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Critical Review of Proposed Residue-Based Selenium Toxicity Thresholds for Freshwater Fish

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ABSTRACT

Proposed fish toxicity thresholds for interpreting the biological significance of selenium concentrations measured in environmental media include 2 to 5 μg/L in water, 4 mg/kg dw in fish whole body tissue, 10 mg/kg dw in fish ovaries, and 3 mg/kg dw in fish diets. Use of these thresholds would likely identify fish populations as being at risk at numerous sites across the U.S. However, selenium effects on fish populations in the field have only been conclusively demonstrated at a few locations. Based on our critical review, these threshold values are not consistent with USEPA methodology for deriving criteria, in many cases are not supported by the scientific literature, and, as a result, are generally overly conservative. Based on currently available information, we believe the scientific literature is not supportive of generic sediment or water thresholds, but is supportive of alternative separate whole body thresholds of 9 mg/kg dw for warmwater fish and 6 mg/kg dw for larval coldwater anadromous fish, an ovary threshold of 17 mg/kg dw for warmwater fish, and fish dietary thresholds of 10 and 11 mg/kg dw for warmwater fish and larval coldwater anadromous fish, respectively.

Key Words: tissue, fish, toxicity, guidelines, selenium

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INTRODUCTION

Selenium is a metalloid element that historically has been of interest due to its potential toxicity to livestock and its necessity as a nutritional supplement in livestock feed in certain areas of the United States (Rosenfeld and Beath, 1946; 1964). Inclusion of selenium in the list of 65 priority pollutants (subsequently expanded to 129) in the mid-1970s provided greater focus in developing aquatic toxicology data for derivation of a selenium water-quality criterion (Adams, 1976; Cardwell *et al.*, 1976; Halter, Adams, and Johnson, 1980; Adams and Johnson, 1981). The first acute criterion recommended by the USEPA for freshwater organisms was 35 μg/L (a chronic criterion was not recommended). The most recent acute and chronic criteria set by the USEPA for protection of freshwater fish and invertebrates are 20 and 5 μg/L, respectively (USEPA, 1987).

These latest criteria were derived based on field studies of fish populations in Belews Lake, North Carolina (Cumbie and Van Horn, 1978). Belews Lake was receiving fly ash from a coal-burning electric power facility that increased selenium concentrations in the lake. Cumbie and Van Horn (1978) observed effects on fish populations at selenium concentrations as low as 10 μg/L, whereas populations in one portion of the lake appeared unaffected at <5 μg/L (USEPA, 1987). Hence, the basis for the current freshwater chronic criterion of 5 μg/L. Since the Belews Lake studies were reported, selenium poisoning of fish populations has been documented at a few additional locations around the U.S., including Hyco Reservoir in North Carolina and Kesterson Reservoir in California.

Selenium is unusual relative to most other metals and metalloids in that many inorganic and organic forms occur in the aquatic environment, and each form is differentially bioavailable and toxic to aquatic organisms. The selenium forms present in an aquatic system are driven by the biogeochemical cycling of selenium that is strongly controlled by site-specific environmental factors such as redox, pH, and biological productivity (Lemly and Smith, 1987; Bowie and Grieb, 1991; Porcella *et al.*, 1991).

Reduction of inorganic selenium species tends to immobilize selenium in an aquatic system, while other processes, such as oxidation and biotransformation, tend to make selenium bioavailable to aquatic organisms. Biological mechanisms such as uptake of sediment selenium by rooted plants, benthic invertebrates, and detritus-eating invertebrates, can act to remobilize selenium into the aquatic food web. Accordingly, lentic systems tend to bioaccumulate selenium much more than lotic systems that have higher flushing rates and lower productivity (Lillebo *et al.*, 1988; Van Derveer and Canton, 1997). For example, Lillebo *et al.* (1988) demonstrated this by plotting bioaccumulation data for impounded and flowing waters; fish selenium residues were approximately six times greater in impounded waters than in flowing waters at a water selenium concentration of 10 μg/L.

Based on increased awareness of the ecotoxicological effects of selenium, a number of water-quality monitoring programs have been implemented to

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evaluate potential selenium contamination at freshwater sites. In order to interpret the significance of the selenium concentrations measured under these programs, several authors have proposed selenium guidelines for various environmental compartments (*e.g.*, Lemly, 1993a; Skorupa, Morman, and Sefchick-Edmonds, 1996). Specifically, guidelines have been proposed for surface water, sediment and various tissues, including ovaries, whole body, diet, liver, eggs, and testes based on the authors' reviews of published and unpublished literature.

These guidelines include recommended toxicity thresholds for abiotic (water, sediment) and biotic (various fish tissues and diet) compartments. In this paper we focus only on the toxicity thresholds for tissues. Given the site-specific factors that influence selenium bioavailability, bioaccumulation, and toxicity in aquatic systems, we feel that proposal of a single guideline value for selenium in surface waters or sediments is inappropriate. Different sites will require different water or sediment selenium concentrations to ensure that concentrations in tissues such as fish ovaries do not exceed a toxic threshold. Site-to-site variability has been demonstrated for fish by Van Derveer and Canton (1997) and for birds by Adams *et al.* (1998). Van Derveer and Canton (1997) used a sediment-based bioaccumulation model to demonstrate that fish in a lotic system in Colorado were not at risk at water selenium concentrations of approximately 30 µg/L, three times higher than concentrations at which effects were observed in Belews Lake. Adams *et al.* (1998) used a statistical bioaccumulation model based on selenium data for bird eggs and surface waters at 17 sites in the U.S.; the 90th and 10th percentile water selenium concentrations associated with a selenium concentration of 20 mg/kg dw in bird eggs ranged from 6.8 to 318 µg/L in their model. Both of these studies support site-specific water quality guidelines for selenium based on a bioaccumulation model that estimates selenium concentrations in the critical tissues for toxicological effects.

For a water selenium threshold to be appropriate it must be based on a bioaccumulation model that accounts for the site-specific factors that influence selenium cycling and bioavailability to aquatic organisms. An effective water quality criterion must be based on aqueous selenium concentrations that are sufficiently low to prevent accumulation in fish food organisms, which in turn would result in the accumulation of selenium to high enough levels in parental fish to cause reproductive impairment. Given this, identification and agreement on tissue toxicity thresholds for use in such a model is critical in predicting whether fish populations are at large-scale risk from selenosis.

We critically reviewed the toxicity thresholds proposed by Lemly (1993a) and Skorupa *et al.* (1996), and the scientific literature on which they are based. We then formulated our own recommendations on the appropriate thresholds following, to the extent possible, the USEPA guidelines used to develop water quality criteria (Stephan *et al.*, 1985). Jarvinen and Ankley (1999) recently summarized fish tissue concentrations and associated effects for several chemicals, including selenium. Their interpretations of the toxicological data

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for the studies we reviewed are also provided in our summaries for comparison.

TISSUE-BASED THRESHOLDS

The primary effect of selenium on fish populations is manifested via maternal transfer from the ovaries to the eggs (Gillespie and Baumann, 1986; Woock *et al.*, 1987; Schultz and Hermanutz, 1990; Hermanutz *et al.*, 1992; Coyle *et al.*, 1993), with the dietary pathway being the most important exposure route for juvenile and adult fish (Sandholm, Oksanen, and Pesonen, 1973; Bertram and Brooks, 1986; Woock *et al.*, 1987; Besser, Canfield, and La Point, 1993; Coyle *et al.*, 1993).

Given the importance of the dietary exposure route for fish, and of the ovaries in transferring selenium to embryos, the most relevant tissues for developing residue-based guidelines are ovaries and food items. Whole body and liver thresholds have also been recommended, although some authors (*e.g.*, Skorupa *et al.*, 1996) have suggested that liver residues may have limited environmental relevance. Accordingly, we focused our review on published laboratory, mesocosm, and field studies in which selenium effects (or no effects) can be correlated with selenium residues in ovaries, whole body tissue, and diet. The thresholds recommended by Lemly (1993a) and Skorupa *et al.* (1996) for these tissues are shown in Table 1.

In reviewing these studies, we established guidelines for critiquing the studies and evaluating the residue-based toxicity data. First, all studies had to be based on environmentally relevant and important exposure pathways, *e.g.*, dietary exposure to organo-selenium or maternal transfer to the embryo. Studies based on other exposure routes, such as water only exposures, were reviewed but not included in the final data analysis. Justification for this is provided in the analysis section of this paper. Second, we only considered toxicological endpoints with clear relevance to population level effects, such as reproduction, survival, growth and teratogenesis. This is consistent with the USEPA's approach for deriving water quality criteria (Stephan *et al.*, 1985). Third, again following USEPA guidance, chronic values were calculated as the geometric mean of the no observable effect concentration (NOEC) and lowest observable effect concentration (LOEC).

The use of NOECs and LOECs in developing chemical guidelines, however, is not a rigorous approach because these values are highly dependent on the study design. The NOEC and LOEC do not provide any information on the magnitude of the effect, only that the effect is statistically significantly greater than that observed in the control organisms. This, in turn, is dependent on the variability in the treatment and control responses and the amount of replication (Gelber *et al.*, 1985). To evaluate the studies further, we fit statistical models (probit, logit) to the toxicity data for the various tissues and endpoints. Although the level of an "acceptable" effect is arguable and variable depending on the site and species evaluated, this approach allows thresholds to be developed for different effect levels. We estimated EC_{10}, EC_{20}, and EC_{50} values

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Table 1. Selenium guidelines (mg/kg dw) proposed by Lemly (1993a) and Skorupa et al. (1996).

	Lemly (1993a)		Skorupa *et al.* (1996)		
	Recommended		Recommended		
Tissue	Guideline	Effect	Guideline	Effect
Ovary	10	Reproductive failure	7-13	Reproductive impairment
Whole body	4	Mortality of juveniles and reproductive failure	4-6	Reproductive impairment
Diet	3	Reproductive failure	3-8	Reproductive impairment

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for the different tissues, where possible, and also compared these results to the guidelines recommended by Lemly (1993a) and Skorupa *et al.* (1996).

For consistency, all tissue concentrations are reported on a dry weight (dw) basis. When selenium concentrations were reported on a wet weight (ww) basis in the original study, Lemly assumed that the moisture content in the tissue was 75%. Jarvinen and Ankley (1999) reported concentrations on a wet weight basis, and assumed that the percent moisture in the tissue was 80% if it was not reported. We converted the concentrations they reported back to a dry weight basis. In contrast to Lemly (1993a), we did not assume that the moisture content in every tissue was 75%. We assumed the moisture content in ovaries was slightly higher, at 85% (Gillepsie and Baumann, 1986). This assumption was only needed for the Hermanutz *et al.* (1992) study. Since the percent moisture in whole body fish tissue typically ranges from 75 to 80%, 75% was used conservatively to adjust whole body concentrations when necessary.

Each of the studies we reviewed is summarized below and, where appropriate, in Tables 2 to 4 relative to interpretations by Lemly (1993a), Skorupa et al. (1996), and Jarvinen and Ankley (1999). The review is divided into coldwater and warmwater species because, as discussed later, there is evidence they may differ in sensitivity. Interpretations of the studies by Lemly (1993a), Skorupa *et al.* (1996), and Jarvinen and Ankley (1999) are also provided for comparison when available.

Coldwater Species

Studies relating selenium residues to toxic effects in coldwater fish are limited to rainbow trout (*Oncorhynchus mykiss*) and chinook salmon (*Oncorhynchus tshawytscha*). Studies on each species are discussed separately below.

*Rainbow Trout (*Oncorhynchus mykiss*)*

Goettl and Davies (1978). Rainbow trout were provided a diet spiked with measured selenite concentrations of 9.0, 2.6, and 1.3 mg/kg dw. After 42 weeks, 21% mortality occurred in trout fed the 9.0 mg/kg dw diet, compared to 0% mortality in the control fish and in fish fed the 2.6 mg/kg dw diet. Treatment replication was not reported so it was not possible to determine the NOEC and LOEC. Assuming that 21% mortality is a significant response, the estimated dietary chronic value for this study is 4.8 mg/kg dw (Table 2). Lemly (1993a) reported that a dietary concentration of 9 mg/kg dw resulted in mortality (Table 2), but he did not calculate a chronic value.

Hodson, Spry, and Blunt (1980). Rainbow trout were exposed to aqueous concentrations of selenite in the laboratory. Some effects on blood chemistry were observed, but as stated by the authors, it is unlikely that the observed level of response would be detrimental to fish in a natural situation. Given the lack of a link to potential population-level effects and that dietary exposure was not evaluated, this study was not considered appropriate in evaluating whole body selenium thresholds.

