

Ex. 6

Declaration of A. Dennis Lemly, Ph.D.

On

**Aquatic Hazard of Selenium Releases From Coal Mining in the
Mud River Ecosystem, West Virginia**

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Background on Selenium Cycling and Bioaccumulation

One of the primary human activities leading to increased selenium in the environment is the mining, processing, and burning of coal for electric power production. Selenium in raw coal and overburden is leached out when these materials are exposed to air and water, and the leachate can pose a significant toxic hazard to aquatic life (Lemly 1985a). The most important principle to understand when evaluating these threats is the ability of selenium to bioaccumulate. This means that a low concentration of selenium in water has the potential to increase by several orders of magnitude by the time it reaches fish and wildlife. For example, a water concentration of 10 ug/L (parts per billion) can increase to over 5,000 times that amount in fish tissues. Bioaccumulation causes otherwise harmless concentrations of selenium to reach toxic levels. Although fish do take up some selenium directly from water, most of it comes from their diet. Therefore, in order to protect fish from selenium poisoning it is essential to keep waterborne selenium below levels that cause bioaccumulation in the food chain (Lemly and Smith 1987). Another important principle is that selenium can cycle in aquatic habitats by moving in and out of sediments. A large portion of the total selenium in a stream or reservoir may be present in sediments, deposited directly from

water or from plants and animals as they die and decompose. However, this pool of selenium is not permanently removed from the system. Biological activity, water chemistry changes, and physical disturbance can mobilize selenium back into water and organisms. This means that the selenium in sediments remains active, and provides a significant source of pollution to bottom-dwelling invertebrates and the fish that feed on them. Case studies show that selenium in sediments can cycle into the water and food chain for decades after selenium inputs are stopped (Lemly 1997).

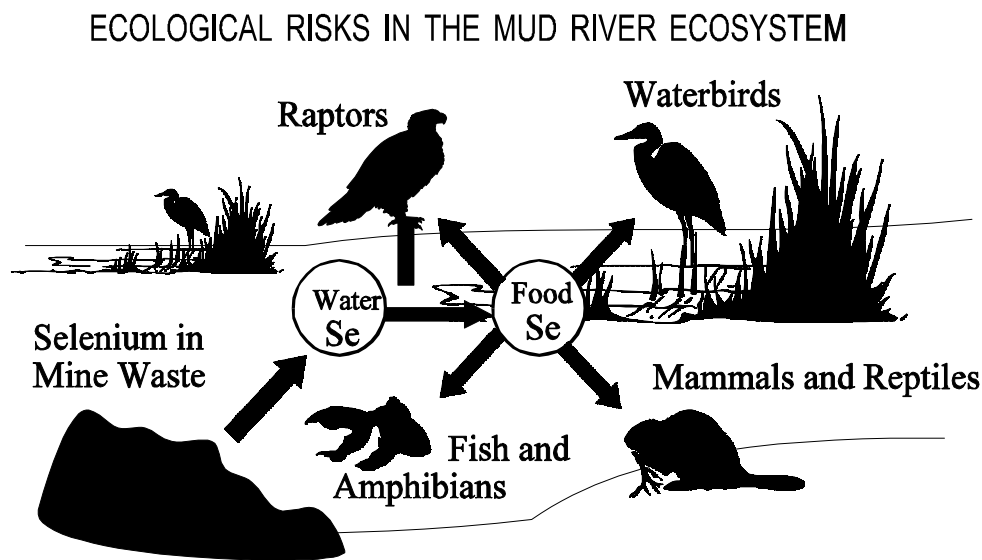


Figure 1. Pathways for selenium movement from mine waste, bioaccumulation in food chains, and dietary exposure of fish and wildlife populations in the Mud River ecosystem.

What are the Toxic Effects and What are the Toxic Concentrations?

Selenium exerts two main types of effects on fish: (1) direct toxicity to juveniles and adults, and (2) reproductive impacts from selenium that is passed from parents to offspring in eggs. Both of these modes of toxicity can occur at the same time so the threat from selenium poisoning is multifaceted. Type 1 toxicity can begin to occur if concentrations in the food chain reach 3 ug/g (parts per million, dry weight) and whole-body residues in fish reach 4 ug/g dw (Cleveland et al. 1993, Lemly 1993a, Hamilton 2003). This form of selenium poisoning involves changes in physiology that causes damage to gills and internal organs, ultimately resulting in death of the fish (Sorensen 1986). There may be no outwardly visible symptoms in this type of selenium toxicity or, if selenium concentrations are high enough, some fish may appear swollen from accumulation of fluid (edema) or have cloudy lenses (cataracts) in their eyes (Lemly 2002a). Type 2 effects occur when selenium present in egg yolk is absorbed by the developing embryo. A variety of developmental abnormalities can result in newly hatched larval fish, such as teratogenic deformities of the spine, head, and fins (Lemly 1993b). Other toxic symptoms include hemorrhaging and swelling or edema (Gillespie and Baumann 1986, Hermanutz et al. 1993). Most of these effects are lethal because they either kill young fish just after hatching or, in the case of some teratogenic deformities, prevent them from feeding normally and escaping predators as they grow (see Figures 2-4).

