ENVIRONMENTAL CONTAMINANTS ENCYCLOPEDIA

SELENIUM ENTRY

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Like a library or many large databases (such as EPA's national STORET water quality database), this document contains information of variable quality from very diverse sources. In compiling this document, mistakes were found in peer reviewed journal articles, as well as in databases with relatively elaborate quality control mechanisms [366,649,940]. A few of these were caught and marked with a "[sic]" notation, but undoubtedly others slipped through. The [sic] notation was inserted by the editors to indicate information or spelling that seemed wrong or misleading, but which was nevertheless cited verbatim rather than arbitrarily changing what the author said.

Most likely additional transcription errors and typos have been added in some of our efforts. Furthermore, with such complex subject matter, it is not always easy to determine what is correct and what is incorrect, especially with the "experts" often disagreeing. It is not uncommon in scientific research for two different researchers to come up with different results which lead them to different conclusions. In compiling the Encyclopedia, the editors did not try to resolve such conflicts, but rather simply reported it all.

It should be kept in mind that data comparability is a major problem in environmental toxicology since laboratory and field methods are constantly changing and since there are so many different "standard methods" published by EPA, other federal agencies, state agencies, and various private groups. What some laboratory and field investigators actually do for standard operating practice is often a unique combination of various standard protocols and impromptu "improvements." In fact, the interagency task force on water methods concluded that [1014]:

> It is the exception rather than the rule that water-quality monitoring data from different programs or time periods can be compared on a scientifically sound basis, and that...
>
> No nationally accepted standard definitions exist for water quality parameters. The different organizations may collect data using identical or standard methods, but identify them by different names, or use the same names for data collected by different methods [1014].

Differences in field and laboratory methods are also major issues related to (the lack of) data comparability from media other than water: soil, sediments, tissues, and air.

In spite of numerous problems and complexities, knowledge is often power in decisions related to chemical contamination. It is therefore often helpful to be aware of a broad universe of conflicting results or conflicting expert opinions rather than having a portion of this information arbitrarily censored by someone else. Frequently one wants to know of the existence of information, even if one later decides not to use it for a particular application. Many would like to see a high percentage of the information available and decide for themselves what to throw out, partly because they don't want to seem uniformed or be caught by surprise by potentially important information. They are in a better position if they can say: "I knew about that data, assessed it based on the following quality assurance criteria, and decided not to use it for this application." This is especially true for users near the end of long decision processes, such as hazardous site cleanups, lengthy ecological risk assessments, or complex natural resource damage assessments.

For some categories, the editors found no information and inserted the phrase "no information found." This does not necessarily mean that no information exists; it

simply means that during our efforts, the editors found none. For many topics, there is probably information "out there" that is not in the Encyclopedia. The more time that passes without encyclopedia updates (none are planned at the moment), the more true this statement will become. Still, the Encyclopedia is unique in that it contains broad ecotoxicology information from more sources than many other reference documents. No updates of this document are currently planned. However, it is hoped that most of the information in the encyclopedia will be useful for some time to come even without updates, just as one can still find information in the 1972 EPA Blue Book [12] that does not seem well summarized anywhere else.

Although the editors of this document have done their best in the limited time available to insure accuracy of quotes or summaries as being "what the original author said," the proposed interagency funding of a bigger project with more elaborate peer review and quality control steps never materialized.

> The bottom line: The editors hope users find this document useful, but don't expect or depend on perfection herein. Neither the U.S. Government nor the National Park Service make any claims that this document is free of mistakes.

The following is one chemical topic entry (one file among 118). Before utilizing this entry, the reader is strongly encouraged to read the README file (in this subdirectory) for an introduction, an explanation of how to use this document in general, an explanation of how to search for power key section headings, an explanation of the organization of each entry, an information quality discussion, a discussion of copyright issues, and a listing of other entries (other topics) covered.

See the separate file entitled REFERENC for the identity of numbered references in brackets.

HOW TO CITE THIS DOCUMENT: As mentioned above, for critical applications it is better to obtain and cite the original publication after first verifying various data quality assurance concerns. For more routine applications, this document may be cited as:

> **Irwin, R.J., M. VanMouwerik, L. Stevens, M.D. Seese**, and **W. Basham.** 1997. Environmental Contaminants Encyclopedia. National Park Service, Water Resources Division, Fort Collins, Colorado. Distributed within the Federal Government as an Electronic Document (Projected public availability

on the internet or NTIS: 1998).

Selenium (Se, CAS number 7782-49-2)

NOTE: This entry contains information on both elemental selenium and selenium compounds.

Brief Introduction:

Br.Class: General Introduction and Classification Information:

Selenium is a naturally occurring, solid substance [953]. It is widely, but unevenly, distributed in the earth's crust. It is commonly found in rocks and soil. Selenium, in its pure form of metallic gray to black hexagonal crystals, is often referred to as elemental selenium or selenium dust. However, in the environment, selenium is not often found in its pure form. It is usually combined in rocks with other substances, such as sulfide minerals, silver, copper, lead, and nickel minerals [953].

Selenium chemistry is complex, as selenium can be in several oxidation states. The most important oxidation states are + 4 and + 6 [291]. The chemical reactions of selenium resemble those of sulfur and are typically nonmetallic in nature [291].

Selenium is a nonmetallic element [190,492] which has tended to be a bigger environmental problem for fish and wildlife in the Western United States, where selenium is more abundant and more bioavailable (mostly due to natural geological circumstances), than in the eastern United States [463]. Exceptions in the Eastern U.S. tend to be circumstances where there is highly contaminated runoff from coal-fired power plant ash piles or other unusual scenarios.

NOTE: Other references refer to selenium as "metallic" [953,672,697,940] or "semimetallic" [445]).

Selenium is an essential nutrient for humans and animals, and both can use inorganic as well as organic selenium compounds [953]. In the body, selenium helps prevent damage to tissues done by oxygen. Selenium, however, harms people and animals when consumed in amounts not much higher than those needed for good nutrition [953]. See the below Br.Haz section for a detailed discussion of beneficial vs. detrimental effects of selenium.

Selenium is listed by the Environmental Protection Agency as one of 129 priority pollutants [58]. Selenium is a toxic pollutant designated pursuant to section 307(a)(1)

of the Clean Water Act and is subject to effluent limitations [366, 40 CFR 401.15 (7/1/87)]. Selenium received special attention in studies of subsurface agricultural irrigation drainage waters of the San Joaquin Valley of California because it was determined to be a "substance of definite concern" [445].

Br.Haz: General Hazard/Toxicity Summary:

Selenium is one of the most fascinating and challenging of all elements involved in poisoning of fish [488]. In spite of its useful functions at very low concentrations, selenium has many toxic impacts upon fish and wildlife at high concentrations [37].

Plant and animal uptake and accumulation of selenium from the environment is influenced by, among other factors: the species of plant or animal; the concentrations and chemical forms of selenium; the medium in which it/they occur(s) (e.g., water and/or diet); the period of exposure; and, in water, the chemical and other characteristics of the water (e.g., dissolved oxygen content, hardness, pH, redox state, salinity, and temperature), including the presence of other chemicals (e.g., sulfate or cadmium) [445]. Hardness is thought to be an important co-factor related to toxicity and bioavailability of many metals, but alkalinity is sometimes a more important co-factor than is generally realized (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

A look at the detailed information presented below reveals that selenium impacts have most often been documented for birds, fish, and wildlife, while severe impacts have rarely been documented for effects on humans. As detailed further below, selenium in the correct (low) doses and forms has many benefits to humans, including its role as part of glutathione peroxidase, an enzyme involved in cellular defense against oxidative damage [893].

Although concentrations of trace amounts of the essential element selenium is desirable, excess levels of selenium are more poisonous than either arsenic or mercury [488]. Excess selenium, even as low as 3-8 ppb in the water) can cause numerous life-threatening changes in feral fresh water fish [488].

Assessment of risk of selenium toxicity to aquatic organisms is complicated by the differential toxicity of different selenium species and by biogeochemical transformations of selenium species (see detailed discussion in the Forms/Preparations/Formulations section

below under the subheading "Common Forms of Selenium") [481].

> Text in paragraph above reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, J.M. Besser, T.J. Canfield and T.W. La Point, "Bioaccumulation of organic and inorganic selenium in a laboratory food chain." Copyright 1993 SETAC].

The toxic potential and availability of selenium compounds is related to chemical form, and most importantly, solubility. Like sulfates, selenates (Se +6) are relatively soluble and are readily taken up by biologic systems [491]. By contrast, selenites (Se +4), selenides (Se -2), and elemental selenium are relatively insoluble. Selenides are so insoluble that in vivo formation of mercury selenide by dietary administration of selenite has been proposed as a method of detoxification of methyl mercury [491].

Although not true in all cases, the relative toxicity of various chemical forms of selenium is generally as follows (from most to least toxic): hydrogen selenide ~ selenomethionine (in diet) > selenite ~ selenomethionine (in water) > selenate > elemental selenium ~ metal selenides ~ methylated selenium compounds [445].

Selenium is a striking example of a contaminant which is predominantly (there are exceptions) referred to as a potentially hazardous contaminant in the fish, wildlife, and environmental toxicology literature but seems to be given the benefit of the doubt as a beneficial element in the human health literature [484,486]. Different species do vary in sensitivity to individual contaminants, but perhaps human health researchers should be looking more closely at fish and wildlife literature and perhaps fish and wildlife researchers should be looking more closely at the human health literature. Is one group (human health investigators) looking harder for positive effects and the other group (wildlife investigators) looking harder for negative effects?

A few summarizations do treat the potential impacts and benefits somewhat more evenly [487,488], but as of 1993, most individual (specialized) research papers and reports still seem to looking at selenium from either in a somewhat negative or positive perspective.

This contrast is partly explained by the fact that the range between insufficient and too much selenium in the diet of animals and humans is unusually narrow [445,488]. Thus, probably one of the most important reasons selenium is treated as a totally positive element in so many papers at the same time it is being treated as a totally

negative hazard in so many other papers has to do with dose. Those looking at very low concentrations of dietary selenium (this group seems to include most researchers working on human health issues) are looking for and finding benefits, while those looking at higher concentrations of dietary selenium (this group includes many researchers working on fish and wildlife issues) are looking for and finding hazards. It is known that different species of living things have different sensitivities to selenium [445], but in the case of selenium in humans versus selenium in fish and wildlife, the difference may partly have to do dose and food chain factors. Many of the most dramatic reports of selenium poisoning have occurred in fish and wildlife resources in ecosystems where elevated selenium levels are biomagnified up through the food chain.

Selenium nevertheless remains somewhat unusual and interesting due to the sheer number of seemingly contradictory literature references to selenium as a contaminant which (depending on dose, form, and numerous other details) can either help cause or help protect against such things as liver damage, cancer, immune system problems, and a host of other potential hazards.

Although Se is an essential micronutrient for various immune mechanisms, an excess of Se may have a deleterious effect on certain immunological functions. As these activities are considered to be important defense mechanisms against tumors and virus infections, a nutritional imbalance of Se could result in an increased risk of these disorders (Nair MP; Schwartz SA. 1990. Immunoregulation of natural and lymphokine-activated killer cells by selenium. Immunopharmacology; 1990 May-Jun; 19(3); P 177-83, Author: Department of Pediatrics, University of Michigan, Ann Arbor 48109-2029).

In the environmental toxicology literature, beneficial aspects of selenium are most often mentioned as the exception rather than the rule. Again, the reason for this most likely has to do with dose and bioconcentration through the food chain; documented problems related to selenium are particularly common in higher food chain levels in western areas which have elevated selenium levels.

> NOTE: One of the paradoxes of the widely publicized selenium toxicity problems at Kesterson National Wildlife Refuge was that selenium deficient soils have been reported near Kesterson [486, (Biol. Trace Element Res., Vol 20 (1-2), 1989]; at Kesterson concentration of selenium from subsurface agricultural drainage water, further concentrated by evaporation, helped produce a buildup of

selenium.

By contrast, if one looks through the human health literature [484,486], one finds reference after reference to the potential benefits of selenium. Selenium is described [484,486] using such glowing phrases as:

> An anti-carcinogenic nutrient, a versatile anticarcinogenic agent, an essential trace element necessary for man, an element with numerous protective functions, a redox switch which helps prevent the malignant transformation of cells, an inhibitor of the replication of tumor viruses, an inhibitor of activation of oncogenes, possibly a modification agent in carcinogen metabolism which protects DNA against carcinogen-induced damage, an anti-carcinogen which accepts biogenic methyl groups, a detoxifying agent for certain metals and xenobiotics, an immuno-potentiating agent, an agent which can bind mercury and cadmium compounds to make them more biologically inert, a part of the anti-oxidative defence system, a protective agent against mercury induced lipid peroxidation, a defence against free radicals, an element indispensable to appropriate immune response, an element which can detoxify various metals by chelating them, a risk factor for cancer in humans which do not get enough of it, helpful in preventing colon and breast cancer, and an element which is possibly helpful in preventing skin, liver, and pancreas cancer.

In the human health literature the potentially harmful aspects of selenium are most often treated as the exception rather than the rule. Most of the documented cases of selenium poisoning seem to be in response to high selenium intake in certain areas of China [893]:

> Yang, G., S. Wang, R. Zhou and S. Sun. 1983. Endemic selenium intoxication of humans in China. Am. J. Clin. Nutr. 37: 872-881. Yang, G., R. Zhou, S. Yin, et al. 1989a. Studies of safe maximal daily dietary selenium intake in a seleniferous area in China. I. Selenium intake and tissue levels of the inhabitants. J. Trace Elem. Electrolytes Health Dis. 3(2): 77-87. Yang, G., S. Yin, R. Zhou, et al. 1989b. Studies of safe maximal daily dietary Se-intake in a seleniferous area in China. II. Relation between Se-intake and the manifestation of clinical signs and certain biochemical alterations in blood and urine. J. Trace Elem. Electrolytes Health Dis. 3(2): 123-130. Although there is sometimes anecdotal information given on harmful effects (like sodium

> selenite causing deficits in mammalian limb development if allowed to reach developing tissue), there are more frequently just blanket statements that selenium toxicity in humans is very rare in the U.S. [484,486].

In a summary of beneficial aspects, Moore et al. [445] stated that:

> Although it is yet to be proven that selenium is an essential micronutrient for plants, it has been shown to be essential for algae, bacteria, and fish and other animals, including humans. Selenium plays important biochemical and physiological roles in animals, including: participating in protein synthesis (e.g., of immunoglobulin and ubiquinone); assisting in the mitochondrial transport of electrons in muscles; facilitating an essential metabolic union of oxygen and hydrogen; and in enzymes containing selenium, such as glutathione peroxidase, playing an important role in catalyzing reactions that protect cell membranes from oxidation damage, thereby complementing the functions of vitamin E. Findings of human health studies suggest that selenium may also play important roles in preventing cardiovascular diseases, arthritis, and certain types of cancer (e.g., breast [female], colon, leukemia, liver, lung [male], ovarian, prostrate, rectal, and skin cancers) [445]. Signs of selenium deficiency in animals include: loss of feathers or hair; reduced growth; degeneration of the liver, pancreas, and heart; myopathy (white muscle disease); periodontal disorders; reproductive impairment; lameness; steatitis; exudative diathesis; immunosuppression; and gastroesophageal ulcers [445]. The difference between essential and toxic doses of selenium is quite narrow. Demayo et al. in 1979 estimated there to be only a fifty-fold safety margin between recommended and toxic dietary concentrations of selenium for animals. Maier et al. (1987) suggest that the safety margin for aquatic life may be approximately 10-fold [445].

Humans require minute quantities of selenium to maintain tissue elasticity and prevent premature aging, muscle pain, and heart disease [173].

So, although excess selenium in certain forms can have dramatic impacts of living things, selenium in certain forms and small quantities often has benefits. Unlike heavy metals such as cadmium, mercury, and lead, selenium in small quantities in the diet of vertebrates and many other living things does have redeeming qualities.

Some contaminants specialists who have looked at some of the human health and animal husbandry literature have wondered whether or not slight elevations of some forms of selenium in fish tissues may possibly be acting partly in a protective manner (to a greater degree than is commonly recognized) to humans and fish and wildlife predators consuming fish contaminated with harmful concentrations of heavy metals such as cadmium, mercury, and lead (Jerry Miller, U.S. Bureau of Reclamation, Salt Lake City, personal communication, 1994). However, care should be taken in generalizing, and the many risks associated with bioconcentration, reproductive risk, and other potential risks of selenium cannot be ignored. See also the Interactions section below for more details on interactions between selenium and mercury.

The benefits, potential impacts, and food web fate of selenium vary not only by the form of selenium involved, but also by the type of organism. For example, monogastric (non-ruminant) animals more readily absorb dietary selenium+4 (selenite) than do ruminants [445].

The range between insufficient selenium in the diet of animals and too much is narrow, and the effects of either problem can be serious [63]. In humans, too much selenium has also reportedly caused baldness, loss of nails and teeth, fatigue, and death [173].

The toxicity to an animal of selenium exposure is influenced by numerous factors, including: the species, sex, lifestage, nutritional status, and health of the organism; the chemical form(s) of selenium; previous exposure history; environmental stresses, including weather; and the presence of other, interactive chemicals [445]. Selenium is more toxic to coho salmon that to chinook salmon and more toxic to trout than to bluegills [445]. Younger animals (especially fish fry and bird embryos) and those consuming low-protein diets are very sensitive to selenium's toxic effects [445]. Waterfowl feeding on zooplankton or on algae may be more sensitive to selenium contamination than those feeding on seeds [222]. Mallards, cinnamon teal, and pintails, which consume large amounts of seeds are therefore less at risk than gadwalls and Norther shovelers, which consume primarily algae and zooplankton [222]. Using the same criteria, green winged teal and widgeon would be at intermediate risk [222]. Chickens and Japanese quail are more sensitive to selenium toxicity than are mallard ducks, which are more sensitive than screech owls and black-crowned night-herons [445].

A comprehensive toxicological profile for selenium and its compounds, especially as it relates to human health, is available from ATSDR [953]. Due to lack of time,

important highlights from this ATSDR document have not yet been completely incorporated into this entry. A synoptic review of selenium hazards to fish, wildlife, and invertebrates was provided by Eisler in 1985 [37]. A more recent summary on selenium issues related to fish was provided by Sorenson in 1991 [488]. However, a tremendous amount of selenium research has been done since then, partially as a result of the widely publicized and dramatic impacts of high selenium levels upon birds and other biota at Kesterson National Wildlife Refuge in California.

Br.Car: Brief Summary of Carcinogenicity/Cancer Information:

EPA 1996 IRIS database information [893]:

Evidence for classification as to human carcinogenicity: weight-of-evidence classification

Classification: D; not classifiable as to human carcinogenicity

BASIS: Based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animal and mutagenicity studies is conflicting and difficult to interpret; however, evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification.

Human carcinogenicity data: Inadequate. Data on the potential carcinogenicity of selenium and various selenium compounds in humans are inadequate.

Animal carcinogenicity data: Inadequate. The carcinogenicity of selenium compounds has been evaluated in several animal studies. However, the data are conflicting and difficult to interpret because of apparent anticarcinogenic activity and high toxicity of some selenium salts.

Available data provide no suggestion that selenium is carcinogenic in man, & evidence for neg correlation between regional cancer death rates & selenium is not convincing (IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985, Multivolume work.,p. V9 256, 1975) [940].

There are many references in the literature to the

ability of selenium to inhibit carcinogenic processes. For example, selenium has been shown to inhibit carcinogenesis in several animal systems. Results show that cadmium stimulates the growth of human prostatic epithelium in vitro between 10(-9) M and 10(-7) M concentrations. Selenium, at concentrations between 10(-12) M and 10(-7) shows no growth stimulatory or inhibitory effects on these cells. However, when present at 10(-8) M level, selenium inhibits the growth stimulation induced by cadmium (Webber MM; Biochem Biophys Res Commun 127, 3: 871-7 (1985) [940]. Some of the literature on inhibition of cancer by selenium is summarized in IRIS [893]. See also: Br.Haz section above.

Although selenium can act as a protective anti-carcinogen [484,486,488,491; see also Br.Haz above for details] there are some references in the literature to selenium as a potential carcinogen:

> Some salts of selenium are carcinogenic [168]. Selenium sulfide produced an increase in hepatocellular carcinomas and adenomas [491].

Br.Dev: Brief Summary of Developmental, Reproductive, Endocrine, and Genotoxicity Information:

Selenium is considered embryotoxic and teratogenic on the basis of animal experiments [37,491].

Excess selenium has been documented to cause the following problems [445]:

> Excess selenium in plants can adversely affect seed germination and growth. In animals, excess selenium can cause: embryo malpositions and deformities (teratogenesis), including hydrocephaly, and malformed beak, eyes, face, feet/hooves and toes, jaws, legs, nose and nasal pits, spinal column, tail, and wings; abnormal development of and damage to/degeneration of internal organs, including the heart, liver, and kidneys; edema; mutagenesis; reproductive impairment, including reduced production, weight, and hatchability of eggs; feed aversion, emaciation, and reduced growth; skin lesions; dullness, roughness, and loss of hair/feathers; cracked, deformed, sore, and sloughed hoofs or nails; stiffness and lameness (perhaps as a result of joint erosion); respiratory failure; paralysis; immune system suppression; a range of behavioral, physiological, biochemical, and histopathological changes; and death [445].

Effects of too much selenium in the diet of animals include birth defects, sterility, and death [63].

There is no evidence from experimental animals administered selenium compounds via the oral route or via injection that selenium compounds are teratogenic in mammals [953].

In human placenta in vitro studies, inorganic selenium compounds reduce the toxicity of human placental toxicants such as cadmium; however, neither the kinetics nor the developmental toxicity of selenium is known for humans [484,485].

Looking through the fish and wildlife literature, one finds reference after reference to potential environmental hazards of excess selenium (toxicity to fish and wildlife, reproductive failures, birds and fish born with birth defects, and all of the well-publicized problems at Kesterson National Wildlife Refuge in California, etc.). In fish, selenium tends to build up in the ova [488]. This may relate to the fact that selenium problems tend to be important in egg bearers (Joe Hunn, NBS, Columbia, MO, personal communication, 1993).

Daily intraperitoneal injections of male rats with selenium dioxide caused changes in the testes with significant testicular degeneration and atrophy at the highest dose tested [953]. On the other hand, selenium deficiency has been reported to cause infertility in livestock and decreased sperm production and motility in selenium-deficient rats [953]. The relevance of these reproductive effects in animals to potential reproductive effects in humans is not known. In samples from more than 200 men, no correlation between seminal plasma selenium and sperm count or mobility was detected [953].

Selenium accumulates in the gonads of bass and bluegills [36].

Impairment of reproduction (including development and survival of embryos) is one of the most sensitive indicators of selenium toxicity among birds (Heinz, Oct 1989; Heinz et al., 1989). Reproduction among fishes can be significantly reduced or even eliminated with little or no adult mortality (Lemly, 1986).

Inorganic selenium compounds have been observed to have both genotoxic and antigenotoxic effects. The antigenotoxic effects generally occur at lower selenium exposure levels than the genotoxic effects [953].

In general, sodium selenite and sodium selenate have

produced mixed results in bacterial mutagenicity test systems [953]. Results with mammalian cell systems are mixed, although sodium selenite is more consistently genotoxic in these systems [953]. At high concentrations, sodium selenite induces unscheduled DNA synthesis and chromosome aberrations in cultured human fibroblasts [953].

Br.Fate: Brief Summary of Key Bioconcentration, Fate, Transport, Persistence, Pathway, and Chemical/Physical Information:

In the environment, selenium combines with oxygen to form several substances. The most common are sodium selenite and sodium selenate [953]. See the Forms/Preparations/Formulations section below for more details.

Preliminary data suggests the potential for bioaccumulation or bioconcentration of selenium is moderate for the following biota: mammals, birds, and fish. It appears to be high to very high for higher plants and low or limited for mollusks, crustacea, lower animals, mosses, lichens, and algae [83].

Daphnids in aqueous selenium exposures may be accumulating a portion of their selenium body burdens via ingestion of selenium enriched algae [481].

The concentrations and chemical forms of selenium in water are influenced by, among other factors, pH and redox conditions. Although dissolved selenium in most natural waters is dominated by the inorganic selenate and selenite forms, substantial concentrations of selenium in +6, +4, and -2 oxidation states can occur simultaneously in natural aerobic surface waters of pH 6.5-9.0 [445].

Selenium concentrations in the soils, how tightly the selenium is bound to soils, and man's activities (such as irrigation practices and mining) can all play a role in determining whether or not selenium becomes an environmental problem in a given area. See also the Bio.Detail section below for details on selenium bioavailability and fate in soils.

As is the case for mercury, microorganisms in sediments or sewage are capable of methylation or demethylation of selenium [488]. Selenium is methylated and probably demethylated in the environment and cycled through a number of components of the food web, complicating chemical determination of the chemical forms available to fish [488]. Dimethylselenide is generated slowly from raw sewage [488].

Selenium, like mercury, has many interactions with sulfur compounds. This affinity for sulfur compounds may account for some of the many synergistic and antagonistic interactions between mercury and selenium (see the Interactions section below for details). Also like mercury, selenium chemistry, transformations, and interactions with other contaminants are complex. In fact, selenium is one of the most complex elements in these regards. Due to these complexities, generalizations about selenium should be approached with caution.