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Table 2. Selenium concentrations in diet (mg/kg dw) associated with effects.

	Our Review		Lemly (1993a)		
Fish Species	Dietary Se Threshold	Endpoint	Dietary Se Threshold	Endpoint	Reference
Rainbow trout	4.8	Geometric mean of concentrations resulting in 0 and 21 percent mortality	9	Mortality	Goettl and Davies, 1978
Rainbow trout	6.9	Geometric mean of NOEC and LOEC for growth and survival	>3	Mortality	Hilton *et al.*, 1980
Rainbow trout	8.7	Geometric mean of NOEC and LOEC for growth and survival	NR		Hicks *et al.*, 1984
Chinook salmon	13	Geometric mean of NOEC and LOEC for survival and growth after 90 days	5	Reduced growth	Hamilton *et al.*, 1990
	13	Geometric mean of NOEC and LOEC for growth after 60 days			
	25	Geometric mean of NOEC and LOEC for survival after 60 days			
Fathead minnow	<55	Lowest concentration tested – reduced growth	NR		Bennett *et al.*, 1986
Fathead minnow	>7.3	Highest concentration tested – no effect on growth	NR		Bertram and Brooks, 1986

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Table 2. Selenium concentrations in diet (mg/kg dw) associated with effects. (continued)

	Our Review		Lemly (1993a)		
Fish Species	Dietary Se Threshold	Endpoint	Dietary Se Threshold	Endpoint	Reference
Fathead minnow	>29.5	Highest concentration measured – no effects on reproduction	20	Reduced growth	Ogle and Knight, 1989
Fathead minnow	<33-60	Reduced growth	NR		Dobbs *et al.*, 1996
Bluegill	<54.4	Only concentration evaluated - 75% mortality	50	Mortality	Finley, 1985
Bluegill	NA	Anomalous response	6.5	Mortality	USFWS, 1990; Cleveland *et al.*, 1993
Bluegill	6.8	Geometric mean of NOEC and LOEC for larval survival to swim-up	13	Reproductive failure	Woock *et al.*, 1987
Bluegill	23.7	Geometric mean of NOEC and LOEC for larval survival	16	Reproductive failure	Coyle *et al.*, 1993
Bluegill	>5.1	No effects on survival	NR		Lemly, 1993c
	<5.1	Survival reduced (simulated winter stress)			
Striped bass	<38.6	Only concentration evaluated - ~100% mortality	35	Mortality	Coughlan and Velte, 1989

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Table 3. Selenium concentrations in whole body tissue (mg/kg dw) associated with effects.

	Our Review		Lemly (1993a)		Jarvinen and Ankley (1999)[a]		
	WB Se		WB Se		WB Se		
Fish Species	Threshold	Endpoint	Threshold	Endpoint	Threshold	Endpoint	Reference
Rainbow trout	WB residue not measured		5	Mortality	Did not cite WB residue		Hilton *et al.*, 1980
Rainbow trout	3.3	Geometric mean of NOEC and LOEC for survival	4	Mortality	NR		Hunn *et al.*, 1987
Chinook salmon	7.6	Geometric mean of NOEC and LOEC for survival and growth after 90 days	3	Reduced growth	4.0	Geometric mean of NOEC and LOEC for survival	Hamilton *et al.*, 1990
	7.4	Geometric mean of NOEC and LOEC for growth after 60 days	10	Mortality			
	16	Geometric mean of NOEC and LOEC for survival after 60 days					
Fathead minnow	<43	Lowest concentration measured – reduced growth	NR		43	Reduced growth	Bennett *et al.*, 1986
Fathead minnow	>2.2	Highest concentration measured – no effect on	NR		2.2	No effect on growth	Bertram and Brooks, 1986

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Table 3. Selenium concentrations in whole body tissue (mg/kg dw) associated with effects. (continued)

	Our Review		Lemly (1993a)		Jarvinen and Ankley (1999)[a]		
Fish Species	WB Se Threshold	Endpoint	WB Se Threshold	Endpoint	WB Se Threshold	Endpoint	Reference
		growth					
Fathead minnow	>7.5	Highest concentration measured -- no effects on reproduction	6	Reduced growth	7.5 5.5	No effects on reproduction Geometric mean of NOEC and LOEC for growth	Ogle and Knight, 1989
Fathead minnow	47.5-76.0	Reduced growth	NR		75.5-89 47.5-76	Reduced survival Reduced growth	Dobbs *et al.*, 1996
Bluegill	4.4	Geometric mean of NOEC and LOEC for survival (waterborne test)	5	Mortality	4 5.4 13.5	No effect on survival (waterborne) Survival reduced 50% (waterborne) No effect on survival (dietary)	USFWS, 1990; Cleveland *et al.*, 1993
Bluegill	WB residue not measured		8	Reproductive failure	NR		Schultz and Hermanutz, 1990

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Fish Species	WB Se Threshold	Endpoint	WB Se Threshold	Endpoint	WB Se Threshold	Endpoint	Reference
Bluegill	10.6	Geometric mean of NOEC and LOEC for larval survival	16	Reproductive failure	[illegible]		Coyle *et al.*, 1993
Bluegill	<18.4	Significant decrease in hatchability and larval survival, significant increase in abnormalities	12	Reproductive failure	18.4	Reduced survival and growth	Hermanutz *et al.*, 1992
Bluegill	>6.0	No effects on survival	NR		5.8	No effects on survival	Lemly, 1993c
	<7.9	34% mortality (simulated winter stress)	NR		7.7	Reduced survival (simulated winter stress)	
Centrarchids	34	EC_{10} based on probit model	15	Teratogenic defects	NR		Lemly, 1993b

WB = Whole body.

NR = Not reviewed.

[a]Whole body concentrations reported by Jarvinen and Ankley were reported on a wet weight basis; if the percent moisture was not provided in the original literature they assumed a moisture content of 80%. The values in this table have been converted to dry weight concentrations using the same percent moisture assumption.

[b]Jarvinen and Ankley (1999) may have misinterpreted the whole body concentrations reported in this study (see text).

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Table 4. Selenium concentrations in ovaries (mg/kg dw) associated with effects.

	Our Review		Lemly (1993a)		Jarvinen and Ankley (1999)[a]		
Fish Species	Ovary Se Threshold	Endpoint	Ovary Se Threshold	Endpoint	Ovary Se Threshold	Endpoint	Reference
Fathead minnow	>10.92	Highest concentration measured – no effects on reproduction	NR		10.9	No effects on survival or reproduction	Ogle and Knight, 1989
Fathead minnow	<39.3	Significantly greater percentage of abnormalities	15	Reproductive failure	NR		Schultz and Hermanutz, 1990
Bluegill	<38.6	65% larval mortality	12	Reproductive failure	46.4[b]	Reduced survival	Gillespie and Baumann, 1986
Bluegill	<30	Significant reduction in percent hatch and larval survival	10	Reproductive failure	29[b]	Reduced survival	Hermanutz *et al.*, 1992
Bluegill	26	Geometric mean of LOEC and NOEC for larval survival	30	Reproductive failure	[c]		Coyle *et al.*, 1993
Bluegill	<49	100% larval mortality	NR		NR		Bryson *et al.*, 1984

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	Our Review		Lemly (1993a)		Jarvinen and Ankley (1999)[a]		
	Ovary Se		Ovary Se		Ovary Se		
Fish Species	Threshold	Endpoint	Threshold	Endpoint	Threshold	Endpoint	Reference
Bluegill	>9.1	88% hatchability, 93% swim-up	NR		NR		Bryson *et al.*, 1985a
Bluegill	<30	76% hatchability, 5.6% swim-up	NR		NR		Bryson *et al.*, 1985a
Bluegill	>14.8	83.8-86.6% hatchability, 91.1-95.5% swim-up	NR		NR		Bryson *et al.*, 1985b
Bluegill	>9.2	86.0% hatchability, 83.3-97.4% swim-up	NR		NR		Bryson *et al.*, 1985b

NR = Not reviewed.

[a]Ovary concentrations reported by Jarvinen and Ankley were reported on a wet weight basis; if the percent moisture was not provided in the original literature they assumed a moisture content of 80%. The values in this table have been converted to dry weight concentrations using the same percent moisture assumption.

[b]Per Gillespie and Baumann (1986), the dry weight concentration was estimated assuming a moisture content of 85%.

[c]It appears Jarvinen and Ankley (1999) may have misinterpreted the ovary concentrations reported in this study (see text).

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Hilton, Hodson, and Slinger (1980). Rainbow trout were fed six different diets containing measured selenite concentrations of 0.2, 0.4, 1.3, 3.7, and 13.1 mg/kg dw. Trout fed the 13.1 mg/kg dw diet had a significantly ($p < 0.05$) lower body weight and higher mortality rate than fish in the other treatments, resulting in a dietary chronic value of 6.9 mg/kg dw (Table 2). For comparison, Lemly (1993a) noted that this study suggests that mortality results at dietary concentrations greater than 3 mg/kg dw. Hilton *et al.* (1980), however, report there were no mortalities in fish fed a diet containing 3.7 mg/kg dw selenium. This study appears to be the basis of Lemly's recommended dietary threshold of 3 mg/kg dw.

Lemly (1993a) also reports from this study that a whole body selenium concentration of 5 mg/kg dw resulted in fish mortality (Table 3). Hilton *et al.* (1980) did not measure whole body selenium concentrations, but concentrations in the carcass, kidney, and liver were approximately 5, 50, and 100 mg/kg dw. Given the relatively high selenium concentrations in the kidney and liver, it is uncertain how well the carcass concentration represents the whole body concentration. Comparison to the interpretation of Jarvinen and Ankley (1999) is not possible because they did not report whole body residues from this study.

Hicks, Hilton, and Ferguson (1984). Juvenile rainbow trout were fed diets containing selenite concentrations of 0.6, 6.6, and 11.4 mg/kg dw for 16 weeks in the laboratory. Trout fed the 11.4 mg/kg dw diet had significantly ($p < 0.05$) reduced growth and increased mortality relative to the controls. No effects were observed in fish fed the 6.6 mg/kg dw diet, resulting in a chronic value of 8.7 mg/kg dw. These results indicate that rainbow trout mortality does not occur at a dietary concentration of 3 mg/kg dw. Lemly (1993a) did not review this study.

Hunn, Hamilton, and Buckler (1987). Rainbow trout were exposed to aqueous concentrations of selenite in the laboratory for 90 days. The test was initiated using sac fry. Percent survival was significantly ($p < 0.05$) reduced relative to the control for fish with a mean whole body selenium concentration of 4.3 mg/kg dw. No significant effects on mortality were observed in fish with a mean whole body selenium concentration of 2.6 mg/kg dw, resulting in a chronic value for this endpoint of 3.3 mg/kg dw (Table 3). Lemly (1993a) similarly reported that mortality was associated with a whole body residue of 4 mg/kg dw. Jarvinen and Ankley (1999) did not cite this study in their review. Because the study only evaluated the water exposure pathway it was not considered in our development of tissue thresholds.

*Chinook Salmon (*Oncorhynchus tshawytscha*)*

Hamilton et al. (1986). Chinook salmon parr were fed a diet containing selenium-contaminated mosquitofish (*Gambusia affinis*) collected from San Luis Drain and Kesterson Wildlife Refuge in California for 6 weeks. Use of mosquitofish from these locations confounds interpretation due to the presence of chemicals in addition to selenium in the tissue. For example,

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mosquitofish from San Luis Drain used in a similar study contained elevated concentrations of boron, chromium, and strontium relative to controls (Hamilton *et al.*, 1990). Moreover, pesticide concentrations have not been measured, but could be relatively high. For example, DDD, DDE, and DDT have been detected in tissue collected in Mud Slough, which runs parallel to the San Luis Drain, at concentrations ranging up to 79.3 µg/kg ww (samples collected in 1992) (Brown, 1997). Hamilton *et al.* (1990) (summarized below) conducted additional tests with chinook salmon, including a test where salmon were exposed to a reference diet fortified with organoselenium. Given that these data are available, and due to the uncertainties in linking effects to selenium residues in fish that may contain elevated levels of several chemicals, the data in Hamilton *et al.* (1986) were not considered appropriate for developing selenium guidelines.

