ECOTOXICOLOGY AND ENVIRONMENTAL SAFETY 39, 1–9 (1998)
ENVIRONMENTAL RESEARCH. SECTION B
ARTICLE NO ES97 1625

REVIEW

A Position Paper on Selenium in Ecotoxicology: A Procedure for Deriving Site-Specific Water Quality Criteria

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

Received October 13, 1997

This paper describes a method for deriving site-specific water quality criteria for selenium using a two-step process: (1) gather information on selenium residues and biological effects at the site and in down-gradient systems and (2) examine criteria based on the degree of bioaccumulation, the relationship between measured residues and threshold concentrations for reproductive effects in fish and wildlife, and any observed reproductive impacts. Several outcomes are possible-criteria can be left unmodified, adjusted upward by a fixed amount (50%), or adjusted downward by one of three amounts (25, 50, or 75%). A criterion (existing or proposed) is lowered or raised by an amount that is proportional to the magnitude of bioaccumulation and toxic effects present-i.e., the degree of biological hazard. Criteria can be modified under two circumstances: (1) diagnostic residues and toxic effects must be coupled (present) in order to lower a criterion or (2) diagnostic residues and toxic effects must be coupled (absent) in order to raise a criterion. Coupling residues and effects makes the procedure sensitive to the natural inter- and intraspecific variation in bioaccumulation and toxic responses exhibited by fish and wildlife in aquatic ecosystems. The goal is to establish criteria that keep food-chain bioaccumulation below levels that result in toxicity to fish and wildlife. Precautions are given for those attempting to apply the generic EPA model for implementing national water quality criteria to a site-specific selenium criterion. © 1998 Academic Press

INTRODUCTION

The United States Environmental Protection Agency (EPA) conducted a comprehensive review of information on selenium ecotoxicology and published revised national freshwater criteria for selenium in 1987 (USEPA, 1987). Since that time, two divergent lines of evidence and thought have emerged regarding whether the criterion for chronic exposure (5 μg/L) is adequate to protect aquatic life. The first line supports the position that the criterion is too high and should be lowered. For example, Winter Stress Syndrome in fish-a disruption of the normal annual cycle of metabolic and physiological changes in cold weather-can substantially increase their sensitivity to selenium and causeconcentrations approaching the criterion value to become toxic (Lemly, 1993a, 1996). In the western United States, selenium resulting from agricultural irrigation has bioaccumulated to toxic levels in food chains and poisoned fish and aquatic birds when waterborne concentrations were well below 5 μg/L (e.g., Hallock and Hallock, 1993; Hamilton *et al.*, 1996; Lemly, 1995a; Stephens *et al.*, 1992; Skorupa and Ohlendorf, 1991). The second line of evidence supports the position that the criterion is too low and should be raised. For example, fauna1 surveys in Colorado streams revealed the presence of abundant fish populations where waterborne selenium consistently exceeds the criterion. These observations have led some to conclude that the national criterion is overly conservative and that it should be relaxed by as much as 26 μg/L in some locations (e.g., Guglielmone, 1995; Canton and Van Derveer, 1997; Van Derveer and Canton, 1997). The principal disagreements between these two schools of thought were recently discussed by Hamilton and Lemly (in press).

The ongoing controversy over whether the EPA criterion is too high or too low makes a very important point. Regardless of what the national criterion is, there will likely be a concerted effort by some to raise or lower it. EPA policy allows the national criterion to be modified by states on a site-specific basis when biological evidence warrants it (Stephan et al., 1985; USEPA, 1985). Such modifications have been pursued; local criteria-below 5 μg/L in some instances and above 5 μg/L in others-are being considered or have been adopted in several western states, for example, Arizona, California, Colorado, and New Mexico (Arizona, 1992; CEPA, 1992; CSWRCB, 1987; Guglielmone, 1995; New Mexico, 1995). However, there is no widely accepted or published method for site-specific criterion revision-by EPA or other sources-that is targeted specifically at

0147-6513/98 $25.00

selenium. Consequently, criteria are being developed with little pertinent direction or guidance, which has caused some to use insufficient data and make assumptions about fish and wildlife exposure/effects that could introduce significant error into the derivation process (Lemly, 1997a; Hamilton and Lemly, in press). This type of error has important implications. A derivation might indicate that changing a criterion is appropriate when, in fact, it is not environmentally acceptable (raising) or biologically necessary (lowering) to do so. A scientifically credible method is needed that can be broadly and uniformly applied, yet is sensitive to selenium's site-specific variations in environmental cycling and biological effects. The procedure described in this paper was developed to address that information need.

RATIONALE

There are two primary considerations in developing site-specific water quality criteria for selenium: (1) local conditions must be evaluated and the resultant information used to decide if a modified criterion is necessary and (2) if changes are warranted, the existing or proposed criterion must be modified by an appropriate amount. A simple, two-step process is proposed for deriving criteria for chronic exposure.

The major principle underlying this process is that reproductive effects in fish and aquatic birds are the most sensitive biological indicators of aquatic ecosystem-level impacts of selenium (Lemly, 1993b, c, 1997b, c; Ohlendorf, 1989; Skorupa *et al*., 1996). Selenium is passed from parents to their offspring in eggs, where it can kill developing embryos outright or induce a variety of lethal or sublethal teratogenic deformities (e.g., Hoffman *et al.*, 1988; Lemly, 1993c). However, parents can consume a selenium-laden diet and experience partial or complete reproductive failure without exhibiting symptoms of selenium toxicosis themselves (Lemly, 1985a, 1997c; Ohlendorf, 1989). Moreover, aquatic food organisms of wildlife strongly bioaccumulate selenium—hundreds to thousands of times the waterborne concentration-but are unaffected by tissue residues that are high enough to cause reproductive failure when consumed by fish and aquatic birds (Lemly, 1985a). Thus, bioaccumulation in aquatic food chains and dietary transfer to eggs cause otherwise innocuous concentrations of waterborne selenium to become toxic. Establishing water criteria that prevent this degree of bioaccumulation in aquatic food chains is the goal of the procedure given in this paper.

