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A PRELIMINARY ASSESSMENT OF SELENIUM IN DRINKING WATER

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FINAL REPORT

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A PRELIMINARY ASSESSMENT OF SELENIUM IN DRINKING WATER

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ABSTRACT

The current interim primary drinking water standard for selenium is undergoing review. As part of that effort, this study defines the major environmental sources of selenium exposure, identifies that portion of an individual's total daily selenium uptake arising from the consumption of drinking water, evaluates the toxicological significance of such uptake, and assesses the adequacy of the current standard in protecting the public health.

This preliminary review is intended to assist the Office of Drinking Water/EPA in defining a priority sequence for the inorganic contaminant standards review process. The data compiled in this document were obtained prior to June 1978.

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EXECUTIVE SUMMARY

The Mitre Corporation/Metrek Division has been assisting the Criteria and Standards Division, Office of Drinking Water, U.S. Environmental Protection Agency in an assessment of the adequacy of the current standard for selenium in drinking water. This report is a preliminary review of the current selenium standard, and is intended to assist the Office of Drinking Water in defining a priority sequence for the inorganic contaminant standards review process.

Selenium is ubiquitous in the environment; it is present in all rocks, soils, plants and animals. The major source of selenium in the environment is the weathering of rocks. Large areas of the Midwest and West contain high natural concentrations of selenium in the soil. Anthropogenic inputs appear to be minor and confined to the immediate vicinity of the source. Most (62 percent) of the selenium entering the environment from man's activities can be traced to coal combustion.

A major use of selenium is in the electronics industry. Xerography is becoming a major use due to the photoconducting properties of selenium. Selenium also finds applications in the glass and chemicals industries and as a nutritional additive in animal feeds.

The average concentration of selenium in the atmosphere appears to be well below 0.01 $\mu g/m^3$. The maximum daily intake from air has been reported as 0.07 μg/day. The literature provides no indication that pollution of the air by selenium is a problem at this time.

Food represents the major source of selenium intake for man. The usual dietary intake averages 170 μg/day, but can vary widely due to dietary preferences. Sea foods (especially shrimp), meat, milk products and grains provide the largest amounts of selenium in the diet; fruits and vegetables contain relatively small amounts.

Drinking water rarely contains selenium at levels above a few micrograms per liter. Most of the analyses reported are well below the current standard of 10 $\mu g/l$. However, selenium is easily leached from soils resulting in high selenium content of some ground water supplies. Levels as high as 12,000 $\mu g/l$ have been reported in well water in isolated cases. Toxic symptoms (e.g., depression, pallor, nervousness, gastrointestinal disturbances, dermatitis and garlic odor of breath and perspiration) have been noted in individuals drinking water containing these high concentrations.

Selenium can be absorbed into the body via ingestion, inhalation and through dermal contact. The only significant exposure route under ambient conditions is ingestion. Selenium is rapidly and efficiently absorbed from the gastrointestinal tract and is widely distributed to all body tissues. There does not appear to be any extensive accumulation in the tissues under normal circumstances.

The major excretory route for selenium in man is the urine. Other routes include feces, breath and perspiration.

The absorption, retention and distribution of selenium within the body and the amounts, forms and routes of excretion are functions of the chemical forms and amounts ingested as well as of the dietary levels of other elements such as arsenic, cadmium, sulfur and mercury. Within certain physiologic limits, the body appears to have a homeostatic mechanism for retaining trace amounts of selenium and excreting the excess.

Selenium is an essential trace element but can be toxic at high concentrations. Chronic selenosis has been reported in humans drinking water containing 9 ppm selenium. This is characterized by bad teeth, jaundice, chloasma, vertigo, chronic gastrointestinal disease, dermatitis, nail and hair changes, arthritis, edema, lassitude and fatigue. Epidemiological evidence indicates that selenium may increase the incidence of dental caries in children. It has been suggested that selenium may be a teratogen in man.