Hamilton et al. (1990). Chinook salmon were fed diets containing either high-selenium mosquitofish collected from San Luis Drain or mosquitofish collected from a reference site that were subsequently fortified with seleno-DL-methionine. Hamilton *et al.* (1990) suggest that the increased toxicity observed in the San Luis Drain diet may be at least partially explained by the presence of other contaminants in the mosquitofish. Given that results are also reported based on selenium-fortified fish from a reference site, we disregarded the data based on fish collected in the San Luis Drain. In the first of two studies, swim-up larvae were exposed to selenium-fortified food for 90 days. The larvae were reared in reconstituted water that simulated a 1:37 dilution from the San Luis Drain (minus trace elements). Selenium concentrations in the water were always below the detection limits of 1.5 to 3.1 µg/L. Dietary selenium concentrations were 3.2, 5.3, 9.6, 18.2, and 35.4 mg/kg dw. In the second study, fingerlings were exposed to the same dietary selenium concentrations for 120 days in brackish water.

Upon termination of the first study at 90 days, survival and growth in chinook salmon fed a diet containing 18.2 mg/kg dw selenium were significantly ($p < 0.05$) reduced relative to control fish. No significant ($p < 0.05$) effects on survival or growth were observed in fish fed a dietary selenium concentration of 9.6 mg/kg dw, resulting in a dietary chronic value of 13 mg/kg dw for these endpoints (Table 2). The reliability of these results is questionable, however, given that control survival decreased from 99% on day 60 to 67% on day 90. It is unusual for high control mortality in an early life-stage test to occur this late in the test and is greater than the acceptable level of control mortality (30%) in salmon early life-stage tests (ASTM, 1998). In addition, survival in the treatment groups was greatly reduced between days 60 and 90 despite whole body selenium residues not substantially changing over this time period. The high mortality rate in the control fish suggests that some of the mortality observed in the treatment groups may be due to factors other than selenium. Given this uncertainty, we feel this test should be repeated to confirm the results and recommend that the 90-day results of this test are not used in developing selenium thresholds at this time.

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The NOEC and LOEC for survival at 60 days were 18.2 and 35.4 mg/kg dw, respectively, resulting in a dietary chronic value of 25 mg/kg dw (Table 2). Based on growth, the NOEC and LOEC at 60 days were 9.6 and 18.2 mg/kg dw, resulting in a dietary chronic value for this endpoint of 13 mg/kg dw (Table 2). Use of the results after 60 days rather than 90 days does not influence the chronic value (*i.e.*, the chronic value for growth at 60 days is equivalent to the chronic value for growth and survival at 90 days), but it does influence the EC_{10}, EC_{20}, and EC_{50} values that are described later in the analysis section.

Lemly (1993a) reported from this study that a dietary selenium concentration of 5 mg/kg dw was associated with reduced growth. However, growth was reduced only at this concentration in the experiment where salmon were fed mosquitofish from the San Luis Drain. As discussed, due to potential contamination by other chemicals, these results are not considered appropriate. The higher toxicity observed using fish from San Luis Drain compared with selenium-spiked fish from the reference site tends to support the hypothesis that other contaminants are contributing to effects. Using the selenium-fortified mosquitofish from the reference site, growth, as discussed above, was only significantly ($p < 0.05$) reduced at dietary selenium concentrations ≥18.2 mg/kg dw. In the brackish water study, salmon were less sensitive than in freshwater.

Following 90 days, the average whole body selenium concentration in fish with significant ($p < 0.05$) mortality was 10.8 mg/kg dw, while no effects ($p > 0.05$) on mortality were observed in fish with a whole body residue of 5.4 mg/kg dw. This results in a whole body chronic value of 7.6 mg/kg dw (Table 3). However, for reasons explained above, the results after 90 days are questionable and probably less reliable than the results after 60 days. Based on the day 60 results, the whole body NOEC and LOEC for survival were 10.4 and 23.4 mg/kg dw, respectively, resulting in a chronic value of 16 mg/kg dw. For the growth endpoint, the whole body NOEC and LOEC were 5.3 and 10.4 mg/kg dw, resulting in a chronic value of 7.4 mg/kg dw. In contrast, Lemly (1993a) reported that growth was reduced in fish fed San Luis Drain mosquitofish and having a whole body residue of 3 mg/kg dw (Table 3). Jarvinen and Ankley (1999) reported the chronic value for survival from this study was 4 mg/kg dw (Table 3).

Warmwater Fish

Selenium residue studies on warmwater fish are limited to fathead minnows (*Pimephales promelas*), bluegill sunfish (*Lepomis macrochirus*), and striped bass (*Morone saxatilis*). Studies for each are described separately below.

Fathead Minnows (*Pimephales promelas*)

Bennett, Brooks, and Boraas (1986). The authors followed selenium through a three-tiered food chain (algae-rotifer-fathead minnow larvae) in three laboratory experiments. In the first experiment, 4-day-old larvae were fed sele-

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nium-contaminated rotifers for 7 days, followed by a control diet for 19 days. In the second experiment, 8-day-old larvae were fed selenium-contaminated rotifers for 9 days. In experiment three, 2-day-old larvae were fed selenium-contaminated rotifers for 7 days before test termination. Mean selenium concentrations in rotifers fed to fathead minnow larvae were >70 and 68 mg/kg dw in experiments one and two, respectively. Larval growth in both of these experiments was significantly ($p < 0.05$) reduced relative to control fish. The mean larval selenium concentration at the end of the 7-day exposure period was 43.0 mg/kg dw in experiment one, and was 51.7 mg/kg dw in experiment two (Table 3). Larval growth in experiment three was significantly reduced at a 90% confidence level, but not at a 95% confidence level. The mean larval selenium concentration was 61.1 mg/kg dw. The mean selenium concentration in rotifers in experiment three was 55 mg/kg dw. Lemly (1993a) did not cite this study in his review. Similar to our review, Jarvinen and Ankley (1999) reported that growth was reduced at a whole body concentration of 43 mg/kg dw.

Bertram and Brooks (1986). Fathead minnows were exposed in the laboratory to either waterborne selenium, selenium-contaminated food, or a combination of the two. Fathead minnows were exposed to waterborne selenium, or the combination of waterborne and dietary selenium, for 8 weeks. Tests of fish exposed only to dietary selenium were terminated after 11 weeks. No effects on growth were observed in fathead minnows fed a diet containing 7.3 mg/kg dw selenium (and 43.5 µg/L selenium in the water), the highest dietary concentration tested (Table 2). No effects on growth were observed in the study in the highest whole body concentration measured, 2.2 mg/kg dw (Table 3). Jarvinen and Ankley (1999) report the same result for whole body tissue. Lemly (1993a) did not cite this study in his review.

Ogle and Knight (1989). Testing was initiated using approximately 60-day-old fathead minnows. Fish were provided a diet spiked with 25% seleno-L-methionine, 25% selenate, and 50% selenite at measured concentrations of 5.2, 10.2, 15.2, 20.3, and 29.5 mg/kg dw. Every 2 weeks, fish were collected and weighed, and one fish from two of four replicates was removed for selenium analysis. Spawning substrates were provided on day 98. On day 105, a male and female were selected and the spawning period was extended for 30 days after the first spawning event in each replicate. Fish were collected for selenium analysis at the end of the spawning period. Eggs were collected and examined for fertility and incubated for determination of hatchability. Survival of larval fish after 14 days was determined. After 98 days of exposure, growth was significantly reduced in fish fed the diet containing 20.3 mg/kg dw selenium.

The ecological significance of this level of growth reduction (16%) is somewhat questionable given the variability in the responses at which significant effects were observed (1.30 ± 0.22 in the control and 1.09 ± 0.16 in fish fed 20.3 mg/kg dw selenium) and, that the fish fed the highest selenium diet (29.5 mg/kg dw) had no significant ($p > 0.05$) effects on any reproductive parameter evaluated: number of spawns per pair, number of eggs per spawn,

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percent hatch, or larval survival to 14 days. For each of these endpoints, the responses were similar to the controls, suggesting that the dietary selenium threshold for reproductive effects is greater than the highest dietary concentration measured (29.5 mg/kg dw, Table 2). Correspondingly, no reproductive effects were observed in fish with a mean ovary concentration of 10.9 mg/kg dw (Table 4) or a whole body concentration of 7.5 mg/kg dw (Table 3). Similarly, Jarvinen and Ankley (1999) reported that no reproductive effects in fish with a mean ovary concentration of 10.9 mg/kg dw or a whole body concentration of 7.5 mg/kg dw.

Schultz and Hermanutz (1990). Fathead minnows were exposed to selenium in experimental streams continuously dosed with 10 μg/L selenite. The streams contained well-developed assemblages of fish food organisms, so an environmentally relevant dietary exposure pathway was simulated. Submerged spawning platforms were provided and checked daily for embryos. Samples of collected embryos were then reared in incubation cups containing stream water with the same selenium concentrations as in the streams from which they were collected. Each sample was observed each post-hatch day for edema and lordosis (larvae were not fed, and observations continued until all larvae died).

Selenium concentrations in the ovaries of the adult females were also determined. The incidence of edema and lordosis in larvae from the dosed stream, 24.6 and 23.4%, respectively, was significantly ($p < 0.05$) greater than in the control stream (0.9 and 5.6%). The mean selenium concentration in ovaries of the parental fish was 39.3 mg/kg dw (assuming ovaries contain 85% moisture [Gillespie and Baumann, 1986]) (Table 4). Lemly (1993a), assumed a 75% moisture content and reported that an ovary concentration of 15 mg/kg dw was associated with reproductive failure (Table 4). The basis for this concentration is unclear, however, as a moisture content of 75% would result in a dry weight concentration of 23.6 mg/kg dw. Jarvinen and Ankley (1999) did not review this study, so their interpretation is not available for comparison.

Dobbs, Cherry, and Cairns (1996). A three trophic level test system consisting of algae, rotifers, and larval fathead minnows was dosed with various concentrations of selenate for 25 days. Fathead minnow larval growth was significantly ($p < 0.05$) reduced relative to the controls in fish fed rotifers with a selenium concentration ranging from approximately 33 to 60 mg/kg dw (Table 2). Whole body selenium concentrations in the affected fish ranged from 47.5 to 76.0 mg/kg dw (Table 3). Jarvinen and Ankley (1999) reported the same concentration range in their review. As the study was conducted after 1993, it was not reviewed in Lemly (1993a).

*Bluegill Sunfish (*Lepomis macrochirus)

Bryson et al. (1984, 1985a, 1985b). Neither Lemly (1993a) nor Jarvinen and Ankley (1999) cited these studies in their reviews of ovary selenium concentrations. Bryson *et al.* (1984) collected bluegills from the selenium-contaminated Hyco Reservoir and from a reference lake. All combinations of males and

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females were artificially crossed in different ash pond concentrations (0, 20, and 50%). Swim-up larvae were fed zooplankton from either Hyco Reservoir or the reference lake. Larvae were observed for 28 days after hatching. All larvae from a Hyco Reservoir female exhibited abnormal development and 100% mortality prior to reaching swim-up. The mean ovary concentration in the females from Hyco Reservoir was 49 mg/kg dw (Table 4).

Bryson *et al.* (1985a), based on a study similar to Bryson *et al.* (1984), reported that egg hatchability from female bluegills averaging 9.1 mg/kg dw in the ovaries was 88%, and that 93% survived through swim-up (Table 4). Swim-up was only approximately 5.6% in larvae from females averaging 30 mg/kg dw in the ovaries (Table 4). The results reported in Bryson *et al.* (1985b) are somewhat difficult to interpret because none of the control fish spawned, but hatching and swim-up success were 83.8 to 86.6 and 91.1 to 95.5%, respectively, from parents with a mean ovary concentration of 14.8 mg/kg dw, and 86.0 and 83.3 to 97.4% from parents with a mean ovary concentration of 9.2 mg/kg dw. The above results indicate that reproductive failure would not be expected to occur at ovary selenium concentrations of 10 mg/kg dw, in contrast to Lemly (1993a).

Finley (1985). Bluegills were fed selenium-contaminated burrowing mayfly nymphs (*Hexagenia limbata*) in the laboratory for 44 days. The mayfly nymphs were collected from Belews Lake and contained an average whole body selenium concentration of 54.4 mg/kg dw. After 44 days, three of four bluegills had died, while no control fish died. The dietary concentration of 54.4 mg/kg dw associated with the observed mortality is comparable to the dietary concentration of 50 mg/kg dw reported by Lemly (1993a).

Gillespie and Baumann (1986). Adult bluegills were collected from the selenium-contaminated Hyco Reservoir and from an uncontaminated reservoir, and all possible combinations of parents were artificially crossed. Zygotes were reared in uncontaminated water and evaluated for percent fertilization and hatching success. In over 18 crosses during a 2-year period, no significant differences in percent fertilization or percent hatch were found between all parent combinations. However, all crosses involving a female from the selenium-contaminated water body resulted in larvae with gross abnormal morphology (65 to 100%), and all larvae died before they reached the swim-up stage.

