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April 25, 2011

Via Electronic Submittal
Jared Blumenfeld, Regional Administrator
U.S. Environmental Protection Agency Region 9
75 Hawthorne Street, WTR-3
San Francisco, California 94105

ATTN: DOCKET NUMBER EPA-R09-OW-2010-0976—Advanced Notice of Proposed Rulemaking: *Water Quality Challenges in the San Francisco Bay/Sacramento-San Joaquin Delta*

Dear Administrator Blumenfeld:

The undersigned clean water associations appreciate the opportunity to comment on the United States Environmental Protection Agency's (EPA) Advanced Notice of Proposed Rulemaking (ANPR) regarding Water Quality Challenges in the San Francisco Bay/Sacramento-San Joaquin Delta. Our associations represent municipal wastewater agencies throughout the state that provide wastewater collection, treatment, biosolids, and recycled water services to millions of Californians. Our members are partners with EPA and the State in protecting the beneficial uses of our waters, including the Bay Delta.

We appreciate EPA's interest in identifying gaps that exist in our current knowledge and understanding of Bay Delta water quality and the health of the fisheries. As EPA is well aware, there are a myriad of state, federal, regional, and local agencies already focused on Delta issues, including the newly formed Delta Stewardship Council. We believe the existing regulatory framework, led by the State Water Resources Control Board (State Water Board) and the regional water boards, is well suited to addressing water quality concerns. This is in part due to the fact that water quality is not the principal factor in the decline of the Delta fishery (see, e.g., Development of Flow Criteria for the Sacramento-San Joaquin Delta Ecosystem, Final Report, August 3, 2010). The State Water Board possesses the authority to manage both water quality

and water quantity, and to consider both flows and other stressors in its Delta programs. In addition, to the extent that pollutant discharges might be demonstrated to be a contributing factor to the fishery decline, the State has authority under California law to regulate all discharges, including nonpoint sources of pollution.

EPA is uniquely suited to add value to the Bay Delta proceedings through its authority over pesticides and the development of information and accurate analytical methods for measurement of contaminants of emerging concern (CECs). Rather than undertaking new regulatory initiatives aimed at water quality criteria for specific contaminants, we encourage EPA to assist the State and local agencies to address emerging issues through improved science, public awareness and cooperative problem solving.

Below are our responses to several of the issues raised in the Federal Register Notice, focusing on pesticides, selenium, ammonia and CECs.[1] EPA should endeavor to distinguish between hypothetical or alleged water quality effects and well supported conclusions linking water quality to significant effects on aquatic life uses. This is a challenge in the Delta, where scientific research often fails to close the loop on scientific understanding and overstatement of that understanding is common.

Contaminants

1. What methods can be used in developing and implementing Total Maximum Daily Loads (TMDLs) to effectively address or incorporate interactive effects between multiple contaminants and other physical, chemical, and biological stressors on individual water bodies or for water bodies within a watershed?

Interactive effects among multiple contaminants are often seen only when the concentrations tested exceed environmentally relevant concentrations. More and more studies are being conducted to assess the effects of mixtures of chemicals. However, many studies that have been conducted to date do not test mixtures at concentrations that co-occur in ambient waters. Ambient concentrations are typically well below the 'no effects' threshold found in such experiments. Studies cited in the *Technical Support Document for Water Quality-based Toxics Control* (TSD) (EPA, 1991) found that synergism is a rare occurrence in combined effluent toxicity studies. It was found:

> . . . in the few studies on the growth of fish, the joint effect of toxicants has been consistently less than additive which suggests that as concentrations of toxicants are reduced towards the levels of no effect, their potential for addition is also reduced. There appear to be no marked and consistent differences between the responses of species to mixtures of toxicants. (TSD at p. 24.)

[1] To facilitate review, specific questions are identified using the numbers published in the Federal Register notice.

Using ambient water as the diluent in effluent toxicity testing allows for identification of synergistic, additive, or antagonistic characteristics when the effluent mixes with the receiving water, and can provide a better description of the toxic effects of the discharge in the receiving water. EPA recommends using ambient water for chronic toxicity tests if the objective is to determine additive or mitigating effects of the effluent to already contaminated receiving water as well as estimate the chronic toxicity of the effluent in uncontaminated receiving water.

Chronic toxicity tests were conducted by Buhl (1998) by mixing 11 inorganics (As3+, Cd, Cr6+, Cu, CN, Pb, Hg, Ni, Se4+, Ag, Zn) at a ratio of their proposed Criteria Continuous Concentration (CCC) in an 8-day static-renewal test. The findings were that proposed site-specific CCC for 11 inorganics are not protective of *Ceriodaphnia dubia* if they occur simultaneously in water. Acute tests were performed on fathead minnow in a 96-hour static-renewal test. The results showed that the proposed site-specific Criteria Maximum Concentration (CMC) for a mixture of these inorganics is protective of fathead minnows. The results of this study suggest that, for more than additive toxicity to occur, there must be a coinciding mixture of many chemicals, all at their water quality criterion concentrations, to exhibit such a toxic effect.

Direct toxicity to Delta species has been evaluated as a potential cause of the Pelagic Organism Decline (POD) for the past several years, although no direct causal relationships have been shown. Werner reported possible synergistic or additive toxicity with ammonia and municipal wastewater effluent. However, the conditions supporting this possibility were only met at concentrations exceeding those that are environmentally relevant in the Sacramento River and Delta. Repeat testing by Werner in 2008 and 2010 over four other test periods did not show any toxicity to delta smelt at effluent concentrations that far exceeded the levels that occur in ambient waters. Furthermore, there was no toxicity to delta smelt, Hyalella, or fathead minnows in streamside “in situ” monitoring at Hood and Rough and Ready Island (in the lower Sacramento River confluence with the San Joaquin River) in 2009. Delta smelt survival at Hood was as good as or better than control survival over 7-day exposures on five test events.

Ammonia

1. What, if any, information is available on the sources or impacts of total ammonia nitrogen in the Bay Delta Estuary that is not reflected or cited above?

Some critical information related to toxicity and food web impacts regarding total ammonia nitrogen impacts in the Bay Delta Estuary was omitted from the ANPR. We have summarized some of the information here, and request that EPA consider this as part of its review of the existing information regarding ammonia nitrogen.

Toxicity

The ANPR cites a finding from an oral presentation (Teh et al. 2009),[2] that ten percent mortality occurred to both *E. affinis* and *P. forbesi* at ambient concentrations present in the river below the Sacramento Regional Wastewater Treatment Plant (SRWTP), to suggest that there is a potential for acute ammonia toxicity for Delta copepods. This interpretation is contrary to the Central Valley Regional Water Quality Control Board (Central Valley Water Board) staff interpretations of these same results. In reviewing these test results, Dr. Chris Foe of the Central Valley Water Board staff noted that the test pH associated with toxicity in Dr. Teh's experiments (i.e., 7.2) was not representative of ambient pH levels in the Sacramento River (Foe 2009).[3] In his summary, Dr. Foe states that:

> *Ten percent mortality occurred to both species at ambient ammonia concentrations present in the river below the SRWTP. However, toxicity was only observed at a lower pH (7.2) than commonly occurs in the River (7.4 to 7.8). Toxicity was not observed when toxicity testing was done at higher pH levels."*
> (Foe 2009, p. 2, emphasis added.)

When acute effects thresholds for environmentally representative pH values are compared to ambient ammonia concentrations in the Delta, there is no evidence of acute toxicity to sensitive Delta species.

Food Web

The state of knowledge regarding algal preferences for ammonium versus nitrate is incorrectly characterized in the ANPR. Other environmental factors which control phytoplankton biomass in the Delta greatly constrain the potential effect of ammonium inhibition on overall productivity. Ammonia concentrations above the postulated inhibition threshold of 4 µM have been shown to stimulate growth of *N-Limited Phytoplankton* as they enter the Delta in the Sacramento River. Evidence from studies conducted in the Delta contradicts the hypothesis that ammonia (or nutrient ratios involving ammonia) promote blooms of microcystis (blue-green algae). Information from the Delta and other estuaries indicates that non-nutrient factors are credible alternative explanations for the observed shift in phytoplankton species composition in the Delta. Experimental data from the Delta contradicts the simplistic assumption that the pelagic food web in the Delta is dependent on diatom biomass for the following reasons:

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[2] Teh, S., S. Lesmeister, I. Flores, M. Kawaguchi, and C. Teh. 2009. *Acute Toxicity of Ammonia, Copper, and Pesticides to Eurytemora affinis and Pseudodiaptomus forbesi.* Central Valley Regional Water Quality Control Board Ammonia Summit, Sacramento, California, August 18-19, 2009.

[3] Foe, C. 2009. *August 2009 Ammonia Summit Summary*. Technical Memo to Jerry Bruns and Sue McConnell, Central Valley Regional Water Quality Control Board, September 24, 2009.

- Diatoms can be toxic to copepods.
- Delta copepods prefer non-diatom prey.
- The reproductive implications of food *choices* are virtually unstudied for the copepods of the San Francisco Estuary.
- Many non-diatom classes of phytoplankton are highly nutritious.
- The interpretation of a specific chlorophyll-a level as an indicator of nutritional sufficiency for Delta copepods is unjustified

None of the publicly available research from the Delta includes direct evidence that nutrient ratios (NH4:NO3, N:P, etc.) influence the taxonomic composition of phytoplankton in the Delta. There is no scientific evidence or consensus that N:P ratios are currently out of alignment in the Delta, or that lowering the N:P ratio would be beneficial for the Delta**.**

There is no evidence that nitrogen and phosphorus are out of "stoichiometric" balance in the Delta. Deviations in atomic TN:TP ratios in water samples from the classic "Redfield Ratio" of 16:1 (named for the oceanographer who determined in 1934 that the mean atomic N:P ratio of marine phytoplankton is 16:1 when neither nutrient limits growth) are often used as a rough indicator of relative N- or P- limitation of phytoplankton growth. Modern surveys indicate that TN:TP <18-22 may indicate N limitation in freshwater and ocean settings; phosphorus limitation is generally not expected unless TN:TP ratios exceed 50:1 (Guilford & Hecky 2000).[4] Boynton et al. (2008)[5] show that TN:TP ratios for 34 coastal, estuarine, and lagoon ecosystems trend somewhat above 16:1. Monthly samples for three IEP Suisun Bay monitoring stations for 2002-2007 provides a mean atomic TN:TP ratio of about 17:1 (16.7:1; Engle *unpublished* data[6]). This ratio is very close to the classic "Redfield Ratio." Lower ratios would be considered by many investigators as potential indicators of relative nitrogen deficiency in the water column. Significant concern exists regarding the low productivity of the Delta (Baxter et al. 2007),[7] and currently only a small fraction of in-Delta freshwater phytoplankton production escapes loss processes such as burial, in-Delta grazing, direct export in water diversions, to be transported into the brackish Delta (confluence zone and Suisun Bay) where the early life stages of POD

[4] Guildford, S.J., and R.E. Hecky. 2000. Total nitrogen, total phosphorus, and nutrient limitation in lakes and oceans: Is there a common relationship? Limnology and Oceanography 45:1213-1223.

[5] Boynton, W.R., J.D. Hagy, J.C. Cornwell, W.M. Kemp, S.M. Greene, M.S. Owens, J.E. Baker, and R.K. Larsen. 2008. Nutrient budgets and management actions in the Patuxent River Estuary, Maryland. Estuaries and Coasts. DOI 10.1007/s12237-008-9052-9.

[6] Data available upon request and attached to the Sacramento Regional County Sanitation District's April 25, 2011 Comment Letter.

[7] Baxter, R., R. Breuer, L. Brown, M. Chotkowski, F. Feyrer, M. Gingras, B. Herbold, A. Müller-Solger, M. Nobriga, T. Sommer, and K. Souza. 2008. Pelagic organism decline progress report: 2007 Synthesis of results. Interagency Ecological Program for the San Francisco Estuary.

fishes rear (Jassby et al. 2002).[8] Because there is experimental evidence from Parker et al. (2010) that Sacramento River phytoplankton entering the Delta upstream from the SRWTP are nitrogen-limited, it is reasonable to predict that reductions in inorganic nitrogen might lower primary productivity in the Sacramento River.

The relationships between cellular indicators of nitrogen or phosphorus deficiency, inorganic nutrient concentrations, phytoplankton taxonomy and stoichiometry, and TN:TP ratios have not been studied in the San Francisco Estuary (SFE). In other words, *bona fide* research which would be required to determine whether current N:P ratios encourage or discourage the growth of particular phytoplankton taxa, or are in any way detrimental to the food web, have not been conducted in the Delta or the rest of the San Francisco Estuary. Central Valley Water Board staff acknowledged in 2010 that no science supports a "target" N:P ratio for the Delta:

> *At this time there is no science to support what [N:P] ratio would be appropriate for the Sacramento River and the Sacramento-San Joaquin Delta*.[9]

There is also no scientific consensus that low N:P ratios favor diatoms over other phytoplankton groups. In fact, low N:P ratios (below the Redfield Ratio) are associated with a shift from diatoms to dinoflagellates in several estuaries[10]—a relationship which is opposite from that proposed for the Delta by some investigators.

Potential negative ramifications of lowering N:P should be considered. For example, the competitive advantage of nuisance species of N-fixing cyanobacteria (e.g., *Aphanizomenon* and *Anabaena*) can increase in estuaries when N:P ratios are reduced if overall nutrient supplies are decreased and if seed populations are present (Piehler et al. 2002);[11] both taxa are present in the upper SFE.[12] Low N:P ratios can also induce blooms of the toxic alga *Microcystis* from resting

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[8] Jassby, A.D., J.E. Cloern, B.E. Cole. 2002 Annual primary production: patterns and mechanisms of change in a nutrient-rich tidal estuary. Limnol Oceanogr 47:698-712.

[9] Staff Response to Comments, regional Water Quality Control Board, Central Valley Region Board Meeting, 9 December 2010 Response to Written Comments for Sacramento Regional County Sanitation District Sacramento Regional Wastewater Treatment Plant Tentative Waste Discharge Requirements at p. 31, emphasis added.

[10] Hodgkiss, I.J., and K.C. Ho. 1997. Are changes in N:P ratios in coastal waters the key to increased red tide blooms? Hydrobiologia 352:141-147.

[11] Piehler, M.F., J. Dyble, P.H. Moisander, J.L. Pinckney, and H.W. Paerl. 2002. Effects of modified nutrient concentrations and ratios of the structure and function of the native phytoplankton community in the Neuse River Estuary, North Carolina, USA. Aquatic Ecology 36:371-385.

