SUMMARY REPORT

Peer Review of

2002 Update Aquatic Life Water Quality Criteria for Selenium

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Prepared for:

U.S. Environmental Protection Agency
Office of Water
Office of Science and Technology
Health and Ecological Criteria Division
401 M Street, S.W.
Washington, D.C. 20460

Prepared by:

Versar, Inc.
6850 Versar Center
Springfield, Virginia 22151

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I. INTRODUCTION

In 1998 EPA held a Peer Consultation Workshop on Selenium Aquatic Life Toxicity and Bioaccumulation. One of the conclusions of that workshop was that it appeared feasible to express the chronic criterion as a tissue concentration. In the document under review, intending to somewhat reduce the need for site-specific criteria adjustments, and to sidestep the controversy involved in setting a reliable water concentration, EPA has expressed the chronic criterion as a whole-body fish tissue concentration.

This is not the first criterion to be expressed in this manner. EPA has already expressed the mercury criterion as a fish tissue criterion. However, whereas the mercury criterion is expressed as a concentration in the diet of the organisms of concern (i.e., humans), the selenium chronic criterion is expressed as a concentration in juveniles or adults of the target species, with the intent of safeguarding reproduction.

EPA does not believe that the evidence supports the concept that there is a uniform tissue concentration that reliably yields effects, irrespective of the duration, route of exposure, and form of selenium. Rather, the proposed chronic criterion is intended for protection of aquatic life exposed to selenium for extended periods via food chain contamination.

In order to provide protection from elevated short exposures to selenium dissolved in the water column, and to meet expectations for using the available data, EPA has also derived a conventional acute criterion, expressed as a concentration dissolved in the water column.

The aquatic life criterion for selenium is used for determining the level of pollution control necessary to attain the aquatic life uses of water bodies, and in particular to allow the propagation of fish and shellfish, per Section 302 of the Clean Water Act. It is necessary that EPA be able to simultaneously defend its criterion as being reliably protective in nearly all situations, and in general being no more stringent than is necessary to achieve protection. That is, the degree of stringency is to be both sufficient and necessary.

The setting of a single value for a criterion involves both technical facets (data assessment) and policy facets (level of protection). EPA values peer reviewers for their technical expertise in assessing the relevant data. For level of protection issues, EPA is most interested in input from the general public - those who would enjoy the benefits of pollution control, and those who would pay the costs. Peer reviewers are thus asked to recognize these distinctions. Nevertheless, the dividing line between technical issues and level of protection policies is indistinct. In the absence of an unambiguously defined goal for the level of protection, peer reviewers may have to approach some of the technical issues from the vantage point of their own conception of the appropriate level of protection.

The document reviewed is a draft revision of EPA's aquatic life criterion for selenium, titled "Aquatic Life Water Quality Criteria for Selenium 2002." The new criterion is intended to replace the current aquatic life criterion originally published in 1987. The new criterion is intended to protect aquatic life; it was not derived with the intent of protecting birds or other wildlife that feed on aquatic life. The document presents acute criteria for fresh and salt waters, expressed as µg/L dissolved selenium, and a chronic criterion for fresh water, expressed as µg/g dry weight (dw) in whole body fish tissue. No saltwater chronic criterion was derived, due to lack of data.

The "Aquatic Life Water Quality Criteria for Selenium 2002" was reviewed by a panel of five reviewers. The charge to the peer reviewers is provided in Section III. The reviewers selected to perform this review are Dr. John M. Besser, Mr. Steve Canton, Dr. A. Dennis Lemly, Dr. Gregory Möller, and Mr. Robin J. (Rob) Reash. A brief description of their experience and educational backgrounds are provided in Section II of the Peer Review Report. Peer Review comments received from each of the five reviewers are provided in Appendices A through E.

The comments and recommendations from the five reviewers have been combined and organized as follows:

- General comments;
- Response to charge;
- Specific comments by page number, referenced by commentor; and,
- Any new information or data that could potentially improve the quality of the document.

II. PEER REVIEWERS

John M. Besser - Dr. Besser is a Research Fisheries Biologist (Aquatic Toxicology) with the United States Geological Survey (USGS), Biological Resources Division, Columbia Environmental Research Center. He has twenty years of experience assessing the bioavailability and toxicity of contaminants, including selenium, in aquatic ecosystems. His current research interests include laboratory and field methods for assessing toxicologic impacts of contaminated sediments on benthic communities; biotic and physicochemical influences on the bioavailability and toxicity of metals and metalloids; and development and validation of sediment quality criteria for protection of freshwater biota. Dr. Besser holds a Ph.D. in Fisheries and Wildlife-Environmental Toxicology from Michigan State University.

Steve P. Canton - Mr. Canton has more than 25 years experience in ecotoxicology, water quality criteria, biological assessments, and aquatic ecology. He has been working on various issues related to selenium toxicity and water quality criteria for various states and cities. Currently Mr. Canton is a Vice President and Senior Aquatic Ecologist with Chadwick Ecological Consultants, Inc., in Littleton, Colorado. He is a certified Senior Ecologist and Lake Manager. He also is a member of the board of directors for the Colorado Lake and Reservoir Management Association and a member of the Colorado Water Quality Monitoring Council. Steve Canton received a M.S. in Zoology with a specialization in Limnology from Colorado State University.

A. Dennis Lemly - Dr. Lemly has spent more than 20 years investigating the effects of selenium in aquatic ecosystems. His research has included studies of selenium bioaccumulation and reproductive toxicity in fish, impacts of selenium on aquatic life, and selenium impacts associated with power plant discharges, agricultural irrigation, and mining effluents. Among his numerous publications, he recently published "Selenium Assessment in Aquatic Ecosystems: A Guide for Hazard Evaluation and Water Quality Criteria" (2002) which presents guidelines for hazard assessment and water quality criteria for selenium. Currently, he has faculty appointments at Duke University and Virginia Tech University. Additionally, Dr. Lemely is a Senior Research Scientist with the U.S. Forest Service at the Southern Research Station, Coldwater Fisheries Research Unit, where he is examining new causes of selenium pollution. Dr. Lemly holds a Ph.D. in Biology from Wake Forest University.

Gregory Möller - Dr. Möller has 25 years of experience in analytical chemistry, environmental toxicology, and biogeochemistry. He is an Assistant Professor of Environmental Chemistry and Toxicology at the University of Idaho, the Technical Director of the University of Idaho Analytical Sciences Laboratory, and the Director of the Idaho Food Quality Assurance Laboratory. He research includes the sources, pathways, biogeochemistry, and control of selenium, particularly in Western phosphate resource areas. Currently, Dr. Möller is a member of the Water Quality Study Team associated with the Western Region Aquaculture Research Center at the University of Washington. Dr. Möller has a Ph.D. in Physical Chemistry from the University of California, Davis.

Robin J. (Rob) Reash - Mr. Reash has worked for more than 15 years performing biological assessments and assessing the affects of water quality on aquatic populations. He is a Principal Environmental Scientist with American Electric Power, Water & Ecological Resource Services, where his work has included examining aquatic species inhabiting selenium-laden coal ash effluents He is a Certified Fisheries Scientist and currently serves a chairman of the Utility Water Act Group Water Quality Committee, a subcommittee member for the Water Environment Research Foundation, and a member of ORSANCO Ohio River biocriteria advisory committee. Mr. Reash has a M.S. in Environmental Biology from the Ohio State University.

III. CHARGE TO THE PEER REVIEWERS

The technical charge for the peer reviewers was provided by the EPA WAM. The reviewers were asked to review of the document and the appropriateness of the criteria and to respond to the following charge questions:

Acute criteria in fresh and salt waters:

1. Are the toxicity tests used to derive the criteria appropriate for such use? Are you aware of other relevant data that were not used?

2. Are the acute criteria appropriate?

3. The criterion did not incorporate a relationship with sulfate. However, if there is need for additional site-specific discrimination, are the data indicating a relationship between toxicity and sulfate concentration sufficient to support expressing the freshwater selenate criterion as a function of sulfate concentration?

Chronic freshwater criterion:

4. Is a concentration in whole-body fish tissue an appropriate basis for expressing the criterion?

5. Are the toxicity tests and other studies used to derive the criterion appropriate for such use? Are you aware of other relevant data that were not used?

6. Is the freshwater chronic criterion appropriate?

7. With the goal of being neither under- nor overprotective, how reliable would you expect the criterion to be in application to different sites? Are there any straightforward ways of improving its site specificity?

8. Although the criterion was not derived using wildlife criteria derivation procedures, EPA noted some evidence that the criterion would protect piscivorus birds. Are you aware of other data relevant to the protectiveness of the criterion for birds?

IV. GENERAL COMMENTS

John Besser
I think the authors have done an adequate job of reviewing, screening, and summarizing the literature on acute and chronic toxicity of selenium (Se) to freshwater biota. Some aspects of the authors' treatment of the acute toxicity data may raise questions, such as the decision to exclude some studies that strongly influenced the previous acute criterion for selenate, and the decision not to adjust the selenate criterion for effects of sulfate. However, the new criterion more closely reflects the bulk of the literature regarding the acute toxicity of selenate. Overall, the acute criterion is based on a large number of studies with a wide range of aquatic taxa, and should be adequate to protect aquatic ecosystems. The saltwater criterion is based on much smaller data set, but the FCV for selenite (the basis for the saltwater CMC) is based on the response of a rather sensitive species, and on this basis I expect that this value will also be adequately protective.

I feel that the chronic criterion is also based on a scientifically valid interpretation of the literature on the toxicity of Se to freshwater biota. However, there is substantially greater uncertainty about the protectiveness of the CCC, due both to the smaller body of literature on which it is based and the greater difficulty in interpreting results of complex chronic studies conducted both in the field and in the laboratory. The greatest threat posed by Se to aquatic biota results from chronic Se exposure, leading to elevated Se concentrations in tissues and to chronic toxic effects, principally during early life stages. Therefore, the decision to derive a tissue-based chronic criterion relieves the authors from the unfair burden of justifying a particular aqueous Se concentration as the direct cause of chronic toxicity in aquatic biota. This still leaves considerable uncertainty about the 'correct' Se concentration for a chronic, tissue-based threshold – uncertainty derived in part from questions about interpretation of critical study results, and about inclusion or exclusion of results of particular studies. Although I have expressed my opinions about some of these specific questions, I think the overall weight of scientific evidence supports the authors' approach. In my opinion, the national CCC value of 7.9 ug/g (dry wt., whole-body fish tissue) reflects the need to accommodate the broad range of environmental factors that may affect Se toxicity, without relying on studies that may be strongly influenced by interactions with other contaminants and other site-specific factors.

Steve Canton
My overall impression is that this is a well written, well thought out review of selenium toxicity and the derivation of meaningful (i.e., regulatorily feasible) criteria that will be protective of aquatic communities. It is obvious that not only considerable time was spent on the literature, but that knowledge of the current thoughts/ideas/discussions/controversies regarding how selenium criteria could or should be set was sought and used. I fully agree with the concept of differing approaches to acute and chronic criteria setting for selenium. This isn't surprising since I proposed such a split in my 1999 paper - which I can't help but mention is in the list of references (thank you!), but does not appear to actually be cited in the document as far as I can tell!

While I appear to have a sizeable number of comments and specific edits below, I believe that these are basically minor edits. Along with some additional "checking of the numbers," these edits would help this document's readability when it is released for public comment (which I am sure will still be lively).

Lastly, I believe the approaches used to derive the acute and chronic selenium criteria are sound and will pass muster as scientifically valid when finalized.

Dennis Lemly
The document is well researched, generally well written, and I agree with the approach of using a fish tissue-based method to derive the criterion for chronic exposure. I also agree with both the Final Acute Value (185 µg/L) and the Final Chronic Value (7.9 µg/g dw) for new national criteria.

Gregory Möller
I am supportive of the approach taken in developing these criteria. Biomonitoring of Se release and potential for chronic impact via fish whole body Se levels represents a reasonable and defendable approach to safeguarding aquatic ecosystems. Pollution is a biological phenomenon and when we measure it in chemical terms we must be able to relate it to any possible negative biological effect. A chronic exposure chemical concentration in an environmental media, such as water or sediment, confounds this relationship because of the required trace element status of selenium in all aerobes and varying degrees of dietary exposure, homeostasis, biotransformation and end effects of various species in a complex ecosystem. The authors have gone to great lengths to be inclusive of the available data and expert opinion in the development of the proposed criteria.

Monitoring of chemical levels in environmental media such as water is usually very satisfying to some because of the precision of the measurements, but this often breaks down when the link to biological phenomena is not available or unclear. There are real challenges in localization of the chemical release and quantifying temporal and spatial variation. With selenium, the complex interplay of inorganic and organic chemical species coupled with diverse biotic and abiotic processes, makes true chemical exposure assessment in a dynamic ecosystem a difficult challenge and in common, non-research, environmental management applications - impossible. Biological systems are "damped" and integrative over time. Hence, receptor monitoring can yield a more accurate assessment of the potential for environmental impact. Spatial variability can still be significant when using organisms and the variability of toxicity among organisms can be great, both within a species and between taxa. However, biologically based monitoring allows for a better ecosystem assessment of migratory populations and real exposure patterns such as concomitant sulfate exposure potentially moderating Se uptake.

The inclusion of an acute water concentration standard adequately recognizes the weaknesses of tissue based monitoring in an acute exposure scenario. Acute disruption of fundamental biological processes and the inability of the organism to overcome the resulting toxicodynamic processes are metabolic in nature and are therefore best quantified by assessment of dose. The acute toxicity of water borne Se to a wide array of aquatic species is well described in the scientific literature. The effects of chronic exposure at low levels in water are confounded by the biogeochemical cycling of Se in aquatic ecosystems and food chain effects that vary considerably with the local environment (e.g., lentic vs. lotic). Management of environmental releases of Se since the 1987 criteria have been difficult and resource intensive due to: 1) limited or conflicting knowledge on site and species specific impacts; 2) unknown field observation variables (e.g., pesticides and metabolites in Se contaminated agricultural drainage, endocrine disrupting chemicals in Se containing waste water discharges); and 3) limited or non-existent, best available technologies (BATs) to treat Se containing discharge waters to meet the aquatic biota criteria.

Rob Reash
The draft "Aquatic Life Criteria for Selenium – 2002" document is a substantial improvement, considering both scientific and policy viewpoints, from EPA's current nationally-recommended aquatic life criteria (last updated in 1987). The scientific basis of the new draft criteria is considerably more robust than the existing nationally-recommended criteria.

During the past 10 – 12 years there has been an enormous amount of information published in the scientific literature on the effects of selenium to aquatic life. The authors should be commended for consolidating this information comprehensively, and organizing the various studies based on relevance to ambient criteria calculations.

The authors, in many respects, were breaking new ground in developing updated acute and chronic aquatic life criteria for selenium. Considerable creativity and "thinking outside the box" was necessary to derive criteria that reflect the realities of scientific evidence. Although careful adherence to EPA's 1985 criteria

methodology was followed when appropriate, the resulting draft criteria were not straightforward "calculated" values, but required best professional judgement (BPJ) decisions. The authors should be commended for considering many factors that were a part of making these decisions. The key areas which required considerable BPJ were 1) the selection of one CMC for dissolved selenium rather than separate criteria for selenate and selenite; 2) the selection of a whole body fish tissue analysis to represent the medium for the chronic criterion; and 3) the choice of toxicological endpoints (and effect levels for these endpoints) to derive a draft nationally-recommended chronic criterion. Comments on these key BPJ decisions are provided below.

The scientific merit of the document is, overall, very good. Some additional information (identified below) should be considered by the authors during development of the final criteria document. Two sections of the Introduction ("Chemical and Physical Properties" and "Sources of Selenium to Aquatic Systems") should be expanded by incorporating more recent references. ATSDR's "Toxicological Profile for Selenium" would be an excellent reference for obtaining additional information on selenium biogeochemistry, sources of selenium, and analytical capabilities/limitations. The knowledge of selenium biogeochemistry has increased greatly since 1987, and I believe the final criteria document should (at least) provide a good general overview of this information.

V. RESPONSE TO CHARGE

***Acute Criteria in Fresh and Salt Waters*:**

1. Are the toxicity tests used to derive the criteria appropriate for such use? Are you aware of other relevant data that were not used?

John Besser
Generally, yes. The criteria are clearly based on an adequate number, quality, and taxonomic distribution of acute toxicity tests with selenate and selenite, at least for freshwater. However, in my opinion, strict adherence to the Guidelines (Stephan et al. 1985) has resulted in the exclusion of many high-quality studies from the calculations of the acute criteria. In several instances (several are discussed below), this has resulted in SMAVs being calculated from a minority of the available tests, with apparently adequate tests being excluded because they were conducted under static condition. Is testing under flow-through conditions necessary for tests as short as 24-h duration? For tests with *Ceriodaphnia* and other daphnids? For a chemical as water-soluble and non-reactive as selenate?