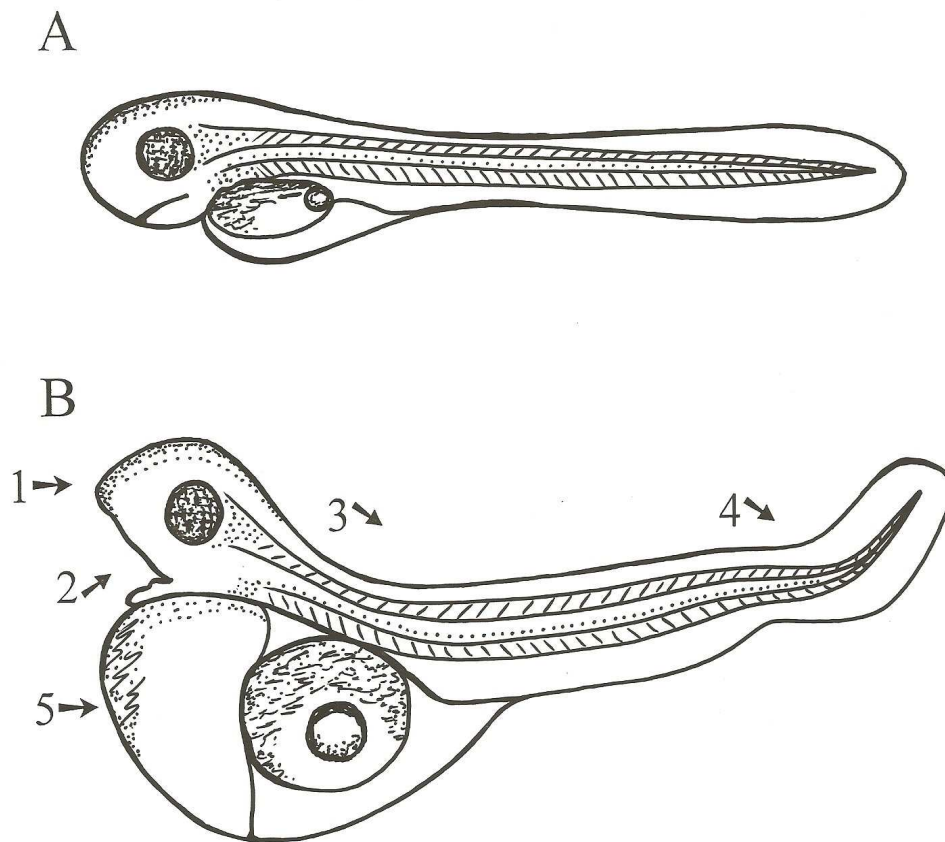


Figure 2. Typical appearance of larval fish at about 2-4 days after hatching. (A) Normal larvae with yolk absorption nearing completion and straight, developing spine, (B) Abnormal development due to selenium-induced terata: (1) deformed, pointed head; (2) deformed, gaping lower jaw; (3) kyphosis (curvature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



Figure 3. One of the most common and outwardly visible teratogenic effects of selenium in fish is deformity of the spine. Shown here are examples of dorso-ventral abnormalities (kyphosis and lordosis).



Figure 4. Lateral curvature of the spine (scoliosis) caused by exposure to elevated selenium.

Individual on right is normal.

Type 2 effects (reproductive failure) begin to occur at egg selenium concentrations of about 9 ug/g dw, which is equivalent to about 16 ug/g dw whole-body in the parent (Coyle et al. 1993, Hermanutz et al. 1993). Adult fish may be unaffected by selenium concentrations that impair their ability to reproduce so the threat of selenium impacts on a fish population must be assessed by something more than routine monitoring surveys, that is, simply finding fish does not indicate the absence of selenium toxicity (Lemly 2002b). Waterborne concentrations of selenium in the 1-5 ug/L range can bioaccumulate and begin the Type 1 and/or Type 2 effects. The exact number is site-specific, and depends on the kind of aquatic system (stream, reservoir, wetland), its biological productivity, and the chemical form of selenium present in the water. Case studies show that if waterborne selenium reaches 10 ug/L, complete reproductive failure can occur in reservoirs, and reproduction may be reduced by 40% in streams (Cumbie and Van Horn 1978, Lemly 1985b, Gillespie and Baumann 1986, Hermanutz et al. 1993).