Selenium is also recognized as an essential element at trace levels. Its metabolic role concerns membrane elasticity, hence it is involved in the production and maintenance of membranes. Other studies indicate that selenium may influence the synthesis of glutathione and protein. Additional evidence suggests that selenium may have therapeutic value against cancer in man. Selenium is also protective against cadmium toxicity.

To determine whether the current drinking water standard is adequate, an evaluation of selenium content from the different sources is necessary. Ambient exposure levels and absorption rates for each exposure route are used to estimate total daily uptake.

Food is the major source of selenium. Somewhat more than 90 percent of the daily uptake can be attributed to this source, depending on the level assumed to be contributed by water. At the current interim drinking water standard of 10 μg/l, water would contribute approximately 10 percent of the daily selenium uptake. No good national estimates of selenium concentrations in drinking water

are available. However, most reported values are below the current standard. Since most of the waters analyzed contained a much lower concentration, water actually contributes less than 10 percent of the total daily uptake. In certain instances, drinking water can be the major source of selenium, however. In those cases where well water samples contained from 9,000-12,000 μg/l selenium, it would be expected that approximately 99 percent of an individual's daily selenium uptake could be attributed to drinking water.

The current uptake of selenium under normal conditions appears to be approximately 200 μg/day. Since toxic symptoms have been observed at levels as low as 600 μg/day, the current uptake represents a safety factor of only three. However, since neither deficiency nor toxicity have been reported at the 200 μg/day level, it is believed that the current uptake is in the required range.

A characteristic of selenium is the rather narrow range between toxicity and nutritional requirement. However, no symptoms of toxicity have been reported at levels generally encountered in the ambient environment. It appears that selenium deficiency may be a more serious health problem than selenium toxicity.

1.0 INTRODUCTION

The Office of Drinking Water (ODW) within the United States Environmental Protection Agency (EPA) in accordance with the Safe Drinking Water Act as amended has promulgated National Interim Primary Drinking Water Regulations for a number of physical, chemical, biological and radiological contaminants in potable water systems. Those interim regulations specify maximum contaminant level (MCLs) for substances in drinking water, and will be replaced by final Primary Drinking Water Regulations, as more definitive information describing the health risks associated with each contaminant is accumulated and analyzed.

The MITRE Corporation/Metrek Division has been assisting the Criteria and Standards Division, Office of Drinking Water, in their assessment of the adequacy of the current standard for selenium in drinking water*. In this preliminary assessment, the biological effects of selenium exposure are reviewed, the major environmental sources of selenium exposure (i.e., air, food, drinking water) are defined, and a discussion of the adequacy of the current interim standard is presented.

This initial review of the current selenium standard is being conducted concurrently with several other contaminants. This document is not intended to be a comprehensive assessment of the selenium standard, but rather a preliminary critique in order to assist

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*Current interim selenium standard - 10.0 μg/l.

the Office of Drinking Water in defining a priority sequence to be instituted for its inorganic contaminant standards review process.

1.1 Background

Selenium occurs in several chemical forms in the environment; i.e., elemental selenium, inorganic complexes (soluble and insoluble forms) and organic forms. Both availability to biological systems and toxicity vary depending on the chemical form present. Unfortunately, the literature does not consistently differentiate between the forms but presents the data based on quantitative analyses of the total selenium present. Where possible data will be presented according to chemical form.

Selenium is widely distributed in the environment, occurring primarily in foods. Some drinking water sources contain selenium at very high concentrations but the overwhelming majority of drinking waters sampled have levels well below the current standard. The amount of selenium in air appears to be insignificant.

Selenium is a paradoxical element; either a deficiency or an excess produces adverse biological effects. There is a relatively narrow margin of safety; a few parts per million can be toxic but trace amounts are required.

There are very few data in the literature which report adverse health effects in man from either a deficiency or an excess

of selenium. Selenium in water has been the cause of severe selenosis in a few isolated instances where geologic factors produced high concentrations of selenium in the well water of the area.

