

SELENIUM - FATE AND EFFECTS IN THE AQUATIC ENVIRONMENT

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ABSTRACT

Selenium has a complex and not fully understood biogeochemistry in the aquatic environment as well as an unusual mode of toxicity (acute via water column exposure; chronic via food chain exposure). It has the narrowest range between nutritional requirements and toxicity of any essential element, and chronic toxicity is not readily predictable. Selenium contamination of waters or even of tissues does not necessarily indicate a problem; there are no generally accepted or universally accepted threshold values for chronic toxicity. Assessing risk must be done site-specifically in a risk assessment framework, focusing on reproductive effects to sensitive exposed fish and waterfowl and on "worst case" hydrologic units. Provided the necessary investigative and monitoring studies are done, any potential selenium problems can be addressed.

INTRODUCTION

Selenium is assuming increasing importance (Renner, 1998) due to increased evidence for selenium contamination of the environment coupled with the fact that selenium is one of the few environmental contaminants that can accumulate through the food chain to cause reproductive effects - to fish and waterfowl. However, environmental issues related to selenium are complex and not universally understood. The purpose of this paper is to briefly review the state of knowledge related to selenium in the aquatic environment.

SELENIUM FACTS

Selenium is a naturally occurring metalloid discovered in 1818 and named after Selene, the Greek goddess of the moon. Selenium enters the aquatic environment as a result of both natural and anthropogenic sources, including: natural weathering; agricultural drainage; combustion of fossil fuels; coal mining; fly ash leaching; sulphide ore mining (e.g., copper, lead/zinc mines); phosphate mining; and, production of animal feed supplements. Selenium used in commerce is a by-product of precious metals recovery from electrolytic copper refinery slimes. Major uses of selenium include, in decreasing importance: photoreceptors/semiconductors; glass; pigments; metallurgical; agricultural/biological.

Selenium was first identified as a naturally occurring poison of range stock in the northern United States (Franke, 1934), over 20 years before it was identified as an element essential for animal health (Schwarz and Foltz, 1957). The difference between its role as a poison and its role as a nutrient is very small, about a factor of 10; it tends to be beneficial in food at concentrations below about 1 $\mu\text{g Se/g}$, and a toxicant at concentrations above about 5 $\mu\text{g Se/g}$ (Oldfield, 1995).

Oldfield (1995) documents world-wide geological differences resulting in some soils which are selenium deficient and some soils which are selenium enriched. Selenium deficiencies in soil, if not rectified, can result in reduced growth and health of livestock (e.g.: Andrews et al., 1968). Humans living in selenium-deficient areas can also be adversely affected by a lack of selenium in their diet (e.g.: Serbo-Croatia - Maksimovic et al., 1992; China - Ge and Yang, 1993; Africa - Vanderpas et al., 1990; Tibet - Moreno-Reyes et al., 1998). In contrast, soils that are naturally enriched in selenium favour the growth of selenium-accumulating plants. Grazers eating such plants can experience a variety of disorders including death. For instance, there is an area in Queensland, Australia, known as the "Poison Strip" (McCray and Nurwood, 1963). Marco Polo, during his 13th century overland trips to China, encountered such an area. His diaries document his horses reluctantly eating the local range plants (there was nothing else to eat), which resulted in clear symptoms of selenium toxicity - their hooves cracked and fell off before they died (Oldfield, 1995). Similarly, the deaths of cavalry horses in Missouri in the 1850s have been linked to selenium poisoning (Trelease and Beath, 1949).

Although there are various natural "poison strips" around the world, the problem most agricultural and livestock farmers face related to selenium is that of deficiency (not to the plants which seem able to accumulate selenoamino acids without harm, but to animals eating those plants). Thus, selenium is used routinely world wide as a supplement in farming, resulting in two of the above noted sources of selenium to the aquatic environment: agricultural drainage and animal feed supplements.

