Biological Assessment to Determine Impacts of Selenium Pollution From Coal Ash Wastewater Discharges on Fish Populations in Lake Sutton, NC

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Background

Progress Energy owns and operates Lake Sutton NC as a cooling reservoir for the L.V. Sutton Steam Plant, a coal-fired electric generating facility. The lake is used as a disposal site for coal ash wastewater from the power plant and it is also open to the public for fishing. The NC Division of Inland Fisheries and/or Progress Energy have monitored fish populations and chemical pollution from coal ash in Lake Sutton since the late 1980's (NCDIF 2010, 2013). Since that time, a substantial amount of information has resulted showing that Lake Sutton is polluted by the trace element selenium. Selenium is a well-documented contaminant in coal ash wastewater and it can cause developmental abnormalities and reproductive failure in fish and wildlife (Lemly 2002a). Although monitoring results indicate that the concentrations of selenium in fish equal or exceed diagnostic toxic levels there has been no detailed biological investigation to determine if impacts are actually occurring. The Southern Environmental Law Center commissioned this study to answer that question. The assessment method used was developed by Dr. Lemly through his work on other selenium-polluted lakes in NC and is published in the peer-reviewed scientific literature (Lemly 1993, 1997a). It consists of examining young fish for the presence of selenium-induced defects and then applying an assessment index to evaluate impacts on the Lake Sutton fish community.

Fish Collection and Examination

Young fish were collected from Lake Sutton during May-September 2013 and preserved in denatured alcohol. Each preserved specimen was categorized according to its fish family and tallied prior to examination; Centrarchidae (bass, sunfish; CE), Cyprinidae (minnows; CY), Clupeidae (shad; CL), Poeciliidae (topminnows; PO), and unidentified (UI). A total of 1,434 fish from Lake Sutton were examined by Dr. Lemly under magnification and any physical abnormalities were noted and recorded. Types of abnormalities investigated included spinal curvature (kyphosis, lordosis, scoliosis), craniofacial defects (including mouth, jaws, and gill cover), fin irregularities (missing, misshaped, vestigial), eye abnormalities (including lens cataracts and exopthalmus), and edema (fluid accumulation and associated swelling. Photographic records were made of representative individuals that exhibited abnormalities

For comparison, young fish were also collected from a reference lake that does not exhibit elevated selenium levels (High Rock Lake, Davidson County, NC) during May-July 2013 and preserved in denatured alcohol. Each preserved specimen was categorized according to its fish family and tallied prior to examination; Centrarchidae (bass, sunfish; CE), Cyprinidae (minnows; CY), Poeciliidae (topminnows; PO), and unidentified (UI). A total of 1,000 fish from the reference lake were examined by Dr. Lemly for abnormalities.

Results

Morphological abnormalities

Fish from Lake Sutton were examined in two batches and consisted of distinctly different sizes and species of fish, and exhibited a markedly different frequency of physical abnormalities. The first batch, collected in May-August, consisted of 939 individuals composed as follows:

639 shiner minnows (Cyprinidae), none of which exhibited abnormalities

100 shad (Clupeidae), none of which exhibited abnormalities

163 large bluegill, other Lepomis, and bass (Centrarchidae; > 6 cm long), none had deformities

35 small bluegill and other Lepomis (Centrarchidae; < 6 cm long), 4 had deformities (11.76%)

2 small crappie (Centrarchidae; < 6 cm long), 1 of which exhibited deformities (50%)

All of the deformities were skeletal, affecting the craniofacial structure. However, none of the 5 deformed individuals exhibited multiple malformations, that is, both craniofacial and spinal or other abnormalities. A complete record of the abnormality tally for all 939 fish in the first batch is presented in Appendix 1. Photographic documentation of the deformities is presented in Figures 1-4.

The second batch of fish, collected in September, consisted of 494 small (< 6 cm long) bluegill and other Lepomis, and one mosquitofish (Gambusia affinis).

494 small bluegill and other Lepomis (Centrarchidae; < 6 cm long), 148 were deformed (29.95%)

1 mosquitofish (Poeciliidae) and it was deformed (100%)

Combined total for all centrarchids < 6 cm long from Lake Sutton = 531 individuals, 153 were deformed (28.81%)

With one exception (a minor fin deformity) all of the 149 deformities were skeletal and affected the spine and/or craniofacial structure. 10 of the deformed individuals exhibited multiple malformations consisting of both craniofacial and spinal abnormalities. A complete record of the abnormality tally for all 495 fish in the second batch is presented in Appendix 2. Photographic documentation of representative deformities is presented in Figures 5-16.

