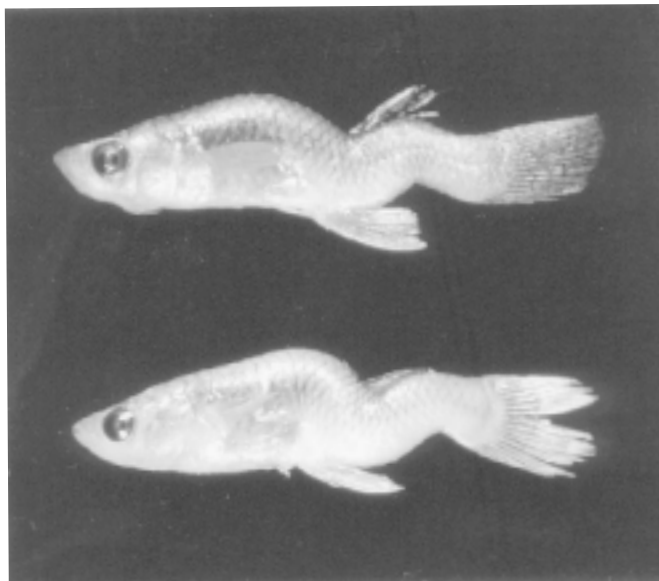


Figure 2. An example of the insidious toxic effects of selenium. These are young mosquitofish (*Gambusia affinis*). They have severe, compound curvature of the spine (kyphosis and lordosis) due to teratogenic effects of selenium that occurred shortly after hatching. Because selenium poisoning can kill early life-stages and reduce reproductive success while leaving adults unharmed, fish populations may disappear gradually without evidence of fish kills or other outward symptoms of trouble. This stealthy mode of action is one characteristic that tends to make selenium a time bomb.

The findings from Belews Lake serve as a clear illustration of how selenium can rapidly, yet insidiously, 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 5 to 10 $\mu\text{g/L}$ range. Without bioaccumulation, these waterborne concentrations would pose relatively 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 U.S. national water quality criterion for selenium from 35 $\mu\text{g/L}$ to 5 $\mu\text{g/L}$ (USEPA, 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 (NCDNRCD, 1986), and in subsequent years a stocking program was successful in reestablishing populations of sport fish (*e.g.*, centrarchids such as

largemouth bass *Micropterus salmoides* Lacepede, and bluegill *Lepomis macrochirus* Rafinesque). 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-level hazards to fish and aquatic birds had changed as well. Results showed that waterborne selenium fell from a peak of 20 µg/L before 1986, to <1 µg/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 to 75% during the period but remained sufficiently elevated (1 to 4 µg/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, indicating 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, 1993b, 1996).

At the ecosystem level, recovery in Belews Lake was very slow. Toxic effects were still evident ten years after inputs of selenium stopped. Projections indicated that several decades could be necessary for the ecosystem to fully recover (Lemly, 1997a). These latent effects illustrate another aspect of the insidious nature of selenium toxicity. Sediment-associated selenium can be mobilized through the food chain gradually, yet continually, and poison subsequent generations of fish despite the fact that adult populations have been reestablished and some reproduction is occurring. Developmental abnormalities in young fish can reveal the impact of latent selenium toxicity on population status (Lemly, 1997b).

Case Example 2. Martin Reservoir, Texas

In contrast to Belews Lake, which received selenium-laden wastewater for over 10 years, the period of selenium input at Martin Reservoir was only 8 months (September 1978 to May 1979). Aqueous discharges from two fly-ash disposal basins at a coal-fired power plant elevated waterborne selenium to an average of 2.6 µg/L (range = 1 to 34 µg/L), with about 5 µg/L present in the primary impact area. That amount was sufficient to cause bioaccumulation in aquatic food chains and substantially elevate tissue concentrations of selenium in fish (bioaccumulation factor for fish = 1190 to 2504 times the water concentration). Within 2 years, the biomass of planktivorous fish declined by over 90%, and a monitoring study conducted by the Texas Parks and Wildlife Department found that major reproductive failure was occurring in other species (*e.g.*, largemouth bass, bluegill; Garrett and Inman, 1984).

Physiological effects of selenium on fish in Martin Reservoir were severe. For example, green sunfish (*Lepomis cyanellus* Rafinesque) and redear sunfish (*Lepomis microlophus* Gunther) were observed to have life-threatening damage to their gills, hearts, livers, and kidneys (Sorensen *et al.*, 1982). Major changes were evident in blood parameters, including the presence of deformed red blood cells and depressed hemoglobin levels. Some individuals had distended abdomens due to accumulated fluid in the body cavity, and all females examined had degenerated ovarian follicles. Elevated tissue levels of selenium and reproductive impairment of fish (ovarian damage) were still evident over 7 years following the discharges (Sorensen, 1986).