Selenium can occur in aquatic environments in five different states or forms, sometimes simultaneously. Selenite [Se (IV)] is the most toxic and bioavailable inorganic form, and is associated with anthropogenic sources. Selenate [Se (VI)] is less toxic and bioavailable than selenite and is associated with natural sources. Elemental selenium [Se (0)] is often found in sediments, generally as a result of microbial reduction. Selenocyanate (SeCN⁻) is common in some industrial wastes; its biogeochemistry is unknown. Reduced selenium [Se -(II)] consists primarily of hydrogen selenide and dimethyl selenium, both of which are volatile, and organic selenium. Organic selenium includes selenoamino acids (e.g., selenomethionine, selenocysteine), selenoproteins, and methylated selenides. The organic selenium forms

are the most ecologically relevant in terms of food chain effects; primary producers and bacteria bioconcentrate inorganic selenium from water or sediment and transform it into selenoamino acids that are incorporated into selenoproteins (Malchow et al., 1995). However, little is known about the organic forms of selenium and analytical procedures are generally lacking.

Selenium toxicity occurs when excess selenium substitutes for sulphur in sulphur-containing amino acids (cysteine and methionine). Selenium acts differently than sulphur, thus selenoamino acids alter the normal function of the proteins into which they are incorporated. This is particularly a problem in rapidly growing fish and waterfowl embryos when the egg yolks are enriched in selenoamino acids.

MEASURING SELENIUM IN THE AQUATIC ENVIRONMENT

Water quality values are generally based on total selenium. For instance, U.S. EPA (1987) sets a freshwater acute criterion value of 20 µg/L. This value has been challenged by Canton (1999), who suggests a conservative acute criterion of 220 µg/L. U.S. EPA (1999) also sets a freshwater chronic criterion value of 5 µg/L, which is presently under review (U.S. EPA, 1998). On the Canadian side of the border, the criterion value becomes a guideline value and drops to 1 µg/L (CCME, 1999).

Both total and dissolved (0.45 µm filtered) selenium are routinely measured in water and effluent samples. Measurement of the dissolved fraction is not as useful as for some other contaminants; binding to particulates does not necessarily reduce bioavailability via food. All analytical methods are not the same. What is used as a sample preservative in the case of one method can be an interferent in the case of another method. Method detection limits are method-dependent and, in some cases, can be well above the above noted ambient water quality values.

Selenium speciation in water or effluent is generally determined operationally based on differences between measured concentrations of Se (IV), inorganic, and total selenium (Cutter, 1977). Thus Se (VI) is generally determined as inorganic selenium minus Se (IV); organic selenium is generally determined as total selenium minus inorganic selenium. Such indirect methods of analyses are not particularly sensitive: inaccuracies in any one measure cascade down to others; matrix differences can result in over- and under-estimates for any or all measurements; various substances can interfere with the analyses (e.g., chloride, nitrite, dissolved organic carbon, transition metals).

Selenium in tissues is generally measured as total selenium. As noted previously, this is not the correct form of selenium related to possible chronic toxicity via the food chain. However, there presently is no dependable analytical technique to measure protein-bound selenium, the form most closely associated with chronic effects.

SELENIUM PROBLEMS IN THE AQUATIC ENVIRONMENT

Although natural terrestrial poisoning has been recognised for over 65 years (Franke, 1934), it is only in the last 20 years that selenium has also been recognised as an environmental problem in the aquatic environment (Frankenberg and Engberg, 1998). Two notable incidents brought this metalloid to the attention not only of the scientific community but also of the general public, and resulted in a lowering of the selenium aquatic criteria/guideline values.

In the late 1970s selenium from coal ash was reported to affect reproduction of fish populations in Belews Lake, North Carolina, at concentrations as low as 10 µg/L (Lemly, 1985). In the mid-1980s, selenium in agricultural drainage was implicated in extensive poisoning of fish and waterfowl at the Kesterton National Wildlife Refuge in California (Hoffman et al., 1988; Ohlendorf and Santolo, 1994). In both the above cases, and in all similar subsequent cases, the cause appeared to be the formation of organic selenium compounds. These then moved up the food chain to fish and waterfowl, resulting in selenium transfer to their eggs and subsequent teratogenesis and reduced hatching success (Maier and Knight, 1994).