Recent news media, human health, cattle and sheep grazing, wildlife, rangeland, and geological controversies about selenium were summarized by Tom Harris in the February 10, 1992 issue of the High Country News (Western U.S. paper published in Paonia, Colorado). According to Harris, more research needs to be done to better define: 1) the role of deep-rooted, selenium-tolerant plants such as Astragalus bisulcatus and woody asters of the genus Xylorrhiza as "converter plants" which draw relatively inert forms of selenium up from the soil and metabolize them into water-soluble selenate--so that when the converter plants die they deposit an enriched halo of water soluble organic selenium compounds that can be taken up by other plants and other biota (the methylated daughter compounds are more bioavailable and more potent than the parent compounds), 2) the role of widespread trucking of cattle and hay in transporting seeds of these "converter plants" into new areas, 3) spreading selenium into new areas through applications of phosphate fertilizers contaminated with selenium, 4) the impact on hunters of consuming selenium contaminated wildlife, including antelope, 5) the extent of selenium contaminated meat (especially the liver, kidney and heart) consumed by humans and wildlife.

Synonyms/Substance Identification:

Metallic Selenium [953]
CI 77805 [940]
ELEMENTAL SELENIUM [940]
SELEN (POLISH) [940]
SELENIUM (COLLOIDAL) [940]
SELENIUM ALLOY [940]
SELENIUM BASE [940]
SELENIUM DUST [940]
SELENIUM ELEMENTAL [940]
SELENIUM HOMOPOLYMER [940]
CASWELL NO. 732 [940]
EPA Pesticide Chemical Code 072001 [940]
Gray selenium [940]

Molecular Formula:
Se [940]

Associated Chemicals or Topics (Includes Transformation Products):

Relationships between this metal versus indicator plants, other metals, and various rock types was summarized by Brooks in 1972 [951]. Some of the same Astragalus plants which are indicators for high selenium are also indicators for uranium prospecting [951].

Certain uranium deposits contain appreciable quantities of selenium [951]. Concentrations of vanadium in selenium rich soils tends to be high [951]. Certain contaminants, such as selenium, thorium 230, and vanadium, tend to occur together in and leach out of uranium mining tailing piles in the U.S.; thorium is quite dangerous and is often leached out of acid process uranium piles (Ward Whicker, Colorado State University, personal communication, 1996).

Site Assessment-Related Information Provided by Shineldecker (Potential Site-Specific Contaminants that May be Associated with a Property Based on Current or Historical Use of the Property) [490]:

Other Associated Materials:

- Carbon disulfide

Metabolism/Metabolites [366]:

In the liver, many selenium compounds are biotransformed to excretable metabolites. Identified metabolites are trimethylselenide in urine and dimethylselenide in breath (Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986.,p. V2).

Impurities [940]:

Commercial grade contains a min of 99% selenium & may contain max 0.2% Tellurium, 0.1% Iron, 0.005% Lead & 0.005% Copper as impurities. The high purity grade ... Is reported to contain a min of 99.99% Selenium. Impurities which may be present @ concn no greater than 1-2 mg/kg each are mercury, tellurium, iron, arsenic & other non-ferrous metals undesirable in electronic & electrostatic applications. Higher concn of "inert" contaminants such as sodium, magnesium, calcium, aluminum & silicon can be tolerated. The ultra high purity grade, prepared only on a laboratory scale, is reported to contain 0.0001-0.001% Impurities. [IARC. Monographs on

the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer,1972-PRESENT. (Multivolume work).,p. V9 246 (1975)].

Water Data Interpretation, Concentrations and Toxicity (All Water Data Subsections Start with "W."):

W.Low (Water Concentrations Considered Low):

No information found.

W.High (Water Concentrations Considered High):

Selenium concentrations in waters of the San Luis Drain and Kesterson Reservoir were 170-420 ug/l (ppb) and 24-430 ug/l, respectively [445].

W.Typical (Water Concentrations Considered Typical):

Typical Ocean Concentrations: EPA 1981: 0.00009 mg/l [83].

Selenium concentrations in the world's oceans range from 0.000052 to 0.00013 mg/l (0.052 to 0.13 ppb) and average 0.09 ug/l (ppb) [445].

Typical Freshwater Concentrations:

EPA 1981: 0.02 mg/l [83].

1971: The average stream concentration was 0.2 ug/L and concentrations rarely exceed 1.0 ug/L [190].

USGS 1974-1981: the 50th percentile of 161 (not especially clean) NASQWAN and NWQSS river sites in the U.S. was <1 ug/l; the 25th percentile was <1 ug/l, and the 75th percentile was 1 ug/l, with concentrations trending downward more often than upward [219]. These riverine sites in the USGS study were mostly in (or downstream of) agricultural and urban areas [219].

Concentrations of selenium in freshwater rivers around the world range from <0.0001 to 0.4 mg/l (<0.1 to 400 ppb) and average 0.2 ppb [445].

California, 1986: Ambient background level for water was 0.2 ug/l [222].

W.Concern Levels, Water Quality Criteria, LC50 Values, Water Quality Standards, Screening Levels, Dose/Response Data, and

Other Water Benchmarks:

W.General (General Water Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Water Concentrations Versus Mixed or General Aquatic Biota):

Notes on total vs. acid soluble vs. dissolved metals:

Although most of the lab tests done to develop water quality criteria and other benchmarks were originally based on "total" values rather than "dissolved" values, and many original criteria for selenium were specifically for "total recoverable selenium [38], some regulatory authorities nevertheless recommend comparing criteria with dissolved or acid soluble metals concentrations. For detailed discussion, see the Laboratory and/or Field Analyses section (far below).

EPA 1996 Great Lakes Guidance:

On November 14, 1996, EPA proposed in the Federal Register that the acute toxicities of selenate, selenite, and one form of organoselenium are additive. They further proposed that all forms be added together to obtain a total for comparison with water quality criteria and that total selenium can be converted to dissolved by multiplying total by 0.996. Further details: EPA suggested in November of 1996 (Federal Register Vol 61, no. 221,pages 5844 to 58449) that:

1) A new acute aquatic life criterion be used in the Great Lakes based on an equation of the presence of selenium 4 (selenite) vs selenium 6 (selenate) vs other forms;

2) the acute toxicity of selenite (Selenium IV) is 12.83 ug/L and that of selenate (Selenium VI) is 185.9 ug/L.

3) all forms of selenium tend to convert back and forth into the other, and the effects are basically additive.

4) Criterion Maximum Concentrations are in total selenium, but the following conversions may be used: A factor to

0.996 may be used to convert to convert total (recoverable) acute criteria for selenite to a dissolved criteria for selenite.

Note: Another conversion factor which is sometimes given: Selenium (+4) conversion for acute and chronic criteria: 0.922 (for example, total recoverable selenium (+4) criteria x 0.922 = dissolved selenium (+4) criteria) [672].

The conversion factors recommended by EPA for converting total recoverable selenium to dissolved concentrations in the January 1997 draft EPA Guidelines for 5 year 305(B) assessments was also 0.922.

Note: None of these generic conversion factors are universal. Both total and dissolved concentrations should be checked at new locations before relying on generic conversion factors (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

5) Depending on the relative proportions of various selenium species, the acute criterion for the Great Lakes is between 13 to 186 ug/L.

EPA 1996 IRIS Database [893]:

Ambient Water Quality Criteria for Aquatic Organisms for Total Selenium [446,689,893]:

Acute Freshwater: 2E+1 ug/l 1-hour avg.
Chronic Freshwater: 5E+0 4-hour avg.

Marine Acute: 3.0E+2 ug/L 1-hour avg.

Marine Chronic: 7.1E+1 ug/L 4-hour avg.

Contact: Criteria and Standards Division / OWRS / (202)260-1315

Discussion: Criterion were derived from a minimum database consisting of acute tests on a variety of species. Requirements and methods

are covered in the reference to the Federal Register. The agency recommends an exceedance frequency of no more than 3 years.

Note: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was underway in March of 1996, and IRIS is updated monthly [893].

Oak Ridge National Lab, 1994: Ecological Risk Assessment Freshwater Screening Benchmarks for concentrations of contaminants in water [649]. To be considered unlikely to represent an ecological risk, field concentrations should be below all of the following benchmarks [649]:

For selenium (CAS 7782-49-2, ug/L):

NATIONAL AMBIENT WATER QUALITY CRITERION - ACUTE: 260

NATIONAL AMBIENT WATER QUALITY CRITERION - CHRONIC: 35

SECONDARY ACUTE VALUE: No information found.

SECONDARY CHRONIC VALUE: No information found.

LOWEST CHRONIC VALUE - FISH: 88.32

LOWEST CHRONIC VALUE - DAPHNIDS: 91.65

LOWEST CHRONIC VALUE - NON-DAPHNID INVERTEBRATES: No information found.

LOWEST CHRONIC VALUE - AQUATIC PLANTS: 100

LOWEST TEST EC20 - FISH: 40

LOWEST TEST EC20 - DAPHNIDS: 25

SENSITIVE SPECIES TEST EC20: 2.60

POPULATION EC2O: No information found.

Other Concern Levels for Water Concentrations:

EPA 1987: EPA freshwater criteria suggests

that selenium concentrations for a four-day average should not exceed 5.0 ug/L, nor a one-hour average above 20.0 ug/L (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

A State of California recommendation based on direct toxicity was that the 2.6 ug/L; however, the water quality criteria based on protection of human health was lower, 0.8 ug/L for impounded waters (versus 10 ug/L for flowing waters), due to bioconcentration concerns [222]. The water quality criteria based on protection of aquatic organisms in the food chain was also quite low, only 0.9 ug/L for impounded waters (versus 11 ug/L for flowing waters), due to bioconcentration concerns [222].

Joseph Skorupa (Fish and Wildlife Service) has suggested that selenium from agricultural irrigation return waters may contribute to problems in various areas throughout the Western United States, and that the long-term safety of aquatic systems containing 2 to 5 ppb of total waterborne selenium can be questioned [463].

A June, 1992, Federal Register discussion of issues related to selenium supplements in animal feed included the following items of interest on selenium toxicity in water [463]:

> Dennis Lemly (Fish and Wildlife Service) commented that although the forms of selenium excreted by animals may not be bioavailable to terrestrial ecosystems, they may be bioavailable in aquatic ecosystems. He suggested that certain areas of the United States, where soil selenium levels are relatively high and bioavailable, are incapable of sustaining even small increases of selenium in the aqueous environment. Joseph Skorupa (Fish and Wildlife Service) suggested that selenium from agricultural irrigation return waters may contribute to problems in various areas throughout the Western United States, and that the long-term safety of aquatic systems containing 2 to 5 ppb of total waterborne selenium can be questioned. Marc Sylvester (Geological Survey) suggested

that selenium in manure spread on farm land in semiarid and arid areas in the Western United States would most likely be oxidized to the selenate form, which is very mobile. Therefore, he indicated that the assumption used in the worst-case analysis that a maximum of 10 percent of the selenium in waste-amended soil will be lost to runoff is unreasonable [463].

CVM met with the scientists from the Department of Interior on May 31, 1990, to discuss their concerns. These scientists stated that research in progress indicates that the specific form of selenium is critical in determining the potential for the occurrence of selenium toxicity because amino forms of selenium may bioconcentrate to toxic levels in fish and birds even when concentrations in water are less than 1 microgram per liter (ppb). For this reason, according to the scientists, information, as opposed to the assumptions used in the worst-case analysis, is needed on the specific forms of selenium entering the environment through animal waste and the fate of these forms [463].

More recently, CVM learned that the Environmental Protection Agency (EPA) is developing a threshold value for dissolved waterborne selenium to protect wildlife using aquatic environments because it appears that the current ambient water quality criteria for selenium of 5 ppb established for aquatic species may not be adequate to protect wildlife [463].

Information from Moore [445]:

See Moore for details on citations: In a review of selenium cycling in aquatic ecosystems, Lemly and Smith (1987) stated that, "...selenium at concentrations greater than 2 to 5 ug/L (ppb) in water can be bioconcentrated in food chains and cause toxicity and reproductive failure in fish." Using his energy-based selenium bioaccumulation model for aquatic birds, DuBowy (1989) determined that the water

quality criterion for selenium would need to be less than 2.8 ppb to protect waterfowl reproduction. A University of California Committee of Consultants formed to evaluate the water quality objectives for the San Joaquin River Basin originally proposed by the CSWRCB (CSWRCB, Aug 1987), recommended a criterion range between 1 and 1.5 ppb waterborne selenium as a "...highly conservative estimate of no adverse effect...[which]...may account for the possible deleterious effect of bioaccumulation" (UC Committee of Consultants on San Joaquin River Water Quality Objectives, Feb 1988). This last range of concentrations is the same as that recommended by scientists from the University of California, Davis, using data from their selenium toxicity research and other scientific literature. They stated that a "...conservative water quality goal for the protection of aquatic organisms, a level where no adverse effects should occur, appears to be between 1.0 and 1.5 ppb" (Davis et al., Jan-Feb 1988). Finally, taking bioaccumulation into effect, the CSWRCB determined that a waterborne concentration of 0.9 ug/l (ppb) selenium would be necessary to ensure no adverse effects on aquatic life (CSWRCB, Mar 1988).

The CCVRWQCB has adopted (CCVRWQCB, Dec 1988) and CSWRCB subsequently approved (CSWRCB, Sep 1989) chronic water quality objectives for selenium of 2-10 ug/l (ppb) (monthly means) for wetlands in the Grasslands area, the San Joaquin River, and Salt and Mud (North) sloughs. The USEPA recently disapproved several of the CCVRWQCB's drainage-related water quality objectives, including some of the selenium objectives. The USEPA stated that the objectives did not satisfy Federal legal requirements because they did not protect designated water uses and they were based, in part, on consideration of economic factors (McGovern, Apr 1990).

In 1986, the U.S. Fish and Wildlife Service evaluated the findings of toxicity research, accounted for known biomagnification through the food chain and associated reproductive toxicity, and recommended the following total recoverable selenium concentrations as target safe levels (MATC's) for cleanup of Kesterson Reservoir and the San Luis Drain: (1) water -

2 ppb, (2) sediment - 4 ppm dry weight, (3) food for warmwater fishes - 5 ppm dry weight, and (4) food for waterfowl - 3 ppm dry weight. They also noted, that in order to protect fish reproduction, their skeletal muscles should not contain more than 5 ppm and their organs (liver and gonads) should not contain more than 10 ppm selenium (both concentrations are for total selenium, dry weight) (Wallenstrom, Aug 1986). Hamilton et al. (1990) suggest that, in order to be safe for fish, dietary concentrations of selenium should be less than 3 ug/g (ppm, dry weight).

Skorupa et al. (Mar 1989) evaluated data developed in both laboratory and field studies and estimated that the maximum acceptable toxicant concentration of dietary selenium for aquatic birds was 5.6 ppm, dry weight. Recently, in an attempt to reduce wildlife contamination hazards, the California Department of Fish and Game adopted a selenium standard for subsurface agricultural drainage water evaporation ponds in the San Joaquin Valley. The CDFG standard requires initiation of special management actions (e.g., hazing) when the selenium concentration in a composite sample of aquatic invertebrates from a pond equals or exceeds 4 ppm, dry weight. Other than CDFG's evaporation pond-specific standard, no regulatory standards currently exist for the protection of fish and wildlife from dietary exposure to selenium."

W.Plants (Water Concentrations vs. Plants):

Shallow Groundwater Ecological Risk Assessment Screening Benchmark for Terrestrial Plants Listed by Oak Ridge National Lab, 1994 [651]:

NOTE: To be considered unlikely to represent an ecological risk, field concentrations in shallow groundwater or porewater should be below the following benchmark for any aqueous solution in contact with terrestrial plants. Toxicity of groundwater to plants may be affected by many variables (pH, Eh, cation exchange capacity, moisture content, organic content of soil, clay content of soil, differing sensitivities of various plants, and various other factors). Thus, the following solution benchmark is a rough screening benchmark only, and site specific tests would

be necessary to develop a more rigorous benchmark for various combinations of specific soils and plant species [651]:

> For CAS 7782-49-2, SELENIUM, the benchmark is 0.7 mg/L (groundwater or porewater).

Algal dry weight and chlorophyll-a concentrations were both reduced in the test waters containing >75 ppb selenium+4. Algal cell replication was reduced in the 100 ppb selenium+4 test waters and ceased in the >125 ppb selenium+4 test waters. Algal selenium depuration mechanisms were overwhelmed in waters with >100 ppb selenium+4. After 10 days, no significantly different effects on growth, reproduction, or survival were observed among daphnia fed high-, mixed-, or low-selenium diets. The authors suggested that the algal selenium fed to the daphnia might have been in a methylated form, thereby explaining the unexpected results of the daphnia toxicity experiment [445].

W.Invertebrates (Water Concentrations vs. Invertebrates):

LC50s for Brachionus calyciflorus and B. plicatilis (both rotifers) were 16.0 and 17.0 mg/L (ppm), respectively, for 24-hr exposures [998].

LC50s for Daphnia magna (water flea) were 0.66, 0.71, 0.43 and 0.43 mg/L for 24-, 48-, 96-hr and 14-day exposures, respectively [998].

LC50 for Mysidopsis bahia (Opossum shrimp) was 0.600 mg/L for a 96-hr exposure [998].

Daphnia mortality was concentration-related in the waterborne selenium+6 experiments. Animals exposed to 1.0 mg/l (1,000 ppb) water experienced 100% mortality. Daphnia exposed to waterborne selenium+6, but fed selenium-laden diets, experienced decreased mortality, except in the 1.0 mg/l (1,000 ppb) water. The effects on growth rate and production of young were similar. With the exception of animals in the 1.0 mg/l (1,000 ppb) water, daphnia receiving selenium-laden algae had a higher growth rate and produced more offspring than those fed the control diet. Radiotracer experiments revealed that uptake of waterborne selenate and depuration of selenate were reduced in the presence of selenium in the diet and waterborne seleno-DL-methionine. Results with waterborne seleno-L-methionine were unclear [445].

The USFWS-NFCRC (Dec 1988) determined the 48-hour LC50's for the cladoceran Daphnia magna and midge Chironomus riparius (animals <1-day-old) exposed to waterborne selenium. Two general test waters, reconstituted freshwater (containing hardness of 134 mg/L [ppm] as calcium carbonate [CaCO3], alkalinity of 60-65 mg/L as calcium carbonate, and 72 mg/L sulfate) or standard ASTM soft water (containing hardness of 40-48 mg/L as calcium carbonate, alkalinity of 30-35 mg/L as calcium carbonate, and 54 mg/L sulfate); were supplemented with selenium+6 (as sodium selenate), selenium+4 (as sodium selenite), a 6:1 mixture of sodium selenate and sodium selenite, or seleno-L-methionine. Mortality for these tests was defined as cessation of mobility [445].

The daphnids did not respond to seleno-L-methionine in a dose-responsive manner; therefore, an LC50 could not be calculated. LC50's for midge exposed to seleno-L-methionine were 5.78 mg/L in hard freshwater and 6.88 mg/L in ASTM soft water. Forty eight-hour LC50's (in mg/L) for the daphnids and midge, respectively, exposed to inorganic selenium were as follows: 4.07 and 16.2 for sodium selenate in hard freshwater and 2.56 and 10.5 in ASTM soft water; 3.02 and 7.95 for sodium selenite in hard freshwater and 0.700 and 14.6 in ASTM soft water; and 2.62 and 9.34 for the 6:1 selenate:selenite mixture in hard freshwater and 1.79 and 14.3 in ASTM soft water [445].

The USFWS-NFCRC (Dec 1988; Dec 1987) also determined the effects of chronic exposures to a 6:1 mixture of sodium selenate:sodium selenite in reconstituted, hard freshwater (same water chemistry as in acute tests) on growth, reproduction, and survival of the same species and age of daphnia and midge used in the acute tests. Daphnia were exposed for 21 days to the following nominal concentrations of waterborne selenium (in ug/L [ppb]): 4 (control), 85, 156, 348, 718, or 1,410. Midge were exposed for 30 days to the following nominal concentrations of waterborne selenium (in ug/L): 10 (control), 303, 837, 1,384, 2,953, or 6,050 [445]. Results:

In the daphnid study, percent survival was significantly reduced at 1,410 ug/L. Both 348 and 718 ug/L caused significant reductions in total young produced, young produced per surviving adult reproductive day, intrinsic rate of natural increase, and adult weight. Exposure to 156 ug/L

caused a significant reduction in adult daphnid weight, but significantly increased length of newborn daphnia. Exposure to 85 ug/L also significantly increased length of newborn daphnia [445].

In the midge study, percent emergence was significantly reduced only at 6,050 ug/L. Emergence time and day of first emergence both increased significantly at all concentrations greater than or equal to 837 ug/L. The authors calculated the no observable effect concentrations for daphnia and midge (for the 6:1 selenate:selenite mixture) to be 85 and 303 ug/L, respectively [445].

W.Fish (Water Concentrations vs. Fish):

Excess selenium, even as low as 3-8 ppb (0.003-0.008 ppm) in the water, can cause numerous life-threatening changes in feral fresh water fish [488].

The Environmental Protection Agency (EPA) 1987 update of the ambient water quality document for selenium [update of reference 38] gives acute (96 hour LC50) values for teleost fish as typically ranging from 620 to 66000 ppb (0.620 to 66.0 ppm); however, where biomagnification is allowed to occur, toxic effects are seen at concentrations as low as 12 ppb in the lab and 2.5 ppb in the field (Heidi Bestgen, Colorado State University, personal communication). Repeated studies have shown that concentrations in water between 2 and 10 ppb can result in reproductive failure and mortality in fish (Will Clements, Colorado State University, personal communication).

LC50s for Cyprinodon variegatus (sheepshead minnow) were 56.0, 26.0, 13.0 and 6.7 mg/L (ppm) for 24-, 48-, 72- and 96-hr exposures, respectively. The no-observed-effect-concentration (NOEC) for death was 2.0 mg/L for a 96-hr exposure [998].

The no-observed-effect-concentrations (NOEC) for death in Lepomis macrochirus (bluegill) ranged from 0.33 to 0.64 mg/L for a 60-day exposure [998].

LC50s for Oncorhynchus kisutch (Coho salmon, silver salmon) ranged from 16.9 to 38.0 mg/L for 96-hr exposures, with most values between 21 and 28 mg/L [998].

LC50s for Oncorhynchus tshawytscha (Chinook salmon) ranged from 46.6 to 96.8 mg/L for 96-hr exposures, with values falling over this entire range [998].

LC50s for Pimephales promelas (fathead minnow) were 1.00 and 0.60 mg/L for 96-hr and 14-day exposures, respectively [998].

Fish toxicity information from Moore [445]:

> See Moore [445] for citation details:
>
> Pyron and Beitinger (1989) studied the acute effects of waterborne selenium on fathead minnows (Pimephales promelas). Six-month-old minnows were exposed to <1 (control), 20, 36, or 66 mg/l (ppm) selenium+6 (as sodium selenate) for 24 hours following which mating behavior, reproductive success, and larval survival were monitored. Fish were fed clean brine shrimp (Artemia sp.) plus commercial flake food diets and exposed to the selenium in reconstituted hard water.
>
> All minnows exposed to 66 ppm selenium+6 waters died within 24 hours of exposure. Surviving fishes in the test and control waters exhibited similar mating behaviors and experienced normal reproduction and hatching success. However, almost all larvae of fish in the 20 and 36 ppm selenium+6 waters exhibited "...gross morphological abnormalities, in particular general edema..." At seven days post hatching, all edamatous larvae had died.
>
> Ogle and Knight (1989) studied the effects of dietary selenium on growth and reproduction of fathead minnows. Beginning at least 105 days prior to egg laying, fish were fed a purified control diet (0.4 ppm) or one of five purified diets to which a mixture of inorganic and organic selenium was added to achieve the following final dietary concentrations: 5.2, 10.2, 15.2, 20.3, or 29.5 ppm (all concentrations in total selenium, dry weight). Proportions of the various selenium forms added to the diet were 25% selenium+6 (as sodium selenate), 50% selenium+4 (as sodium selenite), and 25% selenium-2 (as seleno-L-methionine). Larval progeny were fed newly hatched brine shrimp (ad libitum) and maintained for 14 days. Average water chemistry in the test waters included hardness of 139.4 mg/l (ppm, as calcium carbonate), pH

of 8.19, and 0.8 ug/l (ppb) total selenium.

None of the test diets significantly affected minnow reproduction (including number of spawns per pair, number of eggs per spawn, percent hatch, or percent survival after 14-days). However, beginning on day 56 and thereafter, growth of adult minnows was significantly reduced by the 20.3 and 29.5 ppm mixed selenium diets. The authors suggested that reduced growth may have been caused by a reduction in feeding. The lack of effects on fish reproduction was attributed to reduced bioaccumulation of selenium, possibly as a result of unusual morphology and physiology of the gastrointestinal tract of fathead minnows compared with some other fish species.

The effects of dietary and waterborne selenium on bluegill sunfish (Lepomis macrochirus) were studied by Woock et al. (1987). Adult bluegills were exposed for 323 days (260 days prior to initiation of spawning experiments) to: control trout chow diets (with 8.0% moisture); trout chow diets containing selenium+4 (as sodium selenite, nominal dietary concentrations were 13 or 30 ug/g [ppm]); or trout chow diets containing selenium-2 (as dl- selenomethionine, nominal dietary concentrations were 3, 13, or 30 ug/g [ppm]). In one additional test, fish were exposed to both a selenomethionine diet of 13 ppm and water containing 10 ug/l (ppb) selenium+4 (as sodium selenite). Controls contained <1 ug/g (ppm) selenium in the diet and <1 ug/l (ppb) selenium in water. Average water chemistry in the test waters included: hardness of 16 mg/l (ppm, as calcium carbonate); pH of 6.5; 46 mg/l (ppm) dissolved solids; and 5.2 mg/l (ppm) sulfate. Biological endpoints measured included: weight, lens cataracts, and mortalities of adults; and hatching rate, survival, and teratogenesis among larvae.