The ovary selenium concentration in the female producing 65% abnormal larvae was 38.6 mg/kg dw (assuming a moisture content of 85%) (Table 4). Lemly (1993a) reported that an ovary selenium concentration of 12 mg/kg dw in this study was associated with reproductive failure (Table 4). The basis of this concentration is unclear given that the lowest ovary selenium concentration reported by Gillespie and Baumann was 5.79 mg/kg on a wet weight basis; a moisture content of 75%, as assumed by Lemly (1993a), would result in an ovary concentration of 23.2 mg/kg on a dry weight basis. Jarvinen and Ankley (1999) interpreted this study as indicating that an ovary concentration of 6.96 mg/kg ww resulted in larval mortality prior to swim-up; a moisture con-

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tent of 85% would result in an ovary concentration of 46.4 mg/kg dw (Table 4). This concentration differs from ours because Jarvinen and Ankley (1999) used the average ovary concentration that resulted in mortality of 65 to 100% of the larvae. Since an ovary concentration of 38.6 mg/kg dw resulted in substantial larval mortality (65%), we did not want to bias the average high by including ovary concentrations resulting in even greater mortality. Whole body concentrations in the parent bluegill were not measured in this study, and the dietary selenium concentration to which they were exposed is unknown since they were field collected.

Woock et al. (1987). Parent bluegill were fed a diet containing selenomethionine. In one treatment, fish were also exposed to an aqueous concentration of 10 µg/L selenate. After 260 days, spawning experiments were initiated. Artificial spawning beds were provided and checked daily for fertilized eggs. Fertilized eggs were placed in embryo-larval cups and assessed for percent hatch, larval survival, and abnormalities. Larval survival from parent fish fed 13 mg/kg dw was significantly ($p < 0.05$) reduced, but not in larvae from parents fed 3.6 mg/kg dw, resulting in a chronic value of 6.8 mg/kg dw selenium. Lemly (1993a) associated the dietary concentration of 13 mg/kg dw with reproductive failure. Selenium was not measured in the ovaries or whole body tissue.

USFWS (1990); Cleveland et al. (1993). Juvenile bluegill were exposed to a waterborne 6:1 selenate:selenite mixture for 60 days and to dietary seleno-L-methionine for 90 days. In the waterborne exposure, mortality was significantly ($p < 0.05$) reduced in bluegill with whole body concentrations of 5.0 mg/kg dw, but not in fish with 3.8 mg/kg dw selenium. This results in a chronic value of 4.4 mg/kg dw for this endpoint (Table 3). In the dietary experiment, an anomalous response in bluegill mortality after 90 days was observed. Fish with a whole body concentration of 4.7 mg/kg dw had significantly ($p < 0.05$) elevated mortality relative to the control fish, while mortality in fish with whole body concentrations of approximately 7.5 to 13.5 was not significantly greater. Accordingly, the dietary test cannot be used to calculate a chronic value. Lemly (1993a) similarly reported that a whole body concentration of 5 mg/kg dw was associated with bluegill mortality (Table 3). Jarvinen and Ankley (1999) also reported that the chronic value for increased mortality in the waterborne selenium experiment was between 4 and 5.4 mg/kg dw. They also noted that mortality was not significantly greater than controls in the dietary study up to a whole body residue of 13.5 mg/kg dw.

Hermanutz et al. (1992). Similar to the Schultz and Hermanutz (1990) study described previously, adult bluegill were exposed to selenium in experimental streams dosed with either 10 or 30 µg/L selenite. Bluegill nests were sampled three times per week for embryos and larvae. Percentages of dead embryos and larvae were determined in the laboratory. Randomly selected healthy embryos were reared for several days in incubation cups containing the same stream water and selenium concentrations to which they were exposed in the experimental streams. Hatching percentage, larval survival, and abnormalities were

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recorded. Adults were randomly collected from the experimental streams and analyzed for selenium in their ovaries. Larvae from females with an average ovary concentration of 30 mg/kg dw had a significant ($p < 0.05$) reduction in percent hatch and significantly greater mortality rates in the first four days post-hatch (Table 4). It appears Lemly (1993a) conservatively related the observed effects in larvae to the bluegill with the lowest individual ovary selenium concentration (10 mg/kg dw) and considered this the concentration resulting in reproductive failure. However, live embryos were randomly selected from the experimental streams for embryo-larval studies and there are no data to suggest that all sampled embryos were spawned from the same female, let alone the female with the lowest ovary selenium concentration. Using ovary selenium data from an intermediate day of the test, Jarvinen and Ankley (1999) similarly interpreted the results and reported that an ovary concentration of 29.3 mg/kg dw was associated with reduced survival.

Assuming a percent moisture of 75%, the average whole body selenium residue in the parent fish in the stream was 18 mg/kg dw (Table 3). Lemly (1993a), in reporting a whole body concentration of 12 mg/kg dw, appears to have associated the lowest whole body residue value with reproductive failure (Table 3). We believe the average residue is more appropriate as discussed above. Based on their review, Jarvinen and Ankley (1999) report that a whole body residue of 18.4 mg/kg dw was associated with reduced growth and survival (Table 3).

Coyle et al. (1993). Bluegill sunfish were fed diets containing selenium concentrations of 0.8, 4.6, 8.5, 16.8, and 33.3 mg/kg dw, and simultaneously exposed to an aqueous selenium concentration of 10 μg/L for 140 days. Seleno-L-methionine was used for the dietary exposures and a 6:1 ratio of selenate:selenite was used for aqueous exposures. Spawning frequency, fecundity, and hatching success were monitored during the last 80 days of the test and fry survival was monitored for 30 days after hatch (fry were exposed to the same aqueous selenium concentrations as their parents over the 30 day duration). No effects on spawning frequency, fecundity, or hatching success were observed in any of the treatments. However, larvae from females fed the 33.3 mg/kg dw diet had greatly reduced survival (7%) relative to other treatments (75 to 90%) 5 to 6 days after hatching. Therefore, the dietary chronic value, is 23.7 mg/kg dw (Table 2). Lemly (1993a) determined from this study that a dietary concentration of 16 mg/kg dw resulted in reproductive failure. The basis for this is unclear given that reproductive effects were not observed in fish fed a dietary selenium concentration of 16.8 mg/kg dw.

The mean ovary concentration in the parent females of the affected larvae was 35 mg/kg dw. No significant effects on larval survival were observed in larvae from females with an ovary concentration of 20 mg/kg dw, resulting in a chronic value for the ovaries of 26 mg/kg dw (Table 4). Lemly (1993a) associated an ovary concentration of 30 mg/kg dw reported in this study with reproductive failure (Table 4). Jarvinen and Ankley (1999) reported that an ovary concentration of 41.3 mg/kg ww was not associated with any effects on

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survival, growth, or reproduction, which is curious, because the ovary concentrations presented in Coyle *et al.* (1993) are expressed on a dry weight basis (Coyle, personal communication).

Based on the whole body selenium concentrations in adults on the day spawning was initiated, a chronic value of 10.6 mg/kg dw for whole body tissue is estimated for larval survival over 39 days based on an LOEC of 16 mg/kg dw and an NOEC of 7 mg/kg dw (Table 3). Lemly (1993a) cited the LOEC of 16 mg/kg dw as the effect level for reproductive failure (Table 3). As in their interpretation of ovary concentrations in this study, it appears that Jarvinen and Ankley (1999) may have misinterpreted the whole body concentrations reported in this study as they associate a whole body concentration of 19.0 mg/kg ww with no effects on survival, growth, or reproduction.

Skorupa *et al.* (1996) reviewed this study and noted that, due to the poor larval survival of the controls (20 to 25%) at day 30, this test only had the statistical power to detect "catastrophic reproductive impairment". Coyle *et al.* (1993) suggest that high larval mortality observed beginning at day 7 was likely related to starvation resulting from the unsuccessful transition between endogenous and exogenous feeding (yolk sac absorption occurred between days 5 and 6). Given that larval survival was high in controls (>90%) during endogenous feeding at day 5, and larval survival from females with an ovary selenium concentration of 35 mg/kg dw was extremely low (<10%), a clear selenium-related effect can be observed. Although the high control mortality after swim-up imparts some uncertainty to the results, use of larval survival data on day 5 does not require that catastrophic reproductive impairment must occur to observe significant reproductive effects relative to controls.

Lemly (1993b). Based on centrarchid data for juveniles and adults collected from Belews Lake, North Carolina, Lemly (1993b) used a polynomial regression to fit a cubic model to the relationship between whole body selenium concentrations and the percentage of deformed juvenile and adult fish (Figure 1). Based on his model, a whole body selenium concentration of 15 mg/kg dw would translate to deformities in approximately 4% of the fish. He states that the inflection point for this model occurred between 40 to 50 mg/kg dw, corresponding to a frequency of deformities of about 20 to 30%. No information was provided as to how the curve was constructed, but our scatter plot of the raw data reported in his paper closely resembles the plot in Figure 9 of his paper. Using polynomial regression, we fit a cubic model ($Y_i = B_0 + B_1x + B_2x^2 + B_3x^3$) to the raw data using the statistical computer program SPSS (SPSS, 1998) (Figure 1). Our curves are similar up to an abnormality level of approximately 40%, at which point the Lemly (1993b) curve becomes much steeper. The basis for this discrepancy in the models is unknown, but probably inconsequential given that the approximate EC_{10}, EC_{20}, and EC_{50} values from Lemly's (1993b) model were approximately 31, 47, and 72 mg/kg dw, compared with 29, 47, and 73 mg/kg dw from our cubic model. It is only at concentrations above the EC_{50} where our two models begin to substantially diverge. This divergence would not influence the identification of a whole body threshold for this endpoint since the threshold will be much lower than the 50% effect level.

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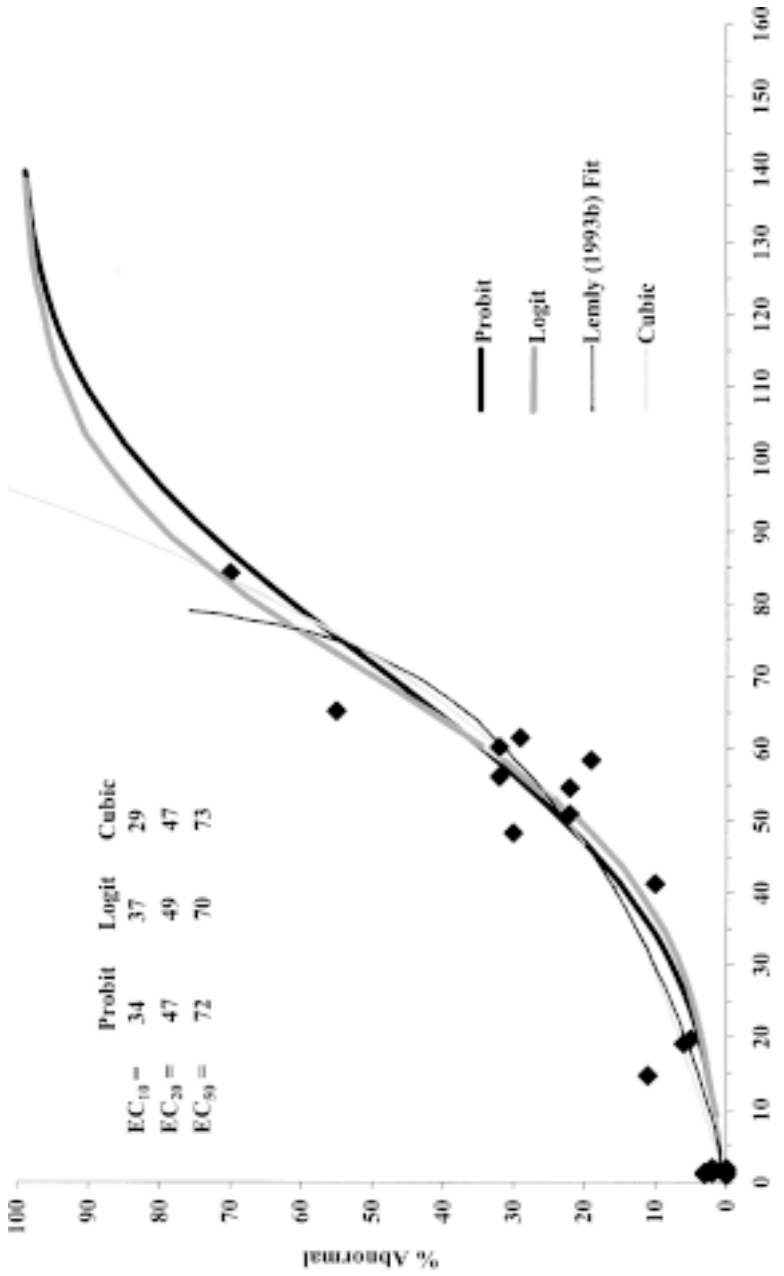


Figure 1. Whole body selenium and teratogenesis in field-collected centrarchids (data from Lemly, 1993b).