Selenium is a regulatory problem, for several reasons. First, it has a complex biogeochemistry in the aquatic environment; it can exist in and transform between several oxidation states, each with varying bioavailability and toxicity. Second, the most significant route of exposure is through the diet. Third, because water quality criteria/guideline values are normally derived based on exposure via the water medium, these are not directly applicable to this unusual exposure route (U.S. EPA, 1998). Fourth, site-specific differences can be major (cf. Ohlendorf, 1999). Fifth and as noted previously, there is a very narrow tolerance between nutritional requirements and toxicity. And, finally, there is a relatively high level of analytical variability and difficulties.

Site-specific differences in selenium bioavailability and toxicity occur for several reasons (Maier and Knight, 1994; Malchow et al., 1995). First, selenium has a complex biogeochemistry. Second, there are differences in exposure pathways (Chapman, 1999). Third, there are differences in species sensitivities

including adaptation (older organisms tend to be more resistant). Fourth, lotic (flowing water) systems do not convert Se (IV) and Se (VI) to the more toxic organo-selenium forms as actively as lentic (standing water) systems (Lemly, 1999a). Fifth, warm water lentic systems seem to be most at risk (DeForest et al., 1999). Sixth, some commercial laboratories have difficulty analysing for total or dissolved selenium at concentrations <10 µg/L.

Lentic (standing water) systems are more at risk of selenium poisoning than lotic (flowing water) systems, again for several reasons. First, lentic systems often contain distinct anaerobic and organically rich zones favouring the formation of organic selenium by bacteria, algae and phytoplankton. Second, residence times are higher in lentic systems. Transformation of inorganic to organic selenium, if it occurs, is faster than in lotic systems; the static nature of lentic systems provides an optimum medium for cycling of organic selenium. Third, there is greater accumulation in sediments, which are both a sink and a source for toxic organic forms of selenium. Fourth, reduced selenium forms tend to predominate. Fifth, the exposure of organisms is greater because of the confined nature of many lentic systems and the fact that many lentic organisms have a small, extensively used home feeding range.

However, all lentic systems (e.g., reservoirs and lakes) are not the same (Ohlendorf, 1999). Not all act as sinks; there have been reports of cases where most of the selenium entering a lentic system from upstream sources continues flowing downstream (U.S. EPA, 1998). But the reality that lotic and lentic systems are interconnected cannot be ignored; these systems cannot be considered in isolation. For instance, backwater areas of streams function as feeding and nursery areas. And streams tend to flow into lakes/wetlands.

Interestingly, although selenium can be a toxicant, it can also reduce the effects of other toxicants. For instance, selenium can reduce bioavailability and toxicity of mercury (e.g.: Nuutinen and Kukkonen, 1998) and has been used for such purposes in Scandinavia. Conversely, other toxicants such as arsenic (Stanley et al., 1994) can reduce selenium bioavailability and toxicity. A variety of interactions are possible; the consequences of most are unknown.

SELENIUM EFFECTS ON WATERFOWL AND FISH

Selenium in aquatic ecosystems can affect waterfowl, resulting in: reduced hatchability of fertile eggs; a high incidence of embryo/chick abnormalities; and, a failure of the adults to breed. In addition, adult waterfowl can demonstrate feather loss and delayed molt, weight loss, tissue lesions, and blood and tissue

chemistry alterations. There have been no documented adverse effects of excess selenium in aquatic systems on terrestrial birds outside the laboratory. Waterfowl differ in their sensitivity to selenium: mallard ducks tend to be the most sensitive; stilts and killdeer tend to be moderately sensitive; and, avocets and snowy plovers tend to be tolerant (Adams, 1997).