The presence or absence of reproductive effects, coupled with information on the degree of accumulation in major ecosystem components, can be used to precisely evaluate local conditions and derive an appropriate site-specific water quality criterion for selenium. This process can be applied consistently and uniformly, regardless of location or habitat type-i.e., in wetlands (seasonal or permanent), streams, rivers, lakes, reservoirs, and other impoundments.

PROCEDURE

Step One

The first step involves gathering information on selenium residues and biological effects. This is done to provide an empirical foundation for evaluating a criterion, which may be national, state, or local, and which may be currently in place or under consideration. A decision tree (Fig. 1) is used to guide the process. It specifies what types of information are necessary for the evaluation and also identifies conditions under which the criterion should be modified. The selenium criterion value is examined in the context of site-specific selenium concentrations and observed or potential toxicity to fish and aquatic birds. Several outcomes and conclusions are possible-the criterion may be appropriate as it is, or it may be inappropriate and need to be raised or lowered. Guidance for measuring selenium residues and interpreting tissue concentrations and biological effects thresholds is available in Table 1 and Fig. 1. If the empirical evidence indicates that a revised criterion is necessary (guided by the decision tree), the investigator should proceed with step two.

Step Two

A simple procedure is followed to determine what the new criterion should be. This technique is based on relationships between the degree of bioaccumulation present at the site (measured selenium residues), the known threshold concentrations for reproductive effects in fish and wildlife (diagnostic residues for teratogenesis and embryo mortality), and any observed reproductive impacts at the site (teratogenesis or embryo mortality).

A more restrictive (lowered) criterion is indicated when the existing or proposed criterion value for waterborne selenium is *not* exceeded, but site-specific residues equal or exceed diagnostic toxic thresholds and reproductive effects are observed. The criterion value should be lowered by an amount that corresponds to the magnitude of bioaccumulation present. Three sets of conditions are possible-each signifies a different level of toxic threat which, in turn, indicates a different amount of revision to the criterion: (1) If residue values fall partly below the threshold toxic ranges (see Diagnostic residue, Table 1) and partly withm the threshold toxic ranges, then the criterion should be lowered by 25%; (2) If residue values fall entirely within the threshold ranges or span the ranges such that some are below, some are within, and some are above, then the criterion should be lowered by 50%; or (3) If all of the residue values exceed the upper end of the threshold ranges then the criterion should be lowered by 75%. Criteria are lowered by an amount that is proportional to the degree of biological hazard present. This process is followed in each instance when a more restrictive criterion is indicated.

No change in the criterion is necessary if: (1) the criterion is not exceeded and no selenium problem is suspected; (2) the criterion is not exceeded, a selenium problem is suspected (diagnostic residues may or may not be present), but there are no reproductive effects; (3) the criterion is exceeded and residues exceed or fall within the toxic threshold ranges or are above the ranges for uncontaminated aquatic systems (Table 1); or (4) the criterion is exceeded, residues are within the ranges for uncontaminated systems, but down-gradient impacts are possible (Fig. 1).

If the residues fall within the ranges of values for reference conditions in uncontaminated systems, yet the existing or proposed criterion value is exceeded, then it may be reasonable to consider raising it. However, raising a criterion is contingent upon firmly establishing that there would be no down-gradient impacts to other aquatic systems, wetlands, or selenium-sensitive species of fish and aquatic birds. If these requirements are met, then the criterion should be raised by 50%. In summary, criteria can be left unmodified, adjusted upward by a fixed amount (50%), or adjusted downward by one of three amounts (25, 50, or 75).

Modified criterion values should be rounded to the nearest 0.5 μg/L. A criterion that falls equidistant from two values is rounded down if the criterion is being lowered and up if the criterion is being raised. For example, a criterion of 5 μg/L that is lowered by 25% (1.25 μg/L) becomes 3.5 μg/L (5.0–1.25 = 3.75, rounded down to 3.5). Lowering the same criterion by 50% yields a value of 2.5 μg/L, and lowering it by 75% results in a final criterion of 1.0 μg/L (1.25 rounded down to 1.0). Criteria that are raised are modified by adding 50% of the criterion value and then rounding up, if necessary, to determine the final value. In theory, criteria could be set at smaller intervals, e.g., 0.1 μg/L. However, it would likely be impossible to scientifically justify or reliably monitor and enforce such criteria. For example, the range of bioaccumulation and effects displayed among fish and aquatic birds (even among the most sensitive species) would offset the perceived benefits of setting criteria at 2.2 and 2.6 μg/L rather than 2.0 and 2.5 μg/L. Rounding to 0.5 μg/L intervals should accomplish the goal of criterion revision—i.e., achieve the desired reduction or prevention of selenium bioaccumulation/effects in aquatic ecosystems—yet also be practical for monitoring, compliance, and treatment/remediation considerations. Diagnostic selenium residues and examples of measured concentrations/effects and appropriate modifications to criteria are given in Table 1.

JUSTIFICATION

What Is an Appropriate Modification of a Criterion?

