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Ecosystem Recovery Following Selenium Contamination in a Freshwater Reservoir

A. Dennis Lemly

United States Forest Service, Southern Research Station, Coldwater Fisheries Research Unit, Department of Fisheries and Wildlife Sciences, Virginia Tech University, Blacksburg, Virginia 24061-0321

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Belews Lake, North Carolina, was contaminated by selenium in wastewater released from a coal-Bred electric generating facility during 1974-1985. Selenium hioaccumulated in aquatic food chains and caused severe reproductive failure and teratogenic deformities in fish. Beginning in 1986, the electric utility company changed its ash disposal practices and selenium-laden wastewater no longer entered the lake. A survey of selenium present in the water, sediments, benthic invertebrates, fish, and aquatic birds was conducted in 1996. Concentrations were compared to pre-1986 levels to determine how much change occurred during the decade since selenium inputs stopped. The data were also examined using a hazard assessment protocol to determine if ecosystem-level hazards to fish and aquatic birds had changed as well. Results reveal that waterborne selenium fell from a peak of 20 µg/liter before 1986, to <1 µg/liter in 1996; concentrations in biota were 85–95% lower in 1996. Hazard ratings indicate that high hazard existed prior to 1986 and that moderate hazard is still present, primarily due to selenium in the sediment-detrital food pathway. Concentrations of selenium in sediments have fallen by about 65–75%, but remain sufficiently elevated (l-4 µg/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 indicate that selenium-induced teratogenesis and reproductive impairment are occurring. Moreover, the concentrations of selenium in benthic food organisms are sufficient to cause mortality in young bluegill and other centrarchids because of Winter Stress Syndrome. At the ecosystem level, recovery has been slow. Toxic effects are still evident 10 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. © 1997 Academic Press

INTRODUCTION

Environmental pollution with selenium from industrial and agricultural sources has killed fish and wildlife at several locations in the United States. Selenium mobilized from the combustion of coal at electric generating stations has contaminated large reservoirs, leading to reproductive failure and elimination of entire communities of fish (Cumbie and Van Horn, 1978; Garrett and Inman, 1984; Woock and Summers, 1984; Woock *et al.,* 1985; Lemly, 1985a,1993c). Irrigation of seleniferous soils has produced subsurface drainage that contaminated wetlands and poisoned fish and migratory birds in the western U.S. (Lemly *et al.,* 1993, Lemly, 1994; Presser, 1994; Presser *et al.,* 1994). The U.S. Environmental Protection Agency (EPA) and some states have formulated and adopted increasingly restrictive water quality standards for selenium because of these incidents of environmental contamination (e.g., NCDEM, 1986; U.S. EPA, 1987; CEPA, 1992).

Although the toxic effects of selenium on fish and wildlife are well documented, relatively little is known about its persistence in contaminated aquatic habitats once waterborne inputs cease. Selenium undergoes a rather complex cycling dynamic in aquatic systems and several mechanisms are possible for moving it between water, sediments, and biota (Lemly and Smith, 1987). The predominant cycling pathways that exist during periods of selenium input may shift dramatically once inputs stop. For example, waterborne routes of exposure to fish and wildlife may be replaced by sediment-detrital pathways (Horne, 1991; Lemly, 1993c). The pathways may also vary substantially depending on the type of system, i.e., a wetland, reservoir, or river.

It is important to determine the time frame necessary for ecosystem recovery under natural conditions if the implications of selenium contamination are to be fully understood. In some situations there might be little need for remediation of a contaminated site once selenium inputs stopped, for example, if natural cleansing of the system was rapid relative to the anticipated hazard and biological impacts. In other cases, selenium contamination might be very persistent and seriously threaten future generations of fish and wildlife unless aggressive cleanup was undertaken. Knowing the approximate time frame needed for recovery of various aquatic systems would be an asset for risk assessors, environmental planners, and fish and wildlife managers. This study was conducted to address that information need.

Belews Lake, North Carolina, was contaminated by selenium in wastewater released from a coal-fired electric generating facility during 1974-l 985. Selenium bioaccumulated in aquatic food chains and caused severe reproductive failure and teratogenic deformities in fish (Cumbie and Van Horn, 1978; Lemly, 1985a,1993c). Begining in 1986, the electric utility

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company changed its coal ash disposal practices and selenium-laden wastewater no longer entered the lake. In the decade since, natural ecosystem processes have operated in the reservoir. There has been no remediation or other manipulations to reduce selenium levels. Thus, Belews Lake provided an ideal field site for investigating the time frame needed for a freshwater reservoir to recover from selenium contamination. A survey of selenium present in water, sediments, benthic invertebrates, fish, and aquatic birds was conducted in 1996. These concentrations were compared to pre- 1986 levels (Cumbie and Van Horn, 1978; Holland, 1979; Lemly, 1985a) to establish how much change has occurred since selenium inputs stopped. The data were also examined using a hazard assessment protocol to determine if ecosystem-level hazards to fish and aquatic birds changed as well.