The effects of selenium on fish include abnormalities (blindness, popeye, osteological deformities), and reduced hatching success. As is the case with waterfowl, there are differences in species sensitivities (Maier and Knight, 1994; Chapman, 1999; Kennedy et al., 2000). Interesting, the same fish species are not always equally sensitive in different studies (Rob Reash, American Electric Power, pers. comm.), suggesting acclimation and/or adaptation.

Determining whether chronic effects of selenium have occurred requires determination as to whether or not reproduction is impaired. In the case of waterfowl, this often involves field studies assessing the reproductive success of marked nests together with selenium tissue concentrations in randomly selected and/or "failed" eggs. Additional measurements to assess exposure can include selenium concentrations in food chain biota or the blood of waterfowl or in livers of pre-fledging birds. In the case of fish, definitive studies generally require raising the eggs from exposed parents in the laboratory to assess hatching success and any deformities related to parental and egg selenium concentrations.

SITE-SPECIFIC SELENIUM CRITERIA

Determining site-specific aquatic criteria for selenium related to possible chronic effects requires four components. First, it is essential that the possibility of adverse effects to fish and waterfowl reproduction be adequately addressed. In this regard it must be remembered that the absence of evidence for effects is not evidence for the absence of adverse effects. Such information is, however, generally only relevant to the status quo and is not predictive for any future possible increases in selenium contamination. Predictions require two additional pieces of information, the second and third components. Second, for the ecosystem in question there must be a reasonable understanding of selenium chemistry in that system and of the factors controlling the conversion of inorganic forms to more toxic organic forms. Third, food chain (exposure) relationships must be understood. In addition, and finally, the possibility of downstream (lake, wetland) or quiescent side channel bioavailability and toxicity, must be fully assessed. Knowing food chain and exposure pathways is essential in all cases.

Relative to a risk assessment scenario, which is ultimately what is required to assess whether or not selenium in the aquatic environment is a potential problem, three separate assessment and measurement

endpoints are required. Assessment endpoints are: reproductive success of sensitive exposed waterfowl populations (e.g., mallards); reproductive success of exposed fish populations; and, aquatic invertebrate community structure and function (related to exposure and food sources). Measurement endpoints are, respectively: waterfowl nest/egg monitoring and selenium concentrations in invertebrates (diet), fish (diet) and eggs; fish egg monitoring/laboratory studies, and selenium concentrations in aquatic invertebrates (diet) and eggs; and, benthos diversity and abundance.

Determining site-specific criteria for selenium in aquatic environments must be done in the context of three key facts. First, site-specific differences exist, and small scale variability in body burdens, for instance, can be great (e.g., Malloy et al., 1999). Second, predictions are uncertain at best. Third, direct determination of any effects under "worst case" conditions is required to be protective or to trigger more realistic studies. No effects means no further actions are needed provided conditions do not change; effects under "worst case" conditions do not necessarily mean such will also occur under more realistic conditions.

Options presently available for developing site-specific selenium criteria for aquatic ecosystems, irrespective of the above, fall into three distinct categories, the first of which is modelling. For example, Van Derveer and Canton (1997) and Canton and Van Derveer (1997) developed a predictive model for the Arkansas River, Colorado, linking water and sediment quality to sedimentary total organic carbon content. This model has not been successfully applied to other freshwater bodies.

Another option exists, in the case where selenium is not bioaccumulated to potentially unsafe levels, and when further increases are not likely. In this case, site-specific criteria can be set based on existing Se (IV) and Se (VI) water column concentrations. This has been done for a site in West Virginia and another in Pennsylvania, resulting in site-specific chronic criteria in terms of total selenium of, respectively, 15.2 µg/L and 10.5 µg/L (John Goodrich-Mahoney, Electric Power Research Institute, pers. comm.).