The main objective of the procedure given in this paper is to modify a criterion (by default, the EPA national criterion) by an *appropriate* amount. Appropriate, as used here, is the extent necessary to prevent bioaccumulation from reaching levels that induce toxic effects in fish and wildlife. A criterion that is appropriate will be both effective and reasonable—i.e., it will provide the needed level of protection to on-site biota and down-gradient habitats without being too conservative or too liberal. This translates to a criterion which neither requires that reference conditions be achieved in every instance nor permits toxic impacts to biota. For example, in situations when a criterion is not exceeded and diagnostic residues are present but no effects are evident, the decision tree indicates that no modification is necessary (Fig. 1). Conversely, when diagnostic residues are present without effects but the criterion is exceeded, no change is indicated. This, in effect, sets two major conditions for triggering criterion modification: (1) diagnostic residues and toxic effects must be coupled (present) in order to lower a criterion, and (2) diagnostic residues and toxic effects must be coupled (absent) in order to raise a criterion. Coupling residues and effects makes the procedure sensitive to the natural inter- and intraspecific variation in bioaccumulation and toxic responses exhibited by fish and wildlife in aquatic ecosystems.

An additional precaution is necessary if the scenario under evaluation consists of nonexceeded criterion, diagnostic residues present, but no observed toxic effects. In this case the recommended decision is to monitor reproduction but not lower the criterion. However, it is essential that all of the fish and aquatic bird species that are characteristic of the site—i.e., typical for the habitat type and are commonly found in nearby reference locations-be included in the assessment of "effects." Selenium-sensitive species can experience reproductive failure and be virtually eliminated, while at the same time some of the most tolerant species remain and only exhibit elevated tissue residues (Lemly, 1985a). This scenario could lead to the false impression that there are no effects and thus no need to lower the criterion when, in fact, selenium-sensitive species have been severely affected. This type of error is also possible when the scenario consists of exceeded criterion but no diagnostic residues or toxic effects among the species that are present. It is possible that sensitive species have been eliminated and the tolerant species that remain exhibit very low bioaccumulation, which may partially explain their ability to tolerate selenium. This could lead to the false assumption that increasing the criterion is appropriate. Thus, in certain situations the biota that "should be pesent" must be considered, particularly if they represent known selenium sensitive species, in addition to what is actually present.

Why Use Percentages and Ranges.7

Percentages are used because this appears to be the simplest practical way of modifying criteria while also addressing the main objective from both an ecological and