Steve Canton
I am well acquainted with the acute selenium toxicity literature, having published a paper on this topic a couple of years ago (again, this paper is in the reference list, but I couldn't find it cited in the document itself!). From that experience, I feel that the literature cited and toxicity data used in this revised criteria document are certainly appropriate for derivation of acute selenium criteria. However, I have to admit my knowledge of saltwater acute selenium toxicity is basically non-existent, so this support is based on my knowledge of the freshwater database (which is considerably more extensive).

In fact, there are data that are relevant, but were not used. Oddly enough, the data I found was "missing" was in fact cited (or at least listed in the references), indicating the authors knew of it's existence. Apparently it was either overlooked or a problem was found with the data. If overlooked, this is easily rectified. If there was a problem with those data, the problem was not specifically documented in the report and would need to be.

The missing data are acute toxicity data for selenite and selenate for the midge *Chironomus riparius* in the paper by Ingersoll, et al. 1990. Use of these data do change the GMAV for *Chironomus*. The odd part is that the toxicity data for *Daphnia magna* from that paper are used, making the absence of the *C. riparius* data a mystery. Other missing data are the acute selenate toxicity values for *Daphnia magna* from a paper by Johnston (1987). Again, this paper is in the reference list, but the data are not included in Table 1a. Lastly, there is a paper by Cumbie and Velte (1986) on selenite toxicity to green sunfish (*Lepomis cyanellus*) that provides another SMAV for that genus and modifies the GMAV. This paper is not in the references, so the authors may not be aware of this study. I've attached a pdf file with the Cumbie and Velte (1986) paper in case GLEC needs a copy (see Appendix B of the Peer Review Report).

The details on the effects of adding these data to the database, with regard to changes in SMAV and GMAV values are presented below in the "Specific Comments" section. Note that if these data are appropriate, the FAV for selenate does change very slightly to 181.4 µg/L, which will affect the final recommendation for the acute selenium criterion of 185 µg/L now in the document.

Dennis Lemly
The toxicity tests used to derive the criterion are appropriate for that purpose. I am not aware of other relevant data that were not used for the acute criterion assessment.

Gregory Möller
Yes, as far as I can tell from the various descriptions. I am not aware of other data.

Rob Reash
The acute toxicity database is comprehensive and appropriate. The selection of acceptable data seems to consistently follow the 1985 guidelines. One acute toxicity study (Bringmann and Kuhn 1959a – page 16) seems quite outdated (> 40 years old), and I have doubts that the QA/QC for this experiment is comparable to that for more standardized, recent tests. The authors should check this study in detail, unless this was previously done. The acute toxicity data for fathead minnow reported by Kimball (manuscript – page 20) seems anomalous compared to other results for this species.

I am not aware of other acute toxicity data that should have been considered to calculate individual Species Mean Acute Values.

***Acute Criteria in Fresh and Salt Waters*:**

2. Are the acute criteria appropriate?

John Besser
The freshwater criteria are probably adequately protective, given the large body of literature on which they are based. I do question whether calculation of separate FAV for selenate and selenite provides more 'benefit' (better resolution of differences in sensitivity between the two species) that is does 'cost' (reduction in the size of the data sets). The overlap in toxicity of selenate and selenite evident in Figures 1 and 2 is probably a result of the decision not to adjust selenate toxicity for effects of sulfate. It may be preferable to pool the GMAVs for selenate and selenite and derive a single FCV for the merged data.

Steve Canton
With the possible exceptions noted above (i.e., possibility of additional data used), I feel the acute criteria are appropriate. Given the relative difference in toxicity between selenite and selenate and the problems with laboratory precision in speciation, I also agree with using the lower of the two CMCs for these selenium forms as the recommended acute criterion for freshwater.

In some respects, I feel the selenate number is lower than would be expected or is possibly justified when reviewing all the available data. I can't help but feel this is an artifact of the small sample size and one particular test value for *Ceriodaphnia* that is considerably lower than other available (but, technically unusable) values for the same species. Interestingly enough, this is very similar to the previous selenate acute FAV in the 1987 document, which was also lower than expected due to a small data base and the seemingly very low values for *Gammarus* - values which were not replicated in subsequent testing conducted and used in the current document. It does make me wonder what repeated testing on *Ceriodaphnia* would show and whether this would remain the most sensitive species!

Despite these "misgivings," given the data available for review, the resulting selenium criteria are calculated correctly and are appropriate for protection of aquatic life from acute selenium toxicity.

Gregory Möller
Yes. The approach appears to be consistent with EPA guidance and the supporting data appears to be sufficiently comprehensive and complete.

Dennis Lemly
The acute criteria are appropriate for their intended purpose, that is, direct waterborne exposure for short durations.

Rob Reash
The authors chose not to distinguish separate CMC's for selenate (Se^{+6}) and selenite (Se^{+4}); instead, they selected the lower of the two CMC values, and expressed this as a "dissolved selenium" criterion. While this decision requires considerable BPJ, I believe that separate criteria should be established for the two inorganic forms, for the following reasons:

- the database for each oxidation state fulfills the minimum database requirement;
- for most species tested, selenite is the more toxic form;
- in some geographic areas, seleniferous soils result in relatively high background levels of selenate in the water column, due to natural weathering action. In such areas where selenate is the

predominant inorganic form, a selenate-specific CMC would seem the most appropriate applicable water quality criterion.

- the establishment of separate criteria is consistent with EPA's policy that the most bioavailable form of metal should be regulated by states and tribes. The toxicological data, in general, indicate that selenite is the more bioavailable form of inorganic Se. In instances where Se in a receiving stream consists predominantly of selenite (even after mixing with wastewater discharges), this more bioavailable form should be regulated.

The authors provide a reasonable defense for expressing the national CMC as a "dissolved selenium" criterion based on the more stringent CMC of the two forms. This is certainly a reasonable BPJ decision, as it eliminates many practical questions and difficulties in situations where a receiving stream or wastewater discharge is comprised of a mixture of both inorganic forms. There are some technical reasons why a dissolved selenium criterion is not appropriate, however. First, expressing a criterion as dissolved requires that implementation of the criterion (i.e., for compliance purposes) requires field filtration of the water sample. The field filtration step does introduce potential sampling errors, most importantly inadvertent contamination. In addition, filtration for trace metals must be performed as soon as possible, as adsorption of dissolved metals to suspended matter (prior to filtration) can occur quickly. While sample contamination can be eliminated using careful QA/QC steps, the necessity of a dissolved analysis should be examined first. Particulate Se seems to represent a very low portion of total inorganic Se in most receiving streams. Thus, the analysis of dissolved selenium versus total recoverable selenium may not be significant from a bioavailable perspective. In most wastewater and receiving stream samples, dissolved selenium typically represents > 90% of total selenium.

The geochemistry of selenium in natural waters is such that separate criteria for the two inorganic forms should be considered. Regarding the geochemistry of inorganic selenium forms, EPRI (1984) states that:

> In most natural waters selenium exists in the +4 oxidation state as H_2SeO_3, $HSeO_3$, or SeO_3^{-2}. Selenium can be found in the +6 oxidation state only under extreme oxidizing conditions in strongly alkaline solutions. (page 3-128)

Because conversion from selenite and selenate is not favored thermodynamically in most natural waters, selenium that is discharged from a wastewater in the form of selenite will likely not be oxidized upon mixing with the receiving stream.

While the expression of separate Se CMC criteria seems justified from a toxicity perspective, there are practical concerns with such criteria. The first are analytical. There are few commercial laboratories that routinely measure the inorganic species of Se, though there are published procedures for these analyses. Thus, expressing criteria in specific oxidations states requires the use of EPA-approved methods for compliance and monitoring purposes. The lack of EPA-approved methods for selenite and selenate analyses poses compliance and monitoring concerns. On the other hand, EPA could begin the process of approving the speciation analytical methods in the near future, which may give sufficient time before states begin to implement the revised acute criteria, if they choose to do so. Another practical problem with separate acute criteria is how to implement these criteria in settings where ambient waters (and/or wastewater discharges) contain both forms of inorganic Se. Two questions arise: 1) what is the applicable criterion to judge compliance with (i.e., is the criterion a weighted criterion based on respective ratios of the two forms?); and 2) how do dischargers demonstrate compliance, especially in situations where the fully-mixed receiving stream shows varying ratios of the two Se forms? As a result of the Great Lakes Initiative (GLI) rulemaking litigation, EPA was mandated to propose new acute criteria for the Great Lakes States. In November 1996, EPA did propose new criteria for selenite and selenate, and proposed an additive model for use in settings when both forms of Se were present in receiving streams and/or wastewater discharges (see 61 Federal Register 5844-58449). While the proposed additivity model does require some implementation guidance, EPA should strongly consider using this framework for establishing (and implementing) the updated acute

criteria.

In summary, I believe EPA should establish separate CMC criteria for selenate and selenite. These criteria would be appropriate where the predominant form of Se in a receiving stream is one form or another (90% or greater of total recoverable Se). These criteria could be implemented in settings where the influence of wastewater does not change the predominant form (established in ambient waters) In situations where a receiving stream does contain varying ratios of the two Se forms, a different approach is needed. EPA could handle this in one of two ways. The first way would be to adopt the additivity model that was proposed in the GLI rulemaking proposal (this model, of course, would include the updated CMC values for both forms). Alternatively, EPA could give states the option of adopting a total recoverable (preferred, but dissolved Se would likely be acceptable) CMC, which should be calculated as the **geometric mean** of the selenate and selenite CMC values. The latter option is, clearly, much less troublesome to implement for CWA compliance purposes.

In summary, while there are practical reasons that establishment of an updated acute criterion using the method proposed by the authors is less troublesome for compliance purposes, the establishment of separate selenate and selenite criteria should be strongly considered by EPA, especially if separate criteria are justified using the 1985 criterion calculation methodology.

One last comment on updated acute criteria is needed. EPA should be aware that many states are continuing to implement the outdated CMC of 20 µg/L total selenium. For states outside of the Great Lakes Basin, this criterion is being implemented for NPDES permit compliance AND total maximum daily load (TMDL) applications. The difference between this criterion and the updated CMC for both selenium forms is considerable (a factor of 10).

***Acute Criteria in Fresh and Salt Waters*:**

3. The criterion did not incorporate a relationship with sulfate. However, if there is need for additional site-specific discrimination, are the data indicating a relationship between toxicity and sulfate concentration sufficient to support expressing the freshwater selenate criterion as a function of sulfate concentration?

John Besser
The data as presented here are not adequate to make this decision. At a minimum, plots of the regressions of sulfate vs. selenate acute toxicity would have helped justify this decision. I would have liked to see how a hypothetical sulfate-adjusted acute value for selenate would compare with the FAV for selenite. This would allow assessment of the authors' valid concern about conditions under which a sulfate-based criterion could be under-protective against toxicity of selenite.

Steve Canton
The discussion of sulfate on pages 15-16 explains what was found and why they didn't use it. The rationale makes pretty good sense and overall, I agree that given the fact that chronic toxicity will require much lower than acute values, the complexity this would add is not necessary. It certainly is not necessary for a national recommended criterion.

However, some of the discussion on page 15 is less convincing than others. For example, the variation in slopes and the fact that the relationship works for most, but not all, species, is not unique to sulfate and selenate. This variation in slopes can also be found in the derivation of hardness relationships for many of the other metals. And, yet, the U.S. EPA has adopted hardness-based criteria for these metals.

So, while this may not be necessary for a national criterion, I would strongly suggest adding an appendix that more fully explains this section and actually presents a potential sulfate-based selenate criteria equation that could be used if a site-specific circumstance warranted its use.

Dennis Lemly
I believe that the strength of the relationship for sulfate influence on selenium toxicity is sufficient to support expressing the freshwater acute selenate criterion as a function of sulfate concentrations.

Gregory Möller
Because of the analogous Se:S biogeochemical cycling, the co-location, co-transport, and therefore co-exposure of the elements is of merit in the assessment of acute ecotoxicity. As with most modifiers of toxicity (antagonists, synergists, etc.), much more is known about the primary compound of intoxication rather than the effects of secondary compounds. However, with an ab initio biochemical pathways examination and observations in the literature, most would judge the moderation of Se toxic endpoints by S as well founded. In my work on selenium releases in natural and disturbed environments, I get far more concerned when I observe Se:S ratios in water greater than 1:1000.

A review of the Se:S data compiled in Table 1a shows the expected lack of effect for selenite:sulfate exposures. However, the selenate data for most species show a distinct relationship as sulfate levels rise. I would not expect the relationship to be the same in all species (for the same reasons I would not expect a similar color of eyes in all species) and reason #1 (page 15, paragraph 4) uses the rate effect differences inappropriately to justify the action of not adjusting for sulfate. The slopes as calculated in page 15, paragraph 4, (0.19 and 0.87) are inaccurately referred to as "sufficiently mild" in reason #2 for not including a sulfate adjustment (page 15, paragraph 4). Using a lower bounding estimate for the Se:S relationship

(0.19?) would be satisfactory to me. Note that using a lower bounding estimate of 0.19 to account for sulfate modification of Se toxicity, a 2000 mg/kg sulfate brine water would have an adjusted acute criteria of (380 + 185) µg/L = 565 µg/L. This is well below the SMAV of 2,073 µg/L for *H. azteca* and therefore protective. Castle et al. (in preparation) have observed that selenium acute toxicity testing breaks down in sulfate brines as a result of animal desiccation.

As discussed in reason #3 (page 16, paragraph 1), a sulfate correction would not be protective of selenite impacted environments. This is the strongest reason for not maintaining a general sulfate correction to the FAV. One can debate scenarios of the potential for selenite occurrence in natural oxic, high sulfate systems, but it can occur. This is an opportunity for site specific adjustments based on the assay of Se speciation in the system, if EPA decides to go down this path. I would be supportive of this mode of incorporating the sulfate adjustment if it were simple.

> *For waters with >90% selenate as a fraction of total selenium, an adjusted selenium concentration of 185 + 0.19 [SO_4^{2-}] µg/L is protective of freshwater aquatic life.*

Justification for inclusion of an adjustment may be found in examining the potential applications of the criteria to different use scenarios including sulfate brines. Since the Se:S relationship passes the reasonable and expected judgment most informed scientists would give it, EPA should move forward with developing sulfate guidance for the acute freshwater criterion.

Rob Reash

If EPA chooses to express separate CMCs for selenate and selenite, the question of whether to express the selenate criterion as a function of sulfate concentration should be answered by consulting the 1985 guidelines. There does seem to be fairly convincing evidence that the sulfate toxicity mitigating interaction is toxicologically apparent. The authors state on page 15 :

> *"The natural logarithm of selenate acute values was a linear function of the natural logarithm of sulfate concentrations. "*

Also on page 15:

> *"...positive slopes for five of six species that had acute values precisely determined."*

The significant LC50 versus sulfate concentration relationship seems to indicate a consistent phylogenetic response. The authors do provide reasons why a sulfate-dependent relationship is not, overall, practical. The authors state that significant differences in the LC50 vs. sulfate regression slope, between species, was indicated. Is this the only criterion by which to judge whether a sulfate relationship should be allocated for selenate? In EPA's "2001 Update of Ambient Water Quality Criteria for Cadmium" (EPA-822-R-01-001), EPA reported that the slope of cadmium LC50 versus hardness for all freshwater species ranged from 0.1086 to 2.031. EPA, then, compared the individual species slopes to the overall pooled slope. The individual species slopes were not significantly different than the overall pooled slope (p=0.27) (page 6). Should a similar analysis be conducted for selenate LC50 versus sulfate levels (i.e., comparison of individual species slopes to a pooled slope)?

The authors state that "…the acute criterion [is] sufficiently high compared to chronically toxic concentrations, [and] it was not clear that the additional complexity of a sulfate formula would have any significance in regulatory application." (pages 15-16). While I believe this statement is generally true, there are some water bodies that, because of their use designation, do not have chronic aquatic life criteria that apply. Thus, the applicable acute (CMC) criteria will determine what effluent limitations are necessary. The authors also state that "If a total selenium criterion were implemented based on the selenate FAV adjusted for the sulfate concentration, then the selenium limit would not adequately protect aquatic organisms when

selenite is the predominant form of selenium and sulfate concentrations are high." (page 16). This statement would be true if EPA chooses to express the updated CMC as a dissolved selenium concentration of 185 µg/L. If EPA choose to adopt separate CMC criteria for selenite and selenate, however, this problem would likely not occur.

The site-specificity usefulness of establishing a protective selenate aquatic life criterion as a function of ambient sulfate levels could be advantageous in certain settings. Water bodies having elevated sulfate levels are those typically influenced by historical mining activities. The application of a site-specific selenate criterion, as a function of receiving stream sulfate level, would seem to make sense from a technical standpoint.