Fish from High Rock Lake, the reference lake, were examined in one batch and consisted of 1,000 individuals composed as follows:

722 small bluegill, other Lepomis, and bass (Centrarchidae; < 6 cm long), 5 exhibited minor fin irregularities (0.69%)

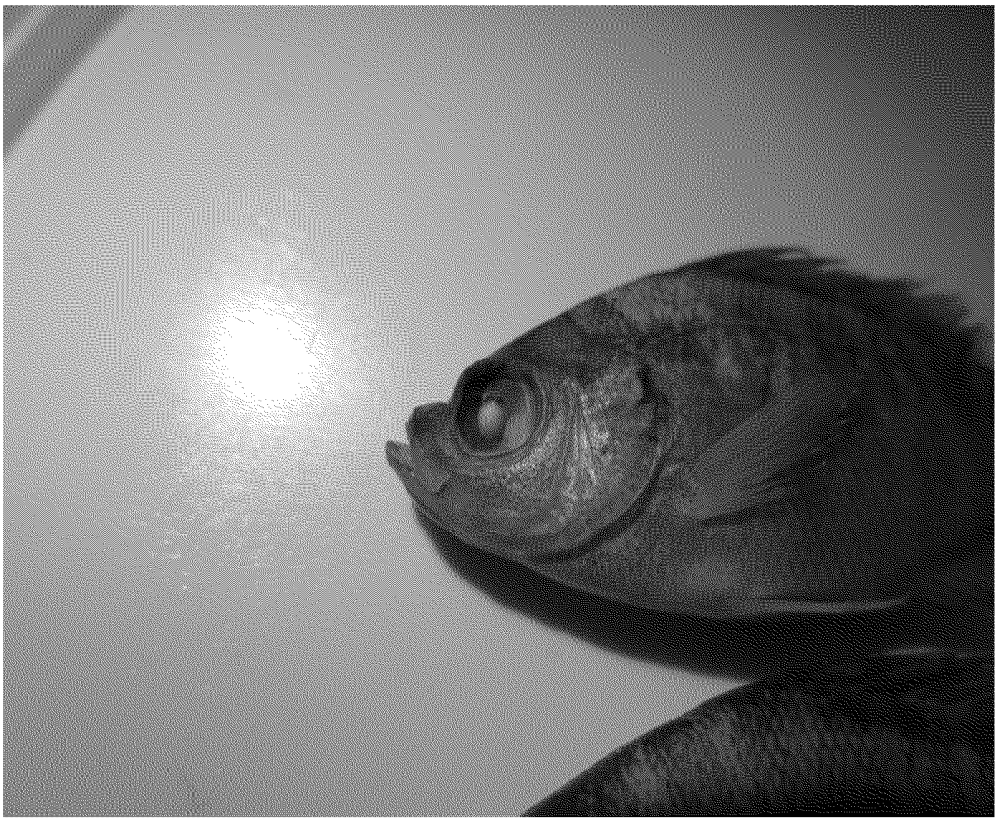
212 minnows (Cyprinidae), 3 exhibited minor fin irregularities (1.41%)

56 mosquitofish (Poeciliidae), none of which exhibited abnormalities

10 unidentified (UI), 1 of which exhibited minor fin irregularities (10%)

All of the abnormalities in fish from the reference lake consisted of minor fin irregularities, none were skeletal. A complete record of the abnormality tally for all 1,000 reference fish is presented in Appendix 3.

Figure 1. Two abnormal bluegill (Lepomis macrochirus) from Lake Sutton (first collection batch) showing the craniofacial defect known as "pugnose". This condition is characterized by deformed "underbite" mouth and jaws, and compressed or shortened head and mouth due to distorted cranial skeletal structure and gill cover. The bottom individual is normal.