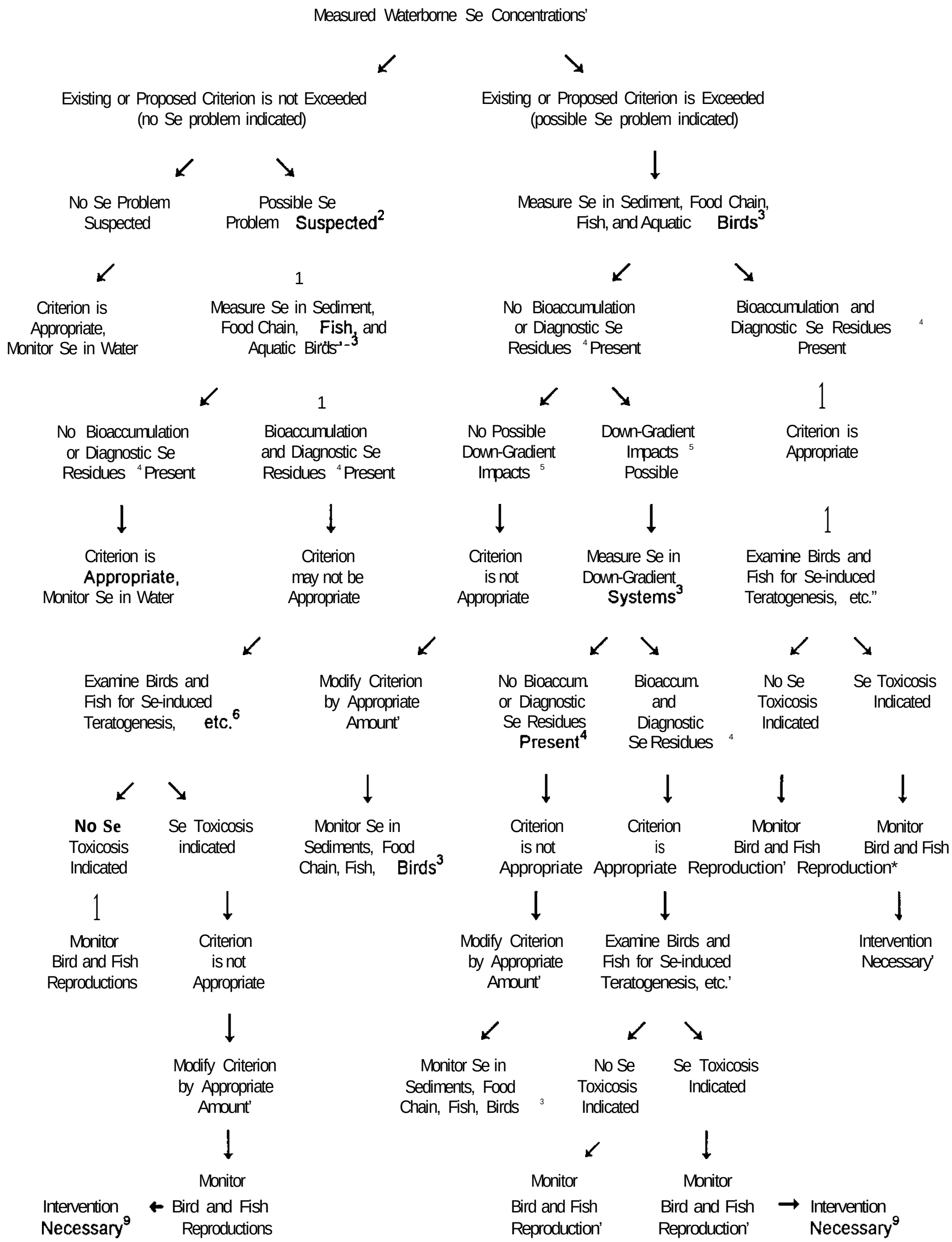

FIG. 1. A decision tree for determining if modification of a selenium water quality criterion is necessary. Numbers indicate the following: [1]A sufficient number of samples to provide some spatial and temporal integration; [2]Possible problem suspected based on observations and available information collected from the site: [3]Measurements according to guidelines and procedures given by Lemly (1995b) and Skorupa and Ohlendorf (1991); [4]Residues that equal or exceed the toxicity thresholds given in Table 1; [5]Down-gradient impacts to other aquatic systems, wetlands, or selenium-sensitive species in the watershed, including confluence with receiving waters that cross state lines or other Jurisdictional boundaries; [6]Teratogenic deformities and/or embryo mortality, measured according to guidelines and procedures given by Lemly (1993c, 1997b), Hoffman *et al.* (1988), and Ohlendorf et *al.* (1986a, 1986b, 1988); [7]Modify according to procedures given in the text: [8]Monitor for evidence of teratogenic deformities and/or embryo mortality and associated selenium residues in eggs, embryos, and larvae: and [9]Regulatory and management actions to effectively control or modify the source of selenium.

a regulatory perspective. The decision tree specifies that selenium concentrations and effects should be monitored in each case when criterion modification is indicated (Fig. 1). It will quickly become apparent if a criterion derived using the percentage method is too conservative or too liberal. The monitoring requirements in the decision tree function as feedback loops that provide an ongoing field test of the effectiveness of the criterion. On-site monitoring of residues

TABLE 1
Selenium Residues that Signify the Thresholds for Reproductive Effects in Fish and Aquatic Birds

Type of Sample	Diagnostic residue[a, b] (reference')	Measured concentration[b] and toxic effect	Appropriate change to a criterion of 5 μg/L[d]
Sediments	2 4 (< 1)	1–5, no effect	None
Food chain'	2–4 (< 2)	3 8, embryo mortality in birds	Lower by 2.5 μg/L (50% decrease)
Fish			
Whole-body	5–7 (< 3)	6–9, no effect	None
Muscle (fillets)	6–8 (< 3)	5–10, no effect	None
Liver	15–20 (< 8)	4–7, no effect	None
Eggs	5–10 (< 3)	6–1 7, terata	Lower by 2.5 μg/L (50%)
Larvae/fry	8–12 (< 3)	512, terata	Lower by 1.5 μg/L (25%)
Aquatic birds			
Muscle	15–20 (< 3)	7 19, no effect	None
Liver	20–30 (< 10)	23–32, terata	Lower by 2.5 μg/L (50%)
Eggs	6–15 (< 3)	4 9, no effect	None
Embryos	10–15 (< 3)	16 25, terata	Lower by 4.0 μg/ L (75%)

Note. Site-specific concentrations and biological effects arc compared to diagnostic residues to determine if modification of a water quality criterion is necessary. The measured concentrations and indicated changes are given as examples of how to appropriately lower a criterion of 5 μg/L, assuming that site-specific waterborne concentrations are below or equal to 5 μg/L. Consult Fig. 1 and the text for guidance on raising criteria.

“Residues that indicate ecosystem contamination is sufficient to cause reproductive impairment of fish and aquatic birds (teratogenic deformity and/or mortality of embryos and larvae/fry).

“All values are in μg/g (parts-per-million) dry weight. Values for reference and contaminated habitats were compiled from data given by Hoffman *et al.* (1988), Lemly (1985a. 1993b,1993c,1997b,1997c), Ohlendorf (1989), Ohlendorf *et al.* (1986a. b, 1988), Skorupa and Ohlendorf(l991). and Skorupa et *al.* (1996).

‘Concentrations typical for uncontaminated aquatic systems.

“Lowering a criterion is indicated only if reproductive effects are present in combination with diagnostic residues; raising a criterion is indicated only if there are no effects, reference levels of residucs are present, and there are no possible down-gradient impacts (Fig. 1).

‘Organisms commonly utilized as food by fish and wildlife, such as macroinvertebrates, aquatic plants and seeds, and forage fish.

and biological effects is an essential part of the validation process for site-specific criteria.

There is a very narrow range between typical reference waterborne concentrations of selenium in aquatic systems (0.1-0.3 μg/L), those that can cause bioaccumulation and toxicity in fish and wildlife (1–3 μg/L), and the current national and state criteria for selenium (2-20 μg/L). Moreover, the values for site-specific criteria that are likely to be developed by states and approved by EPA probably fall within the range l-20 μg/L. Within this narrow range, the most straightforward way to address necessary changes in environmental selenium residues is by using percentages (25, 50 and 75%) that are linked to different degrees of biological effects. This technique is appropriate because the magnitude of teratogenic deformity and/or embryo mortality, and resultant ecosystem-level impacts to fish and aquatic birds, are distinctly greater in cases when a 75% reduction in a criterion is indicated (all residues greater than upper end of the toxicity threshold ranges) than when a 25% reduction is indicated (residues partly below and partly within the threshold ranges).