Mortalities of adults were significant in both 30 ppm diet groups. Dying fish exhibited: food aversion; edema; lethargy; melanism; tetany; and erratic, spiral, or circular swimming. Fish in the 30 ppm selenium+4 diet group were significantly shorter and lighter than fish in other treatment groups. Thirty seven percent of the fish in the 30 ppm selenium-2 diet group developed true lens

cataracts. Neither fish in the control nor any of the other treatment groups had cataracts.

Hatching success was unaffected by selenium exposure. However, larvae borne to adults fed 30 ppm selenium+4, 30 ppm selenium-2, or 13 ppm selenium-2 in the 10 ppb selenium+4 water experienced significant mortality and high frequencies of teratogenesis (including edema, lordosis, and lower jaw gape). Offspring of parents fed the selenium-2 diets experienced significantly greater effects than those of parents fed the selenium+4 diets. The authors also noted that their findings may underestimate the toxic effects of selenium in natural fish diets, because the diets in their study contained high protein levels which could have ameliorated selenium's adverse effects.

The USFWS-NFCRC (Dec 1988) conducted three sets of acute and chronic toxicity tests exposing bluegill to various waterborne and/or dietary concentrations of selenium. In the first set of tests, bluegill were exposed for 96 hours in a generic, hard freshwater or ASTM soft water to various concentrations of selenium+6 (as sodium selenate), selenium+4 (as sodium selenite), a 6:1 mixture of sodium selenate and sodium selenite, selenium-2 (as seleno-L-methionine), or selenium-2 (as seleno-DL- methionine). Water chemistry in this set of tests included: 134 mg/L (ppm) hardness as calcium carbonate, 60-65 mg/L alkalinity as calcium carbonate, and pH of 8.1 in the generic, hard freshwater; and 40-48 mg/L hardness as calcium carbonate, 30-35 mg/L alkalinity as calcium carbonate, and pH of 7.2-7.6 in the ASTM soft water.

The second set of tests consisted of three chronic toxicity experiments. In the first, 3-month-old bluegill were exposed for 30 days in hard freshwater (water chemistry as just described) to a 6:1 waterborne mixture of sodium selenate and sodium selenite at the following nominal, total selenium concentrations (in mg/L [ppm]): 0 (control), 1.4, 2.7, 5.4, 10.9, or 21.7. In the second experiment, 5-month-old bluegill were exposed for 60 days in hard freshwater to the same 6:1 selenate:selenite mixture at the following nominal, total selenium concentrations (in

mg/L): 0 (control), 0.171, 0.341, 0.683, 1.38, or 2.73. Fish surviving this 60-day experiment were placed in selenium-free water for 28 days to study depuration rates. In the third experiment, 3-month-old bluegill were exposed for 90 days to a seleno- L-methionine-enriched diet containing the following nominal concentrations g/Kg [ppm], wet weight): 0 (control), 1.6, 3.3, 6.5, 13.0, or 26.0.

In the third set of tests, 2-year-old bluegill were exposed for a total of 140 days (60 days - pre-spawning phase and 80 days - spawning phase) to 10 ug/L (ppb) selenium (as mixture of selenate and selenite) and to dietary selenium-2 (as seleno-L-methionine) in five concentrations from 0 through 33.3 ug/g (ppm, dry weight), except for one control group which received no dietary selenium. Fry produced during this study were exposed to the same waterborne and, beginning 15 days post hatch, the same dietary concentrations and forms of selenium as their parents for a total of 30 days. Water used in this set of tests was the same hard freshwater described earlier.

Ninety six-hour LC50's (in mg/L [ppm]) produced by the first set of tests for the hard freshwater and ASTM soft water, respectively, were as follows 72-120 and 98 for selenate, 7.8-13.0 and 7.8-13.0 for selenite, 0.009 aneleno-L-methionine, and 0.010 and 0.013 for seleno-DL-methionine. Preliminary findings from the 30-day experiment revealed that bluegill experienced significant mortality in all treatment waters. Findings of the 60-day experiment revealed that percent survival of bluegills was significantly reduced in all treatments containing greater than or equal to 0.683 mg/L selenium. Findings of the third, 90-day dietary experiment revealed no significant dose-related effects on survival, although overall condition of bluegills was reduced at the highest two selenium concentrations.

In the third set of tests, preliminary findings reveal that none of the treatments affected adult bluegills, neither was percent hatch of eggs spawned or growth of surviving fry affected. However, after 30 days, fry in the treatment group receiving the greatest waterborne and dietary concentrations of

selenium had experienced a high rate of mortality.

The USFWS-NFCRC (Dec 1989) conducted acute (96-hour) and chronic (90-day) toxicity studies with juvenile striped bass (Morone saxatilis) exposed to waterborne selenium. In the acute tests, fish were exposed to selenium+6 (as sodium selenate), selenium+4 (as sodium selenite), a 6:1 mixture of sodium selenate and sodium selenite, or seleno-L-methionine. In the chronic test, 80-day-old fish were exposed to the 6:1 mixture of sodium selenate and sodium selenite. Test waters included standard ASTM soft water (containing hardness of 40-48 mg/L [ppm] as calcium carbonate, alkalinity of 30-35 mg/L as calcium carbonate, and pH of 7.2-7.6) or 1.2 g/L (ppth) saline water with an alkalinity of 70-75 mL as calcium carbonate.

96-hour LC50's for ASTM the saline water, respectively, were as follows: selenate -39 and 34 mg/L, selenite 1.0 and 6.0 mg/L, 6:1 selenate:selenite mixture - 15.0 and 18.0 mg and selenomethionine - 0.004 and 0.003 mg/L. Concentrations of the 6:1 selenate:selenite mixture up to 3.0 mg/l did not affect growth or survival of fish after 90 days in the chronic toxicity study.

Hunn et al. (1987) studied the chronic effects of selenium+4 on rainbow trout (Salmo gairdneri). Trout sac fry were fed (ad libitum) a clean Rangen's salmon starter diet supplemented with brine shrimp and exposed for 90 days to the following concentrations of waterborne selenium+4 (as sodium selenite): less than detection (control), 7.8, 12.4, 21.0, 47.2, or 99.5 ug/l (ppb). Water chemistry of test waters included pH of 7.4, 272 mg/l hardness (ppm, as calcium carbonate), and 237 mg/l alkalinity (ppm, as calcium carbonate). Biological endpoints measured during the study included survival, growth (length and weight), and chemical composition and mechanical properties of vertebral bones.

After 90 days, fish exposed to test waters containing the highest selenium+4 concentration experienced significant reductions in growth and survival. Fish exposed to 47.2 ug/l (ppb) selenium+4 also experienced significant mortality, but had an

increase in bone toughness. Bone calcium concentrations were significantly reduced in fish exposed to >12.4 ug/l (ppb) selenium+4.

Hunn et al. (1987) also conducted a 96-hour acute toxicity study using rainbow trout and waterborne sodium selenite. Water chemistry during that study included hardness of 40 mg/l (ppm) and pH of 7.2. The LC50 value determined by that study was 1.80 mg/l (ppm).

Hamilton and Buhl (1990) conducted three sets of experiments to determine the 24- and 96-hour LC50 for various life stages of fall-run chinook salmon (Oncorhynchus tshawytscha) and coho salmon (Oncorhynchus kisutch) exposed to waterborne selenate (as sodium selenate), selenite (as sodium selenite, Na2SeO3), seleno-DL-methionine, boron (as boric acid), and molybdenum (as sodium molybdate) in waters of three different qualities. Up to four life stages were tested in each set of experiments, including eyed eggs, alevins, swim-up fry (8-12 weeks old, post hatch), and advanced fry (14-21 weeks old). The first test water was simulated San Luis Drain effluent (containing the major anions and cations, but not the trace elements) diluted 10-fold in standardized freshwater (SLD/freshwater). Water chemistry in the SLD/freshwater included total hardness of 211 mg/l (ppm, as calcium carbonate), pH of 7.82, conductivity of 721 umhos/cm, and 185 mg/l (ppm) sulfate. The second test water was simulated San Luis Drain effluent diluted 22.5-fold in standardized brackish water (SLD/brackish water). Water chemistry the SLD/brackish water included total hardness of 333 mg/l (ppm, as calcium carbonate), pH of 7.79, conductivity of 2,887 umhos/cm, and 291 mg/l (sulfate. The third test water was standard soft water formulated in accordance with USEPA recommendations. Water chemistry in the soft water included total hardness of 41.7 mg/l (ppm, as calcium carbonate), pH of 7.57, conductivity of 157 umhos/cm, and 47 mg/l (ppm) sulfate.

The first set of experiments involved exposing the two fry life stages of chinook and coho salmon to individual trace elements in the SLD/freshwater of tests consisted of exposing the two fry life stages of chinook and coho salmon to mixtures of trace elements in the SLD/freshwater, SLD/brackish water, and soft

water. The third set of tests involved exposing eyed eggs, alevins, and swim-up fry life stages of chinook salmon to individual trace elements in the soft water. Results from single element tests with selenium and all interactive tests are discussed herein. Individual-effect results for boron and molybdenum are presented in their respective subsections.

The researchers found the intermediate life stag salmon to be more sensitive to the chemicals' toxic effects than the youngest (eyed eggs and alevins) and oldest (advanced fry) life stages. Coho salmon were more sensitive than chinook salmon. Waterborne selenite was more toxic than seleno-DL-methionine, which was more toxic than selenate. The LC50 values for selenite and selenate were not significantly different among water qualities.

Pooled LC50 values (in mg/l [ppm] for 24- and 96 hour tests, respectively) for chinook salmon exposed to selenium were as follows: 46.9 and 13.8 for selenite in SLD/freshwater and 65.6 and 23.4 in SLD/brackish water, 475 and 115 for selenate in SLD/freshwater and 484 and 149 in SLD/brackish water, and >21.6 for seleno-DL-methionine at both times and in both SLD/freshwater and SLD/brackish water. Pooled LC50 values (in mg/l [ppm] for 24- and 96-hour tests, respectively) for coho salmon exposed to selenium were as follows: 28.8 and 7.8 for selenite in SLD/freshwater and 44.1 and 13.6 in SLD/brackish water, and 234 and 32.5 for selenate in SLD/freshwater and >369 and 39.0 in SLD/brackish water. LC50 values (in mg/l [ppm] for 24- and hour tests, respectively) for chinook salmon exposed to selenate in soft water were as follows: >1,000 and >1,000 for eyed eggs, >320 and >320 for 4 for fry. LC50 values (in mg/l [ppm] for 24- and 96-hour tests, respectively) for chinook salmon exposed to selenite in soft water were as follows: >560 and >560 for eyed eggs; 202 and 104 for alevins; and 100 and 65.8, and 27.3 and 13.1 for two different sizes/ages of fry.

The interactive toxicities of various selenium mixtures, including those with added boron and/or molybdenum, were found to be additive. 96-hour LC50 values for chinook salmon exposed to various mixtures of selenate and selenite

ranged from 46.6 to 85.5 mg/l (ppm) in SLD/freshwater and from 51.9 to 96.8 mg/l (ppm) in SLD/brackish water. 96-hour LC50 values for coho salmon exposed to various mixtures of selenate and selenite ranged from 16.9 to 25.8 mg/l (ppm) in SLD/freshwater and from 26.2 to 38.0 mg/l (ppm) in SLD/brackish water.

The USFWS-NFCRC (Dec 1989) studied the effects of a 6:1 waterborne mixture of sodium selenate to sodium selenite on fall-run chinook salmon in 120-day toxicity tests. Swim-up life stage (0.5 g) fish were tested in reconstituted waters designed to mimic the major ionic concentrations (without the trace elements) of San Luis Drain effluent diluted 37-fold in standardized freshwater. Advanced fry life stage fish were tested in the same reconstituted waters diluted 22.5-fold in standardized brackish water (~1.2 ppth salinity). Preliminary results from these experiments reveal growth and survival of swim-up life stage fish were unaffected by selenium concentrations up to 140 ppb and advanced fry life stage fish exposed to 280 ppb selenium experienced significant growth reductions after 90 days and total mortality after 100 days.

The USFWS-NFCRC (Dec 1989; Dec 1987) conducted two sets of tests to evaluate effects upon juvenile fall-run chinook salmon of exposure to selenium in food or water. In the first study, fish were exposed for 30 days in freshwater to D,L-selenomethionine-enriched Oregon Moist Pellet diets containing the following concentrations of selenium (in ppm, wet weight): 0 (control), 15, 30, or 60. Twenty four-hour seawater (30 ppth salinity) challenge tests were conducted weekly during the 30-day period. Fish were then exposed to a selenium-free diet in seawater for an additional 90 days. In the second study, fish were exposed for 7 weeks in freshwater to a 6:1 waterborne mixture of sodium selenate and sodium selenite containing the following concentrations (in ug/l [ppb]): 0 (control), 35, 70, 140, 280, or 560. As in the first study, 24-hour seawater challenges were conducted weekly and, following the initial phase of the study, fish were exposed to selenium-free seawater for an additional 90 days.

Preliminary results from the first (dietary) study reveal that increasing dietary selenium concentrations resulted in reduced survival and reduced growth of fish in the fr the study; however, cumulative mortality in all treatment groups was less than 1%. Results of the seawater challenges showed that fish fed the 30 ppm selenium-2 diet experienced delayed osmoregulatory development and that those receiving the 60 ppm selenium-2 diet failed to develop any osmoregulatory ability. This latter group of fish also failed to exhibit normal gill Na+/K+ ATPase activity. Both the 30 and 60 ppm selenium-2 test groups also experienced increased mortality during the seawater challenges. Migratory behavior of fish from treatment groups was not significantly different than controls. During the 3-month seawater exposure, mortality of fish fed seleniferous diets was approximately 2-7 times higher than controls; however, growth of surviving fish was not different between treatment groups and controls.

Preliminary findings from the second (waterborne) study reveal that after 6 weeks in the freshwater phase of the study, there were no significant differences between control fish and those exposed to waterborne selenium in terms of histopathological changes, growth, or mortality. Neither did control and treatment fish (following 48 days of freshwater exposure) differ in downstream migration behavior, or in growth or survival for up to 3 months in seawater. Following 7 weeks of freshwater exposure, gill sodium/potassium ATPase activity was not significantly different between control and treatment groups exposed to seawater. The only difference between control and treatment groups noted in the study was a markedly higher mortality rate, following 24-hour seawater challenges, in fish exposed to all concentrations of waterborne selenium.

W.Wildlife (Water Concentrations vs. Wildlife or Domestic Animals):

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived for No-Observed-Adverse-Effect (NOAEL) levels (see Tis.Wildlife, B) for these). To be considered unlikely to represent an ecological risk, water

concentrations should be below the following benchmarks for each species present at the site [650]:

CAS 7782-49-2 SELENIUM (AS SELENATE)

SPECIES	WATER CONCENTRATION (ppm)
Mouse (test species)	0.00000
Short-tailed Shrew	0.42900
Little Brown Bat	0.74100
White-footed Mouse	0.27700
Meadow Vole	0.48500
Cottontail Rabbit	0.23000
Mink	0.23800
Red Fox	0.17000
Whitetail Deer	0.09500

Comment: Actually, the number of significant figures for a benchmark value should never be more than one; even if these values have been taken directly from another report, they should be rounded; otherwise the impression is given of a level of accuracy that is simply unwarranted. The uncertainties are too large to justify such a fine distinction (Owen Hoffman, SENES Oak Ridge, Personal Communication, 1997).

To protect livestock/cattle use, selenium levels should be less than 0.01 mg/L [671].

The Environmental Protection Agency (EPA) is developing a threshold value for dissolved waterborne selenium to protect wildlife using aquatic environments because it appears that the current ambient water quality criteria for selenium of 5 ppb established for aquatic species may not be adequate to protect wildlife.

W.Human (Drinking Water and Other Human Concern Levels):

EPA 1995 Region 9 Tap Water Preliminary Remediation Goal and Region 3 RBC for tap water: 180 ug/L [868,903].

EPA 1996 IRIS Database [893]:

Maximum Contaminant Level Goal (MCLG)

MCLG Value: 0.05 mg/L total selenium [893,952].

Status/Year: Final 1991 Econ/Tech?: No, does not consider economic or technical feasibility Reference: 56 FR 3526 (01/30/91)

Contact: Health and Ecological Criteria Division / (202)260-7571 Safe Drinking Water Hotline / (800)426-4791

Discussion: EPA has concluded that selenium should be placed in Category III and promulgates an MCLG of 0.05 mg/L based on a no-effect level obtained from a human study in China (Yang et al., 1989). Yang suggests that 0.400 mg of selenium/person/day is the maximum safe daily intake of selenium and assuming a a daily average consumption of 2 L drinking water per person containing 0.05 mg/L selenium, the resulting selenium ingestion would be 0.1 mg/person/day. The average daily dietary intake in this country is 0.125 mg selenium/person/day. A combined ingestion of water containing 0.05 mg/L and a typical U.S. diet would result in a total daily exposure of 0.225 mg selenium/person/day, well below the limit of 0.400 mg selenium that Yang et al. suggests is safe [893].

Maximum Contaminant Level (MCL) [893]:

Value: 0.05 mg/L total selenium

Status/Year: Final 1991 Econ/Tech?: Yes, does consider economic or technical feasibility Reference: 56 FR 3526 (01/30/91)

Contact: Drinking Water Standards Division / OGWDW / (202)260-7575 Safe Drinking Water Hotline / (800)426-4791

Discussion: EPA has promulgated an MCL equal to the MCLG of 0.05 mg/L.

Ambient Water Quality Criteria for Human Health [893]:

Water & Fish or Fish only: see discussion below. See also 45 FR 79318 (11/28/80); NTIS No. PB81-117814

Contact: Criteria and Standards

Division / OWRS / (202)260-1315

Discussion: The ambient water quality criterion for selenium is recommended to be identical to the existing water standard which is 10 ug/L [893].

Older Values for Water Quality Criteria for Human Health (10-6 Risk Level for Carcinogens) were the same:

Published Criteria for Water and Organisms: 10 ug/L [446,689].

For human health the ambient water quality criterion for selenium is recommended to be identical to the existing water standard which is 10 ug/l. Analysis of the toxic effects data resulted in the calculated level which is protective of human health against the ingestion of contaminated water and contaminated aquatic organisms. The calculated value is comparable to the present standard. For this reason a selective criterion based on exposure solely from consumption of 6.5 g of aquatic organisms was not derived. /Selenium and cmpd/ [366, USEPA/OWRS; Quality Criteria for Water 1986 (1986) EPA 440/5-86-001].

Criteria Federal Register Notice Number: 53 FR 177

Note: Before citing a concentration as EPA's water quality criteria, it is prudent to make sure you have the latest one. Work on the replacement for the Gold Book [302] was underway in March of 1996, and IRIS is updated monthyly [893].

State Drinking Water Standards [940]:

(AL) ALABAMA 10 ug/l [USEPA/Office of Water; Federal-State Toxicology and Risk Analysis

Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)].

(AZ) ARIZONA 10 ug/l [USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)].

State Drinking Water Guidelines [940]:

(AZ) ARIZONA 45 ug/l [USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)].

(ME) MAINE 10 ug/l [USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)].

(MN) MINNESOTA 20 ug/l [USEPA/Office of Water; Federal-State Toxicology and Risk Analysis Committee (FSTRAC). Summary of State and Federal Drinking Water Standards and Guidelines (11/93)].

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are indicated [715]. Exceedances of the criteria should be interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human RMC criteria for selenium in surface waters. These categories of humans not exposed to surface waters with concentrations of selenium exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Camp host: 1548 ug/L

Child Camper: 1422 ug/L
Boater: 5530 ug/L
Swimmer: 2395 ug/L

Human RMC criteria for selenium in ground water. These categories of humans not exposed to ground waters with concentrations of selenium exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 2 ug/L
Camp host: 18 ug/L
Child Camper: 51 ug/L
Worker: 39 ug/L
Surveyor: 387 ug/L

W.Misc. (Other Non-concentration Water Information):

Phytormediation: Plants "are very effective at removing selenium from contaminated soils," asserts Norman Terry of the University of California, Berkeley [1023]. They not only absorb the chemical, they also turn some of it into the less toxic dimethyl selenide gas [1023]. Selenate, the common form of selenium in soil, is about 600 times more toxic than dimethyl selenide gas [1023]. The company originally constructed the wetland, which features cattails and bulrushes, for its beauty [1023]. Now, the wetland is removing 70 to 75 percent of the selenium from the 10 million liters of wastewater that the company pumps through it every day, says Terry [1023]. In central California, at least two farmers are using Indian mustard and tall fescue to extract selenium from irrigation water, reports Gary S. Banuelos of the U.S. Department of Agricultures's Agricultural Research Service in Fresno, Calif [1023]. He advises the farmers on the phytoremediation technology [1023].

A potential complication in comparing contaminants data is that different investigators have sometimes meant different things when they put the words "dissolved" or "total" in front of a reported measurement. In the case of nutrients, the "dissolved" portion is usually simply that portion which has passed through a 0.45-micrometer membrane filter and the "total" measurements implies that it was not filtered and includes both dissolved and other forms of the nutrient [141]. However, usage of the words dissolved and total has not been uniform in the past and there is still considerable debate about which methods should truly be considered "dissolved" or "total" (Merle Schlockey, USGS, personal communication).

Water bodies are often marked by heterogeneity of the distribution of undissolved materials [691]. The size of any effects depends on the difference in density of the undissolved materials and the water, the size of the particles or bubbles of the materials, and various hydrodynamic factors such as the degree of turbulence in the water. Thus, undissolved inorganic materials in rivers and other natural water-bodies tend to increase in concentration with increasing depth because the particles tend to settle [691]. On the other hand, certain biological detritus may tend to rise towards the surface of the water because its density is less than that of water; oils also commonly demonstrate this effect markedly [691]. The surface microlayer is usually higher in concentration of many metallic and organic contaminants than the water column further down.

If the only change one makes is to use the prefix "dissolved" rather than the prefix "total" in an otherwise identical water quality standard, the effect can be a weakening of the standard related to total loading of a system. Many contaminants which are not currently dissolved can become dissolved at a later time, when encountering different conditions (perhaps downstream), such as changes in pH, additions of surfactants or humic substances, bioturbation, methylating organisms, and various other physical, chemical, or biological changes.

One problem with relying too heavily on dissolved fractions of metals is that the dissolved fraction misses the metals carried by colloids. Colloids were found to carry toxic metals 140 miles downstream of mining sources in Leadville, Colorado, to be repeatedly washed from flood deposited lowlands back into the river year after year in spring runoff (Briant Kimball, USGS Salt Lake City, as quoted in U.S. Water News, April 5th, 1995).

See Laboratory section below for EPA generic (guesstimate) conversion factors to convert total to dissolved concentrations.

Some environmental toxicologists make the argument that dissolved metals in surface water and porewaters represent most of what is bioavailable and thus "total" metals parameters are not good as a measure of potential biological effects. This is mostly true in many situations, but it should be kept in mind that fish and other aquatic organisms do not typically live in filtered water and that many fish and other aquatic organisms live in the sediments and in other situations in which they come in contact with toxic or otherwise harmful compounds (as certain colloids, precipitates, oxides, adsorbed metals), etc. Sometimes the effect of total metals is

partially related to physical or chemical aspects, such as when ferric oxide coats or covers benthic organisms. Another factor to consider: contaminants carried downstream by erosion of bottom sediments or colloids can be mobilized when they come in contact with different physical/chemical environments downstream (for example, a tributary bringing low pH into the system).

Misc. Notes on colloids (Briant Kimball, USGS, Salt Lake City Office, Personal Communication, 1995):

> There is no question that dissolved metals are critical to fish and invertebrates, but less well recognized is the potential impact and movement of metals in colloids. The possibility of having colloidal material present means there is a readily available supply of metals in a state in which the metals can quickly be reduced and mobilized. In river banks, reducing environments form just under the surface quickly. Toxic metals of concern would include zinc, lead, copper, and cadmium.
>
> Colloids do move in surface water (for example, transport of metal in colloids 140 miles downstream of Leadville, CO), but also in groundwater, especially related to radionuclides.
>
> Colloidal metals may effect biota more than is widely recognized. Brown trout are effected by colloids which travel kind of like dissolved fractions, don't settle out. There may be little understood colloidal pathways of metals to fish, for example. Colloidal metals become part of the caddis cast which are ingested, once part of acid gut, metals can be released. On the Arkansas River of Colorado below Leadville, the dissolved metals have gone down with treatment, but Will Clements of CSU has discovered the toxicity has not been reduced to the same extent as have the dissolved metals. Treatment has not eliminated colloidal fractions loaded with cadmium and copper, and this is possibly impacting the fish.
>
> In rivers, there is annual flushing of the colloids, loads are much greater during runoff.

Sediment Data Interpretation, Concentrations and Toxicity (All

Sediment Data Subsections Start with "Sed."):

Sed.Low (Sediment Concentrations Considered Low):

Selenium concentrations in freshwater sediments range from 0.20 to 2.00 mg/kg (ppm) [445].

Sed.High (Sediment Concentrations Considered High):

Texas: The statewide 90th percentile value for selenium was 1.9 mg/kg dry weight [7].

Selenium concentrations in freshwater sediments range from 0.20 to 2.00 mg/kg (ppm) [445].

Analyses of sewage sludges from 50 publicly owned treatment works by the U.S. Environmental Protection Agency (1985): The mean concentration of selenium was 2.6 ppm (dry weight) [347].