Because of the multiple exposure pathways, the interim primary drinking water standard for selenium is being reviewed and the relative contributions to an individual's daily selenium uptake arising from specific environmental media are being defined. In this way, if selenium appears to pose a significant health risk as a result of exposure via daily drinking water intake alone, or if drinking water intake contributes significantly to the total daily selenium intake, then an intensive review and analysis of the problem will be initiated.

1.2 Approach

In order to properly assess the health significance of exposure resulting from the ingestion of selenium-contaminated drinking water, it becomes necessary to define an individual's total daily selenium uptake from all pertinent sources, to assess the health impacts associated with that total daily uptake, and to identify that proportion of the total daily uptake arising from the ingestion of drinking water. In this preliminary assessment, the following sequence of steps is followed:

- quantify the major environmental sources of selenium exposure;
- determine the absorption/retention/elimination characteristics of those selenium compounds commonly found in the environment;

- define the toxicological impacts associated with selenium exposure, especially the low-level chronic effects;
- develop estimates of total daily selenium uptake in man based on ambient exposure levels and absorption/retention characteristics;
- assess the public health significance of various levels of selenium in drinking water, given ambient selenium contamination in other environmental media.

In developing this report, time constraints would not permit an exhaustive review of the scientific literature. Review articles were utilized as information sources when the primary citations were unavailable. Those instances when critical data were insufficient or lacking are pointed out in the text.

This review is intended to be a preliminary assessment of the current interim primary drinking water standard for selenium. Should the Office of Drinking Water decide that the selenium standard is high on its priority standards review list, then a comprehensive evaluation will be initiated to define the adequacy of the current interim standard.

2.0 ENVIRONMENTAL SOURCES OF SELENIUM EXPOSURE

Selenium can be found in all rocks, soils, plants and animals. It is present in the earth's crust at an average range of 0.03-0.8 ppm (NAS, 1976). The most commonly accepted average appears to be 0.09 ppm. Many areas of the United States contain high natural concentrations of selenium in the soil and in the plants. Figure 2-1 identifies these seleniferous areas.

Natural sources of selenium include weathering of rocks, volcanism, microbial action, volitilization by plants and animals and spray from large bodies of water (Johnson, 1976). Volcanic activity may be a significant source of the pollutant in air and soil (NAS, 1976). Probably the major source of selenium in the environment is the weathering of natural rock. Natural inputs of selenium appear to be much more important than those contributed by man's activities. It is believed that the impact of any industrial pollution on ambient selenium levels would be restricted to the immediate vicinity of the source (NAS, 1976).

Sources of industrial pollution by selenium include mining and milling; smelting and refining; and the manufacture of glass, steel, electronic components, and various chemicals. Most of the selenium entering the environment from anthropogenic sources can be traced to coal combustion (WHO, 1975; EPA, 1975a). For example, of the 1,215 tons of selenium released to the environment from industrial