[12] Species belonging to the genera *Anabaena* an *Aphanizomenon* are on the list of species from IEP phytoplankton monitoring data in the upper SFE.

stages in sediment (Stahl-Delbanco et al. 2003);[13] and, N:P ratios below the Redfield Ratio (i.e., <16:1) increase the risk of toxic red-tides in estuaries (Hodgkiss & Ho 1997).[14]

Selenium

San Francisco Bay, including the North Bay, has been listed on the State's Clean Water Act section 303(d) list since 1998 because of health advisories pertaining to the consumption of diving ducks. (*California State Water Resources Control Board, 2006 Clean Water Act Section 303(d) List of Water Quality Limited Segments Requiring TMDLs*). In more recent years, concern has emerged that bioaccumulation of selenium may also be impacting aquatic life beneficial uses by contributing to the decline of populations of certain key fish species.[15] While potential impairment to aquatic life beneficial uses (including RARE) because of selenium has not yet been used as a listing rationale, the San Francisco Bay Regional Water Quality Control Board's Preliminary Project Report for a Total Maximum Daily Load (TMDL) for San Francisco Bay proposed a fish tissue target intended to protect a broad array of beneficial uses.[16]

The ANPR poses two questions relevant to our member agencies. First, is the question of whether additional information is available to better characterize selenium sources, loadings and impacts within the watershed of the Bay Delta Estuary. (*ANPR at 35.*) In January of 2011 the San Francisco Bay Regional Water Quality Control Board (San Francisco Bay Regional Water Board) made available its *Preliminary Project Report* for a TMDL for San Francisco Bay (TMDL Report). This report relies on effluent data from 1998 through 2007 to estimate loading from publicly owned treatment works (POTWs) at approximately 226 kilograms (kg) per year. (*TMDL Report at 50.*) More recent data, however, indicate that POTW loading is substantially lower that estimated in the TMDL Report. This different value is likely because, prior to 2008 a number of POTWs used a method (ICP/MS "Collision Cell Mode") that was later found to be positively biased. While the number of samples collected in 2009 and 2010 is smaller, the standard deviation of those samples is less, suggesting that that these data are more representative of actual discharge concentrations. A comparison of the calculations for most of the dischargers listed in Table 11 of the TMDL Report to those using the more recent data set is attached and shows that actual loading is likely half of that estimated (Attachment A).

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[13] Stahl-Delbanco, A., L. Hansson, and M. Gyllstrom. 2003. Recruitment of resting stages may induce blooms of Microcystis at low N:P ratios. J. Plank. Res. 25:1099-1106.

[14] Hodgkiss and Ho (1997), *supra*, note 8.

[15] San Francisco Bay Regional Water Quality Control Board, *Total Maximum Daily Load Selenium in North San Francisco Bay, Preliminary Project Report* (January 2011), available at http://www.swrcb.ca.gov/rwqcb2//water_issues/programs/TMDLs/northsfbayselenium/SeTMDL_PreliminaryReport_01-11.pdf (hereinafter "Preliminary TMDL Report").

[16] Preliminary TMDL Report at 16.

In addition to gathering information to refine mass loading estimates, the Bay Area Clean Water Agencies has undertaken a special study to confirm the preliminary findings regarding the speciation of selenium in municipal wastewater and the percentage that is particle-associated, at a cost of around $50,000.00. Results from this study, which is being coordinated by the San Francisco Estuary Institute, are expected to be available in the fall of this year.

The second question about which the ANPR has requested information is whether there are additional selenium control methods or programs that should be considered for reducing selenium inputs and impacts. Regulatory efforts to reduce selenium, and therefore its impacts on beneficial uses, should focus on the San Joaquin River, which is largest source of selenium loading to the West Delta and North Bay. The annual estimate of dissolved selenium loads for this source ranges from 4,000 kilograms in wet years to 900 in dry years. (*TMDL Report at 62.*) In contrast, the report estimates that annual POTW loading is an order of magnitude lower. Additionally, selenium from POTWs exists predominantly in the form of highly soluble selenate and selenite, making it very difficult to treat.[17] Source control options for POTWs are similarly limited, in contrast to the options that exist for the San Joaquin River. To protect aquatic life beneficial uses from impairment by selenium, the regulatory focus should be on significant sources for which proven source control options are available.

The ANPR recognizes selenium as a potential significant contaminant impairing the biological resources of the Bay Delta. Specifically, the ANPR queries whether the prevailing selenium concentration in the system impairing the biological resources?

Careful review of science confirms the answer. The current selenium concentration in the Bay Delta system is not threatening biological resources. The attached report prepared by the Central Contra Costa Sanitary District contains an analysis of new science and a re-analysis of historical data, and indicates that the prevailing selenium concentration may be beneficial in terms of protection it provides from mercury bioaccumulation and its toxicity in fish and diving ducks.[18] This report confirms the statement in the ANPR that "[b]oth the San Joaquin River and North Bay selenium loads have declined in the last 15 years in response to, first, a control program in the San Joaquin grassland area, and second, NPDES permit requirement established for refineries in the late 1990s." This reduction in loads over the last 15 years has resulted in reduced selenium concentration in aquatic life and eliminated toxicity threat to humans and species of fish and birds at most risk to selenium exposure. In light of the new science, EPA should reconsider the status of selenium as a contaminant of concern for the Bay Delta.

[17] Preliminary TMDL Report at 41.

[18] Central Contra Costa Sanitary District, "*Is the Current Selenium Concentration in the San Francisco Bay-Delta Estuary Imparing the Biological Recources?"* (April 2011) (Attachment B).

Pesticides

Pesticides may be discharged to POTWs in conjunction with both indoor and outdoor pesticide applications. POTWs are not designed to treat pesticides. Pesticides can potentially interfere with treatment plant operation, ability to recycle reclaimed water and biosolids, and compliance with National Pollutant Discharge Elimination System (NPDES) permit effluent limits. When surface water bodies become impaired due to pesticides, POTWs discharging to the water bodies can be impacted through allocations established as part of TMDLs for those water bodies.

When a pesticide is used indoors, it can be discharged to a sewer, either because the use produces wastewater (e.g., human head lice shampoos and pet flea shampoos), or because an indirect pathway for sewer discharge exists (e.g., the treated surface is eventually cleaned with water or a pesticide-impregnated garment is laundered). Some outdoor uses of pesticides also lead to sewer discharges of the pesticides (e.g., filter backwash from swimming pools containing antimicrobial agents). Since POTWs are not designed to treat pesticides, treatment plant effluent and/or biosolids may contain the pesticide. Such pesticide releases may have the potential to cause aquatic toxicity and exceedances of NPDES permit effluent limits.

EPA has the authority to regulate pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). EPA's previous environmental risk assessments for pesticide registration and re-registration have not adequately evaluated and mitigated potential water quality impacts for pesticide discharges into sewers. Pesticide water quality impacts should be properly evaluated and mitigated through EPA's pesticide registration processes instead of regulating municipal dischargers under the Clean Water Act.

1. What, if any, actions should EPA take under its authority to improve the effectiveness of regulating pesticide contamination of the Bay Delta Estuary watershed?

POTWs need assistance from EPA and the Food and Drug Administration (FDA) to protect surface water and biosolids from pesticides. Under California law (similar to laws in most other states), POTWs cannot regulate the sale or use of pesticides. POTWs have limited practical ability to keep residents and small businesses from discharging ordinary consumer products, like pyrethroids, to their indoor drains. For these reasons, attempts to address pesticide discharges through Clean Water Act-based regulation of POTWs effluent and biosolids will not lead to water quality improvement but will unfairly burden local wastewater agencies. The practical and cost effective means of controlling pesticide discharge is for the federal government to use its authorities under FIFRA and Federal Food, Drug, and Cosmetic Act (FFDCA) to regulate pesticide sales and use.

FIFRA provides for federal regulation of pesticide distribution, sale, and use. EPA or FDA must register all pesticides distributed or sold in the United States. Under FIFRA, EPA has

a statutory responsibility to ensure that pesticides are safe and effective for their intended uses and to prevent unreasonable adverse effects to man, other animals, and the environment from their usage. (7 U.S.C. §§ 136(bb), 136a(a), 136a(d)(2), and 136d(b).) The risk benefit standard in FIFRA requires EPA to ensure that pesticides are used in such a manner that mitigation under the Clean Water Act is minimal or unnecessary. Properly implemented, EPA's registration processes can ensure that water quality standards are met and the Bay Delta Estuary aquatic habitat is protected. Additionally, the Endangered Species Act (ESA) of 1973 prohibits any action that can adversely affect an endangered or threatened species or its habitat. In compliance with this law, EPA is required to ensure that use of the pesticides it registers will not harm these species or habitat critical to endangered species' survival.

Although pediculicide [define] uses of pesticides, such as pyrethroid head lice treatments, are not currently subject to regulation under FIFRA, they were subject to such regulation until November 5, 1979, when EPA acted to exempt pediculicides from the requirements of the FIFRA. (44 Federal Register, 63749.) Since pediculicides are considered to be drugs, they are also subject to the FFDCA. The regulation of these products under both the FIFRA and the FFDCA was considered duplicative. In announcing the exemption:

> EPA and FDA concluded that the dual review of pesticide/new drug products offered solely for human use represents an expensive duplication of time and resources for both the Agencies and the sponsors of these products without any significant increase in benefits to public health and/or the environment. It is further concluded that regulations of these products solely by FDA under the FFDCA would adequately serve the intent of FIFRA.

Regulation under the FIFRA and the FFDCA is no longer duplicative. Since 1979, the degree of regulation under FIFRA has changed considerably, most notably with passage of the Food Quality and Protection Act of 1996 (FQPA). This statute requires EPA to review all pesticide registrations on at least a fifteen-year cycle. (7 U.S.C. § 136a(g)(1)(A).) The goal of this requirement is to ensure that all pesticides continue to meet up-to-date standards for safety, public health, and environmental protection. EPA has the authority to require data and take action if needed between registration cycles (7 U.S.C. §§ 136a(c)(2)(B), 136a-1(d)(3)). No similar provisions exist under the FFDCA. Additionally, EPA has emergency suspension authority, which means a pesticide registration can be canceled immediately if there is an emergency, imminent threat to public health or the environment. (7 U.S.C. § 136d(c).) This appears to be a much more direct and powerful tool to regulate pesticides when compared to the FDA's authority to simply require an Environmental Assessment in such circumstances. It is our position that EPA should reassert its control over pediculicides under FIFRA.

EPA should also update and revise data requirements for the registration and registration review of pesticides under FIFRA. The data requirements are intended to ensure that EPA has all the information necessary to evaluate the environmental and human health risks of pesticides.

Current rules do not allow EPA to obtain all the data needed to ensure that pesticides are registered in a manner protective of water quality.

Although aquatic life toxicity testing is required in the pesticide registration and registration review processes, data using the more pesticide-sensitive species and endpoints are generally lacking. For example, a review of registrant generated invertebrate sensitivity data will reveal the majority of their testing is being conducted using the less sensitive *Daphnia* genera as opposed to the more sensitive *Ceriodaphnia dubia*. It is impractical, if not impossible, to conduct laboratory toxicity testing on every relevant species. Therefore, the limited testing conducted as part of the pesticide registration and registration review processes should focus on the more sensitive species and exposure endpoints in order to be a useful surrogate representative of the diverse ecosystem.

Requiring, at a minimum, chronic toxicity species sensitive screening consisting of the fathead minnow (*Pimephales promelas*) seven-day survival and growth test, *Ceriodaphnia dubia* seven-day survival and reproduction test, and four-day green algae cell density test is not overly burdensome or financially costly. In fact, the majority of NPDES dischargers are required to conduct similar screenings annually. Chronic toxicity testing with these three species is conducted nationally and internationally. These methods have been fully evaluated and promulgated in 40 CFR Part 136, and are a required monitoring component of nearly all U.S. dischargers. The cost associated with such a screen ranges from $3,000.00 to $4,500.00. In addition to this minimum testing, toxicity testing with other species should also be considered on a case-by-case basis. For example, it has been well established in the literature that the amphipod Hyalella is particularly sensitive to pyrethroids.

EPA should also consider imposing more stringent conditions on issuing waivers for aquatic toxicity data for pesticide registration and registration review. For example, EPA should withhold registration decisions until required data is submitted and evaluated. By registering pesticides without required aquatic toxicity data, EPA cannot ensure that the pesticide does not pose an unreasonable adverse risk to the environment.

Failure to require such minimal aquatic toxicity testing has shifted the burden and financial responsibility of detecting environmentally harmful pesticide concentrations to NPDES permit holders and other dischargers. Through the “no toxics in toxic amounts” provision of the Clean Water Act, dischargers must demonstrate that effluents and receiving waters are not exhibiting toxicity using the previously mentioned species and endpoints. Having access to reliable acute and chronic toxicity results using these same methods, species, and procedures provided at the time of pesticide registration or registration review will allow dischargers to more effectively “rule in” or “rule out” currently used pesticides when chronic toxicity triggers and/or limits are exceeded.

EPA should also evaluate potential impacts from synergists and multiple active ingredient pesticide formulations during pesticide registration and registration review. Currently these impacts are not evaluated.

When potential water quality impacts are identified during registration or registration review for a pesticide, EPA should implement adequate risk management strategies. EPA should also require risk management strategies for all potential exceedances of water quality criteria (or equivalent values calculated for the purpose of the risk assessment) and all expected incidents of non-compliance by NPDES permit holders.

If risk management strategies include phase-out of the use of a pesticide, EPA needs to develop procedures to ensure the phase-out itself does not lead to adverse water quality impacts. These impacts can be caused by replacement of the phased-out pesticide with another pesticide causing water quality problems. Improper disposal of phased-out pesticides is also a serious concern. For example, during the phase-out of most urban uses of diazinon, a member agency experienced a toxicity incident downstream of a water reclamation facility that appeared to be caused by illegal disposal of a diazinon-based pesticide.

EPA has already taken important steps towards protecting water quality through its various registration processes; however, EPA can further integrate urban water quality protection more effectively into its pesticide review programs. Coordination between EPA's Offices of Pesticide Programs, Water, and Wastewater Management in reviewing pesticide data needs is essential to Clean Water Act implementation; it also provides an appropriate method of meeting FIFRA's goal of preventing unreasonable adverse impacts from pesticide use.

2. How can the process for establishing numeric water quality criteria be streamlined while maintaining technical integrity?

Over the years, various pesticides have been implicated and identified as the source of water quality impairments. With protective aquatic life water quality criteria established for only a few of these compounds, the majority of these pesticide impairments were identified through regulatory-mandated acute and chronic toxicity testing programs. The costs to POTWs associated with these impairments have exceeded millions of dollars. As detailed in the response to question 2 above, the water quality impacts of pesticides should be properly evaluated and mitigated during EPA's registration processes, thus preventing water quality impacts and making mitigation under the Clean Water Act minimal or unnecessary.

We are in favor of the continued use of EPA guidelines and methods for the development of aquatic life-based criteria. EPA has these well-established procedures in place to develop water quality criteria for aquatic life and drinking water standards. EPA should not circumvent these procedures in developing regulatory criteria for pesticides.

Contaminants of Emerging Concern

The ANPR includes a discussion of CECs, including pharmaceuticals, personal care products, solvent stabilizers, flame retardants, pesticides, and other commonly used commercial and industrial compounds. Our clean water associations encourage the EPA to approach the issue of CECs in a cooperative manner with the regulated community. Our members, along with national organizations like the Water Environment Research Foundation, have led a number of groundbreaking efforts regarding CECs, including monitoring, research on effects, and source control campaigns. As mentioned in the ANPR, the State Water Board has also been active in addressing CECs, including convening an expert panel to make recommendations on CEC monitoring in recycled water and an expert panel on CECs in the ocean, estuaries and wetlands. We strongly urge EPA to build on these efforts as it moves forward in addressing CECs.

Specific comments on several of the questions relating to CECs are provided below.

Question 5.a.: **What, if any, additional information is available regarding the effects of CECs on aquatic resources in the Bay Delta estuary?**

Many data gaps still exist regarding the effects of CECs on aquatic organisms. Researchers are attempting to address many of these gaps, including: linking measures of exposure with effects; linking effects observed in the laboratory with effects in the filed; linking effects at the cellular and organ level to effects in the whole organism; and linking effects in individual organisms to effects in populations. As EPA moves forward, it is essential that it that keep in mind that the presence of a compound does not necessarily mean that it is detrimental to aquatic organisms. The toxicological significance of trace amounts of CECs needs to be determined to establish a scientific basis for sensible monitoring and regulatory requirements.