Analyses of 74 Missouri sludges (1985): The mean for selenium was 3 ppm. The range was 1-25 ppm (dry weight) [347].

Sed.Typical (Sediment Concentrations Considered Typical):

No information found.

Sed.Concern Levels, Sediment Quality Criteria, LC50 Values, Sediment Quality Standards, Screening Levels, Dose/Response Data and Other Sediment Benchmarks:

Sed.General (General Sediment Quality Standards, Criteria, and Benchmarks Related to Protection of Aquatic Biota in General; Includes Sediment Concentrations Versus Mixed or General Aquatic Biota):

USFWS: The toxic threshold for selenium transferred to consumer species of fish and wildlife is 3 mg/kg [648].

Sed.Plants (Sediment Concentrations vs. Plants):

No information found.

Sed.Invertebrates (Sediment Concentrations vs. Invertebrates):

Where sediments contain 6-15 ppm selenium (dry weight), the invertebrates may have up to 50 ppm selenium (dry weight) which is a concentration that would be directly toxic to adult fish and waterfowl (Dennis Lemly, Fish and Wildlife Service, National

Fisheries Contaminant Research Center, personal communication, 1991).

Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

Sed.Fish (Sediment Concentrations vs. Fish):

Where sediments contain 6-15 ppm selenium (dry weight), the invertebrates may have up to 50 ppm selenium (dry weight) which is a concentration that would be directly toxic to adult fish and waterfowl (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

Sed.Wildlife (Sediment Concentrations vs. Wildlife or Domestic Animals):

Where sediments contain 6-15 ppm selenium (dry weight), the invertebrates may have up to 50 ppm selenium (dry weight) which is a concentration that would be directly toxic to adult fish and waterfowl (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

USFWS: The toxic threshold for selenium transferred to consumer species of fish and wildlife is 3 mg/kg

[648].

Sed.Human (Sediment Concentrations vs. Human):

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are indicated [715]. Exceedances of the criteria should be interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human RMC criteria for selenium in sediments. These categories of humans not exposed to sediments with concentrations of selenium exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Camp host: 774 mg/kg
Child Camper: 356 mg/kg
Boater: 2765 mg/kg
Swimmer: 1197 mg/kg

Sed.Misc. (Other Non-concentration Sediment Information):

No information found.

Soil Data Interpretation, Concentrations and Toxicity (All Soil Data Subsections Start with "Soil."):

Soil.Low (Soil Concentrations Considered Low):

No information found.

Soil.High (Soil Concentrations Considered High):

Analyses of sewage sludges from 50 publicly owned treatment works by the U.S. Environmental Protection Agency (1985): The mean concentration of selenium was 2.6 ppm (dry weight) [347].

Analyses of 74 Missouri sludges (1985): The mean for selenium was 3 ppm. The range was 1-25 ppm (dry weight) [347].

Most seleniferous soils contain less than 2 ppm selenium [953].

Soil.Typical (Soil Concentrations Considered Typical):

Selenium is more abundant and more bioavailable (mostly due to natural geological circumstances), than in the eastern United States [463].

Typical Igneous Rocks (Earth's Crust) Concentrations: EPA 1981: 0.05 mg/kg dry weight [83].

Typical Soil Concentrations: EPA 1981: 0.2 mg/kg dry weight [83].

Selenium concentrations in soils around the world range widely, from areas of selenium deficiency to those with seleniferous soils (0.1-1,200 mg/kg [ppm], respectively), and average 0.4 ppm [445].

Quebec considers 1 ppm as background [347].

The mean elemental concentration of this metal in plants was 0.1 ppm in the same areas where rocks were 1 ppm [951]. Concentration in soils is 0.5 ppm [951].

Selenium concentration in geological materials: Igneous rocks 0.05 ppm; shales 0 - 0.6 ppm; sandstones 0.0 - 0.05 ppm; limestones 0.08 ppm; soils 0.2 ppm (Wilber CG; Clin Toxicol 17 (2): 171-230, 1980) [366].

The earth's crust is said to have an average selenium concentration of 0.03 of 0.08 ppm (Wilber CG; Clin Toxicol 17 (2): 171-230, 1980) [366].

Soil.Concern Levels, Soil Quality Criteria, LC50 Values, Soil Quality Standards, Screening Levels, Dose/Response Data and Other Soil Benchmarks:

Soil.General (General Soil Quality Standards, Criteria, and Benchmarks Related to Protection of Soil-dwelling Biota in General; Includes Soil Concentrations Versus Mixed or General Soil-dwelling Biota):

Other Maximum Allowable Concentration (MAC) levels of selenium (ppm dry weight): 10 (Stuttgart), 3 (London) [719]

Proposal of Ontario Ministry of Agriculture and Food for MAC in soils treated with sewage sludge: 1.6 ppm dry weight (published in Tokyo; work done for Ontario) [719].

The 1987 soil (clean up) criteria given by the New Jersey Department of Environmental Protection for selenium is 4 mg/kg dry weight [347,386]. Quebec considers 1 ppm as background, 3 ppm as moderately contaminated soils, and 10 ppm as a threshold that requires immediate cleanup [347]. Ontario considers 1.6 ppm selenium as the maximum for proposed redevelopment as agriculture and 5 ppm as the maximum for proposed redevelopment as residential or parkland [347].

Quebec considers 3 ppm as moderately contaminated soil, and 10 ppm as a threshold that requires immediate cleanup [347].

Suggested safe applications (kg/ha) of trace compounds to Missouri soils without further investigations (1988): The maximum cumulative addition of selenium should not exceed 18 kg/ha [347].

Soil.Plants (Soil Concentrations vs. Plants):

Levels of selenium (ppm dry weight) considered phytotoxic: 10 (Vienna), 10 (Warsaw), 10 (Warsaw), 5 (Ontario) [719].

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Terrestrial Plants. To be considered unlikely to represent an ecological risk to terrestrial plants, field concentrations in soil should be below the following dry weight benchmark for soil [651]:

> For CAS 007782-49-2 (SELENIUM), the benchmark is 1 mg/kg in soil (WILL and SUTER, 1994).

Soil.Invertebrates (Soil Concentrations vs. Invertebrates):

No information found.

Soil.Wildlife (Soil Concentrations vs. Wildlife or Domestic Animals):

No information found.

Soil.Human (Soil Concentrations vs. Human):

EPA 1996 National Generic Soil Screening Level (SSL) designed to be conservative and protective at the majority of sites in the U.S. but not necessarily protective of all known human exposure

pathways, land uses, or ecological threats [952]:

SSL = 390 mg/kg for ingestion pathway [952].

SSL = None given for inhalation pathway [952].

SSL = 0.3 to 5 mg/kg for protection from migration to groundwater at 1 to 20 Dilution-Attenuation Factor (DAF) [952].

EPA 1995 Region 9 Preliminary remediation goals (PRGs), 1995 [868]:

Residential Soil: 380 mg/kg wet wt.
Industrial Soil: 8500 mg/kg wet wt.

NOTE:
1) PRGs focus on the human exposure pathways of ingestion, inhalation of particulates and volatiles, and dermal absorption. Values do not consider impact to groundwater or ecological receptors.
2) Values are based on a non-carcinogenic hazard quotient of one.
3) PRGs for residential and industrial landuses are slightly lower concentrations than EPA Region III RBCs, which consider fewer aspects [903].

EPA 1995 Region 3 Risk based concentration (RBC) to protect from transfers to groundwater:

None given [903].

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are indicated [715]. Exceedances of the criteria should be interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human RMC criteria for selenium in soil. These categories of humans not exposed to soil with concentrations of selenium exceeding the below RMCs are not expected to experience

adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 10 mg/kg
Camp host: 258 mg/kg
Child Camper: 178 mg/kg
ATV Driver: 3629 mg/kg
Worker: 387 mg/kg
Surveyor: 3871 mg/kg

Soil.Misc. (Other Non-concentration Soil Information):

Misc. Notes Related to Selenium Bioavailability in Soils [463]:

In areas of the Western United States (states west of and including North Dakota, South Dakota, Nebraska, Kansas, Oklahoma, and Texas), where geological sources of selenium are abundant in the soil profile, selenium tends to be more bioavailable to ecosystems.

A major feature of this area is the presence of pedocal soils. Pedocals are alkaline soils of semiarid and arid climates. Bacterial and chemical processes in these highly oxidizing, alkaline soils favor the formation of calcium and sodium selenates, which are very mobile and water soluble, and are readily available forms of selenium to plants. In addition, many areas in the Western United States are prone to selenium enrichment of the soil because of leaching of underlying seleniferous rocks, such as the shales and clays of the Upper Cretaceous Pierre, Steele, and Niobrara Shales.

Pedalfer soils found in the Eastern United States are acid to neutral soils of humid and semihumid climates. These acid soils favor the formation of more reduced and complexed forms of selenium, such as ferric selenite. These complexes are generally insoluble so as to reduce selenium bioavailability to the point that forages and feeds grown in the Eastern United States contain insufficient selenium for proper animal nutrition.

In short, in areas in the Western United States where selenium is abundant and bioavailable, a more detailed analysis may be required for selenium toxicity than would be required in the east [463].

Phytormediation: Plants "are very effective at removing selenium from contaminated soils," asserts

Norman Terry of the University of California, Berkeley [1023]. They not only absorb the chemical, they also turn some of it into the less toxic dimethyl selenide gas [1023]. Selenate, the common form of selenium in soil, is about 600 times more toxic than dimethyl selenide gas [1023]. See also additional detail in W.Misc. section above.

See also Fate.Detail section below.

Tissue and Food Concentrations (All Tissue Data Interpretation Subsections Start with "Tis."):

Tis.Plants:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Plants:

Selenium is found in livestock feeds [445]. The toxic effects of selenium on livestock have been known for some time [445]. Grazing in seleniferous areas and/or on selenium-accumulator plants has been associated with acute and chronic maladies known as the blind staggers and alkali disease, respectively (although there is some debate regarding whether the blind staggers is actually caused by excess intake of selenium or some other toxic property[ies] of forage such as locoweed [a plant of either the genera Oxytropis or Astragalus which causes severe poisoning when eaten by livestock]) [445].

Livestock foraging on plants containing about 25 ppm selenium suffer from alkali disease, characterized by a lack of vitality, loss of hair, sterility, atrophy of hooves, lameness, and anemia [491].

B) Body Burden Residues in Plants: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

The mean elemental concentration of this metal in plants was 0.1 ppm in the same areas where rocks were 1 ppm [951].

See also Tis.Misc section below.

The contents of copper, molybdenum, sulphur, zinc, selenium, iron, manganese, and the copper/molybdenum ratio were determined in different native plant species from a mountain area of central southern Norway. The overall mean

values and ranges (mg/kg DM) were copper: 6.0, 0.9-27.2; molybdenum: 0.25, 0.01-3.57; zinc: 77, 8-320; selenium: 0.05, less than 0.01-0.32; iron: 208, 15-2245; manganese: 338, 31-3784; sulfur: (g/100 g DM) 0.20, 0.03-0.56; copper/molybdenum: 79, 1-7955. Levels of the individual elements showed considerable variability, both between and within plant groups. Mineral contents were compared with the established requirements for sheep and cattle, the following conclusion being drawn. The levels of zinc, sulphur, iron, and manganese were found to be adequate for ruminants. [Garmo TH et al; Acta Agric Scand 36 (2): 147-161 (1986)] [940].

Tis.Invertebrates:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Invertebrates:

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas:

> Predator Protection Level: The 0.5 mg/kg predator protection level [20] was exceeded in 6 of 77 Trinity River samples. Included in this group were samples of Mississippi map turtles, mosquitofish, carp, spiny softshell turtles, and unionid clams, all from sites downstream of Dallas except for the mosquitofish, which were from site 20.

B) Concentrations or Doses of Concern in Food Items Eaten by Invertebrates:

No information found.

C) Body Burden Residues in Invertebrates: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

The following information summarizes data gathered from the NOAA National Status and Trends (NS&T) Program for the year 1990 [697]:

> For selenium in mussels and oysters combined (n=214), the Geometric Mean was 2.5 ug/g dry and the "high" concentration was 3.5 ug/g dry weight [697]. NOAA "high" concentrations are equal to the geometric mean plus one standard deviation on the log normal distribution [696].

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas:

> Maximum Level: The highest selenium concentration was 0.71 mg/kg. This concentration was found in a composite sample of unionid clam flesh from site 14.

USFWS: Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication to Roy Irwin, 1991). Where sediments contain 6-15 ppm selenium (dry weight), the invertebrates may have up to 50 ppm selenium (dry weight) which is a concentration that would be directly toxic to adult fish and waterfowl (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication, 1991).

Tis.Fish:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Fish (Includes FDA Action Levels for Fish and Similar Benchmark Levels From Other Countries):

Legal Limits for Concentrations in Fish and Fishery Products: The lowest legal limit was 0.05 mg/kg (Chile) [216,418]. Three countries have limits less than or equal to 2.0 mg/kg, but the U.S. apparently has no limit [216,418].

The California Department of Health Services recommended a maximum allowable residue level of 1.0 mg/kg wet weight for muscle (fillet) tissue of edible fish [222]. It was from this that the water concentration concern level of 0.8 ppm was derived (through back calculation from the tissue concern level) [222].

Region III EPA RBC value for fish tissue: 6.8 mg/kg [903].

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas):

> Predator Protection Level: The 0.5 mg/kg predator protection level [20] was exceeded in

6 of 77 Trinity River samples. Included in this group were samples of Mississippi map turtles, mosquitofish, carp, spiny softshell turtles, and unionid clams, all from sites downstream of Dallas except for the mosquitofish, which were from site 20.

Bureau of Land Management RMC Benchmarks, 1995: Risk Management Criteria (RMC) were developed for the mostly dry BLM lands in the western U.S. These risk management criteria should be used by the land manager as a cautionary signal that potential health hazards are present and that natural resource management or remedial actions are indicated [715]. Exceedances of the criteria should be interpreted as follows [715]:

Less than criteria: low risk
1-10 times the criteria: moderate risk
10-100 times the criteria: high risk
>100 times the criteria: extremely high risk

Human RMC criteria for selenium in fish consumed by humans. These categories of humans not exposed to fish with concentrations of selenium exceeding the below RMCs are not expected to experience adverse toxic effects [715]:

Child resident (living on properties adjacent to BLM lands): 392 ug/kg
Camp host: 807 ug/kg
Child Camper: 2222 ug/kg

B) Concentrations or Doses of Concern in Food Items Eaten by Fish:

The effects of dietary and waterborne selenium on bluegill sunfish (Lepomis macrochirus) were studied by Woock et al. (1987) [445]. Mortalities of adults were significant in both 30 ppm diet groups.

Toxic effects have been documented in fish consuming diets containing 10 to 33 mg/kg selenium, concentrations similar to those in prey organisms from selenium impacted habitats [481]. Dietary Se-methionine is more toxic to fish than dietary inorganic selenium [481].

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> of oganic and inorganic selenium in a laboratory food chain." Copyright 1993 SETAC].

Selenium whole-body levels above 0.5 mg/kg are considered harmful to fish and predators [20].

However, some more recent researchers have concluded that harmful effects of selenium on predators has seldom been documented for concentrations below 3 mg/kg and often been documented for concentrations above 30 mg/kg (the "3/30 guideline", Jerry Miller, U.S. Bureau of Reclamation, Salt Lake City, personal communication, 1994). For example:

> Hamilton et al. (1990) suggest that, in order to be safe for fish, dietary concentrations of selenium should be less than 3 ug/g (ppm, dry weight) [445]. USFWS: The toxic threshold for selenium transferred to consumer species of fish and wildlife is 3 mg/kg (ppm) [648].
>
> The effects of dietary and waterborne selenium on bluegill sunfish (Lepomis macrochirus) were studied by Woock et al. (1987) [445]. Mortalities of adults were significant in both 30 ppm diet groups.

Toxic effects have been documented in fish consuming diets containing 10 to 33 mg/kg selenium, concentrations similar to those in prey organisms from selenium impacted habitats [481]. Dietary Se-methionine is more toxic to fish than dietary inorganic selenium [481].

> Text in paragraph above reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, J.M. Besser, T.J. Canfield and T.W. La Point, "Bioaccumulation of oganic and inorganic selenium in a laboratory food chain." Copyright 1993 SETAC].

Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication to Roy Irwin, 1991).

Ninety day survival of fish was reduced in fish fed a diet containing > or = 9.6 ug of Se/g, and reduced growth was noted in a diet containing > or

= 5.3 ug/g (ppm) [195].

> Text in paragraph above reprinted with permission from Environmental Toxicology and Chemistry, Volume 9, S.J. Hamilton, K.J. Buhl, N.L. Faerber, R.H. Wiedmeyer and F.A. Bullard, "Toxicity of organic selenium in the diet to chinook salmon." Copyright 1990 SETAC].

Reduced fish growth, whole-body concentrations of selenium and survival were strongly correlated to concentrations of selenium in diets (Hamilton, 1990) [445].

C) Body Burden Residues in Fish: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

The toxic effect threshold of selenium for health and reproduction is 4 mg/kg dry weight, whole body [648].

In order to protect fish reproduction, their skeletal muscles should not contain more than 5 ppm and their organs (liver and gonads) should not contain more than 10 ppm selenium (both concentrations are for total selenium, dry weight) (Wallenstrom, Aug 1986) [445].

An estimate of a no effect selenium concentration in fish was a whole-body concentration of 1.1 mg/kg wet weight [222]. This was the no effect level for the fish itself, not predators which might be eating many fish of the same species. A predator protection level based on this data would therefore have to be well below 1.1 mg/kg wet weight.

The geometric mean of whole-body (wet weight) concentrations of fish in a 1980-1981 national survey was 0.47 mg/kg [23]. A more recent (1976-1984) NCBP survey report gave the national geometric mean level for selenium in whole-body fish as 0.42 mg/kg, the maximum level as 2.3 mg/kg, and the 85th percentile level as 0.73 mg/kg wet weight [384].

Whole body levels above 1.0 are not unusual in some areas [488]. At polluted sites, selenium whole-body levels are often above 4.28 mg/kg dry weight and liver (hepatic) levels are often above 6.7 mg/kg dry weight [488]. Normally, muscle contains between 0.6 and 0.6 ppm and ovaries vary between 5.9 and 12.1 ppm dry weight [488].

The range of concentrations of selenium in a

studies of edible fish tissues in Pennsylvania in 1977 (included sites which were not especially clean) was from ND to 3.34 mg/kg wwt [57].

Whole body concentrations of selenium in mosquitofish from highly contaminated areas of California were twice as high as the highest reported liver concentrations from the literature [488]. A North Carolina study of several species of fish suggested the highest concentrations of selenium were in the liver [488]. A separate literature review that the liver of selenium-exposed fish accumulates higher levels of selenium than skeletal muscles. However, selenium concentrations in the liver typically correlate with selenium concentrations in other tissues, even though the distribution patterns within tissues, as well as absolute values of selenium, vary considerably with the species considered [488].

Some references state that selenium accumulates in the axial muscles of fish, so fillet levels are typically closer to whole-body concentrations than are most other contaminants [27,136]. A more recent, and somewhat contrasting summary documents the tendency of selenium to concentrate more highly in the liver, gonads, and kidneys of fish than in muscle [488]. Selenium accumulates in the gonads of bass and bluegills [36].

Sunfish exposed to aqueous selenium accumulate selenium in the liver (7-27 ppm, wet weight) and skeletal muscle, although the levels in skeletal muscle are about half of those in the liver [488].

Selenium concentrations in muscle samples from trout and dace from the Pecos River near Pecos National Monument & Historical Park were 1.9 and 13.6 mg/kg wwt (Milford Fletcher, National Park Service, Personal Communication). These two samples were had higher concentrations of selenium concentrations than the concentrations (0.29 to 1.41 mg/kg wwt) reported for trout collected upstream in the Fish and Wildlife Service 1991 study of the Terrero Mine waste study area [479]. Two other Pecos River fish muscle samples had selenium concentrations of <1.0 mg/kg (Milford Fletcher, National Park Service, Personal Communication).

An extremely high mean of 197 ppm dry weight was reported for whole-body samples of mosquito fish from a polluted area in California, a level so high is it twice as high as the highest liver

concentrations previously published [488]. Some of the high concentrations in mosquitofish from polluted areas in California prompted one wag to joke that letting the contaminated mosquitofish dry out on a sidewalk would be creating a material classified as a "hazardous waste."

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas):

> Due to cost, we analyzed selenium in only 50 Trinity River samples. Selenium was found above the detection limit (0.09 mg/kg) in all but one sample.
>
> Gradient Monitoring Levels: The highest concentration of selenium in 24 samples of Trinity River mosquitofish was 0.52 mg/kg (site 20). For contrast, mosquitofish from a pond severely contaminated by agricultural drainage at Kesterson National Wildlife Refuge in California had selenium concentrations ranging from 26 to 98 mg/kg [77,139].

Tis.Wildlife: Terrestrial and Aquatic Wildlife, Domestic Animals and all Birds Whether Aquatic or not:

A) As Food: Concentrations or Doses of Concern to Living Things Which Eat Wildlife, Domestic Animals, or Birds:

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas:

> Predator Protection Level: The 0.5 mg/kg predator protection level [20] was exceeded in 6 of 77 Trinity River samples. Included in this group were samples of Mississippi map turtles, mosquitofish, carp, spiny softshell turtles, and unionid clams, all from sites downstream of Dallas except for the mosquitofish, which were from site 20.

B) Concentrations or Doses of Concern in Food Items Eaten by Wildlife, Birds, or Domestic Animals (Includes LD50 Values Which do not Fit Well into Other Categories, Includes Oral Doses Administered in Laboratory Experiments):

See also [201] information in Tis.Wildlife, A) above.

Oak Ridge National Lab, 1994: Risk Assessment Screening Benchmarks for Wildlife derived from No-Observed-Adverse-Effect (NOAEL) levels (mg contaminant per kg body weight per day). To be considered unlikely to represent an ecological risk, wet-weight field concentrations should be below the following (right column) benchmarks for each species present at the site [650]:

For CAS 7782-49-2, SELENIUM (AS SELENATE):

SPECIES	NOAEL (mg/kg/day)	FOOD CONCEN-TRATION (ppm)
Mouse (test species)	0.07500	0.00000
Short-tailed Shrew	0.09400	0.15700
Little Brown Bat	0.11900	0.35600
White-footed Mouse	0.08300	0.53800
Meadow Vole	0.06600	0.58200
Cottontail Rabbit	0.02200	0.11200
Mink	0.02400	0.17200
Red Fox	0.01400	0.14400
Whitetail Deer	0.00600	0.20200

Comment: Actually, the number of significant figures for a benchmark value should never be more than one; even if these values have been taken directly from another report, they should be rounded; otherwise the impression is given of a level of accuracy that is simply unwarranted. The uncertainties are too large to justify such a fine distinction (Owen Hoffman, SENES Oak Ridge, Personal Communication, 1997).

Skorupa et al. (Mar 1989) evaluated data developed in both laboratory and field studies and estimated that the maximum acceptable toxicant concentration of dietary selenium for aquatic birds was 5.6 ppm, dry weight [445].

In an attempt to reduce wildlife contamination hazards, the California Department of Fish and Game adopted a selenium standard for subsurface agricultural drainage water evaporation ponds in the San Joaquin Valley. The CDFG standard requires initiation of special management actions (e.g., hazing) when the selenium concentration in a composite sample of aquatic invertebrates from a pond equals or exceeds 4 ppm, dry weight [445].

Selenium whole-body levels above 0.5 mg/kg are considered harmful to fish and predators [20].

However, some more recent researchers have concluded that harmful effects of selenium on predators has seldom been documented for concentrations below 3 mg/kg and often been documented for concentrations above 30 mg/kg (the "3/30 guideline", Jerry Miller, U.S. Bureau of Reclamation, Salt Lake City, personal communication, 1994).

USFWS: The toxic threshold for selenium transferred to consumer species of fish and wildlife is 3 mg/kg [648].

The toxic effect threshold of selenium for health and reproduction is 4 mg/kg dry weight, whole body [648]. Aquatic sediments containing more than 3 ppm selenium (dry weight) usually cause selenium levels in benthic invertebrates to exceed safe concentrations (3-5 ppm dry weight for fish and wildlife that feed upon them (Dennis Lemly, Fish and Wildlife Service, National Fisheries Contaminant Research Center, personal communication to Roy Irwin, 1991).

An estimate of a no effect selenium concentration in fish was a whole-body concentration of 1.1 mg/kg wet weight [222]. This was the no effect level for the fish itself, not predators which might be eating many fish of the same species. A predator protection level based on this data would therefore have to be well below 1.1 mg/kg wet weight.

Livestock foraging on plants containing about 25 ppm selenium suffer from alkali disease, characterized by a lack of vitality, loss of hair, sterility, atrophy of hooves, lameness, and anemia [491].

Information from Moore [445]:

> See Moore [445] for details on citations:
>
> Smith et al. (1988) studied the effects of dietary selenium-2 (as seleno-DL- methionine) on black-crowned night-herons (Nycticorax nycticorax). Thirteen and 23 days, respectively, prior to egg laying, pairs of adult herons were placed on 10 or 30 ppm (dry weight) selenium-2 commercial diets (containing 7-10% moisture). Birds on control diets received 0.1 ppm selenium (dry weight). Adults were necropsied after an average of 92 days on the test diets and hatchlings were euthanized at three days of age. Biological

endpoints measured during the study included: weight loss; reproductive success; eggshell thickness; hatchling survival; and various morphological, hematological, and biochemical parameters.