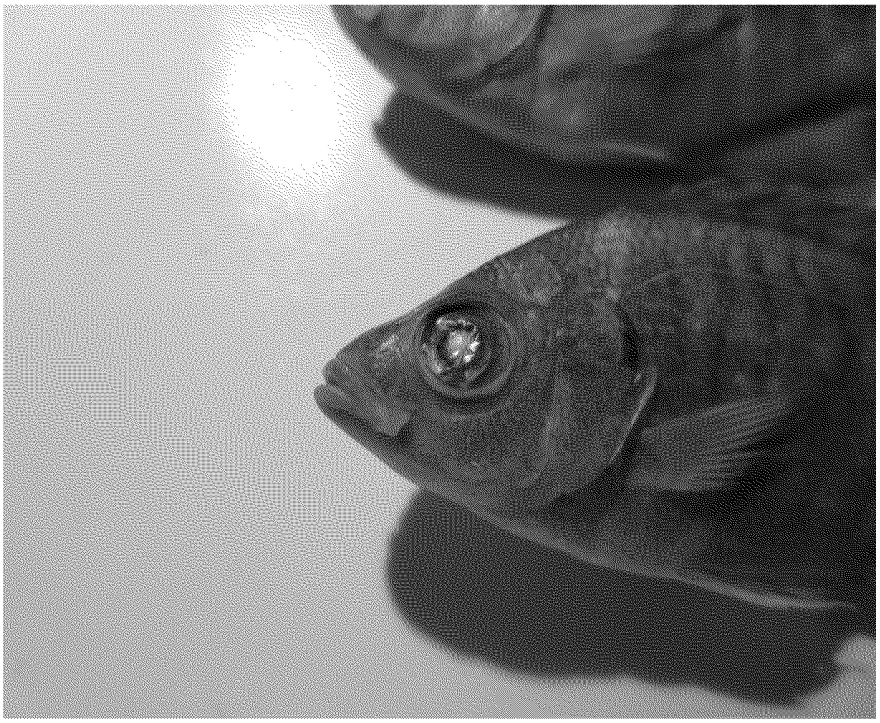


Figure 2. Comparative head features of a “pugnose” and normal bluegill from Lake Sutton.

Figure 3. Normal (bottom) and “pugnose” (top) crappie (Pomoxis sp.) from Lake Sutton (first collection batch).

Figure 4. Photo showing all deformed individuals from the first collection batch. All exhibited the same "pugnose" craniofacial abnormalities.

Figure 5. Abnormal bluegill (Lepomis macrochirus, top) from Lake Sutton (second collection batch) showing multiple defects of the mouth (which is permanently distended and less than 20% of its normal size) and other craniofacial structures including "gaping" distorted, permanently deformed gill cover. Bottom individual is normal.

Figure 6. Photo showing additional head detail of individuals in Figure 5.

Figure 7. A "pugnose" bluegill (Lepomis macrochirus, top) from the second collection batch in Lake Sutton. Bottom individual is normal.

Figure 8. Photo showing additional head detail of individuals in Figure 7.

Figure 9. Abnormal bluegill (Lepomis macrochirus, top) from Lake Sutton (second collection batch) showing deformities of the lumbar and caudal regions of the spine (kyphosis and lordosis). Bottom individual is normal.

Figure 10. Photo showing additional detail of individuals in Figure 9.

Figure 11. Abnormal bluegill (Lepomis macrochirus, top) from Lake Sutton (second collection batch) exhibiting spinal kyphosis and lordosis. Bottom individual is normal.

Figure 12. Photo showing additional detail of individuals in Figure 11.

