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Commentary

Review of residue-based selenium toxicity thresholds for freshwater fish

Steven J. Hamilton*

US Geological Survey, Columbia Environmental Research Center, Field Research Station, 31247 436th Avenue, Yankton, SD 57078-6364, USA

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Abstract

A variety of guidelines have been proposed in recent years for linking selenium concentrations in the whole body of fish or in diet with adverse effects in fish. Diverging viewpoints seem to be forming separating groups supporting either the low-selenium guidelines proposed by the government and academic researchers or the high-selenium guidelines proposed by other researchers. Recently, an article was published that reviewed selected studies and recommended guidelines for selenium concentrations in the whole body of fish and in diet that were higher than those proposed by other researchers (≈4 μg/g in whole body and 3–4 μg/g in diet). That article also recommended separating guidelines for coldwater fish (6 μg/g in whole body and 11 μg/g in diet) and warmwater fish (9 μg/g in whole body and 10 μg/g in diet). The approaches, information, and guidelines presented in the article are reviewed and problems in their interpretation and conclusions are discussed. The majority of the selenium literature supports a whole-body threshold of 4 μg/g in fish and 3 μg/g in diet.

Keywords: Selenium; Diet; Fish; Threshold; Tissue-based criteria

1. Introduction

The criteria for selenium in the aquatic ecosystem has become a controversial topic in recent years as evidenced by debate articles in the journal *Human and Ecological Risk Assessment* (Chapman, 1999; Lemly, 1999a; Hamilton, 1999; Ohlendorf, 1999; DeForest et al., 1999; Fairbrother et al., 1999), response articles (Skorupa, 1999; Fairbrother et al., 2000), and debates at national scientific meetings, i.e., "Selenium in the Environment: A Ticking Time Bomb or No Big Deal?" (SETAC, 1999). There seems to be a divergence between academia or government-backed articles proposing low-selenium criteria (SWRCBC, 1987; UCC, 1988; DuBowy, 1989; Skorupa and Ohlendorf, 1991; Pease et al., 1992; Peterson and Nebeker, 1992; Lemly, 1993a, 1996; Maier and Knight, 1994; Engberg, 1999; Skorupa, 1998; USDOI, 1998) and nongovernmental articles proposing high criteria (Canton and Van Derveer, 1997; Van Derveer and Canton, 1997; Canton, 1999; DeForest et al., 1999; Adams et al., 2000; Brix et al., 2000).

The US Environmental Protection Agency (USEPA) is currently in the process of revising the selenium chronic criterion for the protection of aquatic life (C. Delos and K. Sappington, USEPA, written communications), which was established in 1987 (USEPA, 1987). One step in the USEPA revision process was a peer consultation workshop on the bioaccumulation and aquatic toxicology of selenium, held to discuss the technical issues underlying the freshwater aquatic life chronic criterion (USEPA, 1998). The nine-member peer review group was composed of representatives from federal agencies, academia, private consultants, and industry. The subjects of interest in the workshop included the potential development of a water-based criterion, a tissue-based criterion, and a sediment-based criterion. The general consensus of the peer review group was that the relationship between water-borne and sediment selenium concentrations to the tissue accumulation of selenium was poor because of the importance of dietary exposure in determining the potential for chronic effects. Consequently, there has been recent interest in promoting a tissue-based criterion

*Fax: +1-605-665-9335.
E-mail address: steve_hamilton@usgs.gov (S.J. Hamilton).

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or threshold (DeForest et al., 1999; Brix et al., 2000; Hamilton, 2002).

2. Critique of a tissue-based selenium threshold paper

A recent article by DeForest et al. (1999) reviewed the proposed residue-based toxicity thresholds for freshwater fish. Similar information was given in Brix et al. (2000). They proposed whole-body thresholds of 9 μg/g (all given as dry weight) for warmwater fish and 6 μg/g for larval coldwater anadromous fish, ovary threshold of 17 μg/g for warmwater fish, and fish dietary thresholds of 10 μg/g for warmwater fish and 11 μg/g for larval coldwater anadromous fish. These values are substantially different from those proposed by Maier and Knight (1994; 4.5 μg/g in tissue and 4 μg/g in diet), Lemly (1993a, 1996; 4 μg/g in whole body, 10 μg/g in ovary, and 3 μg/g in diet), and Hamilton (2002; 4 μg/g in tissue).