Organ weights, hemoglobin concentrations, hematocrits, egg fertility, eggshell thickness, the Ratcliffe Index of eggshell quality, and 72-hour hatchling survival were unaffected in adults receiving either the 10 or 30 ppm selenium-2 diet. However, adult herons fed the 30 ppm selenium-2 diet lost significantly more weight than herons fed other diets. Hatching success was not different from controls, and teratogenesis was not observed and hematology was unaffected in embryos/hatchlings produced by adults fed the 10 ppm selenium-2 diet, however their hatchlings did have significantly shorter femur and radius-ulna lengths and significantly higher liver malondialdehyde concentrations.

Martin (1988) conducted four experiments to assess the effects of selenium and boron on avian reproduction. One experiment involved feeding <1-year- old Japanese quail (Coturnix japonica) laying ration control diets or such diets supplemented with selenium+4 (as sodium selenite), selenium-2 (as selenomethionine), or boron (as sodium borate) to achieve the following dietary concentrations: 10, or 15 ppm selenite; 5 or 8 ppm selenomethionine; or 25, 50, or 100 ppm borate (all values in dry weight). In another experiment, fresh, fertile Pekin duck eggs were injected with 0.1 ml of saline solutio of the following concentrations of selenium+4 (as sodium selenite): 0.0, 0.3, 0.4, 0.6, 0.8, 1.0, 1.4, 1.8, or 2.2 ppm. The remaining experiments involved Single Comb White Leghorn chickens (Gallus domesticus) in dietary and egg-injection exposures to selenium+4 (as sodium selenite), selenium-2 (as selenomethionine), and/or boron (as sodium borate). In the chicken dietary study, hens were fed either clean laying ration (control) diets or such diets supplemented with selenium and/or boron to achieve one of the following concentrations: 12 ppm selenite, 12 ppm selenite plus 500 ppm borate, 10 ppm selenomethionine, or 10 ppm selenomethionine plus 500 ppm borate (all values in dry weight). In the chicken egg-injection study,

fresh, fertile eggs injected with one of the following concentrations of selenium and/or boron: 0.0, 0.6, 0.8, 1.0, 1.2, 1.4, 1.6, or 1.8 ppm selenite; 0.3, 0.5, or 1.0 ppm selenomethionine; 0.0, 11.3, 15.8, 16, or 18.1 ppm borate; 1.8 ppm selenite plus 16 ppm borate; or 0.5 ppm selenomethionine plus 16 ppm borate. Food and water were provided ad libitum in all studies.

The results of the selenium tests, both singly and in combination with boron, are discussed herein. The independent results of boron are discussed in boron subsection.

In the quail study, all concentrations of both forms of selenium- supplemented diets resulted in abnormal embryos, significantly reduced hatchability of fertile eggs, and possibly increased mortality. The percent hatchability of eggs and the percent of abnormal embryos, respectively, at each dietary concentration were as follows: 15 ppm selenite, 25.6% and 70.3%; 10 ppm selenite, 10.2% and 83.9%; 7.5 ppm selenite, 65.6% and 8.8%; 8 ppm selenomethionine, 10.4% and 66%; and 5 ppm selenomethionine, 56.4% and 14%.

In the duck study, hatchability of fertile eggs was significantly reduced at injected selenite concentrations of >0.6 ppm. No abnormal embryos were found in the control eggs, however abnormal embryos were discovered in all but one (0.4 ppm) of the selenite treatment groups. Embryo abnormality and mortality generally increased with increasing concentrations of injected selenite.

In the dietary chicken study, egg production generally decreased when birds were placed on the treatment diets. Egg hatchability was significantly reduced and abnormal embryos were produced by all the treatment diets except the selenite diet. The percent hatchability of eggs and the percent of abnormal embryos, respectively, at each dietary concentration were as follows: 12 ppm selenite, 84.9% and 0%; 12 ppm selenite plus 500 ppm borate, 51.9% and 14.9%; 10 ppm selenomethionine, 23.2% and 33.8%; and 10 ppm selenomethionine plus 500 ppm borate, 27.7% and 25%.

In the chicken egg-injection study, no

abnormal embryos were found in the control groups; however, abnormal embryos were produced by injecting >0.8 ppm selenomethionine. Egg hatchability was significantly reduced by injecting >1.8 ppm selenite or >0.5 ppm selenomethionine. Injections of borate with either selenite or selenomethionine produced both abnormal embryos and significantly reduced egg hatchability.

Hoffman and Heinz (1988) reviewed the effects on plasma biochemistry of dietary sodium selenite and selenomethionine on mallard (Anas platyrhynchos) hatchlings (see study by Heinz et al., 1987). Plasma glutathionine peroxidase activity was significantly increased in mallards fed 5, 10, and 25 ppm sodium selenite and those fed 10 ppm selenomethionine. Mallards fed 25 ppm sodium selenite also experienced increased concentrations of plasma uric acid (an indication of altered renal function) and sorbitol dehydrogenase activity (an indication of liver toxicity) was increased in birds fed 10 ppm selenomethionine.

Hoffman et al. (1989) studied the effects of dietary selenium on the physiology of mallard ducklings, specifically hepatic glutathione metabolism and lipid peroxidation. One-day-old ducklings were provided with duck mash diets (containing 7-9% moisture) with added vitamin E and zinc, and supplemented with selenium+4 (as sodium selenite) or selenium-2 (as seleno-ethionine), creating final dietary selenium concentrations of 0.1, 10, 20, and 40 ppm. After six weeks, selenomethionine accumulated to higher concentrations in the liver and more strongly affected hepatic glutathione metabolism and lipid peroxidation than did selenite. The 20 and 40 ppm selenomethionine diets significantly increased the ratio of oxidized to reduced hepatic glutathione and significantly increased hepatic lipid peroxidation (as estimated by malondialdehyde concentrations). The ratio of oxidized to reduced hepatic glutathionine was unaffected by the selenite diets; however, the 40 ppm selenite diet significantly increased hepatic lipid peroxidation.

Heinz and Gold (1987) studied the behavior of 6-lard ducklings whose parents were fed diets

supplemented with selenium-2. Pairs of adult mallards were fed a control diet or diets supplemented with selenium-2 (as seleno-DL-methionine) to achieve final dry weight concentrations of 1, 2, 4, or 8 ppm. The adults received these diets beginning at least four weeks prior to egg laying and ducklings were fed untreated commercial duck starter mash. Six days after hatching, ducklings were exposed to a fright stimulus. The authors determined that there were no significant differences in response to the fright stimulus among ducklings whose parents were fed the control or selenium-laden diets.

Heinz et al. (1987) and Hoffman and Heinz (1988) studied the effects of dietary selenium+4 (as sodium selenite) and selenium-2 (as seleno-DL-methionine) on adult mallards and their young. Beginning at least four weeks prior to the laying of the first eggs, mallard pairs were fed commercial duck mash diets (with 7-10% moisture) nominally containing: 0 ppm selenium (the control diet); 1, 5, 10, 25, or 100 ppm selenium+4; or 10 ppm selenium-2. Ducklings were reared on duck mash diets containing the same selenium concentrations as in their parents' diets. Most adults were killed following laying of all 31 eggs needed for the study and surviving ducklings were killed at three weeks of age. Biological endpoints measured during the study included: adult survival; weights of adults, eggs, and hatchlings; eggshell quality; egg fertility; hatching success of fertile eggs; and number of 21-day-old hatchlings produced and hatchling survival to 21 days.

Percent fertility, hatching success of fertile eggs, and weights of eggs laid were not significantly different among treatment groups. Adults fed 100 ppm selenium+4 experienced significant weight loss and mortality and produced eggs prior to death. Adults fed 25 ppm selenium+4: experienced significant weight loss; produced 44% fewer eggs that hatched; produced smaller embryos and ducklings, and a significantly lower percentage and number of ducklings that survived to 21 days of age; produced a significant percentage of abnormal embryos (22.2%); and produced poorer quality eggshells. Adults fed 10 ppm selenium+4 produced a significant percentage of abnormal

embryos (11.2%). Adults fed 10 ppm selenomethionine: produced 40% fewer eggs that hatched; produced a significantly lower percentage and number of ducklings that survived to 21 days of age; and produced a significant percentage of abnormal embryos (18.3%), many more of which (a total of 13.1%) were teratogenic than those abnormalities produced in the selenite treatment groups. The effects of two seleno-amino acids in the diets of mallard ducks were studied by Heinz et al. (1989) and Hoffman and Heinz (1988). Beginning three weeks prior to pairing and continuing through egg laying, adult mallards were fed duck mash diets (containing ~10% moisture) supplemented with seleno-DL-methionine or seleno-DL-cystine to achieve final dietary concentrations of 0.1-0.2, 1, 2, 4, 8, or 16 ppm selenomethionine or 16 ppm selenocystine. Adult ducks were sacrificed after laying all eggs needed for the study. Hatchlings were fed clean duck mash diets for six days, after which they were sacrificed.

Adult mallards on the test diets experienced no mortality nor did they show other signs of selenium toxicity. Neither did the test diets affect the initiation or rate of egg laying, the fertility of eggs, egg weights or eggshell thickness, or hatchling weights or sex ratio. However, after three weeks on both of the 16 ppm diets (at the time of pairing), adult females experienced significant, temporary weight loss, and hatching success of fertile eggs produced by hens on the 16 ppm selenomethionine diet was significantly reduced. Hens fed the 8 or 16 ppm selenomethionine diets produced a significantly higher percentage of malformed embryos (6.8% and 67.9%, respectively), produced fewer eggs that hatched (totals of 64% and 11%, respectively), and significantly lower numbers and percent survival (81% and 0%, respectively) among 6-day-old ducklings. Weights of surviving 6-day-old ducklings were also significantly less in the 8 ppm selenomethionine dietary group.

In review of the embryotoxic effects found in the above-two studies, Hoffman and Heinz (1988) noted that although the percent of abnormal embryos (including those exhibiting edema and/or stunted growth) was similar, the frequency of teratogenesis (including multiple

malformations) among mallards fed selenomethionine was significantly higher than among those fed sodium selenite. The authors suggested that the increased embryotoxic effects associated with selenomethionine might have been related to its much greater accumulation in mallard eggs.

Heinz et al. (1988) also studied the toxicity of dietary selenium+4 and selenium-2 to mallard ducklings. For six weeks, one-day-old ducklings were provided with duck mash diets (containing 7-9% moisture) supplemented with selenium+4 (as sodium selenite) or selenium-2 (as selenomethionine), creating final dietary selenium concentrations of 0.1, 10, 20, 40, and 80 ppm. Variables measured during the study included food consumption, accumulation of selenium in the liver, organ and body weights, primary feather and tarsus lengths, and mortality. Other than enlarged livers associated with the selenite diet, neither of the 10 ppm diets produced significant effects. All of the 20, 40, and 80 ppm diets significantly reduced food consumption and growth. Mortality was 25% and 12.5% in the 40 ppm selenite and selenomethionine diets, respectively, and equaled or approached 100% in both of the 80 ppm diets. The authors noted that mortality may have been influenced by reduced consumption of food. There appeared to be little relationship between liver selenium concentrations and mortality. The authors also noted that survival and growth of ducklings were less sensitive to dietary selenium than was reproduction among mallards.

The USFWS-PWRC (Jan 1989) conducted two sets of dietary selenium-2 studies with mallards to assess the effects of overwintering in a selenium- contaminated environment. Adult male mallards were fed selenomethionine-enriched mash diets (containing the following nominal selenium concentrations: 0 [control], 10, 20, 40, or 80 ppm) for 16 weeks, followed by 16 weeks on selenium-free diets. Adult female mallards were exposed to 15 ppm selenomethionine diets for 21 weeks.

Preliminary results of the male dietary study reveal that: weight and survival were unaffected in birds receiving the 10 ppm diet, the 20 ppm diet caused some mortality, and all

birds exposed to the 80 ppm diet died. Once removed from the seleniferous diet, weights of surviving birds returned to normal. In the female dietary study, preliminary results reveal that reproductive impairment was experienced by birds laying eggs within two weeks after cessation of the seleniferous diet. No such effects were evident after that time.

The USFWS-PWRC (Jan 1990; Jan 1989; Jan 1988) studied the interactive effects of three trace elements and nutrition on mallard ducklings. Various concentrations of arsenic+5 (as sodium arsenate), boron (as boric acid), and/or selenium-2 (as seleno-DL-methionine) were added to ducklings' diets containing low (7%) or normal (21%) amounts of protein. Two separate sets of tests were conducted over 4 weeks. In the first, six groups of ducklings were fed diets containing both levels of protein, and arsenic+5 and selenium-2, singly and in combination. In the second experiment, six groups of ducklings were fed diets containing both levels of protein, and boron and selenium-2, singly and in combination. The effects of selenium and both dietary levels of protein are discussed here. The interactive effects of selenium with arsenic and selenium with boron are discussed in subsections 3.5 ("Arsenic") and 3.6 ("Boron"), respectively. Ducklings exposed to the low-protein diet or the diet containing 60 ppm selenium-2 experienced a reduced growth rate. The combination of these diets caused hepatic histological lesions and 100% mortality. When exposed to a diet containing a normal level of protein and 60 ppm selenium-2, ducklings experienced hepatic histological lesions and 40% or 50% mortality. Ducklings were not killed, but their growth was reduced when they were fed diets containing 15 ppm selenium-2 and the low level of protein. These preliminary results suggest that low-protein diets can exacerbate the toxic effects on mallard ducklings of dietary exposure to selenium-2.

In another study, independent and interactive effects of dietary boron and selenium-2 on mallard reproduction were tested using a 3x3 replicated factorial design (Smith and Heinz, Mar 1990; USFWS-PWRC, Jan 1988). The 9 diets contained either no boron or selenium

(control), or boron (as boric acid) in concentrations of 450 or 900 ppm, and/or selenium-2 (as seleno-DL- methionine) in concentrations of 3.5 or 7.0 ppm (all dry weight concentrations). Adults were provided the test diets prior to pairing and were maintained on those diets until sacrificed. Ducklings received the same dietary concentrations of boron and/or selenium-2 as their parents. All birds were sacrificed 14 days post hatch. The independent effects of selenium-2 are discussed here and its effects in combination with boron in subsection 3.6 ("Boron").

None of the selenium-2 test diets had any effects on egg size or weight. Hatching success of fertile eggs was decreased to ~60% of the control value by the 7.0 ppm selenium-2 diet. Early embryonic survival (0-7 days) was not affected by dietary selenium-2 concentrations in this study; however, late embryonic survival (day 8 to hatch) was different among treatment groups. Embryo mortality was 49% in the group receiving 7.0 ppm selenium-2.

Raabe et al. (Sep 1988) studied the acute biological effects of inhaled dimethyl selenide, about which very little is known. Young-adult male Fisher-344 rats were exposed to one of four airborne concentrations (0, 1,607, 4,499, or 8,034 ppm) of dimethyl selenide for one hour. Significant changes occurring one day following exposure to the selenium gas included: inflammation of lungs and/or liver; and changes in protein, RNA, and/or DNA content in lungs and/or liver. Spleen protein and RNA content of rats exposed to the highest concentration of dimethyl selenide were significantly elevated one week following exposure. No other adverse effects were observed during the 7-day study and, with the exception of biochemical changes in the spleen of rats exposed to 8,034 ppm selenium gas, all organs of all rats recovered completely by seven days post exposure. The authors determined that (in acute exposures), "...inhaled dimethylselenide vapor is relatively nontoxic in rats."

C) Body Burden Residues in Wildlife, Birds, or Domestic Animals: Typical, Elevated, or of Concern Related to the Well-being of the Organism Itself:

The following text is quoted from the Trinity River Report [201] for reference comparison with values from other areas:

> The selenium level in one composite sample of softshell turtles from the Rio Grande River at Big Bend National Park was 0.64 mg/kg [65]. This level is somewhat higher than those (0.26 to 0.43 mg/kg) found in softshell turtles from three sites (1, 15, 18) on the upper Trinity River. However, it is about the same (0.67 mg/kg) as the concentration recorded for a sample from highly polluted site 11 just downstream of Dallas.

Tis.Human:

A) Typical Concentrations in Human Food Survey Items:

No information found.

B) Concentrations or Doses of Concern in Food Items Eaten by Humans (Includes Allowable Tolerances in Human Food, FDA, State and Standards of Other Countries):

Crit. Dose: 0.015 mg/kg-day [Study 1 NOAEL] UF: 3 MF: 1 [893].

RfD: 5E-3 mg/kg-day Confidence: High [893,952].

C) Body Burden Residues in Humans: Typical, Elevated, or of Concern Related to the Well-being of Humans:

Milk Concentrations [940]:

The mean value for selenium in human milk has been reported to be 0.018 ppm with a range of 0.007 to 0.033 ppm. [Wilber CG; Clin Toxicol 17 (2): 171-230 (1980)].

Tis.Misc. (Other Tissue Information):

A number of plants have been described as indicators of higher-than-normal concentrations of this element in the soil [951]. Toxicity to plants is moderate [951].

Monogastric (non-ruminant) animals more readily absorb dietary selenium+4 than do ruminants [445].

Liver References to Selenium:

NOTE: many of these references are listed elsewhere in this document as well, but are split out here

separately for those just interested in liver issues.

Whole body concentrations of selenium in mosquitofish from highly contaminated areas of California were twice as high as the highest reported liver concentrations from the literature [488]. A North Carolina study of several species of fish suggested the highest concentrations of selenium were in the liver [488]. A separate literature review that the liver of selenium-exposed fish accumulates higher levels of selenium than skeletal muscles. However, selenium concentrations in the liver typically correlate with selenium concentrations in other tissues, even though the distribution patterns within tissues, as well as absolute values of selenium, vary considerably with the species considered [488].

Sunfish exposed to aqueous selenium accumulate selenium in the liver (7-27 ppm, wet weight) and skeletal muscle, although the levels in skeletal muscle are about half of those in the liver [488].

Those fish accumulating the highest levels of selenium in the liver have the lowest hematocrits, lowest hemoglobin levels, lowest erythrocyte numbers, abnormally small and irregularly shaped erythrocytes, and reductions in both mean corpuscular volumes and mean corpuscular hemoglobin concentrations [488]. Green sunfish with the highest levels of selenium in the liver have pericarditis and myocarditis [488].

Se can either help cause or help protect against such things as liver damage [445,484.486,488].

Selenium has been associated with abnormal development of and damage to/degeneration of internal organs, including the heart, liver, and kidneys [445].

Se...possibly helpful in preventing skin, liver, and pancreas cancer [484,486].

Some references state that selenium accumulates in the axial muscles of fish, so fillet levels are typically closer to whole-body concentrations than are most other contaminants [27,136]. A more recent, and somewhat contrasting summary documents the tendency of selenium to concentrate more highly in the liver, gonads, and kidneys of fish than in muscle [488]. Selenium accumulates in the gonads of bass and bluegills [36].

At Kesterson NWR, in order to protect fish reproduction, their skeletal muscles should not contain more than 5 ppm and their organs (liver and gonads) should not contain more than 10 ppm selenium (both concentrations are for total selenium, dry weight) (Wallenstrom, Aug 1986) [445].

The rate of absorption as elemental selenium ... Is low. ... Liver & kidney are principal sites of deposition. Excretion of selenium is by urine, feces, sweat, & breath. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2130][366]

Liver and kidney contained the highest concentrations of most metals (in rats) [366].

In the liver, many selenium compounds are biotransformed to excretable metabolites. Identified metabolites are trimethylselenide in urine and dimethylselenide in breath. /Selenium compounds/[Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986.,p. V2

Heinz et al. (1990) calculated that mallards fed selenium- enriched diets (10 ppm seleno-DL-methionine) accumulated 95% of peak selenium concentrations in liver and breast muscle in 7.8 and 81 days, respectively [445].

Selenium in living tissue is often found associated with protein. This is because some proteins (including enzymes) require selenium and selenium can readily substitute for sulfur in some sulfur-amino acids such as cysteine, cystine, and methionine (amino acids are the building blocks of proteins). In plants, selenium accumulates in seeds (Burau, Jul-Aug 1985). Chronic exposure of animals to selenium causes accumulation in the following tissues: liver, kidneys, pancreas, spleen, heart, lungs, ovaries, testes, gills, brain, and blood (NRC-Subcommittee on Selenium, 1976; Eisler, Oct 1985; Gillespie et al., 1988; Maier et al., 1987; Ogle et al., 1988; USEPA, Sep 1987) [445].

In studies with mallards first fed then removed from highly selenium- enriched diets (up to 160 ppm seleno-DL-methionine), Heinz et al. (1990) found that one-half of the selenium was lost from blood and breast muscle tissues in 9.8 and 23.9 days,

respectively. In the liver, selenium was initially lost rapidly and then more slowly. Selenium concentrations in blood, breast muscle, and liver tissues were predicted to return to background levels in 58.4, 120.4, and 161.8 days, respectively [445].

There appeared to be little relationship between liver selenium concentrations and mortality. The authors also noted that survival and growth of ducklings were less sensitive to dietary selenium than was reproduction among mallards [445].

Hamilton et al. (1990) suggest that, in order to be safe for fish, dietary concentrations of selenium should be less than 3 ug/g (ppm, dry weight) [445].

Liver enlargement was noted for many species of fish exposed to selenium [488].

Bio.Detail: Detailed Information on Bioconcentration, Biomagnification, or Bioavailability:

Preliminary data suggests the potential for bioaccumulation or bioconcentration of selenium is moderate for the following biota: mammals, birds, and fish. It appears to be high to very high for higher plants and low or limited for mollusks, crustacea, lower animals, mosses, lichens, and algae [83]. The best potential mediums for biological monitoring (including gradient monitoring) appear to include higher plants [83].

Plants easily take up selenate compounds from water and change them to organic selenium compounds such as selenomethionine (a transparent solid in pure form) [953].

Bioaccumulation of selenate can be diminished in the presence of chromate, molybdate, sulfate, and selenomethionine [445]. One rodent study suggested that more selenium is accumulated if methyl mercury is present. Selenium accumulation in all organs (especially the kidney) was accelerated (8-fold increase in 41 days in the kidneys) in rats treated with mercury and selenium [488].

Some references state that selenium accumulates in the axial muscles of fish, so fillet levels are typically closer to whole-body concentrations than are most other contaminants [27,136]. A more recent, and somewhat contrasting summary documents the tendency of selenium to concentrate more highly in the liver, gonads, and kidneys of fish than in muscle [488]. Selenium accumulates in the gonads of bass and bluegills [36].

A nationwide study of selenium in bivalves showed less variation in levels from various stations than was found for most other contaminants [62].

Laboratory aquatic microcosm experiments have clearly demonstrated that the bioconcentration potential of selected organic selenium compounds, including selenomethionine, is much greater than for common inorganic selenium forms such as selenate

and selenite [445]. This is especially true at very low waterborne concentrations [445]. Specifically, the USFWS-NFCRC (Dec 1989) conducted microcosm experiments with algae (Chlamydomonas reinhardtii) and daphnids (Daphnia magna) and discovered that bioconcentration factors for dissolved selenomethionine were much greater than for selenite, which were greater than for selenate [445]. Besser et al. (1989) discovered that algae and daphnia exposed to waterborne selenomethionine in microcosms for 28 days accumulated tissue concentrations of selenium tens of thousands of times greater than those in the water [445]. In light of the high toxicity of dietary selenomethionine to fish and wildlife, the speciation of waterborne selenium may be more important in determining safe levels than the concentration of total selenium [445].

Research in progress indicates that the specific form of selenium is critical in determining the potential for the occurrence of selenium toxicity because amino forms of selenium may bioconcentrate to toxic levels in fish and birds even when concentrations in water are less than 1 microgram per liter (ppb) [463].

Due to bioconcentration concerns, the State of California water quality criteria based on protection of aquatic organisms in the food chain was quite low, only 0.9 ug/L for impounded waters (versus 11 ug/L for flowing waters) [222].

The mean elemental concentration of this metal in plants was 0.1 ppm in the same areas where rocks were 1 ppm [951].

Bioconcentration factors (BCFs) estimated from 1 ug/L (ppb) selenium as Se-methionine exposures were approximately 16,000 for algae, 200,000 for daphnids, and 5,000 for bluegills [481]. Algae and daphnids concentrated Se more strongly from selenite (BCFs of 220 to 3,600) than selenate (BCFs 65 to 500), while bluegills concentrate se about equally from both species [481]. Daphnids in aqueous selenium exposures may be accumulating a portion of their selenium body burdens via ingestion of selenium enriched algae [481]. Bluegills in Se-contaminated systems accumulate inorganic Se species primarily via food chain uptake, although organoselenium compounds such as Se-methionine may contribute significantly to Se bioaccumulation by bluegills via both aqueous and food-chain uptake [481].

> Text in paragraph above reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, J.M. Besser, T.J. Canfield and T.W. La Point, "Bioaccumulation of oganic and inorganic selenium in a laboratory food chain." Copyright 1993 SETAC].

Misc. Notes Related to Selenium Bioavailability in Soils:

> In areas of acid or neutral soils, the amount of biologically available selenium should steadily decline. The decline may be accelerated by active agricultural or industrial practices. In dry areas with alkaline soils and oxidizing conditions, elemental selenium and selenides in rocks and volcanic soils may oxidize sufficiently to maintain the availability of

biologically active selenium.