Question 5.b: **What, if any, specific information exists to identify the sources and nature of discharges of CECs into the Bay Delta Estuary?**

We agree with the statement in the ANPR that CECs can be introduced into the aquatic environmental through a variety of sources, including not only municipal wastewater systems but also industrial wastewater systems, urban stormwater, animal husbandry operations, and agricultural runoff. To be effective, efforts to address CECs must consider all of these sources, not just POTWs. Our associations encourage on-going efforts by EPA to investigate pharmaceuticals at hospitals, medical clinics, doctors' offices, and long-term care facilities. We encourage EPA to broaden these efforts to include facilities dealing with animals, such as concentrated animal feeding operations and veterinary clinics.

Question 5.c: **What, if any, monitoring mechanisms or methodologies are available to assist in identifying CECs?**

To properly characterize sources of CECs, accurate analytical methods are essential. While EPA has developed two new analytical methods to measure low-level concentrations of pharmaceuticals (EPA Method 1694) and steroids/hormones (EPA Method 1698), more effort is needed from EPA in this area. EPA Method 1698 is particularly problematic, as it relies on uncommon and expensive instrumentation as well as complex extraction and cleanup procedures. Despite its cost and complexity, EPA Method 1698 has recoveries as low as 23% and as high as 275% for some constituents. We encourage EPA to improve CEC analytical techniques, with a focus on development of a better method for quantifying steroids and hormones. Without good analytical techniques, accurate characterization of sources and impacts of CECs is impossible.

Additionally, our associations believe that the most useful approaches to identify the sources, fate, transport and effects of CECs in the environment is through special studies, rather than by a traditional regulatory approach or via routine compliance monitoring programs typically used for conventional and priority pollutants. The state of the science is not yet sufficiently developed to set regulatory standards, and therefore it is premature to require routine monitoring for many, if not most, CECs. Instead, special studies designed to answer particular questions related to the sources, fate, transport and effects of various CECs (or classes of CECs) are part of the important foundational work necessary to determine which compounds are of greatest concern and how best to address them. Much work in this area is already being undertaken by academic experts and applied research institutions such as the Southern California Coastal Water Research Project (SCCWRP) or the San Francisco Estuary Institute (SFEI), and they are well-positioned to assist in this role.

Question 5.d: **What, if any, methods are most effective to minimize introduction of CECs into the Bay Delta Estuary?**

The area in which EPA can make the biggest difference in minimizing introduction of CECs into the environment is by providing leadership in source reduction for CECs. For instance, EPA should work with the Drug Enforcement Agency (DEA) in its efforts to amend controlled substance regulations to make it easier for state and local drug take-back programs to be implemented. EPA also should encourage product stewardship efforts related to pharmaceuticals and personal care products. EPA should work with the FDA as it reviews environmental impacts of new drugs to ensure that water quality impacts are adequately considered. EPA should continue to investigate potential reductions in pharmaceutical discharges from medical facilities. EPA should play a leadership role in engaging in discussions with manufacturers and others regarding the redesign of products that contain chemicals of concern that have a pathway to enter the water environment via the sewer (e.g. through Green Chemistry or other product reformulation or product stewardship efforts). These efforts are often beyond the scope and legal authority of local agencies providing wastewater services to

undertake, plus local agencies usually have a limited ability to engage manufacturers when the concern is focused only on a local area.

EPA has well-established procedures in place to develop water quality criteria for aquatic life and drinking water standards. EPA should not circumvent these procedures in developing regulatory criteria for CECs, but rather supplement as needed to account for effects such as endocrine disruption. In developing regulatory criteria, EPA should prioritize any constituents that are found present in the environment at concentrations known to cause detrimental human health and/or aquatic life impacts.

Thank you for the opportunity to submit the above information. We stand willing and able to work with USEPA staff in the synthesis of information obtained in response to the ANPR and in the formulation of a plan of action based on good science going forward.

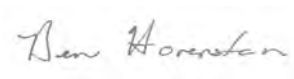
Sincerely,

Amy Chastain, BACWA

Roberta Larson, CASA

Debbie Webster, CVCWA

John Pastore, SCAP



Ben Horenstein, Tri-TAC

Attachment A

Selenium Annual Loading Comparison for North Bay POTWs

	Time Period	# of Samples	Mean	Stdev	Min	Max	Average Flow, MGD	Est. Loading (kg/yr)	Loading Change (kg/yr)
City of Benicia	1999-07	97	0.81	0.51	<0.3	5	3.0	3.5	
	2009-10	23	0.43	0.21	0.17	1.1	3.0	1.8	-1.7
Central Contra Costa Sanitation District	1998-07	99	0.34	0.50	<0.05	4	45.8	21.8	
	2009-10	24	0.38	0.12	<0.1	0.58	45.8	24.3	+2.5
Central Marin Sanitation Agency	1998-07	98	0.75	0.68	0.17	6.4	11.0	12.3	
	2009-10	6	0.36	0.24	0.13	0.76	11.0	5.4	-6.9
Delta Diablo Sanitation District	1999-06	100	4.21	7.54	<1	37	11.5	64.5 (34)	
	2009-10	40	0.67	0.11	0.41	0.86	11.5	10.6	-53.9
East Bay Municipal Utility District	1998-07	294	0.34	0.19	<0.2	1.6	74.6	34.8	
	2009-10	24	0.27	0.04	<0.3	0.44	74.6	27.5	-7.3
Fairfield-Suisun Sewer District	1998-03	95	0.75	0.38	0	2	17.0	19	
	2009-10	4	0.50	0.26	0.24	0.76	17.0	11.7	-7.3
Las Gallinas Valley SD Permit	2001-03	10	0.64	0.17	0.5	0.9	3.5	3.3	
	2009-10	21	0.34	0.22	<0.06	0.74	3.5	1.7	-1.6
Mt. View Sanitary District	1999-06	37	0.62	0.60	<0.02	5	2.0	2.3	
	2009-10	9	0.49	0.54	<0.1	1.64	2.0	1.4	-0.9
City of Petaluma	1999-07	60	0.65	0.23	0.35	1.4	7.6	6.9	
	2009-10	13	0.32	0.33	0.017	1.2	7.6	3.3	-3.6
City of Pinole and Hercules	2000-07	47	0.91	0.66	<0.1	4	3.2	4.0	
	2009-10	5	0.46	0.11	0.28	0.54	3.2	2.0	-2.0
Sausalito-Marin Sanitary District	1999-07	85	1.36	0.91	0.5	17.5	1.6	5.5	
	2009-10	23	0.47	0.30	0.23	1.2	1.6	1.0	-4.5
Vallejo Sanitation & Flood Control District	2000-07	79	0.84	0.52	<0.7	10.6	8.0	20.3	
	2009-10	23	0.47	0.09	0.32	0.65	8.0	5.2	-15.1
West County/City of Richmond	2000-07	60	1.73	0.97	0.25	9	14.1	33.7	
	2009-10	39	0.42	0.23	0.038	1	14.1	8.1	-25.6

* For values below MDL, 1/2 MDL was used in mean calculations.

** Loading for 2009 – 2010 was calculated using the same average flows as assumed in the TMDL.

*** This table contains data for only 13 of the 22 agencies listed in the TMDL, however, these agencies represent more than 80% of the flow and mass loading.

Attachment B

Is the Current Selenium Concentration in the San Francisco Bay – Delta Estuary Impairing the Biological Resources? A Reality Check

INTRODUCTION

The local refineries and the agricultural drainage input of selenium, along with the invasion of the North San Francisco Bay (NSFB) by the overbite clam (C*orbula amurensis*) in the late 1980s, resulted in elevated concentrations of selenium in diving ducks and white sturgeon (SWRCB, 1991; Carlton et al., 1990; Nichols et al., 1990). The selenium concentration in muscle tissue of the above species exceeded the California Department of Health Services (DOHS) toxicity thresholds for the protection of human health (SWRCB, 1991). Similarly, the liver selenium concentrations of diving ducks and white sturgeon were at potential health and reproductive impairment levels (SWRCB, 1991).

Major changes have occurred in the last 20 years. The local refineries have substantially reduced their input of selenium to the NSFB (Abu-Saba & Ogle, 2005; Presser & Luoma, 2006; Tetra Tech, 2008a). Similarly, the selenium load from agricultural drainage has been substantially curtailed (Nicole et al., 2008), and the overbite clam population has declined, especially in the San Pablo Bay (Poulton et al., 2004). Furthermore, the toxicity thresholds for the protection of human health and aquatic life have been developed, revised, and/or updated (Baginska, 2008; Lemly & Skorupa, 2007; Linville, 2006; OEHHA, 2008; Stanley et al., 1996; Tashjian et al., 2006;

Tetra Tech, 2008b). Additionally, substantial monitoring data has become available to accurately assess the current impact of selenium on human health and aquatic life (Takekawa et al., 2002, DeVink et al., 2008; Hoffman et al., 1998; SFEI Annual Reports, 1993-2008; Wainwright-De La Cruz et al., 2008).

This paper presents a current synthesis of knowledge related to the impact of selenium on the aquatic life in the NSFB and on human health.

Contrary to recent publications (Beckon, 2008; Luoma et al., 2008; RWQCB, 2009; RWQCB, 2011; SFEI, 2009), the available data suggests that the selenium concentration in the NSFB no longer poses a threat to aquatic life and to human health. Interestingly, data suggests that the current selenium concentration in the NSFB system instead might be beneficial in terms of (a) reduced bioaccumulation of methylmercury in fish and diving ducks, (b) counteracting mercury toxicity in these and other species, and (c) potential reduction in mercury exposure to the consumers of these species (Hoffman et al., 1998; Peterson et al., 2009; Stewart et al., 2004; Belzile et al., 2006; Ralston et al., 2007).

IMPACTS ON HUMAN HEALTH

A. Where We Were in the 1980s

The threat to human health was primarily due to the consumption of diving ducks and white sturgeon. The invasion of the overbite clam in NSFB exacerbated the bioaccumulation of selenium in these species.

The overbite clam invaded the estuary in 1986 (Carlton et al., 1990; Nichols et al., 1990; Linville et al., 2002). By June 1987 the overbite clam was a dominant species of bivalves and numbered up to 16,000 individuals per square meter in Suisun Bay and San Pablo Bay (Carlton et al., 1990; Nichols, 1990). This species turned out to be an efficient selenium concentrator in the NSFB because of its high feed rate and slow body loss rate of selenium (Lee et al., 2006; Linville et al., 2002; Presser & Luoma, 2006). Since the overbite clam was a principal food source for adult white sturgeon and diving ducks, it became a direct link to the elevated selenium concentration in white sturgeon and diving ducks. By 1989 the muscle tissue selenium concentration of diving ducks more than tripled, and it almost doubled in adult white sturgeon, compared to 1986 (SWRCB, 1991). These muscle tissue concentrations of diving ducks and adult white sturgeon exceeded the DOHS screening values for the protection of human health (see Figure 1). The DOHS issued health advisories for the consumption of diving ducks and white sturgeon in the San Francisco Bay (Fan & Lipsett, 1980). Later the California Regional Water Quality Control Board San Francisco Bay Region (RWQCB) placed the NSFB on the Section 303(d) List for selenium (RWQCB, 1998, 2000 & 2006).

B. Where We Are Today

In June 2008, the California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (OEHHA) updated the selenium reference dose (Rfd) to 5 µg per day from the previous 3 µg per day. Additionally, the OEHHA updated the selenium background dietary level to 114 µg per day from the previous 170 µg per day (OEHHA, 2008). These revisions by OEHHA are considered protective of human health.

Considering a diving duck tissue consumption rate of 16 g per day (used in the 1987-88 advisory) and a white sturgeon consumption rate of 32 g per day (currently recommended by the OEHHA), the new selenium muscle tissue advisory levels for diving ducks and fish are 14.8 and 7.4 µg/g wet weight, respectively.

Figures 2 and 3 present the muscle tissue concentration of selenium in diving ducks and white sturgeon, respectively, in the San Francisco Bay from 1986 through 2006 in relation to the OEHHA 2008 human health toxicity thresholds. Clearly, the current selenium concentration in these species no longer poses a threat to human health.

There appears to be three main reasons for the recent (post-1999) decline in the selenium concentration in diving ducks and white sturgeon compared to the pre-1996 period.

1. In 1996 the Grassland Bypass Project was implemented to reduce selenium discharge from the agricultural drainage in the San Joaquin River. This project reduced the selenium discharge by a factor of about two by 1999 compared to the discharge from 1986 through 1996 (Nicole et al., 2008).
2. In 1998 the local refineries implemented an effluent selenium reduction program. This program reduced the refineries selenium discharge by a factor of four by 2000 (Abu-Saba & Ogle, 2005; Presser & Luoma, 2006; Tetra Tech, 2008a).
3. The population of overbite clams in NSFB, especially in the San Pablo Bay, dropped substantially. The population dropped from an average of 5,000 per square meter in 1988-1989 to approximately 200 per square meter in 1999-2000 (Poulton et al., 2004).

Figure 4 depicts the relationship of the above described major changes to the muscle tissue selenium concentration in diving ducks and white sturgeon.

IMPACTS ON WILDLIFE RESOURCES

A. Impact of Selenium on the Health Conditions of Diving Ducks

Liver selenium concentrations of greater than 10 µg/g dry weight (dw) in egg-laying females are considered to cause reproductive impairment and greater than 33 µg/g dw are considered harmful to the health of birds (i.e. poor body condition, histopathological lesions, etc). The above thresholds are based upon studies in captive mallards (Custer & Custer, 2000; Custer et al., 2008; DeVink et al., 2008; Heinz, 1996).

Figure 5 presents the liver selenium concentration of diving ducks in the NSFB and the Canadian Boreal regions in relation to these toxicity thresholds. Clearly, in 1986-87 (SWRCB, 1991), the liver selenium concentration in NSFB diving ducks was substantially elevated—the mean concentration was up to a factor of three over the 33 µg/g toxicity thresholds. Interestingly however, in spite of these substantially elevated selenium concentrations, no histopathological abnormalities in the diving ducks in NSFB were found during this period (SWRCB, 1991).

Takekawa et al. (2002), conducted a study on the effect of selenium on the body conditions of the diving ducks in NSFB. The study was based upon specimens collected in 1986-87. The liver selenium concentration ranged from 34 to 140 µg/g dw, with a mean of 48 µg/g dw. Takekawa et al. (2002), concluded that "Despite this [elevated selenium in the liver] we found no relationship between

liver selenium concentration and any of the body condition indices we tested in scaup."

Hoffman et al. (1998), studied health conditions of diving ducks from the Suisun Bay compared to the diving ducks from the Tomales Bay (a coastal reference site known to be less contaminated with selenium compared to Suisun Bay). This study was based upon specimens collected in 1989. As expected, the study found that the liver selenium concentration in diving ducks (greater scaup and surf scoters) from Suisun Bay was five to six times higher than those of the same species from the Tomales Bay. The mean liver selenium concentration in greater scaup and surf scoters from Suisun Bay clearly exceeded the toxicity threshold of 33 µg/g dw by a factor of 2 and 3.6 respectively. Interestingly however, there were no significant differences in body, heart, and liver weights of diving ducks between these two locations.

Similarly, DeVink et al. (2008), published results of a detailed study on the effects of selenium on the body condition and breeding prosperity of diving ducks in the Canadian boreal region. The liver selenium concentration in this study ranged from 9 to 75 µg/g with a mean of 34 µg/g dw. They concluded that "Moreover, higher (selenium) concentrations in scoters do not appear detrimental to female (and male) body condition or breeding prosperity. Therefore, we conclude that selenium is likely not the cause of decline or lack of population recovery of scaup or scoters."