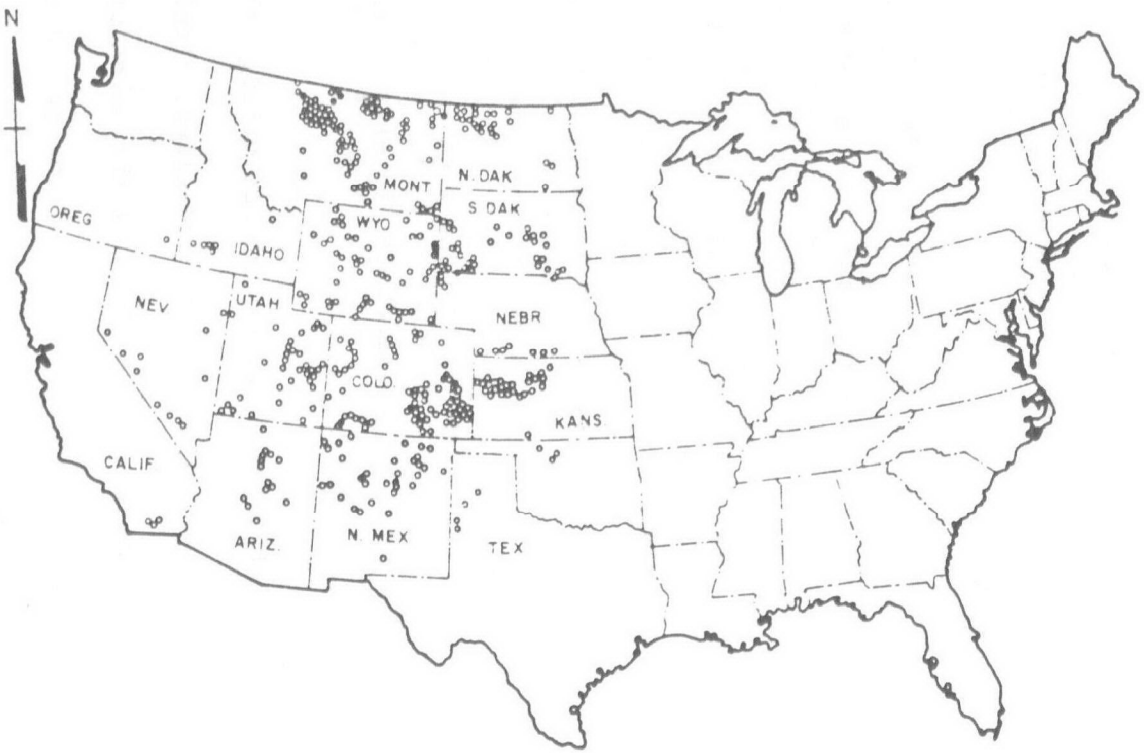

FIGURE 2-1
DISTRIBUTION OF SELENIFEROUS VEGETATION

Source: Lansche, 1967, as cited in Stahl, 1969

sources in 1970, 62% was from coal combustion (NAS, 1976). The burning of fossil fuels, in general, constitutes a major source of selenium input to the atmosphere, emitting an estimated 4,000 tons per year (Lakin, 1973). The burning of other organic matter also adds selenium to the atmosphere.

2.1 Selenium Concentrations in Ambient Air

A significant amount of the selenium in air probably has a natural source. At this time the portion attributable to natural sources cannot be quantified. Pollution from industrial sources seems to be confined to a small area in the immediate vicinity of the source. At a distance of 2 km from an electrolytic copper plant, the selenium concentration dropped from 0.50 $\mu g/m^3$ to 0.07 $\mu g/m^3$. At another plant, the concentration fell from 0.39 $\mu g/m^3$ to an undetectable level 2 km from the plant (NAS, 1976).

The average concentration of selenium in ambient air is probably well below 0.01 $\mu g/m^3$. It seems unlikely that pollution of the air by selenium is a problem at this time (NAS, 1976). Table 2-1 summarizes the data available concerning the levels of selenium in ambient air.

Daily selenium intake from air has been estimated at 0.07 μg/day (Woolrich, 1973), 0.02 μg/day (Casarett and Doull, 1975) and <1 μg/day (Schroeder et al., 1970).