MATERIALS AND METHODS

Study Site

Belews Lake is a Duke Power Company impoundment situated on Belews Creek, a small tributary to the Dan River, located in the northwestern Piedmont area of North Carolina (Fig. 1). At the dam site, the drainage area of the Belews Creek watershed is approximately 20,000 hectares (76 square miles). Construction of the reservoir began in spring 1970 and the lake reached full pool elevation in April 1973, somewhat ahead of the design schedule. This early filling of the lake allowed over 1 year of environmental stabilization to occur before the first generating unit of the Belews Creek Steam Station went on-line. At full pool, Belews Lake has a surface area of approximately 1560 hectares, a shoreline of 110 kilometers, a maximum depth of 42 meters, and a mean depth of 14 meters (Weiss and Anderson, 1978).

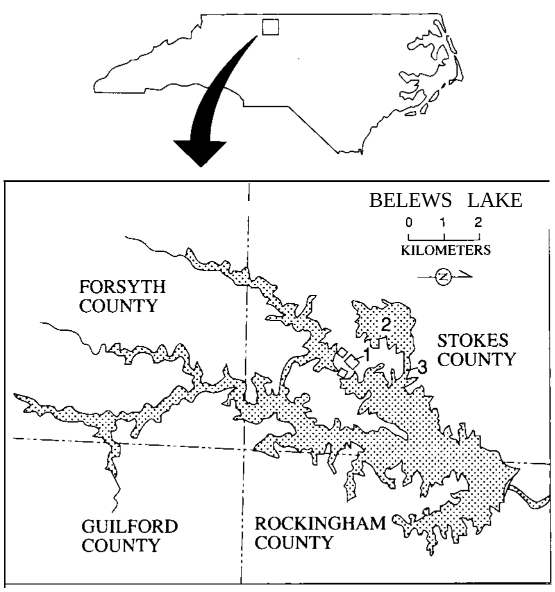

FIG. **1.** Geographic location of Belews Lake, North Carolina. 1, Belews Creek Steam Station; 2, coal ash settling basin; 3, ash basin return water canal.

Belews Lake was constructed to provide a source of condenser cooling water for the Belews Creek Steam Station, the largest coal-fired electric generating station on the Duke Power system and one of the largest in the United States. The station has two generating units; the first began operating in August 1974, the second in December 1975. Each of the two units has a generating capacity of 1140 MW and can consume up to 500 tons of coal per hour. Cooling water is supplied to each unit from Belews Lake at a maximum rate of 35 m^3/sec (2500 cfs), which is sufficient to circulate the entire lake volume through the power plant every 37 days. The average flow into the lake from the drainage basin is approximately 2.6 m^3/sec; however, forced evaporation caused by the heated discharge from the Belews Creek Steam Station reduces the average outflow to 2.1 m^3/sec. Consequently, the volume replacement time of Belews Lake increased from about 1000 days to 1500 days when the power plant began operating (Cumbie, 1978).

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 μg/liter) return flows from the ash basin entered the west side of Belews Lake through an ash sluice water canal (Fig. 1). Selenium bioaccumulated in aquatic food chains and caused reproductive failure in fish (Cumbie and Van Horn, 1978). In response to these problems with the fishery, Duke Power Company switched to a dry-ash handling system in December 1985 (NC-DEM, 1986). Fly-ash is now disposed in a landfill on the power plant site. Bottom ash and stormwater runoff from the landfill are conveyed into the old ash basin, which has been diverted to drain into the nearby Dan River.

Collection of Water, Sediments, and Biota

Samples were taken from Belews Lake and a reference lake (High Rock Lake), both of which lie in the Piedmont area of North Carolina. All samples were collected and analyzed during April and May 1996. Samples were taken from five locations around the perimeter of the lakes. These locations were in the littoral zone of the main lake basins, approximately 5-10 m from shore. All samples were refrigerated at +2°C until selenium analyses were performed (within 2 weeks). Water samples (five from each lake) were taken from approximately 1 m below the surface with an acid-rinsed linear polyethylene (LPE) bottle, filtered through 0.45-μm mesh netting into a 250-ml acid-rinsed LPE bottle, and acidified to pH <2.0 with ultrapure nitric acid (selenium content of acid <0.01 μg/liter).

