How Might Selenium Moderate the Toxic Effects of Mercury in Stream Fish of the Western U.S.?

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The ability of selenium (Se) to moderate mercury (Hg) toxicity is well established in the literature. Mercury exposures that might otherwise produce toxic effects are counteracted by Se, particularly when Se:Hg molar ratios approach or exceed 1. We analyzed whole body Se and Hg concentrations in 468 fish representing 40 species from 137 sites across 12 western U.S. states. The fish samples were evaluated relative to a published wildlife protective Hg threshold (0.1 µg Hg·g⁻¹ wet wt.), the current tissue based methylmercury (MeHg) water quality criterion (WQC) for the protection of humans (0.3 wt.), the current tissue based methylmercury (MeHg) water quality criterion (WQC) for the protection of wildlife (0.3 µg Hg·g⁻¹ wet wt.) and to presumed protections against Hg toxicity when Se:Hg molar ratios are >1. A large proportion (56%) of our total fish sample exceeded the wildlife Hg threshold, whereas a smaller, but significant proportion (12%), exceeded the MeHg WQC. However, 97.5% of the total fish sample contained more Se than Hg (molar ratio >1) leaving only 2.5% with Se:Hg ratios <1. All but one of the fish with Se:Hg <1, were of the genus Pychocheilus (pikeminnow). Scientific literature on Se counteracting Hg toxicity and our finding that 97.5% of the freshwater fish in our survey have sufficient Se to potentially protect them and their consumers against Hg toxicity suggests that Se in fish tissue (Se:Hg molar ratio) must be considered when assessing the potential toxic effects of Hg.

Introduction

Selenium is an essential nutrient for all life forms that have nervous systems, but Se can be toxic when present at high levels in the environment. There is no physiological requirement for Hg, but it bioaccumulates in the aquatic food chain and fish are the chief exposure route for wildlife and humans. In fish, MeHg constitutes 95–97% of the total Hg in fish files (1). Therefore, since total Hg is more easily measured in fish tissue, total Hg measurements are recommended for fish surveys by EPA (2).

At high exposures, Se and Hg can each be individually toxic, but evidence supports the 1971 observation by Parizek et al. (3) that co-occurring Se and Hg antagonistically reduce each other’s toxic effects. In 1972, Ganther et al. (4) found that tuna containing an ≈1:1 molar ratio of Se:Hg reduced toxic effects of MeHg. He attributed the reduced toxicity to Se in the tuna. Various hypotheses for the Se protective mechanism have been proposed (5, 6). One of the most comprehensive involves formation of highly stable organic MeHg-selenocysteine (MeHg-SeCys) that forms in the brain and nervous systems of Hg stressed organisms (7). This form and its products are highly stable, thus making the Se biochemically unavailable (8, 9). Sequestration (deactivation) of Se by high concentrations of MeHg inhibits normal selenoenzyme antioxidant activities that result in the adverse effects associated with Hg toxicity. However, during Hg stress, redistribution of Se from somatic cells and dietary sources to preferentially supply the brain replaces some of the Se lost to HgSe and MeHg-SeCys formation. This reduces the toxic effects by maintaining selenium-dependent enzymes (selenoenzymes) required for brain function and protein synthesis (10, 11).