A final option exists in the case where selenium is bioaccumulated to potentially unsafe levels and/or further increases are likely. In this case, long-term field studies or a combination of field and laboratory studies are required. Long-term field studies can involve exposing reproducing fish populations via mesocosms such as is presently being done with bluegills in West Virginia (Rob Reach, American Electric Power, pers. comm.). An example of laboratory studies is provided by Kennedy et al. (2000).

In any case, selenium monitoring/investigative studies must be aimed at answering two major questions. First, are there any adverse environmental impacts at present? Second, will there be any adverse impacts in future? Key study components need to be aimed at: determining the present health of exposed waterfowl, fish and benthos; determining selenium mobilization mechanisms; detailed mapping of the exposed watershed to determine high risk areas; monitoring of water, sediment and biota based on food-chain relationships; and, investigative/predictive studies in the laboratory and/or high risk areas.

TREATMENT OF SELENIUM CONTAMINATED WATERS

A variety of physical-chemical methods are available for treating selenium-contaminated waters, including iron precipitation and membrane separation. The former involves co-precipitation with iron salts at an acidic pH, is only effective with Se (IV) and will not reduce selenium concentrations below existing criteria/guideline values. However, the most promising techniques appear to be biological, including the use of sulphate reducing bacteria, bioreactors (Owens, 1998) and wetlands. Wetlands seem to be the most effective technique but are also controversial. Lemly (1999b) considers wetlands treating selenium to be a potential problem. However, others believe that such wetlands provide both a reasonable and cost-effective approach, and one which presently provides the greatest overall reduction in selenium levels (Hansen et al., 1998; Knight et al., 1999). There is evidence that biological selenium volatilization is a significant pathway for selenium removal in such treatment wetlands (Hansen et al., 1998; Fan et al., 1998; Ansedde et al., 1999). There is also evidence that permanent flooding can at least partially immobilize (Home, 1991), as can converting wetlands to grassland (Wu et al., 1995). Further, addition of nitrate (e.g., as fertilizer) may help control the oxidation and mobilization of selenium in fresh waters (Wright, 1999). However, accumulation of selenium in wetlands has resulted in deformities or other effects on young of waterfowl using these areas; (Lemly, 1999b). Management actions to deal with such issues have involved obviating the attractiveness of the wetlands to waterfowl (e.g., steep banks, no extended shorelines, minimal open water) and/or minimizing food chain exposures (e.g., excluding fish, maximizing plants that are a poor food source for waterfowl). Whether such management actions are sufficient and in fact the whole philosophy of using wetlands to treat selenium contaminated waters remains a topic of active debate (Lemly, 1999b).

SUMMARY

Selenium water column concentrations serve as triggers for further investigation, not as pass/fail decision criteria; they indicate hazard, not risk. Selenium contamination per se does not necessarily indicate a problem.

Similarly, selenium body burden concentrations only indicate hazard, not risk. There presently are no generally accepted or universally applicable tissue residue threshold values which unambiguously indicate that chronic toxicity will occur. Values available in the literature are not generally applicable to other species or necessarily to the same species in different situations. Moreover, it is not presently possible to make definitive predictions based on tissue residues in food organisms.

Assessing the risk posed by selenium in the aquatic environment requires site-specific studies, in a risk assessment framework. The primary focus should be on reproductive effects to exposed fish and waterfowl, and on "worst case" hydrological units (Lemly, 1998, 1999a).