Samples of lake sediments (10 from each lake) and benthic macroinvertebrates (10 from each lake) were taken with an

Ekman dredge. The top 3 cm of a dredge haul was used as a sediment sample. This layer was removed and placed into an acid-rinsed LPE bottle. Macroinvertebrates were removed from seived sediment samples, rinsed in distilled water to remove debris, and placed into acid-rinsed LPE bottles.

Fish were captured with seines. Three life stages were collected (fry, fingerlings, adults), representing four functional feeding groups: omnivorous bottom feeders (common carp, *Cyprinus carpio),* topwater feeders (mosquitofish, *Gambusia affinis),* insectivores (bluegill, *Lepomis macrochirus),* and carnivores (largemouth bass, *Micropterus salmoides).* Early life stages were examined for teratogenic deformities. For each lake, mature ovaries were removed from 10 adult fish of each species and used for selenium analysis (five ovaries pooled per sample for mosquitofish). Birds were killed with shotguns and the livers (10 for each lake) were removed and analyzed for selenium content. American coot (*Fulica americana)* was the bird species sampled.

Analysis of Terutogenic Deformities in Fish

A survey of externally visible abnormalities in young fish was conducted. Two sets of samples were taken: one of the fry stage (<3 cm total length for bluegill, largemouth bass, and carp; <1 cm for mosquitofish), and one of the fingerling stage (4-6 cm for bluegill, largemouth bass, and carp; l-l.5 cm for mosquitofish). For each set, approximately 50-150 individuals of each species were placed on ice and returned to the laboratory to be examined for malformations. Three categories of teratogenic abnormalities were enumerated: (1) spinal deformities (lordosis—concave curvature of lumbar and caudal regions of spine; scoliosis-lateral curvature of spine; kyphosis-convex curvature of thoracic region of spine resulting in humpback), (2) missing or abnormal fins, and (3) abnormally shaped head or mouth. The results of this survey were compared to those of one conducted in 1992 (Lemly, 1993c).

Determination of Selenium

Specimens and samples were manipulated with stainless-steel instruments during preparation and analysis. All selenium measurements were made using hydride generation techniques with a Varian SpectrAA- atomic absorption spectrophotometer equipped with a Varian PSC-56 autosampler and VGA-76 hydride generator. Fish and bird tissue and macroinvertebrates were freeze-dried with a Virtis SRC-4X sublimator, and moisture content was determined by difference between wet and dry weight. Dried samples were blended and prepared for analysis using a dry-ash oxidative fusion technique (May, 1982; Hansson *et al.,* 1987). After the oxidation step, samples were treated with hot hydrochloric acid to convert all selenium present to the selenite form. Water samples were prepared for analysis using a two-step persulfate oxidation/hydrochloric acid reduction procedure (Ingersoll *et al.,* 1990). Selenium was removed from sediments using acid digestion techniques (Fio and Fujii, 1988) and determinations were made on the extracts. Smoke-free conditions were maintained during all analytical procedures.

Four types of samples were used to assess quality control in selenium determinations: procedural blanks, pre- and postdigestion spikes, replicate analyses, and reference materials with known selenium content. Limits of detection averaged 0.070 μg/g Se for tissue and sediments, and 0.0056 μg/liter Se for water. All reagent blanks had selenium concentrations below the limits of detection. Recovery of selenium spikes ranged from 83 to 112% for predigestion samples (average = 94%), and 88–105% for postdigestion samples (average = 97%). The mean relative percentage difference of duplicate analyses was 3.5 (*n* = 28). The selenium concentration determined for National Bureau of Standards RM 50 (Albacore Tuna) consistently fell within the certified acceptable range.