In summary, I believe a sulfate interaction effect for selenate should be strongly considered by EPA, if the existing criterion calculation guidelines support this. EPA should assess the hardness versus toxicity relationships for other metals as a basis for comparison. If EPA chooses not to express a selenate CMC as a function of ambient sulfate levels, the agency should recognize the scientific evidence of the toxicity relationship, and consider including language that allows a site-specific criterion demonstration that incorporates this relationship.

Chronic freshwater criterion:

4. Is a concentration in whole-body fish tissue an appropriate basis for expressing the criterion?

John Besser
Yes. Selection of tissue as the measure of Se exposure has many benefits, given the many known and suspected (and probably unknown) sources of variation in Se bioavailability in the environment. Tissue Se concentrations integrate many influences on a site- and species-specific basis, which would be impossible for any water-based chronic Se criterion. As the authors admit, whole-body Se concentrations are probably not the 'best' tissue for evaluating the probability of toxic effect. Since the most sensitive responses to Se exposure in fish (and wildlife) are almost always expressed in early life stages, Se concentrations in ripe ovaries are probably the best predictor of these effects. However, whole-body Se concentrations do reflect Se concentrations in gonads and other target organs, and they can be obtained year-round from a variety of species using relatively simple methods of collection, sample preparation, and analysis. Analysis of tissues is more complex and expensive than analysis of water, but not by a large margin. The disadvantages of a tissue-based criterion relative to traditional water-based values are largely logistic (how many of what species to collect and when) and economic (greater cost of sample preparation and analysis), rather than scientific.

Although the use of the whole-body Se concentration for the chronic criterion is appropriate, it does present some difficulty in interpreting results of toxicity tests for which whole-body Se concentrations were not measured or reported. This is especially a concern given the relatively weak correlation of whole-body and liver Se concentrations. Since liver Se concentrations (extrapolated to whole-body concentrations) influence chronic values for several studies, this possible source of error could affect the reliability of the FCV. The number of studies used to derive the liver:whole body regression is surprisingly small (see comment #9 (Appendix A of Peer Review Report) or Specific Comment for page 44, below), and perhaps additional data could be found to improve the fit of the predictive equation.

Steve Canton
In many ways, I truly believe that some kind of tissue based method is the best way to work with a bioaccumulative metal that expresses its toxicity primarily through dietary uptake. Having looked closely at this issue, I have not found a better way.

We came across this issue when looking at what appeared to be healthy stream fish communities in western U.S. streams where background selenium can often be over 10-15 µg/L. Yet, we know that in other waters with much lower selenium levels, uptake of selenium has caused reproductive failure. As noted in the document, we approached in this issue in our publications by using the base of the food chain - sediment selenium concentrations. This was simply a back-hand approach to bioaccumulation effects, and while not a true measure of uptake by fish, it was functionally similar to using whole-body fish tissue concentrations.

Whole-body fish tissue is also not specifically linked to reproductive failure. That endpoint is best expressed in the selenium content of the eggs/ovaries. However, I also have to agree with the authors of the document that basing a national criterion on reproductive tissues is unworkable (since eggs/ovaries may sometimes be available for only one month of the year, given the spawning characteristics of any particular fish species). As such, it makes good sense to use whole-body fish tissue as a surrogate for this mode of toxicity. And, based on the analysis in the document, it certainly appears that there is a very strong correlation between egg/ovary selenium concentrations and whole-body concentrations. This is a very long way to say, yes, the concentration in whole-body fish tissue is an appropriate basis for expressing the chronic selenium criterion.

Dennis Lemly
The selenium concentration in whole-body fish tissue is an appropriate basis for expressing a criterion for chronic exposure because it integrates the 3 major pathways for selenium uptake (water, planktonic food chain, benthic-detrital food chain), and encompasses the most sensitive biological endpoints (fish reproduction and teratogenic effects).

Gregory Möller
Yes. The proposed approach to limiting the environmental impact of low level anthropogenic selenium release employs fish tissue as an indicator of unacceptable risk. This approach is the most direct and uses resident species in the local food chain as a sentinel of threat. Occupying a key position of the food chain of an aquatic ecosystem as well as maintaining independent commercial and recreational value, fish are an excellent choice for monitoring and assessment. Fish whole body Se levels serve alternately as a direct, upper-trophic level dose-response assessment and as an exposure indicator for aquatic birds, especially piscivorus types.

Rob Reash
I believe that expressing the chronic criterion as a whole-body total Se analysis is appropriate. The authors provide a fairly convincing argument that a whole body analysis is the appropriate medium for the criterion, considering both technical and practical factors. I do agree with the authors that female ovarian tissue is the most direct tissue of analysis to assess sensitive reproductive effects. A considerable amount of reliable ovarian Se tissue data are found in the published literature (especially for sunfish species), and the analysis of ovarian Se levels (in conjunction with carcass Se levels) may provide considerable insight in the development of a site-specific criterion, where there is some evidence of a population decline.

It is recognized that analysis of an internal organ for criterion compliance purposes introduces potential sources of error (sampling and analytical). Some of these potential sources of error, regarding analysis of ovary tissue, are: 1) ovary maturity level (i.e., are immature ovaries appropriate for Se analysis?); 2) post-spawning ovarian tissue loss; and 3) unintentional contamination of tissue (improperly cleaned instrument). The analysis of whole body tissue would seem to eliminate all of these potential problems.

One technical concern with the analysis of whole body Se is the influence of gonad size and maturity on this analysis. Selenium does accumulate preferentially in gonad tissue, especially in settings where selenium contamination occurs (Gillespie and Baumann, 1986). Thus, the spawning condition of female fish may affect the whole body concentration of Se, especially in pre-spawn fish having a high gondasomatic index. Gillespie (1985) showed that sexually mature male and female bluegill sunfish (collected from contaminated Hyco Reservoir) had higher levels of Se in carcass tissue (whole body minus gonads) compared to fish that had immature gametes. This difference was particularly evident for female bluegill: immature fish had mean carcass and ovary Se concentrations of 4.06 and 6.66 ppm (wet weight), whereas fish with mature ovaries had mean carcass and ovary Se levels of 7.25 and 7.13 ppm, respectively.

The above findings suggest that the level of sexual maturity (at least in bluegill sunfish) does have some effect on whole body Se levels. Hence, the stage of sexual maturity may introduce a confounding factor to random, non-discriminatory collection of fish for analysis of whole body Se levels. Certainly, a well-designed sampling program (incorporating statistical methods to allow for identification of confounding, or co-variable, effects) would greatly minimize the probability of making Type I or Type II errors.

The stage of sexual maturity is one of many factors that EPA, eventually, should address for consistent implementation of the fish tissue criterion. A list of technical and regulatory implementation issues that I recommend be considered by EPA, prior to issuance of the final criteria document, are provided in Appendix 1 (see "Miscellaneous Comments" and Appendix E in the Peer Review Report).

I do believe that EPA should issue technical guidance (in conjunction with the final criteria update or as a separate document) that provides pragmatic guidelines on how fish are to be collected and analyzed for whole body Se analysis. In a sense, this would be very similar to the technical guidance EPA is currently developing for implementation of the new methylmercury human health water quality criterion, which is also expressed as a fish tissue concentration. In both cases, states, tribes and other stakeholders will need concise guidance on: 1) how criterion compliance is determined; and 2) how site-specific or region-specific considerations may be taken into account.

In summary, expressing the nationally-recommended freshwater CMC as a whole body fish analysis seems appropriate. The analysis of whole body tissue has advantages over the analysis of individual organs, e.g., the probability of inadvertent contamination is substantially less. There are some practical technical and regulatory guidelines that EPA should strongly consider issuing, in conjunction with the final criteria. Most states (and most regulated industry) are not familiar with implementing (and determining compliance with) a fish tissue criterion. I believe that the effort EPA would spend on developing this guidance would be regarded as prudent and well spent.

Chronic freshwater criterion:

5. Are the toxicity tests and other studies used to derive the criterion appropriate for such use? Are you aware of other relevant data that were not used?

John Besser
I think the reviewers have generally done a good job of screening the literature to select studies that give the most definitive evidence of tissue thresholds associated with toxicity. This task is more difficult and more subjective than screening aqueous toxicity tests, but the authors have identified the most critical issues associated with test validity, primarily by requiring a substantial contribution of dietary Se exposure and by avoiding possible influence of contaminants other than Se. In some cases, I question or disagree with specific decisions made by the authors in including or excluding studies (see attached comments).

In comments #10 and #12 (See comments in Appendix A or comments for pages 53-57 and 55 under "Specific Comments"), I list several studies that were either unknown to the authors or were improperly excluded from discussions and calculations leading to the FCV.

Steve Canton
In general, I would say that the data/studies used are appropriate for deriving the final chronic value. I was a bit surprised that there is no citation for a recent analysis of tissue thresholds for fish by De Forest, et al. (1999. Critical review of proposed residue-based selenium toxicity thresholds for freshwater fish. *Human and Ecological Risk Assessment* 5: 1187-1228). This study conducted many of the same analyses as those presented in the draft criteria document (and came to some fairly close agreement on some numbers).

With regard to the studies used in this document, the authors listed a number of fairly straightforward "criteria" that they used to determine if a study was valid for use (pages 47-48). This "data review protocol" should ensure the use of only "appropriate" studies. However, as they found out, there were few studies that met all the criteria. As such, they did end up using a number of studies that did not meet even half of their criteria. And there are some inconsistencies in how similarly-derived endpoints are used in the analysis (detailed comments are below). However, I think the studies used are appropriate, although I might come up with different chronic values from one or two of them (again, details below).

I was unclear as to why with the Dobbs et al. study with fathead minnows, the <73 µg/g value was used as the chronic value. Following the logic employed by the authors with other studies, I would think the 73 would be considered a LOAEC and the next lower value with no effects measured a NOAEC. If so, with a geometric mean calculated and used, the chronic value for this study would become 57 µg/g, not 73. This would change the GMAV for fathead minnows to 38.18 µg/g (down from 41.46 µg/g).

I would also note that the studies at the Monticello artificial streams (Hermanutz, et al) were used to provide a chronic value for bluegill (12.12 µg/g). However, the discussion of this paper in appendix H also notes the follow up study reported in the same study. These studies also include measurement of elevated whole-body selenium values, but found no chronic effects. Their conclusion was that residual dietary inputs were still present resulting in the elevated tissue levels. Since the authors of this revised criteria document note that studies with dietary input are important (and actually preferred) for their analysis of chronic criteria, I am not sure why those "no effect" tissue levels were not also included in the analysis. The data is certainly as valid as those from the companion study. This would add two more data points for bluegill of > 14.5 µg/g and > 17.3 µg/g, with a resulting change in the GMAV to 11.65 µg/g.

I am not aware of other chronic data on fish populations that were not used. There are some other studies on

other taxonomic groups - and at least some of these I found in the list of references. But, they are not relevant to a whole-body fish tissue-based chronic selenium criterion.

Dennis Lemly
The toxicity tests and other studies used to derive the criterion are appropriate for that purpose. I am not aware of other relevant data that were not used for the chronic criterion assessment.

Rob Reash
I believe that the general approach used to calculate a chronic value (weight of evidence using a regression analysis of internal concentration versus effect) is an appropriate BPJ decision. The authors correctly discuss the limitations of establishing a no-effect chronic level in a site-specific setting (page 46). The determination of the LOAEC is extremely elusive in field settings, and extrapolation of chronic effect levels among systems may be inappropriate from a generic standpoint because of unique biogeochemical processes.

The decision to express the chronic effect as a 20% departure from control response seems appropriate. This value recognizes the variability in precision of detecting a true effect level among the different studies examined.

I have not evaluated the Toxic Effects Analysis Model, but its application to determining a chronic criterion seems appropriate.

The experimental design requirements for "acceptable chronic tests" used in the logistic regression analysis seem appropriate.

Pages 56-61 were difficult to read due to the poor sentence structure. Suggested rewording of some sentences is included on pages with hand-written comments.

On page 57 (2nd paragraph) the authors discuss reasons why chronic values from field studies were not included in the calculation of the Species Mean Chronic Value calculation:

> *....given the need to protect sensitive populations of species, the chronic values for the studies in which eggs and larvae were obtained from bluegill adults that were exposed to elevate selenium for multiple generations.....were not included in the SMCV calculation.*

The authors, apparently, made this decision because of evidence indicating that chronic values from field studies (to the degree they could be elucidated) are higher than corresponding values from laboratory studies. A few comments on this. First, the data in Table 4 seem to indicate that bluegill sunfish are the most sensitive species in the chronic value database, although there are many chronic values that are imprecisely determined (i.e., designated as "less than" values). Thus, it is unclear what the phrase "…given the need to protect sensitive populations of species…" (page 57) means. If bluegill sunfish are adequately protected by the chronic criterion, it does appear that all other species would be protected also.

While the inclusion or exclusion of chronic values from field studies is a BPJ decision, the issue is an important one for implementation of the chronic criteria. I believe the authors were correct in discussing this issue. In field settings, populations may use several mechanisms to survive and reproduce successfully, even when internal Se levels seem to exceed laboratory-derived chronic values. Long-term tolerance induction (due to genetic or physiological adaptation) certainly may occur in settings where Se levels are less than acutely toxic. If field-determined chronic values are excluded in calculating SMCV values, then some considerations of applying the criteria in environments where Se tolerance may have occurred, is appropriate I believe. This issue is discussed in more detail under charge question #7.

Chronic freshwater criterion:

6. Is the freshwater chronic criterion appropriate?

John Besser
The criterion selected by the authors is consistent with my reading of the Se toxicity literature. In my opinion, there is substantial uncertainty about toxicity of Se to fish in the range of whole-body Se concentrations, 5 to 10 ug/g (dry wt). Toxicity thresholds less than 10 ug/g tend to be associated with additional stressors other than Se (e.g., winter stress; Lemly 1994) or effects that may not rise to the threshold of 'unacceptable effects' (e.g., increased frequency of larval hemorrhage without evidence of reduced survival; Hermanutz et al. 1996, as reported by Tao et al. 1999). The selection of the 7.9 ug/g criterion (based on Lemly 1996) is a reasonable attempt to be protective in this range of uncertainty, but a case can be made (see comment #11: Appendix A of the Peer Review Report or Specific Comment regarding page 55, below) for including the lower threshold suggested by Tao et al (1999), which would push the GMCV for bluegill and the FCV towards the lower end of this range.

Steve Canton
Given the amount of text in the document regarding the need for data quality and various statistical tests used to derive various acute and chronic values, I find it more than ironic that the chronic criterion is simply chosen as the lowest number in the database! Harkens back to the "good old days" of the 1987 document where a similar exercise was conducted. Then, 5 µg/L was chosen because, basically, this was the concentration in an arm of Belews Lake where no effects had been documented.

Regardless, I'm not sure the 7.9 µg/g value, in and of itself, is a valid choice since in the Lemly study he notes that there was no difference in the tissue concentrations for those fish that died or lived. And this study did not have enough treatments to determine an effect level similar to a geometric mean of LOAEC and NOAEC, or develop a concentration-response curve to allow calculation of an EC20. So, I do not believe choosing this one value from this one study as the chronic criterion is appropriate.

The explanation given for use of this number has to do with that particular study and the apparent adverse additive effects of cold temperatures on the fish. However, these values well within the range derived from the more complete studies used to develop the GMAV for bluegills (especially considering sampling and analytical variability). As such, I don't think this number necessarily needs to be singled out for the final chronic value.

Therefore, I would hope for a more "scientific" method for deriving the final chronic value from the available data. I'm not sure what this is, though! Assuming no changes to the values in the document (although I do feel some should change), there are some simple approaches. The authors may simply take the geometric mean of the chronic levels (20.24 µg/g), which seems a bit high. Or the geometric mean of only the fish data (16.83 µg/g) - still high. Or the 5th percentile of the chronic data (9.93 µg/g) - getting closer. Or the 5th percentile of the fish data only (9.82 µg/g).

But, the point is, after all the work put into the document to be more "scientific" in the derivation of the appropriate chronic criterion, it seems kind of a let down to resort to the lowest number found.

Even if a more statistical approach were used, given the fairly low number of values to use for any mathematical exercise, I have to admit that I, too, would probably end up recommending use of the lowest GMAV (9.5 µg/g) for the final chronic value. This would be more defensible than the 7.9 value. And this

is quite similar to values recommended by DeForest, et al. (1999). Again, note that if some of the additions/corrections to the data that I suggest are used, this would result in a final chronic value of 11.6 µg/g.

Dennis Lemly
The freshwater chronic criterion is appropriate if it is understood that site-specific modifications will likely be necessary under some circumstances.