Methylmercury is, by biochemical definition, an irreversible inhibitor of selenoenzymes since it transfers from the thiol of cysteine to the selenol of selenocysteine at the enzymes active site (7, 12). Since selenocysteine is a critical component of protein synthesis and must be formed de novo during each cycle of cellular protein synthesis (7, 12), inhibition of its formation critically impairs cell metabolism. Based on rat (7, 13) and mice studies (14), MeHg toxicity appears to occur when molar concentrations of MeHg exceed those of Se and covalent bonding of MeHg to the Se of selenocysteine occurs, thereby irreversibly inhibiting Se-dependent enzymes (7, 13). In adult onset, molar surpluses of Se over Hg (Se:Hg molar ratio >1) tend to protect the brains of Hg-stressed organisms. Fetal and young organisms are at much greater risk of toxicity from Hg exposure because the rapid rate of cell division in these organisms requires a steady supply of Se. Watanabe et al. (14) demonstrated that in utero Se nutritional status affects MeHg neurotoxicity. Additionally, Ralston et al. (7) found that neurofunctional defects (hind leg crossing) can be stabilized, and growth impairments in young rats can be reversed by increasing the amount of Se in their diets, even while maintaining high MeHg exposures. Peterson et al. (15) showed that total Hg (THg) in fish filets exceed that in whole fresh water fish (0.185 µg THg·g⁻¹ wet wt. in whole fish = 0.3 µg THg·g⁻¹ wet wt. in fish filet). If, as Gather (4) suggested, the molar ratio of Se:Hg in fish filet is ~1 it follows that the mass of Se in filet might be approximately the same or greater than that of Hg. Harris et al. (16) and Korbas et al. (17), recently determined that various forms of Se complex with MeHg in fish filets, making the 95–97% of MeHg in fish tissue (1) less toxic to the fish and presumably to consumers (4) of the fish than previously thought. Harris et al. (16) indicated that zebrafish larvae are 20 times less sensitive to cystine-bound MeHg (MeHg(Cys)), the predominant form of MeHg found in fish tissue, than they are to MeHgCl, that is commonly used in toxicity tests. This was corroborated when Calaberto et al. (18) discovered that fish tissue maintains the MeHg(Cys) association after passing through an artificial digestion process. The MeHg(Cys) does not dissociate into toxic MeHg forms as previously suspected.
Selenium’s effect in counteracting Hg toxicity increases throughout Se’s nutritionally relevant range and has been demonstrated in all insect, fish, bird, and mammal species tested to date (13). However, effects remain controversial. A review of adult effects resulting from fetal exposure in MeHg exposed animal models by Newland et al. (6) suggests that diets rich in Se do not uniformly protect against MeHg’s effects. The review by Yang et al. (5) points out that “a large number of scientific studies have provided strong evidence of the protective role of Se in preventing the detrimental effect of CH3Hg+.” Ralston et al. (7) found that MeHg toxicity in rats could not be predicted from tissue MeHg content alone, but that toxicity was directly related to the Hg:Se molar ratios in the tissue. Thus, it appears that selenium-dependent protection against Hg-toxicity depends not on Hg concentrations per se, but rather on the total mass ratio of Se to Hg.

Gaither (4) first mentioned the Se:Hg molar ratio of 1:1 as protective against Hg toxicity in fish. Luten et al. (19) drew a similar conclusion relative to both freshwater and marine fish.

Since the evidence indicates that Se:Hg molar ratios influence the toxicity of either element and that these ratios are useful in interpretation of toxicity, we developed the fish tissue data in this paper from that perspective. The purpose of this paper is to describe the Se:Hg molar ratios in whole stream fish (n = 468) collected from 137 sites across 12 western U.S. states and to relate those ratios to a published wildlife methylmercury (MeHg) consumption threshold (0.1 µg Hg·g⁻¹ wet wt.) (20). In addition, we comment on these molar ratios relative to the current methylmercury (MeHg) water quality criterion (WQC) for protection of humans (0.3 µg Hg·g⁻¹ wet wt.) (21) and on potential fish tissue Se toxicity.

Materials and Methods

Procedures for sample site selection, Hg analysis, Hg quality assurance, and quality control (QA/QC), and results of fish tissue Hg analyses were reported previously (15). Each is described briefly as follows.

Probability Sample Design. For Se analysis, we selected 468 freeze-dried samples that previously had been analyzed for Hg (15). All piscivores (n = 206) were analyzed, since those fish commonly contain the highest Hg concentrations and are among commonly sought game fish. Presumably they pose the greatest potential risk of Hg toxicity relative to fish reproduction or consumption by other fish. In addition, we analyzed a random sampling (n = 262) of the remaining nonpiscivorous fish.