The DeForest et al. (1999) article seems to have fallen short of their objective of critically reviewing the proposed tissue-based thresholds for freshwater fish because they excluded the results of water-borne studies and selectively discussed results from dietary studies. Their review focused primarily on Lemly (1993a) and they correctly cite several errors in two summary tables. Those errors were corrected in Lemly (1996), which they do not cite. They also did not include information from the review article on selenium toxicology by Maier and Knight (1994) in their review. Maier and Knight (1994) independently proposed threshold concentrations for selenium effects that were similar to those of Lemly (1993a, 1996).

2.1. Errors in Lemly (1993a)

Despite the errors in Lemly (1993a), the proposed tissue-based thresholds were still supported unchanged in Lemly (1996). The residue-based thresholds proposed by DeForest et al. (1999) seem overly high and are not supported by the majority of the selenium literature. The review by Deforest et al. (1999) seems to be incomplete and does not include important articles that further supported the thresholds proposed by Lemly (1996).

Numerous authors cite Lemly (1993a) as the first comprehensive review of the selenium literature and proposal of selenium residue-based thresholds. Few authors cite Lemly (1996), which has conclusions similar to those of Lemly (1993a), but different supporting data in Tables 1 and 2, which had similar supporting citations between the two publications. No one in their publications has noted the difference in values given in Tables 1 and 2 in those two publications (Tables 1 and 2).

2.2. Additional articles supporting Lemly's proposed values

Several articles not cited in Lemly (1993a, 1996) or published later support the 4 μg/g whole-body concentration for toxic effects in fish (Hilton and Hodson, 1983; Cleveland et al., 1993; Lemly, 1993b; Hamilton et al., 1996,2001a, b) (Table 3). This effect concentration in the whole body was supported by Skorupa et al. (1996), who proposed 4–6 μg/g, and Maier and Knight (1994) who proposed 4.5 μg/g.

Likewise, several articles not cited in Lemly (1993a, 1996) or published later support the 3-μg/g dietary toxicity threshold for fish (Cleveland et al., 1993; Lemly, 1993b; Hamilton et al., 1996, 2001a, b) (Table 4). These articles report effect concentrations of 4.6–6.5 μg/g, which suggests a threshold concentration at a lower concentration, i.e., conservatively <4.6 μg/g. Those articles lend further support to the 3 μg/g threshold of effects suggested by Hilton et al. (1980), Lemly (1993a, 1996) and Skorupa et al. (1996) and the 4 μg/g threshold suggested by Maier and Knight (1994).

2.3. Information not cited in Deforest et al.

DeForest et al. (1999) cited selenium contamination problems at Belews Lake, North Carolina, Hyco Reservoir, North Carolina, and Kesterson Reservoir, California, but did not cite selenium contaminant problems at Sweitzer Lake, Colorado (Barnhart, 1957; Birkner, 1978; Butler et al., 1989, 1991, 1994, 1996) or Martin Lake, Texas (Sorensen, 1991).

Similarly, DeForest et al. (1999) cited Van Derveer and Canton (1997) as demonstrating that fish in lotic systems in Colorado were not at risk at water selenium concentrations of approximately 30 μg/L. However, they failed to mention that the articles by Canton and Van Derveer (1997) and Van Derveer and Canton (1997) had incorrectly interpretated exposure survey reports as being exposure-response studies, ignored the importance of the water-borne entry of selenium in aquatic food webs, overlooked key studies from the extensive body of selenium literature, and failed to consider the offstream consequences of proposing high instream selenium standards (Hamilton and Lemly, 1999). Offstream concerns of selenium contamination have also been discussed in Skorupa (1998) and Lemly (1999b). These offstream concerns about selenium contamination were substantiated by Radtke et al. (1988) and Radtke and Kepner (1990), who concluded that elevated selenium concentrations in sediment and biota in the backwaters of the lower Colorado River were carried by water from the upper Colorado River basin and not derived from local agricultural or industrial sources.