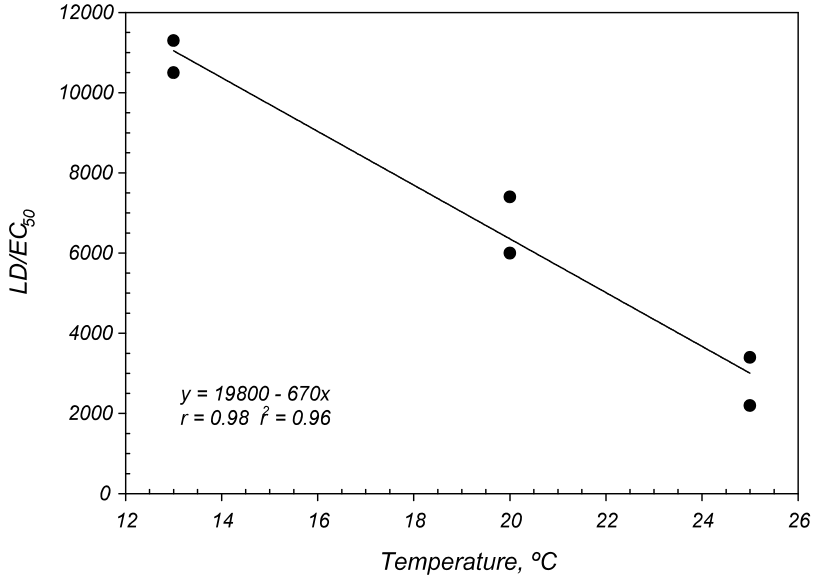
Gregory Möller
No, not entirely. A weakness of the approach occurs on page 58 when the FCV is lowered on the basis of a single additive stress study (Lemly 1993a). Up to this point in the discussion, the process was systematic and orderly. Recognition of this study is valid, but additive, synergistic, potentiated or antagonistic effects as a whole have not been included in the discussion in great detail and certainly not in the FCV calculation. The antagonistic effects of sulfate are explored in the acute criteria development. The potential for beneficial or adaptive effects at low to moderate exposures such as increased immune function, increased growth rates, adaptive enzyme systems for oxidative stress or adaptive biotransformation and elimination observations (thereby increasing tolerance) in naturally exposed populations (specifically excluded in the FCV) are all not quantified in the final development. Species specific responses and cold vs. warm environmental biodynamics of selenium are inadequately treated to justify a cold stress modification to the FCV.

Lemly 1993a appears to be a well conducted study but interpretation of its conclusions should be limited to cold stress on warm water species. Fisheries scientists I have consulted doubt that salmonids would respond similarly and that the bluegill experiment is noteworthy but not definitive when applied to freshwater species as a whole. Indeed, salmonids are biochemically better suited for cold and therefore experience less cold stress. Natural behavior is observed by fish in seeking out a suitable thermocline as a survival response. Given the myriad of potential environmental and biological (species) modifiers to the toxic and beneficial effects of selenium, I would therefore strongly recommend elimination of the results of Lemly 1993a as a modifying datum in the formulation of the criteria. This is especially true in the absence of study replication and the overly broad application of the study interpretation to all fish, cold and warm water species.

It is interesting that the Adams 1976 data from Table 1a presents a different picture of temperature effect on selenium toxicosis. Below, I plot the data from page 20 and demonstrate a negative slope relationship of selenite LD/EC_{50} with increasing temperature. Although one data set is an acute toxicity trial and the other is a chronic trial, I see the Adams 1976 data as significant in limiting any determination of the Lemly 1993a study as definitive. It is clear that the temperature effects of selenium ecotoxicity are not adequately studied or understood to justify incorporation into the FCV. I have often thought it curious that the 4 ºC increase in Belews Lake temperature as a result of power plant cooling has received only minor attention in addressing its aquatic ecosystem decline. Unless EPA wants to get deep into the game of modifying all similar criteria on the basis of temperature effects, warm and cold, it is best not to invoke it in this singular case on the basis of a single, unreplicated study.

In regard to the development of the GMCV, I would recommend EPA perform a 3-parameter log normal regression treatment (vide infra) and shown in the spreadsheet attachments: *Muscle to Whole Body Conversion* (page 1-3), *Ovary to Whole Body Conversion* (page 1-3), *Liver to Whole Body Conversion* (page 1-3) (see Appendix D of the Peer Review Report). This approach will increase the validity of the tissue-whole body Se model derived to calculate the GMCV. Please note that the attached worksheets have not been audited for error and the data and equations developed are shown for information purposes only.

Temperature Effect on Acute Toxicity of Selenite to Fathead Min
Data from Table 1a, Adams 1976



There is a substantial lifespan-through-spawning fish study that is presently concluding and the results should be examined for inclusion into the salmonid GMCV developed in the draft proposal. I have attached a project study report "Effects of dietary selenium on cutthroat trout (*Oncorhynchus clarki*) growth and reproductive performance" by Dr. Ronald Hardy of the University of Idaho (Appendix I - included in Appendix D of the Peer Review Report). Professor Hardy, a former NMFS researcher, is director of the Aquaculture Research Institute and author of the textbook Fish Nutrition.

This 3-year lifespan study examined fish spawned from fish taken from the Blackfoot River (Se affected watershed) and Henry's Lake (background watershed) near the Western Phosphate Resource Area (WPRA) in and around the Caribou National Forest of South Eastern Idaho. The WPRA has had active mining of phosphate for fertilizer and manufacturing for over 80 years and Se release was first observed in 1996. Additional description of the area, selenium releases and the study are found below and in the attached report. In short, fish from the Blackfoot River (affected) and Henry's Lake (not affected) were examined by molecular biology techniques for genetic differentiation, and thereby survivorship bias and none was found (not shown in this report). Over 6800 eggs from the Blackfoot River fish were examined over two years and the % deformed fry were observed at typical or below levels indicated as normal or background (1999 0.76% and 2000 2.6%, Hardy, Appendix 2). A 2-3 year feeding trial of fish spawned from captured adults was started following assessment of reproductive success. Studies were conducted on Henry's Lake and Blackfoot River cutthroat trout groups using a diet modified by 2, 4, 6, 8, 10 mg/kg Se (Henry's Lake fish) or 5, 10, 15 mg/kg Se (Blackfoot River fish) as selenomethionine (control diet 1-2 mg/kg Se). Growth, feed conversion ratios, Se retention and reproductive success were examined. The spawning for the final group of fish is underway now (May 2002). The update summary reports:

> Groups of Henry's Lake cutthroat trout were fed six experimental diets containing 0-10 mg added selenium as selenomethionine/kg dry diet for 124 weeks (868 days, 2.5 years). In the highest dietary selenium groups, whole body Se concentrations reach a high of 12.5 ug Se/g dry tissue after 44 weeks of feeding, the last Se analysis until spawning. No reduction in appetite, mortality, or difference in size was detected among dietary treatment groups during this period. Fish grew at rates that exceed growth rates of cutthroat trout at state and federal hatcheries. Thus, no effects on fish growth, feed intake, or survival were found when fish were fed levels of dietary selenium, supplied as selenomethionine, as high as 10 ug Se/g diet throughout the entire life cycle of the fish.

> Groups of Blackfoot River cutthroat trout were troublesome from the beginning of exogenous feeding. No diet formulation developed for rainbow trout, open-formula, experimental, or commercially-available, supported normal growth or health of the fish. Contacts with state and federal agencies revealed that in all situations where cutthroat of wild origin are reared in captivity, the fry were extremely difficult to rear, suffering large losses and poor growth when fed any commercial or agency-specified diet. A completely new diet formulation was developed at the Hagerman Fish Culture Experiment Station, tested for eight weeks, and tested informally at the Jackson Hole National Fish Hatchery (USFWS) on cutthroat fry and fingerlings of wild origin. Results were positive, and the feeding trial with this group of fish was re-started using this diet formulation as the base to which selenomethionine is added. Once the fish reached the post-juvenile stage, they were weaned to the formulation used to rear the Henry's Lake fish (Se level differed, of course).
>
> No signs of toxicity have been observed in Blackfoot River cutthroat after nearly two years of feeding diets supplemented with 0, 5, 10, and 15 ug Se (as selenomethionine)/g diet. Whole body and egg Se levels of Henry's Lake fish reflected dietary Se intake. Egg levels were much higher in dietary treatment groups than levels typically observed in eggs of wild fish taken from the Blackfoot River. Thus, the objectives of this study, to orally dose cutthroat trout with the form of selenium found in their food chain and produce fish with a range of intake levels and body levels greater than that found in the Blackfoot River watershed, and to determine the acute and chronic effects of Se intake on growth, feed intake, survival and reproductive performance, have been met, or will shortly be met once egg incubation is completed, and their tissues and eggs are analyzed for Se.
>
> Depuration rates of Se from juvenile cutthroat trout varied with dietary treatment group, appearing to depend upon the whole body level (and body burden) at the onset of depuration. Cutthroat trout containing high concentrations of Se reached approximate baseline levels after 32 weeks of depuration (feeding the control diet). During this period, the fish grew approximately 75%. Growth dilution was insufficient to account for the decrease in Se concentration in the fish, suggesting that Se was excreted, most likely in connection with protein turnover. These results suggest that juvenile cutthroat trout, exposed to high environmental Se levels in the upper sections of the Blackfoot River system, are likely to depurate to much lower levels after leaving upstream nursery areas and migrating downstream to post-juvenile and adult rearing areas, where the major portion of their life cycle is spent and where Se concentrations in the river system are low relative to contaminated areas in upstream tributaries.

This data suggests that cutthroat trout and salmonids in general, may be more tolerant to environmental selenium levels and that a salmonid GMCV may be a more accurate representation of an aquatic life protection threshold in these habitats. The cutthroat trout study will be presented at the Fall SETAC meeting and it is currently in preparation for journal submission.

My suggestions for modifying the current approach are thus: Calculate a new salmonid GMCV level including the Hardy data as it occurs in the attached report and as complete data are available (Blackfoot River fish) available early this summer. Compare the GMCVs and if the Bluegill GMCV data remains lowest, use it for the FCV. I would encourage a recalculation of the Bluegill GMCV using data obtained from the more statistically rigorous tissue-whole body, 3 parameter log normal regression approach I have shown. This would be a defendable criteria development that avoids species specific levels. A 9.5 mg/kg FCV (Bluegill GMCV) better approaches the draft salmonid GMCV of 11.64 mg/kg and is still protective of aquatic birds.

Rob Reash
I believe that the freshwater criterion is appropriate based on:

- the medium to express the chronic criterion; and
- the selection of acceptable test data to determine an EC20 (regression analysis).

I do not believe that the lowering of the bluegill sunfish SMCV from 9.5 mg/kg to 7.9 mg/kg, based on the

Lemly (1993) study, is appropriate. As the authors state:

> *The Lemly (1993a) laboratory results, indicating a chronic value <7.9 ug/g dw, are not completely comparable to the other results used to calculate the bluegill GMCV. Lemly (1993a) involved an additional natural stress, exposureto a winter low temperature of 4° C. This appeared to reduce the tissue concentration associated with reduced survival. (page 58)*

I noted, from reviewing this study, that there were extreme responses of bluegill in the two test temperatures (4º and 20º C). While some adverse effects were observed at high selenium/low temperature exposures, there were no adverse effects seen at fish exposed to the higher water temperature (20º C). This raises two points: 1) the Lemly study did not determine the chronic *water temperature* temperature threshold; and 2) if the interactive effect of water temperature and selenium exposure is a consistent physiological "effect", then a greater range of water temperatures should have been tested. Many water bodies in the U.S. have water temperatures approaching 30º C during summer months. Is the interactive effect of water temperature and selenium exposure significant at temperatures higher than 20º C?

In summary, I believe that that combined BPJ decision to 1) exclude the field-derived chronic values for bluegill sunfish from the criterion calculation, and 2) lower the FCV from 9.5 mg/kg to 7.9 mg/kg (based on the Lemly study) is overly conservative. The Lemly study, while providing useful information on the interactive effect of Se exposure and temperature, is not sufficient (by itself) to lower the FCV, especially when other studies did not test a water temperature-Se exposure relationship.

Chronic freshwater criterion:

7. With the goal of being neither under- nor overprotective, how reliable would you expect the criterion to be in application to different sites? Are there any straightforward ways of improving its site specificity?

John Besser
In general, I expect the tissue-based chronic criterion would be more reliably applicable across the range of site conditions than a water-based criterion. However, no criterion based on single-chemical tests, whether based on concentrations in water or tissue, can predict site-specific influences of water quality or interactions with other contaminants.

There is credible evidence that Se exposure can cause or contribute to toxic effects at levels substantially less than the FCV (e.g., Hamilton et al,. 2001a,b; see Appendix E) under some site-specific conditions. In some cases, apparent Se toxicity thresholds for these studies approach nation-wide or regional 'background' tissue Se concentrations. Given both the essential nature of Se and the mandate for derivation of WQCs (i.e., to protect 95% of species tested), I believe it is unrealistic to expect a national WQC for Se to protect all species at all sites. However, it should be acknowledged that protection of at-risk fish species (e.g., species federally listed as Endangered or Threatened, or species occurring in habitats that have been shown to be at greater risk) may require a greater level of protection than that provided by the national WQC.

Steve Canton
The best indication of the overall protectiveness of this value is found in Table 4 with the various bluegill chronic values. While the GMAV is determined to be 9.5 µg/g, based on laboratory studies, the geometric mean of chronic values for field-derived bluegills (data not used in the derivation of the GMAV) is 31.6 µg/g - over three times higher. This strongly indicates that laboratory-based chronic values will be protective (perhaps even overprotective) of wild populations.

The document compares the criterion to the nation-wide database collected as part of the National Contaminant Biomonitoring Program and NAWQA. Review of these databases show that 98% of the fish collected and analyzed for whole-body selenium would meet the criteria (overall mean of 2.99 µg/g - see page 61). The authors conclude that the criterion will pose no real problem of "background" values being greater than the criterion. However, the NAWQA database has few fish data, so is of limited use. The Contaminant Biomonitoring program is certainly more extensive (with an overall mean of 1.9 µg/g), but contains few if any data from areas in the West where the presence of marine shales have resulted in elevated selenium levels in a number of water bodies. For example, there are no data points for Wyoming, only one from eastern Colorado (none from the westslope of Colorado where there are extensive marine shale deposits), only one from central South Dakota (not western South Dakota where marine shales are prevalent), and so on.

If the "reference" sites in the western U.S. that potentially drain marine shales are considered alone (i.e., Green River, UT, Utah Lake, UT, Colorado River, CA/AZ, San Joaquin River, CA, North Platte River, NE, South Platte River, CO, Platte River, NE), the whole-body selenium fish tissue values even with this limited number of sampling sites in the west range from 0.8 to 10.5 µg/g. The mean is 4.4 µg/g (over twice that of the national database) and instead of only 2% of the data exceeding the proposed criterion, over 10% of the data exceeds the value. This limited database would seem to suggest that in some regions of the U.S., this proposed criterion could cause "unavoidable exceedences" to be reported relatively frequently.

Another example that seems to indicate this value might be overprotective comes from a recent study on the

Republican River (CO, KS, NE) which analyzed fish population structure and population health with comparisons to whole-body selenium levels (May, T.M., M.J. Walther, J.D. Petty, J.F. Fairchild, J. Lucero, M. Delvaux, J. Manring, M. Armbruster, and D. Hartman. 2001. An evaluation of selenium concentrations in water, sediment, invertebrates, and fish from the Republican River Basin: 1997-1999. *Environmental Monitoring and Assessment* 72: 179-206). Over 20% of the fish species one year and close to 50% of the species in the second year of this long-term study had whole-body selenium levels greater than the proposed value of 7.9 µg/g - including sunfish species. The study analyzed population and age structure of the fish species collected and concluded there was no evidence of reproductive impairment in these populations despite the elevated selenium whole-body levels.

These quick analyses suggest that regionally (especially in large portions of the western U.S.), the proposed value of 7.9 µg/g may be overprotective. Note, however, that if this chronic value is recalculated with the additional data I've suggested, the lowest GMAV is around 11.6 µg/g (for both trout and sunfish). This value is not exceeded at the reference sites in the National Contaminant database and even in a river with elevated selenium (Republican River from example above), only one or two fish show values higher than that criterion.

One straightforward approach to site-specificity would be do differentiate between waters based on the fish communities present. The final chronic value chosen is specifically protective of bluegills (and presumably other centrarchids). However, in many western streams (for example), this fish family is rarely present (if at all). This is due to biogeography - not selenium. In this case, perhaps the salmonid value of 11.64 µg/g could be applied to the cold-water streams. And in warmwater, lower-elevation streams with cyprinids predominant, the fathead minnow value could be applied. This is one idea for site-specific use of the data presented.