The most recent liver selenium concentration in diving ducks in NSFB ranges from 7 to 119 µg/g dw with a mean of 47 µg/g dw (Wainwright-De La Cruz et al., 2008). This concentration is approximately one half of that observed in 1987-1989 (Hoffman et al., 1998; SWRCB, 1991), which showed no significant negative effect on the body conditions or histopathological abnormalities in the diving ducks of NSFB (see Figure 5). Although the liver selenium concentration of the diving ducks in NSFB exceeds the mallard toxicity thresholds, there appears to be no negative impact of these elevated selenium concentrations on the body conditions of these species. This observation implies that the diving ducks in NSFB are probably more tolerant to selenium than captive mallards. As DeVink et al. (2008) noted, the toxicity threshold of 33 µg/g dw (for mallards) may not be applicable to diving ducks. Thus, the currently available data suggests that the prevailing selenium concentrations in the NSFB do not appear to pose a threat to the health of the diving ducks.

B. Impact of Selenium on Oxidative Stress Status of Diving Ducks

Elevated hepatic selenium concentrations are known to cause oxidative stress with altered glutathione metabolism in captive mallards (Heinz et al., 1989; Hoffman & Heinz, 1998; Hoffman et al., 1989, 1991 & 1992). The oxidative stress may have a direct or an indirect affect on the body conditions of diving ducks, especially during spring migration and, thus, ultimately affecting the

reproductive success of these species (Anteau et al., 2007). The oxidative stress status of the NSFB diving ducks has been a concern locally (Wainwright-De La Cruz et al., 2008).

Hoffman et al. (1998), studied the status of oxidative stress in the diving ducks from Suisun Bay compared to the diving ducks from the Tomales Bay *(a reference site)*. The hepatic selenium concentrations of greater scaup and surf scoters in the Suisun Bay were higher by a factor of five and six, respectively than those of the same species from the Tomales Bay. However, in spite of these highly elevated hepatic selenium concentrations, the diving ducks from the Suisun Bay appeared to be physiologically better equipped to handle oxidative stress compared to the diving ducks from the Tomales Bay. The reduced glutathione (GSH) and glucose-6-phosphate dehydrogenase (G-6-PHD) concentrations in greater scaup from the Suisun Bay were higher by a factor of approximately two and three, respectively, than those of the same species from the Tomales Bay. Similarly, the GSH and G-6-PHD concentrations in surf scoters from the Suisun Bay were higher by a factor of two and seven, respectively, than those of the same species from the Tomales Bay (Figure 6). The GSH and G-6-PDH are major combatants of oxidative stress in birds (Hoffman et al., 1998; Hoffman & Heinz, 1998). As previously mentioned, the most recent liver selenium concentration of diving ducks (surf scoters) in NSFB ranges from 7-119 µg/g dw with a mean of 47 µg/g dw (Wainwright-De-La Cruz et al., 2008). This concentration is substantially lower than observed earlier

(Hoffman et al., 1998). The earlier higher range was shown to have no negative impacts on the status of oxidative stress of diving ducks inhabiting the NSFB compared to those at the Tomales Bay. Therefore, the available data suggests that the prevailing selenium concentrations in the NSFB do not appear to cause undue oxidative stress in diving ducks.

C. Impact of Selenium on the Reproduction and Egg Hatchability in NSFB Birds

Figure 7 presents the egg selenium concentration of the diving ducks and wading birds in San Francisco Bay in relation to the selenium toxicity thresholds. As is evident from this data, the mean selenium concentration in eggs of the diving ducks and other wading birds in the San Francisco Bay are well below current teratogenesis/egg hatchability/duckling growth and survival thresholds.

Furthermore, the egg mean selenium concentration of the diving ducks in the San Francisco Bay complex is approximately 40 percent lower than the concentration in eggs of diving ducks in Canada, which has been shown to have no impact on the breeding prosperity of the diving ducks (DeVink et al., 2008).

Therefore, there appears to be no evidence suggesting that the prevailing selenium concentrations in the San Francisco Bay complex are causing harmful impacts on the diving and wading bird egg hatchability or reproductive success.

IMPACT OF SELENIUM ON SAN FRANCISCO BAY FISHERIES

The United States Environmental Protection Agency (US EPA, 2004) originally proposed a fish tissue criterion of 7.9 µg/g dw as a tissue selenium target for the protection of fishery resources. Lemly and Skorupa (2007) critiqued this proposed value and suggested that the target should be lowered to 5.8 µg/g dw mainly because of Winter Stress Syndrome concerns. The US EPA's (2004) proposed fish tissue numeric target was based upon whole body concentration of selenium in juvenile bluegill. Lemly (1993) discovered that this species was more sensitive to selenium exposure in winter than in summer. In response to the Lemly and Skorupa (2007) critique, the US EPA investigated the effect of Winter Stress Syndrome on bluegill. Recently, the US EPA (2008) issued the results of this study, which concluded that (a) the juvenile bluegill did not decrease in body condition factor and lipid content (Winter Stress Syndrome) as reported by Lemly (1993); (b) the toxicity of selenium to juvenile bluegill was approximately one half of that observed by Lemly (1993), i.e., the new toxicity threshold for bluegill is approximately 11.1 µg/g dw compared to 5.8 µg/g proposed by Lemly (1993); and most importantly (c) the US EPA (2008) study showed that under a similar temperature and exposure period, bluegill receiving a natural diet accumulated 2.5 times less selenium compared to an artificial diet spiked with seleno-L-methionine, the diet employed by Lemly (1993). The USEPA has not yet revised its proposed fish tissue numeric criterion of 7.9 µg/g dw.

Tetra Tech (2008b) performed a thorough review of several selenium fish toxicity studies and calculated the effect thresholds for each study and species. The species of most concern for the NSFB are the Sacramento spittail and the white sturgeon. The feeding behavior of these two species exposes them to substantial higher levels of selenium compared to other resident species of the NSFB. For example, the striped bass bioaccumulates selenium approximately 10 times less than the white sturgeon (SWRCB, 1991). The lowest effect threshold for the NSFB resident species was for the white sturgeon. Tetra Tech (2008b) calculated a toxicity threshold of 3.9, 4.0, and 6.2 µg/g for liver, muscle and whole body respectively for the white sturgeon.

Baginska (2008) and RWQCB (2011) proposed a fish tissue numeric target of 6.0 to 8.0 µg/g dw for the protection of fishery resources of the San Francisco Bay. The Baginksa (2008), RWQCB (2011), and the Tetra Tech (2008b) toxicity thresholds are based, in part, on the Linville (2006) white sturgeon study.

Linville (2006) exposed female white sturgeon in mid-viotellogenic stages of oogenesis to diets containing either 1.4 µg/g dw (control) or 34 µg/g dw selenium (treatment) for about six months. The test end points were reproductive performance (fecundity, fertilization success, and neurulation), weight and length of larvae, and larvae developmental abnormalities (ederma and skeleton deformities, such as lordosis, kiphosis, and scoliosis). Linville (2006) found that 34 µg/g dw dietary selenium exposure of adult female white sturgeon had no significant impact on reproductive performance and weight or length of larvae compared to the control. Parallel to the

maternal exposure experiments she also microinjected white sturgeon larvae with seleno-L-methionine. There were significant effects on larval deformities in both experiments. Linville (2006) concluded that "A hazard threshold of 3 (ED_5) to 8 (ED_{10}) µg/g in developing white sturgeon is suggested for this species."

Interestingly however, Linville's (2006) experimental data does not support her suggested thresholds. In microinjection experiments, the ED_5 and ED_{10} are approximately 8 and 9 µg/g dw, respectively, as calculated from the logit equation (see Figure 8). Similarly, in the maternal exposure experiments, the ED_5 and ED_{10} are 10 and 15 µg/g dw respectively, as calculated from the logit equation (see Figure 9). Furthermore, it appears that Linville (2006) suggested thresholds are based upon pooled microinjection and maternal exposure experiments. Although the larvae direct microinjection experiment may have academic utility, it is not applicable to the Bay's natural conditions because, (a) larvae in the Bay are exposed to selenium from the yolk sac in a natural complex form, generally less acutely toxic than seleno-L-methionine and (b) the larvae in the Bay are not instantly exposed to a toxic seleno-L-methionine concentration as it happens during micro-injection. Instead, the larvae in the Bay gradually obtain selenium from the yolk sac over a period of several days if not weeks, which allows larvae to develop the ability to produce selenium detoxification enzyme (superoxide dismutase) and thus detoxify selenium to some extent (Palace et al., 2004). Probably, because of micro-injection stress, the form and the route of selenium exposure there was 45 to 70 percent more mortality of larvae in micro-injection experiments compared to maternal exposure; and overall, the larval development

abnormalities were two to three times more in microinjection experiments compared to maternal dietary exposure experiments. Therefore, pooling the data for these two separate experiments is not appropriate.

Our review of Linville (2006) maternal dietary exposure data (see Table 1) shows that the Treatment T1 (larvae selenium concentration of 11.6 µg/g) is a no-observed adverse effect level (NOAEL) (zero abnormalities). Since Treatment T3 produced more larvae abnormalities (13 percent) compared to Treatment T1 (0 percent) at a substantially lower selenium concentration (7.75 vs. 11.6 µg/g), Treatment T3 cannot be considered the lowest observed adverse effect level (LOAEL). The LOAEL cannot be lower than the NOAEL; therefore in this case, Treatment T2 (larvae selenium concentration of 18.4 µg/g) becomes the LOAEL. This LOAEL is supported by her logit probability analysis that estimates an ED_{10} and ED_{20} of 15 and 20 µg/g respectively (see Figure 9). This LOAEL is further supported by the general rule in ecological risk assessment; i.e., it is not feasible to describe population impacts below 20 percent effect level (Suter et al., 2000; EPA, 1991). The NOAEL and LOAEL associated adult muscle tissue concentrations are 9.95 µg/g dw and 15.30 µg/g dw, respectively (Table 1, under the column titled, Larvae & Muscle). Therefore, the resulting white sturgeon larvae development toxicity threshold, in terms of adult muscle tissue concentration, is 12 µg/g dw (geometric mean of 9.95 and 15.3 µg/g dw).

Tashjian et al. (2006), conducted an extensive study on the effects of selenium on chronic toxicity in juvenile white sturgeon. The study end points were survival, growth,

behavioral effects, activity level, and liver, gill and muscle tissue histopathology. The results show that for all test end points, the selenium dietary exposure toxicity threshold (geometric mean of NOAEL and LOAEL) is 14.0 µg/g dw. This threshold is substantially higher than the previously considered 9 to 10 µg/g for this species (Luoma et al., 1992). The corresponding muscle tissue concentration threshold from Tashjian et al. (2006), is 20.3 µg/g dw.

Since the toxicity threshold for white sturgeon developmental abnormalities of 12 µg/g dw (calculated from Linville (2006) experimental data) is lower than Tashjian et al. (2006) for juvenile white sturgeon chronic toxicity (20.3 µg/g dw), it should be protective of white sturgeon for all the end points studied in Linville (2006) and Tashjian et al. (2006) combined.

Figure 10 compares the recent selenium muscle tissue concentration of adult white sturgeon with the calculated toxicity threshold (12 µg/g dw or 2.8 µg/g ww) from Linville (2006) data. Figure 11 compares the current selenium dietary exposure from the NSFB bivalves to the Tashjian et al. (2006), reported dietary exposure toxicity threshold. As is evident from Figures 10 and 11, the current selenium muscle tissue concentration of white sturgeon and its selenium dietary exposure are well below the respective toxicity thresholds. It should be noted here that the most recently NSFB modeled (predicted) white sturgeon muscle tissue concentration using a trophic transfer factor of 1.7 is 10.7 µg/g dw or 2.5 µg/g ww (Tetra Tech, 2010). This prediction is in line with the maximum concentration observed in the most recent (2006) sampling and analysis (see figure 10).

Beckon (2008) presented a paper at the 2008 CALFED Conference on the toxicity of selenium to salmonids. After review and re-analysis of Hamilton et. al. (1990), Beckon concludes, "Young salmon suffer 10 percent mortality due to selenium at a fish tissue concentration of about 1.8 µg/g (whole body dw)…These data suggest that selenium may have killed one quarter of the young Chinook salmon migrating down the San Joaquin River."

Our review of Hamilton et al. (1990) data (see Table 2) shows the following:

1. The fish tissue selenium concentrations up to 4 µg/g dw (SLD diet) and 5 µg/g Se-Met diet had no significant (α=0.05) impact on the salmon survival compared to the control (see Table 2).

2. The fresh water dietary exposure toxicity threshold (geometric mean of NOAEL and LOAEL) for Chinook salmon is in the range of 7 µg/g dw (SLD diet) and 13 µg/g dw (Se-Met diet) (see Table 2).

3. The brackish water dietary exposure toxicity threshold for growth (length and weight) for Chinook salmon is in the range of 7 µg/g dw (SLD diet) and 25.4 µg/g dw (Se-Met diet) (see Hamilton et. al., 1990 Table - 6).

Note that the SLD diet was found to have elevated concentrations of boron, chromium, and strontium compared to the control and Se-Met diets, which might have increased observed SLD diet toxicity compared to the control and Se-Met diets.

The Chinook salmon larvae/fingerling food mostly consists of insects and amphipods (zooplankton), etc. (Beckon & Maurer, 2008). The available data on the actual selenium concentration of zooplankton in the NSFB (Tetra Tech, 2008c) and the modeled concentrations (Schlekat et al., 2000) show that the dietary selenium exposure concentration for Chinook salmon fingerling in the NSFB are well below the selenium toxicity threshold calculated from the Hamilton et al. (1990), study. Therefore, Tashjian et al. (2006), and the re-analysis of Linville (2006) and Hamilton et al. (1990) data suggests that the current dietary exposure concentrations of selenium in the Bay do not appear to impair the Bay fishery resources.

WATER COLUMN TOXICITY

The selenium water quality criteria promulgated for San Francisco Bay upstream to and including Suisun Bay and Sacramento-San Joaquin Delta are 5.0 µg/L (4-day average) and 20 µg/L (1-hour average). However, Lemly and Skorupa (2007) critiqued these criteria and suggested that the criterion should be lowered to 2 µg/L.

The San Francisco Estuary Institute (SFEI) has been monitoring the water column selenium concentration in the San Francisco Bay. From 2002 to 2006, the highest concentration observed in the water column of the open Bay was 2 µg/L. The Bay-wide average concentration for 2007 was 0.10 µg/L, slightly lower than the long-term average of 0.12 µg/L (Figure 6 from the SFEI 2008c). Therefore, the prevailing selenium water column concentrations are well below the Lemly & Skorupa (2007) suggested 2 µg/L threshold.

SEDIMENT TOXICITY

No evidence currently exists linking sediment or sediment-pore water toxicity to selenium in the San Francisco Bay. Under the California Bay Protection and Toxic Cleanup Program (BPTCP), the SWRCB commissioned a study entitled "Sediment Quality and Biological Effects in San Francisco Bay" (SWRCB, 1998). This study observed sediment toxicity to amphipods and/or sediment pore water toxicity to sea urchin embryos at several segments of the Bay. In 2002 this observed toxicity resulted in the SWRCB designating specific segments of the Bay as impaired due to selenium concentrations in sediment and adding those sites to the Section 303(d) List.

However, the sediment samples that showed toxicity in the BPTCP study (SWRCB, 1998) had several other contaminants, such as copper, chromium, mercury, lead, nickel, polynuclear aromatic hydrocarbons (PAH), and polychlorinated biphenyl (PCB);

the concentrations of which exceeded the established toxicity thresholds. The limited toxicity identification studies of these BPTCP sediment samples confirmed toxicity due to copper, chromium, and mercury. None of these studies confirmed selenium as the source of the observed toxicity.