DeForest et al. (1999) chose to disregard the results of the SLD diet despite the more realistic exposure scenario

Table 1
Selenium concentrations in tissue associated with toxic effects in fish and aquatic organisms

Species[a]	Tissue	Lemly (1993a) selenium concentration (μg/g)[b]	Lemly (1996) selenium concentration (μg/g)[b]	Effect	Reference
Rainbow trout	Whole body	3	2	Blood changes	Hodson et al. (1980)
	Liver	12	51	Blood changes	Hodson et al. (1980)
	Whole body	5	5	Mortality	Hilton et al. (1980)
	Whole body	4	1	Mortality	Hunn et al. (1987)
Chinook salmon	Whole body	9.5	20	Reduced smolting	Hamilton et al. (1986)
	Whole body	3	2	Reduced growth	Hamilton et al. (1990)
	Whole body	10	5	Mortality	Hamilton et al. (1990)
Fathead minnow	Whole body	6	5	Reduced growth	Ogle and Knight (1989)
	Ovaries	15	24	Reproductive failure	Schultz and Hermanutz (1990)
	Whole body	8	16	Reproductive failure	Schultz and Hermanutz (1990)
Striped bass	Skeletal muscle	14	14	Mortality	Coughlan and Velte (1989)
	Whole body	NG[c]	2	Mortality	Saiki et al. (1992)
Bluegill	Skeletal muscle	20	20	Mortality	Finley (1985)
	Liver	32	34	Mortality	Finley (1985)
	Carcass	8	24	Reproductive failure	Gillespie and Baumann (1986)
	Ovaries	12	23	Reproductive failure	Gillespie and Baumann (1986)
	Whole body	5	5	Mortality	USFWS (1990)
	Whole body	16	19	Reproductive failure	Coyle et al. (1993)
	Ovaries	30	34	Reproductive failure	Coyle et al. (1993)
	Eggs	40	42	Reproductive failure	Coyle et al. (1993)
	Ovaries	10	18	Reproductive failure	Hermanutz et al. (1992)
	Skeletal muscle	10	16	Reproductive failure	Hermanutz et al. (1992)
	Liver	22	29	Reproductive failure	Hermanutz et al. (1992)
	Whole body	12	18	Reproductive failure	Hermanutz et al. (1992)
	Whole body	15	15	Teratogenic defects	Lemly (1993c)
Green alga	Whole organism	20	20	Reduced cell replication	Foe and Knight (1986)
Cyanobacterium	Whole organism	700	394	Reduced chlorophyll a	Kiffney and Knight (1990)
Cladoceran	Whole organism	20	15	Reduced weight	Ingersoll et al. (1990)
	Whole organism	30	32	Reproductive failure	Ingersoll et al. (1990)
Aquatic birds	Liver	10	NG	Reproductive failure	Skorupa et al. (in press)
	Eggs	3	NG	Reproductive failure	Skorupa et al. (in press)

[a] Rainbow trout (*Oncorhynchus mykiss*), Chinook salmon (*Oncorhynchus tshawytscha*), fathead minnow (*Pimephales promelas*), striped bass (*Morone saxatilis*), bluegill (*Lepomis macrochirus*), green alga (*Selenastrum capricornutum*), cyanobacterium (*Anabaena flosaquae*), cladoceran (*Daphnia magna*).
[b] Selenium concentrations on a dry weight basis.
[c] Not given in Lemly (1993a).

compared to the selenomethionine- (SEM) based diet in the studies with chinook salmon (*Oncorhynchus tshawytscha*) (Hamilton et al., 1990). Although there were differences in the diet formulation between the SLD-based diet and the SEM-based diet, reduced survival occurred in both dietary selenium exposures at 9.6 μg/g, and the whole-body selenium residues were remarkably similar (6.5 μg/g in the SLD diet and 5.4 μg/g in the SEM diet). Other adverse effects from the two diets were also similar between the two diets. The slight reduction in growth that occurred earlier and at slightly lower dietary concentrations in the SLD diets compared to the SEM diets was a minor discussion point in Hamilton et al. (1990).

DeForest et al. (1999) cited Brown (1997) to imply that pesticide residues in western mosquitofish (*Gambusia affinis*) used in the San Luis Drain (SLD) diet tested in Hamilton et al. (1990) may have influenced the results of dietary exposures with chinook salmon. The possibility of confounding effects from pesticides or other

Table 2
Concentrations of selenium known to be toxic in the diets of fish and wildlife