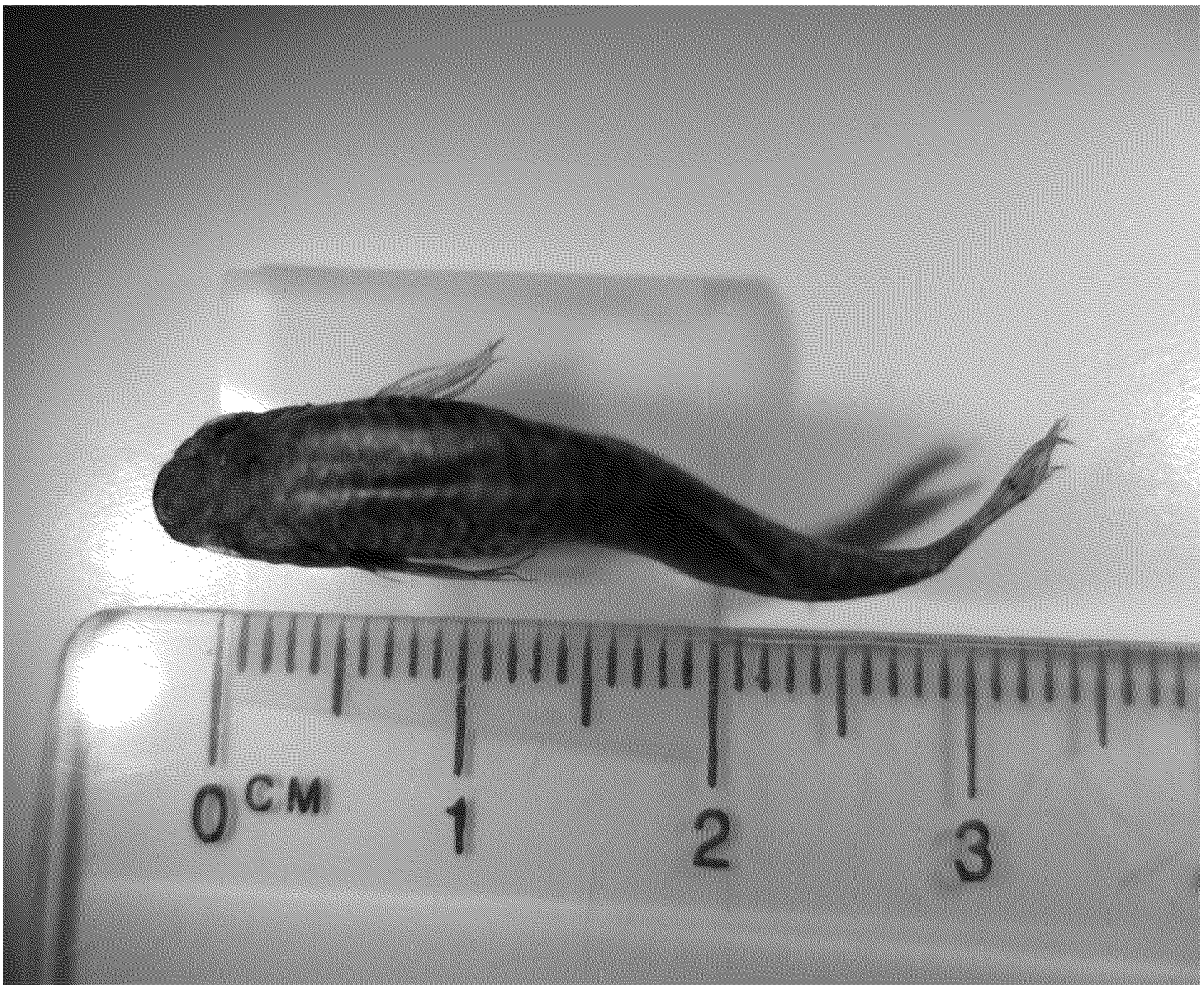


Figure 13. A deformed mosquitofish (Gambusia affinis) from Lake Sutton (second collection batch) exhibiting lateral curvature of the spine (scoliosis).

Figure 14. A group of bluegill (Lepomis macrochirus) from Lake Sutton (second collection batch) exhibiting a spinal deformity that causes downturned caudal fins due to malformed vertebrae and fin rays.

Figure 15. Another group of bluegill (Lepomis macrochirus) from Lake Sutton (second collection batch) exhibiting caudal deformity due to spinal defects.