Despite the relatively short period of selenium input, large-scale ecosystem damage occurred in Martin Lake. This is a good illustration of what can happen once the fuse of the selenium time bomb has been ignited. Even a brief contamination event can set an inevitable cascade of events into motion — bioaccumulation, tissue damage and reproductive failure in fish, sediment-detrital remobilization — resulting in dramatic impacts that persist for many years. The example of Martin Reservoir underscores the need to closely evaluate human activities that are emerging as potentially important factors in the mobilization and bioaccumulation of hazardous concentrations of selenium in aquatic ecosystems (see Escalating Threats, below). Events that increase waterborne selenium by even a few $\mu\text{g}/\text{L}$ can rapidly push aquatic ecosystems over the threshold and explode the time bomb.

ESCALATING THREATS OF CONTAMINATION

There are at least four emerging issues that add substantially to the time bomb aspect of selenium in the United States and elsewhere: (1) mobilization of selenium due to open-pit phosphate mining, (2) use of constructed wetlands to treat selenium-laden wastewater from oil refineries and agricultural irrigation, (3) sub-surface disposal of seleniferous fly ash from coal-fired power plants, and (4) mobilization of selenium from animal feedlot wastes.

Open-Pit Phosphate Mining

In the U.S., 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 U.S. The first 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 either 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 U.S. Forest Service and administered by the U.S. 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/g, parts per million), seepage water (up to 1500 µg/L), tailings ponds (up to 1500 µg/L), and streams near the waste dumps (up to 474 µg/L; Herring *et al.*, 1999; Desborough *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 bouveri* Richardson). A preliminary hazard assessment of selenium in the watershed indicated that waterborne selenium concentrations in the Blackfoot River and 14 of its tributaries met or exceeded toxic thresholds for fish. Concentrations of selenium in fish tissues exceeded toxic thresholds for reproduction at one location and approached the threshold at two 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. Continued expansion of phosphate mining is anticipated, and large mineral leases are awaiting development both on and off national forest lands. Phosphate mining has the potential to elevate selenium levels in aquatic ecosystems across extensive areas of the intermountain West. The fuse of the selenium time bomb may already have been ignited 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 advo-

cated for removing 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 increases the likelihood that they will 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 hazards that are just as serious as the ones they were constructed to solve in the first place. That result did take place at the Chevron USA Oil Refinery in Richmond, California, in the mid-1990s. The constructed wetland was effective in removing selenium from the wastewater stream. However, the habitat feature it provided attracted large numbers of migratory waterfowl and shorebirds, some of which were poisoned by selenium. That finding prompted the implementation of a remedial management plan which ended its use as a treatment wetland for selenium removal (Skorupa, 1998). Thus, the wastewater treatment goals were not attainable within the context of environmental compatibility.

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 effort to document or reveal ecological hazards (*e.g.*, Hansen *et al.*, 1998; Terry and Zayed, 1998). Consequently, the methods have inherent dangers that are not readily apparent to potential users. These oversights are a major shortcoming that is pervasive in the wetland treatment technology field. Many wetland treatment methods are being marketed on a national scale without full knowledge or disclosure of the risks they pose to fish and wildlife. Constructing a wetland to treat selenium-laden wastewater may well activate the selenium time bomb 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 to 300 $\mu\text{g Se/g}$) and other combustion wastes are generated in the process. The current annual production of coal ash in the U.S. is about 120 million tons and is projected to steadily increase in the next century (USEPA, 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. The problem with this disposal method is that over time landfills can become unstable and either the surface clay cap or the underlayment clay develops cracks, rainwater or groundwater infiltrates, and leaching of selenium begins to occur. If that happens, selenium-laden seepage (50 to 200 $\mu\text{g Se/L}$) can 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, Burnet, and Harnby, 1983).

One example of this problem occurred in 1991 in eastern Pennsylvania, where plans were being made to construct a 65 ha landfill to dispose fly-ash from five 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* Mitchell), which is a highly valued sport fish in the region. This prompted an investigation by the Pennsylvania Department of Environmental Resources (DER) into the leaching behavior of selenium in fly ash. Evidence from the initial investigation, which found that selenium leaching was a legitimate concern, led DER to revoke the disposal permit for two of the five facilities identified in the original application (Pennsylvania Bulletin, 1992), and called 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 repurposed to handle only construction and demolition waste — no fly-ash was allowed. The events in this case example represent major adjustments to Pennsylvania laws brought about by environmental concerns over selenium in fly-ash landfills. Moreover, the actions taken by DER set an important precedent by establishing very rigid requirements for landfills that are proposed in the future.