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The use of a cubic model for fitting a curve to biological response data is questionable, however, because it will predict levels of effect greater than 100%. Sigmoid models such as probit and logit models that asymptote at a 100% response have been generally demonstrated to appropriately describe biological response data (Gelber *et al.*, 1985). Accordingly, we also fit probit and logit models to the raw data; both appeared to provide a good fit to the data (Figure 1). As shown in Figure 1, the EC_{10}, EC_{20}, and EC_{50} for the probit and logit models are 34, 47, and 72 and 37, 49, and 70 mg/kg dw, respectively. The 15 mg/kg whole body residue associated with teratogenesis reported by Lemly (1993a) appears to be overly conservative for this endpoint given that even the EC_{05} from this study is 24 mg/kg dw using the probit model. In addition, Lemly (1998) suggests that if the proportion of juveniles or adults with terata is less than 20%, the anticipated impact on fish populations is expected to be negligible. Considering this, we would select an EC_{10} of 34 mg/kg dw as a conservative effects threshold for this study. Regardless, as demonstrated later in this paper, teratogenesis in juvenile and adult fish is a less sensitive endpoint than larval mortality.

Lemly (1993c). Field-collected juvenile bluegills were exposed in the laboratory for 180 days to a dietary seleno-L-methionine concentration of 5.1 mg/kg dw and a waterborne 1:1 selenate:selenite concentration of 4.8 μg/L. In one group of fish, winter conditions were simulated by gradually reducing the water temperature to 4°C and decreasing the photoperiod; in the other group, the water temperature was maintained at 20°C. In the warmwater group, mortality (5.8%) was not significantly reduced relative to the warmwater control fish (4.3%). In the coldwater group, mortality was 33.8% compared to 2.8% in the coldwater control. The whole body tissue concentrations in the warmwater and coldwater groups were 6.0 and 7.9 mg/kg dw, respectively, Jarvinen and Ankley (1999) interpreted the results similarly. While these data provide interesting information on the potential interactive effects of temperature and selenium, water-quality criteria guidelines are not currently developed using studies with thermally stressed organisms (Stephan *et al.*, 1985). Consequently, we did not consider these data in our final assessment.

*Striped Bass (*Morone saxatilis*)*

Coughlan and Velte (1989). Striped bass were fed selenium-contaminated red shiners (*Notropis lutrensis*) in the laboratory for 78 days. The red shiners were collected from Belews Lake and contained an average whole body selenium concentration of 38.6 mg/kg dw. After 78 days, all striped bass had died or were killed because they were near death. The dietary concentration of 38.6 mg/kg dw is comparable to the dietary concentration of 35 mg/kg dw reported by Lemly (1993a).

ANALYSIS AND RECOMMENDATIONS

Most of the study results summarized above are discussed in terms of whether the responses in the treatment groups are significantly different than

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the responses in the control groups. We also evaluated the concentration-response data using regression models when sufficient data were available. This approach allows different effect levels to be estimated and allows data from multiple studies with similar endpoints to be pooled.

Ovaries. In many of the studies in which adverse effects were associated with ovary residues, the range of ovary selenium concentrations measured was insufficient for developing an adequate concentration-response relationship. The Coyle *et al.* (1993) study is the only one for which a concentration-response relationship could be developed based on data from a single study. The ovary-based chronic value from this study for larval mortality at day 5 was 26 mg/kg dw. Fitting probit and logit models to the day 5 larval mortality data (following Abbott's correction[2]) results in EC_{10}, EC_{20}, and EC_{50} values of 24, 27, and 33 mg/kg dw and 26, 29, and 34 mg/kg dw, respectively (Figure 2). The EC_{10} and EC_{20} values bracket the chronic value (26 mg/kg dw). Of the individual studies, this study seems to be the most appropriate for recommending an ovary-based selenium guideline at this time because it was based on a wide range of exposure concentrations (*i.e.*, a sufficient number to bracket the effects threshold), it evaluated environmentally realistic exposure pathways (*i.e.*, organoselenium in the diet and inorganic selenium in the water), and it evaluated the critical exposure pathway for larvae (*i.e.*, maternal transfer from the ovary to the egg). However, we did not want to disregard the data from other studies that evaluated larval mortality from parents exposed to selenium, so we pooled all the relevant data.

The studies pooled were Ogle and Knight (1989), Bryson *et al.* (1984, 1985 a,b), Hermanutz *et al.* (1992), and Coyle *et al.* (1993). Using Abbott's correction on the larval mortality data, these data tend to follow a characteristic sigmoid distribution when plotted (similar to the Coyle *et al.* data in Figure 2). Both the probit and logit models fit the raw data quite well (Figure 3). The EC_{10}, EC_{20}, and EC_{50} values are 17, 20, and 27 mg/kg dw based on the probit model, and 18, 21, and 27 mg/kg dw based on the logit model. The probit EC_{10} is 70% greater than the ovary guideline of 10 mg/kg dw recommended by Lemly (1993a) which, as discussed above, appears to be derived from the Hermanutz *et al.* (1992) study by associating the lowest measured ovary selenium concentration in parent fish with significant observed larval mortality. These data suggest that the ovary chronic value of 26 mg/kg dw from Coyle *et al.* (1993) may be nonconservative as the EC_{50} is of similar magnitude. Accordingly, we recommend that the EC_{10} of 17 mg/kg dw be used as the ovary-based threshold for larval mortality.

The statistical regression approach allows different levels of protection to be evaluated and does not unduly place all the weight on one study. A key uncertainty in this approach is the applicability of these models to coldwater fish (all relevant ovary data currently available are for warmwater species). As

[1] Corrected % Mortality = (Observed % Mortality – Control % Mortality)/(100 – Control % Mortality)

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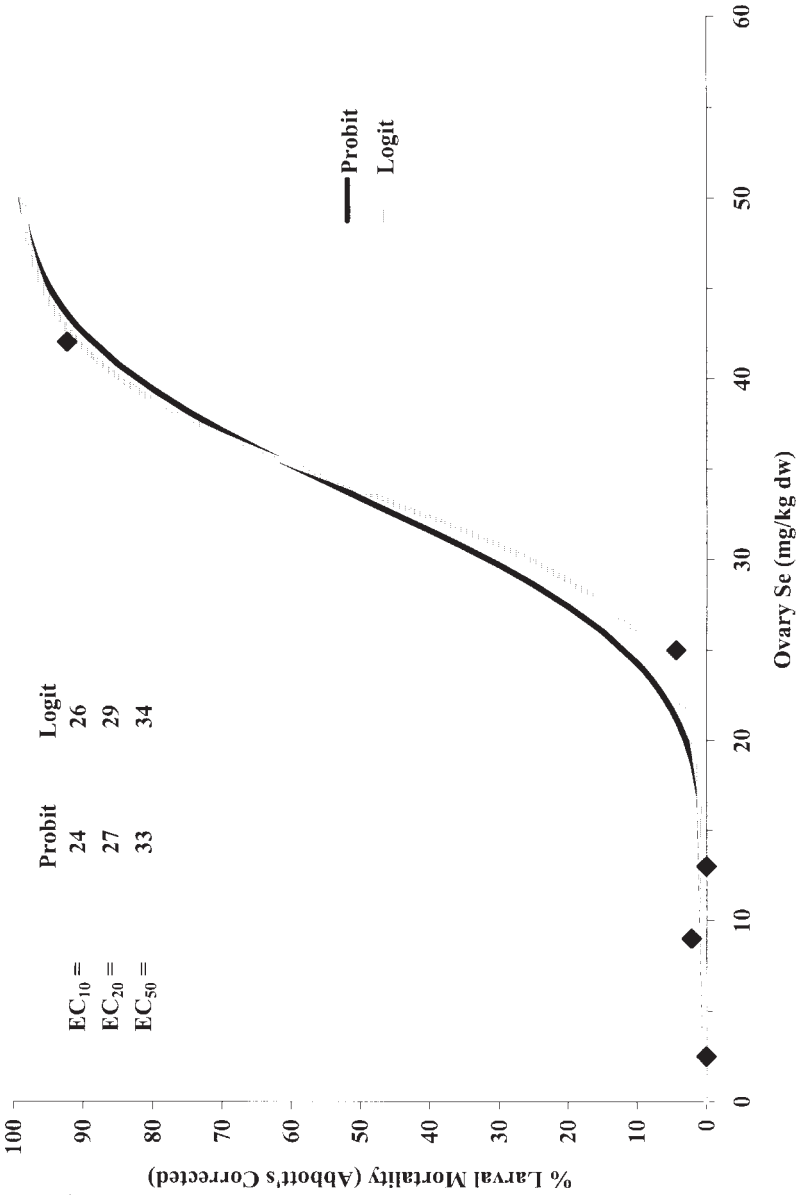


Figure 2. Ovary selenium vs. larval mortality at day 5 (data from Coyle *et al.*, 1993).

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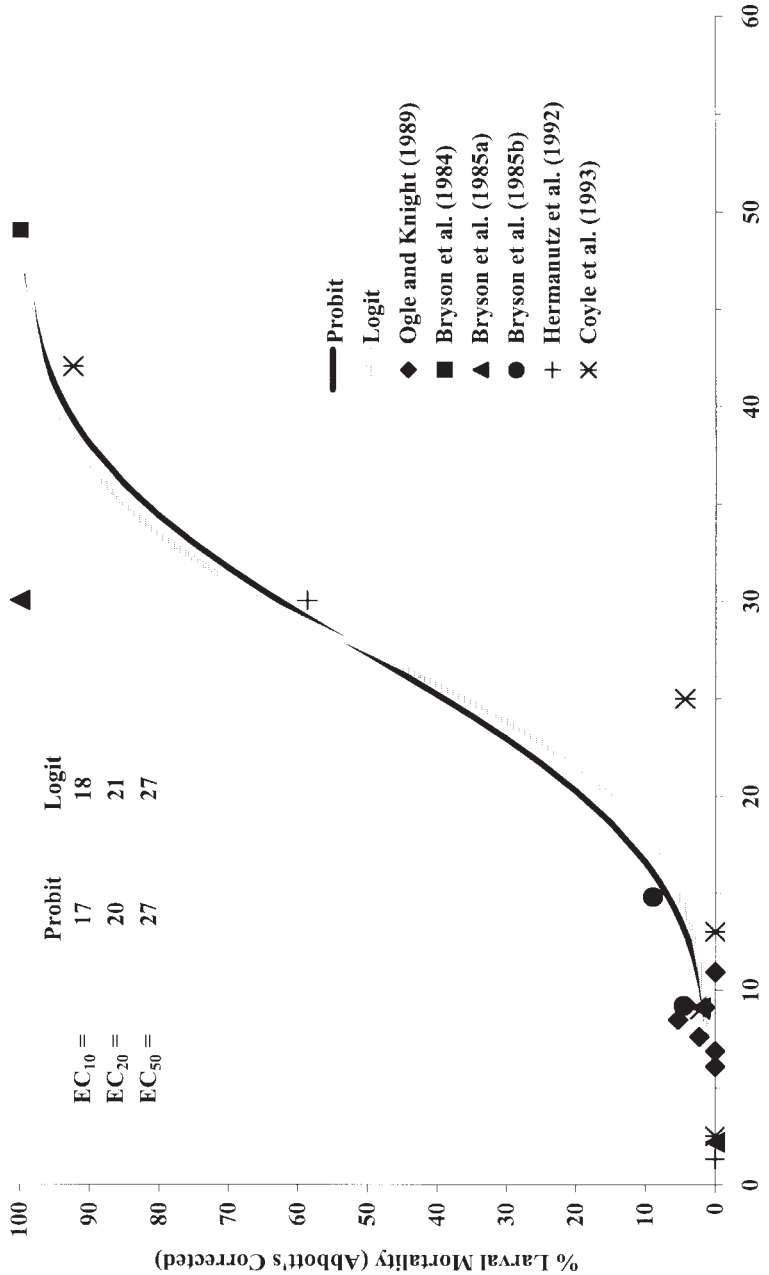


Figure 3. Warmwater fish: ovary selenium vs larval mortality.