REFERENCES

- Adams, W. J. 1997. Understanding selenium in the: aquatic environment. A symposium held in Salt Lake City, Utah, March 6-7, 1997. Kennecott Utah Copper, Salt Lake City, UT, USA.
- Andrews, E. D., W. J. Hartley, and A. B. Grant. 1968. Selenium-responsive diseases of animals in New Zealand. *N. Z. Vet. J.* 16:3-17.
- Ansede, J. H., P. J. Pellechia, and D. C. Yoch. 1999. Selenium biotransformation by the salt marsh cordgrass *Spartina alterniflora*: Evidence for diimethylselenoniopropionate formation. *Environ. Sci. Technol.* 33:2064-2069.
- Canton, S. P. 1999. Acute aquatic life criteria for selenium. *Environ. Toxicol. Chem.* 18:1425-1432.
- Canton, S. P., and W. D. Van Derveer. 1997. Selenium toxicity to aquatic life: An argument for a sediment-based water quality criteria. *Environ. Toxicol. Chem.* 16:1255-1259.
- CCME. 1999. Canadian environmental quality guidelines. Canadian Council of Ministers of the Environment, Winnipeg, MB, Canada.
- Chapman, P. M. 1999. Selenium - A potential time bomb or just another contaminant? *Human Ecol Risk Assess.* 5:1123-1138
- Cutter, 1977. Species determination of selenium in natural waters. *J. Analytica ChimicaActa* 98:59-66.
- Fan, T. W. M., R. M. Higashi, and A. N. Lane. 1998. Biotransformations of selenium oxyanion by filamentous cyanophyte-dominated mat cultured from agricultural waters. *Environ. Sci. Technol.* 32:3185-3193.
- Franke, K. W. 1934. A new toxicant occurring naturally in certain samples of plant foodstuffs. 1. Results obtained in preliminary feeding trials. *J. Nutr.* 8:597-608.

Frankenberger, W. T. Jr., and R. A. Engberg. 1998. *Environmental Chemistry of Selenium*. Marcel Dekker, Inc. New York, NY, U.S.A.

Ge, K., and G. Yang. 1993. The epidemiology of selenium deficiency in the etiological study of endemic diseases in China. *Am. J. Clin. Nutr.* 57:259S-263S.

Hansen, D., P. J. Duda, A. Zayed, and N. Terry. 1998. Selenium removal by constructed wetlands: role of biological volatilization. *Environ. Sci. Technol.* 32:591-597.

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

Horne, A. J. 1991. Selenium detoxification in wetlands by permanent flooding: I. Effects on a macroalga, an epiphytic herbivore, and an invertebrate predator in the long-term mesocosm experiment at Kesterton Reservoir, California. *Water Air Soil Pollut.* 57/58:43-52.

Kennedy, C. J., L. E. McDonald, R. Loveridge, and M. M. Strosher. 2000. The effects of selenium contamination in adult cutthroat trout (*Oncorhynchus clarki lewisi*) on eggs, larvae and fry. *Arch Environ. Contam. Toxicol.* (In Press).

Knight, R. L., R. H. Kadlec, and H. M. Ohlendorf. 1999. The use of treatment wetlands for petroleum industry effluents. *Environ. Sci. Technol.* 33:973-980.

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

Lemly, A. D. 1993. Guidelines for evaluating selenium data from aquatic monitoring and assessment studies. *Environ. Monit. Assess.* 28:83-100.

Lemly, A. D. 1998. A position paper on selenium in ecotoxicology: a procedure for deriving site-specific water quality criteria. *Ecotox. Environ. Safety* 39:1-9.

Lemly, A. D. 1999a. Selenium transport and bioaccumulation in aquatic systems: a proposal for water quality criteria based on hydrological units. *Ecotox. Environ. Safety* 42:150-156.

Lemly, A. D. 1999b. Selenium impacts on fish: An insidious time bomb. *Human Ecol. Risk Assess.* 5:1123-1138

Maier, K. J., and A. W. Knight. 1994. Ecotoxicology of selenium in freshwater systems. *Rev. Environ. Contam. Toxicol.* 134:31-49.

Maksimovic, Z., V. Jovic, S. Djujic, and M. Rsumovic. 1992. Selenium deficiency in Yugoslavia and possible effects on health. *Env. Geochem. Health* 14:107-111.