Species	Lemly (1993a) dietary selenium concentration (μg/g)[a]	Lemly (1996) dietary selenium concentration (μg/g)[a]	Effect	Reference
Rainbow trout	9	9	Mortality	Goettl and Davies (1978)
	>3	13	Mortality	Hilton et al. (1980)
	10	11	Kidney damage	Hilton and Hodson (1983)
Chinook salmon	6.5	6.5	Mortality	Hamilton et al. (1989)
	5	5	Reduced growth	Hamilton et al. (1990)
Fathead minnow	20	20	Reduced growth	Ogle and Knight (1989)
Striped bass	35	39	Mortality	Coughlan and Velte (1989)
Bluegill	50	54	Mortality	Finley (1985)
	6.5	6.5	Mortality	USFWS (1990)
	NG[b]	5	Mortality	Lemly (1993b)
	13	13	Reproductive failure	Woock et al. (1987)
	16	33[c]	Reproductive failure	Coyle et al. (1993)
Mallard duck[d]	>4	11	Reproductive failure	Heinz et al. (1987)
	>4	9	Reproductive failure	Heinz et al. (1989)

[a] Selenium concentrations on a dry weight basis.
[b] Not given in Lemly (1993a).
[c] Exposure included 10 μg/L in water.
[d] Mallard duck (*Anas platyrhynchos*).

Table 3
Selenium concentrations in tissue associated with toxic effects in fish

Exposure route, species	Tissue	Selenium concentration (μg/g)	Effect	Reference
Diet				
Rainbow trout	Carcass	4.0–4.5	Kidney damage and reduced weight	Hilton and Hodson (1983)
Fathead minnow	Whole body	43–61	Reduced growth	Bennett et al. (1986)
Bluegill	Whole body	25	Mortality	Bryson et al. (1984)
	Whole body	4.3[a]	Mortality	Cleveland et al. (1993)
	Whole body	7.9	Mortality	Lemly (1993b)
Channel catfish	Muscle	3.5	Reduced growth	Gatlin and Wilson (1984)
Razorback sucker	Whole body	3.6–8.7	Mortality	Hamilton et al. (1996)
	Whole body	5.4	Mortality	Hamilton et al. (2001a)
	Whole body	6.1	Mortality	Hamilton et al. (2001b)
Water				
Bluegill	Whole body	5.1[b]	Mortality	Cleveland et al. (1993)
Razorback sucker	Whole body	5.9	Reduced growth	Hamilton et al. (2000)
Bonytail	Whole body	9.4	Reduced growth	Hamilton et al. (2000)

[a] Derived from Fig. 3 in Cleveland et al. (1993).
[b] Derived from Fig. 2 in Cleveland et al. (1993).

contaminants in Kesterson studies has been explored, but none have been reported (i.e., Moore et al., 1990; Ohlendorf et al., 1993). Nevertheless, the toxicity of water from the SLD to fish has been reported and linked to high concentrations of major ions present in atypical ratios, to high concentrations of sulfates, or to both (Saiki et al., 1992).

In fact, in several other selenium contaminant studies, concerns about the influence of other interacting chemicals have been expressed, but none confirmed. For example, Sorensen (1986) stated that "Fish kills [at Belews Lake, NC, and Martin Lake, TX] were considered a direct result of selenium release into the main basin of the lakes because several hundred

Table 4
Selenium concentrations known to be toxic in the diets of fish

Species	Dietary selenium concentration (μg/g)	Effect	Reference
Fathead minnow	55–70[a]	Reduced growth	Bennett et al. (1986)
Bluegill	45[b]	Mortality	Bryson et al. (1984)
	6.5[c]	Mortality	Cleveland et al. (1993)
	5.1[d]	Mortality	Lemly (1993b)
Razorback sucker	2.4–5.1[e]	Mortality	Hamilton et al. (1996)
	4.6[f]	Mortality	Hamilton et al. (2001a)
	4.6[f]	Mortality	Hamilton et al. (2001b)

[a] Rotifers fed selenium-laden algae.
[b] Burrowing mayfly nymphs (*Hexagenia limbata*) collected from Belews Lake, North Carolina.
[c] Selenomethionine incorporated into an Oregon moist pellet diet.
[d] Exposure included water-borne exposure to 4.8 μg/L selenium and winter stress (4c).
[e] Zooplankton collected from Sheppard Bottom ponds 1, 3, and 4 at Ouray NWR, Utah.
[f] Zooplankton collected from three sites near Grand Junction, Colorado.

analyses for metals, metalloids, physiochemical parameters, and pesticides provided essentially negative results except for sufficiently high levels of selenium in the water (about 5 μg/L) to warrant concern.'' Others have reached similar conclusions concerning fishery problems at Belews Lake (Lemly, 1985), water and biota collected from Kesterson Reservoir area, California (Saiki and Lowe, 1987), trace elements in fish from the Merced River, and from Salt Slough, San Joaquin Valley, California (Nakamoto and Hassler, 1992), studies of Hyco Reservoir, North Carolina (Bryson et al., 1984; Gillespie and Baumann, 1986), and phosphate-mining activities in the Blackfoot River watershed of southeastern Idaho (Watson, 1998).