Abu-Saba and Ogle (2005), after a thorough review of the BPTCP data and the basis of Section 303(d) listing of these segments of the Bay, concluded, "Based upon the overwhelming weight of evidence presented…it is concluded that selenium is not impairing the BPTCP sites that were added to the Section 303(d) List in 2002 and delisting these sites for impairment by selenium is warranted."

Currently the United States Forest Service (USFS) and the United States Fish and Wildlife Service (USFWS) recommends 2 µg/g dw as a selenium sediment toxicity threshold (Lemly, 2008).

The Regional Monitoring Program (RMP) has extensively monitored sediment selenium concentrations in San Francisco Bay. Figure 12 presents the recent (2005-2006) selenium sediment concentration in the NSFB in relation to the USFS and USFWS's recommended sediment selenium concentration toxicity threshold. The data clearly show that the NSFB sediment selenium concentration is well below the USFS and USFWS sediment selenium concentration toxicity threshold of 2 µg/g dw. South Bay sediment selenium concentrations are also below the 2 µg/g dw toxicity threshold for the same period. In 2005 the mean selenium concentration (+/- SD) of South Bay

sediments was 0.56 +/- 0.36 µg/g dw (range was from 0.36 µg/g to 1.58 µg/g dw (range was from 0.10 µg/g to 0.15 µg/g dw Se). Therefore, the current sediment selenium concentration does not justify a cause for the finding of aquatic life impairment for all Bay segments.

PROJECTED SITE-SPECIFIC WATER QUALITY CRITERIA FOR SELENIUM FOR SAN FRANCISCO BAY USING PRESSER & LOUMA (2010) ECOSYSTEM-BASED MODEL:

The mean dissolved selenium concentration in the San Francisco Bay for 1993-2005 is reported to be approximately 0.17 ug/L (Tetra Tech, 2008a). The mean selenium content of the particulate matter in the San Francisco Bay for 1997-1999 is reported to be approximately 0.7 ug/g dw (Tetra Tech, 2008c). Therefore the San Francisco Bay site-specific K_d is approximately 4100, (0.7 x 1000 ÷ 0.17).

The Asiatic clam median selenium concentration is reported to be about 12 ug/g dw (Stewart et al., 2004). Using a conservative Asiatic clam food selenium content of 3 ug/g dw the Trophic Transfer Factor (TTF) for asiatic clam is 4, (12 ÷ 3).

The mean selenium concentration of White Sturgeon for 1997-2009 is reported as approximately 1.44 ug/g ww (SFEI 1997-2006 and SFEI 2008b) or 6.8 ug/g dw assuming a 79 percent moisture content, with an Asiatic clam median selenium concentration of 12 ug/g dw, the TTF for White Sturgeon is approximately 0.6 (6.8 ÷ 12).

By plugging the above San Francisco Bay site-specific K_d and /TTFs and Linville's data supported White Sturgeon toxicity target of 15 ug/g dw into Presser & Louma model, the Site-Specific Water Quality Criteria for Selenium for San Francisco Bay is approximately 1.5 ug/L (15 x 1000) ÷ (0.6 x 4 x 4100).

Interestingly, the projected Site-Specific Water Quality Criteria for Selenium for the San Francisco Bay of approximately 1.5 ug/L compares well with the soon to be released USEPA's draft National Water Quality Criteria of 1.3 ug/L for lentic water and 2.6 ug/L for lotic water.

The current (SFEI 2010) dissolved selenium concentration of San Francisco Bay (0.16 ug/L) is substantially lower than the projected criteria of 1.5 ug/L. Thus the White Sturgeon population in the San Francisco Bay does not appear to be threatened by selenium. The RMP (SFEI 1997-2008) supports this conclusion.

POTENTIAL BENEFITS OF PREVAILING SELENIUM CONCENTRATIONS FOR THE PROTECTION OF AQUATIC LIFE IN NSFB

Increased selenium concentrations in the aquatic environment are known to reduce methylmercury bioaccumulation and its toxicity in aquatic and terrestrial organisms (Belzile et al., 2006; Chen et al., 2001; Cuvin-Aralar & Furness, 1991; El-Demerdash,

2001; Frisk et al., 2003; Hansen, 1988; Hoffman & Heinz, 1998; Peterson et al., 2009; Ralston et al., 2007).

Two local studies suggest that the prevailing selenium concentration in the NSFB might be beneficial in terms of protection against methylmercury bioaccumulation and its toxicity to diving ducks and fish. Hoffman et al. (1998), studied the hepatic mercury, selenium and the oxidative stress status of diving ducks in Suisun Bay compared to Tomales Bay. The data from this study is summarized in Figure 13. The data suggest that in all three species of diving ducks, increased hepatic selenium concentrations appear to reduce the bioaccumulation of mercury. Note that the biota (resident small fish and mussels – food source of these bird species) is generally not more contaminated with mercury at the Tamales Bay than the Suisun Bay (Kimbrough et al., 2008; Luoma & Linville, 1995; Marshal, 2007; O'Conner, 1992; Phillips, 1988; RWQCB, 1998a).

Stewart et al. (2004), studied the food web pathway of selenium in white sturgeon in the San Francisco Bay. Out of 37 white sturgeon collected over a 2-year period, one individual appeared to be mainly piscivorous (based upon $\delta^{15}N$ analysis). This individual had about 4 times less hepatic selenium concentration compared to the group mean (5.5 vs 22 µg/g); however, this individual's muscle tissue mercury concentration was about 3 times higher than the group average (3.68 vs 1.12 µg/g). Although Hoffman et al. (1998) and Stewart et al. (2004), data is limited, it does suggest that if diving ducks and white sturgeon were to switch to a relatively low-selenium diet from a

selenium enriched diet (overbite clams), the muscle tissue mercury concentration in both species is expected to increase at least by a factor of two to three. This may have serious consequences in terms of potential increased mercury toxicity to fish and diving ducks, as well as a threat to human health from consumption of increased mercury-contaminated fish and diving ducks from NSFB. Therefore, it appears that further reduction in selenium may exacerbate mercury contamination in aquatic life in NSFB.

SUMMARY

Since 1986, regulators have required reductions in selenium discharge to the San Francisco - Bay Delta through NPDES permit limits on refineries and agricultural drainage. These management decisions have paid off. Selenium levels in agricultural drainages and refineries have been reduced by a factor of 2 and 4 respectively in the mid-to-late '90s. Consequently the selenium in diving ducks and white sturgeon have been reduced 57 and 67 percent respectively in 2005. Based on the above discussion, we find that the current available data on selenium concentrations in water column, sediment, diving duck tissue, bird eggs, and fish tissue do not appear to threaten aquatic life, wildlife and/or human health. Further reductions in selenium in the NSFB, has a potential to exacerbate mercury contamination in fish, wildlife, and humans.

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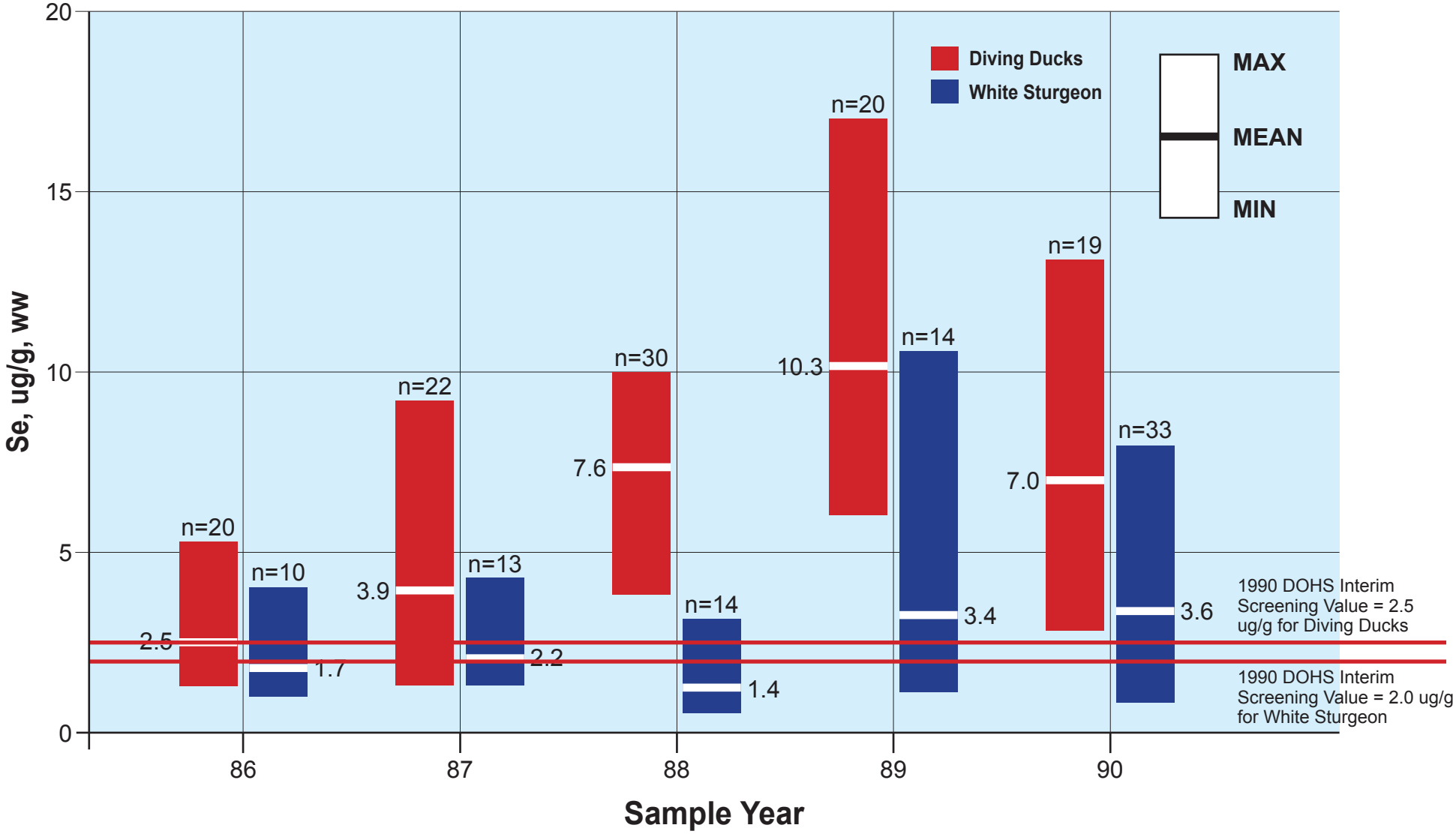
Figure 1

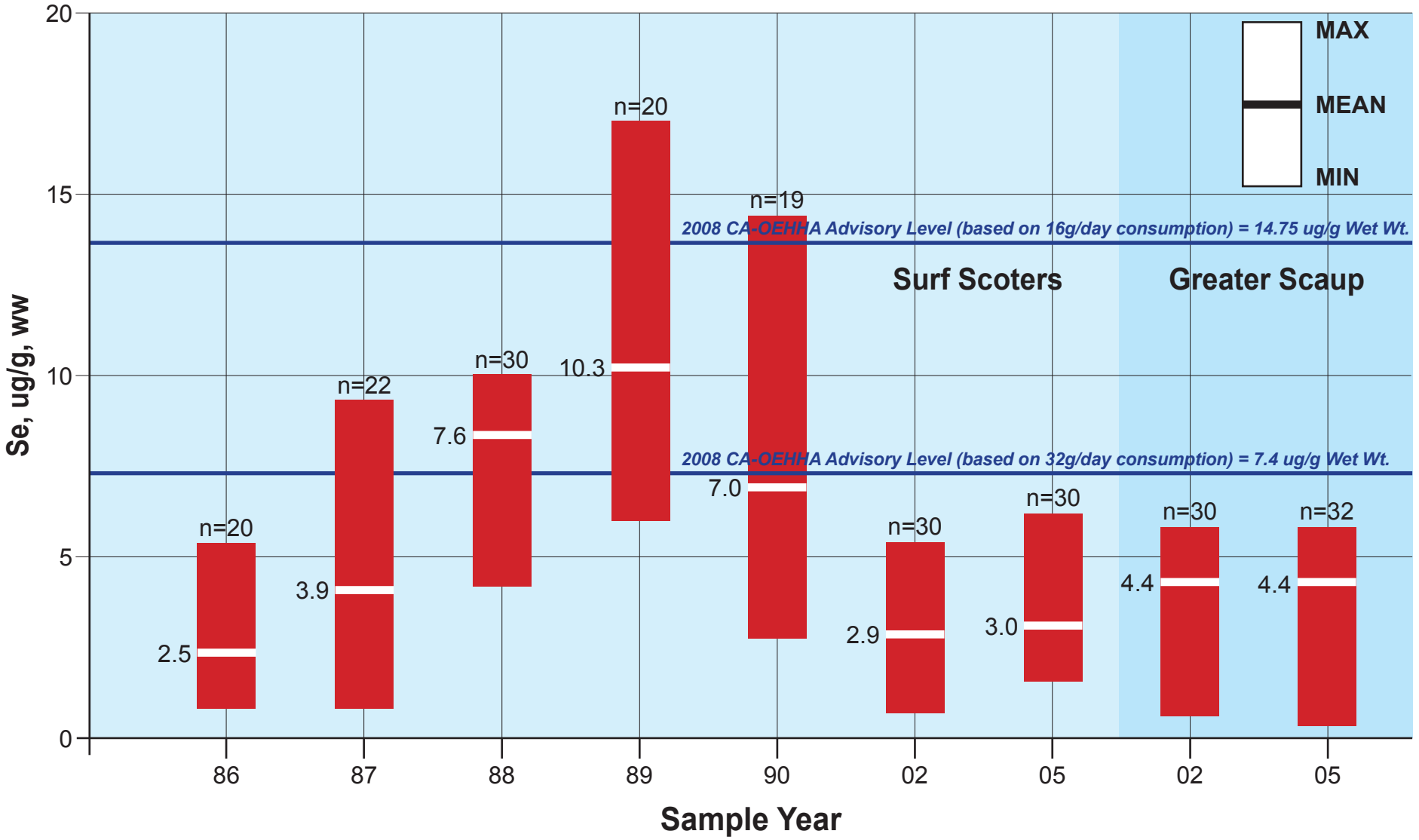
Selenium Concentration* in Diving Ducks and White Sturgeon (1986-1990) in Relation to CA DOHS 1990 Interim Screening Value