The use of ranges for threshold values rather than a single number makes the criterion modification process sensitive to natural variation in bioaccumulation and toxic responses of biota. It prevents the process from being overly conservative in cases when a lowered criterion is indicated. Using reference values prevents the process from being too liberal in cases when it is appropriate to raise a criterion. Increases are contingent upon finding reference levels of bioaccumulation. Thus, modification is always linked to a “no effect” range. Allowing a 50% increase in a criterion is reasonable if there is no evidence of bioaccumulation or threats to down-gradient systems. Moreover, the feedback loop provided by monitoring will indicate if further revisions (upward or downward) should be undertaken once a criterion is raised by this amount. This is also true for criteria that are lowered. It is possible that more than one revision will be necessary before an appropriate criterion is achieved.

IMPLEMENTING CRITERIA

EPA provides states the option of developing site-specific criteria and regulations governing their implementation if adequate justification is given. Establishing criteria that are sensitive to selenium’s local variations in aquatic cycling and toxic effects should be the goal of the derivation process at the state and local levels. The main purpose of this paper is to provide a procedure for deriving criteria at those levels.

However, it is also important to consider how criteria will be implemented and enforced-i.e., whether there will be provisions for averaging concentrations over several days in order to meet criterion levels, whether periodic exceedance of criteria will be allowed, and whether mixing zones will be used to dilute effluents and achieve criterion concentrations. In many cases, states may choose to follow the example set by EPA (e.g., Stephan *et al.*, 1985; USEPA, 1985, 1987), which allows averaging, exceedances, and mixing zones to factor into the implementation of criteria. However, the guidance provided by EPA is generic and can greatly reduce the effectiveness of site-specific selenium criteria. It is essential that selenium's profile of environmental cycling and toxicity be used to formulate implementation policies at state and local levels. There are several important precautions that should be taken by water quality regulators if they intend to rely on EPA guidance documents to implement site-specific selenium criteria.

EPA guidelines allow the national criterion for chronic exposure to be exceeded periodically (once every 3 years, on average) as long as the 4-day average concentration is 5 μg/L or less (USEPA, 1987). During exceedances, the permissible ambient (ecosystem-wide) waterborne concentration can be as high as 20 μg/L (Fig. 2). Stephan *et al.* (1985) gives the rationale for this approach: "the averaging period of four days was selected by the U.S. EPA on the basis of data concerning how rapidly some aquatic species react to increases in the concentrations of some pollutants, and three years is the Agency's best scientific judgement of the average amount of time aquatic ecosystems should be provided between excursions." The wording of the statement reveals that this is a generic model for contaminant exposure-response and associated derivation of criteria—i.e., the words "some aquatic species" and "some pollutants." The EPA model is flawed when applied to selenium because little, if any, data specific to selenium were used to develop it in the early 1980s, and no attempt has been made by the Agency to test or validate its assumptions using selenium data that have become available since that time.

Some of the specific flaws that invalidate the EPA model when it is applied to selenium are: (1) The EPA guidance document clearly indicates that the process for national criteria is molded to fit wastewater treatment facilities (POTWs) that discharge a point source into a flowing receiving water (Stephan *et al.*, 1985, pp. 11–12). However, the most widespread threats of selenium poisoning in aquatic life are in lentic systems (reservoirs, wetlands, and off-channel bays and impoundments) and are due to selenium sources other than POTWs (Lemly, 1993b). The environmental dynamics of selenium in lentic ecosystems are quite different than the riverine conditions used for the EPA model. (2) The 4-day average is based on organism responses to waterborne exposure alone. However, food chain bioaccumulation and dietary intake resulting from short-term pulses are

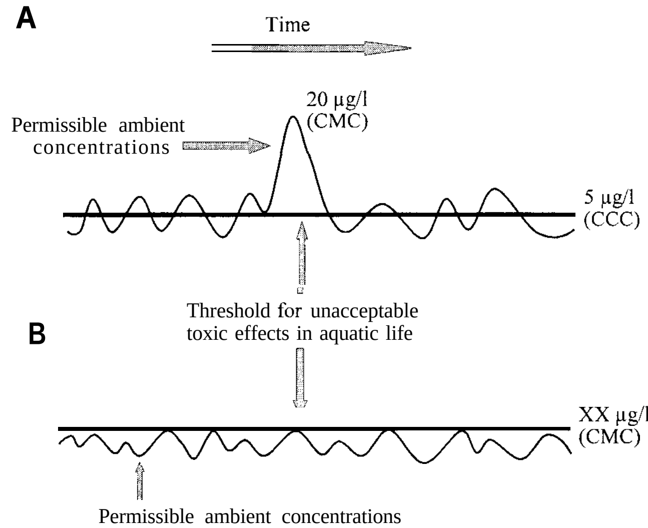

FIG. 2. Contrasts between the existing EPA national water quality criterion for selenium (chronic exposure, A) and the process for deriving site-specific criteria (this paper, B). A principal difference is that in implementing national criteria, EPA guidelines allow 4-day averages and exceedances up to the Criterion Maximum Concentration (CMC, 20 μg/L). This can offset the protection to aquatic life that is afforded by the Criterion Continuous Concentration (CCC, 5 μg/L). To provide full protection, site-specific criteria set biologically based concentration limits as the CMC and do not make provisions for averages, exceedances, or mixing zones (see text for details).