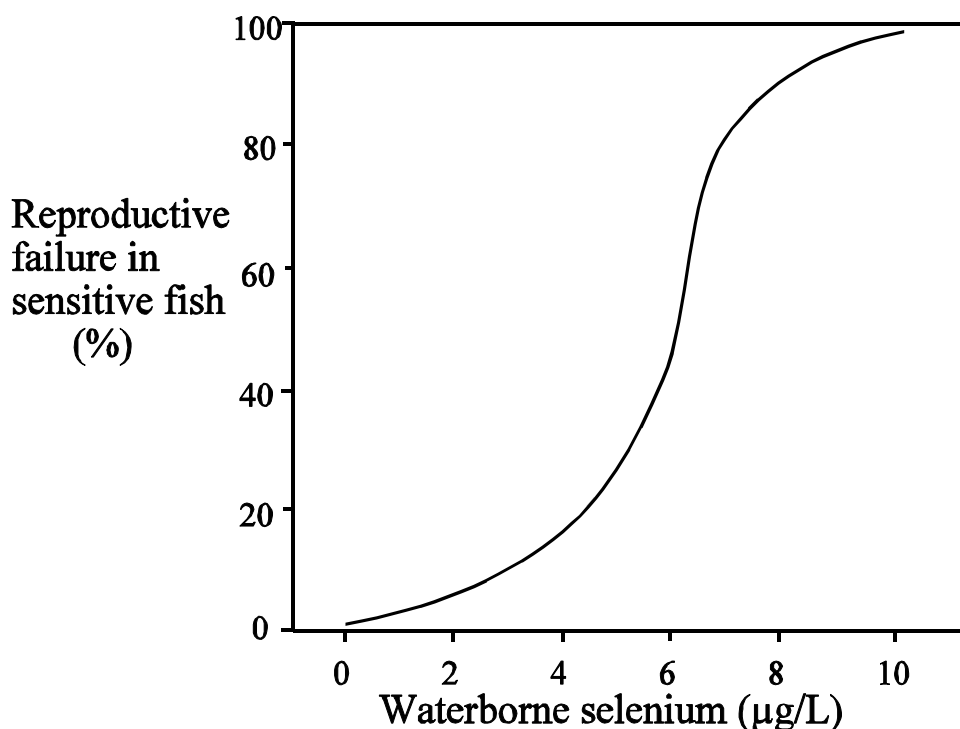


Figure 5. Relationship between the concentration of selenium in habitats favorable for bioaccumulation (e.g., reservoirs and wetlands) and the degree of reproductive failure in sensitive fish species (e.g., bluegill). A small increase in waterborne selenium can result in catastrophic impacts on reproductive success.

What Concentrations are Present in the Mud River Ecosystem?

Selenium analyses from the West Virginia Department of Environmental Protection, coal mine discharge monitoring reports, and U.S. Geological Survey have documented selenium levels in water and fish tissues over the past three years (2005-2007). These records indicate the following concentrations:

Water

Coal mine discharges:

2006 = <5-36ug/L; 2007 = <5-82 ug/L

Mud River:

2006 = 4-14 ug/L; 2007 = 22 ug/L

Upper Mud River Reservoir:

2006 = 1-6 ug/L; 2007 = 3-7 ug/L

Fish (whole body, dry weight)

2005 Mud River = 1-19 ug/g; Upper Mud River Reservoir = 22-60 ug/g

2006 Mud River = 2-27 ug/g; Upper Mud River Reservoir = 6-60 ug/g

2007 Mud River = 1-17 ug/g; Upper Mud River Reservoir = 3-53 ug/g

What is the Hazard of Selenium from Coal Mining?

Aquatic hazard ratings can be derived for selenium monitoring data based on waterborne, food-chain, or fish tissue residues in relation to known toxic effects (Lemly 2002b). A scoring system produces ratings that reflect the degree of hazard present: none, minimal, low, moderate, or high. For the Mud River ecosystem, this can be done by comparing available data for selenium in water and fish tissues to toxic thresholds, and to the magnitude of toxicity expected as concentrations exceed these thresholds. With respect to water, concentrations of selenium in coal mine discharges are up to 16 times the level that can bioaccumulate and cause major Type 1 and Type 2 toxicity in fish (5 ug/L) and up to 8 times the amount that can completely eliminate reproduction in sensitive species (10 ug/L). The hazard of waterborne selenium in coal mine discharges is high.