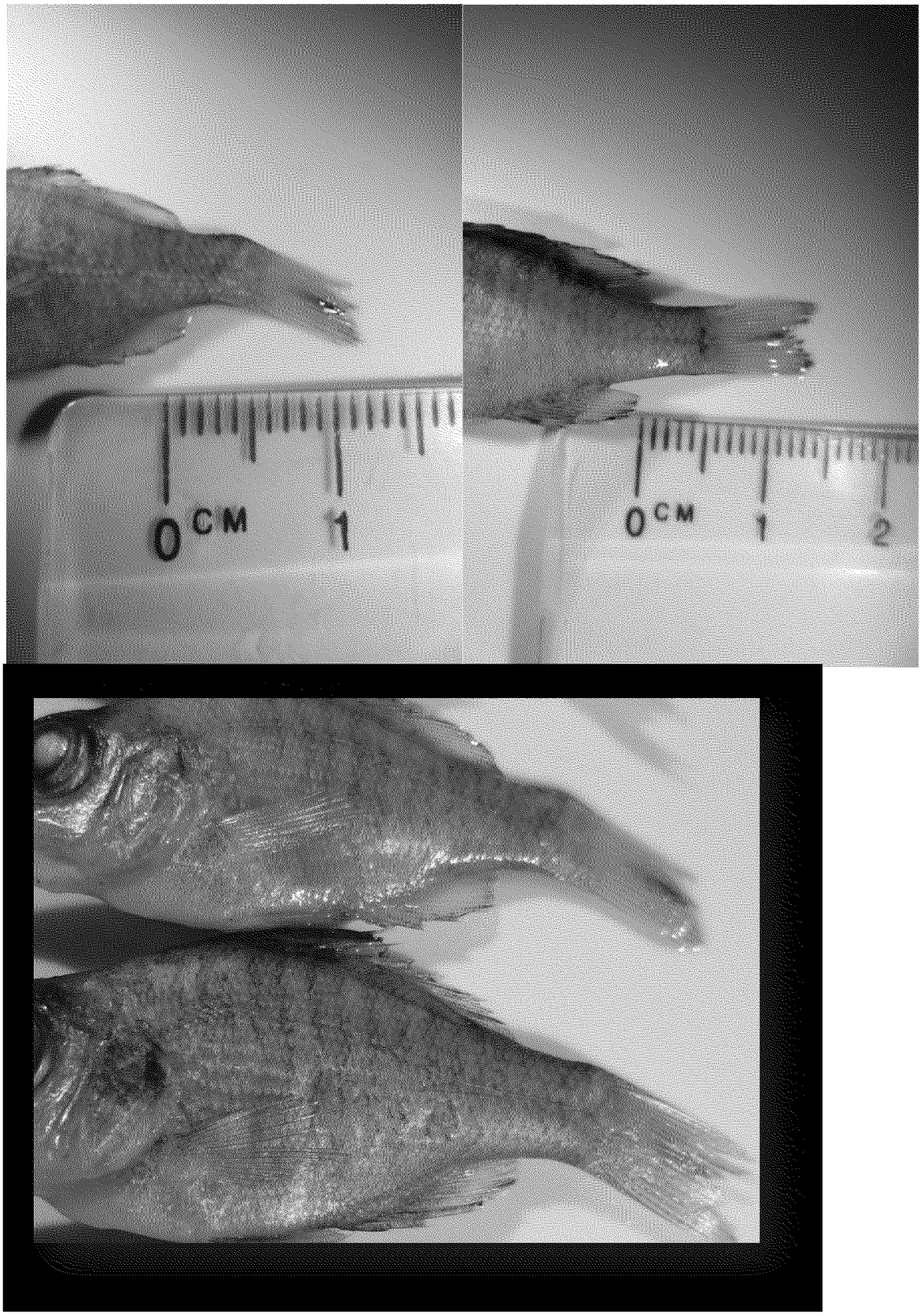


Figure 16. Additional examples of caudal deformities in bluegill (Lepomis macrochirus) from Lake Sutton (second collection batch). Individual in upper right photo is normal.

Teratogenic Deformity Index (TDI)

All species of fish collected from the reference lake exhibited a low incidence of abnormalities (<1.5%) which is consistent with background conditions and normal selenium levels. However, some species collected from Lake Sutton expressed symptoms consistent with elevated selenium and associated toxic responses manifest in deformed skeletal features, particularly the spine and craniofacial region. Only the Lepomis species group (consisting primarily of bluegill and redear sunfish) produced appropriate size, age, and number of individuals for TDI assessment (< 6 cm long juveniles, 200 individuals; Lemly 1997a). Deformity rates were markedly elevated in this group (28.81%). Applying the TDI method prescribed by Lemly (1997a) yields a TDI of 7.20% teratogenic mortality (28.81% deformed X 25% mortality for juvenile fish. Note that a mortality rate of 25% is used because only severe deformities of the spine and craniofacial structure are considered to be lethal, which on average, amounts to about 25% of total deformities in a selenium-impacted fish population. Other, less severe deformities such as minor fin abnormalit ies may generally not be lethal and are not factored into teratogenic mortality. TDI thus yields a conservative estimate of mortality and population-level impacts.). The final assessment is 71.19% surviving normal fish + 21.61% surviving teratogenic fish = 92.8% total survivors. Total teratogenic mortality is thus estimated at 7.2%, which is categorized by a Level 2 TDI rating (Lemly 1997a, 2002b). A Level 2 TDI rating means that selenium pollution from coal ash is poisoning enough individuals to have a negative impact on the population of bluegill and other Lepomis in Lake Sutton. However, there are other aspects of poisoning that are taking place as well. Even within the single group that could be assessed (Lepomis), it is not correct to interpret/infer that morphological abnormalities and TDI ratings are the "total impact". Externally visible deformities are reliable biomarkers of selenium poisoning but they are just a symptom of a much larger underlying problem. Only impacts resulting from teratogenic mortality can be estimated by TDI, not total mortality/impacts. This is because a large part of selenium's toxicity is expressed just before or soon after hatching. These effects have been well studied in NC lakes and are reported in the scientific literature (e.g., Gillespie and Baumann 1986, Woock et al. 1987). Embryo mortality and post-hatch toxicity prior to swim-up of larval bluegill typically range from 25-100% for individual spawns when the selenium concentration in eggs is at the levels reported from Lake Sutton (Table 1). This mortality could easily be double, triple, or even quadruple the observed teratogenic mortality (7.2%) assessed from deformities that persist in the 2-5 cm length stage. Therefore, it is scientifically and toxicologically reasonable to estimate that the total selenium impact on the bluegill/Lepomis group in Lake Sutton is currently in the range of 20-30% total population mortality per year.