TABLE 2-1

SELENIUM CONTENT OF AMBIENT AIR

Selenium Concentration ($\mu g/m^3$)	Location	Reference	Remarks
0.006 (avg)	Buffalo, NY	Pillay et al., 1971	14 samples with range of 0.0036-0.0095 $\mu g/m^3$
0.001	Cambridge, MA	Hashimoto et al., 1967	Based on concentration in rain and snow
0.0025	Niles, MI	Dams et al., 1970	Concentration in suspended particulates
0.0038	East Chicago, IN	Dams et al., 1970	Concentration in suspended particulates
0.0014	Boston area	Gordon et al., 1973	---
0.0044 (max)	Northwest Indiana	Harrison et al., 1971	Data from 24 hour samples at 25 sites

2.2 Selenium Concentrations in the Diet

Food represents the major source of selenium intake for man (Sterrett, 1977; WHO, 1973). There is a wide range in the selenium content of foods. Several factors influence these differences:

- The class of food - seafood, meat and grains tend to have high concentrations of selenium while fruits and vegetables have rather low concentrations.
- The origin of the food - foods from areas with high concentrations of selenium in the soil tend to contain more selenium.
- Food processing - more refined and/or processed foods usually contain less selenium; cooking and heating may reduce the selenium level due to volitilization (WHO, 1973).

Due to the nature of food distribution throughout the United States, there is no reason to expect either an inadequate or an excess amount of selenium in our diets (NAS, 1976).

Selenium content of foods has been monitoried by the FDA in its Total Diet Studies. Data from this study and other sources are presented in Table 2-2. The usual dietary intake has been reported as 150 μg/day (Mahaffey et al., 1975; ICRP, 1975), 60-150 μg/day (Schroeder et al., 1970), 170 μg/day (FDA, 1977), and 200 μg/day (EPA, 1976).

2.3 Selenium Content in Drinking Water

Data reporting the selenium content of waters are limited. The forms of selenium in drinking water have not been investigated; moreover, the current standard for selenium in drinking water is based on the total selenium content (EPA, 1976).

TABLE 2-2

SELENIUM CONTENT OF FOODSTUFFS

FOOD CLASS*	CONCENTRATION (ppm)	REFERENCE
I. Dairy Products	0.063	FDA, 1977
	Trace	Johnson and Manske, 1977
	0.069	Morris and Levander, 1970
	0.37	Schroeder et al., 1970
Whole milk	0.13	Morris and Levander, 1970
Human milk	0.018	Shearer et al., 1975
II. Meat, Fish and Poultry	0.221	FDA, 1977
	0.25	Johnson and Manske, 1977
Meat	0.224	Morris and Levander, 1970
	0.92	Schroeder et al., 1970
	0.48	NAS, 1976
Processed meat	0.33	NAS, 1976
Seafood	0.532	Morris and Levander, 1970
	0.99	Schroeder et al., 1970
III. Grains and Cereals	0.244	FDA, 1977; Johnson and Manske, 1977
	0.387	Morris and Levander, 1970
	0.15	Schroeder et al., 1970
IV. Potatoes	0.005	FDA, 1977
	0.005	Morris and Levander, 1970
	Not detected	Schroeder et al., 1970
	Trace	Johnson and Manske, 1977
V. Leaf Vegetables	0.002	FDA, 1977
	0.012	Morris and Levander, 1970
	0.17	Schroeder et al., 1970
VI. Legume Vegetables	0.004	FDA, 1977
	0.006	Morris and Levander, 1970
	0.01	Schroeder et al., 1970
	Trace	Johnson and Manske, 1977
VII. Root Vegetables	0.001	FDA, 1977
	0.021	Morris and Levander, 1970
	0.06	Schroeder et al., 1970
	Trace	Johnson and Manske, 1977
Garlic	0.276	Morris and Levander, 1970
VIII. Garden Fruits	0.001	FDA, 1977
	0.005	Morris and Levander, 1970
	Not detected	Schroeder et al., 1970
	Trace	Johnson and Manske, 1977
IX. Fruits	0.001	FDA, 1977
(Canned and Fresh)	0.006	Morris and Levander, 1970
	Not detected	Schroeder et al., 1970
X. Oil and Fats	0.002	FDA, 1977
	Trace	Johnson and Manske, 1977
XI. Sugar and Adjuncts	0.002	FDA, 1977
	Trace	Johnson and Manske, 1977
	0.15	Schroeder et al., 1970
Brown sugar	0.012	Morris and Levander, 1970
White sugar	0.003	Morris and Levander, 1970
XII. Beverages	0.001	FDA, 1977
	0.057	Schroeder et al., 1970
Coffee (ground)	0.124	FDA, 1977
Coffee (instant)	0.069	FDA, 1977
Coffee (ground and instant)	0.101	Shah et al., 1971
Tea	0.116	FDA, 1977; Shah et al., 1971