Hazard Assessment

A protocol for aquatic hazard assessment of selenium (Lemly, 1995) was applied to pre- and post-1986 data sets from Belews Lake to determine the toxic threat to fish and aquatic birds. This procedure establishes a separate hazard rating for five ecosystem components (water, sediments, benthic macroinvertebrates, fish, aquatic birds) based on where the highest concentrations of selenium fall on corresponding hazard profiles given in the protocol. The hazard profiles were developed from a comprehensive synthesis of field and laboratory data on selenium cycling, food-chain bioaccumulation, and reproductive toxicity to fish and aquatic birds. The hazard rating focuses on reproductive endpoints, which are the most sensitive indicators for predicting ecosystem-level impacts of selenium contamination (Lemly, 1993a). Numerical scores are assigned according to the hazard ratings: None, 1; Minimal, 2; Low, 3; Moderate, 4; High, 5. A final ecosystem-level hazard assessment is determined by adding the scores for each ecosystem component and comparing the total to the following evaluation criteria: 5, No Hazard; 6-8, Minimal Hazard; 9-l 1, Low Hazard; 12-15, Moderate Hazard; 16-25, High Hazard. The assessment protocol can also be applied if selenium data are missing for one ecosystem component. In that case, the following modified criteria are used for calculating the final hazard evaluation (Lemly, 1996): 4, No Hazard; 5-7, Minimal Hazard; 8-10, Low Hazard; 11-14, Moderate Hazard; 15-20, High Hazard. Modified criteria were necessary for pre-1986 data sets because no information on aquatic birds was available. Selenium concentrations in whole-body fish samples and aquatic bird livers were converted to equivalent egg concentrations (whole-body values x 3.3 for fish; liver values x 0.33 for birds; Lemly, 1995) for use in the hazard assessment protocol.

RESULTS

Compared to pre-1986 levels, concentrations of selenium present in Belews Lake in 1996 were markedly lower in all ecosystem components (Table 1). Waterborne selenium

TABLE 1
Selenium Concentrations and Aquatic Hazard Ratings for Belews Lake, North Carolina, and a Reference Lake

Ecosystem component	Concentration of selenium[a]	Hazard assessment: Component hazard[b]	Component score	Total score	Ecosystem hazard
		Pre-1986			
Belews Lake					
Water	5–20	High	5		
Sediments	4–12	High	5		
Invertebrates	15-57	High	5		
Fish eggs				20	High
Carp	45-119	High	5		
Bluegill	40–133	High	5		
Bass	77-159	High	5		
Mosquitofish	63-120	High	5		
		1996			
Belews Lake					
Water	<1	None			
Sediments	1–4	Moderate	4		
Invertebrates	2-5	Moderate	4		
Fish eggs				15	Moderate
Carp	5-18	Moderate	4		
Bluegill	3-20	Moderate	4		
Bass	4–16	Moderate	4		
Mosquitofish	5-20	Moderate	4		
Bird eggs	2-5	Minimal	2		
High Rock Lake					
Water	<1	None			
Sediments	<1	None	1		
Invertebrates	<2	None	1		
Fish eggs				5	None
Carp	<3	None	1		
Bluegill	<3	None	1		
Bass	<3	None	1		
Mosquitofish	<3	None	1		
Bird eggs	<3	None	1		

[a] Selenium concentrations in μg/liter (parts per billion) for water; μg/g (parts per million) dry weight for sediments and biota.

[b] Hazard ratings were determined by comparing selenium concentrations to hazard profiles from Lemly (1995)

reached a peak of 20 μg/liter (average = 10 μg/liter) before selenium inputs stopped, then fell to near reference levels (<1 μg/liter) in 1996. Since 1986, selenium concentrations in biota have dropped by about 85–95%; concentrations in sediments have fallen by about 65-75%. Hazard estimates indicated that high hazard existed in Belews Lake prior to 1986 and that moderate hazard still persists (Table 1). The frequency of developmental abnormalities in young fish was high relative to the reference lake, both in 1992 and in 1996 (Table 2). Whereas fingerling size fish exhibited deformities in 1992, the deformities were confined to fry in 1996.

DISCUSSION

Despite the substantial decline in selenium concentrations in Belews Lake over the past decade, two direct lines of evidence indicate that the toxic threat to fish and aquatic birds has not been eliminated. First, selenium concentrations in benthic invertebrates were high enough to cause Winter Stress Syndrome (WSS) in young bluegill and other centrarchids. Described in detail by Lemly (1993b), this condition develops when otherwise sublethal effects (metabolic stress) due to selenium are present concurrently with the arrival of cold water temperature in late autumn. Cold weather and the associated short photoperiod of winter environmentally “programs” the fish for reduced activity and food intake, and they do not respond to the metabolic stress with increased feeding. If exposure to selenium persists, stored body lipid necessary for overwintering is depleted, body condition drops, and many of them will die. Dietary selenium concentrations of 5 μg/g dry weight are sufficient to cause WSS to develop and kill as many as 30% of young-of-the-year bluegill (Lemly, 1993b). The invertebrate

TABLE 2
Prevalence of Teratogenic Deformities in Young Fish from Belews Lake, North Carolina, and a Reference Lake