Stream and river sampling sites were drawn from Arizona, California, Colorado, Idaho, Montana, Nevada, North Dakota, Oregon, South Dakota, Utah, Washington, and Wyoming, on a probability basis, from the perennial stream network appearing on the 1:100, 000-scale digital line graph database of the United States Geological Survey (22–24). At each site, up to nine individual fish (three individuals from up to three different piscivore and nonpiscivore species) could be collected, but not all sites yielded fish.

Sample Collection and Processing for Hg Analyses. We collected fish from streams and rivers according to wadeable and nonwadeable electrofishing protocols (25, 26). Fish were wrapped in aluminum foil, double-bagged in resealable freezer bags, and shipped on ice to the laboratory within 36 h of being caught (25, 26). At the laboratory, they were inspected for condition and stored frozen at −20 °C until processing (15).

Freeze-Dried Sample Preparation. A second set of wet homogenate subsamples were freeze-dried for Se analysis at the same time the above samples were prepared. Since Se analysis by Instrumental Neutron Activation Analysis (INAA) requires a very small, but uniformly mixed sample, the freeze-dried samples were prepared according to a procedure prescribed by the University of Missouri Research Reactor. The full procedure is described in the Supporting Information (Methods - Se Sample Preparation).

Mercury Analysis. All Hg analyses were done on frozen wet homogenate samples by combustion atomic absorption spectrometry (CAAS) using a direct mercury analyzer (Milestone DMA80; Milestone, Monroe, CT or LECO model AMA 254; LECO Corporation, St. Joseph, MI) and EPA Method 7473 (27). Samples were analyzed in triplicate, and reanalyzed if the relative standard deviation (RSD) exceeded ±3%. The result for each sample was reported as the mean wet weight Hg concentration. All Hg analyses were performed within time frames that assured against nondegradation and/or changes in the Hg content of fish tissue (28).

Mercury Detection Limit and Quality Assurance. The analytical method detection limit (MDL) was calculated using the method of Taylor (29) as published in 1986 by the U.S. EPA in 40 CFR Part 136, Appendix B, Revision 1.11. The MDL was based on repeated analyses between 2000 and 2004 (n = 875) of a low-level standard (NIST 2976 mussel tissue) and expressed as µg Hg·g⁻¹ wet wt. (assuming a water content of 70% for the mussel species used for the standard (30)). The MDL was calculated to be 0.015 µg Hg·g⁻¹ wet wt.

We assessed analytical precision using 376 duplicate analyses of fish tissue homogenate samples within a single sample batch. Precision expressed as relative percent difference of duplicate measurements was 6.4%. We assessed systematic error of our Hg analyses by repeated analyses of two standard reference materials (SRMs) during sample analytical runs: a high-level SRM (DORM-2 dogfish tissue; Institute for National Measurement Standards (INMS), Ottawa, ON, Canada) and a low-level SRM (NIST 2976 mussel tissue; National Institute of Standards & Technology (NIST), Gaithersburg, MD). For the DORM-2 SRM (certified as 4.64 ± 0.26 µg Hg·g⁻¹ dry wt.), the measured mean value was 4.58 µg Hg·g⁻¹ dry wt. (n = 1099, SD = 0.33 µg Hg·g⁻¹ dry wt., relative standard deviation [RSD] = ±7.3%), indicating a small negative bias (−1.2%). For the low-level NIST 2976 SRM (certified as 0.061 ± 0.004 µg Hg·g⁻¹ dry wt.), the measured mean value was 0.070 µg Hg·g⁻¹ dry wt. (n = 876, SD = 0.021 µg Hg·g⁻¹ dry wt., RSD = ±29.8%), indicating a positive bias (14.8%) at lower concentrations.

Selenium Analysis. All Se analyses were performed on freeze-dried fish homogenate samples by standard comparator INAA according to the analysis protocol of the University of Missouri Research Reactor (31–33). The procedure is described briefly in Supporting Information Methods: Se Analysis.