- Malchow, D. E., A. W. Knight, and K. J. Maier. 1995. Bioaccumulation and toxicity of selenium in *Chironomus decorus* larvae fed a diet of seleniferous *Selenastrum capricornutum*. *Arch. Environ. Contam. Toxicol.* 29:104-109.
- Malloy, J. C., M. L. Meade, and E. W. Olsen. 1999. Small-scale spatial variation in selenium concentrations in chironomid larvae. *Bull. Environ. Contam. Toxicol.* 62:122-129.
- McRay, C. W. R., and I. D. Nurwood. 1963. Selenosis in northwestern Queensland associated with a marine cretaceous formation. *Queensland J. Agric. Sci* 20:475-498.
- Moreno-Reyes, R., C. Suetens, F. Matthieu, F. Begaux, D. Zhu, M. T. Rivera, M. Boelaert, J. Neve, N. Perlmutter, and J. Vanderpas. 1998. Kashin-Beck osteoarthropathy in rural Tibet in relation to selenium and iodine status. *New Engl. J. Med.* 339:1112-1120.
- Nuutinen, S., and J. V. K. Kukkonen. 1998. The effect of selenium and organic material in lake sediments on the bioaccumulation of methylmercury by *Lumbriculus variegatus* (Oligochaeta). *Biogeochem.* 40:267-278.
- Ohlendorf, H. M., and G. M. Santolo. 1994. Kesterton Reservoir - past, present and future: An ecological risk assessment. In: *Selenium in the Environment*, pp. 69-118. (Frankenberg, W. T. Jr. and S. Benson, Eds.). Marcel Dekker, Inc. New York, NY, U.S.A.
- Oldfield, J. E. 1995. Selenium in maps. *Bull. Selenium-Tellurium Dev. Assoc.*, April 1995, 1-7.
- Owens, L. P. 1998. Bioreactors in removing selenium from agricultural drainage water. In: *Environmental Chemistry of Selenium*, pp. 501-514. (Frankenberg, W. T. Jr. and R. A. Engberg, Eds.). Marcel Dekker, Inc. New York, NY, U.S.A.
- Renner, R. 1998. EPA decision to revise selenium standard stirs debate. *Environ. Sci. Technol.* 32:350A.
- Schwarz, K., and C. M. Foltz. 1957. Selenium as an integral part of Factor 3 against dietary necrotic liver degeneration. *J. Am. Chem. Soc.* 79:3292-3293.
- Stanley, T. R. Jr., J. W. Gann, G. J. Smith, and R. Rosscoe. 1994. Main and interactive effects of arsenic and selenium on mallard reproduction and duckling growth and survival. *Arch. Environ. Contam. Toxicol.* 26:444-451.
- Trelease, S. F., and O. A. Beath. 1949. *Selenium: Its Geological Occurrence and Its Biological Effects in Relation to Botany, Chemistry, Agriculture, Nutrition, and Medicine*. Champlain Printers. Burlington, VT, U.S.A.
- U.S. EPA. 1987. Ambient water quality criteria for selenium - 1987. PB88-142-237. U.S. Environmental Protection Agency, Washington, DC, U.S.A.
- U.S. EPA. 1998. Report on the peer consultation workshop on selenium aquatic toxicity and bioaccumulation. EPA-822-R-98-007. U.S. Environmental Protection Agency, Washington, DC, U.S.A.

U.S. EPA. 1999. National recommended water quality criteria. EPA 833/Z-99-001. U.S. Environmental Protection Agency, Washington, DC, U.S.A.

Vanderpas, J., B. Contempre, N. L. Dual, N. Perlmutter, M. Boelaert, and C. Mathieu. 1990. Iodine and selenium deficiency associated with cretinism in northern Zaire. *Am. J. Clin. Nutr.* 52:1087-1093.

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

Wright, W. G. 1999. Oxidation and mobilization of selenium by nitrate in irrigation drainage. *J. Environ. Qual.* 28:1182-1187.

Wu, L., J. Chen, K. K. Tanji, and G. S. Banuelos. 1995. Distribution and biomagnification of selenium in a restored upland grassland contaminated by selenium from agricultural drain water. *Environ. Toxicol. Chem.* 14:733-742.