Year and fish species	Lake" and number examined		Total with teratogenic deformities (%)[b]	
	Fry[c]	Fingerlings	Fry	Fingerlings
1992[d]				
Common carp		BEL-29		2 (7)
		HRL-28	—	0 (0)
Bluegill		BEL-7 1	—	4 (6)
		HRG64	—	0 (0)
Mosquitofish		BEL-103		4 (4)
		HRL-90		0 (0)
Largemouth bass	—	BEL–21	—	1 (5)
		HRL-34		0 (0)
1996				
Common carp	BEG.52	BEL-73	3 (6)	0 (0)
	HRL-60	HRL-55	0 (0)	0 (0)
Bluegill	BEL-105	BEL-100	4 (4)	0 (0)
	HRL-119	HRL- 100	0 (0)	0 (0)
Mosquitofish	BEL-140	BEG100	6 (4)	0 (0)
	HRL–100	HRL-100	0 (0)	0 (0)
Largemouth bass	BEL-101	BEG75	3 (3)	0 (0)
	HRL-88	HRL-80	0 (0)	0 (0)

[a] BEL, Belews Lake; HRL, High Rock Lake.
[b] Rounded to the nearest percent.
[c] No fry were collected in 1992.
[d] Data taken from Lemly (1993c).

food organisms of fish in Belews Lake contained 2-5 μg/g selenium (Table 1). The presence of elevated concentrations of selenium in food organisms suggests that WSS is probably slowing the recovery of centrarchids in Belews Lake.

The second line of evidence is even more compelling. Selenium-induced teratogenic deformities occurred in young fish (Table 2). Teratogenesis is a well-known biomarker of selenium toxicosis in wild birds and fish (Ohlendorf *et al.,* 1986; Hoffman *et al.*, 1988; Lemly, 1993c). Selenium is passed from parents to their offspring in eggs. Absorption of selenium from the yolk as young fish develop causes morphological abnormalities if concentrations in eggs reach about 15-20 μg/g (Gillespie and Baumann, 1986; Woock *et al.*, 1987; Coyle *et al.*, 1993). Lemly (1993c) developed a predictive model for evaluating the role of teratogenic effects in reproductive failure of centrarchid fishes based on tissue concentrations of selenium. Given the concentrations present in ovaries of bluegill and largemouth bass from Belews Lake (5-20 μg/g, Table 1), approximately 10–20% reproductive failure is predicted for these species. Moreover, the prevalence of teratogenic deformities was similar in 1992 and 1996, suggesting that recovery from this facet of reproductive failure over the past 4 years has been negligible.

The difference in frequency of teratogenic deformities between fry and fingerlings has important implications for detecting and evaluating reproductive impacts from selenium contamination. In 1992, when deformities were found in fingerlings, the fish population of Belews Lake was in an early stage of recovery. Although several species had been stocked and were naturally reproducing, some had not colonized the entire lake and none had completely repopulated to their former numbers (Lemly, 1993c). The abundance of predatory species such as largemouth bass was low. Lemly (1993c) hypothesized that low predation pressure allowed deformed individuals to survive to juvenile and adult life stages. He further stated that if the fish population were in a more normal balance, selenium teratogenesis would produce deformed larvae that would fall prey to piscivorous species and thus not be seen in the juvenile and adult populations. This scenario is supported by findings of the present study (Table 2). Since 1992 the fish population in Belews Lake has recovered further and there is a typical mix of predators and forage species. Predation pressure on young fish should be considerably greater now than when the 1992 survey was made. The 1996 survey found that teratogenic deformities were clearly evident in fry, but not fingerlings. These deformities are permanent and do not disappear as fish grow (Fishbein, 1977; Harbison, 1980). Thus, these deformed individuals either died due to selenium toxicity or other causes, or fell prey to piscivorous species. Results from the 1992 survey reveal that deformed fry would survive to fingerling size and beyond. The absence of deformities in fingerlings in 1996 was probably due to predation.

The point made here is that in order to assess reliably the severity of selenium-induced teratogenesis, it is necessary to focus on the larval and fry life stages. Otherwise, an important component of selenium's reproductive toxicity to fish can be entirely overlooked. Even then, it is likely that the true frequency of teratogenic deformities will be underestimated because of the removal of some individuals by predation.

In addition to the direct lines of field evidence that indicate continuing selenium toxicity to fish in Belews Lake, hazard assessment indicates that the ecosystem has not recovered. Results indicate that high hazard existed prior to 1986 and that moderate hazard is still present (Table 1). Current hazard is primarily due to the persistence of a contaminated sediment-detrital food pathway. Concentrations of selenium in sediments have fallen by about 65–75%, but remain sufficiently elevated to contaminate benthic food organisms of fish and aquatic birds. According to the original definition of aquatic hazard ratings for the assessment protocol used in this study, moderate hazard indicates a persistent toxic threat sufficient to impair but not eliminate reproduction. Depending on species sensitivity, effects could range from severe to negligible (Lemly, 1995). The field evidence from Belews Lake confirms that a rating of moderate hazard is valid and correct. Teratogenesis and WSS are sufficient to negatively affect reproduction, but they do not entirely eliminate it for any of the species examined.