Selenium Limit of Quantitation and Quality Assurance. The limit of quantitation (LOQ) for the INAA Se analysis of fish homogenate under this protocol is on the order of 2 ng, which on a 0.025 g sample yields a fractional mass LOQ of 0.08 µg·g⁻¹ dry wt. The LOQ is based on 10 times the square root of the integrated baseline over an energy range of 160.2–163.7 keV. In gamma-ray spectroscopy, the standard deviation of the background for the measurement is the square root of the number of counts in the integrated baseline and the LOQ is 10 times one standard deviation of the background (34).

SRM NIST (1577 Bovine Liver; ca. 30 mg per sample) was used as an external quality control standard for the INAA measurements for two reasons. First, INAA Se analyses require small sample masses (30 mg). Thus, the 250 mg DORM-2 masses recommended by both NIST and National Research Council of Canada are incompatible with the INAA method. Second, DORM-2 and bovine liver standards behave identically relative to the INAA method. The certified value for Se in SRM 1577 is 1.1 ± 0.1 µg Se·g⁻¹ dry wt. Analysis of replicate
TABLE 1. Mass and Molar Concentrations of Mercury and Selenium and Surplus Se Concentrations in Various Fish Groups

<table>
<thead>
<tr>
<th>Fish group</th>
<th>Mean total length (mm)</th>
<th>Mean wet wt. (g)</th>
<th>Mean Hg concentration (µmol Hg·g⁻¹ wet wt.)</th>
<th>Mean Se concentration (µmol Se·g⁻¹ wet wt.)</th>
<th>Surplus Se concentration (µmol Se·g⁻¹ wet wt.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Piscivores</td>
<td>128.1 (165, 569)</td>
<td>0.0066</td>
<td>0.865 (0.0007, 0.0461)</td>
<td>0.0494 (0.0010, 0.0851)</td>
<td>0.0089 (0.0006, 0.009)</td>
</tr>
<tr>
<td>Piscivores</td>
<td>153.6 (180, 535)</td>
<td>0.0070</td>
<td>1.386 (0.012, 1.319)</td>
<td>0.103 (0.0010, 0.662)</td>
<td>0.063 (0.0025, 0.177)</td>
</tr>
<tr>
<td>Species</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Bullheads</td>
<td>193 (165, 220)</td>
<td>0.0087</td>
<td>1.289 (0.014, 0.453)</td>
<td>0.016 (0.0057, 0.0691)</td>
<td>0.0160 (0.0036)</td>
</tr>
<tr>
<td>All piscivores</td>
<td>325 (122, 640)</td>
<td>0.0094</td>
<td>1.740 (0.0075, 5.460)</td>
<td>0.0099 (0.0016)</td>
<td>0.0089 (0.0017)</td>
</tr>
</tbody>
</table>

Selenium mean concentration (minimum, maximum) - Surplus Se concentration (µmol Se·g⁻¹ wet wt.) mean (std. error) - Hg concentration for all piscivores in Table 1 (µmol Hg·g⁻¹ dry wt.) by fish group in Table 1 indicate all of the piscivore groups pose a toxicity risk relative to the wildlife threshold of 0.1 µg Hg·g⁻¹ wet wt., but the nonpiscivore groups present a mixed picture. Several individual pike-minnow, walleye, sauger, bass, and pike exceed the MeHg WQC (0.3 µg·g⁻¹ wet wt. for fillet) as it relates to whole fish concentrations ≥0.185 µg·g⁻¹. Based on an assessment using the MeHg WQC many individual fish in our sample likely would be recommended for limited or non-consumption by either wildlife or humans.

**Selenium: Mercury Molar Ratios:** Based on Se soil concentrations across our study area ranging from 0.17 to 0.74 µg·g⁻¹ dry wt. (35), we expected to see many fish types and regions in the western U.S. with fish Se: Hg molar ratios <1. However, there is a general geographic pattern of Se:Hg molar ratios >1 (surplus Se), but surplus Se is not uniformly present in all fish (Figure 2 and Supporting Information Table S1).