equally or perhaps more important in causing selenium toxicity to aquatic life (Lemly, 1985a, 1993b, 1997c). This component of selenium cycling is overlooked in the EPA model. Moreover, exposure-bioaccumulationresponse times for selenium in fish and aquatic birds (waterborne or dietary intake) are on the order of weeks or months rather than 4 days (e.g., Lemly, 1982, Heinz *et al.*, 1988; Coyle *et al.*, 1993; Heinz and Fitzgerald, 1993)—the EPA model assumptions are not correct. (3) The concentrations of waterborne selenium allowed by EPA during exceedances (up to 20 μg/L) are not environmentally acceptable for lentic systems or lotic systems that will deliver selenium into off-channel bays, wetlands, reservoirs, or other down-gradient lentic systems. Studies such as those by Cumbie and Van Horn (1978), Bryson *et al.* (1984), Lemly (1985), Gillespie and Baumann (1986), and Hamilton *et al.* (1996) reported that concentrations of 10–20 μg/L can quickly reach dietary levels that are toxic to fish and aquatic birds. Consider, for example, a scenario in which an exceedance causes waterborne selenium in a reservoir or wetland to reach 15 μg/L—an acceptable concentration in the EPA model (Fig. 2). By the time ambient locations reach this level, the entire "bioaccumulation engine" of the ecosystem will have been fueled by the influx of new selenium, which substantially escalates the toxic threat to aquatic life (Lemly, 1985b). (4) The 3-year period between excursions (exceedances), although perhaps reflecting the best scientific judgement available for *some* pollutants in the early 1980s, is not appropriate for selenium. Once an aquatic ecosystem has captured the selenium dose delivered by an exceedance, it can continue to cycle it tightly within the system for many years. For example, studies indicate that the recovery period for reservoirs contaminated by 10 μg/L selenium could be > 10 years, perhaps several decades, due to recycling of selenium from sediments into benthic–detrital food chains and associated dietary and reproductive toxicity to fish (Garrett and Inmann, 1984; Lemly, 1997c).

Similar problems are evident with the use of dilution or mixing zones, which are areas exempt from ambient criteria. This concept was developed for application to flowing waters (Stephan *et al*., 1985). It has no credible basis for application to selenium in lentic/wetland systems because the "dilution zone" may constitute the entire body of open water. Even in riverine habitats, the notion of mixing zones is a totally artificial process because EPA has not referenced data verifying that a mixing zone can effectively dilute a selenium-laden effluent and also be environmentally compatible with fish and wildlife habitat uses, which it must be under Federal statutes such as the Migratory Bird Treaty Act and the Endangered Species Act (Margolin, 1979). Selenium strongly bioaccumulates in food organisms and makes the dilution zone an area of extremely high exposure for fish and wildlife. On the contrary, several case studies found that using mixing zones to dilute seleniferous water creates more biological hazards than it resolves (e.g., Skorupa, 1998). The apparent benefits gained by achieving target concentrations in a mixing zone may be more than offset by detrimental effects that are caused by other aspects of the selenium cycle. The threat of toxic impacts overrides the need to attempt "dilution as a solution."

Given these flaws, it is important to closely examine the rationale for, and distinction between, national and site-specific criteria. EPA criteria are intended to provide protection for most aquatic species most of the time, not everything all of the time (Stephan *et al*., 1985). Because of this basic caveat, as well as the fact that there are differences in ecosystem and aquatic species sensitivity to selenium, there may be a plausible argument for allowing some leeway in meeting the national criterion-i.e., a reasonable averaging of concentrations over time if monitoring indicates that there are no biological effects (but not 20 μg/L exceedances). However, at a local level, the national criterion's intent to protect "most species" still leaves large gaps that could lead to substantial inconsistencies (toxic effects at or below the criterion level, lack of effects above the criterion). Site-specific water quality criteria should reflect the sensitivity of local biota and close the gaps.

Site-specific criteria for selenium should designate a biologically based concentration limit using the procedure described in this paper (Figs. 1–2). If full protection of aquatic life is desired, then there should be no provision for averages, exceedances, or mixing/dilution zones in the implementation of criteria.

EXAMPLES OF CRITERION DERIVATION

Example l-Lowering a Criterion

The existing criterion is 5 μg/L and the following site-specific information is available (μg/g concentrations are in dry weight):

Waterborne concentrations of selenium are 1–3 μg/L.

Concentrations of selenium in sediments are 224 μg/g.

Concentrations of selenium in aquatic food-chain organisms are 1–7 μg/g.

Concentrations of selenium in randomly sampled aquatic bird eggs are 4412 μg/g.

Teratogenic deformities occurred in 2% of randomly sampled aquatic bird eggs.

Embryo mortality occurred in 4% of randomly sampled aquatic bird eggs.

Concentrations of selenium in whole-body samples of fish are 6610 μg/g.

Teratogenic deformities occurred in 5% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 8-12 μg/g.

Evaluation. The site-specific residues span the toxic threshold ranges-some are below, some are within, and some are above-and reproductive effects are present.

Conclusion. These conditions warrant a 50% (2.5 μg/L) reduction in the criterion. The final criterion for chronic exposure is set at 2.5 μg/L.

Example 2—Raising a Criterion

The existing criterion is 5 μg/L and the following site-specific information is available (μg/g concentrations are in dry weight):

Waterborne concentrations of selenium are 5-12 μg/L.

Concentrations of selenium in sediments are < 1 μg/g.

Concentrations of selenium in aquatic food-chain organisms are < 2 μg/g.

Concentrations of selenium in randomly sampled aquatic bird eggs are < 3 μg/g.

Teratogenic deformities occurred in 0% of randomly sampled aquatic bird eggs.

Embryomortality occurred in < 1% of randomly sampled aquatic bird eggs.

Concentrations of selenium in whole-body samples of fish are < 3 μg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are < 3 μg/g.

Evaluation. The site-specific residues were equivalent to reference levels and no reproductive effects were evident. Samples from down-gradient receiving waters and wetlands indicated the same pattern-no bioaccumulation or effects.