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discussed below, some coldwater fish may be more sensitive than warmwater fish. Another uncertainty is the paucity of warmwater species for which ovary data are available. Further testing should be conducted to understand the variability in sensitivities between species.

Whole Body. The whole body guideline of 4 mg/kg dw recommended by Lemly (1993a) is based on mortality of rainbow trout exposed to aqueous selenite (Hunn *et al.,* 1987). In reviewing the whole body residue-based toxicological data, we noted two potentially significant relationships that may exist. The first potentially significant relationship is heavily dependent on the chinook salmon data from Hamilton *et al.* (1990). As discussed above, the toxicity results after 90 days are questionable due to the rapid decline in survival of control fish between day 60 and 90 of the test. If the results after 90 days are indeed reliable, and the observed mortality can be attributed to selenium, the results suggest coldwater fish (represented by chinook salmon) may be more sensitive than warmwater fish (represented by bluegill) (Figure 4). However, if the results after 60 days are more reflective of the sensitivity of chinook salmon to selenium, there would be limited evidence to suggest that coldwater fish are more sensitive than warmwater fish. In fact, the sensitivity of coldwater and warmwater fish appear to be quite similar based on the day 60 data (Figure 4). The second potentially significant relationship that became apparent was that effects are observed in fish at lower whole body selenium concentrations when they are exposed to only waterborne selenium than when they are exposed to dietary selenium. Cleveland *et al.* (1993), for example, exposed bluegills to an aqueous 6:1 selenate:selenite mixture for 60 days, and in a separate experiment, exposed bluegills to dietary organoselenium for 90 days. Observed mortality was higher at lower whole body residues in fish exposed only to waterborne inorganic selenium (Figure 5). This has important implications in establishing a whole body selenium guideline because, as discussed previously, natural fish populations are primarily exposed to selenium via their diet. Cleveland *et al.* (1993) support this observation, noting that toxicity to natural populations of bluegill and probably other aquatic organisms would probably occur at much lower waterborne concentrations than those tested in this study owing to food-chain exposure.

Besser *et al.* (1993) also observed that bluegills from both aqueous and food-chain exposures to seleno-methionine accumulated greater selenium concentrations than bluegills in comparable exposures with inorganic selenium species. Bluegills in food-chain exposures accumulated consistently greater selenium concentrations than those in aqueous exposures, and bluegills exposed to both aqueous and foodborne selenium accumulated most of their total selenium residues from their food. Bertram and Brooks (1986) measured different uptake kinetics between waterborne selenium and dietary selenium, suggesting the existence of two functional compartments for selenium accumulation in fish. One compartment may contain an unbound inorganic pool and the other may contain organically bound compounds. The depuration rate of the unbound inorganic pool is rapid. The authors noted that accumulation of waterborne selenium and dietary selenium is approximately additive,

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and that in all cases the potential burden of selenium in fish tissues is related to the concentration of selenium in the fish diet.

We hypothesize that waterborne inorganic selenium exposures result in toxicity at lower whole body residues than those based on dietary organoselenium exposures because the inorganic selenium in fish is rapidly depurated. In natural populations, most of the selenium exposure and resulting bioaccumulation is from dietary organoselenium, resulting in higher whole body concentrations. Comparison of whole body selenium concentrations in field-collected fish to laboratory-derived toxicity data based on waterborne exposures to inorganic selenium is, therefore, not a valid comparison. Given that Lemly's (1993a) recommended whole body residue guideline of 4 mg/kg dw is based on a study involving water only exposures, we recommend guidelines based only on studies including dietary exposures.

For coldwater fish, Hamilton *et al.* (1990) is the only study in which fish were fed diets containing organoselenium. As discussed above, the whole body chronic values in this study are 7.4 mg/kg dw for reduced growth and 7.6 mg/kg dw for reduced survival following 60 and 90 days of exposure, respectively. Based on the probit and logit models, the EC_{10}, EC_{20}, and EC_{50} values for mortality in this study are 12, 17, and 27 mg/kg dw after 60 days and 1.7, 2.8, and 7.0 mg/kg dw after 90 days (Figure 6). Based on the growth endpoint, EC_{10}, EC_{20}, and EC_{50} values are 6, 11, and 20 mg/kg dw after 60 days of exposure to selenium, and 5, 7, and 14 mg/kg dw after 90 days (Figure 7).

The lowest whole body chronic value for warmwater fish from a dietary study, 10.6 mg/kg dw, is based on larval mortality (Coyle *et al.*, 1993). Similar to the ovary data discussed above, we pooled the whole body selenium and larval mortality studies from all relevant studies for warmwater fish (*i.e.*, Ogle and Knight, 1989, Hermanutz *et al.*, 1992, Coyle *et al.*, 1993). The whole body EC_{10}, EC_{20}, and EC_{50} values, using both the probit and logit models, are 9, 11, and 15 mg/kg dw, respectively (Figure 8). It should be noted that the concentration-response relationship is not well defined as there are only two whole body concentrations where larval mortality was greater than 10% following Abbott's correction for control mortality. As for the ovaries, we recommend the EC_{10} values as whole body thresholds. Given that the results of the Hamilton *et al.* (1990) study after 90 days are questionable and need to be confirmed, we recommend that the EC_{10} for growth at 60 days (*i.e.*, 6 mg/kg dw), the most sensitive endpoint measured, be considered the whole body threshold for larval coldwater anadromous fish; the EC_{10} of 9 mg/kg dw is recommended as the threshold for warmwater fish. It is important to note that the coldwater anadromous fish threshold is for larval fish, while the warmwater threshold is for adults. The coldwater anadromous fish threshold specifically does not apply to adult fish as maternal transfer of selenium has not been evaluated in these fish.

Dietary. The Hamilton *et al.* (1990) chinook salmon study is currently the most relevant for determining a dietary threshold for coldwater fish because it is the only coldwater study in which fish were fed organoselenium. Probit

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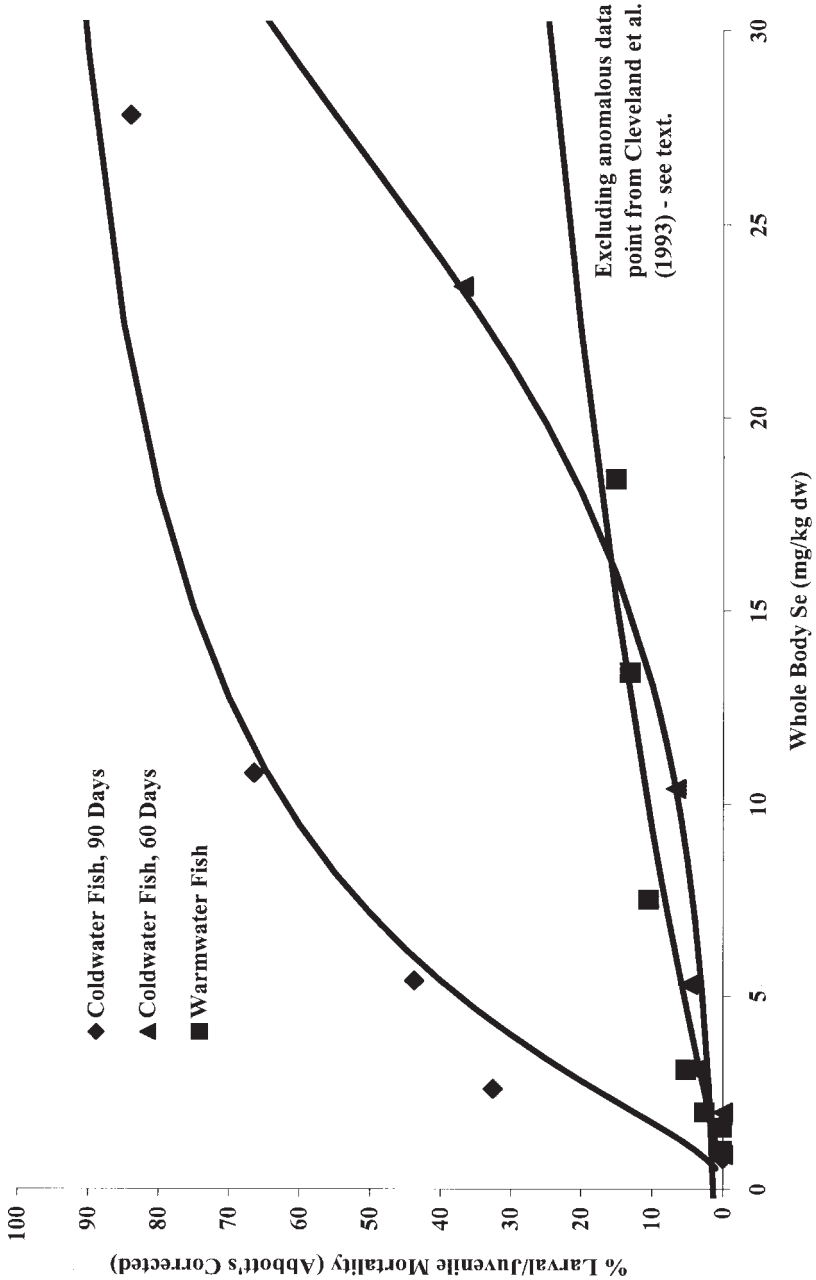


Figure 4. Relative sensitivities of coldwater and warmwater fish based on whole body residues.

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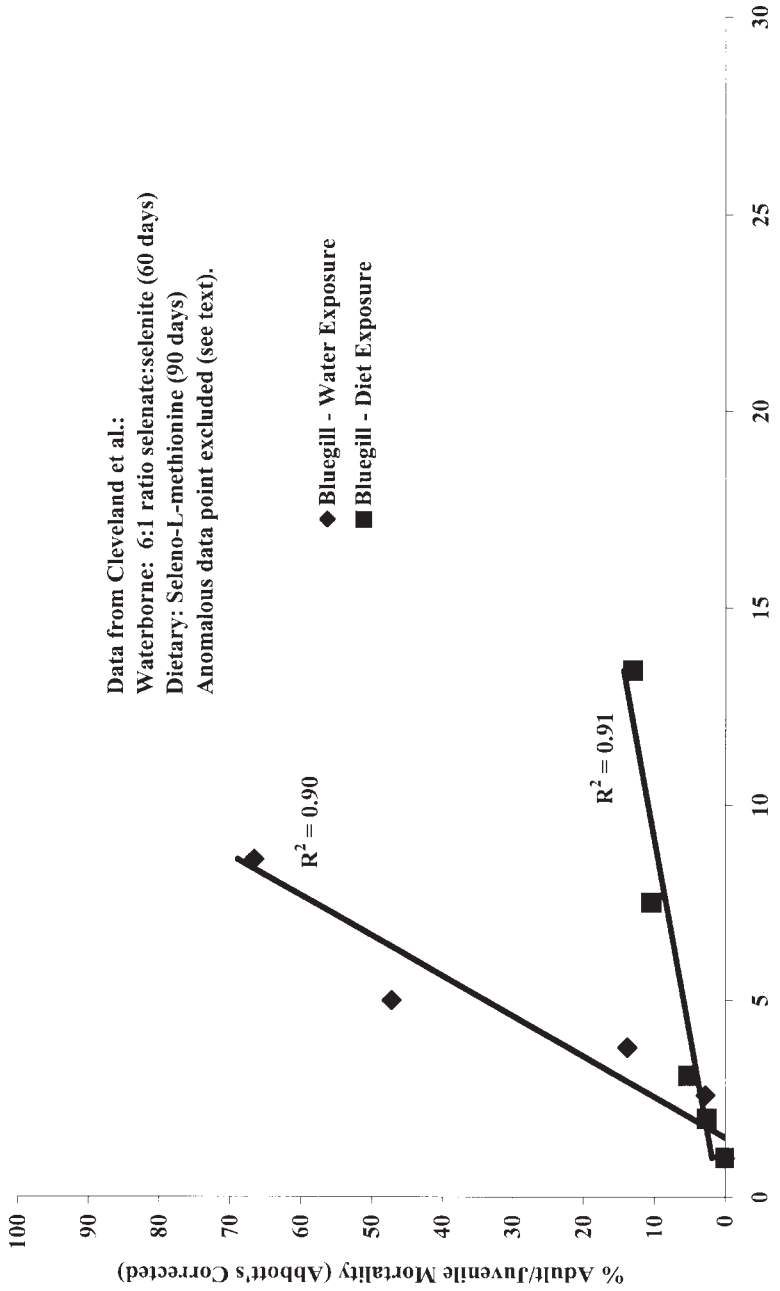


Figure 5. Relative sensitivities of bluegills based on dietary and waterborne exposures.