*According to FDA

The selenium content of water is a function of the pH of the water. Acid waters (pH 6.3-6.7) tend to precipitate selenium as a basic ferric selenite, while in alkaline waters (pH 8) selenium may be oxidized to soluble, toxic selenate (Lakin, 1973). Thus, areas with alkaline waters may be of special concern.

Natural waters are usually low in selenium content with an average concentration of 0.25 μg/l (Johnson, 1976). Available data suggest that surface waters rarely contain toxic levels of selenium or even amounts which would be significant in terms of nutritive requirements (NAS, 1976).

Drinking water rarely contains selenium at levels above a few micrograms per liter. However, the concentration of selenium in wells in seleniferous areas can be quite high. Levels as high as 210 μg/l have been reported in South Dakota (NAS, 1977). Under average conditions, drinking water cannot be considered a significant source of selenium (NAS, 1976). Table 2-3 provides a summary of the maximum values reported in drinking water samples analyzed for selenium. No explanation was given for the extremely high values reported in the table for Alabama and Utah. Though no fatalities occurred due to ingestion of these waters, symptoms of selenosis were reported. The estimated daily intake from drinking water ranges from negligible amounts (Sakurai and Tsuchiya, 1975) to <1 μg/day (Schroeder et al., 1970).

TABLE 2-3

MAXIMUM SELENIUM CONCENTRATIONS REPORTED IN DRINKING WATER SUPPLIES

Maximum Selenium Concentration (μg/l)	Location	Reference	Remarks
70	9 areas of U.S.	McCabe et al., 1970	Based on 2595 distribution samples; 10 samples (0.4%) exceeded 10 μg/l limit.
15	Interstate carriers	EPA, 1975	Based on analyses of 418 samples, only 1 (0.2%) failed the mandatory limit of 10 μg/l.
10	194 finished water supplies	Taylor, 1963	Results of a two year study; mean concentration was 8 μg/l.
0.11	Cambridge, MA	Hashimoto et al., 1967	Tap water samples
0.090	Cambridge, MA	Hashimoto et al., 1967	Well water samples
11	New York State	Public Water Supply Report, 1974	Found in 64% of the 312 distribution samples analysed; average concentration was 3.16 μg/l
12,000	Alabama	EPA, 1975a	Two well water supplies with concentration of 8,000 and 12,000 μg/l.
9,000	Utah	EPA, 1975a	Well water samples
20	National data	Lassovszky, 1978	Based on 1332 distribution samples from surface water sources; 2 samples (0.1%) exceeded the 10 μg/l limit.
30	National data	Lassovszky, 1978	Based on 2898 distribution samples from groundwater source; 11 samples (0.3%) exceeded the 10 μg/l limit.

Interim Primary Standard - 10 μg/l

2.4 Other Sources of Exposure

Smoking is an additional source which could add significant amounts of selenium to the body via the respiratory tract. The selenium content of cigarette tobacco has been reported at average concentrations of 0.08 ppm (Olson and Frost, 1970) and 0.35 ppm (Schroeder et al., 1970). The paper from these cigarettes adds an additional 0.05 ppm selenium (Olson and Frost, 1970; Schroeder et al., 1970). Pipe and cigar tobacco reportedly contain 0.08 and 0.33-1.01 ppm selenium, respectively (Olson and Frost, 1970).