Concentrations present in the Mud River are lower, but maximums still exceed the threshold for major toxic impacts by a factor of 3-4. Consequently, the hazard of Mud River water is also high. Concentrations present in Mud River Reservoir do not exceed the 10 ug/L threshold for catastrophic impacts, but they still represent a substantial threat because they exceed the 2-5 ug/L thresholds for toxicity and reproductive failure. Case study data indicate that this is a serious risk. For example, field studies show that when waterborne selenium in southern reservoirs reaches 2-5 ug/L, bioaccumulation can be sufficient to cause Type 1 and Type 2 toxicity in fish (Cumbie and Van Horn 1978, Holland 1979, Sorensen and Bauer 1984, Sorensen 1988). Because concentrations of selenium in Mud River Reservoir are at this level (average of 3-4 ug/L, maximum of 7 ug/L), the hazard of selenium is high. In addition to the preceding hazard ratings, waterborne selenium concentrations measured in the Mud River and Mud River Reservoir are from 1.5 to 4.5 times the current EPA freshwater criterion for the protection of aquatic life (5 ug/L). Both maximum and average concentrations of waterborne selenium increased from 2006 to 2007 in coal mine discharge, Mud River, and Upper Mud River Reservoir.

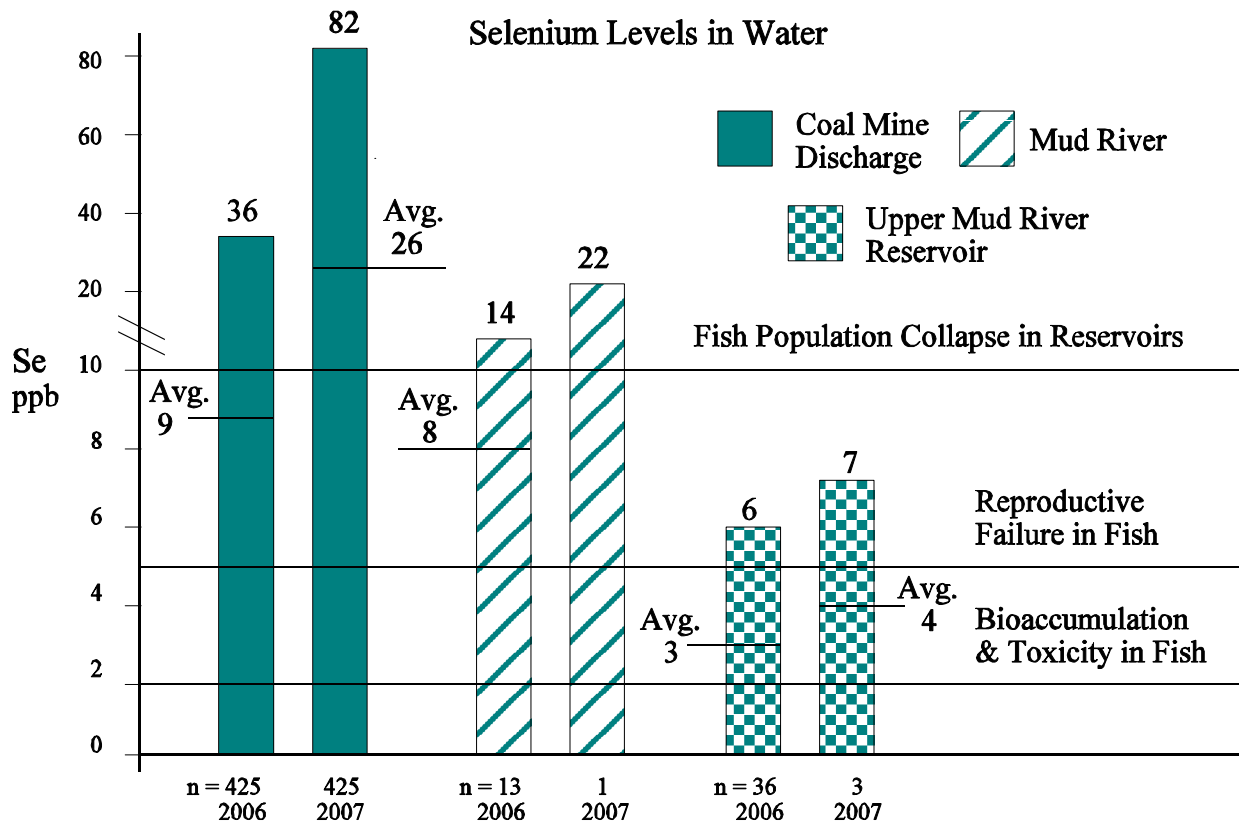


Figure 6. Selenium levels measured in water relative to concentrations that are toxic to fish.