Figure 2 suggests that Se:Hg molar ratios might decline with increasing fish size, possibly reducing Se protection in larger fish. We tested this by linear regression of surplus Se against total fish length for piscivores and nonpiscivores. The relationship for piscivores is poor (r² = 0.085) and the one for nonpiscivores is worse (r² = 0.0004). We conclude from this that Se protection against Hg toxicity in larger fish probably remains intact. The proportion of piscivores with Se:Hg <1 (11 of 206) was substantially greater than that of nonpiscivores (1 of 262 fish; P < 0.001, for Fisher's exact test of the difference between proportions).
Pikeminnows. All of the fish in Figure 2 that have a Se:Hg molar ratio < 1 were pikeminnows (Ptychocheilus spp.), except the one largescale sucker (Catostomus macrocheilus), ranging in total length from about 300 to about 550 mm. There were 23 smaller and one larger pikeminnows with a Se:Hg molar ratio > 1. This suggests that some combination of fish species, fish size and possibly environment might play a role in determining Se:Hg ratios. Northern pikeminnows represent the top of the freshwater aquatic food chain and are known to be voracious piscivores (36). Zimmerman (36) found that the stomach of pikeminnows, relative to their total weight (index of feeding (IF)) was more than twice that of smallmouth bass (Micropterus dolomieu). This perhaps increases Hg bioaccumulation in large pikeminnows over other piscivores due to the potential uptake from the large stomach mass. However, since pikeminnow size alone appears not to control Se:Hg molar ratios, other factors must contribute. Pikeminnows having a molar ratio of Se:Hg < 1 came from seven sites: five in Oregon, one in Montana, and one in Washington. This suggests that local or regional environmental factors such as, wetland extent (37), forested regions (38), agricultural areas (39), and/or several water quality variables including pH, alkalinity, DOC, and SO₄ (40–43) might contribute to a molar surplus of Hg relative to Se. Zimmerman (36) suggested that some combination of these factors do influence the chemical burdens of fish with his finding that northern pikeminnow had significantly higher IF values in the Snake River than in the Columbia River and that stomach fullness, while significantly greater in summer than in spring in the unimpounded lower Columbia River, did not differ between seasons in the impounded reaches of the Columbia and Snake Rivers.

Mercury vs Selenium. High Hg concentrations in fish tissue from our samples were found only when Se concentrations in the same tissue were low (Figure 3). This is consistent with Belzile et al. (44) who found reduced bioaccumulation of Hg in all lake trophic levels (including young-of-the-year fish) downwind from the Sudbury, Ontario smelters. They concluded “Selenium plays an important role in limiting the whole-body assimilation of Hg at lower levels of the aquatic food chain.” Bioaccumulation differs from, but is not entirely unrelated to, Hg toxicity potential. Concentration gradients in our study are not as well-defined geographically as those at Sudbury, but our results do suggest that fish species and environmental variability influence Se: Hg molar ratios in freshwater fish.

Mercury Criteria vs Se:Hg Molar Ratios. Peterson et al. (15) estimated the proportion of stream length across the western U.S. where the total Hg in fish tissue exceeded the wildlife threshold (20) and the current MeHg WQC (21). Those estimates are accurate relative to the wildlife and human health benchmarks for Hg alone, however, they likely exaggerate Hg toxicity potentials relative to an assessment based on Se:Hg molar ratios. Considering all fish in our sample (n = 468), 56% exceeded the wildlife Hg threshold (0.1 µg Hg g⁻¹ wet wt.) (20) and 12% exceeded the MeHg WQC (0.3 µg Hg g⁻¹ wet wt.) (21). When
examined by major feeding groups, 33% of the nonpiscivores (n = 262) and 84% of the piscivores (n = 206), respectively, exceeded the wildlife Hg threshold. Five percent of the nonpiscivores and 25% of the piscivores exceeded the MeHg WQC. Based on Hg concentrations alone, a large proportion of the fish in our sample would exceed the MeHg WQC and possibly be unfit for consumption. However, if we consider that a molar ratio surplus of Se:Hg >1 in fish might be sufficient to prevent Hg toxicity in the fish and consumers of the fish (4), only 12 samples (those below the 1:1 line in Figure 3) would be considered unsuitable for wildlife consumption. By allowing for Se and Hg measurement uncertainty and applying that to the 1:1 line in Figure 3, only one more fish with a Hg molar surplus was added to the group. Thus, based on their Se:Hg molar ratios, 13 fish (2.7% of our total sample) might pose Hg toxicity problems for wildlife consumers. However, if we assess the potential toxicity of the 13 fish with Hg surplus (Hg >Se) based on the current MeHg WQC of 0.3 µg Hg · g⁻¹ wet wt., only 6 of the 13 fish have excess Se and 13 fish have a Hg > their 1:1 Se:Hg molar ratio. Thus, potential Hg toxicity in our entire fish sample might be no more than seven (1.3% of our sample). Since all of these fish are northern pikeminnows, this could be important to northwestern Native Americans because they commonly consume northern pikeminnows. Here we have compared the Hg surplus, relative to the Se:Hg ratio in whole fish to a human consumption criterion. We realize that such a comparison might not be directly pertinent to those human consumers of western U.S. fish who eat only the filet tissue. A comparison might not be directly pertinent to those human consumers. Thus, in our sample there are ~6 times more fish in the potential Se toxicity category than those in the potential Hg toxicity category.