Again, if the criterion is recalculated as I suggest, this may be less of an issue since the salmonid and centrarchid chronic values would be similar, indicating cold and warmwater streams would have equal protection.

Dennis Lemly

Given that the acceptable level for impacts is an EC_{20} (page 47), the criterion should provide adequate protection for most species of fish most of the time. However, if Centrarchids have whole-body tissue residues near the criterion level (7-8 µg/g dw) concurrently with winter conditions (cold temperature and short photoperiod), then unacceptable (>20%) mortality of juveniles may occur due to Winter Stress Syndrome. In this situation, which could be widespread, and probably others yet to be identified, site-specific revision of the criterion will be necessary. However, such modifications can't be done from a tissue-basis alone. Even though EPA has chosen to "sidestep the controversy involved in setting a reliable water concentration" (Versar background document, page 2), the point of regulation for selenium will still be water, whether promulgated as a criterion by EPA, as it has been in the past, or stepped down to states and tribes to deal with on their own, as it will be now. Thus, as a purely practical matter, a national tissue-based criterion will not eliminate the need for states and tribes to set water concentration-based limits on selenium sources, and also develop and implement TMDLs to reduce selenium to acceptable levels in fish tissues. The tissue criterion is a biological target but the mechanism for meeting the target will still be manipulating waterborne selenium. States and tribes will have to decipher what waterborne concentration (standard) is required to keep whole-body residues in fish at or below 7.9 µg/g dw. This necessitates developing new (and/or re-examining old) state- or site-specific, water-based standards. Thus, formulating selenium standards is now a more circuitous process – EPA gives the biological target and it is up to states/tribes to find the waterborne concentration necessary to achieve it, which could be more difficult and time-consuming than the old (1987) water-based criterion technique. I don't disagree with the tissue-based approach, but it is important that EPA not leave states and tribes on their own to wrestle with the issue using trial and error methodologies to set a water standard. With the switch to a tissue-based criterion, proper guidance for site-specific modifications is more necessary than ever before. Because of selenium's unusual ecological risk factors (complex aquatic cycling

pathways and multiple modes of toxicity), implementation guidance for this trace element must be selenium-specific in order for states and tribes to formulate appropriate standards. EPA's current implementation guidance is woefully inadequate because it is generic – it does not provide the necessary degree of specificity. However, in response to this information need (a need that I identified long before the EPA Peer Consultation Workshop or this Draft Criteria Document), I have developed and published a peer-reviewed procedure for deriving site-specific chronic criteria for selenium. The method uses water and tissue concentrations, diagnostic residues, and biological effects to set local criteria for hydrological units. This technique appears as Chapter 7 in my new book *Selenium Assessment in Aquatic Ecosystems* (Lemly 2002). I also present methods for setting environmentally safe ecosystem loading limits (TMDLs, Chapter 8) and delineating hydrological units (Chapter 6). Together, these 3 chapters can provide the guidance necessary to address site-specific questions. The book would appear to be essential for states and tribes in order for them to properly translate the new tissue-based criterion into state- or site-specific standards and limits on selenium sources/discharges. I recommend that, at a minimum, Lemly (2002) be added to the references cited at the end of the "Implementation" section (page 66), as well as to the document's reference list (page Ref-47). I am enclosing a copy of my book along with this review for EPA's information and consideration. If EPA endorses my procedure, it would be appropriate to formally mention it in the last paragraph on page 66 as the site-specific methodology recommended for use by states/tribes to set local water standards to meet the tissue criterion. I have supplied suggested wording in the margin. There is no hidden agenda here – I do not receive royalties from the sale of this book (my Federal employee status prevents it). I wrote it as a service to all those involved in selenium pollution issues, and it appears that it will have much more application now that a national tissue-based criterion is being proposed by EPA. My only interest is in making sure that those who need to conduct hazard assessments and set site-specific standards for selenium have proper guidance on how to go about it.

Gregory Möller

The criterion is intended to safeguard natural resources. The presented data suggest that an FCV of 9.5 mg/kg (Bluegill GMCV) would be sufficiently protective based on the preponderance of current data. Invocation of temperature effects via the Lemly 1993a study into the criteria development complicates the application across cold and warm water species. Unless EPA desires to develop species specific criteria, temperature effects are best not included in the FCV. The 9.5 mg/kg fish tissue value better approaches the salmonid GMCV (cold water fishes) and therefore is directly applicable to a wider range of sites. A 9.5 mg/kg FCV balances the available data consistent with current practice. We do not currently apply warm water stresses to cold water fishes in application of chemical water quality standards. Application of a cold stressed, warm water species FCV is likely to cause concern in the Western and Northern part of the US where the environment and resident species are significantly different.

In the discussion of potential ecosystem impacts of various FCV whole body fish Se levels, one needs to keep track of wet weight and dry weight representations – too often a source of confusion. Food, and therefore selenium, is presented in nature as wet weight. It is very important to note that the wet weight transformation of a 9.5 mg/kg dry weight FCV, calculated with the EPA 80% moisture correction (page 46), yields a whole body wet weight level of 1.9 mg/kg. This number is in the range of commercially produced fish chows and the range of the basal diet fed to the control fish in the attached Hardy study (1-2 mg/kg) (included in Appendix D of the Peer Review Report).[1] The Lemly 1993a study used a Tetramin® control feed with 0.8 mg/kg selenium. This has equally important considerations in the projection of a 9.5 mg/kg dw FCV to ecological risk assessment and food chain effect. The attached work of Hardy (Table 11) shows that a cutthroat trout whole body Se level of 9.37 ± 4.67 mg/kg dw is attained after 2.5 years of dietary exposure at 9.5 mg/kg Se in the feed.[2] This analysis suggests that concerns should be minimal and an FCV value of

[1] The fish diet Se value, 1-2 mg/kg is a dry weight value. The moisture content of the feed by proximate analysis was 6.3% (Hardy Table 1). A wet weight value is substantially the same.

[2] Calculated from 8.0 mg/kg selenomethionine added to a 1.5 mg/kg basal diet concentration.

9.5 mg/kg dw is protective.

I have concerns that overly protective selenium criteria can venture into the realm of the adaptive response of the antioxidant enzyme system that includes Se-glutathione peroxidase. In work examining liver Se relationships with other metals, I developed the hypothesis that oxidative stress could cause an increase in bulk hepatic tissue and body burden Se levels (Möller 1996) and this could contribute to the observation of selenium problems in multiply contaminated zones such as agricultural drainage ponds. One of my students completed a 30 day, randomized block, static replacement pilot study with juvenile Fathead minnows. All fish were fed commercial trout chow that had background 1 mg/kg total Se. The water for treatment fish contained the herbicide Paraquat, a redox cycling compound understood to cause oxidative stress. The table below shows the whole body Se levels increased 128% compared to the controls. Increases were also noted for iron and manganese, metals also involved in the antioxidant enzyme system. These pilot study results suggest the potential for Se ecosystem effects that are unrelated to Se release. Selenium (Hoffman et al. 1998a, 1998b) itself has been identified as an oxidative stress inducing compound and thus joins the ranks of other NADH reducible metals, complexes and organic molecules that can induce this effect in organisms. We are presently developing a total set of enzyme, antioxidant and free radical assays to take this study into a formal phase.

Fathead minnow: whole body antioxidant metal increase with Paraquat (50 µg/L) exposure (n=5 groups).

mg/kg dw	Control	Paraquat Treated	% Increase	p Value
Se	0.53	1.21	128	0.27
Mn	12.8	16.2	27	0.10
Fe	142	619	334	0.23

Site specificity of the criteria may be enhanced by commenting on population level concern. Lotic aquatic systems often have confined populations whereas lentic systems are often migratory. There have been several observations of varying degree of impact in lentic vs. lotic Se exposure and this may be the result of the in-migration and out-migration behaviors of the respective populations. The major chronic toxicity endpoint of concern for selenium is reproductive failure. You will not find many biologists that will disagree that reproductive failure is a population level concern. Inserting a population reference will prevent the observation of one fish with whole body Se exceeding the FCV from being interpreted as an indicator of ecosystem collapse.

There is significant regulatory guidance concerning population level concerns in environmental management:

> *"ecological effects of most concern are those that can impact populations (or higher levels of biological organization)."* (USEPA 1997).
>
> *"Superfund remedial actions generally should not be designed to protect organisms on an individual basis (the exception being designated protected status resources, such as listed or candidate threatened and endangered species or treaty-protected species that could be exposed to site releases), but to protect local populations and communities of biota."* (USEPA 1999).
>
> *The ecological entity to be protected "can be a species (e.g., eel grass, piping plover), a functional group of species (e.g., piscivores), a community (e.g., benthic invertebrates), an ecosystem (e.g., lake), a specific valued habitat (e.g., wet meadows), a unique place (e.g., remnant of native prairie), or other entity of concern."* (USEPA 1998).

The biogeochemical cycling of selenium in aquatic ecosystems makes population concerns important. I would recommend inserting into the criteria the sentence:

"The potential for reproductive failure in selenium exposed organisms makes population level protection important."

Rob Reash

These are appropriate questions to ask. Because the FCV was selected based on protecting the most sensitive species in the chronic database (bluegill sunfish), the criterion will likely not be under-protective in most settings. There may be some settings where a more stringent criterion is justified, e.g., water bodies where chronic reproductive effects on fish species are apparent, even though the national FCV criterion is not exceeded. Most studies have shown that, once loadings of selenium to a waterbody are decreased, the levels of Se in biota will also decrease, but at a much slower rate than levels in the water column. There are also settings where the national FCV criterion may be over-protective. One example would be water bodies that do not have persistent populations of sunfish, e.g., coldwater streams or Western streams and rivers that have a natural depauperate fish community). In such cases, EPA should consider providing states and tribes the option of revising the national FCV criterion to reflect representative species on a water body-specific, regional, or statewide scale.

In settings where long-term Se loading has occurred (e.g., fly ash pond receiving streams, refinery wastewater receiving streams), application of the final FCV criterion may not be appropriate. Fish populations, exposed to Se in the food chain for several generations, may exhibit acceptable reproductive success due to several mechanisms. There is some evidence that the toxicity of selenium is antagonized in the presence of other trace metals. Recovery of fish populations may be evident even though whole body (or other tissue) Se levels exceed the FCV criterion. A good example of this is Hyco Reservoir, where bioaccumulation-induced toxic effects were observed in some sunfish populations. In 1990 Carolina Power & Light Company installed a dry fly ash disposal system, essentially eliminating loading of Se to the reservoir. During annual monitoring studies conducted after 1990, utility biologists saw evidence that the reproductive success of bluegill and largemouth bass was considerably greater compared to pre-1990 years; yet, extrapolated whole body tissue levels of Se (based on muscle and liver Se levels) in these two species were higher than 7.9 mg/kg (CP&L, 1993). In such "closed system" settings, the export of selenium from the system is very slow. Thus, a considerable time period may be required before Se levels in fish return to "background" levels. In settings such as this, loading of selenium to the water body has been eliminated, yet fish tissue levels will remain somewhat elevated for several years. The application of the national FCV criterion to such a water body may not be appropriate.

Chronic freshwater criterion:

8. Although the criterion was not derived using wildlife criteria derivation procedures, EPA noted some evidence that the criterion would protect piscivorus birds. Are you aware of other data relevant to the protectiveness of the criterion for birds?

John Besser
The authors made little attempt to review the large literature of Se toxicity to birds, so I will not attempt to list the many studies they did not consider. I think they have wisely focused on the most definitive, controlled dietary exposures conducted by Heinz and colleagues. My reading of the avian toxicity literature (not my specialty) suggests that toxic effects of Se on birds, expressed as concentrations in either diet or egg tissue, tend to occur in approximately the same range as for fish. Thus I would expect that the FCV for toxicity to fish should be adequately protective for birds as well.

Steve Canton
Most other publications I am aware of regarding dietary uptake thresholds for birds are for birds with insectivorous diets (e.g., Adams et al. 1998 - which is in the list of references, but is not cited in the discussion of safe levels for birds on pages 59-60 of the draft criteria document). This, and other papers by those authors, support the conclusion that the value chosen (or even if modified as suggested above and below) would be protective of birds.

Dennis Lemly
I am not aware of other data relevant to the protectiveness of the criterion for birds.

Gregory Möller
Opresko et al. (1995) developed dietary selenium thresholds for piscivorus birds using mallard toxicity data for selenite (Heinz et al. 1987) and selenomethionine (Heinz et al. 1989). Selenomethionine most closely resembles actual diets. Heinz et al. (1989) exposed mallards to selenomethionine fortified feed and evaluated reproductive success and hatchling survival. The NOAEL and LOAEL for reproductive impairment observed 4 and 8 mg/kg. Opresko et al. (1995) estimated dietary selenium thresholds for these piscivorus birds using the ingestion rate and body weight for mallards reported in Heinz et al. (1989) and species-specific ingestion rates and body weights for piscivorus birds (belted kingfisher, great blue heron, osprey). As shown in the draft criteria document (page 60), these dietary thresholds ranged from 10.6-12.2 mg/kg, suggesting that a fish tissue-based criterion of 9.5 mg/kg (Bluegill GMCV) would be protective of piscivorus birds.

The chronic toxicity of selenomethionine to the piscivorus black-crowned night heron was evaluated by Smith et al. (1988) in a 94 day reproductive study. Their work observed a dietary NOAEL of 10 mg/kg based on reproductive effects. Thus, a whole body fish tissue criterion of 9.5 mg/kg is less than the chronic NOAEL for this piscivorus bird.

The trophic transfer of selenium from food to bird egg was analyzed in field-collected data for several species of birds at 15 sites in the Western U.S. (Adams et al. 1998). This work suggests that trophic transfer is much less in the field than in the laboratory studies performed by Heinz et al. (1989). The field data indicate that on average the trophic transfer is 1.1 while the laboratory study indicates trophic transfer factors of 2-4 from food to mallard duck eggs suggesting a positive bias of the laboratory determined rate vs. the field determined rate.[1] Additionally, by research design, the field study integrates species variability, genetic differentiation

[1] For comparison, the average selenium food to egg transfer factor in the Hardy Cutthroat trout study was 1.2 (Hardy, Table 11).

and food Se speciation diversity into the development of a food to bird egg trophic transfer rate. A high trophic transfer rate in the laboratory study of Heinz et al (1989) is not unexpected since selenomethionine, the dosing agent in this study is more actively incorporated into tissue than selenite, selenate or selenocysteine (Burke, 1986), all of which would be components of a natural diet in varying proportions. Use of the 1.1 trophic transfer factor to assess selenium transfer from food to bird eggs indicates that at a dietary concentration of 9.5 mg/kg would yield a bird egg concentration of 10.45 mg/kg. This level is below the calculated concern thresholds of 16 mg/kg (Fairbrother et al. 2000), 12-15 mg/kg (Adams et al. 2002 In Preparation) and 12.8 mg/kg (Ohlendorf 2002, in press). It is above an earlier 6-8 mg/kg conservative threshold suggested by Skorupa et al. (1996) in a U.S. Fish and Wildlife Service guideline. The preponderance of work in this area suggests that a 9.5 mg/kg FCV (developed from the Bluegill GMCV) would be protective of birds.

A large population-scale study of avian selenium effects in the selenium contaminated WPRA ecosystem is in it final stages. The study is being conducted by Professor John Ratti and Professor Edward Garton of the Wildlife Resources Department of the University of Idaho. The project has evolved from an egg study to a nesting and reproductive success study. In 1999 and 2000, approximately 250 and 350 eggs were collected, respectively, representing about 20 species. Seven nesting success indicators were greater on mining impacted sites and eight nesting success indicators were greater on background sites, allowing limited differentiation of reproductive success in the two environments. In 2001, the project attempted to use four species for reproductive success studies consisting of the American Robin, Red-Winged Blackbird, Coot and Yellow-Headed Blackbird. Approximately 450 eggs were collected but because of the low water year, only the robin and red-winged blackbird could be represented in the study. The study measured hatching and fledging success using a significant number of nesting sites that represented background and mining disturbed areas. The researchers conducted a stratified random sample of aquatic/riparian habitat patches for the entire study area incorporating sampling strata based on a combination of National Wetland Inventory polygons and mining vs. reference regions. They used complete counts of 57 sites to determine bird abundance, total number of nests started and nest success (both hatching and fledging) for 4 species (red-winged blackbirds, yellow- headed blackbirds, American coots and American robins). The field study found more than 600 nests but droughty conditions limited abundant nest data only for red-wing blackbirds (aquatic) and robins (terrestrial).