In areas of the Western United States (states west of and including North Dakota, South Dakota, Nebraska, Kansas, Oklahoma, and Texas), where geological sources of selenium are abundant in the soil profile, selenium tends to be more bioavailable to ecosystems [463]:

> A major feature of this area is the presence of pedocal soils. Pedocals are alkaline soils of semiarid and arid climates. Bacterial and chemical processes in these highly oxidizing, alkaline soils favor the formation of calcium and sodium selenates, which are very mobile and water soluble, and are readily available forms of selenium to plants. In addition, many areas in the Western United States are prone to selenium enrichment of the soil because of leaching of underlying seleniferous rocks, such as the shales and clays of the Upper Cretaceous Pierre, Steele, and Niobrara Shales [463].
>
> Pedalfer soils found in the Eastern United States are acid to neutral soils of humid and semihumid climates. These acid soils favor the formation of more reduced and complexed forms of selenium, such as ferric selenite. These complexes are generally insoluble so as to reduce selenium bioavailability to the point that forages and feeds grown in the Eastern United States contain insufficient selenium for proper animal nutrition [463].
>
> In short, in areas in the Western United States where selenium is abundant and bioavailable, a more detailed analysis may be required for selenium toxicity than would be required in the east [463].

Bioaccumulation [445]:

> See Moore et al. [445] for literature citation details:
>
> Selenate is readily accumulated by terrestrial plants. In contrast, many aquatic organisms more readily take up, accumulate, and metabolize selenomethionine and selenite (Besser et al., 1989; Ogle et al., 1988). Within these organisms, selenium is biochemically reduced to elemental selenium and selenide forms, including methylated selenium compounds and seleno-amino acids. Bioaccumulation of selenate can be diminished in the presence of chromate, molybdate, sulfate, and selenomethionine.
>
> Some plants, selenium accumulators, can take up and accumulate very high concentrations of selenium in their tissues (over 1,000 ppm) without injurious effects. Primary or obligate selenium accumulators grow only where soil selenium concentrations are high enough to satisfy

metabolic needs and include many species of Astragalus and some species of Brassica, Haplopappus, Machaeranthera, Oonopsis, and Stanleya, and Zylorhiza. Secondary or facultative selenium accumulators can tolerate, but do not require elevated soil selenium concentrations. Facultative selenium accumulators include many species of Aster and some species of Astragalus, Atriplex, Castelleja, Comandra, Grayia, Grindelia, Gutierrezia, Machaeranthera, and Mentzelia.

Selenium can also be bioconcentrated by a large number of aquatic plants and animals that form the bases for important fish and wildlife food chains. Hamilton et al. (1990) note that algae and zooplankton can accumulate selenium in tissues to concentrations several-hundred times those found in water. The USFWS-NFCRC (Dec 1989) conducted microcosm experiments with algae (Chlamydomonas reinhardtii) and daphnids (Daphnia magna) and discovered that bioconcentration factors for dissolved selenomethionine were much greater than for selenite, which were greater than for selenate. Besser et al. (1989) discovered that algae and daphnia exposed to waterborne selenomethionine in microcosms for 28 days accumulated tissue concentrations of selenium tens of thousands of times greater than those in the water. According to Lemly (1989), bioconcentration factors for aquatic plants and animals exposed to 5-30 ug/l (ppb) waterborne selenium typically range from 500 to 35,000. Using both field-collected and laboratory data, DuBowy (1989) developed an energy-based selenium bioaccumulation model for aquatic birds that he used to calculate a bioaccumulation factor of 1,430 for waterfowl (ratio of wet weight concentration of selenium in breast muscle tissue to selenium concentration in water). Bioconcentration factors in aquatic systems are especially high when selenium occurs at very low waterborne concentrations.

Although opinions are mixed, there is some evidence that selenium can also biomagnify through the food chain Lemly (1989) noted that biomagnification factors (between food-chain trophic levels) for 5-30 ug/l (ppb) waterborne selenium in aquatic systems typically range from 3 to 7 times.

Selenium in the diet constitutes the most important source for uptake in animals (Lemly, 1986; Lemly and Smith, 1987). Although data are lacking, selenium in natural diets most likely occurs primarily in organic forms (Demayo et al., 1979b; Maier et al., 1987; NRC-Subcommittee on Selenium, 1976). Selenomethionine may be the dominant form of selenium in plant tissues and selenocysteine may dominant in animal tissues (USFWS-PWRC, Jan 1990). Dietary and possibly waterborne organic

selenium compounds (such as selenomethionine) are more effectively and quickly taken up, accumulate to higher concentrations, and are retained longer by animals than are inorganic forms of selenium (Besser et al., 1989; Burau, Jul-Aug 1985; Davis et al., Jan-Feb 1988; Heinz et al., 1988; NRC-Subcommittee on Selenium, 1976; Ogle et al., 1988). Heinz et al. (1990) calculated that mallards fed selenium- enriched diets (10 ppm seleno-DL-methionine) accumulated 95% of peak selenium concentrations in liver and breast muscle in 7.8 and 81 days, respectively.

Selenium absorption in the gastrointestinal tract is dependent on a variety of factors, including: its chemical form(s) (e.g., fish take up selenomethionine through the gastrointestinal tract more readily than either selenium+4 or selenium+6 [USEPA, Sep 1987]); the animal's nutritional (protein) status; and whether or not the animal is a ruminant (monogastric [nonruminant] animals more readily absorb dietary selenium+4 than do ruminants) (NRC-Subcommittee on Selenium, 1976). In dietary selenium studies with Atlantic salmon smolts (Salmo salar), Bell and Cowey (1989) found the digestibility of DL-selenomethionine to be better than selenium+4 (as sodium selenite), which was more easily digested than was DL- selenocystine.

Selenium in living tissue is often found associated with protein. This is because some proteins (including enzymes) require selenium and selenium can readily substitute for sulfur in some sulfur-amino acids such as cysteine, cystine, and methionine (amino acids are the building blocks of proteins). In plants, selenium accumulates in seeds (Burau, Jul-Aug 1985). Chronic exposure of animals to selenium causes accumulation in the following tissues: liver, kidneys, pancreas, spleen, heart, lungs, ovaries, testes, gills, brain, and blood (NRC-Subcommittee on Selenium, 1976; Eisler, Oct 1985; Gillespie et al., 1988; Maier et al., 1987; Ogle et al., 1988; USEPA, Sep 1987).

Selenium loss (depuration) in animals occurs through urination, exhalation, perspiration, and with feces and milk. Urination is commonly the most important depuration mechanism; however, at higher selenium intakes, large quantities of volatile selenium can also be lost through exhalation (Demayo et al., 1979b). Selenium is excreted in urine as the trimethyl selenonium ion and in feces as either elemental selenium or a metal selenide (NRC- Subcommittee on Selenium, 1976). Inorganic selenium is lost more rapidly by some species than is organic selenium (Maier et al., 1987).

In studies with mallards first fed then removed from

highly selenium- enriched diets (up to 160 ppm seleno-DL-methionine), Heinz et al. (1990) found that one-half of the selenium was lost from blood and breast muscle tissues in 9.8 and 23.9 days, respectively. In the liver, selenium was initially lost rapidly and then more slowly. Selenium concentrations in blood, breast muscle, and liver tissues were predicted to return to background levels in 58.4, 120.4, and 161.8 days, respectively.

Eisler (Oct 1985) and Ohlendorf and Skorupa (1989) provide tissue selenium concentrations for fish and wildlife collected from both seleniferous and uncontaminated environments.

Williams (Dec 1988) conducted a 96-hour study of the effects of various concentrations of waterborne selenium+6 (as sodium selenate [Na2SeO4]) and sulfate (as sodium sulfate [Na2SO4]) on bioaccumulation and growth of green algae (Selenastrum capricornutum). Selenium in the test solutions also included modest amounts of selenite and organoselenium (~4% and <6%, respectively). Test solutions contained the following average concentrations and approximate molar ratios (in parentheses) of selenate (in ug/l [ppb]) and sulfate-sulfur ([SO4-S] in mg/l [ppm]), respectively: 107 and 3.34 (1:75), 11.3 and 3.34 (1:750), 107 and 33.2 (1:750), and 11.3 and 33.2 (1:7,000). Separate algal growth tests (controls) were conducted in waters containing 3.34 or 33.2 mg/l (ppm) sulfate-sulfur with no additions of selenium. Algae were cultured in modified Woods Hole media, with reduced concentrations of magnesium sulfate (MgSO4; to 3.34 mg/l S [ppm]), increased concentrations of magnesium carbonate (MgCO3; to 3.65 mg/l Mg [ppm]), and no tris buffer or EDTA [445]. More detail:

> Increasing concentrations of sulfate, while maintaining concentrations of selenate and increasing ratios of sulfate to selenate concentrations, both resulted in reduced bioaccumulation of selenium by the algae. Algal growth was reduced over the two controls in all four selenium test waters and growth reductions increased with increasing concentrations of selenium in tissues. Of the four test waters, the greatest growth occurred in the high sulfate - low selenate solutions [445].

Interactions:

Under some circumstances, selenium may interact in an antagonistic or protective manner with arsenic, cadmium, copper, lead, mercury, silver, thallium, zinc, and the herbicide paraquat [445]. Another reference stated that selenium can serve as an

antidote to the toxic effects of metals such as arsenic, mercury, cadmium, copper, and thallium [491].

Bioaccumulation of selenate can be diminished in the presence of chromate, molybdate, sulfate, and selenomethionine [445]. It has also been noted that selenium toxicity can be reduced through the administration of methionine and vitamin E [445]. These interactions, however, are dependent upon the specific forms of the chemicals involved, and mixing different forms of the same chemicals can sometimes have the opposite effects [445]. For example, dietary sulfate, which can have a protective effect on selenate toxicity, does not affect the toxicity of selenite or organoselenium. Dimethyl selenide (normally not very toxic) has a synergistic effect with some mercuric salts, and although drinking water containing arsenic can ameliorate the toxic effects of dietary selenium, their toxic effects can be additive when provided together in drinking water [445].

One study revealed the inhibitory effect of sodium selenite on induction of bladder cancer by butylbutanolnitrosamine in rats. The incidence of carcinoma in the control group was 87.5% whereas in the sodium selenite group--50% (P less than 0.01) (Frolov AG, 1990. The effect of sodium selenite on the butyl butanol nitrosamine induction of bladder tumors in rats. Original title: Vliianie natriia selenita na induktsiiu butilbutanolnitrozaminom opukholei mochevogo puzyria u krys. Vopr-Onkol; 1990; 36(6); P 697-700 (Russian).

Interactions between Selenium and Mercury:

> NOTE: Some of the interactions between selenium and mercury were mentioned in the Brief Introduction above. These are important enough that they are repeated here with additional information:

Selenium, like mercury, has many interactions with sulfur compounds. This affinity for sulfur compounds may account for some of the many synergistic and antagonistic interactions between mercury and selenium (see details below). Also like mercury, selenium chemistry, transformations, and interactions with other contaminants are complex. In fact, selenium is one of the most complex elements in these regards. Due to these complexities, generalizations about selenium should be approached with caution.

Antagonism/Synergism: Four studies have suggested that the presence of selenium reduces the toxicity of (is antagonistic to) mercury (Hg), cadmium (Cd), arsenic (As), and silver (Ag) [488]. The exact mechanism of action for selenium induced protective effects of vertebrates has not been elucidated [488]. Sorenson (1991) suggested caution in attempting to use selenium supplementation to decrease the severity of point-source mercury contamination, since synergistic interactions

have also been observed, and since synergistic interactions on even a single life stage (as demonstrated on carp by Huckabee and Griffith) have a potential of eliminating an entire population of fish [488]. Although selenium and mercury interacted synergistically rather than in an antagonistic (protective) fashion, the author of the study which showed synergistic effects cautions that it was an early lab study and should not totally override all later field studies (John Huckabee, Electric Power Institute, personal communication, 1994).

Nevertheless, most mercury and selenium experts suggest caution in using selenium to treat surface waters in an effort to reduce mercury problems, since there is such a small safety window between too little and too much selenium. Interactions are known to be concentration (dose) dependent [488]. Interactions between Se and mercury can be synergistic at low aqueous mercury concentrations (< or = 0.07 ppm) and antagonistic at high mercury levels (> or = 0.10 ppm) and high selenium levels [488]. In Sweden, selenium supplementation was tried as a remedy for mercury problems in lakes and some reduced mercury concentrations seemed to be the result; however, too much selenium was eventually used and negative effects on fish reproduction was eventually seen (John Rudd, Freshwater Research Institute, Winnipeg, Manitoba, personal communication, 1994). Moderate selenium toxicity to adult fish may be irrelevant if the larval fish are much more sensitive to selenium toxicity and thus the fish do not make it to adulthood.

One rodent study suggested that more selenium is accumulated if methyl mercury is present. Selenium accumulation in all organs (especially the kidney) was accelerated (8-fold increase in 41 days in the kidneys) in rats treated with mercury and selenium [488].

Selenium has been referred to as an agent which can bind mercury and cadmium compounds to make them more biologically inert, as a protective agent against mercury induced lipid peroxidation, as an element which can detoxify various metals by chelating them [484,486].

Under some circumstances, selenium may interact in an antagonistic or protective manner with mercury and several other contaminants [445]. Some contaminants specialists who have looked at some of the human health and animal husbandry literature have wondered whether or not slight elevations of some forms of selenium in fish tissues may possibly be acting partly in a protective manner (to a greater degree than is commonly recognized) to humans and fish and wildlife predators consuming fish contaminated with harmful concentrations of heavy metals such as cadmium, mercury, and lead (Jerry Miller, U.S.

Bureau of Reclamation, Salt Lake City, personal communication, 1994). However, care should be taken in generalizing, and the many risks associated with bioconcentration, reproductive risk, and other potential risks of selenium cannot be ignored.

Dietary sulfate, which can have a protective effect on selenate toxicity, does not affect the toxicity of selenite or organoselenium; dimethyl selenide (normally not very toxic) has a synergistic effect with some mercuric salts; and although drinking water containing arsenic can ameliorate the toxic effects of dietary selenium, their toxic effects can be additive when provided together in drinking water [445].

Metal selenides are formed with cadmium, copper, and mercury. Many organic selenides also are common [445].

Gary Heinz and Dave Hoffman of the Patuxent Fish and Wildlife Research Center in Laurel, MD, believe that, as of November 1993, there is usually not enough data available to tell exactly what various combinations of various forms of selenium and mercury mean as body burdens in various life stages of waterfowl. Organic selenium (typically organoselenium compounds such as Se-methionine) combined with organic mercury (typically methyl mercury) appears to be the most dangerous combination, but not all research has used these two. Some workers have suggested that a ratio of one mole of selenium/one mole of mercury can have protective effects for certain adult organisms, but ratios found in nature seldom work out to this exact ratio. Some preliminary experimental data from Patuxent seems to hint that certain doses of organic selenium combined with certain doses of organic mercury can have "more than additive" effects (deformities and death) on mallard duck embryos; however, the reverse (antagonism) may be true for adult ducks and the complexity of potential combinations of forms and concentrations of the two compounds makes generalizations difficult (Gary Heinz and Dave Hoffman, Patuxent Fish and Wildlife Research Center, National Biological Survey, personal communication, 1994).

Interactions between Selenium and Sulfur:

Ratios of Se/S have been used to evaluate Se mobility and sources of atmospheric pollutants; it Boston, the Se/S ratios helped determine that a considerable portion of the air pollution is due to petroleum combustion [488].

Selenium, like mercury. has many interactions with sulfur compounds. This affinity for sulfur compounds may account for some of the many synergistic and antagonistic interactions between mercury and selenium.

Dietary sulfate, which can have a protective effect on selenate toxicity, does not affect the toxicity of selenite or organoselenium; dimethyl selenide (normally not very toxic) has a synergistic effect with some mercuric salts; and although drinking water containing arsenic can ameliorate the toxic effects of dietary selenium, their toxic effects can be additive when provided together in drinking water [445].

Uses/Sources:

Selenium is a rare element forming only 9E-06 percent (9 X 10 to the power of - 6%) of the Earth's crust [291]. Selenium is an essential nutrient for humans and animals, and both can use inorganic as well as organic selenium compounds [953]. In the body, selenium helps prevent damage to tissues done by oxygen [953].

Plants take up selenium from soil, groundwater, sewage sludge, and air pollution [83]. A number of plants have been described as indicators of higher-than-normal concentrations of this element in the soil [951]. Animals take up selenium from industrial sources, contaminated air, contaminated water, and contaminated food [83]. Other than areas impacted by agricultural drainage, very high concentrations of selenium in fish and wildlife occur primarily in areas where selenium is naturally high in the soils, where there is an influence of sewage sludge, or where coal fired power plants are present [37,44].

Man's uses of selenium include photocopying, glass manufacturing, the production of stainless steel, fungicides, lubricants, electronic devices, pigments, dyes, insecticides, and veterinary medicine [38,61]. Selenium is used in the production of photocells, exposure meters, and solar cells [291]. Selenium also finds extensive application in rectifiers, a result of its ability to convert alternating electric current to direct current [291]. Other applications of the element include its use in the glass industry to de-colorize glass, as a photographic toner, as an additive in steel production, and in xerographic reproduction [291]. Selenium dioxide is a good oxidizing agent and is used in certain organic syntheses [291].

Used in electrodes [490].

Major Uses [940]:

In the glass industry as a decolorizing agent. [Friberg, L., Nordberg, G.F., Kessler, E. and Vouk, V.B. (eds). Handbook of the Toxicology of Metals. 2nd ed. Vols I, II.: Amsterdam: Elsevier Science Publishers B.V., 1986.,p. V2 485].

As ingredient of toning baths in photography; as pigment in mfr ruby-, pink-, orange-, or red-colored glass; as metallic base in making electrodes for arc lights, electrical instruments & apparatus; as rectifier in radio & television sets; in selenium photocells, in semiconductor fusion

mixtures, selenium cells [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

In telephotographic apparatus; as vulcanizing agent in processing of rubber; as catalyst in determination of nitrogen by kjeldahl method; for dehydrogenation of organic compounds [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

In steel & copper (degasifier & machineability improver) [Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1032].

Selenium is used extensively in the manufacture and production of glass, pigments, rubber, metal alloys, textiles, petroleum, medical therapeutic agents, and photographic emulsions. [US Dept of Interior/Fish & Wildlife Service Contaminant Reviews; Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review Biol Rept No (85)1.5 p.5 (1985)].

Natural Sources [366]:

There are no true deposits of selenium anywhere and it cannot economically be recovered from the earth directly. [International Labour Office. Encyclopedia of Occupational Health and Safety. Vols. I&II. Geneva, Switzerland: International Labour Office, 1983. 2018].

Constitutes about 0.09 Ppm of the earth's crust. Occurs in nature usually in the sulfide ores of the heavy metals; found in small quantities in pyrite; in the minerals clausthalite (PBSE), naumannite ((Ag,Pb)Se), tiemannite (HgSe); in selenosulfur. [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Present in the major oceans and in inland waters resulting in the presence of selenium in drinking water. **PEER REVIEWED** [IARC. Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man. Geneva: World Health Organization, International Agency for Research on Cancer, 1972-1985. (Multivolume work).,p. V2 248 (1973)].

Selenium accompanies sulfur in volcanic effluents. Soils in the neighborhood of volcanos tend to have enriched amounts of selenium. [Wilber CG; Clin Toxicol 17 (2): 171-230 (1980)].

Selenium concentration in geological materials: Igneous rocks 0.05 ppm; shales 0 - 0.6 ppm; sandstones 0.0 - 0.05 ppm; limestones 0.08 ppm; soils 0.2 ppm. [Wilber CG; Clin Toxicol 17 (2): 171-230 (1980)].

The earth's crust is said to have an average selenium

concentration of 0.03 of 0.08 ppm. [Wilber CG; Clin Toxicol 17 (2): 171-230 (1980)].

Selenium is the most strongly enriched element in coal, being present as an organoselenium compound, a chelated species, or as an adsorbed element. [US Dept of Interior/Fish & Wildlife Service Contaminant Reviews; Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review Biol Rept No (85)1.5 p.3 (1985)].

Artificial Sources [940]:

Various industries discharge small amounts of selenium into the air, water, and soil of the immediate vicinity. [Wilber CG; Clin Toxicol 17 (2): 174 (1980)].

Forms/Preparations/Formulations:

Common Forms of Selenium (Selenium Species):

The concentrations and chemical forms of selenium in water are influenced by, among other factors, pH and redox conditions. Although dissolved selenium in most natural waters is dominated by the inorganic selenate and selenite forms, substantial concentrations of selenium in +6, +4, and -2 oxidation states can occur simultaneously in natural aerobic surface waters of pH 6.5-9.0 [445]. One author found the following six forms of dissolved selenium in water samples taken from three California water bodies (Kesterson Reservoir, the San Joaquin River, and the Salton Sea), all of which have received subsurface agricultural drainage water: selenate, selenite, seleno-amino acids, dimethyl selenonium ion ((CH3)2Se+1), dimethyl selenide, and dimethyl diselenide [445].

In the environment, selenium naturally occurs in four valence states, +6, +4, 0, and -2. Selenium dioxide (SeO2), a selenium+2 form, does not occur naturally in the environment, but is produced through the combustion of fossil fuels [445]. Selenium in the +6 oxidation state (as the selenate ion [SeO4-2]) is very common, soluble, and stable in alkaline, oxidized waters and soils. Selenate is the most common form of selenium in subsurface agricultural drainage waters of the San Joaquin Valley and in the San Joaquin River [445]. Selenium+4 (as the selenite ion [SeO3-2]) is somewhat less soluble than selenate and forms under slightly less oxidized conditions. Formation of the insoluble, inert, and stable selenium (elemental or "metallic" selenium, a mineral) is favored in acidic and reducing conditions. Further chemical reduction leads to the formation of selenium-2 (selenide), which is the basis of: inorganic

metal selenides (which are generally insoluble); many organic selenium compounds, including amino acids (very soluble) and methylated, volatile selenium forms (relatively insoluble); and hydrogen selenide. Metal selenides are formed, for example, with cadmium, copper, and mercury. Examples of organoselenium compounds include: the seleno- amino acids, selenocysteine ($C_3H_7NO_2Se$), selenocystine ($C_6H_{12}N_2O_4Se_2$), and selenomethionine ($C_5H_{11}NO_2Se$); and the common methylated selenide compounds dimethyl selenide ($(CH_3)_2Se$) and dimethyl diselenide ($(CH_3)_2Se_2$). Hydrogen selenide (H_2Se) is a highly toxic, volatile, and relatively insoluble inorganic compound that is formed in some industrial settings, but is not found in the natural environment [445].

For a discussion of various forms of selenium see W.General section above and Federal Register Vol 61, no. 221,pages 5844 to 58449.

Radionuclides:

The symbol for Selenium-75 is ^{75}Se, the atomic number is 34, the half-life is 118 days, and X-ray emission is the major form of decay [674].

Various forms of selenium commonly referred to in the environmental toxicology literature under different names and classification categories include:

Inorganic Selenium Compounds and Species: Bluegill sunfish tend to take inorganic selenium primarily through the food chain [481]. The most important oxidation states are +4 and +6 [291]. Inorganic compounds include:

1. Selenates (selenium+6): Selenium in the +6 oxidation state (as the selenate ion [SeO_4^{-2}]) is very common, soluble, and stable in alkaline, oxidized waters and soils [445]. Selenate is the most common form of selenium in subsurface agricultural drainage waters of the San Joaquin Valley and in the San Joaquin River [445].

2. Selenites (such as selenium +4 as sodium selenite): Monogastric (non-ruminant) animals more readily absorb dietary selenium +4 than do ruminants [445]. The low waterborne concentrations in the ocean may be due to precipitation of selenium +4 with iron and manganese oxides [445]. Selenium +4 (as the selenite ion [SeO_3^{-2}]) is somewhat less soluble than selenate and forms under slightly less oxidized conditions. Formation of the insoluble, inert, and stable selenium (elemental or "metallic" selenium, a mineral) is

favored in acidic and reducing conditions [445].

3. Selenium dioxide (SeO_2), a selenium +2 form, does not occur naturally in the environment, but is produced through the combustion of fossil fuels [37].

4. Selenides (selenium -2): Further chemical reduction leads to the formation of selenium -2, which is the basis of:

A. Hydrogen selenide. Hydrogen selenide (H_2Se) is a highly toxic, volatile, and relatively insoluble inorganic compound that is formed in some industrial settings, but is not found in the natural environment [445].

B. Various inorganic metal selenides (which are generally insoluble). Metal selenides are formed with cadmium, copper, and mercury. Many organic selenides also are common (see summary below) [445].

Organic Selenium (organoselenium) Compounds and Species: There are numerous references in the literature documenting that certain organic forms of selenium are more bioavailable, more easily bioconcentrated, or more toxic than are certain inorganic forms of selenium. Organoselenium compounds can constitute 30-60% of the selenium in some fresh and marine waters [445]. Organic selenium includes:

1. Amino acids (very soluble) and methylated, volatile selenium forms (relatively insoluble) [445]: Examples of organoselenium compounds include the seleno-amino acids, selenocysteine ($C_3H_7NO_2Se$), selenocystine ($C_6H_{12}N_2O_4Se_2$); and the common methylated selenide compounds dimethyl selenide ($(CH_3)_2Se$) and dimethyl diselenide ($(CH_3)_2Se_2$) [445].

NOTE: Dimethylselenide is generated slowly from raw sewage [488]. Selenium is methylated and probably demethylated in the environment and cycled through a number of components of the food web, complicating chemical determination of the chemical forms available to fish [488].

2. Selenomethionine ($C_5H_{11}NO_2Se$): Selenomethionines (Se-methionines, such as selenium-2 as selenomethionine) are organoselenium forms which are so prominent in the environmental toxicology literature that they are discussed

separately.