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and logit models were fit to the larval mortality (Abbott's corrected) and growth data at 60 and 90 days (Figures 9 and 10). The lowest EC_{10}, EC_{20}, and EC_{50} values for mortality from either model are 19, 27, and 41 mg/kg dw after day 60 and 3.4, 5.3, and 12 mg/kg dw after day 90. Based on the growth endpoint, the lowest EC_{10}, EC_{20}, and EC_{50} values from either model are 11, 17, and 30 mg/kg dw after day 60 and 10, 15, and 24 mg/kg dw after day 90. For the same reasons discussed above, further studies on chinook salmon should be conducted to verify these results, as well as studies of nonanadromous coldwater fish in order to determine if they are similarly sensitive.

For warmwater fish, similar to the ovary versus larval mortality analyses described above, we compared larval mortality data to dietary selenium concentrations fed to the parent fish. The relevant studies for this analysis were Ogle and Knight (1989), Bryson *et al.* (1985b), Woock *et al.* (1987), and Coyle *et al.* (1993). Probit and logit models were fit to the larval mortality and dietary selenium data from these studies. The model fits were relatively poor, primarily due to the extreme differences in larval mortality in the Ogle and Knight (1989) and Woock *et al.* (1987) studies at a dietary concentration of about 29 mg/kg dw. If the Ogle and Knight (1989) fathead minnow data are removed, the fits of the probit and logit models become much better (Figure 11). Larval mortality may not have been as high in the Ogle and Knight (1989) study because the fathead minnow diets were spiked with 50% selenite, 25% selenate, and 25% organoselenium, and organoselenium is more toxic than inorganic selenium (Woock *et al., 19*87). The EC_{10}, EC_{20}, and EC_{50} values using the model excluding the Ogle and Knight (1989) data are 10, 13, and 19 based on both the probit and logit models. Using data from all appropriate studies, we recommend the EC_{10} of 10 mg/kg dw as the dietary threshold for larval mortality in warmwater fish. Similarly, the EC_{10} of 11 mg/kg dw is recommended as the dietary threshold for reduced growth in larval coldwater anadromous fish. The EC_{10} values from the probit and logit models for both warmwater fish and coldwater anadromous suggest that the dietary guideline of 3 mg/kg dw recommended by Lemly (1993a) may be overly conservative. It must be reemphasized that the Hamilton *et al.* (1990) study should be repeated to determine if the 90-day results are indeed valid and that further studies should be conducted to examine the sensitivities of chinook salmon relative to other coldwater fish (particularly nonanadromous fish). Moreover, this threshold for larval coldwater anadromous fish may be relevant for modeling a lotic system, but probably not a lentic system.

SUMMARY AND CONCLUSIONS

The most appropriate tissue for developing a residue-based selenium guideline is the ovary. It is the best predictor of selenium concentrations to which embryos and larvae are exposed. Use of an ovary-based guideline is also more appropriate than a dietary-based guideline because ovary residues represent the integration of waterborne and dietary exposures that natural fish populations receive. One of the key uncertainties in establishing residue-based guide-

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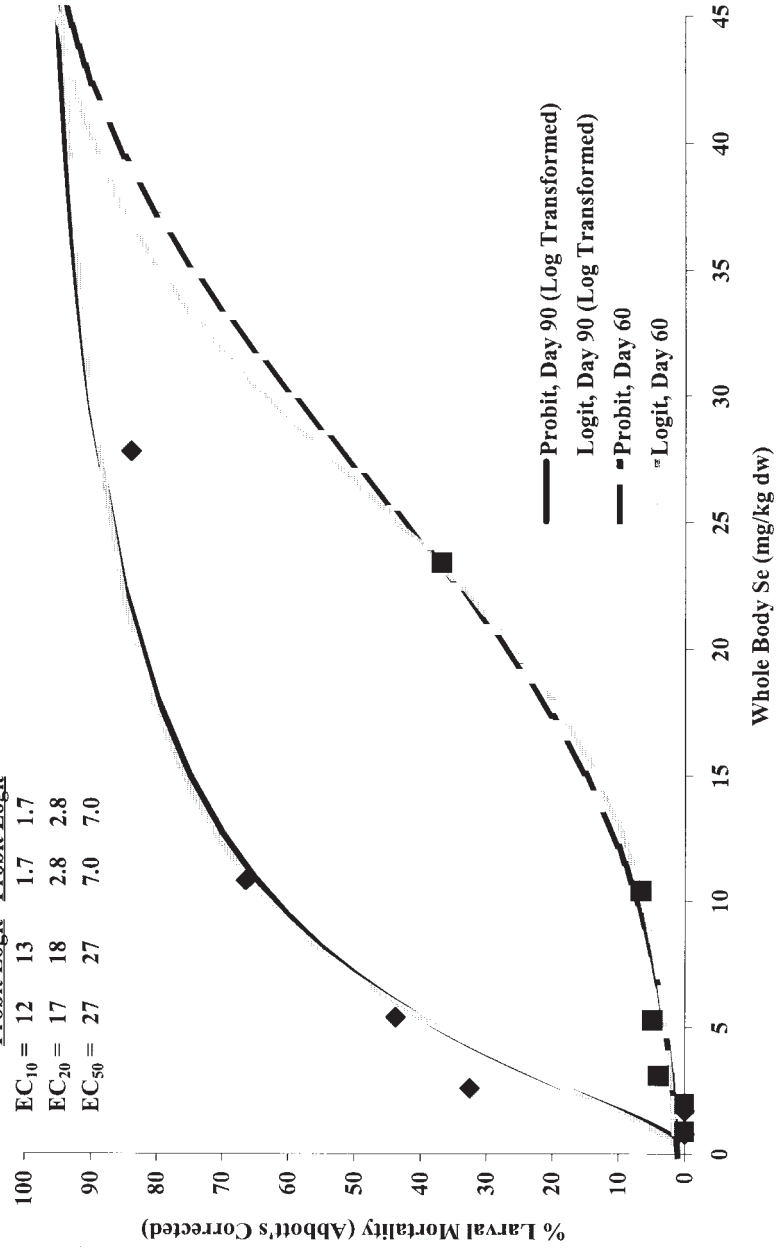


Figure 6. Whole body selenium vs. larval mortality in chinook salmon (data from Hamilton *et al.*, 19.90).

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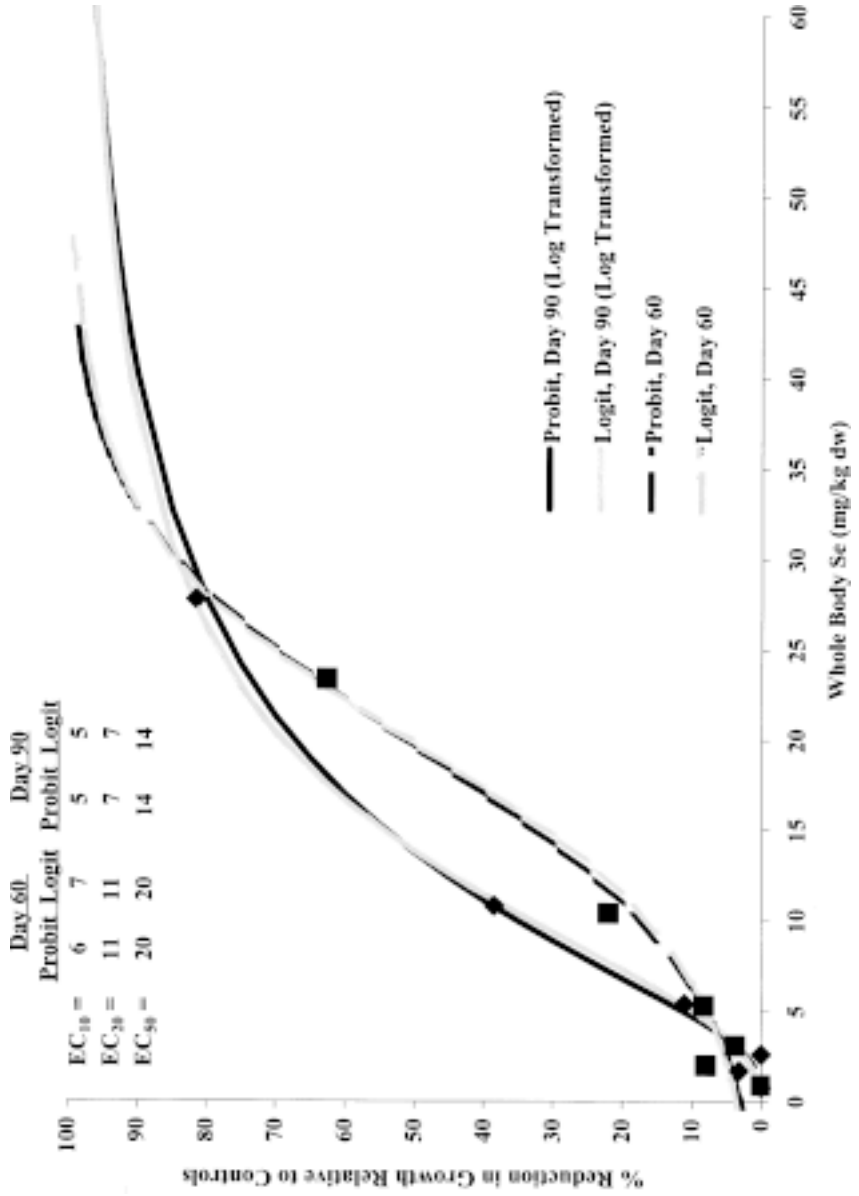


Figure 7. Whole body selenium vs. larval growth in chinook salmon (data from Hamilton *et al.*, 1990).

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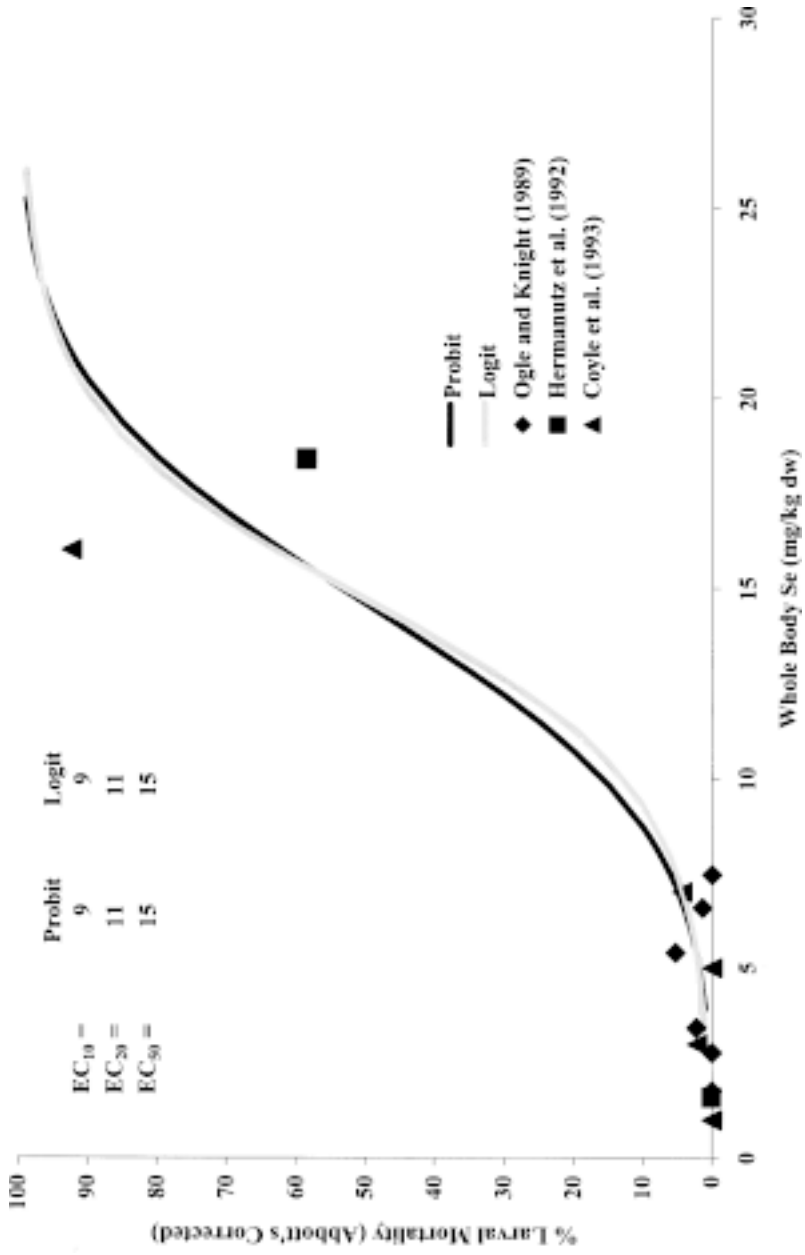


Figure 8. Whole body selenium vs. larval mortality in warmwater fish.