The field research teams took one egg from each nest for Se analysis and monitored nest activity over the season. With this data, logistic regression can be used to evaluate how hatching success and fledging success decreased with increasing Se levels. More than 45% of the eggs had Se levels above 5 mg/kg and many were above 12 or 16 mg/kg, levels that have been identified in the literature as significant in exposure and risk (vide supra). However the surprising result was that all of the logistic regressions for hatching success and fledging success in both red-wings and robins showed positive slopes for egg Se concentration. <u>In all cases increased Se levels in bird eggs were associated with higher levels of nesting success.</u> Likewise the field teams found not a single case of terata in more than 1,000 eggs and fledglings examined from over 20 species. The investigators expected that reproductive success would start to decline at very high levels of Se but the nests with the highest levels of Se (around 30 mg/kg) both hatched and fledged young successfully. The researchers have hypothesized that the observed beneficial effect of selenium exposure may be a result of the migratory behaviors of the WPRA nesting bird populations throughout the largely marginal or deficient Se areas of the West and Northwest areas of North America.

These results, in light of the observations at Kesterson and elsewhere, suggest that birds may have variable responses to Se exposure and therefore are poor candidates as sentinel indicators. The migratory behaviors of many birds may limit opportunistic exposure to isolated selenium contaminated zones. Indeed, some feeder fish sampled from the primary contaminated areas of the WPRA exceed the proposed whole body Se criteria. Primary Se release sites have demonstrated water Se levels in excess of 2 mg/L and some secondary waters show significant exceedances of the current 5 ug/L Se criteria. Yet, avian population modeling at the site indicates that if there was a "magical" conversion of mine sites into background sites (i.e., the population

dynamics, including reproductive success, of mined areas were substituted into the model for background sites) no population level change would occur. Twenty year bird population modeling in this study shows stable populations. This suggests that a satisfactory level of protection is afforded birds under the proposed criteria approach. The WPRA bird studies will be presented at the Fall SETAC meeting and they are currently in preparation for journal submission. Additional Spring 2002 nest surveys are underway.

Rob Reash

This discussion in the criteria document is good and technically accurate, but I have a few comments. First, the discussion of potential impacts to piscivourous waterfowl is not comprehensive. If EPA was truly interested in a comprehensive evaluation of how protective the FCV criterion was to wildlife, than a more extensive review would be necessary. This raises the question of whether wildlife effects should be discussed in the criteria document. Opinions on this issue will vary between "absolutely" and "no". EPA should acknowledge that Se effects on wildlife species have been documented, and a compilation of wildlife effects data is not that unreasonable, however EPA should clearly state that a wildlife criterion methodology has not been finalized for national usage. The data acceptability guidelines for the development of aquatic life criteria should, in likewise manner, be elucidated for wildlife criteria.

I believe that the wildlife effects discussion should be placed in an appendix.

VI. SPECIFIC COMMENTS

Gregory Möller
The document uses µg/g as the concentration unit. Consider using the preferred SI unit mg/kg.

Gregory Möller
Page 2, Paragraph 1, Line 3
It is doubtful that "substantial" concentrations of Se(II) are ever found in oxygenated alkaline waters. USEPA 1987a is a meta-analysis and a weak reference for this.

John Besser
Page 2, Paragraph 2 (Besser comment #1)
In the second paragraph, statements are redundant re: 'producing gaseous dimethyl selenide' and 'methylate selenium... to volatile $(CH_3)_2Se$'.

Gregory Möller
Page 2, Paragraph 2, Last Sentence
Please provide a reference for the last sentence.

Gregory Möller
Page 2, Pargraph 3, Line 3
"uncontaminated" is an awkward, inaccurate descriptor. Try: non-seleniferous

Gregory Möller
Page 2, Paragraph 3, Line 5
Try alkaline rather than "drier".

Gregory Möller
Page 2, Paragraph 3, Line 11
Delete "in high concentrations" – these are imprecise words.

Gregory Möller
Page 3, Paragraph 2, Line 3
Try biosynthesis rather than "manufacture".

Gregory Möller
Page 3, Paragraph 2, Line 3
Delete "the damaging (oxidizing)".

Gregory Möller
Page 2, Paragraph 3, Line 14, continuing page 4, Paragraph 1, Lines 2 and 3
This is a broad overstatement. Nutritional research has demonstrated variable uptake - some homework needed here.

From the attached Hardy study (page 3-4) (included in Appendix D of the Peer Review Report):

> The biological availability of selenium for fish differs with selenium source. Bell and Cowey (1989) reported that the selenium present in fish meal has a low availability to rainbow trout, while that of selenomethionine is high. Lorentzen et al. (1994) observed differences in bioavailability between selenite and selenomethionine on the basis of muscle and whole-body selenium concentrations. Fish fed diets supplemented with selenomethionine had 3-5x higher muscle

selenium levels than fish fed equivalent dietary selenium levels, with sodium selenite as the supplement. Studies of bioavailability are principally focused on avoiding selenium deficiency by taking into consideration the bioavailability of selenium from various dietary sources.

Steve Canton
Pages 4 and 5
This section roughly outlines the methods for deriving the criteria proposed in the document. The conclusion that acute and chronic criteria derivation can and should follow different approaches is broached here. This might be a good place to cite Canton (1999), which recommended this split in approaches (I know - this is my paper, but I can't help but want to see it cited in the document since it is in the list of references!).

Steve Canton
Page 6, Paragraph 4, Last Sentence
This last phrase "which is the most sensitive SMAV for selenite in the database" is a bit overstated. Even though other data were not used because of the criteria in Stephan, et al. (1985), they show a similar range.

John Besser
Page 7 (Besser comment #2)
Gammarus: The inclusion of the single toxicity value for adults biases the SMAC/GMAC upwards. In other cases (e.g., *Onchorhynchus* ELS tests, page 8), data from tests with 'less sensitive' life stages are excluded.

Steve Canton
Page 8, Paragraph 4
As noted earlier, there is another study available providing acute toxicity values for green sunfish (*Lepomis cyanellus*) - the study by Cumbie and Velte (1986). As I note in my comments to Table 1, this study results in a SMAV of 15,138 for green sunfish and changes the resulting GMAV for *Lepomis* to 20,771. I have included a copy of this paper with this review, should the authors wish to use the data.

John Besser
Page 9 (Besser comment #3)
The FAV for selenite is greater than TWO (not one) SMAVs.

Steve Canton
Page 9, Paragraph 1
The last sentence could make the reader "uncomfortable" saying the FAV is higher than the lowest SMAV. To add a level of comfort, the authors could also note that the CMC is half the lowest GMAV.

Steve Canton
Page 9
I only cursorily checked the saltwater values for mathematical errors (found none). I don't have the knowledge of this literature to do more.

Steve Canton
Page 12, Paragraph 4 - *Daphnia*
The authors include a study by Johnston (1987) in the references, but the data for acute toxicity to *D. magna* is not included in the table. If the data from that study are included (LC50 of 750), the SMAV becomes 1,826 and the GMAV for *Daphnia* becomes 882.1 Since this is one of the four most sensitive genera, it results in a slight change in the FAV and CMC (noted on comments to page 33 and in comments above).

John Besser
Pages 12-13 (Besser comment # 4)
Ceriodaphnia, Daphnia: Exclusion of static/measured studies biases two SMAVs *(C. dubia, D. pulex*) selenate substantially lower. Although the Guidelines specify using data from flow-through tests preferentially over those from static tests, the use of static conditions should not be a problem in general for selenate, which is chemically stable and highly soluble. More importantly, static test methods are standard (and appropriate) for tests with *Ceriodaphnia* and *Daphnia*.

John Besser
Page 13 (Besser comment #5)
Gammarus: The exclusion of two studies that suggest MUCH greater toxicity of selenate (at lower sulfate?) is troubling.

Steve Canton
Page 13, Paragraph 3
I found it ironic that the data used in the 1987 criteria document for *Gammarus*, which resulted in a very low FAV for selenate at the time, are now not even used (replaced by more recent, flow-through tests). Subsequent testing seems to indicate that this amphipod is more tolerant of selenate than the earlier tests would indicate.

John Besser
Page 14 (Besser comment #6)
Onchorhynchus: Two static tests that were excluded produced much lower LC50s for rainbow trout.

Steve Canton
Page 14, Paragraph 2
The authors missed the acute toxicity data from Ingersoll, et al. 1990 for *Chironomus riparius* for both selenite and selenate (noted on tables). If used, this genus becomes the eighth most sensitive rather than *Paratanytarsus*.

John Besser
Page 15 (Besser comment #7)
Sulfate dependence: Even if slopes are 'mild' and differ among species (but are always positive), an average slope to correct for sulfate concentrations should improve the reliability of acute WQCs for selenate. The concern about under-protection in cases where selenite is the predominant species may be valid, but I doubt that selenite is likely to be the predominant species in sulfate-rich waters.

Steve Canton
Page 16, Paragraph 2
The wide range of sensitivities in the four most sensitive genera is a direct result of the need to use the single GLEC flow-through acute test for *Ceriodaphnia* - which has an LC50 5-times lower than the other tests for this genus. This, plus the still limited database for selenate, again results in a lower FAV for selenate than selenite even though most of the data would indicate otherwise (as noted later in document).

Steve Canton
Page 16, Paragraph 5
The changes/additions in available toxicity data noted below for Tables 1 and 2, result in changes in Table 3 (noted on comments to Pages 37-38). The result is that there are now matching data for 22 species and 18 are more sensitive to selenite.

Steve Canton
There are two page 16's!

Steve Canton
Page 18, Table 1a, Midge
The authors seemed to have missed the data for *Chironomus riparius* from Ingersoll, et al. 1990 (however, other data on *Daphnia magna* from that paper are used). If their data are included (LC50 = 14,600), then the GMAV changes, as noted with comments on Table 2a below.

Steve Canton
Page 22, Table 1a, Bluegill
Again, as noted above, I've attached a file with a study by Cumbie and Velte (1986) that provides LC50 values selenite for green sunfish (flow-through, measured values). This doesn't change the bluegill SMAV, but does add data to modify the GMAV for *Lepomis*, as noted in Table 2a.

Steve Canton
Page 23, Table 1a, *Daphnia magna*
Again, the data from Johnston 1987 are missing (LC50 = 750). This lowers the SMAV to 1,826 (down from 2,118).

Steve Canton
Page 24, Table 1a, *Chironomus*
Again, missing data on *C. riparius* from Ingersoll, et al. 1990. The LC50 is 10,500, changing the GMAV to 15,775 (down from 23,700 on page 24).

Steve Canton
Page 31, Table 2a
selenite ranking: If new data on *C. riparius* and *L. cyanellus* are included in calculations, the GMAV for *Lepomis* changes to 20,771 and the GMAV for *Chironomus* changes to 26,328. This changes the ranks - *Lepomis* becomes 20th most sensitive (not 23rd) and *Chironomus* becomes 24th most sensitive (not 26th).

Steve Canton
Page 33, Table 2a - selenate ranking
If the new *C. riparius* data are included, the GMAV becomes 15,775 and *Chironomus* would be ranked 8th most sensitive.

Steve Canton
Page 34, Table 2a - selenate ranking
If the Johnston 1987 data are included, the GMAV for *Daphnia* becomes 882.1.

Steve Canton
Page 36, Table 2b - FAV and CMC values
With the new values noted in Table 1a above, the FAV for selenate would change slightly to 362.7 µg/L and the CMC to 181.4 µg/L. These are slight modifications in the existing calculations, but represent perhaps "more correct" numbers incorporating all the data (*at least I think this is all the data unless the authors find that those studies I think need adding are deficient in some way and shouldn't be used!*).

Steve Canton
Page 37-38, Table 3a
Data added in the tables above mean additional information is available for this table. *Chironomus riparius* has SMAV for selenite of 14,600, for selenate 10,500, and the ratio is 1.390. *Lepomis cyanellus* has an SMAV for selenite of 15,138, with no value for selenate and no ratio possible. For *Daphnia magna* the SMAV for selenate changes to 1,826, changing the ratio to 0.496.

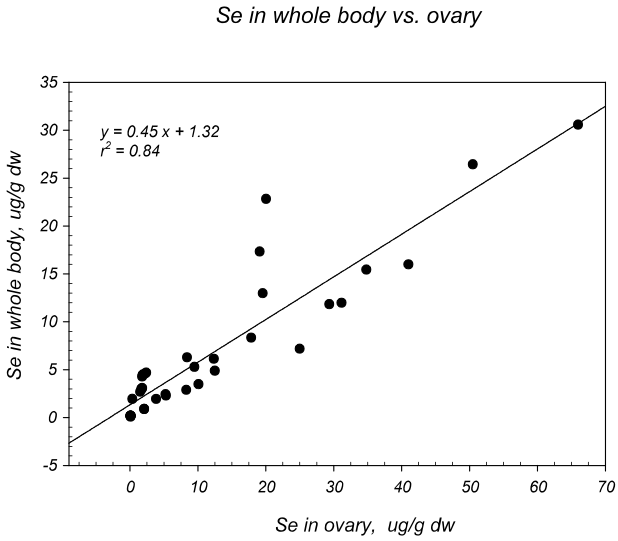
Steve Canton
Page 43, Paragraph 2
This paragraph notes that the authors specifically used studies with either diet + water or diet alone studies for reviewing chronic effects. This seems to indicate that the follow-up studies on the Monticello streams, where diet was the only exposure route, should also be used to provide chronic effects levels (see notes below).

John Besser
Page 44 (Besser comment #8)
Although the availability of data for Se concentrations in larval fish tissue is a legitimate practical limitation for those implementing the proposed chronic WQC, I see no reason why Se concentrations in larval tissues should be excluded from the analysis used to derive the chronic WQC. This is especially relevant because most of the effects described in Table 4 are observed in larval or juvenile fish, not adults.

John Besser
Page 44 (Besser comment #9)
Although the text states that data from twelve studies were used for the regressions of whole-body Se vs. tissue Se, only six studies are cited in Appendix G. Also, it is not clear whether regressions for muscles and ovary tissues were used preferentially to the weaker correlation for liver tissue, or whether predictions based on more than one tissue were averaged to reduce uncertainty.

Steve Canton
Page 44, Paragraph 4
Too bad the relationship between whole-body and liver is so much weaker. As noted when I reviewed the studies provided, it appears that many of the chronic values for trout came from this equation.

Gregory Möller
Page 44, Paragraph 2, 3; Page 45 Figure 4; Page 46, Paragraph 1
EPA must correct an error that occurs in Figure 2 on page 45 and in equation II found on page 46. In checking the data from Appendix G for development of equations that relate muscle, ovary and liver Se concentrations to whole body concentrations, I find an error in the determination of equation II. The regression statistics developed for the ovary conversion are incorrect. The corrected analysis, $[Se]_{whole\ body} = 0.45[Se]_{ovary} + 1.32$, is shown in the figure below. A spot check of the data (p H-21) calculated by the equation below yielded a correct result and this suggests that the error is a typo and was not propagated through the calculated tissue conversions. It appears that the r^2 value was inadvertently substituted for the slope value in the written equation. However, ordinary linear regression is not the best approach.



The tissue conversion approach shown by EPA outlines a method to increase the amount of data available for whole body Se levels vs. effects from the cited literature using linear regression. Although the relationships are clear, a review of the statistical approach used may offer alternative modes of analysis that will increase the rigor of this operation and the subsequent use of the modified data in the calculation of effects relationships. Specifically, many biostatisticians may expect to see the regression be a log-log equation especially since the data range exceeds an order of magnitude. Confirming this is the heteroscedasticity (non-uniform variance) of the scatter about the regression line which gets larger as the concentrations get larger. To correct for this I worked with a biostatistician colleague on a possible alternate approach to model the relationship between whole body fish Se concentrations and muscle, ovary and liver concentrations. Descriptions of the approach basics are found in Helsel and Hirsch (1992) and similar statistics texts.

The results of this effort are found in the attached spreadsheets: *Muscle to Whole Body Conversion* (page 1-3), *Ovary to Whole Body Conversion* (page 1-3), *Liver to Whole Body Conversion* (page 1-3) (included in Appendix D of the Peer Review Report). In these spreadsheets, the muscle, ovary or liver tissue Se data is tested for lognormality and goodness of fit (page 1). The whole body data is similarly tested (page 2). The estimated lower bound of the tissue values was found by optimizing the r^2 of the fit plot regression.[1] On page 3 of the spreadsheets, I show the 3 parameter lognormal plot and regression equation as well as the 2 parameter normal plot and regression equation.[2] In all cases (muscle, ovary and liver) the goodness-of-fit statistics are better for the 3 parameter log normal regression model. In the third graph of page 3 in each series, I examine the assumption of no difference between species that is implicit in the development of the equations I, II, and III on page 46 of the draft criteria document. For the muscle analysis, I find satisfactory support for the assumption. In the case of ovary and liver, this assumption is weaker. Examination of the trout liver values compared to the bluegill and bass demonstrates significant separation of the observations. The possible explanations for the apparent difference are many and include trout having an enhanced Se hepatic biotransformation and elimination efficiency. However, given the small number of observations, separating trout data on the basis of species difference is probably not justified in the current treatment.