As coal-fired power production continues in the next century, the number of fly-ash landfills will steadily increase — so will 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 landfill is a potential selenium time bomb to down-gradient aquatic habitats, and every new landfill increases the extent of the threat on a regional and national scale.

Feedlot Wastes

Selenium is widely used as a nutritional supplement to livestock diets, and it is not uncommon for the liquid manure associated with swine or cattle feedlots to contain 50 to 150 $\mu\text{g Se/L}$ (Oldfield, 1998). Because excreted selenium has been physiologically processed by the animal, the chemical forms present in this matrix likely consist of various organic metabolites or

other organic compounds. Importantly, organic selenium has a much greater bioaccumulation potential in aquatic ecosystems than inorganic selenium. 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, Canfield, and La Point, 1993). This magnitude of bioaccumulation means that a waterborne concentration of only 0.1 $\mu\text{g/L}$ 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 (5 to 15 $\mu\text{g/g}$).

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 are known as "hog factories" or "confinement facilities", each with 5000 to 20,000 animals. 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), and likely contributed to outbreaks of a toxic dinoflagellate (*Pfiesteria piscidia*) in the Pamlico River Estuary (Glasgow *et al.*, 1995).

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 U.S. Although there is a pressing need to evaluate this threat, there has been no monitoring for acute or residual selenium contamination of aquatic life in any of the pollution events associated with feedlot operations, and there is no monitoring of selenium movement off fields sprayed with excessive amounts of liquid manure. Concerns for aquatic impacts of livestock wastes are focused on nutrient enrichment, bacterial contamination, and depression of dissolved oxygen — there is no recognition of selenium issues.

CONCLUSIONS

The threat of widespread impacts from the selenium time bomb 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 by far the most serious threat

to fishery resources. The fuse of the time bomb may already have been ignited at some locations. Only environmentally sound risk assessments followed by prudent management actions can prevent the selenium time bomb from exploding.

REFERENCES

- Besser, J. M., Canfield, T. J., and La Point, T. W. 1993. Bioaccumulation of organic and inorganic selenium in a laboratory food chain. *Environ. Toxicol. Chem.* 12, 57–72.
- Commonwealth of Pennsylvania. 1991. *Permit for Solid Waste Disposal and/or Processing Facility — No. 301106*. Pennsylvania Department of Environmental Resources, Harrisburg, PA, USA.
- Coyle, J. J., Buckler, D. R., Ingersoll, C. G., Fairchild, J. F., and May, T. W. 1993. Effects of dietary selenium on the reproductive success of bluegills (*Lepomis macrochirus*). *Environ. Toxicol. Chem.* 12, 551–565.
- Cumby, P. M. and Van Horn, S. L. 1978. Selenium accumulation associated with fish mortality and reproductive failure. *Proc. Annual Conf. Southeast. Assoc. Fish Wildlife Agencies* 32, 612–624.
- Desborough, G., DeWitt, E., Jones, J., Meier, A., and Meeker, G. 1999. Preliminary mineralogical and chemical studies related to the potential mobility of selenium and associated elements in Phosphoria Formation strata, southeastern Idaho. *Open-File Report 99–129*. U.S. Geological Survey, Denver, CO, USA.
- Garrett, G. P. and C. R. Inman. 1984. Selenium-induced changes in fish populations of a heated reservoir. *Proc. Annual Conf. Southeast. Assoc. Fish Wildlife Agencies* 38, 291–301.
- Glasgow, H. B., Jr., Burkholder, J. M., Schmechel, D. E., Tester, P. A., and Rublee, P. A. 1995. Insidious effects of a toxic estuarine dinoflagellate on fish survival and human health. *J. Toxicol. Environ. Health* 46, 501–522.
- Hansen, D., Duda, P. J., Zayed, A., and Terry, N. 1998. Selenium removal by constructed wetlands: Role of biological volatilization. *Environ. Sci. Technol.* 32, 591–597.
- Herring, J. R., Desborough, G. A., Wilson, S. A., Tysdal, R. G., Grauch, R. I., and Gunter, M. E. 1999. Chemical composition of weathered and unweathered strata of the Meade Peak Phosphatic Shale Member of the Permian Phosphoria Formation. A. Measured sections A and B, central part of Rasmussen Ridge, Caribou County, Idaho. *Open-File Report 99–147–A*. U.S. Geological Survey, Denver, CO, USA.
- Lemly, A. D. 1985a. Toxicology of selenium in a freshwater reservoir: Implications for environmental hazard evaluation and safety. *Ecotoxicol. Environ. Safety* 10, 314–338.
- Lemly, A. D. 1985b. Ecological basis for regulating aquatic emissions from the power industry: The case with selenium. *Reg. Toxicol. Pharmacol.* 5, 465–486.
- Lemly, A. D. 1993a. Teratogenic effects of selenium in natural populations of freshwater fish. *Ecotoxicol. Environ. Safety* 26, 181–204.
- Lemly, A. D. 1993b. Metabolic stress during winter increases the toxicity of selenium to fish. *Aquatic Toxicol.* 27, 133–158.
- Lemly, A. D. 1995. A protocol for aquatic hazard assessment of selenium. *Ecotoxicol. Environ. Safety* 32, 280–288.
- Lemly, A. D. 1996. Winter Stress Syndrome: An important consideration for hazard assessment of aquatic pollutants. *Ecotoxicol. Environ. Safety* 34, 223–227.
- Lemly, A. D. 1997a. Ecosystem recovery following selenium contamination in a freshwater reservoir. *Ecotoxicol. Environ. Safety* 36, 275–281.