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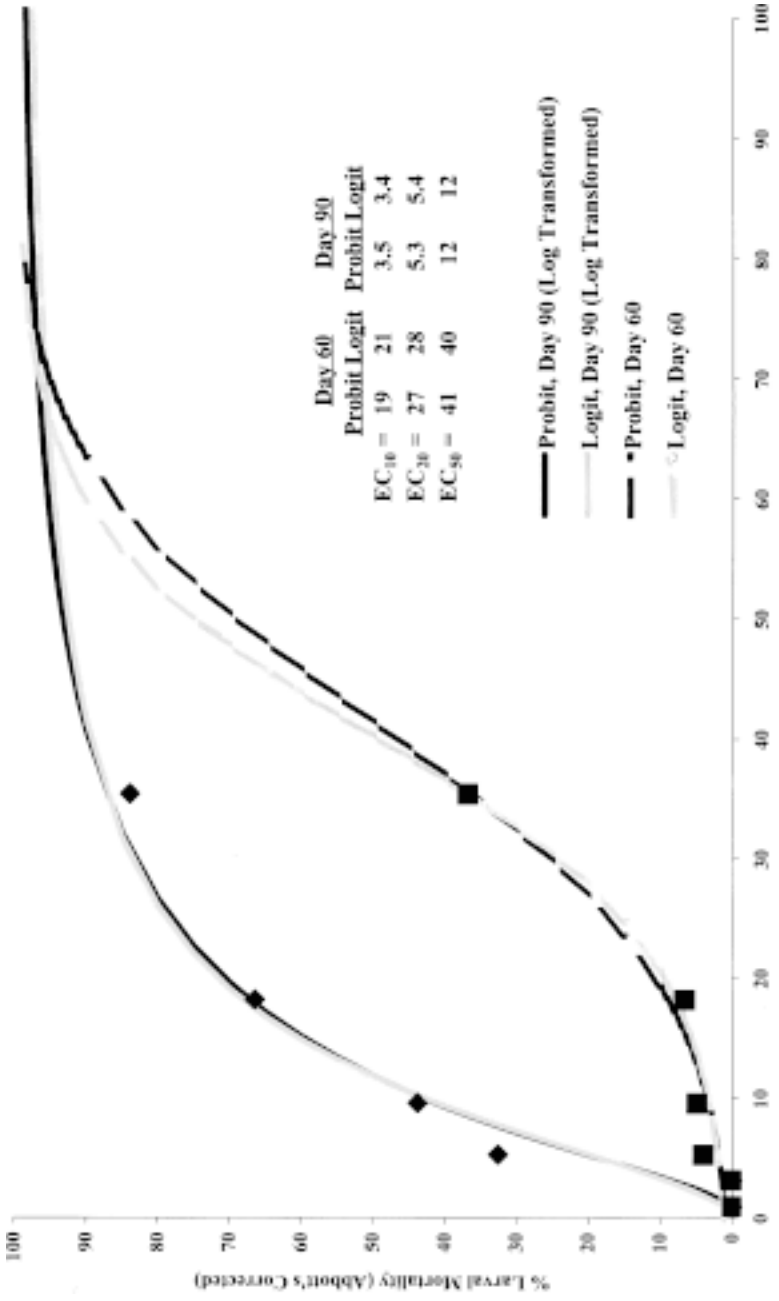


Figure 9. Dietary selenium vs. larval mortality in chinook salmon (data from Hamilton *et al.*, 1990).

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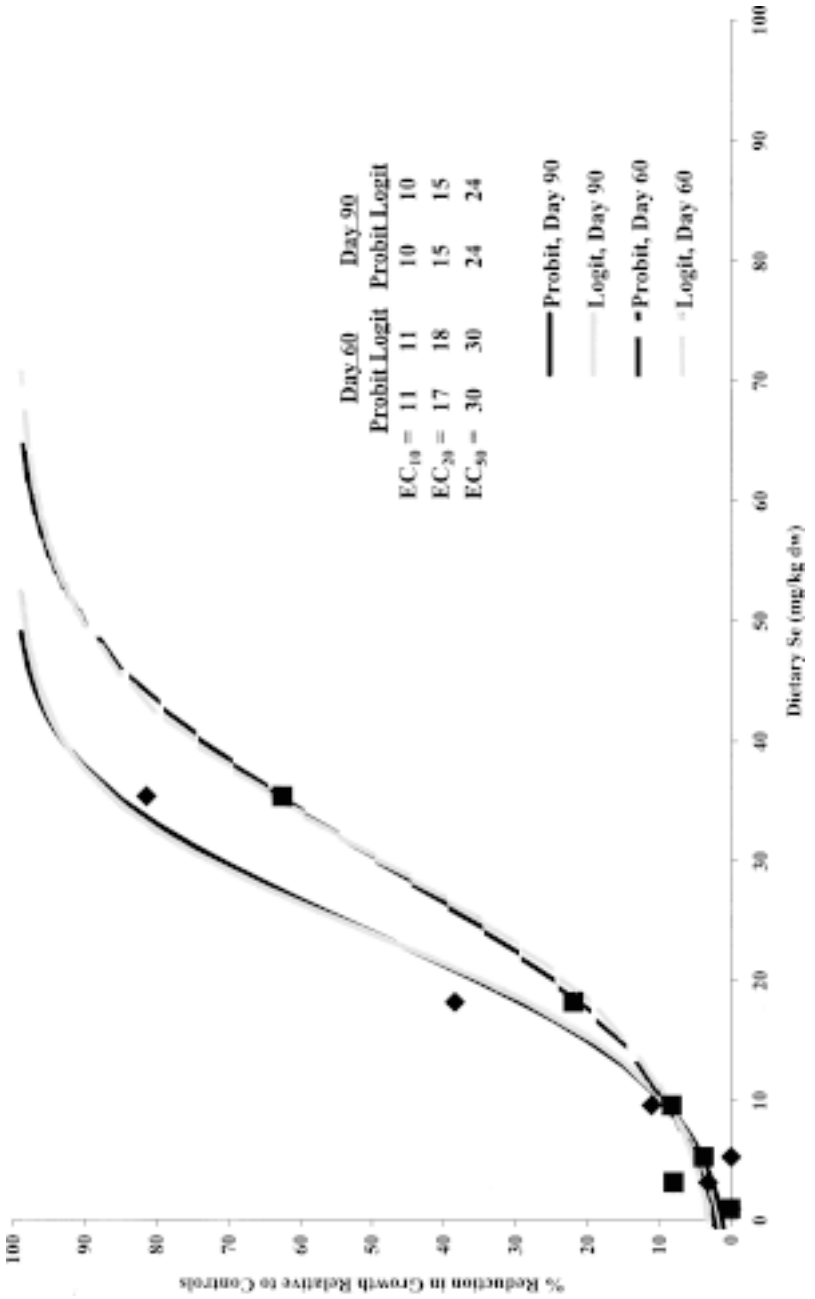


Figure 10. Dietary selenium vs. larval growth in chinook salmon (data from Hamilton *et al.*, 1990).

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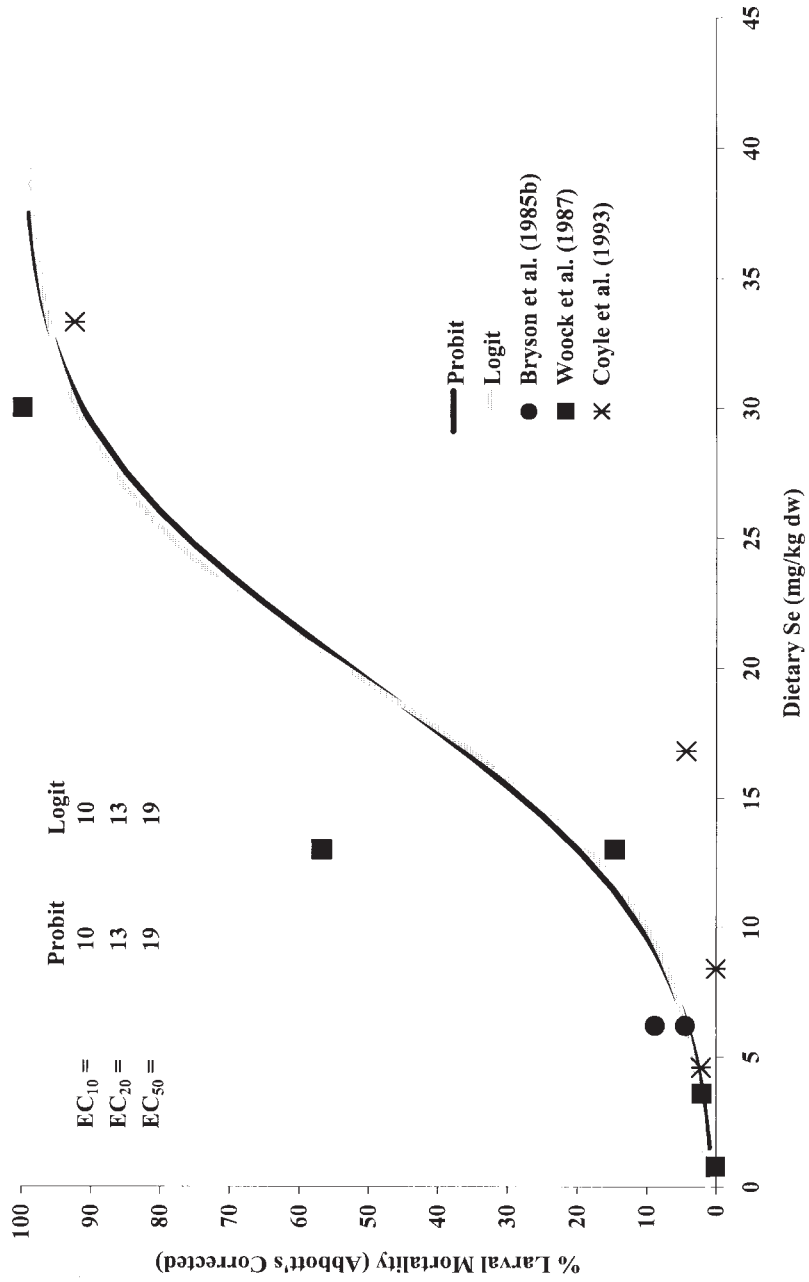


Figure 11. Dietary selenium vs. larval mortality in warmwater fish.

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lines at this time is the limited number of fish species for which there are residue-effects data. The vast majority of the data are for rainbow trout, chinook salmon, fathead minnows, and bluegill. Additionally, the lack of maternal transfer data for nonanadromous coldwater species represents a significant uncertainty given that the whole body residue data suggest that coldwater species are more sensitive.

In summary, the guidelines recommended by Lemly (1993a) and Skorupa *et al.* (1996) are generally not supported by our review of the scientific literature, particularly for warmwater fish. In some cases the tissue concentrations associated with toxic effects are based on non-environmentally relevant exposure pathways, while in others the interpretations of the toxic tissue concentrations appear unsubstantiated by the data presented in the original studies. The tissue concentrations we recommend as guidelines, compared with those recommended by Lemly (1993a) and Skorupa *et al.* (1996), are presented in Table 5. Based on the tissue residue selenium data reported in the scientific literature, we believe our recommended tissue guidelines provide a defensible set of values for use in bioaccumulation models for conducting risk assessments or deriving site-specific water quality criteria. Based on the guidelines recommended by Lemly (1993a) and Skorupa *et al.* (1996), fish populations at many locations across the U.S. might be predicted as being at risk from selenosis. Our review suggests that selenium risks to fish populations is not as common as some believe, but further studies are needed to confirm the sensitivities of fish to selenium, particularly coldwater species.

Table 5. Comparison of our recommended thresholds (mg/kg dw) to those of Lemly (1993a) and Skorupa et al. (1996).

Tissue	Our Review	Lemly (1993a)	Skorupa *et al.* (1996)
Fish ovaries	17	10	7-13
Fish whole body	6 (coldwater anadromous fish)	4	4-6
	9 (warmwater fish)		
Fish diet	11 (coldwater anadromous fish)	3	3-8
	10 (warmwater fish)		

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