Conclusion. The criterion for chronic exposure should be raised by 50%, to a final value of 7.5 μg/L.

Example 3—No Modification Necessary

The existing criterion is 5 μg/L and the following site-specific information is available (μg/g concentrations are in dry weight):

Waterborne concentrations of selenium are 2-5 μg/L.

Concentrations of selenium in sediments are 1-4 μg/g.

Concentrations of selenium in aquatic food-chain organisms are l-6 μg/g.

Concentrations of selenium in randomly sampled aquatic bird eggs are 2–8 μg/g.

Teratogenic deformities occurred in 0% of randomly sampled aquatic bird eggs.

Embryo mortality occurred in < 1% of randomly sampled aquatic bird eggs.

Concentrations of selenium in whole-body samples of fish are 3–6 μg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 3-8 μg/g.

Evaluation. Residues of selenium are at toxicity thresholds but no reproductive effects are evident (the fraction of a percentage of embryo mortality present is considered a background level and not due to selenium).

Conclusion. No change to the criterion for chronic exposure is indicated but monitoring of bird and fish reproduction—i.e., sampling for evidence of teratogenesis and embryo mortality-and associated selenium residues is recommended.

Example 4-Examining a Proposed Criterion

The proposed criterion for chronic exposure is 15 μg/L and the following site-specific information is available (μg/g concentrations are in dry weight):

Waterborne concentrations of selenium are 10–20 μg/L.

Concentrations of selenium in sediments are l-2 μg/g.

Concentrations of selenium in aquatic food-chain organisms are 1–3 μg/g.

Concentrations of selenium, terata, and embryo mortality in randomly sampled aquatic bird eggs are unavailable because few aquatic birds nest at the site.

Concentrations of selenium in whole-body samples of fish are 2-5 μg/g.

Teratogenic deformities occurred in 0% of fish from ichthyoplankton samples.

Concentrations of selenium in ichthyoplankton samples are 3-6 μg/g.

Possible impacts to down-gradient systems-unknown.

Evaluation. No reproductive effects are evident in fish and potential threats to aquatic birds are likely minimal because few of them were found to nest or feed at the site. However, the habitat usage and selenium status of down-gradient receiving waters and associated wetlands have not been characterized. Moreover, concentrations of selenium in fish are at the lower end of the diagnostic toxicity range.

Conclusion. The proposed criterion of 15 μg/L is rejected because of possible threats to down-gradient systems, as well as the presence of elevated selenium residues in fish and food-chain organisms. Monitoring of selenium residues in fish and down-gradient systems is recommended.

CONCLUSIONS

During the past two decades, selenium has gained widespread attention as an important aquatic contaminant, and it is currently classified as one of EPA's priority pollutants. EPA published a revised (lowered) national freshwater criterion for selenium in 1987 but there is considerable disagreement as to the applicability of the national criterion at a state and local level-some believe it should be lowered, some believe it should be raised. This has led to more and more instances of states and municipalities attempting to develop site-specific criteria. However, there are no published or widely accepted methods available from EPA or other sources that give specific guidance for selenium. The procedure given in this paper should fill that information gap. It provides a simple, straightforward method for using information on the selenium status of aquatic ecosystems and resultant threats/impacts to biota as the basis for deriving criteria. The method should be a useful tool for those involved in water quality assessment and regulation throughout North America and abroad.

REFERENCES

Arizona (State of Arizona). (1992). *Arizona Administrative Code. Title 18. Environmental Quality. Chapter I I. Water Quality Boundaries and Standards. Supplement 92-4.* Arizona Department of Environmental Quality, Phoenix, AZ.

Bryson, W. T.. Garrett, W. R., Mallin, M. A. MacPherson, K. A., Partin, W. E., and Woock, S. E. (1984). *Roxboro Steam Electric Plant 1982 Environmental Monitoring Studies. Vol. 2. "Hyco Reservoir Bioassay Studies."* Technical Report. Carolina Power and Light Company, New Hill, NC.

Canton, S. P., and Van Derveer, W. D. (1997). Selenium toxicity to aquatic life: An argument for sediment-based water quality criteria. *Environ. Toxicol. Chem.* 16, 125551259.

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

Coyle, J. J., Buckler, D. R., Ingersoll, C. G., Fairchild, J. F., and May, T. W. (1993). Effect of dietary selenium on the reproductive success of bluegills *(Lepomis macrochirus). Enciron. Toxicol. Ckem.* 12, 551–565.

CSWRCB (California State Water Resources Control Board). (1987). *Regulation of Agricultural Drainage to the San Joaquin River*. Technical Report WQ-85-01. California SWRCB, Sacramento, CA.

Cumbie, P. M., and Van Horn, S. L. (1978). Selenium accumulation associated with fish mortality and reproductive failure. *Proc. Annu. Conf. Southeast. Assoc. Fish & Wildlife Agencies 32*, 612–624.

Garrett, G. P., and Inmann, C. R. (1984). Selenium-induced changes in the fish population of a heated reservoir. *Proc. Annu. Conf. Southeast. Assoc.* Fish *& Wildlife Agencies* 38, 291–301.

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

Guglielmone, K. (Ed.). (1995). *Proceedings of A Forum on Selenium Ecotoxicology*. Brown and Caldwell, Denver, CO.

Hallock, R. J., and Hallock, L. L. (Ed.). (1993). *Detailed Study of Irrigation Druinage in and near Wildlife Management Areas, West-Central Nevada, 1987–90. Part B. Effect on Biota in Stillwater and Fernley Wildlife Management Areas and Other Nearby Wetlands.* Water-Resources Investigations Report 92-4024B. U.S. Geological Survey, Carson City, NV.