[1] Optimizing the r^2 was accomplished by plotting the standard normal variate (adjusting for ties) on the x-axis against ln(x-l) on the y-axis, changing l until r^2 was maximized. This was done using Excel's RSQ(ARRAY 1, ARRAY 2) function, with ARRAY 1 being z and ARRAY 2 being ln(x-l).

[2] The exploratory calculation for regressions of the 2nd kind was performed on another spreadsheet and is not shown.

The results of the analysis are thus:

- The ordinary least squares regression presented by EPA is invalid.
- All of the data (whole body, muscle, ovary, and liver) are log normally distributed.
- A 3-parameter, log normal regression represents a more accurate representation of the tissue-whole body Se relationships.
- Batching different fish species in the data sets is satisfactory for muscle, but less so for ovary and liver.

The modeled numerical relationships between whole body and tissue specific Se concentrations are:

$$[Se]_{whole\ body} = 1.63\ ([Se]_{muscle} + 0.78)^{0.80} - 1.23 r^2 = 0.96\ (r = 0.98)$$

$$[Se]_{whole\ body} = 1.57\ ([Se]_{ovary} + 0.74)^{0.68} - 1.08\ r^2 = 0.85\ (r = 0.92)$$

$$[Se]_{whole\ body} = 0.24\ ([Se]_{liver} + 2.47)^{1.04} - 0.67\ r^2 = 0.78\ (r = 0.88)$$

Steve Canton
Page 46, Paragraph 1 (after equations)
There seems to be some inconsistency between this paragraph, which says wet weight numbers were converted to dry weight assuming a moisture content of 80% and the text on Page H-21, where it says 85% moisture content used for ovary data. Perhaps this sentence should be qualified to say this is for flesh or other tissue, but that it can vary with type of tissue.

Note, also, that we now may have layers of conversions used to develop the actual chronic level based on whole-body tissue data - i.e., conversion from wet to dry weight, and then possible conversion from a wet-to-dry concentration for a particular organ to dry weight for whole-body concentrations. I understand that there really isn't anyway around this, but some mention of the potential sources of error in this multiple conversion pathway should be mentioned.

Steve Canton
Page 47, Paragraph 1
This is where they explain why they use an EC20 for effect levels, based on the revised ammonia criteria document. This is a fairly recent document - they could have used an IC25 and cited an older guidance for chronic effects used in the whole effluent toxicity testing program for 15 years.

Steve Canton
Page 47, Paragraph 2
Looking for chronic effects through the mere presence of deformities may be overstating an actual chronic impact. I tend to think of chronic effects as those that adversely affect the fitness of the population to maintain itself (i.e., something that would adversely affect reproduction, survival, growth to maturity, etc.).

Steve Canton
Page 47, Paragraph 3
This paragraph starts with the phrase "Only data sets that met the following conditions..." In fact, that is not the case. As I read through Appendix H (and the text on the following pages of the main text), it became clear that only a few of the studies actually met all four test conditions listed here (in fact, the Lemly study later used to set the final chronic value didn't meet half the criteria!). The authors probably have no choice in "bending" these rules so they can actually use what appears to be otherwise good data. As such, they may want to make these less "must have" conditions, and more "would like to have" conditions!

Steve Canton
Page 49, Paragraph 1
I am glad they could find something other than a fish to show a chronic effect - but since they are heading to fish tissue as the measurement endpoint for the final chronic value, do we really care about a rotifer? Maybe leave this in the appendix, but take it out of the main document (unless the authors want to show that the final number chosen would be protective of non-fish aquatic life?).

Steve Canton
Page 50, Paragraph 2, fourth line from bottom of paragraph
Considering the MATC is back-calculated from liver data, use of three decimal places is really overstating the precision of this data point! (This goes for the top of Page 51 as well).

Steve Canton
Page 52, Paragraph 1
This is a continuation of the discussion of fathead minnow values from Dobbs et al. 1996. For some reason, the authors use the LOAEC (<73 µg/g) as the chronic value from this test. Yet, the study does appear to have a NOAEC value as well (45 µg/g). Why not calculate a geometric mean (like other studies) from these data and use 57 µg/g as the chronic level from this study?

Steve Canton
Page 52, Paragraph 3
Again, the use of three decimal places is a bit optimistic for precision! In the last sentence, could add the analysis in Appendix H where it is noted that no effects were found for any reproductive parameter.

John Besser
Pages 53-57 (Besser comment #10)
Several studies relevant to this section seem to have been overlooked:

(a) Finley (1985). This study is listed in Appendix F (Unused data) with the justification of 'selenium...component of effluent, fly ash, formulation, mixture [etc]', even though the exposure was based on field collected diets from the same source as Bryson et al (1984, 1985a) and Gillespie and Baumann (1986). The omission or inclusion of this study would probably have little or no effect on the SMCV for bluegill.

(b) Cleveland et al. (1993). This study is cited previously with regard to the 'questionably low' tissue Se concentrations associated with toxicity in a water-only study, and it is also listed in Appendix F (Unused data) under the 'mixture' category. In fact, the dietary component of the study was conducted with an appropriate diet, spiked with Se-methionine. The study showed no effect of Se-contaminated diet on survival of juvenile bluegill at tissue Se concentrations up to 13 ppm dwt in a 90-d study. However, they reported small, but statistically significant, reduction in condition factor, K. It should be noted that weight (needed to calculate K) was not reported, and was presumably not significantly reduced. Although this finding may not be considered an 'unacceptable effect' (i.e., no effect on survival or growth, less than 20% reduction in condition factor), this dietary study in this paper should be discussed. [This apparent affect on condition factor would be associated with a chronic value of approximately 6.0, less than the GMCV and draft chronic criterion, but similar to the chronic value for the effect on hemorrhage described by Hermanutz et al (1986), and discussed below.]

(c) Hermanutz et al (1992). . This peer-reviewed study, which is similar to the subsequent, unpublished study that is discussed at length (Hermanutz et al. 1996; see comment #11 - Specific Comment regarding page 55 or see Appendix A of the Peer Review Report), is listed in the References but is not mentioned in this section or in Appendix F. The earlier study did not include the 'controversial' 2.5 ppb treatment, but it

showed more pronounced toxicity in the 10 ppb treatment (significant effects on adult growth and survival and embryo hatch, survival, edema, and hemorrhage). The chronic value for this study would be <23 ppm. The authors may be justified for omitting this 'less than' value from the calculations for the GMCV for *Lepomis*, as they did the results of several field-based studies, but the results of this study should be discussed.

Dennis Lemly
Pages 53-57
I have several comments written on the margin of pages 53-57 (included as part of Appendix C of the Peer Review Report). These are intended to improve the validity of the discussion, not change the EC_{20s} or chronic values.

The statement on **page 53, paragraph 3**, "In some field studies, chronic tolerance to selenium appears to be much higher than in laboratory studies", is misleading and does not correctly interpret the literature cited. The underlying problem is that the authors are improperly equating different exposure levels, life stages, and biological effects endpoints in order to support this assertion. It is important to know that data for the polluted areas of Belews Lake and Hyco Reservoir come from ecosystems that were saturated with selenium, including high residues in fish. It is not surprising, therefore, that the resulting chronic values and EC_{20s} based on these data are much higher than for controlled laboratory and stream studies where exposures (food chain residues) were manipulated and were limited to low-to-moderate concentrations. This is why the lab and stream studies were done in the first place, that is, to ascertain the low-level and threshold effects, since the field food chains were grossly polluted and preliminary evidence suggested severe impacts on fish. Concentrations in field biota were typically well above the highest levels of contamination observed in the lab/stream – not surprisingly, samples of tissue yielded relatively high chronic values and EC_{20s}. There were no "low selenium" field exposures to provide an estimate of the threshold response or to bracket the lowest effect level as there were for the controlled studies. If you only have high levels of contamination to sample, you're only going to get high tissue residues, chronic values, and EC_{20s}. What the authors suggest as tolerance in natural field settings can be explained simply on the basis of different exposure conditions.

Another problem concerns the authors' inappropriate mixing of endpoints. For example, the EC_{20} for deformities in Belews Lake fish, and the chronic value for the Bryson and Gillespie (BG) field studies, are not directly comparable to the effect thresholds determined in the Hernmanutz and Coyle (HC) studies. For one thing, the HC number represents an effect threshold whereas the chronic values for the BG studies are a "concentration in the female parent associated with this high occurrence of mortality of hatched larvae" (**page 53, paragraph 4**), clearly not a threshold, and the Belews number is a 20% effect level, also not a threshold – the authors incorrectly compare three different effect levels. No wonder the HC threshold is "approximately 3 times lower than those recorded above" (**page 54, paragraph 2**). Also, the Belews number is for teratogenic deformities, the BC number is for parent fish, but the HC studies used larva/fry survival – the authors mix three different endpoints. It is not valid to compare different effect levels and endpoints, incorrectly equate them, and say (speculate) that the difference is due to tolerance. Moreover, the Belews number is generated from data for juvenile and adult fish, which automatically yields a two-fold higher value than for larvae and fry (see Lemly 2002, page 94, for a figure that explains this difference). Thus, the life stage of fish must be accounted for when evaluating teratogenic deformity data – yet another endpoint consideration that the authors failed to recognize. With such a mixing of effect levels and biological endpoints, it is not surprising that the chronic value is higher for field studies, but this does not indicate tolerance. The authors have presented an analysis that seems plausible on the surface, but it is not technically valid if one examines the data sets, endpoints, and associated effect levels carefully.

There are 4 major points that should be addressed in order to provide a valid interpretation of the data for the discussion on **pages 53-58**: (1) Don't compare tissue residues from fish with different levels of exposure (i.e., 30% or greater difference in environmental concentrations and/or dietary intake) and then try to infer that

differences in the resultant chronic values or EC_{20s} are due to tolerance – only compare same-level exposures; (2) Don't mix threshold values with high mortality impacts or EC_{20s} – these are not the same effect levels; (3) Don't mix the endpoints – keep fry survival, parental concentrations, and teratogenic effects separate; and (4) When evaluating teratogenic effects, don't mix life stages – keep larvae/fry separate from juveniles/adults.

The authors need to make the appropriate data comparisons and then modify the discussion. The suggestion of tolerance is simply not valid as they present it, and should be dropped altogether. However, on **page 58**, the decision that Bryson and Lemly not be used in the bluegill GMCV is valid. The authors exclude these studies because they (authors) infer tolerance developed from multiple-generations of exposure when, in fact, the reason is different levels of exposure across generations (i.e., declining selenium in the food chain over time coupled with recolonization and artificial stocking of fish; see Lemly 1997a for data and discussion; also Bryson 1985a, page 2-9, paragraph 2), not generational differences in sensitivity. The authors arrived at the right conclusion, but for the wrong reason. Moreover, the 19 µg/g dw "no effect" level that the criterion document gives (**page 56, last sentence**) for bluegill larvae in Bryson's study is not reliable because: (1) There were no replicates for the tissue analyzed, and thus "sample sizes were not sufficient for statistical comparison of concentrations" (page 2-8 in Bryson) – i.e., no geometric mean can be determined, and (2) The trend in selenium concentrations relative to percent effluent in the nonaffected area was reversed compared to that for both the control (Roxboro City Lake) and the affected area (Table 2.8, page 2-17, in Bryson), that is, as percent effluent increased so did selenium concentrations in the control and affected areas, but the relationship was opposite in the nonaffected area (from which the 19 µg/g value is taken). This apparent discrepancy may have been due to influences of mercury on selenium bioaccumulation and toxicity (Bryson, page 2-9, paragraph 3), but it remains an unanswered question that clouds the interpretation of data from the "unaffected" portion of Hyco Reservoir. It is incorrect for the authors to infer that Bryson's 19 µg/g dw value is indicative of tolerance – this statement (**last sentence on page 56**) needs to be removed from the criterion document, as well as the **last sentence on page 58**.

Steve Canton
Page 53, Paragraph 1
As I read through this discussion, it seemed to me that this study did not meet 3 of the 4 conditions outlined earlier on Page 47 as necessary before a study would be used!

Steve Canton
Page 53, Paragraph 2
I agree the Ogle and Knight fathead minnow data should not be used.

John Besser
Page 55 (Besser comment #11)
The interpretation of the studies conducted by Hermanutz et al (1996) is problematic. The endpoints and statistical analysis of these data described here differ from the Hermanutz (1996) report that was provided for review. [These changes were apparently reported by Tao et al (1999), which was not provided.]

I feel that the authors have re-interpreted some of the analyses of Tao et al inappropriately. For the 'field nest data', they state that Tao et al. reported statistically significant increases in larval hemorrhage from the 2.5 ppb treatment, relative to controls. These analyses were based on ANOVA with Dunnett's one-tailed mean comparisons, with significance of p=0.05 for untransformed data and p=0.022 for rank-transformed data. However, the authors reject these findings and state that critical p-values for the significance of these comparisons should have been reduced from p=0.05 to p=0.05/6 (because six mean comparisons were made), in order to keep the experiment-wise error rate to the desired 0.05. Based on this change, the authors conclude that the whole-body LOAEC for this study should be 26 ppm (10 ppb treatment) rather than 5.6 ppm (2.5 ppm treatment).

I believe that this 'correction' is in error and under-estimates the actual statistical significance of the data. Dunnett's test controls the experiment-wise error rate at a specified value (in this case, p=0.05). The test compares each treatment mean to the control mean and, and the least significant difference used for these comparisons is adjusted to account for the number of comparisons made (SAS/STAT, version 8.02; SAS Institute, Cary NC).

If the original statistical analysis by Tao et al is correct, the effects on hemorrhage in the 2.5 ppb treatment would be considered statistically significant and the appropriate whole-body LOAEC would be 5.55 ug/g (dwt).. This change could affect the calculation of the SMCV for bluegill, which would then be the geometric mean of 8.95 (Coyle et al), <7.9 (Lemly), and <5.55 (Hermanutz/Tao); or 7.30 ug/g. However, because the effect observed in the 2.5 ppb treatment (larval hemorrhage) was not associated with significant effects on other parameters, such as survival, it may not be considered an 'unacceptable effect' that would require adjustment of the SMCV or the FCV.

Steve Canton
Page 55, Paragraph 3
Technically, this study has only two treatments - i.e., again, it doesn't meet the criteria on Page 47. But, the data are probably useable - that's why the language on Page 47 needs to be toned down a bit.

Steve Canton
Page 56, Paragraph 1
This is very interesting - no effect levels for bluegills. But, the data aren't used. The authors suggest that these data indicate a combined dietary + water exposure is more important than diet alone. Yet, some of the rainbow studies used are diet only, and the authors note on Page 43 that diet alone studies would be used. Given the need for as much data as possible, there is no reason these data should not be included in the calculation of the SMCV and GMCV.

Steve Canton
Page 56, Paragraph 2
This study also does not meet the conditions outlined on Page 47. Plus, the author notes that the whole-body tissue levels are not different in the fish that survived and those that died. Given the lack of a wider concentration gradient so an effect level could be calculated, this 7.9 value should not be given any more weight than the other bluegill data (i.e., it is not strong enough data to pull out and use alone as the determinant of the final chronic value!).

Steve Canton
Page 57, Paragraph 2
I can see the reasoning for not using the field collected fish studies, although they do appear to indicate that there will be a "safety factor" with any lab-derived number since they appear to be less sensitive to chronic selenium effects (also noted in the Lemly 1993b study with "centrarchids" as discussed on Page 58). However, I do not see any reason not to use the additional data from the Monticello streams with the no-effect tissue levels.

Steve Canton
Page 58, Paragraph 3
The addition of "winter stress" to chose a final chronic value seems contrary to the use of the data in the prior discussions. These discussions and the calculations of chronic effects seemed to concentrate on the production of fit larval fish (i.e., survival, growth, or deformities in larvae). Larval fish are probably not present in winter, so keying in on the winter stress issue is not really necessary (or necessarily supported). As noted above, this study does not meet the authors' test conditions and the 7.9 may not actually be the chronic effect level since the surviving fish had the same tissue concentration.

Given the considerable discussion earlier in the document of following EPA guidance for development of criteria, resorting to picking the lowest number from the database seems odd. Choosing the lowest GMCV would have precedent in the "most sensitive genus" approach for recalculation of acute criteria after deletion of non-resident species leaves you under the 8-family rule. In this case, that would be 9.5. Also, given the analytical and sampling error inherent in measuring whole-body tissue levels, 7.9 is probably functionally equivalent to 9.5. (And, again, if those additional data from the Monticello streams are incorporated, this value would change to 11.6).