> Selenomethionine may be the dominant form of selenium in plant tissues and selenocysteine may dominant in animal tissues (USFWS-PWRC, Jan 1990) [445]. Dietary and possibly waterborne organic selenium compounds (such as selenomethionine) are more effectively and quickly taken up, accumulate to higher concentrations, and are retained longer by animals than are inorganic forms of selenium (Besser et al., 1989; Burau, Jul-Aug 1985; Davis et al., Jan-Feb 1988; Heinz et al., 1988; NRC-Subcommittee on Selenium, 1976; Ogle et al., 1988) [445].

Although not true in all cases, the relative toxicity of various chemical forms of selenium is generally as follows (from most to least toxic): hydrogen selenide ~ selenomethionine (in diet) > selenite ~ selenomethionine (in water) > selenate > elemental selenium ~ metal selenides ~ methylated selenium compounds [445]. The toxicity of dietary selenocystine to mallard ducks was much less than selenomethionine and may be somewhat less than selenite (Heinz et al, 1989; Heinz et al., 1987) [445].

Differential Toxicity and Food Chain Characteristics of Different Forms of Selenium:

> Fish take up selenomethionine through the gastrointestinal tract more readily than either selenium +4 or selenium +6 [445].
>
> Organoselenium compounds such as Se-methionine may contribute significantly to Se bioaccumulation by bluegills via both aqueous and food-chain uptake, whereas inorganic compounds are accumulated mostly through the food chain [481]. Dietary organic selenium in the form of Se-methionine is more toxic to fish than dietary inorganic selenium [481].
>
> > Text in paragraph above reprinted with permission from Environmental Toxicology and Chemistry, Volume 12, J.M. Besser, T.J. Canfield and T.W. La Point, "Bioaccumulation of oganic and inorganic selenium in a laboratory food chain." Copyright 1993 SETAC].
>
> Selenite is 10 times for toxic to fish than selenate, and organic forms are even more toxic than selenite (Denny Buckler, FWS Columbia, personal communication). The USFWS-NFCRC (Dec 1989) conducted microcosm experiments with algae (Chlamydomonas reinhardtii) and daphnids (Daphnia magna) and discovered that bioconcentration factors for dissolved selenomethionine were much greater

than for selenite, which were greater than for selenate [445].

Selenium absorption in the gastrointestinal tract is dependent on a variety of factors, including: its chemical form(s) (e.g., fish take up selenomethionine through the gastrointestinal tract more readily than either selenium+4 or selenium+6 [USEPA, Sep 1987]); the animal's nutritional (protein) status; and whether or not the animal is a ruminant (monogastric [non-ruminant] animals more readily absorb dietary selenium +4 than do ruminants)[445].

Information from HSDB [940]:

The forms of selenium in soil depend on soil pH and redox. At equilibrium, most soil selenium should be elemental selenium. [Parr, J.F., P.B. Marsh, and J.M. Kla (eds.). Land Treatment of Hazardous Wastes. Park Ridge, New Jersey: Noyes Data Corporation, 1983. 186].

In areas of acid or neutral soils, the amount of biologically available selenium should steadily decline. The decline may be accelerated by active agricultural or industrial practices. In dry areas with alkaline soils and oxidizing conditions, elemental selenium and selenides in rocks and volcanic soils may oxidize sufficiently to maintain the availability of biologically active selenium. [US Dept of Interior/Fish & Wildlife Service Contaminant Reviews; Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review Biol Rept No (85)1.5 p.4 (1985)].

Chem.Detail: Detailed Information on Chemical/Physical Properties:

Solubilities [940]:

Sol in aq potassium cyanide soln, potassium sulfite soln, dilute aqueous caustic alkali soln [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

INSOL (sic, actually "relatively insoluble") IN WATER; SOL IN SULFURIC ACID [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Density/Specific Gravity [940]:

4.26-4.81 [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Vapor Pressure [940]:

> 0.001 mm Hg at 20 deg C [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. 2].

Boiling Point [940]:

690 deg C [Sax, N.I. Dangerous Properties of Industrial Materials. 6th ed. New York, NY: Van Nostrand Reinhold, 1984. 2390].

Melting Point [940]:

170-217 deg C [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Molecular Weight [940]:

78.96 +/- 3 [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Color/Form [940]:

LIQUID IS A BROWNISH RED [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Selenium exists in several allotropic forms. 3 Are generally recognized ... Selenium can be prepd with either amorphous or crystalline structure. ... Amorphous is either red, in powder form or black, in vitreous form. Crystalline monoclinic prism is deep red; crystalline hexagonal form, the most stable variety, is a metallic gray. [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-34].

Odor [940]:

Odorless [Mackison, F. W., R. S. Stricoff, and L. J. Partridge, Jr. (eds.). NIOSH/OSHA - Occupational Health Guidelines for Chemical Hazards. DHHS(NIOSH) PublicationNo. 81-123 (3 VOLS). Washington, DC: U.S. Government Printing Office, Jan. 1981. 1].

Other Chemical/Physical Properties [940]:

Combines directly with hydrogen, with the halogens (excluding iodine); oxidized to selenious acid by nitric acid, to selenic acid by sulfuric acid; reduces hot aqueous soln of silver and gold salts with formation of silver selenide and metallic gold, respectively [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Dark red-brown to bluish-black solid; sol in carbon disulfide, methylene iodide, benzene or quinoline; density: 4.28; Softens @ 50-60 deg c & becomes elastic @ 70 deg c; formed when molten selenium is cooled rapidly; when freshly precipitated, reacts with water @ 50 deg c forming selenious acid & hydrogen /amorphous selenium/ [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Dark red, transparent crystals; two monoclinic forms: both forms are metastable & change into gray form on heating; melting point: below 200 deg c; density (alpha-form) 4.46 /Red selenium/ [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Soluble in concentrated nitric acid; crystalline selenium is a p-type semiconductor /red selenium/ [Sax, N.I. and R.J. Lewis, Sr. (eds.). Hawley's Condensed Chemical Dictionary. 11th ed. New York: Van Nostrand Reinhold Co., 1987. 1032].

Lustrous gray to black hexagonal crystals; insol (sic, actually "relatively insoluble") in alcohol; very slightly sol in carbon disulfide (2 mg/100 ml); sol in ether; conducts electricity & rectifies alternating current; conductivity increases up to 1000 times on exposure to light; density 4.81 @ 20 deg C/4 deg C; melting point 217 deg C; Mohs' hardness: 2.0; Latent heat of fusion 16.4 cal/g; Latent heat of vaporization 20.6 kcal/mol; Linear coefficient of thermal expansion per degree C= 37X10-6; Specific heat (28 deg C): 0.084 cal/g/deg C; Surface tension (217 deg C): 92.5 dynes/cm; Thermal conductivity (25 deg C): 0.0007-0.00183 cal/(cm)(deg C)/sec. /Gray selenium/ [The Merck Index. 10th ed. Rahway, New Jersey: Merck Co., Inc., 1983. 1212].

Melting point: transition point to hexagonal: 60-80 deg c /amorphous selenium/ [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Exhibits both photovoltaic action, where light is converted directly to electricity; exhibits photoconductive action, where electrical resistance decreases with increased illumination [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-34].

Soluble in chloroform /gray selenium/ [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

Sol in carbon disulfide: 0.1 G/100 cc @ 46.6 Deg c /red selenium/ [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-125].

BP: 684.9 +/- 1 deg C /Gray selenium/ [Weast, R.C. (ed.) Handbook of Chemistry and Physics, 68th ed. Boca Raton, Florida: CRC Press Inc., 1987-1988.,p. B-34].

Fate.Detail: Detailed Information on Fate, Transport, Persistence, and/or Pathways:

See also: Information in Bio.Detail above for fate of selenium in soils; and information in Forms/Preparations/Formulations above.

Microorganisms in sediments and/or sewage are capable of elemental methylation [488]. This process is well publicized for mercury. Not so well known is the fact that selenium is methylated and probably demethylated in the environment and cycled through a number of components of the food web, complicating chemical determination of the chemical forms available to fish [488]. Dimethylselenide is generated slowly from raw sewage [488].

Biogeochemical Cycling: In terrestrial environments, insoluble (biologically unavailable) selenium in soils may be oxidized by weathering and converted into soluble and bioavailable forms like selenate and selenite [445]. In soil water, selenate and, to a lesser extent selenite and possibly water-soluble organoselenium, are bioavailable and readily taken up through plant roots. Plants chemically reduce a portion to selenium-2 and incorporate it into tissues in soluble and/or protein-bound amino acids such as selenocystine, selenocysteine, and selenomethionine [445]. Animals, including humans, then consume plants and other animals containing selenium. Plants, animals, and microorganisms release volatile forms of selenium (primarily dimethyl selenide and to a lesser extent dimethyl diselenide) into the atmosphere where it is oxidized, converted to elemental selenium, and returned to the earth with rain, snow, or precipitating particulate matter. In arid environments with alkaline soils (like the San Joaquin Valley), selenium returned to soils in plant and animal wastes is readily oxidized to selenate and once again becomes available for biological uptake and cycling [445].

Selenium cycling in aquatic systems is somewhat different than in terrestrial environments [445]. Waterborne selenium can remain in a dissolved form, or be removed from solution through biological uptake, precipitation, or volatilization [445].

As in terrestrial systems, selenium can be taken up through the roots of higher aquatic plants. Many other aquatic organisms (e.g., some bacteria, fungi, algae, and invertebrates that form important components in aquatic food chains) also readily take up waterborne selenate, selenite, and/or selenomethionine.

Selenium is also removed from the water column through adsorption and complexation onto clay and particulates, and through reaction and precipitation with some metals (e.g., selenite readily reacts with iron to form ferric selenite [Fe2(SeO3)3] or ferroselite [FeSe2]). As a result of these processes and deposition of dead plant and animal tissue, a substantial portion of the selenium in aquatic systems builds up in sediments, becomes

buried through subsequent sedimentation and/or is exposed to further chemical and microbial reduction. This reduction leads to the formation of insoluble elemental selenium, metal selenides, and organic selenium-2, including methylated selenium-2 forms. Selenium builds up most in sediments and tissues of plants, fish, and wildlife in slow-moving or still, biologically productive aquatic systems such as sloughs, wetlands, and some evaporation ponds. Microcosm studies revealed that selenium more readily accumulated in fine-textured, highly organic pond sediments than in sandy riverine sediments [445].

Several mechanisms exist in aquatic sediments to return (remobilize) selenium out of the sediments and back into the water column and/or atmosphere. Such mechanisms include: oxidation and methylation by plant roots and microorganisms, mixing and oxidation as a result of feeding by fish and wildlife or burrowing by benthic organisms, mixing and oxidation associated with water movements, and oxidation by plant photosynthesis. Additionally, plant roots, bottom-feeding fish and wildlife, and benthic invertebrates can all take up selenium directly from sediments. Selenium can also be released into the water and/or atmosphere in volatile, methylated forms by plants, animals, algae, and as a result of microbial activity. Although there is general agreement regarding the various biogeochemical cycles involving selenium, little quantitative information is available regarding process rates or the specific roles of biota in these cycles [445].

Notes on Selenium in Soil [366]:

> The forms of selenium in soil depend on soil pH and redox. At equilibrium, most soil selenium should be elemental selenium. [Parr, J.F., P.B. Marsh, and J.M. Kla (eds.). Land Treatment of Hazardous Wastes. Park Ridge, New Jersey: Noyes Data Corporation, 1983. 186].
>
> In areas of acid or neutral soils, the amount of biologically available selenium should steadily decline. The decline may be accelerated by active agricultural or industrial practices. In dry areas with alkaline soils and oxidizing conditions, elemental selenium and selenides in rocks and volcanic soils may oxidize sufficiently to maintain the availablity of biologically active selenium. [US Dept of Interior/Fish & Wildlife Service Contaminant Reviews; Selenium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review Biol Rept No (85)1.5 p.4 (1985)].

Absorption, Distribution and Excretion [366]:

> The rate of absorption as elemental selenium ... Is low. ... Liver & kidney are principal sites of deposition. Excretion of selenium is by urine, feces, sweat, & breath. [Clayton, G. D. and F. E. Clayton (eds.). Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. 3rd ed. New York: John Wiley Sons, 1981-1982. 2130].

Nonradioactive and radioactive metal salts were administered intravenously to Sprague Dawley rats. The highest amount of each metal approached the maximum tolerated dose. Cobalt (Co), silver (Ag), and manganese (Mn) were eliminated rapidly. The elimination of 20 to 50% of the dosage was observed for copper (Cu), thallium (Tl), bismuth (Bi), lead (Pb), cesium (Cs), gold (Au), zinc (Zn), mercury (Hg), selenium (Se), and chromium (Cr). The slowest excretion rate was measured for arsenic (As), cadmium (Cd), iron (Fe), methyl mercury (MeHg), and tin (Sn). No substantial elimination rate decline was observed for MeHg and Fe, and the decline was small for Tl, Cs, Hg, Sn, Co, Ag, Zn, Cr, and As. Elimination of Ag and Mn via feces was fast, with more than 70% eliminated on the first day. Cu, Tl, Pb, and Zn were excreted at a slower rate, with 30.6 to 38.3% excreted on the first day. The rest of the metals were eliminated slowly by the intestinal route. Co was removed rapidly via urine, while Pb, Sn, Zn, MeHg, Ag, Fe, Mn, and Cd were eliminated slowly. The biliary excretion of Ag, As, and Mn was fast, with 25.5, 30.2 and 16.2% eliminated in two hours. Cu, Se, Cd, Pb, Bi, and Co were eliminated at an intermediate rate via the biliary route. Ag, As, Mn, Cu, Se, Cd, Pb, Bi, and MeHg were highly concentrated in bile relative to plasma. Liver and kidney contained the highest concentrations of most metals. The intestinal route was the major path of elimination for Ag, Mn, Cu, Tl, Pb, Zn, Cd, Fe, and MeHg. Co, Cs, Au, Se, and Cr, were removed predominantly by urine. For Bi, Hg, As, and Sn the two routes were similar. [Gregus Z, Klaassen CO; Toxicol Appl Pharm 85 (1): 24-38 (1986)].

Laboratory and/or Field Analyses:

Many methods have been used to monitor for selenium [861, 953,1001,1003,1004,1005,1006]. EPA methods recommended depend on the application: whether for drinking water [40 CFR Part 141 and 1005,1006,1008], NPDES discharge permits [40 CFR 136 and 1005,1006], CERCLA [861,1005,1006], RCRA [861,1005,1006], or low-detection-limit water-quality based permitting [1001,1003,1004]. Other agencies (USGS, APHA, ASTM, NOAA, etc. also publish different "standard methods." If one simply wants to know whether or not the concentration exceeds EPA criteria or various low concentration benchmarks for humans, fish, or wildlife, it is not always too clear which "standard method" is optimum, although some might argue that for water, the 1996 EPA methods 1639 (lab method) and 1669 (field method, see details below) should apply.

Acceptable containers (after proper cleaning per EPA protocols) for Antimony, Arsenic, Cadmium, Copper, Lead, Nickel, Selenium, Silver, Thallium, and Zinc: 500-mL or 1-L fluoropolymer, conventional or linear polyethylene, polycarbonate, or polypropylene containers with lid [1003].

Low concentration criteria or benchmarks require relatively rigorous analyses using either hydride generation or graphite furnace Atomic Absorption rather than ICP methods. Detection

limits should be no higher than comparison benchmarks or criteria for various media (water, sediments, soil, tissues, etc), some of which are low (see sections above). Unless required to be even lower for comparisons with lower benchmarks, the detection limits should usually not exceed the following default concentrations often recommended by the National Park Service (Roy Irwin, National Park Service, Personal Communication, 1996):

0.50 ppm dry weight in tissues,

Detection Limits as low as 0.05 ppb are possible if required by lower benchmarks or criteria [953]

1.0 ppm dry weight in sediments and soils,

Note: for certain soils, detection levels may need to be as low as 0.3 mg/kg for protection from migration to groundwater, depending upon the exact Dilution-Attenuation Factor (DAF) [952]. Solids detection Limits below 1 ppb are possible if required by lower benchmarks or criteria [953]

0.001 ppm (mg/L) in water

Note: water detection limits may need to be this low when considering risk to aquatic organisms, since:

1) Long term safety of 2 to 5 ppb of total waterborne selenium to fish and other aquatic organisms can be questioned [463].

2) Excess selenium, even as low as 3-8 ppb in the water) can cause numerous life-threatening changes in feral fresh water fish [488].

Detection Limits as low as 0.001 ppb are possible if required by lower benchmarks or criteria [953]. In some situations (as when background concentrations are low), water detection limits of 0.83 ug/L may be appropriate, using EPA method 1639, since EPA Water Quality Criteria are as low as 5 ug/L [1001,1003]. EPA Method 1638 allows a detection limit of 1.2 ug/L [1003,1003].

Need to Analyze for Separate Selenium Species and then Add Them before Comparison with Certain Benchmarks:

On November 14, 1996, EPA proposed in the Federal Register that the acute toxicities of selenate, selenite, and one form of organoselenium are additive. They further proposed that all forms be added together to obtain a total for comparison with water quality criteria and that total selenium can be converted to dissolved by multiplying total by 0.996 (Federal Register Vol 61, no. 221,pages 5844 to 58449):

Dissolved vs. Total vs. Acid Soluble:

Although most of the lab tests done to develop water quality criteria and other benchmarks were originally based on "total" values rather than "dissolved" values, the lab settings were typically fairly clean and the numbers generated by the lab tests are therefore often even more comparable to field "dissolved" values than to field "total" values (Glen Suter, Oak Ridge National Lab, Personal Communication, 1995).

As of January 1995, the U.S. EPA was recommending that states use dissolved measurements in water quality standards for metals, in concert with recommendations EPA previously made for the Great Lakes [672]. The conversion factors recommended by EPA for converting total recoverable metals criteria to dissolved metal criteria were given as follows [672]:

Selenium (+4) conversion for acute and chronic criteria: 0.922 (for example, total recoverable selenium (+4) criteria x 0.922 = dissolved selenium (+4) criteria). EPA 1996 Great Lakes: EPA suggested in November of 1996 (Federal Register Vol 61, no. 221,pages 5844 to 58449) that: the following conversion may be used: A factor to 0.996 may be used to convert to convert total (recoverable) acute criteria for selenite to a dissolved criteria for selenite.

The conversion factors recommended by EPA for converting total recoverable selenium to dissolved concentrations in the January 1997 draft EPA Guidelines for 5 year 305(B) assessments was also 0.922.

Note: None of these generic conversion factors are universal. Both total and dissolved concentrations should be checked at new locations before relying on generic conversion factors (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

Filtration and Acidification of Water Samples:

For ICP water samples for metals, EPA recommends the following (40 CFR Part 136, Appendix C, pertaining to ICP analyses using method 200.7, 1994 edition of CFR Part 40):

1) For samples of "total or total recoverable elements," samples should be acidified to a pH of two or less at the time of collection or as soon as possible thereafter.

Note: In more recent (1996) guidance related to the more rigorous method 1669, EPA clarified (some would say confused or added data variability) the issue of when to acidify by stating:

"Preservation recommendations for Antimony, Arsenic, Cadmium, Copper, Lead, Nickel, Selenium, Silver, Thallium, and Zinc: Add 5 mL of 10% HN03 to 1-L sample; preserve on-site or immediately upon laboratory receipt" [1003].

Note: the nitric acid (triple distilled or not?) and dilution water (contaminated or not?) and containers (proper type, cleaned correctly or not?) used are all potential sources of contamination (see more detailed note below related to data variation factors).

2) For determination of dissolved elements, the samples must be filtered through a 0.45 micron membrane filter as soon as soon as practical after collection, using the first 50-100 ml to rinse the filter flask. Acidify the filtrate with nitric acid to a pH of 2 or less. Normally 3 mL of (1+1) of nitric acid per liter should be sufficient to preserve the sample.

3) For determination of suspended elements, the samples must be filtered through a 0.45 micron membrane filter as soon as soon as practical after collection. The filter is then transferred to a suitable container for storage and shipment, with no preservation required.

Contaminants data from different labs, different states, and different agencies, collected by different people, are often not very comparable (see also, discussion in the disclaimer section at the top of this entry).

As of 1997, the problem of lack of data comparability (not only for water methods but also for soil, sediment, and tissue methods) between different "standard methods" recommended by different agencies seemed to be getting worse, if anything, rather than better. The trend in quality assurance seemed to be for various agencies, including the EPA and others, to insist on quality assurance plans for each project. In addition to quality control steps (blanks, duplicates, spikes, etc.), these quality assurance plans call for a step of insuring data comparability [1015,1017]. However, the data comparability step is often not given sufficient consideration. The tendency of agency guidance (such as EPA SW-846 methods and some other new EPA methods for bio-

concentratable substances) to allow more and more flexibility to select options at various points along the way, makes it harder in insure data comparability or method validity. Even volunteer monitoring programs are now strongly encouraged to develop and use quality assurance project plans [1015,1017].

At minimum, before using contaminants data from diverse sources, one should determine that field collection methods, detection limits, and lab quality control techniques were acceptable and comparable. The goal is that the analysis in the concentration range of the comparison benchmark concentration should be very precise and accurate.

It should be kept in mind that quality control field and lab blanks and duplicates will not help in the data quality assurance goal as well as intended if one is using a method prone to false negatives. Methods may be prone to false negatives due to the use of detection limits that are too high, the loss of contaminants through inappropriate handling, or the use of inappropriate methods.

Other more detailed information on sources of potential variation in contaminants data:

> Variation in concentrations of contaminants may sometimes be due to differences in how individual investigators treat samples in the field and lab rather than true differences in environmental concentrations. It was recognition that collectors and labs often contaminate samples that led EPA to develop the 1600 series of water protocols for low detection limit applications [1001,1002,1003,1004]. In comparing contaminants data from different labs, different states, and different agencies, one should keep in mind that they are often not very comparable. They may be as different as apples and oranges since:
>
> > 1) Different Agencies (EPA, USGS, NOAA, and various State Agencies) publish different lab and field protocols. Each of these protocols is different and has typically changed over time.
> >
> > > Note: Even "Standard EPA Methods" which are supposedly widely used by consultants, industry, and academia, have been variable over time and between application category (Drinking Water vs. NPDES, vs. RCRA, vs. CERCLA, vs. Water-Quality Based permits, etc.).
> > >
> > > Preservation and other details of various EPA lab and field protocols have changed over the years, just as they have at USGS and various States and other agencies. USGS data from 30 years ago may be different than USGS data today due to differences (drift) in lab and

field protocols rather than differences in environmental concentrations.

2) Independent labs and field investigators are not always using "the latest and greatest methods," and it is difficult for them to keep up with all the changes from various agencies in the midst of their "real world" busy lives. Updates are not always convenient to obtain. For example, EPA changes are scattered through various proposed Federal Register Notices, various updates of CFRs, and numerous publications originating in many different parts of EPA and their contractors. The wording is sometimes imprecise and is often inconsistent between EPA methods for different applications.

3) The details of the way one person collects, filters, and acidifies water samples in the field may be different than the way another does it. Sources of potential variation include the following:

A) The protocol phrases "As soon as practical or as soon as possible." Different situations can change the elapsed time considered by the field collector to be "as soon as practical." It may take different amounts of time to get to a safe or otherwise optimum place to filter and/or acidify and cool the samples. In one case precipitation and other changes could be going on in the collection bottle while the bottle is on the way to filtration and acidification. In other cases, the field collector filters and acidifies the samples within minutes. Weather, safety concerns, and many other factors could play a role.

B) Differences in numerous other details of the method used can drastically change the results. Some cold, wet, hurried, or fire ant-bitten collectors might decide that it is not "practical" to filter and acidify quite so immediately in the field, and may decide the shore, a vehicle, a motel room, or even a remote lab are more "practical" locations. Filtering and acidifying in the field immediately has been thought of as a better option for consistency (see copper and silver entries for examples of what can happen if there is a delay). However, in recent methodology designed to prevent some the contamination and variability listed above, EPA has recently suggested that waiting until

the sample arrives at the lab before acidifying is OK [1003].

C) What kind of .45 micron filter was used? The flat plate filters that were used for years tended to filter .45 micron sizes at first and then smaller and smaller sizes as the filtering proceeded and the filter loaded up with particulate matter. As the filter clogged, the openings grew smaller and colloids and smaller diameter matter began to be trapped on the filter. For this reason, both the USGS and EPA 1600 series protocols have gone to tortuous-path capsule filters that tend to filter .45 micron sizes more reliably over time. Example of specifications from EPA method 1669:

> Filter—0.45-um, 15-mm diameter or larger, tortuous-path capsule filters, Gelman Supor 12175, or equivalent [1003].

D) "Normally 3 mL of (1+1) of nitric acid per liter should be sufficient to preserve the (water) sample" (40 CFR Part 136, Appendix C, pertaining to ICP analyses using method 200.7, 1994 edition of CFR Part 40). Sometimes it is not, depending on alkalinity and other factors. What field collectors sometimes (often?) do is just use pop tabs of 3 mL of nitric acid and hope for the best rather than checking to see that the acidity has been lowered to below a pH of two. EPA CFR guidelines just call for a pH of below two, whereas samples meant to be "acid soluble" metals call for a pH of 1.5 to 2.0 [25]. See also, various USEPA 1984 to 1985 Ambient Water Quality Criteria Documents for individual metals.

> Note: Some shippers will not accept samples with a pH of less than 1 for standard shipping (John Benham, National Parks Service Personal Communication, 1997).

E) One person might use triple distilled concentrated nitric acid rather than reagent grades of acid to avoid possible contamination in the acid, while another may not. When using very low detection limits, some types of acid may introduce contamination and influence the results. Using a 10% dilution of nitric acid as called for by EPA [1003] is another

potential source of contamination, since the dilution water and/or containers may be contaminated. Sometimes people may be incorrectly determining that background concentrations are high due to contamination sources such as these (Pat Davies, Colorado Division of Wildlife, personal communication, 1997).