** In muscle tissue, average and range*
SOURCE: Selenium Verification Study. (SWRCB 1991)

Figure 2

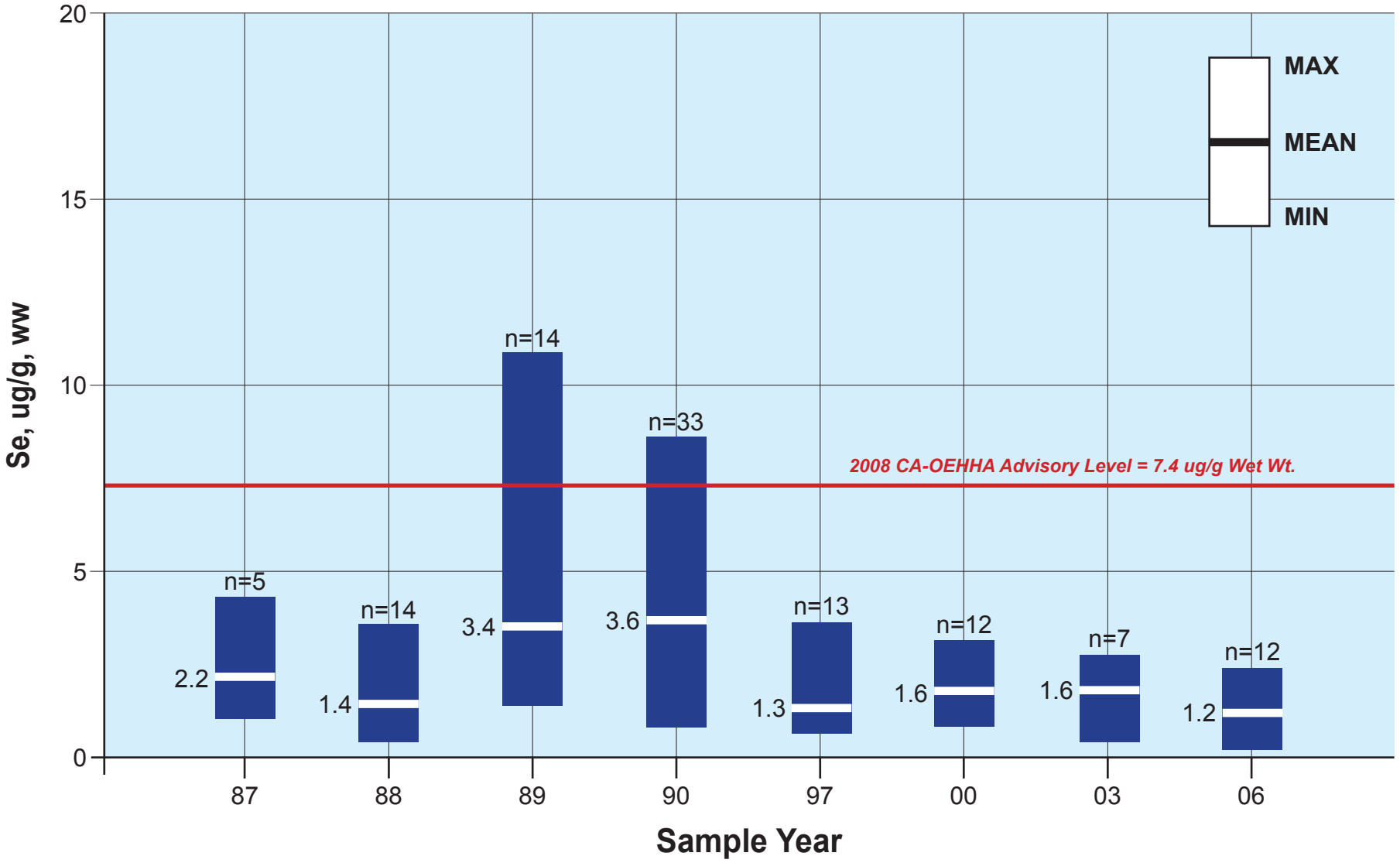
Selenium Concentration* in Diving Ducks (1986-2005) in Relation to CA OEHHA 2008 Tissue Advisory Level



** In muscle tissue, average and range*
SOURCE: Selenium Verification Study. (SWRCB 1991) and (SFEI 2008)

Figure 3

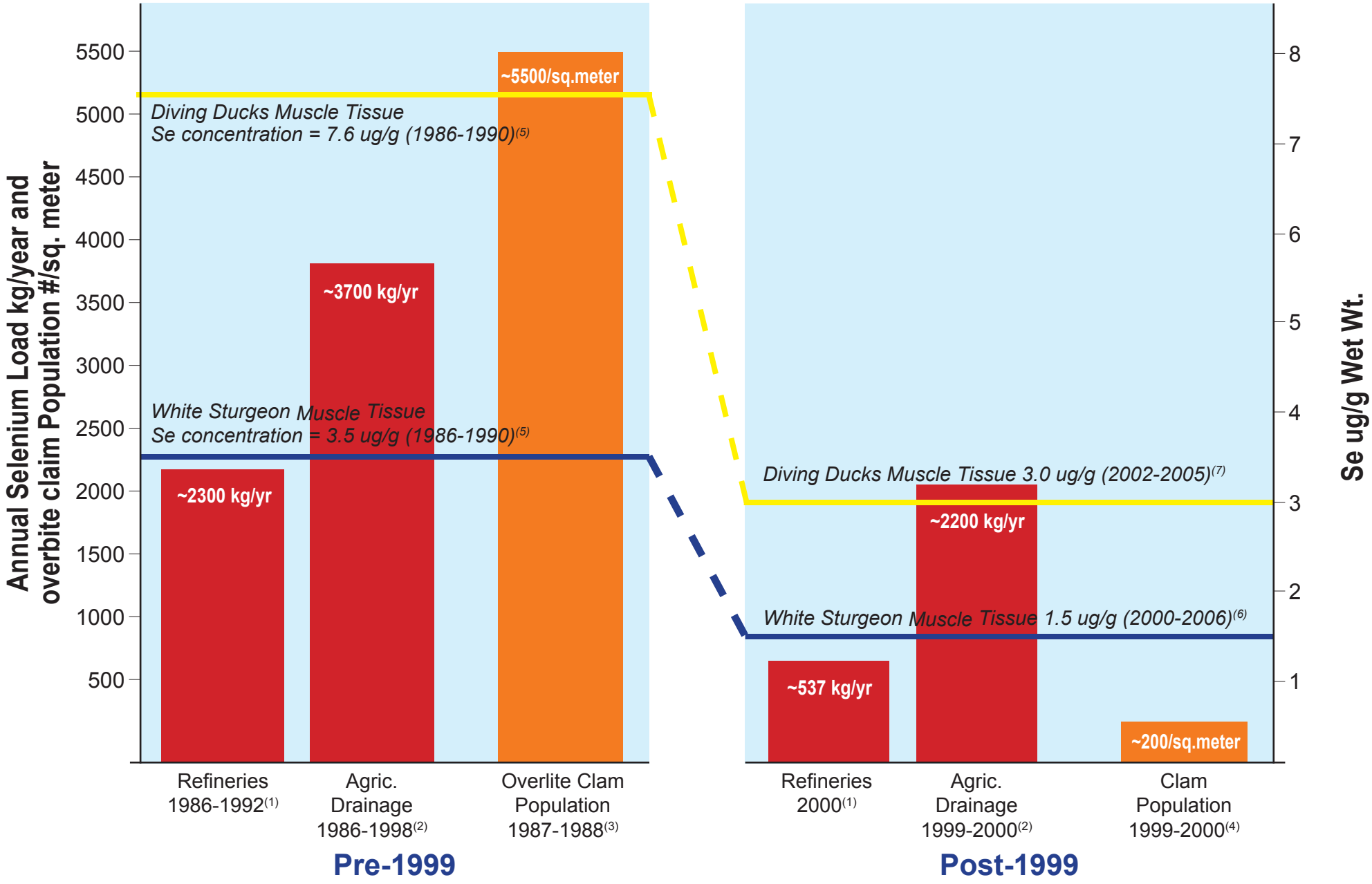
Selenium Concentration* in White Sturgeon (1987-2006) in Relation to CA OEHHA 2008 Tissue Advisory Level



** In muscle tissue, average and range*
SOURCE: Selenium Verification Study. (SWRCB 1991) and RMP Annual Reports 1997-2006

Figure 4

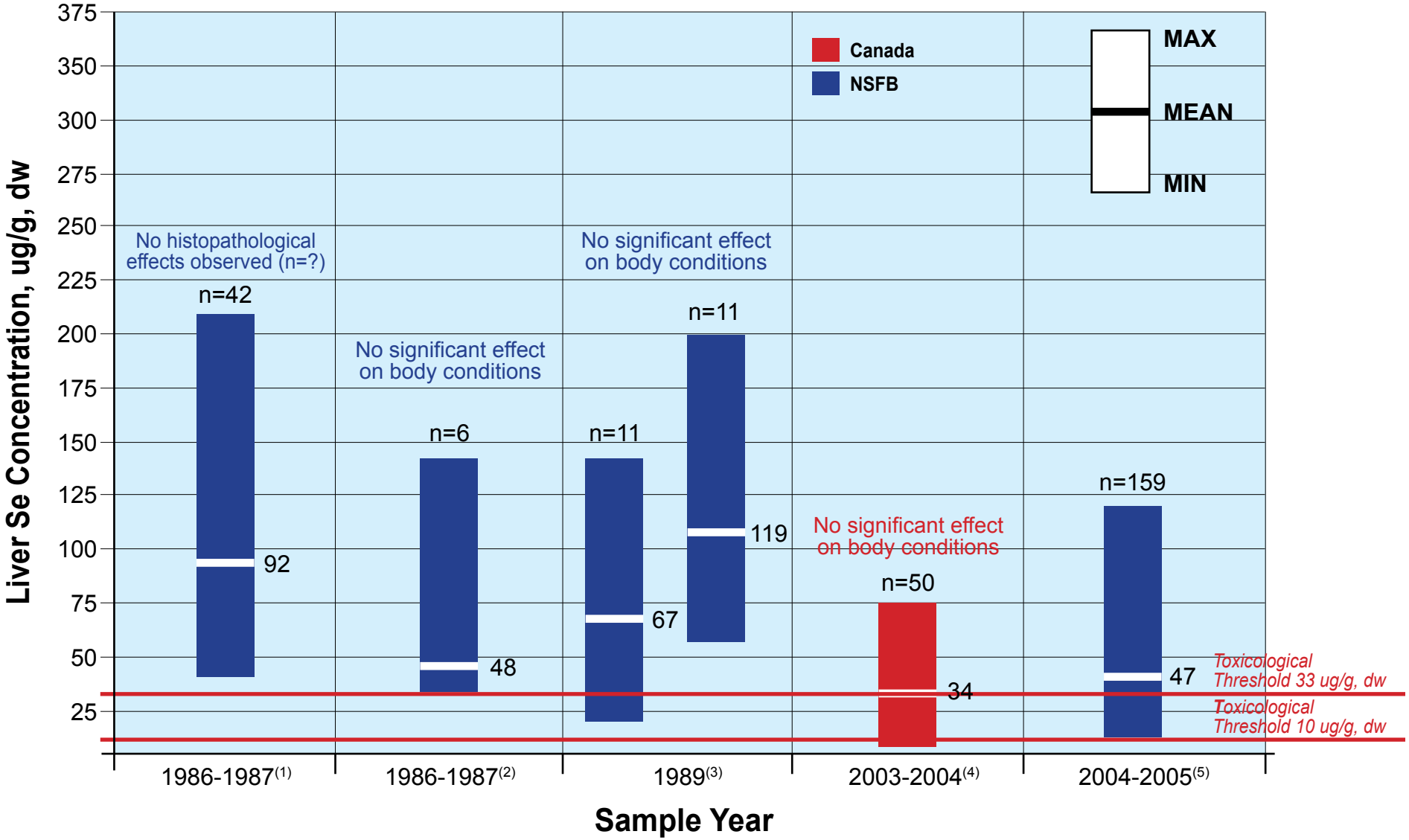
The Status of Key Indicators Impacting the Se Concentration of Diving Ducks and White Sturgeon in NSFB Pre and Post 1999



1. Tetra Tech (2008)(a), 2. Nicole et al (2008), 3. Carlton et al (1990),
4. Poulton et al (2004), 5. SWRCB (1991), 6. SFEI (1997-2006), 7. SFEI (2008)

Figure 5

Hepatic Se Concentration of Diving Ducks from NSFB* and Canadian Boreal Region in Relation to MallardsToxicity Thresholds

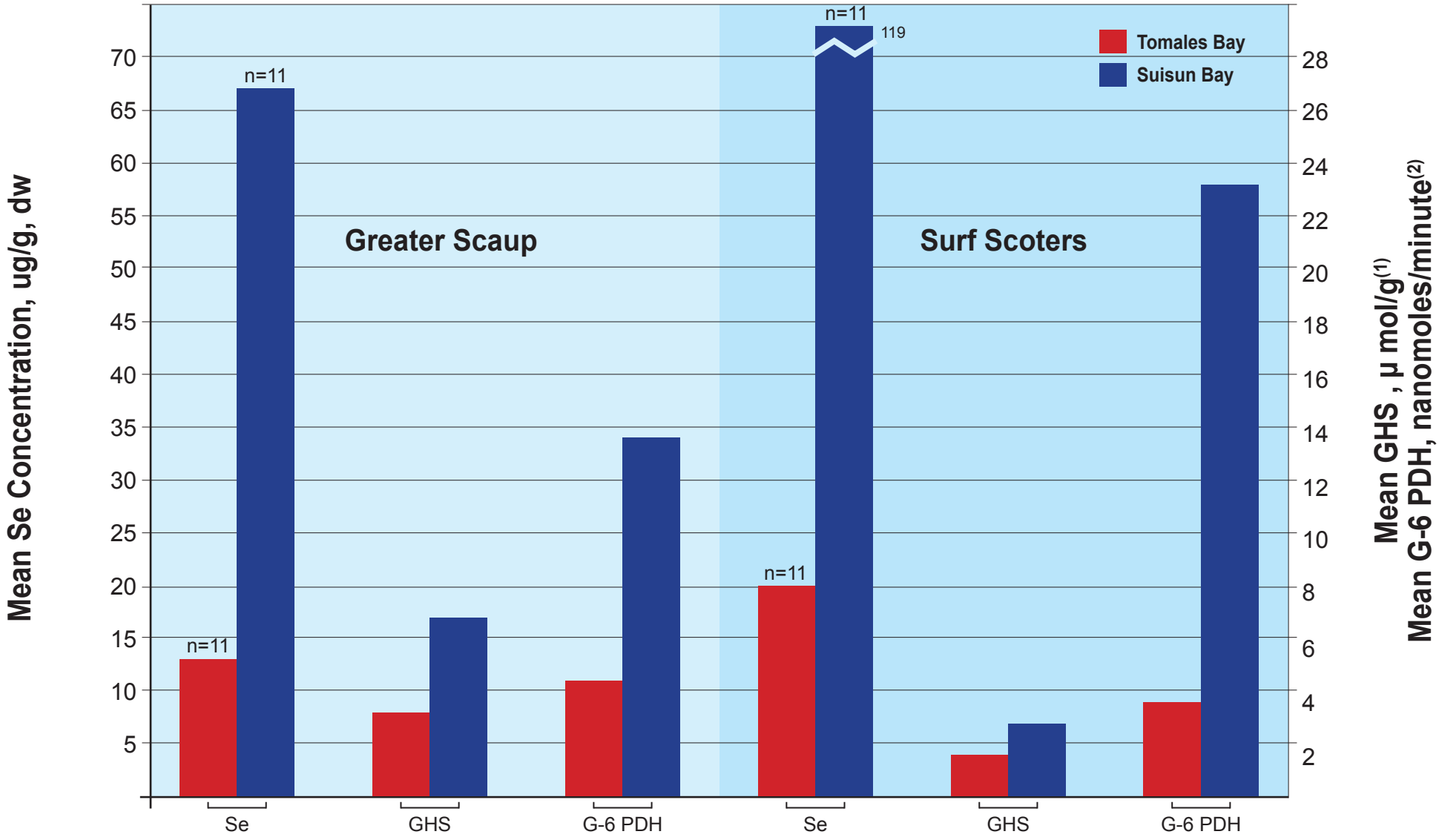


* North San Francisco Bay

1. SWRCB (1991)
2. Takekawa et al (2002)
3. Hoffman et.al. (1998)
4. DeVink et al (2008)
5. Wainwright - De La Cruz et al (2008)