Examining tissue residue data can further reveal the hazard of selenium. Concentrations of selenium in fish from Mud River exceeded the threshold for both Type 1 and Type 2 toxicity (4ug/g and 16 ug/g respectively) by 1.7-6.7 times. This exceedance indicates that the hazard of selenium in Mud River is high. Tissue selenium residues in fish from Mud River Reservoir were substantially greater than in the river, with concentrations exceeding the effects thresholds by 4-15 times. This suggests that greater bioaccumulation and food-chain transfer of selenium was taking place in the reservoir, which is consistent with patterns seen elsewhere in southern impoundments.

The magnitude of bioaccumulation and amount of exceedance of the toxic thresholds indicates that hazard in Mud River Reservoir is high.

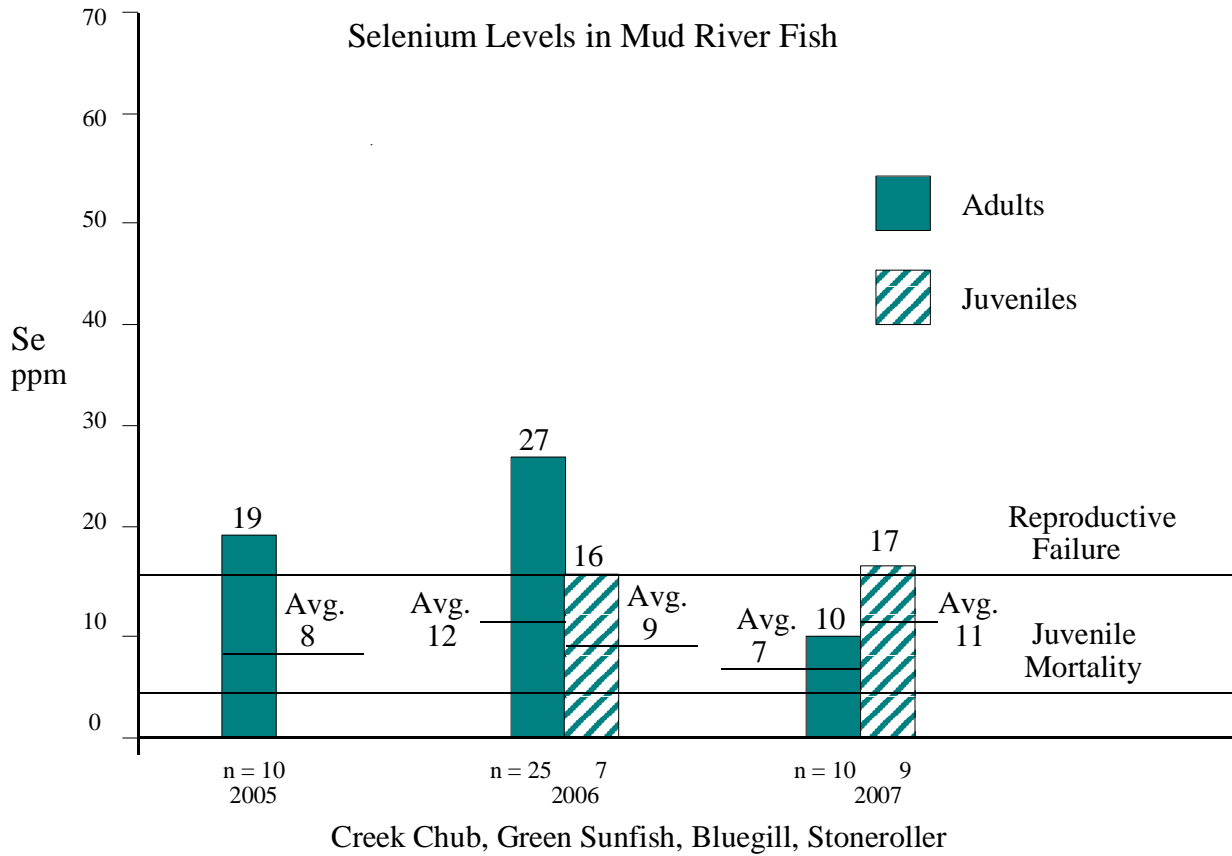


Figure 7. Selenium concentrations in Mud River fish relative to known toxic effect levels.