Monetized value of toxic impacts

Loss of fish due to toxic effects of water pollution imparts several well recognized and calculable economic costs. These costs may include ecological, recreational, commercial, subsistence, property value, and aesthetic components (Kopp and Smith 1993). It is important to understand that each fish carries multiple values and when that fish is lost, all of those values are lost. Thus one must calculate and add all the cost components together to arrive at the true and full monetized negative cost impact (Gentner and Bur 2009). For the present investigation, three of the principal components of cost were calculated and added: (1) replacement value, (2) recreational sport value, and (3) food/subsistence value. Replacement cost can be determined by multiplying the number of fish poisoned times the monetized value of an individual fish in terms of its physical replacement cost, that is, the cost to spawn and grow (via hatchery), collect (via field sites), or otherwise obtain and stock a replacement fish (USFWS 1995). Those costs were calculated for lost bluegill/Lepomis in Lake Sutton. The first step in this process was to use catch records from the two most recent Progress Energy monitoring reports (Progress Energy 2011, 2012) to determine the relative abundance of bluegill/Lepomis in the total fish population, which indicates an average of 63.5%. The next step was to estimate the total fish standing crop, which was done using catch per unit effort electrofishing results from Lake Sutton combined with fisheries monitoring information from other NC lakes (Lemly and Skorupa 2012, supporting information file). Total fish standing crop estimates for Lake Sutton range from 34,050 individuals per hectare to 54,470 per hectare, or an average of 44,260 X 63.5% = 28,105 bluegill/Lepomis per hectare. The total standing crop of the bluegill/Lepomis species group in Lake Sutton is thus estimated at 28,105 individuals per hectare X 445 hectares = 12,506,725. A teratogenic mortality rate of 7.2% translates to a loss of 900,484 individuals from the bluegill/Lepomis species group annually. The replacement cost for these lost fish is calculated from NC DENR figures (NCAC 1993) which gives a dollar amount of $5 for each individual bluegill/Lepomis ("Sunfish – All Sizes - $5/fish"). The resultant dollar cost of poisoned fish amounts to $4,502,420 for the 2013 sampling year. It is scientifically and toxicologically reasonable to expect that poisoning rates were similar in preceding years when tissue selenium concentrations were at or above current levels. Monitoring data shows that those levels were present in all but 2 (1989 and 1992) of the previous 26 years for which records are available, going back to 1987 (Table 1; NCDIF 2013). The cumulative cost of selenium poisoning on the bluegill/Lepomis group in Lake Sutton is calculated to be $4,502,420 per year X 25 years, or $112,560,500.

The other two aspects of cost which were included are the recreational sport value and the food value of fish that have been poisoned, but would have been present and reached catchable size for sport enjoyment by recreational anglers and consumption by both anglers and subsistence fishermen (there is significant subsistence use of Lake Sutton fish; SELC 2013).

Those costs were calculated by combining the recreational value of lost harvestable size fish with the food value of those harvestable size fish (Lemly and Skorupa 2012). Approximately 10% of a fish population is expected to mature to harvestable/edible size, which results in a catchable/edible population of bluegill/Lepomis in Lake Sutton of 1,250,672 (12,506,725 X 10%). A total of 90,048 harvestable/edible individuals would be expected to be missing from the population due to teratogenic mortality (1,250,672 X 7.2%). The dollar value of missing angler/sport fish is $5 X 90,048 = $450,240 annually; the dollar value of missing food/subsistence fish is $7.50 (1 fish per meal) X 90,048 = $675,360 annually; total = $1,125,600 annually. As with replacement cost, adding the damage costs expected in previous years yields a total of $1,125,600 X 25 = $28,140,000 in cumulative monetized losses.

The total monetized value for fishery losses due to selenium poisoning in Lake Sutton is calculated to be $5,628,020 in 2013 for the bluegill/Lepomis species group, and the total cumulative losses for this group are valued at $140,700,500 for the period 1987-2013. However, this value is based on teratogenic mortality alone. As discussed in the TDI section, total mortality of bluegill/Lepomis is projected in the 20-30% range. The resultant value of losses would be correspondingly increased, which means that annual monetary losses under existing pollution levels could amount to as much as $22,512,080, and the aggregate loss value for the period would be approximately $562,802,000. Moreover, this figure does not include the value of losses to other important species such as largemouth bass (Micropterus salmoides), catfish (Ictalurus sp.), crappie (Pomoxis sp.), and carp (Cyprinus carpio), which were not collected in sufficient numbers to assess impacts.