3.0 ABSORPTION, RETENTION AND ELIMINATION OF SELENIUM IN HUMANS

Selenium is absorbed into the body via ingestion, inhalation and, to a lesser extent, through dermal contact. The concentration of selenium in the body is a function of intake levels, absorption rates, metabolic requirements and excretion rates.

Selenium is widely distributed in the internal organs. The liver and kidneys contain the largest amounts.

The amount of selenium in the diet has a major influence on the amount excreted via the different pathways. The main excretion route in humans is generally the urine but other routes include the feces, breath and perspiration.

The absorption, retention and distribution of selenium within the body and the amounts, forms and routes of excretion vary with the chemical forms and amounts ingested and with the dietary levels of other elements such as arsenic and sulfur (Underwood, 1977). Within certain physiologic limits, the body appears to have a homeostatic mechanism for retaining trace amounts of selenium and excreting the excess material (Casarett and Doull, 1975).

3.1 Absorption Characteristics

Absorption characteristics vary greatly with the chemical forms and amounts of selenium taken into the body and with the dietary levels of other elements such as arsenic, cadmium and mercury. Following absorption, selenium is transported by albumin to more stable binding sites in blood and tissues (Underwood, 1977).

3.1.1 Pulmonary Absorption

Due to the low concentrations in ambient air, the intake of selenium via the respiratory route is considered negligible (Sakurai and Tsuchiya, 1975). However, selenium may pose a possible hazard since it exists in air in a readily respirable physical state. In addition, selenium is localized on the surface of particulate matter and thus is readily available to dissolve and interact *in vivo* (Hausknecht and Ziskind, 1976).

Practically no quantitative data concerning pulmonary absorption of gaseous or particulate selenium compounds were found in the literature. The International Commission on Radiation Protection suggests an absorption rate of 70 percent for inhaled selenium compounds (ICRP, 1959). In subsequent calculations in this document, a pulmonary absorption rate of 70 percent has been assumed.

3.1.2 Gastrointestinal Absorption

Selenium is rapidly and efficiently absorbed from the gastrointestinal tract (McKee and Wolf, 1963; Diplock and Hoekstra, 1976). Rat studies indicate higher gastrointestinal absorption of selenium from grains grown in seleniferous areas than from selenites and selenates and very low absorption from selenides and elemental selenium (Underwood, 1977). Blood levels respond readily to the concentration of selenium in the diet (Lee, 1977). It is believed that the small intestine is the primary site of absorption (Lee, 1977). An absorption rate of 90% has been reported for humans (ICRP, 1959).

In addition, 50-80% of the ingested selenium is excreted via the kidneys in humans (Waldbott, 1973) so it can be assumed that at least these amounts are absorbed. In another study of three young women, intestinal absorption rates were reported as 70%, 64% and 44% (Underwood, 1977). Estimated absorption rates ranging from 95-100% have been reported for other mammals (NAS, 1976) which tends to support human rates reported. In subsequent calculations in this document, a GI absorption rate of 90 percent has been assumed.

3.1.3 Dermal Absorption

The absorption of selenium through the skin has been known to occur. This is usually in an industrial setting and does not appear to present a problem in the ambient environment.

3.2 Retention Characteristics

The selenium that is retained in the body is widely distributed. Although tissues do not appear to accumulate selenium to any great extent, some accumulation does occur in hair, liver and the kidneys and to a lesser extent in muscles. The kidneys retain the highest concentrations. Table 3-1 shows the distribution of selenium in the tissues of six humans at autopsy. Selenium is not stored but rather is rapidly eliminated from the body after the dietary source is removed. However, the liver may store selenium for a few weeks (Lee, 1977). At high doses, a balance between intake and excretion is found which prevents further accumulation (Schroeder et al., 1970).