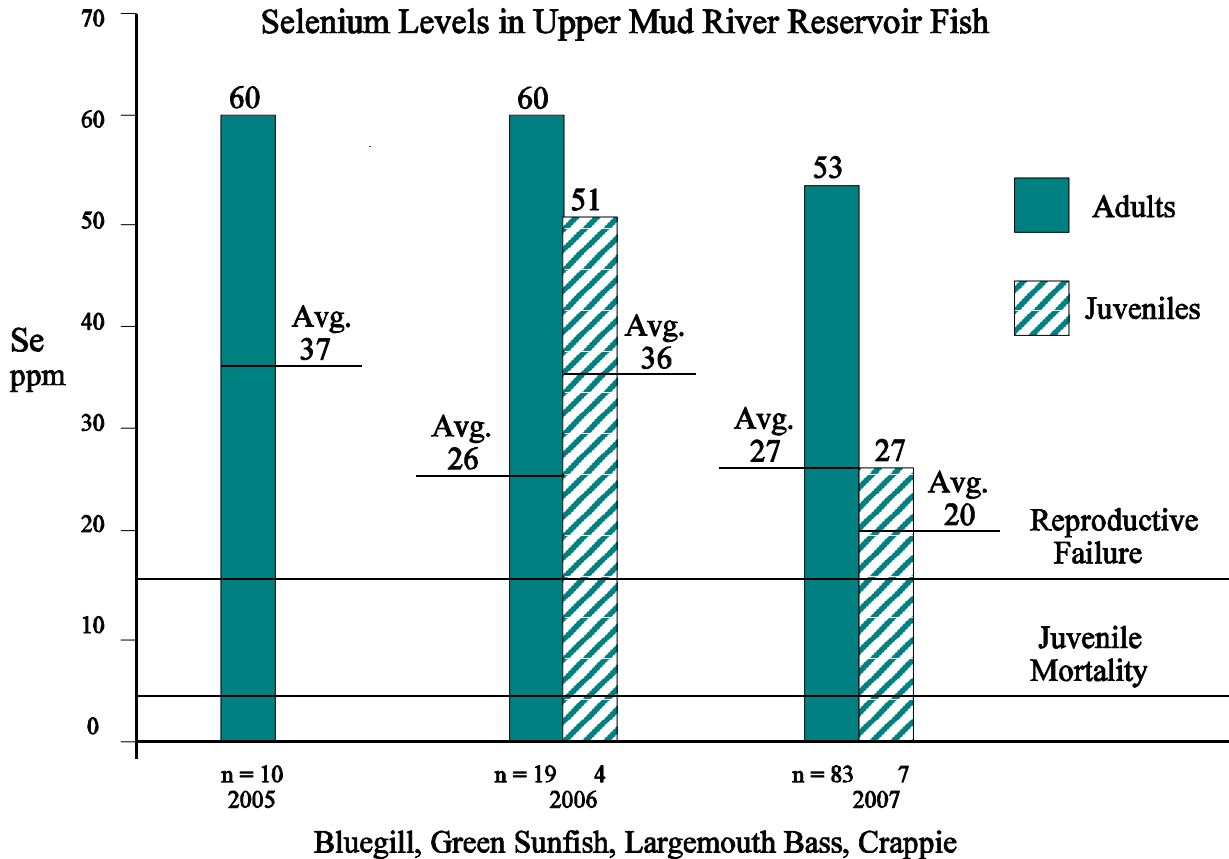


Figure 8. Selenium concentrations in Upper Mud River Reservoir fish relative to known toxic effect levels.

Another way to evaluate hazard is to examine residues in relation to the degree of effects one would expect to see for those tissue concentrations, rather than simply comparing them to a toxicity threshold. In this way, actual effects levels can be used as a check, or validation test, of hazard ratings that were based on exceedance of a lowest effect threshold. Fish collected from Mud River had residues up to 27 ug/g dw. Experimental studies show that this concentration in stream fish is associated with 60% reproductive failure and 15% adult mortality (Hermanutz et al.

1993), as well as significant physiological effects (Lohner et al. 2001a, 2001b, 2001c). This degree of effects supports a high hazard rating, which is consistent with the threshold exceedance rating. Fish in Mud River Reservoir had residues up to 60 ug/g. Experimental studies in southern impoundments show that this concentration in tissues is associated with 75% mortality of juveniles, complete reproductive failure in adults, and collapse of fish populations (Cumbie and Van Horn 1978, Finley 1985, Lemly 1985b, Gillespie and Baumann 1986). This degree of effects supports a hazard rating of high, which is consistent with the threshold exceedance rating. A final measure of hazard can be obtained by examining selenium residues in fish food organisms and comparing them to known toxic dietary levels. Invertebrates from Upper Mud River Reservoir contained up to 39 ug/g dw selenium. This exceeds the dietary toxicity threshold for both Type 1 and Type 2 toxicity in fish (4 and 13 ug/g dw respectively) by 3-10 times, indicating high hazard. Field and laboratory investigations have found that concentrations of 13-39 ug/g dietary selenium can cause 40-100% reproductive failure and 30-75% mortality of juvenile fish (Finley 1985, Woock, et al. 1987, Coyle et al. 1993, Lemly 1993a). This degree of effects supports a high hazard rating, which is consistent with the threshold exceedance rating.