> Note: Just using triple distilled nitric acid may not be the total answer to potential contamination. The key issue to be sure that the acid used is free of the metals being analyzed. In guidance for EPA method 1669, the use of "ultrapure nitric acid; or Nitric acid, dilute, trace-metal grade" is specified [1003]. In guidance for EPA method 1638, the use of "Nitric acid—concentrated (sp gr 1.41), Seastar or equivalent" is specified [1003].

F) Holding times can strongly influence the results and there can be quite a bit of variation even within EPA recommended 6 month limits (see Silver entry for details). Holding times recommended for EPA for water samples of metals other than mercury or chromium VI have usually been listed as 6 months (Federal Register, Volume 49, No. 209, Friday, October 28, 1984, page 43260). In the 1994 version of the CFR, NPDES holding times for mercury and Chromium VI are the same ones listed in 1984, but no EPA holding times are given for other metals (40 CFR, Part 136.3, Table 2, page 397, 1994). EPA sources stated this was a typo, that no one else brought it to their attention in the last 3 years, that 6 months is still an operable holding time for "other metals" including this one, and that 6 months is actually an artifact from the days when 6 month composite samples were used for NPDES permits rather than having been originally scientifically derived.

> Counterpoint: Although some information suggests that 6 months is probably too long for some contaminants in some scenarios (see silver and copper entries), not all of the information in the literature casts the 6 month metals holding time in such questionable light. In one study, two EPA research chemists found that preservation under certain

conditions of drinking water (EPA Method 200.8) metals samples to a pH of less than 2 effectively stabilized the metal concentrations for 6 months. They found that trace metal standards in the 10 to 50 ug/L concentration could be held in 1% nitric acid if a 5% change of concentration was acceptable [1009]. Some metal concentrations changed more than 5% (Zinc up to 24%, Selenium up to 23%) [1009]. Vanadium, Manganese and Arsenic changed up to 5-7% [1009]. In some of the trials, metals were higher after 6 months due to leaching from containers, while in some they were lower [1009]. The changes were nevertheless considered not of great consequence related to drinking water MCLs and EPA method 200.8 [1009]. However, it is not clear that the careful measures utilized (like rechecking to make sure the pH was less than 2, the use of particular kinds of water samples, the use of particular acids, etc.) in this one study replicates what goes on in day to day ("real world") contaminants lab work around the country.

Some EPA sources state that 6 months should be OK if the sample bottle is vigorously shaken and re-acidified in the lab prior to lab analyses, a practice not universally or even particularly commonly done in labs today. The degree to which a water sample is re-acidified, re-checked for pH, shaken before analysis, and the length of time it sits before and after these steps, seems to vary a lot between laboratories, and EPA guidance for various methods is not consistent. Some labs recheck pH, some don't. Some shake, some don't, etc. For drinking water, preservation is considered complete after the sample is held in pH of less than 2 for at least 16 hours [1007]. New EPA Method 1638 specifies:

> "Store the preserved sample for a minimum of 48 h at 0–4°C to allow the acid to completely dissolve the metal(s) adsorbed on the container walls. The sample pH should be verified as <2 immediately before withdrawing an aliquot for processing or direct analysis. If,

> for some reason such as high alkalinity, the sample pH is verified to be >2, more acid must be added and the sample held for sixteen hours until verified to be pH <2" [1003].

> For many other methods, the minimum holding time in acid is not stated or is different (see various EPA and other Agency methods).

G) If present, air in head space can cause changes in water sample concentrations (Roy Irwin, National Park Service, Personal Communication, based on several discussions with EPA employees and various lab managers in February 1997).

> Note: air from the atmosphere or in headspace can cause oxidation of anaerobic groundwater or anaerobic sediment samples. This oxidation can cause changes in chemical oxidation states of contaminants in the sample, so that the results are not typical of the anaerobic conditions which were present in the environment prior to sampling (John Benham, National Park Service, Personal Communication, 1997).

H) When is the sample shaken in the lab or the field? If the filter is acidified in the field, it will be shaken on the way back to the lab. If lab acidified, how much and when is the sample shaken and then allowed to sit again for various times periods before analyses? Many methods treat this differently, and what many field collectors and labs actually do before analyzing samples is different as well. For EPA method 1638, the word shake appears in the "Alternate total recoverable digestion procedure":

> "..Tightly recap the container and shake thoroughly" [1003].

I) If one field filters and acidifies, one often changes metal concentrations and colloidal content compared to samples not treated in this manner. Acidifying effects microbial changes. If one holds the samples a while before filtering and acidifying, the situation changes. In collection bottles,

there are potential aging effects: temperature changes, changes in basic water chemistry as oxygen and other dissolved gasses move from the water into the headspace of air at the top, potential aggregation of colloidal materials, precipitation of greater sizes over time, development of bigger and more colloids, and more sorption (Roy Irwin, National Park Service, personal communication, 1997).

4) The guidance of exactly where to take water samples varies between various state and federal protocols. Taking water samples at the surface microlayer tends to increase concentrations of various contaminants including metals. Other areas of the water column tend to produce different concentrations. Large quantities of anthropogenic substances frequently occur in the surface microlayer at concentrations ranging from 100 to 10,000 times greater than those in the water column [593]. These anthropogenic substances can include plastics, tar lumps, PAHs, chlorinated hydrocarbons, as well as lead, copper, zinc, and nickel [593]. Sometimes a perceived trend can be more the result of the details of the sample micro-location rather than real changes in environmental concentrations (Roy Irwin, National Park Service, personal communication, 1997). The new EPA method 1669 mentions the microlayer, and states that one can use a fluoropolymer closing mechanism, threaded onto the bottle, to open and close a certain type of bottle under water, thereby avoiding surface microlayer contamination [1003]. However, even this relatively new EPA method 1669 also gives recommendations for ways to sample directly at the surface, and does not discourage the use of surface samples.

5) Although the above examples are mostly related to water samples, variability in field and lab methods can also greatly impact contaminant concentrations in tissues, soil, and sediments. Sediment samples from different microhabitats in a river (backwater eddy pools vs. attached bars, vs. detached bars, vs. high gradient riffles vs. low gradient riffles, vs. glides, etc.) tend to have drastically different concentrations of metals as well as very different data variances (Andrew Marcus, Montana State University, personal communication, 1995). Thus, data is only optimally comparable if both data collectors were studying the same mix of microhabitats, a stratified sampling approach which would be unusual when comparing random data from different investigators.

6) Just as there are numerous ways to contaminate, store, ship, and handle water samples, so are there different agency protocols and many different ways to handle samples from other media. One investigator may use dry ice in the field, another may bury the samples in a large amount of regular ice immediately after collection in the field, while a third might place samples on top of a small amount of ice in a large ice chest. The speed with which samples are chilled can result in different results not only for concentrations of organics, but also for the different chemical species (forms) of metals (Roy Irwin, National Park Service, personal communication, 1997).

7) In comparing contaminants metals data, soil and sediment contaminant concentrations should usually be (but seldom has been) normalized for grain size, total organic carbon, and/or acid volatile sulfides before biologically-meaningful or trend-meaningful comparisons are possible (Roy Irwin, National Park Service, Personal Communication, 1997).

8) There has been tremendous variability in the precautions various investigators have utilized to avoid sample contamination. Contamination from collecting gear, clothes, collecting vehicles, skin, hair, collector's breath, improper or inadequately cleaned sample containers, and countless other sources must carefully be avoided when using methods with very low detection limits [1003]. The EPA 1600 series method, some of which are described below, are designed to minimize some the common sources of contamination.

Highlights from EPA Method 1639: Determination of trace elements in ambient waters by stabilized temperature graphite furnace atomic absorption:

This 1996 proposed EPA method provides procedures to determine dissolved elements in ambient waters at EPA water quality criteria (WQC) levels using stabilized temperature graphite furnace atomic absorption (GFAA) [1003]. It may also be used to determine total recoverable element concentrations in these waters [1003].

As of March 1997, the EPA 1600 series methods had not yet been officially approved in 40 CFR for use in NPDES permits, but the improvements in these methods were suggested by EPA staff to be wise practice when attempting low detection limit analyses for metals.

This method was developed by integrating the analytical procedures contained in EPA Method 200.9 with the stringent quality control (QC) and sample handling procedures necessary to avoid contamination and ensure the validity of analytical results during sampling and analysis for metals at EPA WQC levels [1003]. This method contains QC procedures that will ensure that contamination will be detected when blanks accompanying samples are analyzed [1003]. This method is accompanied by Method 1669: Sampling Ambient Water for Determination of Trace Metals at EPA Water Quality Criteria Levels (the "Sampling Method") [1003]. The Sampling Method is necessary to ensure that contamination will not compromise trace metals determinations during the sampling process [1003].

Many of the requirements for this method are similar to those for other EPA 1600 series methods [1003].

This method may be used with the following metals [1003]:

Antimony (Sb), CAS 7440-36-0
Cadmium (Cd), CAS 7440-43-9
Trivalent Chromium, CAS 16065-83-1
Nickel (Ni), CAS 7440-02-0
Selenium (Se), CAS 7782-49-2
Zinc (Zn), CAS 7440-66-6

For dissolved metal determinations, samples must be filtered through a 0.45-um capsule filter at the field site [1003]. The filtering procedures are described in the Sampling Method [1003]. Except for trivalent chromium, the filtered samples may be preserved in the field or transported to the laboratory for preservation [1003]. Procedures for field preservation are detailed in the Sampling Method; procedures for laboratory preservation are provided in this method [1003]. To determine trivalent chromium, a field preparation step, which is described in the Sampling Method, is used to isolate the trivalent chromium [1003].

To determine total recoverable analytes in ambient water samples, a digestion/extraction is required before analysis when the elements are not in solution (e.g., aqueous samples that may contain particulate and suspended solids) [1003].

Construction materials—Only the following materials should come in contact with samples: fluoropolymer (FEP, PTFE), conventional or linear polyethylene, polycarbonate, polypropylene, polysulfone, or ultrapure quartz [1003]. PTFE is less desirable than FEP because the sintered material in PTFE may contain contaminates and is susceptible to serious memory contamination

[1003]. Fluoropolymer or glass containers should be used for samples that will be analyzed for mercury because mercury vapors can diffuse in or out of the other materials resulting either in contamination or low-biased results [1003]. All materials, regardless of construction, that will directly or indirectly contact the sample must be cleaned using EPA procedures and must be known to be clean and metal free before proceeding [1003].

The following materials have been found to contain trace metals and must not be used to hold liquids that come in contact with the sample or must not contact the sample itself, unless these materials have been shown to be free of the metals of interest at the desired level: Pyrex, Kimax, methacrylate, polyvinylchloride, nylon, and Vycor [1003]. In addition, highly colored plastics, paper cap liners, pigments used to mark increments on plastics, and rubber all contain trace levels of metals and must be avoided [1003].

Serialization—It is recommended that serial numbers be indelibly marked or etched on each piece of Apparatus so that contamination can be traced, and logbooks should be maintained to track the sample from the container through the labware to injection into the instrument [1003]. It may be useful to dedicate separate sets of labware to different sample types; e.g., receiving waters vs. effluents [1003]. However, the Apparatus used for processing blanks and standards must be mixed with the Apparatus used to process samples so that contamination of all labware can be detected [1003].

Do not dip pH paper or a pH meter into the sample; remove a small aliquot with a clean pipet and test the aliquot [1003]. When the nature of the sample is either unknown or known to be hazardous, acidification should be done in a fume hood [1003].

Store the preserved sample for a minimum of 48 h at 0–4°C to allow the acid to completely dissolve the metal(s) adsorbed on the container walls [1003]. The sample should then verified to be pH < 2 just before withdrawing an aliquot for processing or direct analysis [1003]. If for some reason such as high alkalinity the sample pH is verified to be > 2, more acid must be added and the sample held for 16 h until verified to be pH < 2 [1003].

One of the requirements for the alternate total recoverable digestion procedure is to tightly recap the container and shake thoroughly [1003].

Highlights from EPA Method 1669 for Sampling Ambient Water for Trace Metals at EPA Water Quality Criteria Levels [1003]:

As of March 1997, the 1600 series methods had not yet been officially approved in 40 CFR for use in NPDES permits, but the improvements in these methods were suggested by EPA staff to be wise practice when attempting low detection limit analyses for metals.

This "field method details" protocol is for the collection and filtration of ambient water samples for subsequent determination of total and dissolved Antimony, Arsenic, Cadmium, Copper, Chromium III, Chromium VI, Lead, Mercury, Nickel, Selenium, Silver, Thallium, and Zinc, at low (Water Quality Criteria Range) concentrations [1003]. It is designed to support the implementation of water quality monitoring and permitting programs administered under the Clean Water Act [1003].

This method is not intended for determination of metals at concentrations normally found in treated and untreated discharges from industrial facilities [1003]. Existing regulations (40 CFR Parts 400–500) typically limit concentrations in industrial discharges to the mid to high part-per-billion (ppb) range, whereas ambient metals concentrations are normally in the low part-per-trillion (ppt) to low ppb range [1003]. This guidance is therefore directed at the collection of samples to be measured at or near the water quality criteria levels [1003]. Often these methods will be necessary in a water quality criteria-based approach to EPA permitting [1001]. Actual concentration ranges to which this guidance is applicable will be dependent on the sample matrix, dilution levels, and other laboratory operating conditions [1003].

The ease of contaminating ambient water samples with the metal(s) of interest and interfering substances cannot be overemphasized [1003]. This method includes sampling techniques that should maximize the ability of the sampling team to collect samples reliably and eliminate sample contamination [1003].

Clean and ultraclean—The terms "clean" and "ultraclean" have been used in other Agency guidance [1004] to describe the techniques needed to reduce or eliminate contamination in trace metals determinations [1003]. These terms are not used in this sampling method due to a lack of exact definitions [1003]. However, the information provided in this method is consistent with summary guidance on clean and ultraclean techniques [1004].

Preventing ambient water samples from becoming contaminated during the sampling and analytical process is the greatest challenge faced in trace metals determinations [1003]. In recent years, it has been

shown that much of the historical trace metals data collected in ambient water are erroneously high because the concentrations reflect contamination from sampling and analysis rather than ambient levels [1003]. Therefore, it is imperative that extreme care be taken to avoid contamination when collecting and analyzing ambient water samples for trace metals [1003].

There are numerous routes by which samples may become contaminated [1003]. Potential sources of trace metals contamination during sampling include metallic or metal-containing sampling equipment, containers, labware (e.g. talc gloves that contain high levels of zinc), reagents, and deionized water; improperly cleaned and stored equipment, labware, and reagents; and atmospheric inputs such as dirt and dust from automobile exhaust, cigarette smoke, nearby roads, bridges, wires, and poles [1003]. Even human contact can be a source of trace metals contamination [1003]. For example, it has been demonstrated that dental work (e.g., mercury amalgam fillings) in the mouths of laboratory personnel can contaminate samples that are directly exposed to exhalation [1003].

For dissolved metal determinations, samples must be filtered through a 0.45-um capsule filter at the field site [1003]. The filtering procedures are described in this method [1003]. The filtered samples may be preserved in the field or transported to the laboratory for preservation [1003].

This document is intended as guidance only [1003]. Use of the terms "must," "may," and "should" are included to mean that EPA believes that these procedures must, may, or should be followed in order to produce the desired results when using this guidance [1003]. In addition, the guidance is intended to be performance-based, in that the use of less stringent procedures may be used so long as neither samples nor blanks are contaminated when following those modified procedures [1003]. Because the only way to measure the performance of the modified procedures is through the collection and analysis of uncontaminated blank samples in accordance with this guidance and the referenced methods, it is highly recommended that any modifications be thoroughly evaluated and demonstrated to be effective before field samples are collected [1003].

The method includes a great many details regarding prevention of field contamination of samples, including clothing needed, clean hands vs. dirty hands operations, and numerous other details [1003].

Surface sampling devices—Surface samples are collected using a grab sampling technique [1003]. Samples may be collected manually by direct submersion of the bottle into the water or by using a grab sampling device [1003]. Grab samplers may be used at sites where depth profiling is neither practical nor necessary [1003].

An alternate grab sampler design is available [1003]. This grab sampler is used for discrete water samples and is constructed so that a capped clean bottle can be submerged, the cap removed, sample collected, and bottle recapped at a selected depth [1003]. This device eliminates sample contact with conventional samplers (e.g., Niskin bottles), thereby reducing the risk of extraneous contamination [1003]. Because a fresh bottle is used for each sample, carryover from previous samples is eliminated [1003].

Subsurface sampling devices—Subsurface sample collection may be appropriate in lakes and sluggish deep river environments or where depth profiling is determined to be necessary [1003]. Subsurface samples are collected by pumping the sample into a sample bottle [1003]. Examples of subsurface collection systems include the jar system device or the continuous-flow apparatus [1003].

Advantages of the jar sampler for depth sampling are (1) all wetted surfaces are fluoropolymer and can be rigorously cleaned; (2) the sample is collected into a sample jar from which the sample is readily recovered, and the jar can be easily recleaned; (3) the suction device (a peristaltic or rotary vacuum pump, is located in the boat, isolated from the sampling jar; (4) the sampling jar can be continuously flushed with sample, at sampling depth, to equilibrate the system; and (5) the sample does not travel through long lengths of tubing that are more difficult to clean and keep clean [1003]. In addition, the device is designed to eliminate atmospheric contact with the sample during collection [1003].

Selection of a representative site for surface water sampling is based on many factors including: study objectives, water use, point source discharges, non-point source discharges, tributaries, changes in stream characteristics, types of stream bed, stream depth, turbulence, and the presence of structures (bridges, dams, etc.) [1003]. When collecting samples to determine ambient levels of trace metals, the presence of potential sources of metal contamination are of extreme importance in site selection [1003].

Ideally, the selected sampling site will exhibit a high degree of cross-sectional homogeneity [1003]. It may be

possible to use previously collected data to identify locations for samples that are well mixed or are vertically or horizontally stratified [1003]. Since mixing is principally governed by turbulence and water velocity, the selection of a site immediately downstream of a riffle area will ensure good vertical mixing [1003]. Horizontal mixing occurs in constrictions in the channel [1003]. In the absence of turbulent areas, the selection of a site that is clear of immediate point sources, such as industrial effluents, is preferred for the collection of ambient water samples) [1003].

To minimize contamination from trace metals in the atmosphere, ambient water samples should be collected from sites that are as far as possible (e.g., at least several hundred feet) from any metal supports, bridges, wires or poles [1003]. Similarly, samples should be collected as far as possible from regularly or heavily traveled roads [1003]. If it is not possible to avoid collection near roadways, it is advisable to study traffic patterns and plan sampling events during lowest traffic flow [1003].

The sampling activity should be planned to collect samples known or suspected to contain the lowest concentrations of trace metals first, finishing with the samples known or suspected to contain the highest concentrations [1003]. For example, if samples are collected from a flowing river or stream near an industrial or municipal discharge, the upstream sample should be collected first, the downstream sample collected second, and the sample nearest the discharge collected last [1003]. If the concentrations of pollutants is not known and cannot be estimated, it is necessary to use precleaned sampling equipment at each sampling location [1003].

One grab sampler consists of a heavy fluoropolymer collar fastened to the end of a 2-m-long polyethylene pole, which serves to remove the sampling personnel from the immediate vicinity of the sampling point [1003]. The collar holds the sample bottle [1003]. A fluoropolymer closing mechanism, threaded onto the bottle, enables the sampler to open and close the bottle under water, thereby avoiding surface microlayer contamination [1003]. Polyethylene, polycarbonate, and polypropylene are also acceptable construction materials unless mercury is a target analyte [1003]. Assembly of the cleaned sampling device is as follows:

Sample collection procedure—Before collecting ambient water samples, consideration should be given to the type of sample to be collected, the amount of sample needed, and the devices to be used (grab, surface, or subsurface

samplers) [1003]. Sufficient sample volume should be collected to allow for necessary quality control analyses, such as matrix spike/ matrix spike duplicate analyses [1003].

Highlights from EPA Method 1638: Determination of Trace Elements in Ambient Waters by Inductively Coupled Plasma — Mass Spectrometry:

This 1996 proposed EPA method is for the determination of dissolved elements in ambient waters at EPA water quality criteria (WQC) levels using inductively coupled plasma-mass spectrometry (ICP-MS) [1003]. It may also be used for determination of total recoverable element concentrations in these waters [1003]. This method was developed by integrating the analytical procedures in EPA Method 200.8 with the quality control (QC) and sample handling procedures necessary to avoid contamination and ensure the validity of analytical results during sampling and analysis for metals at EPA WQC levels [1003]. This method contains QC procedures that will assure that contamination will be detected when blanks accompanying samples are analyzed [1003]. This method is accompanied by Method 1669: Sampling Ambient Water for Determination of Trace Metals at EPA Water Quality Criteria Levels ("Sampling Method") [1003]. The Sampling Method is necessary to assure that trace metals determinations will not be compromised by contamination during the sampling process [1003].

This method may be used with the following metals:

Antimony (Sb), CAS 7440-36-0
Cadmium (Cd), CAS 7440-43-9
Copper (Cu), CAS 7440-50-8
Lead (Pb), CAS 7439-92-1
Nickel (Ni), CAS 7440-02-0
Selenium (Se), CAS 7782-49-2
Silver (Ag), CAS 7440-22-4
Thallium (Tl), CAS 7440-28-0
Zinc (Zn), CAS 7440-66-6

As of March 1997, the EPA 1600 series methods had not yet been officially approved in 40 CFR for use in NPDES permits, but the improvements in these methods were suggested by EPA staff to be wise practice when attempting low detection limit analyses for metals [1003].

This method is not intended for determination of metals at concentrations normally found in treated and untreated discharges from industrial facilities [1003]. Existing regulations (40 CFR Parts 400–500) typically limit concentrations in industrial discharges to the mid to

high part-per-billion (ppb) range, whereas ambient metals concentrations are normally in the low part-per-trillion (ppt) to low ppb range [1003].

The ease of contaminating ambient water samples with the metal(s) of interest and interfering substances cannot be overemphasized [1003]. This method includes suggestions for improvements in facilities and analytical techniques that should maximize the ability of the laboratory to make reliable trace metals determinations and minimize contamination [1003]. These suggestions are ...based on findings of researchers performing trace metals analyses [1003]. Additional suggestions for improvement of existing facilities may be found in EPA's Guidance for Establishing Trace Metals Clean Rooms in Existing Facilities, which is available from the National Center for Environmental Publications and Information (NCEPI) at the address listed in the introduction to this document [1003].

Clean and ultraclean—The terms "clean" and "ultraclean" have been applied to the techniques needed to reduce or eliminate contamination in trace metals determinations [1003]. These terms are not used in this method because of their lack of an exact definition [1003]. However, the information provided in this method is consistent with the summary guidance on clean and ultraclean techniques [1003].

The procedure given in this method for digestion of total recoverable metals is suitable for the determination of silver in aqueous samples containing concentrations up to 0.1 mg/L [1003]. For the analysis of samples containing higher concentrations of silver, succeedingly smaller volume, well-mixed sample aliquots must be prepared until the analysis solution contains <0.1 mg/L silver [1003].

Sample preservation—Preservation of samples and field blanks for both dissolved and total recoverable elements may be performed in the field at time of collection or in the laboratory [1003]. However, to avoid the hazards of strong acids in the field and transport restrictions, to minimize the potential for sample contamination, and to expedite field operations, the sampling team may prefer to ship the samples to the laboratory within two weeks of collection [1003]. Samples and field blanks should be preserved at the laboratory immediately upon receipt [1003]. For all metals, preservation involves the addition of 10% HNO_3 to bring the sample to pH <2 [1003]. For samples received at neutral pH, approx 5 mL of 10% HNO_3 per liter will be required [1003].

Do not dip pH paper or a pH meter into the sample; remove a small aliquot with a clean pipet and test the aliquot

[1003]. When the nature of the sample is either unknown or known to be hazardous, acidification should be done in a fume hood [1003].

Store the preserved sample for a minimum of 48 h at 0–4°C to allow the acid to completely dissolve the metal(s) adsorbed on the container walls [1003]. The sample pH should be verified as <2 immediately before withdrawing an aliquot for processing or direct analysis [1003]. If, for some reason such as high alkalinity, the sample pH is verified to be >2, more acid must be added and the sample held for sixteen hours until verified to be pH <2 [1003].

In some situations (as when background concentrations are low), water detection limits as low as 0.029 ug/L may be necessary for silver, using EPA method 1638, since EPA Water Quality Criteria are as low as 0.31 ug/L [1001].

In some situations (as when background concentrations are low), water detection limits as low as 0.0097 ug/L may be necessary for antimony, using EPA method 1638, since EPA Water Quality Criteria are as low as 14 ug/L [1001] [1003].

In some situations (as when background concentrations are low), water detection limits as low as 0.0079 ug/L may be necessary for thallium, using EPA method 1638, since EPA Water Quality Criteria are as low as 1.7 ug/L [1001] [1003].

In some situations (as when background concentrations are low), water detection limits as low as 0.14 ug/L may be necessary for zinc, using EPA methods 1638 or 1639, since EPA Water Quality Criteria are as low as 28 ug/L [1001] [1003].

EPA 1996 IRIS Database, information related to older methods for drinking water [893]:

Monitoring Requirements: Ground water systems monitored every 3 years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

Analytical Methods: Atomic absorption/furnace technique (EPA 270.2; SM 304): Previous PQL= 0.01 mg/L.

See also: EPA EMMI database [861].