Dennis Lemly
Page 58, Paragraph 3
This paragraph states: "This appeared to reduce the tissue concentration associated with reduced survival". Lemly 1993a, Figure 1, shows an increase in selenium in the group that exhibited reduced survival (shown as cold + Se, Figure 9). This sentence needs to be reworded to something like "Cold water temperature increased the sensitivity of fish to selenium, but appeared to increase the tissue concentration associated with reduced survival (5-6 µg/g dw @ 20° C versus 7-8 µg/g dw @ 4° C)". I use the word "appeared" because in the discussion of Lemly 1993a, he attributes the difference in residues to the reduced lipid content of the affected group (lipid has low Se relative to other body constituents), which served to increase the concentration in remaining whole-body tissues. Thus, a change in body chemistry, rather than a change in total body burden of selenium, was likely responsible for the "apparent" increase in tissue concentrations.

Steve Canton
Pages 60-61
The comparison of the final chronic value to background levels in fish was discussed at length above. To reiterate, the potential for "unavoidable exceedences" will probably be higher in the western U.S. where marine shales are present and the streams under represented in the National Contaminant database.

Steve Canton
Pages 63-65, Table 4
For fathead minnows, as noted earlier, the Dobbs et al. study could produce a chronic value of 57 (geometric mean of LOAEC and NOAEC). This would change the SMCV and GMCV from 41.46 to 38.18. The follow-up study in the Monticello streams should be used, providing two additional chronic values for bluegill; >14.5 and >17.3. When combined with the other appropriate data for bluegills, the SMCV and GMCV would change to 11.65.

Steve Canton
Page 66, Paragraph 1
If the additions/corrections/modifications to the acute and chronic databases I suggest above are used, the values here would change slightly. The acute value would become 181 µg/L and the chronic level would be 11.6 µg/g dry wt in whole body fish tissue. One aspect missing from this (and the mercury criteria document, by the way) is any discussion of how the tissue level would be measured in the field. Would this be composite of all fish present? The highest level recorded at a site regardless of fish species? Geometric mean of replicates within and among species? Some discussion of how the data should be collected and interpreted

is needed. This goes beyond the other implementation problems of "backing up" the tissue numbers to effluent limits (and is a lot easier than figuring out how that will be done!).

Steve Canton
Appendix D, Page D-2, Paragraph 2
The tissue-based chronic number does help the problems of site-specific bioaccumulation potential greatly. However, the analysis seems to indicate that there might be differences based on species composition. For example, the final chronic value for minnows is 3 times higher than that for trout (or the "revised" number for bluegill).

John Besser
Appendix E, Pages E-13 through E-16 (Besser comment #12)
The authors adequately describe the on-site toxicity studies with conducted with the endangered razorback sucker by Hamilton et al. (2001a,b) and identify the principal limitation of these studies, i.e., the lack of consistent response of toxic effects to a gradient of dietary Se concentrations. However, there is no mention of two recent reports of toxicity studies with razorback suckers:

> Beyers, DW, Sodergren, C. 2001a. Evaluation of interspecific sensitivity to selenium exposure: Larval razorback sucker versus flannelmouth sucker. Larval Fish Laboratory. Department of Fishery and Wildlife Biology, Colorado State University, Fort Collins, Colorado.
>
> Beyers, DW, Sodergren, C. 2001b. Assessment of exposure of larval razorback sucker to selenium in natural waters and evaluation of laboratory-based predictions. Larval Fish Laboratory. Department of Fishery and Wildlife Biology, Colorado State University, Fort Collins, Colorado.

These studies describe both acute and chronic toxicity studies with endangered species of fish from the Colorado River system. Although these results may not meet the standards for inclusion in WQC calculations, these studies should be reviewed and discussed, either in Appendix E or as part of the derivation of the acute and/or chronic criteria.

John Besser
Appendix H, Page H-3 (Besser comment #13)
The value for Se in fathead minnow tissue for the 202 ppb treatment should be 55 ppm, rather than 75 ppm (based on Figure H-1).

Steve Canton
Appendix H, Page H-4
As noted earlier - why not use the geometric mean of the NOAEC (45 from table on Page H-3) and 75 or 73 (both with significant weight differences)? This would provide a chronic value from this study of 57 µg/g.

John Besser
Appendix H, Pages H-22 through H-23 (Besser comment #14)
The analysis of the study by Bryson et al (1984) uses a chronic value of <61 ug/g in adult muscle tissue, estimated using the muscle:whole-body regression equation, rather than <24.7 ug/g, based on the measured concentration in juvenile bluegills at the end of the study. Despite the authors' reluctance to use tissue data from juvenile fish, these results (from 60-d juveniles, by my estimation) are less uncertain than values estimated by regression. They are also similar to the chronic value of <28 ug/g in tissues of juvenile fish that were progeny of adult females from the same location (Gillespie and Baumann 1986).

John Besser
Appendix H, Page H-36 (Besser comment #15)
The discussion of the Effects Data for the Hermanutz et al (1996) study completely ignores the statistical analysis of the Nest Observation data by Tao et al (1999). As discussed above (#11), these analyses, which determined significant increases in larval hemorrhage, appear to be valid and should not be ignored.

Steve Canton
Appendix H, Page H-36, Paragraph 2
Use of NOAEC and LOAEC are strange here, considering there were only two treatments to begin with. Difficult to say where the NOAEC would really be. Although Studies II and III are discussed, only data from Study II is used for chronic value. As noted earlier, Study III meets the same requirements as Study II and those data should be used.

Steve Canton
Appendix H, Page H-40
The results of this general centrarchid study closely matches the other studies with field-derived test organisms. This would tend to suggest that lab-based chronic values will certainly be protective (if not overprotective) in the field.

VII. MISCELLANEOUS COMMENTS

Steve Canton
This completes my review of the document. Overall, I certainly enjoyed reading this and appreciate the amount of effort expended to produce this document. Sorry if my comments started to take on a "conversational" tone. It seemed easier and less stilted than trying to write in the third-person all through the review. Should you have any questions or need additional information regarding my review comments, feel free to contact me at the phone number or e-mail address at the head of this document.

Dennis Lemly
Since the new chronic criterion is tissue-based, the title of the document would be more accurate if the word tissue was added, i.e., Aquatic Life Water *and Tissue* Quality Criteria for Selenium. Similarly, on page 1, 2nd paragraph, 1st sentence ....water *and tissue* quality criteria....

Gregory Möller
Selenium in the Western Phosphate Resource Area - Background
I have described several studies underway related to selenium release in the Western Phosphate Resource Area. I add the following background to describe the context and importance of these studies. The Western Phosphate Resource Area is responsible for 4% of the world's phosphate ore production and currently accounts for 15% of the domestic US production. It is regarded as a strategic national resource as it is the only source of elemental phosphorous in the nation. A one-hundred year ore supply has been documented and this resource will become increasingly important in the near future as phosphate in Florida becomes less available. Currently 5 companies are engaged in active mining in the WPRA and the phosphate industry accounts for over 70% of the non-farm income in the 3 S.E. Idaho counties.

In late 1996, selenium leaching from phosphate mining sites was observed following a case of equine selenosis in a down-gradient pasture (Möller and Talcott 1997). Since that time numerous studies have been conducted in the area to examine sources, pathways, receptors and controls of selenium in this unique lotic ecosystem. Selenium has been found to reside in the middle waste shales of the phosphoria formation in concentrations of up to 200 mg/kg or larger. Unlike selenium in the well studied areas of Belews Lake (flyash, powerplant discharge) and Kesterson National Wildlife Refuge (agricultural sump and drainage water), mobilized contamination from mining leachate at the WPRA can be regarded as monotonic in selenium.[1] Phosphoria, the calcium phosphate mineral mined in the WPRA, is actively used as a binding agent for heavy metals in HM contaminated site remediation. Other constituents of potential concern (i.e. Zn, Cd) have been identified in environmental surveys, however, they are of minor occurrence, geographically isolated and limited in relative risk. Sediment release from active and reclaimed mine sites into the Blackfoot River watershed has been an active environmental management concern.

Unique about the WPRA are the relative isolation of Se as a contamination vector in the affected watershed and the decades-long history of phosphate mining (and presumptive Se release) in the area. These attributes make the WPRA an exceptional field laboratory for examining Se dynamics and impacts to a watershed. With the levels of release observed, phosphate mining in the WPRA is most certainly a target for the new criteria. Indeed a recent assessment identifies WPRA phosphate mining as the number one, human related factor in selenium environmental risk:

> *A selenium time bomb situation is developing in the United States and elsewhere that may result in substantial impacts on fish populations. The selenium time bomb has three components: (1) high food-chain bioaccumulation, (2) steep toxic response curve for fish, and (3) insidious mode of toxicity. If the threshold for selenium toxicity is exceeded, the time bomb explodes and a cascade*

[1] Se:S ratios in typical WPRA primary leachate are about 1:500.

> *of events is set into motion that will result in major ecosystem disruption. Several human-related factors are emerging that are capable of igniting the fuse of the time bomb by increasing waterborne concentrations of selenium and providing conditions favorable for bioaccumulation. Some of these factors are* ***(1) mobilization of selenium due to open-pit phosphate mining,*** *(2) use of constructed wetlands to treat selenium-laden wastewater from oil refineries and agricultural irrigation, (3) landfill disposal of seleniferous fly ash from coal-fired power plants, and (4) mobilization of selenium from animal feedlot wastes. Collectively, these threats may be sufficient to cause widespread, unanticipated toxic effects in fish populations. Only environmentally sound risk assessments followed by prudent management actions can defuse the selenium time bomb — once it explodes, it is too late to avoid significant impacts.*
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> ***Selenium Impacts on Fish: An lnsidious Time Bomb.*** A. D. Lemly
> Human and Ecological Risk Assessment: Vol. 5, No. 6, pp. 1139–1151 (l999)

The alarmist viewpoint expressed in the above paper has certainly not been borne out in 6 years of intensive, academic study of this mature site with substantial Se release and watershed deposition. Risk assessments based on water Se levels appear to overstate any negative biological effects, especially population level effects, in this lotic ecosystem. There have been no recorded observations of bird or fish population crashes in the area. Likewise there have been no observations, anecdotal or otherwise, of fish or bird terata in this actively fished and hunted area regarded for its trophy stock. Thus far in the examination of the area for biological impacts, two confirmed cases of livestock impacts have been observed. Direct selenium biological effects observations have been limited to one case of chronic equine selenosis (hoof wall dysplasia and alopecia) in confined animals with flood irrigated pasture and water from high concentration mine site runoff and one confirmed case of sheep deaths on seleniferous reclaimed mineland pasture following a late June snowfall. Livestock producers in this area, like most, supplement the mineral feed mix supplied to the animals with selenium, as is typical practice for animal health maintenance in the US.

The continuing large-scale wildlife effects field studies by University of Idaho researchers suggest the following:

- Lotic cold water ecosystems maintain selectively different selenium biodynamics that lentic warm water systems.

- Ecosystems with monotonic Se impacts are superior for isolating Se biodynamics in field studies than those with multiple stressor exposures such as agricultural pesticides in irrigation drainage.

- Current Se hazard assessment approaches and attitudes (vide supra) would have predicted wide-scale ecosystem collapse for the WPRA long ago. The lack of wildlife field observations of population level or organism level effects suggest the moderation of effects in this ecosystem may be due to: 1) site specific species, 2) lack of additional chemical or physical stressors found in more challenged environments such as power plant cooling ponds and agricultural drainage sumps, 3) lentic and lotic system site specific differences, 4) the marginal Se status of the surrounding areas, or 5) weakness in the current approach to Se hazard assessment.

- Migratory populations of birds may experience a beneficial reproductive success effect from Se exposures at sites with significant selenium release.

- Birth defect rates for sampled cutthroat trout (1999, 2000) from the Se impacted Blackfoot River watershed are typical of background reproductive success statistics.

- For the Henry's Lake fish, no effects on fish growth, feed intake, or survival were found when fish were fed levels of dietary selenium, supplied as selenomethionine, as high as 10 mg Se/kg diet throughout the entire life cycle of the fish.

- Fish selenium depuration rates were highest for the highest dietary exposure groups and the residual whole body levels were lowest (Hardy, Figure 5).

- Reproductive success of the 2.5 year diet study fish is still in analysis. Preliminary visual analysis of the Henry's Lake treatment group data shows high variability of possible effects compared to controls, but this casual analysis does not reveal a dose-response relationship or a discernable pattern. Analysis of this recent data set (Hardy, Table 10) and the development of the Blackfoot River fish data set are incomplete at this time.

- Primary WPRA Se risk is at or in proximity to 1st order source release zones.

The results of these studies in the WPRA represent significant new knowledge in the management of environmental selenium.

Challenges in Criteria Revision

I am aware of the challenges that EPA has in their work towards a revision of the aquatic biota criteria. I have been a quiet witness to the uncomfortable level of subjective passion that has characterized the scientific debate on Se ecosystem effects. As a practicing scientist working in related areas I am embarrassed by it all. I encourage passion for scientific discovery in my students and I drill them in disciplined objectivity about outcomes.

> *Science is first of all a set of attitudes. It is a disposition to deal with the facts rather than what someone has said about them...Science is a willingness to accept facts even when they are opposed to wishes... the opposite of wishful thinking is intellectual honesty. Scientists have simply found that being honest - with oneself as well as others - is essential to progress. Experiments do not always come out as one expects, but the facts must stand and the expectations fall. The subject matter, not the scientist knows best.*
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> - Skinner 1953, Science and Human Behavior.

Working though this passion is not easy for EPA in the preparation of the criteria. The draft document is respectful and science based. I teach my students: "regulatory science is science on a deadline" and that decisions in the regulatory arena need to be made in a timely fashion. I am also aware that several years of large-scale Se focused ecosystem research in the WPRA is near completion. In fact since studies are active, it is a large resource of information to address current data needs. This year the University of Idaho has received a $900K grant from EPA to continue its work exploring the sources, pathways, receptors and control of selenium in the WPRA, in addition to the $2M of exploratory research thus far. This new knowledge will allow version 2.0 of the selenium criteria to be scientifically defendable and inclusive of a wider range of research observations.

Recommendations

1. Insert a sulfate adjustment for the acute freshwater criterion: *"For waters with >90% selenate as a fraction of total selenium, an adjusted selenium concentration of 185 + 0.19 [SO_4^{2-}] µg/L is protective of freshwater aquatic life."*
2. Incorporate the Hardy data into the SMCV and GMCV calculations.
3. Recalculate the whole body-tissue selenium conversion using 3 parameter log normal regression. Use this data to recalculate SMCV and GMCV values.
4. Do not use a cold stress modifier to the FCV.

5. Strive to have an FCV that is not over- or under- protective of cold or warm water species. In the current analysis 9.5 mg/kg dw whole body Se approaches that goal.
6. Use a population level reference to broaden site specific application: *"The potential for reproductive failure in selenium exposed organisms makes population level protection important."*

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Regulatory and Technical Implementation Considerations in Determining Whether a Whole Body Fish Se Level Attains the Fish Tissue Criterion

A. *Sampling design*

1. Locations to be sampled
   a. ambient (background)
   b. fully-mixed discharge/receiving stream
   c. far-field (no wastewater influence) location?

2. Target species
   a. is bluegill sunfish (or other sunfish) the default target species?
   b. appropriate alternate species (in order of preference)

3. Temporal considerations
   a. what season to sample (coincide with critical low-flow conditions?)
   b. if fish are collected in different seasons, can sample data be pooled?
   c. what is minimum sample size for a seasonal sample?

4. Fish selection
   a. separate analyses for males and females?
   b. should level of sexual maturity be consistent among fish from different sites? Or, should this simply be noted prior to sample preparation?
   c. are length and weight measurements necessary? Should fish age be determined?

B. *Analytical considerations*

1. Composite versus individual fish
   a. what is minimum analysis required – a single composite sample?
   b. in what cases is data on Se in individual fish important?

2. QA/QC
   a. what specific QA/QC procedures should be followed (can existing EPA documents be referenced?) Is analysis of a Certified Reference Material required?
   b. should states require that lab QA/QC data be submitted?

C. *Compliance with the criterion*

1. How is compliance determined?
   a. is the criterion a not-to-exceed concentration or an average?
   b. what is the averaging period?
   c. when the criterion is not attained, should confirmatory analyses be conducted to determine what sources are causing the exceedance?

2. Background (ambient) samples
   a. should these be required when assessing a site influenced by wastewater discharges?

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VIII. RECOMMENDED REFERENCES

Steve Canton

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Assessment of exposure of larval razorback sucker to selenium in natural waters.
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APPENDIX A

JOHN BESSER

APPENDIX B

STEVE CANTON

APPENDIX C

DENNIS LEMLY

APPENDIX D

GREGORY MÖLLER

APPENDIX E

ROB REASH