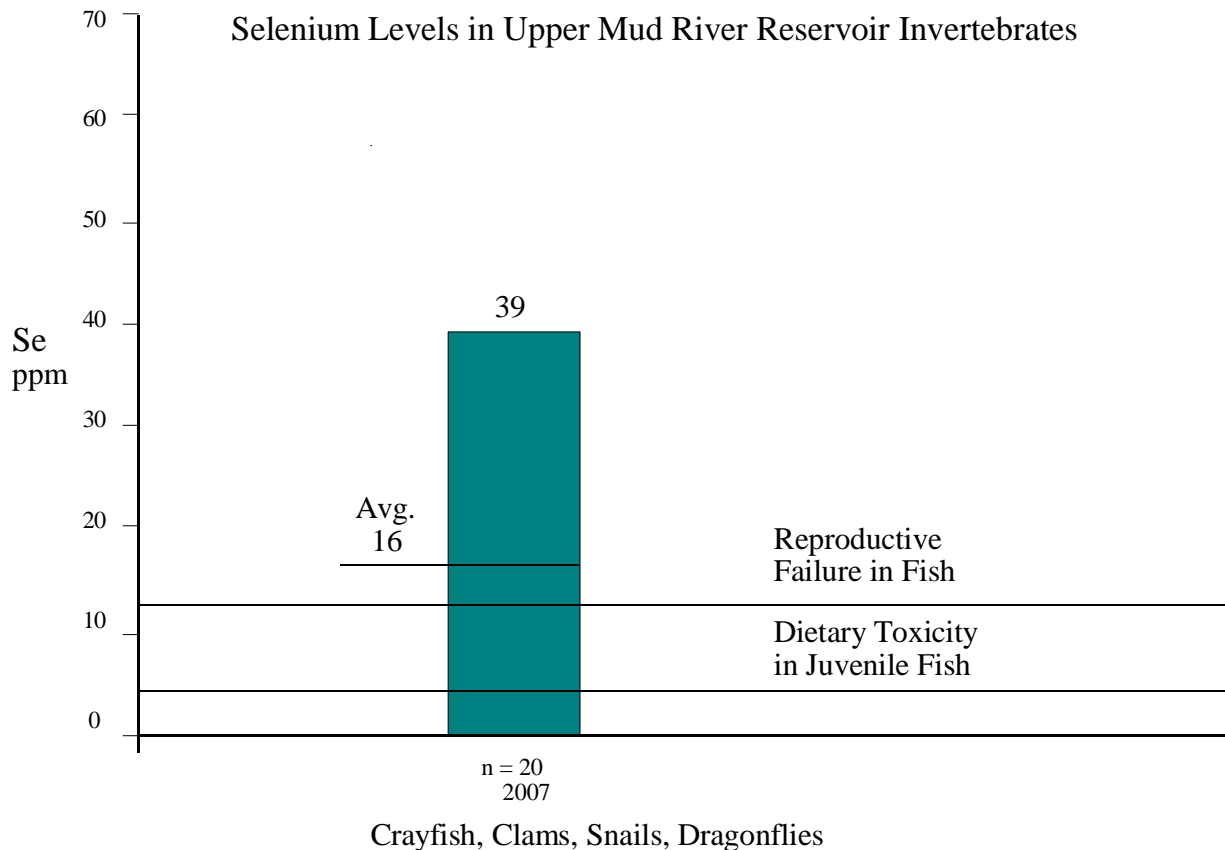


Figure 9. Selenium concentrations in fish food organisms relative to known toxic effect levels.

Are Toxic Effects Occurring in the Mud River Ecosystem?