2.4. Water-borne versus dietary exposure

DeForest et al. (1999) did not include results from water-borne studies, but rather limited their analyses to dietary studies. In doing so, they eliminated several studies that relate directly to the tissue threshold of 4 μg/g suggested by Lemly (1993a, 1996), 4.5 μg/g of Maier and Knight (1994), and 4 μg/g of Hamilton (2002). For example, they discard the results of Hunn et al. (1987), who reported adverse effects in rainbow trout (*Oncorhynchus mykiss*), with 5.2 μg/g (assuming 75% moisture) in the whole body because it was a water-borne exposure.

Critically reviewing a residue-based toxicity threshold should include consideration of the results of water-borne studies. A selenium residue in a fish is the result of all exposures, dietary, water-borne, and sedimentary. The exposure routes are concurrent and inseparable. For example, four studies with young fall chinook salmon used different test waters and exposure routes, but had remarkably similar results based on whole-body selenium residues (Hamilton et al., 1986, 1990; Hamilton and Wiedmeyer, 1990). In separate dietary studies, fish were exposed to either SEM in a commercially prepared diet or to the same diet made with fish meal containing elevated concentrations of naturally incorporated seleno-compounds, and reduced growth occurred in fish with whole-body residues of 4.0–5.4 μg/g (Hamilton et al., 1990). In separate water-borne studies, fall chinook salmon were exposed to water-borne selenium in two different water qualities and adverse effects (reduced growth and survival) occurred in fish with whole-body residues of 3.8–4.9 μg/g (Hamilton et al., 1986; Hamilton and Wiedmeyer, 1990). Even though the routes of exposure were different in these studies, a common whole-body selenium residue of 4–5 μg/g was associated with the same adverse effects.

The convergence of adverse effects from water-borne and dietary exposures with a variety of fish suggests that once tissue selenium concentrations reach a critical threshold, regardless of the route of exposure, adverse effects will occur. This supposition is supported by results from several studies, including Hodson et al. (1980), where rainbow trout were exposed to 53 μg/L of selenium for 308 days, but no effects were observed on the survival, growth, condition factor, or several blood and plasma measurements because whole-body selenium residues were only 1.8 μg/g. Hamilton and Wiedmeyer (1990) found no effects on mortality or growth of 2-g fall chinook salmon exposed to water-borne selenium concentrations as high as 140 μg/L for 60 days in a blended brackish water (~1‰ salinity) because whole-body selenium residues were only 1.3 μg/g. Bertram and Brooks (1986) reported no effects on fathead minnow (*Pimephales promelas*) exposed to 7.3 μg/g in the diet and 43.5 μg/L in water for 56 days because whole-body

residues were only 2.2 μg/g. These water-borne and combined diet and water-borne exposure studies help define the upper end of the no-effect tissue threshold (1.3–2.2 μg/g) and the lower end of the effect tissue threshold (3.8–4.0 μg/g). Consequently, a threshold tissue concentration of 4 μg/g would seem reasonable.

DeForest et al. (1999) discussed their supposition that water-borne exposures result in mortality at lower whole-body selenium concentration than dietary exposures, and used Cleveland et al. (1993) as their focal point. The authors did not mention that the water-borne study was conducted with 5-month-old fish and the dietary study with 3-month-old fish, which may have influenced the data interpretation. More importantly, the selenium residue at day 60 linked to reduced mortality in the water-borne study was 4.3 μg/g and in the diet study was 5.1 μg/g. These values are very close to each other, especially considering no standard deviation or standard error was given in Cleveland et al. (1993) for readers to judge the variation of the values. If toxicity were observed at 4.3 and 5.1 μg/g, then some concentration less than these would approach the toxic effects threshold. Consequently, the data in Cleveland et al. (1993) would also support a proposed threshold of 4 μg/g. URS (2000) used a USEPA procedure (Stephan et al., 1985) with data from Cleveland et al. (1993) to calculate a whole-body toxicity threshold for selenium of 3.4 μg/g for the dietary study and 3.3 μg/g for the water-borne study. Thus, they revealed, contrary to DeForest et al. (1999), that there was no difference between water-borne and dietary exposure of bluegill (*Lepomis macrochirus*).