Figure 6

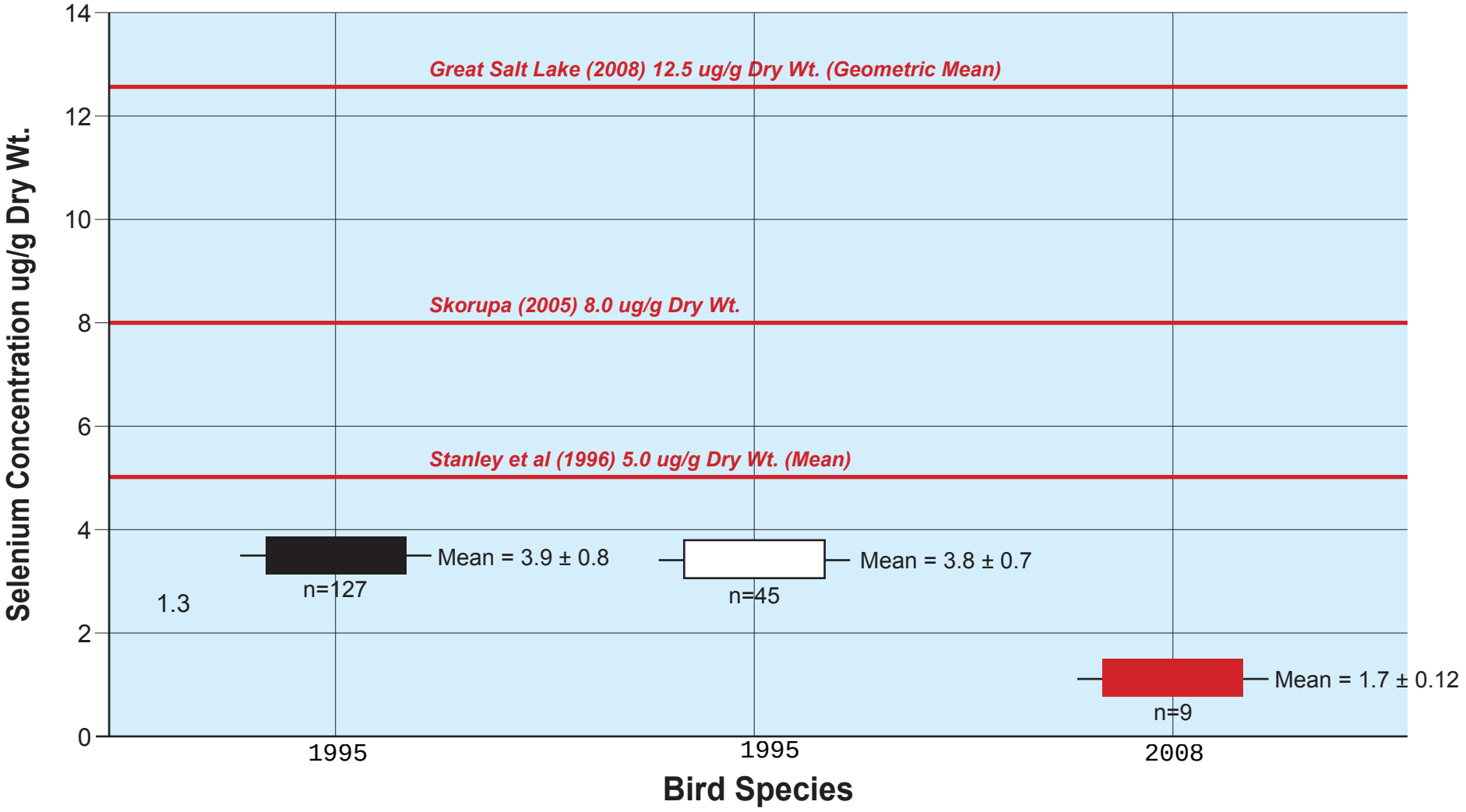
Hepatic Selenium Concentrations and the status of Oxidative Stress in Diving Ducks from Suisun Bay Compared to Tomales Bay*



** From Hoffman et al (1998)*
1. Reduced glutathione
2. Glucose - 6 - phosphate dehydrogenase

Figure 7

Selenium Concentration in eggs of Diving Ducks and Wading Birds from San Francisco Bay in Relation to egg Selenium concentration Thresholds



Black-crowned night-heron (Hothem et al 1995)

Snowy Egret (Hothem et al 1995)

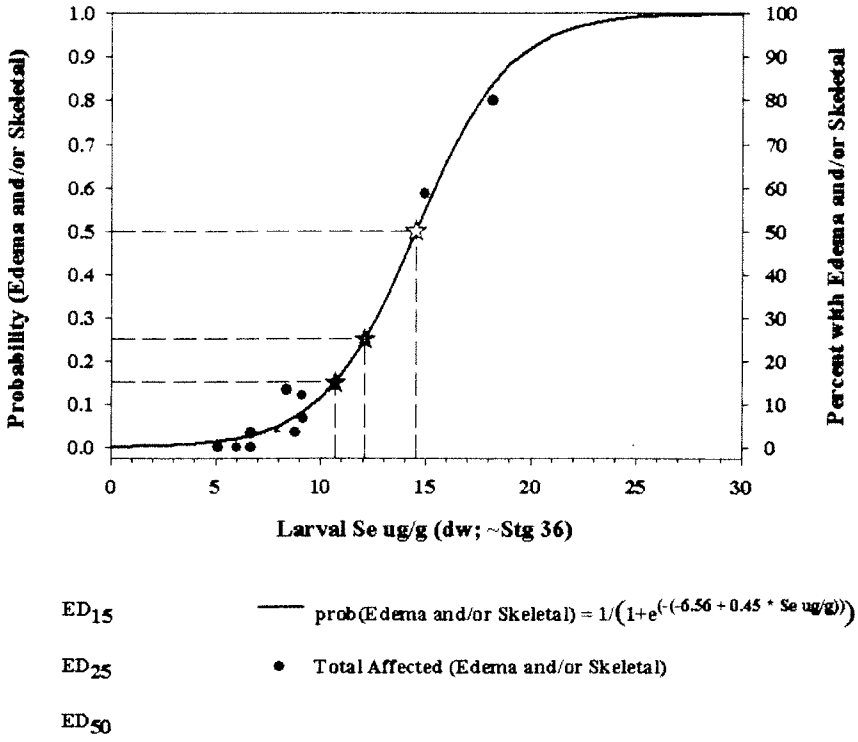
Diving Duck (Wainwraight—De la Cruz (2008)

Figure 8

White sturgeon larvae following microinjection of seleno-L-methionine

Occurrence of skeletal deformities and/or edema related to larval selenium exposure

Microinjection Study Two



Percentage of larvae with edema and/or skeletal deformities related to selenium concentrations in larvae (μg/g; dw) at the start of larval development (*ca.* stage 36). Larvae were microinjected with seleno-L-methionine in ***microinjection study two.*** Data includes two progeny cohorts and spans early larval development (*ca.* stages 36 – 45). Logit analysis shows a significant association between selenium concentrations in larvae and the occurrence of defects ($\chi 2 = 122.7$, $p < 0.0001$; prob(*defects*) = $1/(1+e^{(-(-6.56 + 0.45 * (\text{Se in Larvae ug/g}))})$). The left y-axis shows the probability of larvae to develop edema and/or skeletal deformities at a given larval selenium concentration, as determined by logit analysis. The right y-axis shows the actual percentage of affected larvae in samples containing *ca.* 30 larvae each. ED_{50} (14.5 μg/g), ED_{25} (12.1 μg/g) and ED_{15} (10.7 μg/g) values were calculated from the logit equation.

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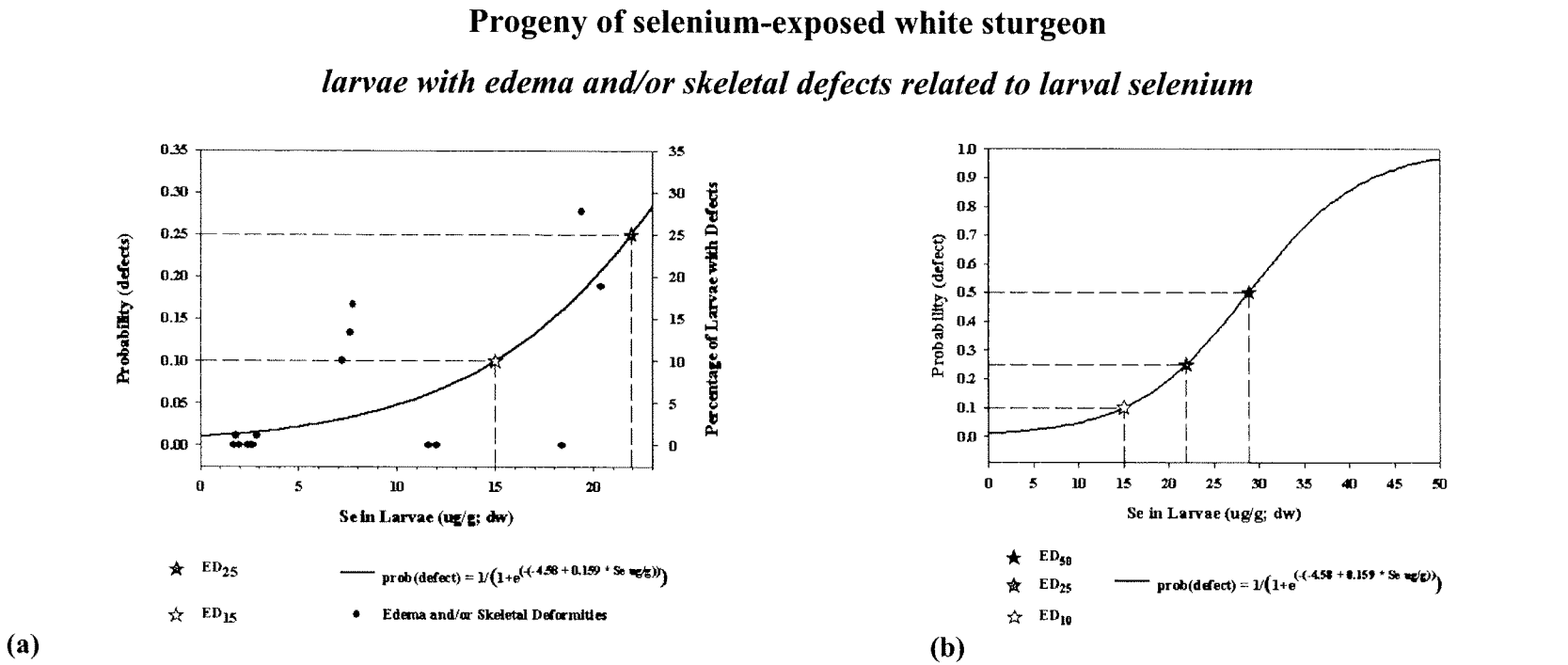
Figure 9



Percentage of larvae with edema and/or skeletal deformities related to selenium concentrations in larvae (µg/g; dw). Larvae were progeny of adult white sturgeon females exposed to dietary selenium. Data includes three progeny cohorts from the control group and three from the treatment group. Data for three larval development stages are included (stages 36, 40 and 45). Logit analysis shows a significant association between selenium concentrations in larvae and the occurrence of defects ($\chi^2 = 66.5$, $p < 0.0001$; prob*(defects)* $= 1/(1+e^{(-(-4.58 + 0.159 * (\text{Se in Larvae ug/g}))})$). (a) The right y-axis shows the actual percentage of affected larvae in samples containing 30 – 90 larvae each. The left y-axis shows the probability of larvae to develop edema and/or skeletal deformities at a given larval selenium concentration. (b) ED_{50} (28.83 µg/g), ED_{25} (21.92 µg/g) and ED_{10} (15.01 µg/g) values were calculated from the logit equation.

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TABLE 1

Selenium Concentrations (ug/g dw) in Adult Tissue from Female White Sturgeon and Their Progeny Relative to Incidence of Development Defects*

Exposure	Fish / Progeny ID	Liver	Muscle	Ovarian Tissue	Egg	Larvae[1]	Percent Developmental Defects[2]
Control	C3	1.33	1.28	1.49	2.46	2.43	0[3]
Control	C4	0.80	1.22	1.65	1.61	1.69	0
Control	C5	2.16	1.48	1.38	2.68	2.67	0
Treatment	T3	11.00	11.10	5.74	7.61	7.75	13.33 +/- 3.33
Treatment	T1	8.72	9.93	6.53	11.00	11.6	0
Treatment	T2	11.60	15.30	13.20	20.50	18.4	27.78 +/- 2.94

Exposure to either Control (*ca*. 1.4 ug/g) or Treatment (*ca*. 34 ug/g) dietary selenium for approximately six months during vitellogenesis. Selenium concentration and occurrence of developmental defects are shown for the progeny of each female (n=25 – 30 larvae for stage 36; n= 60 – 90 larvae for stages 40 and 45).

[1] Newly hatched larvae (stage 36)

[2] Occurrence of edema and/or skeletal deformities at the end of yolk sac development

[3] Data from stage 36 only, due to very low hatch

*Reproduced from Linville (2006) Table 3-18

TABLE 2

Mean and Std. Dev. (in parentheses) of Percent Survival of Chinook Salmon Exposed to Selenium in the Diet and Reared in a Reconstituted Fresh Water.

DIET TYPE	DIETARY SELENIUM CONC. (ug/g dry wt)	EXPOSURE PERIOD 30 Days			60 Days			90 Days		
Control [1]	1.0	99.0	(1.0)	1.0 [4]	99.0	(1.0)	0.9 [4]	66.7	(4.1)	0.8 [4]
SLD [2]	3.2	99.0	(1.0)	3.0	97.3	(1.6)	3.3	56.0	(11.0)	2.7
	5.3	98.0	(2.0)	3.9	93.0	(2.7)	4.5	44.7	(10.4)	4.0
	9.6	100.0	(0)	6.8	95.0	(1.7)	8.4	31.2	(15.7)**	6.5**
	18.2	99.0	(1.0)	10.6	92.4	(4.8)	13.3	20.9	(11.7)**	14.3**
	35.4	99.0	(1.0)	21.1	89.0	(3.1)*	29.4	18.2	(11.0)**	33.5**
SeMet [3]	3.2	100.0	(0)	2.4	100.0	(0)	2.0	72.5	(12.5)	1.7
	5.3	100.0	(0)	3.1	95.0	(1.7)	3.1	45.0	(10.2)	2.6
	9.6	99.0	(1.0)	5.1	94.1	(4.0)	5.2	37.5	(13.1)*	5.4
	18.2	99.0	(1.0)	8.5	92.4	(4.8)	10.4	22.4	(11.3)**	10.8**
	35.4	84.0	(9.8)	16.0	62.5	(14.0)**	23.8**	10.7	(6.8)**	27.8**

Asterisks indicate significant difference from the control:

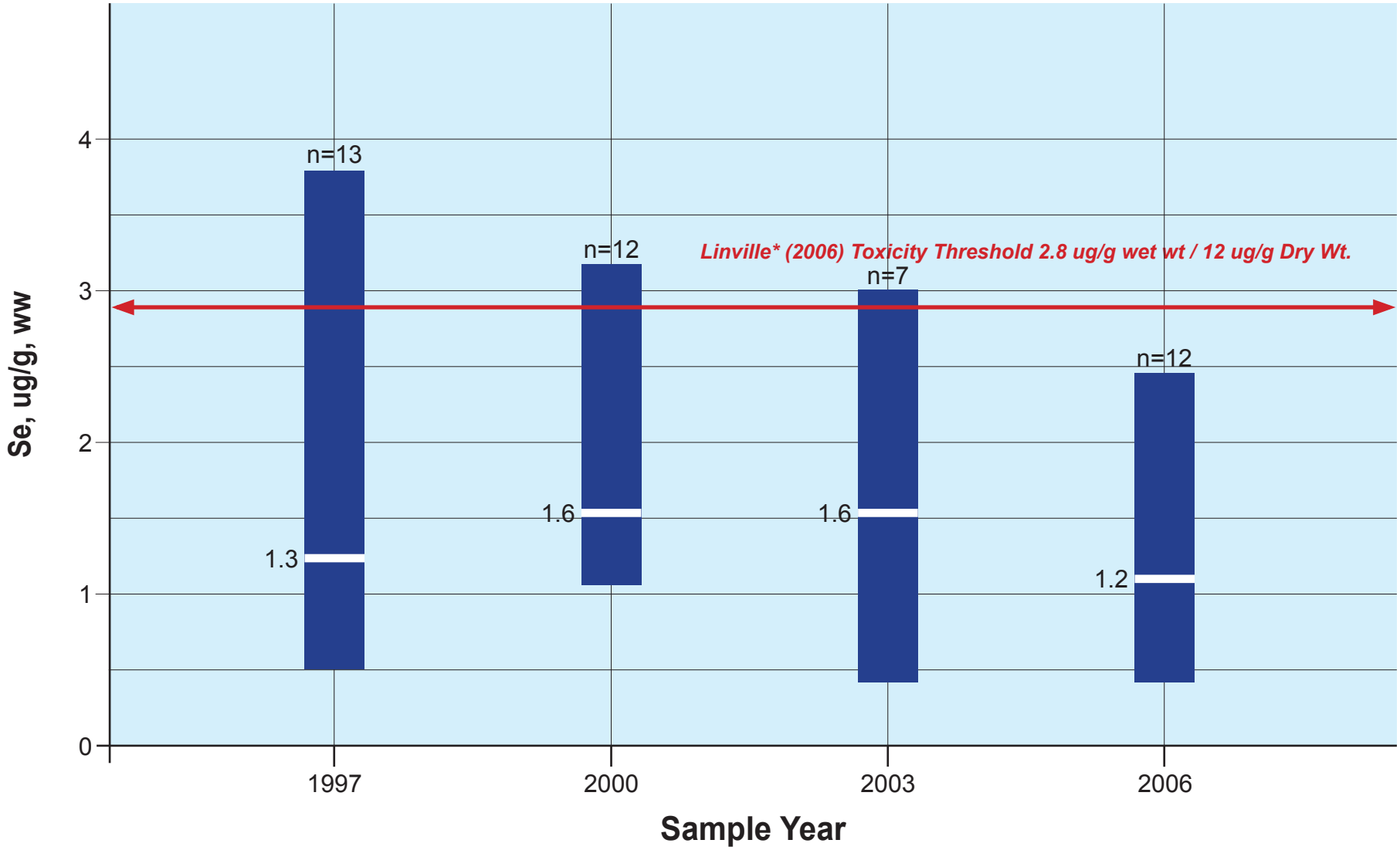
ANOVA: * p≤ 0.10 ** p < 0.05

1. Diet from low selenium mosquito fish
2. Diet from high selenium mosquito fish collected from selenium-laden San Luis Drain
3. Low Selenium mosquito fish fortified with selenium-DL-Methionire
4. Fish tissue Se. concentration µg/g . dw.

* Reproduced from Hamilton et. al. (1990)

Figure 10

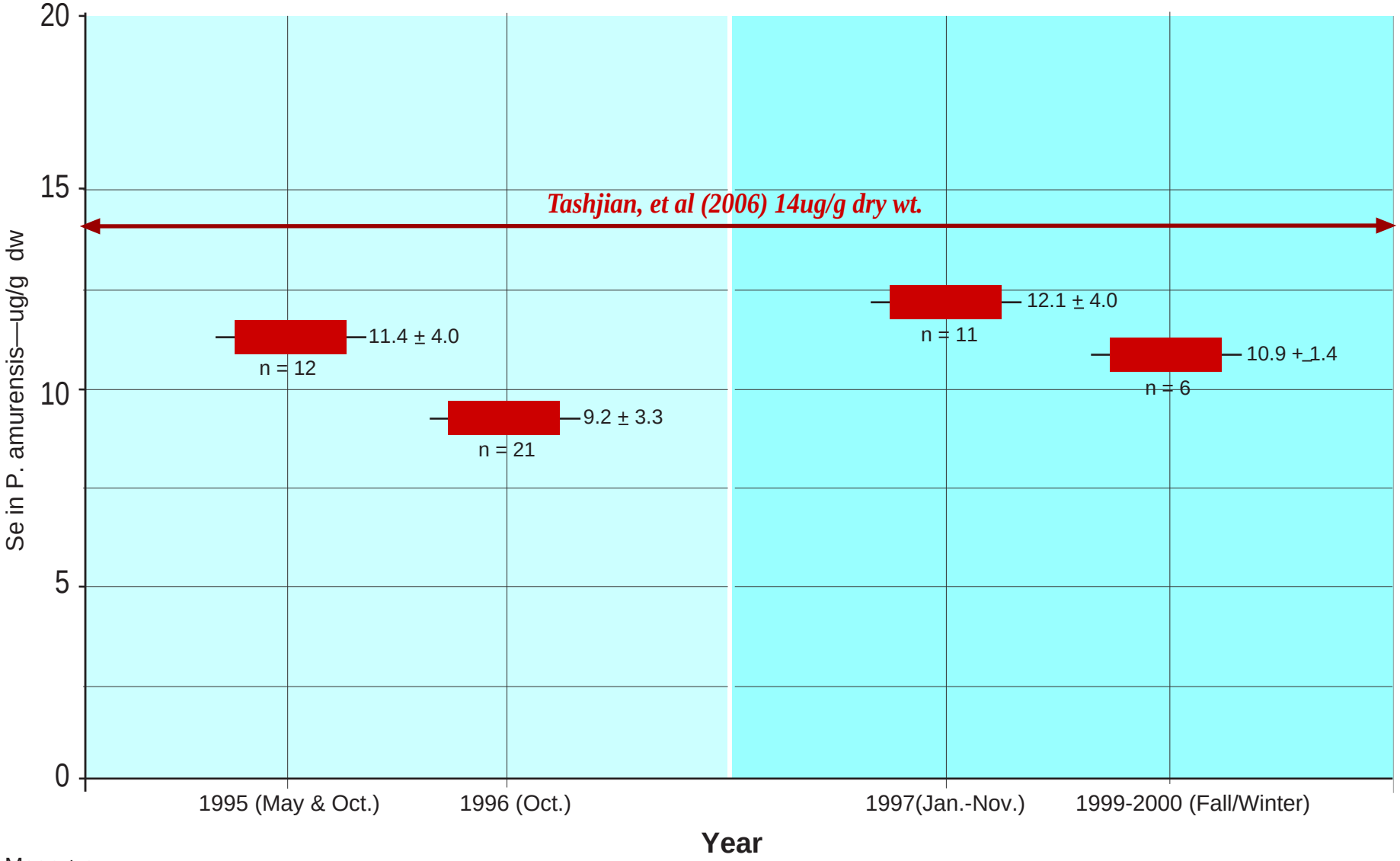
Selenium Concentration in Adult White Sturgeon in San Francisco Bay (1997-2006) in Relation to Linville* (2006) Toxicity Threshold



** Our calculated value from Linville (2006) maternal experiments. (see text)*
SOURCE: RMP Annual Reports 1997-2006: muscle tissue, average and range

Figure 11

Selenium Concentration* (ug/g dry wt.) in P. amurensis from North San Francisco Bay in Relation to Selenium Dietary Exposure Threshold

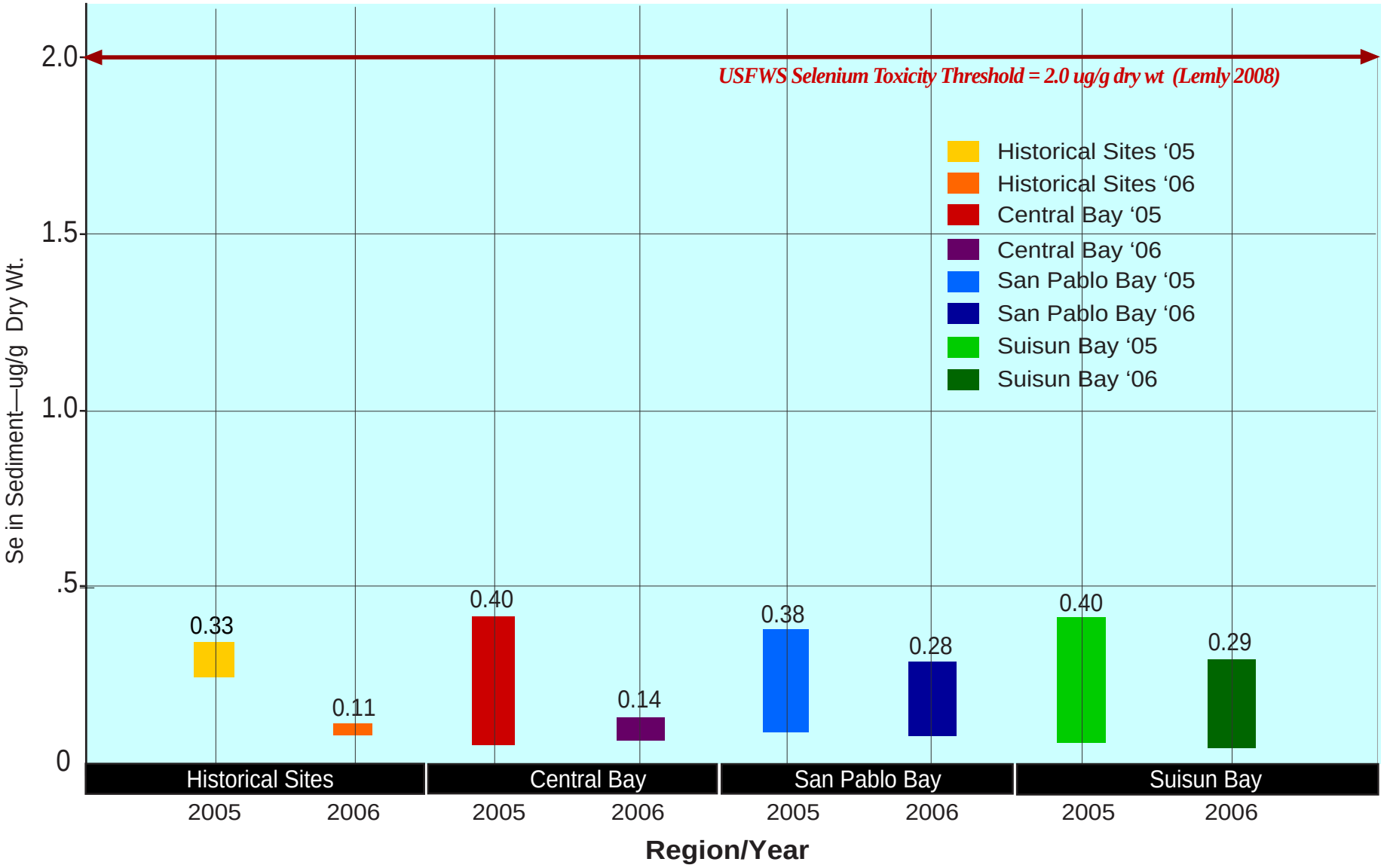


* Mean ± s

SOURCE: Linville et. al. (2002), Stewart et. al (2004) and Stewart (2008)

Figure 12

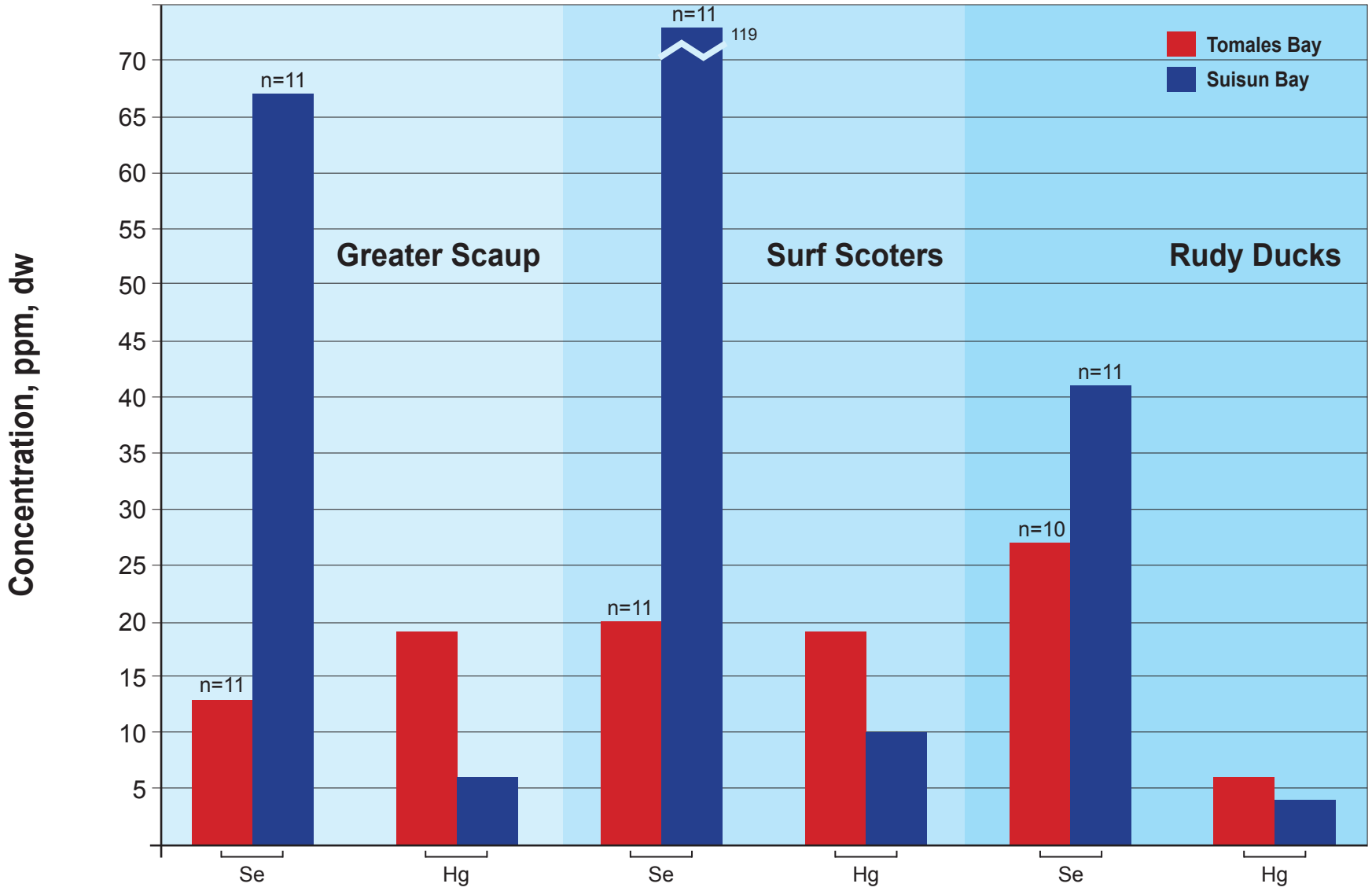
Selenium Concentration range (ug/g dry wt.) in Sediments (Surface) from North San Francisco Bay (2005 - 2006) in relation to USFWS Selenium Toxicity Threshold



* Source 2005-2006 RMP Annual Reports.

Figure 13

Elevated Hepatic Selenium vs. Mercury Assimilation in Diving Ducks from Suisun Bay and Tomales Bay*



** From Hoffman et al (1998)*