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Ecological Aspects of Mercury–Selenium Interactions in the Marine Environment

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In the marine environment, mercury is accompanied by selenium in all investigated species of mammals, birds, and fish—possibly due to a normal homeostatic regulation. It therefore seems likely that selenium will exert its protective action against mercury toxicity in the marine environment, decreasing its detrimental effects on reproduction, behavior, growth, etc. of the organisms and thus protect the population and ecosystem. On the other hand, the increased retention of mercury caused by selenium may lead to a higher level of biomagnification in the food chain and a higher body burden in the individual. This might counteract the positive effect of decreased intoxication.

It is well known that the heavy metals which are large and have high polarizability and several easily excited outer electrons form strong covalent bonds with the "soft bases," i.e., elements of large size with accessible low lying empty orbitals like sulfur and selenium.

Protective action against heavy metal toxicity is exhibited by sulfhydryl compounds, and BAL, a dithiol, is used against metal poisoning.

Experimental Data for Various Types of Organisms

A strong correlation between selenium and mercury has been found in marine mammals and fish like tuna and swordfish, and several workers have demonstrated that selenium protects against both inorganic and organic mercury and also other heavy metals in experimental animals. The first report on the protective effect of selenite against mercury toxicity appeared in 1967 (*1*).

Koeman et al. (*2*) determined the content of mercury and selenium and some other elements in marine organisms. The results showed that in the liver and also in the brains of seals, dolphins, and porpoises from all over the world, the mercury concentration is strongly correlated with that of selenium, but not with any of the other elements analyzed (Cd, As, Zn, Sb). Moreover, it is indicated that the increase in mercury and selenium concentrations occurs in a 1:1 molecular ratio.

In fish and marine birds, most of the mercury is present in the form of methylmercury. This does not seem to be the case with, for example, seals, contrary to what might be expected for an animal at the end of the food chain. In adult and juvenile common seals, 2–14% and 16–78%, respectively, of the mercury in liver and brain was found to be in the form of methylmercury. Porpoises were found to have a low portion of methylmercury in liver and brain tissues, whereas muscle tissue contained methylmercury only. In all subcellular fractions analyzed of tissues from different organs of a seal the mercury/selenium ratio was found to approximate the 1:1 molecular ratio. Herring and mackerel, which are important feeding organisms for marine mammals, were analyzed, and the selenium to mercury ratio appears to be approximately 16:1. The conclusion is derived that the 1:1 ratio in marine mammals is established within the organisms themselves.

Also some marine fish-eating birds, guillemots and a razor bill, were analyzed, and no clearcut correlation between selenium and mercury could be established. The selenium level was higher than the mercury level in all organs, and the mercury concentrations were low compared to those found in seals of comparable age.

Ganther et al. (*3*) analyzed tuna and found the Hg/Se ratio to be 1:15 and 2:3, respectively, in fish with low and high mercury levels (0.2 and 2.9 mg/kg). The increment in mercury content between low- and high-Hg tuna was in an approximate 1:1 molar ratio with the increment in Se.

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Not very much information as to the Hg/Se ratios in fresh water organisms seems to be available at present. Experiments with mink fed mercury-contaminated fresh water fish [pike containing almost 6 mg methylmercury and 0.2 mg selenium/kg (*4*)] with a Se/Hg ratio of 0.13:1 have been performed (*5*). The content of mercury and methylmercury increased with the time of exposure, while selenium remained constant or increased only slightly. The ratios of selenium to mercury on a molar basis were 0.7:1 for liver after 25 days of exposure and 0.3:1 after 100 days. For kidney the ratios were 0.5:1 and 0.3:1, respectively. The low and decreasing Se/Hg ratios in mink liver and kidney might reflect the low selenium content in the pike diet rather than a different mechanism for the accumulation of the elements in mink as compared to marine mammals.

The correlation between the two elements in humans was investigated by Kosta, Byrne, and Zelenko (*6*). Approximately a 1:1 molar ratio in those organs which accumulate and retain mercury strongly, namely, thyroid, pituitary, and kidney, was found in humans following long-term exposure to high levels of inorganic mercury. Since selenium will normally be present in at least typical physiological levels whereas mercury levels in nonexposed persons should be insignificant, a molar ratio will only be observed for rather elevated levels of mercury. The ratio of the increments over normal levels approached the molar ratio in many cases.

In vitro studies of the binding of methylmercury by selenocysteamine were performed by Sugiura and co-workers (*7*). The selenohydryl group was found to have a high affinity for mercury in comparison to sulfhydryl and amino groups. A selenocysteamine–methylmercury complex was isolated. This supports the theory of a direct Hg–Se linkage. A high and enhanced tissue retention of both selenium and mercury has been observed by many workers (*6, 8–10*). According to Kosta, Byrne, and Zelenko, this suggests their removal from biological turnover.

The antagonistic interrelationship between selenium and mercury has been established in many studies. Ohi and co-workers (*11*) found a protective effect of selenite on the toxicity of methylmercury in rats regarding both growth rate and mortality as did Stillings et al. (*9*), and Ganther et al. (*3*), and Parizek et al. (*12*).

Experiments with Japanese quail fed diets containing methylmercury and selenium have been performed by Ganther et al. (*3*), Ganther and Sunde (*8*), and Stoewsand, Bache, and Lisk (*10*). Stoewsand, Bache, and Lisk found that an addition of 5 mg/kg Se to a diet containing 20 mg/kg Hg as MeHgCl (Hg/Se ratio 1:0.75) protected against intoxication when fed simultaneously. A dietary pretreatment with selenium or selenium + methylmercury before the addition of methylmercury delayed the onset of intoxication, but after 9 weeks the mortality was as high as when no selenium was administered. No correlation between the levels of methylmercury in the organs or in produced eggs with mercury intoxication was found. The results of the long-term studies by Ganther et al. indicated that the growth, survival and reproductive performance of Japanese quail fed tuna diets containing 1 mg/kg of mercury or less were comparable to those of control birds fed a corn-soy diet devoid of mercury. At the 1 mg/kg Hg level the Hg/Se ratio on a molecular basis was approx. 1:5. A dietary mercury level of 10 mg/kg (Hg/Se ratio 7:1) was not tolerated.

The mechanisms of the antagonistic interactions between selenium and mercury are not fully understood. One obviously likely mechanism is the formation of a Hg-Se compound of low biological availability and activity. Selenium affecting the activities of enzymes cleaving the carbon-mercury bond in organic mercury compounds may be another mechanism contributing to a decrease in organic mercury toxicity. Fang (*13*) found an enhanced activity of PMA cleavage enzymes in rat liver from rats supplemented with 0.5 mg/kg or 5 mg/kg sodium selenite in the drinking water. This increase was dose dependent. Svenson and van de Ven (unpublished data) studied the breakdown of methylmercury in seal liver homogenates and found some indications of selenium interaction with the breakdown.

Conclusions and Tentative Implications for the Marine Environment

The 1:1 molar ratio of the mercury and selenium increment in various types of organisms suggests a direct Hg–Se linkage. This coaccumulation could be thought of as a result of a compensation by the organism for the depletion of the physiologically essential levels of selenium as mercury is accumulated and linked to the selenium already present—a normal homeostatic regulation.

The ability of selenium compounds to decrease the toxic action of both organic and inorganic mercury in experimental animals has been established beyond doubt.

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birds, and fish. It therefore seems likely that selenium will exert its protective action against mercury toxicity in the marine environment, decreasing its detrimental effects on reproduction, behavior, and growth of the organisms and thus protect the population and ecosystem.

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