2.5. Coldwater fish versus warmwater fish

Another flaw in the supposition of DeForest et al. (1999) that coldwater fish are more sensitive to selenium toxicity than warmwater fish is that they reviewed selected literature and not a more complete set of selenium publications. The result is that they recommend 6 μg/g as the whole-residue threshold for coldwater fish and 9 μg/g as the threshold for warmwater fish. Several studies in Tables 1 and 3 reveal that whole-body selenium residues of 4–6 μg/g cause adverse effects regardless of whether fish were coldwater or warmwater and regardless of the route of exposure (Hilton et al., 1980; Hilton and Hodson, 1983; Hunn et al., 1987; Hamilton et al., 1990, 1996, 2001a, b; USFWS, 1990; Cleveland et al., 1993; Lemly, 1993a, b, c). DeForest et al. (1999) have not provided an adequate foundation for differentiating the importance of whole-body selenium residues between coldwater fish and warmwater fish. If 4–6 μg/g causes adverse effects in fish, then some concentration lower should be selected as the threshold concentration, i.e., 4 μg/g, not 6 or 9 μg/g as proposed by DeForest et al. (1999).

Two other publications mention the possible differences between coldwater fish and warmwater fish (USDOI, 1998; URS, 2000). Table 32 in USDOI (1998), citing Lemly (1996), gives the no-effect selenium concentration for whole-body residues as <3 μg/g in warmwater fish and <2 μg/g in coldwater fish; the level of concern as 3–4 μg/g and 2–4 μg/g, respectively; and toxicity threshold as >4 μg/g for warmwater and coldwater fish. Although Lemly (1996) does not differentiate between warmwater and coldwater fish, USDOI (1998) cited Lemly (1996) and reported a slight difference in guideline values between warmwater and coldwater fish. Even so, the values in USDOI (1998) were less than those of DeForest et al. (1999), but similar to those reported by others (Maier and Knight, 1994; Hamilton, 2002). USDOI (1998) did not discuss the basis for suggesting a difference between warmwater and coldwater fish in their sensitivity to selenium toxicity.

URS (2000) also suggests the selenium literature has some evidence of coldwater fish being more sensitive to selenium than warmwater fish. They followed the USEPA method (Stephan et al., 1985) employed by DeForest et al. (1999) to calculate the selenium tissue threshold as the geometric mean of the no observable effect concentration (NOEC) and the lowest observable effect concentration (LOEC). Application of the procedure to day 60 data for bluegill from Cleveland et al. (1993) yielded a whole-body toxicity threshold of 3.4 μg/g in their dietary study. Using day 90 data for chinook salmon from Hamilton et al. (1990), URS (2000) reported a whole-body toxicity threshold of 1.5 μg/g. Thus, they concluded there was evidence of differences in sensitivity between warmwater fish (3.4) and coldwater fish (1.5).

However, URS (2000) seems to have used inappropriate data for chinook salmon in their calculation. They note that growth of chinook salmon was reduced at 30 and 60 days of exposure to the 3.2 μg/g SLD diet and then use the whole-body selenium residue at day 90 for that treatment in the USEPA method calculation (i.e., NOEC 0.8 μg/g and LOEC 2.7 μg/g). At day 90, growth was not reduced in the 3.2-μg/g diet treatment, but was reduced in the 5.6-μg/g diet treatment. For day 60 data (NOEC 0.9 μg/g, LOEC 3.3 μg/g) the geometric mean whole-body toxicity threshold is 1.7 for chinook salmon. If day 60 data from Hamilton et al. (1990) were used in the comparison, one might still conclude there was a difference in sensitivity between coldwater fish with a threshold of 1.7 and warmwater fish with a threshold of 3.4 (Cleveland et al., 1993). However, if day 90 data were used, there would be no difference between coldwater fish with a whole-body toxicity threshold of 3.3 (NOEC 2.7 μg/g, LOEC 4.0 μg/g; Hamilton et al., 1990) and warmwater fish with a threshold of 3.9 (NOEC 3.3 μg/g, LOEC 4.6 μg/g; Cleveland et al., 1993). Considering the incongruity between day 60 and day 90

data from these two studies, there seems to be little support for differentiating sensitivity to selenium toxicity between coldwater and warmwater fish.