Concentrations of selenium in the eggs of aquatic birds (equivalent concentrations) collected from Belews Lake were elevated, but were not as high as those in fish eggs (Table 1).

One reason may have been that the bird species sampled (American coot) was not foraging on benthic invertebrates or other selenium-contaminated food organisms to the extent that fish were. However, stomach contents were not examined, so this cannot be confirmed as the primary cause. Another possible reason is that differential accumulation of selenium occurred between birds and fish even though they were both consuming similar diets. Other research has found that coots typically contain somewhat less selenium than fish collected from the same aquatic habitat (Ohlendorf *et al.*, 1986, 1987). Differences in diet and/or bioaccumulation would explain the relatively low selenium in coots from Belews Lake. The hazard rating from the assessment protocol (minimal for birds) is supported by field data on coot reproduction. Concentrations of selenium in birds from Belews lake were not high enough to cause teratogenic effects or embryomortality (Skorupa and Ohlendorf, 1991; Skorupa *et al.*, 1996). However, the threat to other aquatic birds that utilize Belews Lake but were not sampled in this study (e.g., dabbling ducks, *Anus* sp.) may be greater because they are generally more sensitive to selenium (Skorupa et al., 1996) and have food habits that could result in greater dietary exposure than coots. Thus, the hazard estimates for coots cannot be generalized to all waterbirds.

Given that selenium concentrations have been substantially reduced, why does moderate hazard persist at the ecosystem level? One might assume that reducing concentrations in water, sediments, and biota would translate to reduced toxicity. This is true to some extent, but the relationship is not one-to-one. The toxicity profile for selenium is quite steep, i.e., the transition from no effects to severe effects occurs over a very narrow range of selenium concentrations (Lemly, 1985b). It begins at low concentrations in water and sediment because of the propensity for high bioaccumulation in food chains. Relatively little reduction in toxic impact is realized unless the lower end of the profile is reached, i.e., l-2 μg/liter in water and about 1 μg/g in sediments. The levels of contamination in sediments and biota prior to 1986 were quite high and toxic effects on fish were severe, which led to complete reproductive failure in most species (Lemly, 1985a). Although large reductions in selenium have occured since 1986 (85–95%), toxic effects have not necessarily been reduced by an equivalent amount. The threshold concentrations for preventing bioaccumulation have not yet been reached for sediments in Belews Lake. Consequently, contaminated benthic-detrital food chains persist and subtle, but nontheless important, modes of selenium toxicity (WSS and teratogenesis) are still operating.

Despite the recovery that has occurred, fish in Belews Lake have not regained the balanced populations that were present in the lake before it was contaminated (personal communication with Hugh Barwick, Duke Power Company, Huntersville, NC). Spawning is taking place and young-of-the-year fish are evident, but teratogenesis and WSS are offsetting part of the natural reproduction. However, once these hazards during early life are passed, survival and growth of juvenile and young-adult fish are generally good. Large year-to-year fluctuations in predator-prey numbers are typical of warm-water reservoir fisheries. Overlaying the natural reduction in numbers expected during "down" years with a selenium-contaminated benthic-detrital food chain could result in partial collapse of some populations, e.g., largemouth bass. Thus, some artificial stocking may be necessary to maintain a quality sport fishery in Belews Lake.

The ability of a reservoir or wetland to naturally cleanse itself of selenium contamination depends on many factors such as hydrologic retention time, chemical conditions that regulate selenium cycling dynamics, and sedimentation rate (to bury and isolate sediment-associated selenium) just to name a few (Lemly and Smith, 1987). Belews Lake has a long hydrologic retention time (1500 days) and relatively low sedimentation rates (<0.5 cm/year). These were among the primary physical factors that contributed to selenium accumulation originally (Cumbie, 1978; Duke Power Company, 1980), and continue to cause problems today. A principle component of the selenium paradigm in Belews Lake or any other contaminated body of water is that as long as selenium is bioavailable from sediments, fish and wildlife are at risk. This is true regardless of the concentrations in water or other ecosystem components. The extent to which this risk is expressed as a toxic impact depends on site- or ecosystem-specific factors that regulate the cycling dynamics of selenium. Results from this and other studies of Belews Lake indicate that some of the conditions which favor good warm-water fish production, i.e., oligo-mesotrophic reservoirs with low inorganic sedimentation rates, also favor the accumulation and retention of selenium.