Our finding that nearly all (97.5%) of the freshwater fish in our survey have sufficient Se to potentially protect them and their consumers against Hg toxicity suggests that consideration of Se–Hg interactions might improve our understanding of risks associated with fish tissue Hg toxicity. Several researchers (13, 19, 45, 50) recommend measuring Se concurrently with Hg in fish tissue and considering the Se–Hg interaction. The focus of future research should be on the Se protective mechanism itself, on the effects of co-occurring Se and Hg, and on establishing the Se:Hg molar ratios of whole fish versus filets in streams, lakes, and reservoirs in various geographic settings.

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shark. Luten et al. (19) reported similar results for marine fish filets. However, all of their freshwater fish species (pike, perch, and pike-perch; n = 21) exhibited Se:Hg molar ratios <1. This is in near total contrast to our results, which is not surprising, since theirs was a European study and the soils of north-central Europe and the Scandinavian countries are depleted of Se (46). This likely contributes to the high Hg levels relative to the low Se levels observed in their freshwater fish (47). Kehrig et al. (48) measured Se and Hg in hepatic and muscle tissue of four fish species in a tropical estuary. They found the Se:Hg molar ratios were >1 (5 to 70 times >) in both tissue types of all fish. Because reports of Se:Hg ratios in freshwater fish are rare and because geographic regions differ, more documentation is needed. This is particularly true for regions of the eastern U.S. and for lakes and reservoirs that might produce Se:Hg fish tissue ratios considerably different from the ones we report here for stream fish of the western U.S.

Potential Se Toxicity. Small amounts of Se are required by all cells of virtually all forms of animal life, but Se levels above certain threshold limits can be harmful. Lemly’s (49) whole body 4.0 µg Se · g⁻¹ dry wt. (1.0 µg · g⁻¹ wet wt., or 0.01267 µmol · g⁻¹ wet wt.) toxic effect threshold (TET) is the concentration at which fish experience reproductive failure and juvenile mortality. This TET is widely cited in the literature. Thus, we used this benchmark to assess Se toxicity potential in our fish sample. There are 456 fish in Figure 3 that have a Se:Hg molar ratio >1. Presumably, all of these fish are protected against Hg toxicity. However, there are 68, or 15% of the 456 fish that have Se concentrations that exceed the Lemly (49) TET of 1.0 µg Se · g⁻¹ wet wt. above the 1:1 line. This raises potential selenium toxicity (selenosis) concerns for those fish and their consumers. Thus, in our sample there are ~6 times more fish in the potential Se toxicity category than those in the potential Hg toxicity category.
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Supporting Information Available
Table S1 lists all fish groups analyzed, their mercury and selenium concentrations and the selenium surpluses for each group. Additionally, details of the fish tissue sample preparation method and the selenium neutron activation analysis are described. This material is available free of charge via the Internet at http://pubs.acs.org.

Literature Cited