Table 1. Historical and recent concentrations of selenium in centrarchid fishes (bluegill andother Lepomis, and bass Micropterus sp.) from Lake Sutton relative to known toxic thresholds*

Year	Mean Tissue Se ug/g dw				Toxic Thresholds
	Liver	Egg	Muscle	Whole-body	(Lemly 2002b)
1987	105.5	86.5	15.7	26	Liver = 12 ug/g dw
1989	NA	24	4.4	7.5	Egg = 10 ug/g dw
1991	14.5	59	10.7	18	Muscle = 8 ug/g dw
1993	28.5	102	18.5	31	Whole-body = 4 ug/g dw
1995	69	99	18	30	
1997	75.5	124	22.5	37.5	
1999	30	127	23	38.5	
2001	30	104.5	19	31.5	
2003	30	77	15.5	23	
2005	25.5	91	16.5	27.5	
2007	27	79	18	27.5	
2010	24	NA	17.5	NA	
2011	24	NA	17	NA	

*1987-2007 data were extracted from NC Division of Inland Fisheries summary for selenium in Lake Sutton (NCDIF 2013); 2010-2011 data are from Progress Energy 2011 and 2012 environmental monitoring reports.

NA = not analyzed

Largemouth Bass

The largemouth bass is a key ecological, recreational, and human subsistence-use species in Lake Sutton. Recent fisheries monitoring by Progress Energy and the North Carolina Division of Inland Fisheries shows that the population of "catchable" size bass has declined by over 50% since 2008 (NCDIF 2010, Progress Energy 2010). Also, fish body condition (one measure of fish health) is considerably lower in Lake Sutton than nearby rivers and impoundments. Finally, size distribution data show that there are very few small individuals (<10 cm) present in the population, coupled with unusually high mortality of adult bass and unstable population structure (NCDIF 2008, 2010). These circumstances indicate that, on the whole, largemouth bass are not doing well in Lake Sutton. Selenium toxicity has been suspected as a contributing factor for many years (NCDIF 2008). That is a very reasonable suspicion. Studies of other selenium-polluted power plant lakes in NC show that largemouth bass are very susceptible to selenium poisoning and they are one of the first species to be detrimentally affected due to selenium-induced reproductive impairment, as well as bluegill and other Lepomis (Cumbie and Van Horn 1978, Birchfield et al. 1983, Lemly 1985, Baumann and Gillespie 1986). Multiple sampling efforts made in the present study failed to produce a single bass less than 6 cm long. Due to lack of small individuals, it was not possible to assess selenium-induced teratogenic deformities but selenium concentrations in bass tissues were twice the toxic threshold for reproductive effects in 2010-2011 (Table 1). The rarity of small bass is, in itself, strongly suggestive of selenium toxicity in a coal ash--influenced lake (Birchfield et al. 1983). The toxicological response profiles of bluegill and bass to selenium exposure in both laboratory tests and real-world field conditions are very similar in terms of teratogenic deformities and reproductive effects. It is therefore scientifically reasonable to expect that the impact of selenium on the bass population in Lake Sutton is at least as great as it is on the bluegill/Lepomis species group.

Discussion and Conclusions

The occurrence of spinal, craniofacial, and other skeletal abnormalities in combination with elevated tissue levels of selenium confirms that teratogenic selenium poisoning is taking place in the fish community of Lake Sutton. The types of deformities seen are classic biomarkers of selenium toxicity (Lemly 1993, 2002a, 2002b, Janz et al. 2010). The response of fish in terms of expressed frequency of abnormalities is consistent with the differential sensitivity of fish to selenium toxicity, with centrarchids (bluegill/Lepomis, crappie, bass) being among the most sensitive and typically exhibiting the most deformities (Lemly 1993). It is important to note that no largemouth bass of appropriate size (i.e., small enough), and insufficient numbers of crappie (Pomoxis), were collected for analysis. Consequently, no scientifically valid assessment and

interpretation of teratogenic mortality and associated economic cost valuations could be made for these major ecological, recreational, and human subsistence species. It is therefore very likely that the results given in this report substantially underestimate total fisheries impacts and associated monetized costs. Nevertheless, the losses taking place each year in the Lake Sutton fishery due to coal ash impacts on the bluegill/Lepomis species group alone are substantial and important ecologically and economically.