CONCLUSIONS

The fish community of Belews Lake has regained a mix of species and numbers that, to the casual observer, might suggest that recovery is complete. Indeed, a great deal of recovery has taken place and, overall, the lake is in far better biological condition now than it was in 1986. However, selenium toxicity is still evident 10 years after selenium inputs were stopped. At the ecosystem level, recovery has been slow and the sediment-associated selenium will likely continue to be a significant hazard to fish and aquatic birds for several more years. It is clear that the time frame necessary for complete recovery from selenium contamination in freshwater reservoirs can be on the order of decades. Given this possibility, industrial and natural sources of selenium should be scrutinized in the environmental planning process so that threats to fish and wildlife can be recognized and avoided.

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REFERENCES

CEPA (California Environmental Protection Agency) (1992). Derivation of *Site- Specific Water Quality Standards for Selenium in San Francisco Bay.* Technical Report. CEPA, Oakland, CA.

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.

Cumbie, P. M. (1978). *Belews Luke Environmental Study Report: Selenium and Arsenic Accumulation.* Technical Report 78–04. Duke Power Co., Charlotte, NC.

Cumbie, P. M., and Van Horn, S. L. (1978). Selenium accumulation associated with fish mortality and reproductive failure. *Proc. Ann. Conf. Southeastern Assoc. Fish Wildl. Agencies 32, 612-624.*

Duke Power Company. (1980). *Toxic Effects of Selenium on Stocked Bluegill (Lepomis macrochirus) in Belews Luke, North Carolina, April-September, 1979.* Technical Report, Duke Power Co., Charlotte, NC.

Fio, J. L., and Fujii, R. (1988). *Comparison of Methods to Determine Selenium Species in Saturation Extracts of Soils from the Western San Joaquin Valley, California.* Open-File Report 88–458. U.S. Geological Survey, Sacramento, CA.

Fishbein, L. (1977). Toxicology of selenium and tellurium. In *Advances in Modern Toxicology,* Vol. 2, *Toxicology of Trace Elements* (R. A. Goyer and M. A. Mehlman, Eds.), pp. 191–240. Wiley, New York.

Garrett, G. P., and Inman, C. R. (1984). Selenium-induced changes in the fish populations of a heated reservoir. *Proc. Ann. Conf. Southeastern Assoc. Fish Wildl.* Agencies 38, 291–301.

Gillespie, R. B., and Baumann, P. C. (1986). Effects of high tissue concentrations of selenium on reproduction by bluegills. *Trans. Am. Fish. Soc.* 115, 208–213.

Hansson, L., Petterson, J., and Olin, A. (1987). A comparison of two digestion procedures for the determination of selenium in biological material. *Talanta 34,* 820–833.

Harbison, R. D. (1980). Teratogens. In *Casarett and Doull's Toxicology: The Basic Science of Poisons* (J. Doull, C. D. Klaassen, and M. 0. Amdur, Eds.), pp. 158-175. Macmillan, New York.

Hoffman, D. J., Ohlendorf, H. M., and Aldrich, T. W. (1988). Selenium teratogenesis in natural populations of aquatic birds in central California. *Arch. Environ. Contam. Toxicol. 17, 5 19-525.*

Holland, E. A. (1979). *Arsenic and Selenium in the Water, Sediments, and Biota near a Coal-Fired Power Plant-Belews Luke, North Carolina.* Master's Thesis. University of North Carolina, Chapel Hill, NC.

Home, A. J. (1991). Selenium detoxification in wetlands by permanent flooding. I. Effects on a macroalga, an epiphytic herbivore, and an invertebrate predator in the long-term mesocosm experiment at Kesterson Reservoir, California. *Water Air Soil Pollut. 57-58, 43-52.*

Ingersoll, C. G., Dwyer, F. J., and May, T. W. (1990). Toxicity of inorganic and organic selenium to *Daphnia magna* (Cladocera) and *Chironomus riparius* (Diptera). *Environ. Toxicol. Chem. 9,* 1171-l 181.

Lemly, A. D. (1985a). Toxicology of selenium in a freshwater reservoir: Implications for environmental hazard evaluation and safety. *Ecotoxicol. Environ. Saf.* 10, 314–338.

Lemly, A. D. (1985b). Ecological basis for regulating aquatic emissions from the power industry: The case with selenium. *Regul. Toxicol. Pharmacol. 5,* 465–486.