Hamilton, S. J., and Lemly, A. D. Do not let sediment muddy up the water quality criteria for selenium. *Environ. Toxicol. Chem.*, in press.

Hamilton, S. J., Buhl, K. J., Bullard, F. A., and McDonald, S. F. (1996). *Evaluation of Toxicity to Larval Razorback Sucker of Selenium-Laden Food Organisms from Ouray NWR on the Green River, Utah.* Technical Report. U.S. Geological Survey, Midwest Science Center, Ecotoxicology Research Station, RR 1, Yankton, SD.

Heinz, G. H., and Fitzgerald, M. A. (1993). Overwinter survival of mallards fed selenium. *Arch Enciron. Contam. Toxicol. 25,* 90–94.

Heinz, G. H., Hoffman, D. J., and Gold, L. G. (1988). Toxicity of organic and inorganic selenium to mallard ducklings. *Arch. Environ. Contam. Toxicol. 17,* 561–568.

Hoffman, D. J., Ohlendorf. H. M., and Aldrich, T. W. (1988). Selenium teratogenesis in natural populations of aquatic birds in central California. *Arch. Environ. Contam.* Toxicol. 17, 519–525.

Lemly, A. D. (1982). Response ofjuvenile centrarchids to sublethal concentrations of waterborne selenium. 1. Uptake, tissue distribution, and retention. *Aquat. Toxicol. 2, 2355252.*

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

Lemly, A. D. (1985b). Ecological basis for regulating aquatic emissions from the power industry: The case with selenium. *Regul. Toxicol. Pharmacol. 5, 4655486.*

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

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

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

Lemly, A. D. (1995a). *Hazard of Selenium to Fish and Migratory Birds at Ouray National Wildlife Refuge, Utah.* Technical Report. Department of Fisheries and Wildlife Sciences, Virginia Tech University, Blacksburg, VA.

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

Lemly, A. D. (1996). Winter Stress Syndrome: An important consideration for hazard assessment of aquatic pollutants. *Ecotoxicol. Enciron. Saf. 34,* 223–227.

Lemly, A. D. (1997a). Environmental hazard of selenium in the Animas La Plata water development project. *EcotoxicoL Environ. Saf. 37, 92-96.*

Lemly, A. D. (1997b). A teratogenic deformity index for evaluating impacts of selenium on fish populations. *EcotoxicoL Environ. Saf. 37, 2599266.*

Lemly, A. D. (1997c). Ecosystem recovery following selenium contamination in a freshwater reservoir. *Ecotoxicol. Enciron. Saf. 36, 2755281.*

Margohn, S. (1979). Liability under the Migratory Bird Treaty Act. *Eco. Law Q. 7, 98991010.*

New Mexico (State of New Mexico). (1995). *Standards for Interstate and Intrastate Streums. New* Mexico Water Quality Control Commision, Santa Fe, NM.

Ohlendorf, H. M. (1989). Bioaccumulation and effects of selenium in wildlife. In *Selenium in Agriculture and the Environment.* Special Publication No. 23, pp. 1333177. Soil Science Soc. Am., Madison, WI.

Ohlendorf, H. M., Hoffman, D. J., Saiki, M. K., and Aldrich, T. W. (1986a). Embryonic mortality and abnormalities of aquatic birds: Apparent impacts of selenium from irrigation drainwater. Sci. *Total Environ. 52, 49963.*

Ohlendorf, H. M., Hothem, R. L., Bunck, C. M., Aldrich, T. W., and Moore, J. F. (1986b). Relationships between selenium concentrations and avian reproduction. *Trans. N. Am. Wildlife Nat. Resour. Conf.* 51, 330–342.

Ohlendorf, H. M., Kilness, A. W., Simmons, J. L., Stroud, R. K., Hoffman, D. J., and Moore, J. F. (1988). Selenium toxicosis in wild aquatic birds. *J. Toxicol. Environ. Health 24,* 67–92.

Skorupa, J. P. (1998). Selenium poisoning of fish and wildlife in nature: Lessons from twelve real-world examples. In *Environmental Chemistry of Selenium* (W. T. Frankenberger, and R. A. Engberg, Eds.), pp. 3 15–354. Marcel-Dekker, New York.

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

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

Stephan, C. E., Mount, D. I., Hansen, D. J., Gentile, J. H., Chapman, G. A., and Brungs, W. A. (1985). *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses.* Publication PB85-227049. National Technical Information Service, Springfield, VA.

Stephens, D. W., Waddell, B., Peltz, L. A., and Miller, J. B. (1992). *Detailed Study of Selenium and Selected Elements* in *Water, Bottom Sediment, and Biota Associated with Irrigation Drainage in the Middle Green Ricer Basin, Utah, 1988–90.* Water-Resources Investigations Report 92-4084. U.S. Geological Survey, Salt Lake City, UT.

USEPA (U.S. Environmental Protection Agency). (1985). *Technical Support Document for Water Quality-Based Toxics Control.* Publication PB86-150067. National Technical Information Service, Springfield, VA.

USEPA (U.S. Environmental Protection Agency). (1987). *Ambient Water Quality Criteria for Selenium—1987.* Publication EPA-440/5-87-006, USEPA, Office of Water Regulations and Standards, Washington, DC.

Van Derveer, W. D., and Canton, S. P. (1997). Selenium sediment toxicity thresholds and derivation of water-quality criteria for freshwater biota of western streams. *Environ. Toxicol. Chem. 16,* 1260–1268.