It is prudent to consider current ecological conditions in relation to the future of Lake Sutton and the L.V. Sutton plant, which will possibly include termination of coal-fired operations coupled with dilution/reduction/termination of coal ash wastewater flow into the lake. The persistence of toxic impacts on fish after cessation of selenium-laden wastewater inputs may be considerable and protracted, as other lakes in NC have shown (Lemly 1997b). Given the magnitude of sediment-associated selenium contamination currently in Lake Sutton (21-28 ug/g reported in 2011 samples, Progress Energy 2012; toxic threshold = 2 ug/g, Lemly 2002b) there is a huge repository of selenium that is biologically available to enter the benthic food chain and, thereby, the diet of fish (Lemly and Smith 1987). Once incorporated into the aquatic food chain, sediment-associated selenium is no different toxicologically, and is of no less biological hazard to fish, than the waterborne source which initially carried selenium into the lake. Thus, biological impacts can persist as if the power plant was still operating. Natural sedimentation rates combined with microbial volatilization and biological removal into macroorganisms (insects, annelids, crustaceans, mollusks, etc.) typically results in about a 1-2% attenuation of sediment selenium concentrations per year (Frankenberger and Engberg 1998, Lemly 2002b). Taking the upper limit of this range means that, at best, the sediment associated selenium in Lake Sutton would continue to equal or exceed the fish toxicity threshold for another 46 years following complete termination of waterborne selenium inputs. Associated negative impacts on the fishery, recreational anglers, and subsistence fishermen would also be expected to persist for that length of time as well.

The results of this investigation present a stark contrast to a recent conclusion on fish community health made by Progress Energy (2012) "no deformities were observed or reported from the lake during 2011". This statement apparently stemmed from the results of casual observations made by biologists who collected fish for the 2012 Progress Energy monitoring report coupled with lack of any voluntary reports of fish deformities made by anglers. There are important flaws with this blanket comment by Progress Energy which implies that pollution-related abnormalities were non-existent. First, no targeted investigation was made to assess deformities by specific examination of appropriate numbers and age/size classes of fish. Second, casual observation by biologists or the public would likely only detect overtly noticeable selenium-induced deformities (that is, twisted spines, deformed mouth and head, etc.). These severely deformed individuals are almost always eliminated before they reach a

size typically caught and inspected by recreational anglers or survey biologists because of direct selenium toxicity or through increased vulnerability to predation due to impaired swimming ability or erratic behavior (Lemly 1993, 2002a). It would indeed be unusual for a biologist or sportsman to see and report an overtly noticeable selenium-induced deformity in an adult fish where predatory species exist. The comment in the Progress Energy report reveals no scientifically valid information about what is actually happening with respect to selenium-induced deformities and associated population-level impacts in Lake Sutton fish.

Even though the results of this investigation reveal important and rather striking biological information on deformities and associated fishery impacts they merely scratch the surface with respect to total selenium toxicity in Lake Sutton. First, no assessment was possible for bass, crappie, carp, catfish, and other important recreational and subsistence fish species. Second, as discussed in the section Teratogenic Deformity Index, even within the single group that could be assessed (Lepomis), it is not correct to interpret/infer that morphological abnormalities and TDI ratings are the "total impact", which could easily be several times the 7.2% teratogenic mortality level. Third, as discussed in the section on largemouth bass, the degree of toxicological similarities between bluegill and bass means that is reasonable to expect that similar losses are taking place in bass as well. Fourth, the fact that NC DENR/DIF has stocked fish on occasion, for example, Florida strain largemouth bass and channel catfish (Hotz, A. 2009, Marsh 2013), and that connection of Lake Sutton with the Cape Fear River and tributary creeks provides a conduit for immigration of fish into Lake Sutton (Progress Energy 2011, 2012, Marsh 2012), could obscure what would otherwise be a much greater impact of reduced reproductive success and teratogenic mortality on fish populations Lake Sutton.

As a final point, information from the latest Progress Energy reports (Progress Energy 2011, 2012) is very useful in evaluating the adequacy of NC state and EPA national selenium criteria. The monitoring data from those reports combined with toxicological assessment from the present study confirms that current standards do not protect fish from selenium poisoning in Lake Sutton. The measured average waterborne concentration of selenium in Lake Sutton was 3.5, 3.1 and 2.7 ug/L in 2009, 2010, and 2011, which is well below the EPA national and NC state criterion of 5 ug/L. However, markedly elevated selenium levels were found in fish tissues each year, far exceeding toxic thresholds, and up to 28.81% deformities were present in the fish in 2013. These toxic impacts resulted from approximately 2.7 ug/L waterborne selenium. Clearly, a tightening of the waterborne selenium standard is warranted for Lake Sutton. Biological/toxicological assessment of the Lake Sutton fishery suggest that an ecologically appropriate standard should be in the 1.5-2.0 ug/L range, as has been recommended in the scientific literature (e.g. Peterson and Nebeker 1992, Lemly and Skorupa 2007).

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