2.6. Diet selenium threshold

DeForest et al. (1999) proposed a dietary selenium threshold of 11 μg/g for coldwater fish and 10 μg/g for warmwater fish. The available information suggests similar sensitivity between coldwater fish and warmwater fish to dietary selenium toxicity. Tables 2 and 4 reveal that 4.6–6.5 μg/g dietary selenium causes adverse effects in fish regardless of whether they are coldwater species or warmwater species (Hamilton et al., 1989, 1990, 2001a, b; USFWS, 1990; Cleveland et al., 1993; Lemly, 1993a, b). If these dietary concentrations cause adverse effects in fish, primarily mortality, then a lower concentration must be selected as a dietary threshold concentration, i.e., 3 μg/g.

Professional judgment is an important consideration in the interpretation of data that can be frequently difficult and complex, conflicting or ambiguous, or incomplete (USEPA, 1992). Over 20 years ago, Hilton and colleagues conducted several selenium toxicity studies in the late 1970s and early 1980s and, based on their scientific judgment, they hypothesized that >3 μg/g dietary selenium would be harmful to fish over the long term (Hilton et al., 1980). Research in the late 1980s through the early 2000s has substantiated the speculation of John Hilton and colleagues.

3. Divergence of selenium thresholds

Much of the controversy in recent years concerning the selenium criterion for aquatic life and the dichotomy in proposed toxicity thresholds has been between government/academia published papers and nongovernmental papers. It is incumbent on federal government scientists to be an advocate for the environment on behalf of the general public as stated in the mission statement of the US Department of the Interior. Some may state this is a biased position. The chief biologist of the National Biological Service (NBS), and later the Biological Resources Division of the US Geological Survey, Dennis Fenn noted that the line is thin between judgment informed by sound scientific data and speculative judgment based on little data and much personal interest (Fenn and Milton, 1997); yet he concluded NBS scientists must be advocates for the environment (Fenn and Milton, 1997; Fenn, 1997). As Fenn stated, a basic premise of the scientific method is that the scientist has no vested interest in the outcome of the observations.

DeForest et al. (1999) have attempted to critically evaluate selenium thresholds for fish. Others have attempted similar critical evaluations of thresholds using limited datasets for fish (Brix et al., 2000) and birds (Adams et al., 1998, 2000; Fairbrother et al., 1999). Skorupa (1999) critiqued the article by Fairbrother et al. (1999) and noted the selective use of data from several studies that resulted in higher selenium threshold values for birds than proposed by government researchers. Fairbrother et al. (2000), in turn, responded to Skorupa (1999). Skorupa (personal communication) had similar comments on the draft of Adams et al. (1998). Articles that use limited datasets do little to enhance the body of knowledge about selenium. In contrast, to meet our responsibilities as federal researchers for stewardship of our natural resources for the benefit of our citizens, it is incumbent on us to ensure that the full range of relevant information is acquired and presented to the public. This responsibility requires us to not only point out deficiencies of selective information presented in scientific papers such as DeForest et al. (1999) and Brix et al. (2000), yet work to complement their data with the widest possible range of data.

Arguments in the articles by DeForest et al. (1999), Brix et al. (2000), Fairbrother et al. (1999), and Adams et al. (1998) for high threshold values were supported by statistics. However, Skorupa (1999) pointed out how selective use of data points can lead to the arrival at erroneous conclusions. Many of the concerns raised in this critique of DeForest et al. (1999) match those expressed by Stoto (1990) who noted that errors in conclusions could result from incomplete and inaccurate reporting of data, i.e., incomplete and inaccurate review of the selenium literature.

4. Conclusions

DeForest et al. (1999) and Brix et al. (2000) have used selective data to present high toxicity threshold for selenium in the tissue and diet of fish. They have cited older literature containing errors (Lemly, 1993a) while omitting later literature with corrected values (Lemly, 1996), excluded data from publications based on minor justifications, and overlooked key studies from the extensive body of selenium literature. The proposed high-selenium thresholds by DeForest et al. (1999) and Brix et al. (2000) does not stand on equal footing with reviews of more extensive datasets by USDOI (1998), Lemly (1996), Maier and Knight (1994), and Hamilton (2002). Recent studies continue to support the dietary selenium threshold of 3 μg/g and the whole-body selenium threshold of 4 μg/g for fish.

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