In June, 2007, biologists from the West Virginia Department of Environmental Protection examined recently hatched fish collected from Upper Mud River Reservoir and found deformity frequencies as high as 35% in the samples (see Attachment 1). Actual photographs of these fish illustrate the normal condition (Figures 10-11) as compared to individuals having positive, tell-tale biomarkers of selenium toxicity (Figures 12-13), consisting of curvature of the spine (scoliosis, kyphosis), edema, and yolk-sac deformity (see also Figures 2, 3 & 4, above). These findings

confirm that selenium poisoning is taking place in the fish community of Upper Mud River Reservoir. Reproductive effects such as these would be expected given the selenium levels measured in adult fish. Photographs of larval fish published in research studies of selenium toxicity show that the outward appearance of edema and yolk-sac deformities in Mud River Reservoir match those resulting from selenium poisoning (Gillespie and Baumann 1986, Woock et al. 1987).



Figure 10. Dorsal view of normal fish larva from Upper Mud River Reservoir, June, 2007.

Note well developed pectoral fins and straight spine.



Figure 11. Side view of normal fish larva from Upper Mud River Reservoir, June 2007.

Note normal eye development, straight spine, and complete yolk absorption with no evidence of edema or a swollen, deformed yolk sac.



Figure 12. Dorsal view of abnormal fish larva from Upper Mud River Reservoir, June 2007.

Note deformed spine in “S” shape, typical of scoliosis due to selenium poisoning.

This individual also has deformed pectoral fins, which is another characteristic biomarker of selenium toxicity.



Figure 13. Side view of abnormal fish larva from Upper Mud River Reservoir, June 2007.

Note the distended, fluid-filled yolk sac (edema) with delayed yolk absorption. This individual also has dorso-ventral curvature of the spine (kyphosis) and deformed pectoral fins and eyes (both eyes are on the same side of the head). All of these abnormalities are characteristic biomarkers of selenium poisoning.

Conclusions

Based on comparisons between the concentrations of selenium found in West Virginia monitoring investigations and the known toxic effects of those concentrations as demonstrated in research studies of streams and reservoirs across the Southeast and Midwest, using the same fish species and environmental exposure conditions present in West Virginia, it is highly probable that a substantial amount of both Type 1 and Type 2 selenium poisoning is taking place in the Mud River ecosystem. This assessment is validated by the finding of selenium-induced deformities in newly hatched fish collected from Upper Mud River Reservoir in 2007. The Mud River ecosystem is on the brink of a major toxic event. If waterborne selenium concentrations are not reduced, reproductive toxicity will spiral out of control and fish populations will collapse. The warning signs are evident. If a catastrophic event is to be avoided, now is the time to take action.

I declare under penalty of perjury that the foregoing is true and correct.

Date: April 17, 2008



A. Dennis Lemly

Qualifications as an Expert Witness: see attached curriculum vitae

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APPENDIX A

CURRICULUM VITAE

A. Dennis Lemly, Ph.D.
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I have spent over 25 years investigating the effects of selenium pollution in aquatic ecosystems. I have extensive experience conducting field and laboratory research on selenium toxicology, primarily involving aquatic cycling, bioaccumulation, and effects on fish. These studies include intensive investigations of the two most substantial cases of selenium pollution that have taken place in the USA; (1) Belews Lake, North Carolina, where 19 species of fish were eliminated, and (2) Kesterson Marsh, California, where thousands of aquatic birds were poisoned. My career began in the late 1970's with studies of the landmark pollution event at Belews Lake, which established the fundamental principles of selenium bioaccumulation and reproductive toxicity in fish. In the 1980's, I was a research project manager for the U.S. Fish and Wildlife Service, directing studies that determined impacts of selenium from agricultural irrigation on aquatic life at Kesterson and in 14 other western states. In the 1990's, the emphasis of my research shifted to the development of methods and guidelines for hazard assessment and water quality criteria for selenium, which led to the publication of a reference book (see item 42 below). This handbook contains the first comprehensive assessment tools for evaluating selenium pollution on an ecosystem scale. I have consulted on selenium contamination issues around the world, including such problems as power plant discharges in Australia, gold mining effluents in Russia, agricultural irrigation drainage in Egypt, and landfill leachate in Hong Kong. I provide the methods and technical guidance necessary to identify, evaluate, and correct aquatic selenium problems before they become significant toxic threats to fish and wildlife populations. I have devised and applied techniques for protecting aquatic life in habitats from the Arctic to the tropics, and from high mountain streams to coastal lagoons. My selenium evaluation guidelines and water quality criteria recommendations have been adopted by the U.S. Environmental Protection Agency, the U.S. Fish and Wildlife Service, 23 states and over 60 nations and provinces around the world. I earned graduate degrees from Wake Forest University and I currently hold joint appointments with the USDA-Forest Service and the Graduate Faculty at Wake Forest University (Research Professor of Biology) and Virginia Tech University (Professor of Fisheries Science).

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PUBLICATIONS ON SELENIUM:

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