Lemly, A. D. (1993a). Guidelines for evaluating selenium data from aquatic monitoring and assessment studies. *Environ. Monitor. Assess. 28,* 83–100.

Lemly, A. D. (1993b). Metabolic stress during winter increases the toxicity of selenium to fish. *Aquat. Toxicol. 27, 133-158.*

Lemly, A. D. (1993c). Teratogenic effects of selenium in natural populations of freshwater fish. *Ecotoxicol. Environ. Saf. 26,* 181–204.

Lemly, A. D. (1994). Irrigated agriculture and freshwater wetlands: A struggle for coexistence in the western United States. *Wetlands* Ecol. *Manage.* 3, 3-15.

Lemly, A. D. (1995). A protocol for aquatic hazard assessment of selenium. *Ecotoxicol. Environ. Saf. 32,* 280–288.

Lemly, A. D. (1996). Evaluation of the hazard quotient method for risk assessment of selenium. *Ecotoxicol. Environ. Saf. 35, 156162.*

Lemly, A. D., and Smith, G. J. (1987). *Aquatic Cycling of Selenium: Implications for Fish and Wildlife.* Fish and Wildlife Leaflet 12. US. Fish and Wildlife Service, Washington, DC.

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.

May, T. W. (1982). Recovery of endogenous selenium from fish tissues by open system dry ashing. *J. Assoc. Off. Anal. Chem. 65,* 1140–1145.

NCDEM (North Carolina Division of Environmental Management) (1986). *North Carolina Water Quality Standards Documentation: The Freshwater Chemistry and Toxicity of Selenium with an Emphasis on its Effects in North Carolina.* Report No. 86–02. North Carolina Department of Natural Resources and Community Development, Raleigh, NC.

Ohlendorf, H. M., Hoffman, D. J., Saiki, M. K., and Aldrich, T. W. (1986). Embryonic mortality and abnormalities of aquatic birds: Apparent impacts of selenium from irrigation drainwater. Sci. *Total Environ. 52,* 49–63.

Ohlendorf, H. M., Hothem, R. L., Aldrich, T. W., and Krynitsky, A. J. (1987). Selenium contamination of the Grasslands, a major California waterfowl area. Sci. *Total Environ. 66, 169-183.*

Presser, T. S. (1994). The Kesterson Effect. *Environ. Manage.* 18, 437–454.

Presser, T. S., Sylvester, M. A., and Low, W. H. (1994). Bioaccumulation of selenium from natural geologic sources in the western states and its potential consequences. *Environ. Manage.* 18, 423–436.

Skorupa, J. P., and Ohlendorf, H. M. (1991). Contaminants in drainage water and avian risk thresholds. In *The Economics and Management of Water and Drainage in Agriculture* (A. Dinar and D. Zilberman, Eds.), pp. 345-368. Kluwer Academic, Boston, MA.

Skorupa, J. P., Morman, S. P., and Sefchick-Edwards, J. S. (1996). *Guidelines for Interpreting Selenium Exposures of Biota Associated with Nonmarine Aquatic Habitats.* Technical Report. U.S. Fish and Wildlife Service, Sacramento, CA.

U.S. EPA. (1987). *Ambient Water Quality Criteria for Selenium-1987.* EPA-440/5–87-006. U.S. Environmental Protection Agency, Washington, DC.

Weiss, C. M., and Anderson, T. P. (1978). *Belews Lake: A Summary of a Seven Year Study (August 1970–June 1977) to Assess Environmental Effects of a Coal-Fired Power Plant on a Cooling Pond.* Technical Report ESE No. 475. Department of Environmental Sciences and Engineering, University of North Carolina at Chapel Hill, Chapel Hill, NC.

Woock, S. E., and Summers, P. B. (1984). Selenium monitoring in Hyco Reservoir (NC) waters (1977-1981) and biota (1977-1980). In *Workshop Proceedings: The Effects of Trace Elements on Aquatic Ecosystems,* pp. 6-l-6-27, Publication EA-3329. Electric Power Research Institute, Palo Alto, CA.

Woock, S. E., Bryson, W. T., Macpherson, K. A., Mallin, M. A., and Partin, W. E. (1985). *Roxboro Steam Electric Plant-Hyco Reservoir 1984 Bioassay Study.* Technical Report. Carolina Power and Light Company, New Hill, NC.

Woock, S. E., Garrett, W. R., Partin, W. E., and Bryson, W. T. (1987). Decreased survival and teratogenesis during laboratory selenium exposures to bluegill, *Lepomis macrochirus. Bull. Environ. Contam. Toxicol. 39, 998-1005.*