- Lemly, A. D. 1997b. A teratogenic deformity index for evaluating impacts of selenium on fish populations. *Ecotoxicol. Environ. Safety* 37, 259–266.
- Lemly, A. D. 1999. *Preliminary assessment of selenium hazards on Caribou National Forest, Idaho*. Technical Report. U.S. Forest Service, Southern Research Station, Coldwater Fisheries Research Unit, Department of Fisheries and Wildlife Sciences, Virginia Tech University, Blacksburg, VA, USA. 20 pp.
- Lemly, A. D., Finger, S. E., and Nelson, M. K. 1993. Sources and impacts of irrigation drainwater contaminants in arid wetlands. *Environ. Toxicol. Chem.* 12, 2265–2279.
- Murtha, M. J., Burnet, G., and Harnby, N. 1983. Power plant fly ash — disposal and utilization. *Environ. Progress* 2, 193–198.
- NCDNRCD (North Carolina Department of Natural Resources and Community Development). 1986. *North Carolina Water Quality Standards Documentation: The Freshwater Chemistry and Toxicity of Selenium with an Emphasis on Its Effects in North Carolina*. Report Number 86–02. NCDNRCD, Division of Environmental Management, Raleigh, NC, USA.
- Oldfield, J.E. 1998. Environmental implications of uses of selenium with animals. In: *Environmental Chemistry of Selenium*, pp. 129–142. (Frankenberger, W. T., Jr. and Engberg, R. A., Eds.). New York, Marcel Dekker.
- Pennsylvania Bulletin. 1992. Permit No. 301106. Antrium Fly Ash Disposal Facility. *Pennsylvania Bulletin* 22, 311.
- Schildgen, B. 1996. Murphy's Laws. 1. Hogs rule. 2. You pay. *Sierra* (May to June): 29–31.
- Skorupa, J. P. 1998. Selenium poisoning of fish and wildlife in nature: Lessons from twelve real-world experiences. In: *Environmental Chemistry of Selenium*, pp. 315–354. (Frankenberger, W. T., Jr. and Engberg, R. A., Eds.). New York, Marcel Dekker.
- Sorensen, E. M. B. 1986. The effects of selenium on freshwater teleosts. In: *Reviews in Environmental Toxicology* 2, pp. 59–116. (Hodgson, E., Ed.). Amsterdam and New York, Elsevier.
- Sorensen, E. M. B., Bauer, T. L., Bell, J. S., and Harlan, C. W. 1982. Selenium accumulation and cytotoxicity in teleosts following chronic, environmental exposure. *Bull. Environ. Contam. Toxicol.* 29, 688–696.
- Terry, N. and Zayed, A. 1998. Phytoremediation of selenium. In: *Environmental Chemistry of Selenium*, pp. 633–655. (Frankenberger, W. T., Jr. and Engberg, R. A., Eds.). New York, Marcel Dekker.
- USEPA (United States Environmental Protection Agency). 1987. Ambient water quality criteria for selenium — 1987. *EPA-440/5-87-006*. USEPA, Office of Water Regulations and Standards, Washington, DC, USA.
- USEPA (United States Environmental Protection Agency). 1998. Wastes from the combustion of coal by electric utility power plants. Report to Congress. *EPA/530-SW-98-002*. USEPA, Washington, DC, USA.