TABLE 3-1

SELENIUM IN HUMAN TISSUES, ppm WET WEIGHT

Tissue	9 Months Male	41 yr Male	68 yr Male	69 yr Male	52 yr Female	68 yr Female	Mean
Liver	0.33	0.42	0.81	0.65	0.72	0.28	0.54
Lung	0.24	0.26	0.20	0.14	0.05	0.10	0.15
Heart	0.25	0.25	0.26	0.25	0.37	-	0.28
Kidney	0.70	0.75	1.84	1.52	1.12	0.61	1.09
Spleen	0.47	0.29	0.32	0.28	-	-	0.34
Bone (rib)	0.42	-	-	-	-	-	(0.42)
Muscle	0.18	0.17	0.36	-	0.38	0.11	0.24
Pancreas	0.34	0.29	0.27	-	-	-	0.30
Testes	0.15	0.36	0.38	-	-	-	0.30
Brain	0.04	0.21	-	-	-	-	0.13
Small intestine	0.18	0.32	0.12	-	-	-	0.21
(Fat) intestine	-	-	N.D.	-	-	-	-
Fat	-	0.04	-	-	-	-	-
Human milk	-	-	-	-	0.24	-	-
Breast	-	-	-	-	-	0.11	-
Mean							0.36

Source: Schroeder et al., 1970

The long-term fate of an oral dose of ^{75}Se-labeled seleno-methionine was reported by Griffiths et al. (1976). In this study involving four women, whole body radioactivity decreased exponentially with a half-time of 90 to 207 days.

3.3 Elimination Characteristics

Selenium is excreted from the human body via urine, feces, respiration, milk and perspiration. The urine is the predominant route of elimination under chronic conditions and may be an indicator of exposure (EPA, 1975). The feces is considered a minor route. In humans 50-80% of ingested selenium is excreted through the kidneys (Waldbott, 1973). The urine is believed to contain at least twice as much selenium as the feces (Casarett and Doull, 1975). Urinary output has been estimated at 50% or more of the ingested dose in humans where input and output are considered to be in an approximate balanced state (Sakurai and Tsuchiya, 1975). Animal studies tend to show agreement with these figures. Rats given a diet containing selenium as Na_2SeO_4 over a two-week period excreted about 50% of the ingested selenium in the urine and 12% in the feces within two weeks (Diplock and Hoekstra, 1976). In contrast to this, the Task Group on Reference Man reports only 33% excretion via the urine, 13% via the feces and 53% via sweat (ICRP, 1975).

Elimination characteristics are highly dependent upon chemical form, amount ingested and the presence of modifying factors

(Underwood, 1977). Animal experiments suggest that at low concentrations the urinary and fecal excretion routes attain a steady state. It appears that the body has effective removal mechanisms so that toxic levels do not occur (Stahl, 1969). As the dosage becomes larger, respiration and perspiration become important routes of excretion. The formation of volatile selenium is significantly greater and elimination via the lungs and perspiration increases (Diplock and Hoekstra, 1976). Selenium also appears to accumulate in hair which serves as an elimination mechanism (Casarett and Doull, 1975).

In the human body, natural detoxification occurs through reduction of selenium compounds to elemental selenium which is excreted through the kidneys and liver. Elemental selenium is also converted to dimethyl selenide which is excreted through the breath and perspiration (Stahl, 1969).

There are few data available which quantify the rates of excretion from the different routes. The selenium balance for reference man has been reported. Of the average 150 μg/day intake from foods and fluids (unknown inputs via atmosphere), 50 μg/day are reportedly excreted via the urine, 20 μg/day via the feces, 80 μg/day via perspiration, 0.3 μg/day are deposited in hair and trace amounts are lost through other fluids (ICRP, 1975).

Human body burden has been reported by two sources. The figures reported are 14.6 mg, with a range from 13.0-20.3 mg (Schroeder et al., 1970) and 15 mg (Casarett and Doull, 1975).

4.0 TOXICITY

Selenium is considered an essential trace element in animals. It is a micronutrient at levels up to 1 ppm (Waldbott, 1973) but toxic at levels above 4 ppm (Lakin, 1973). Toxic levels for man are estimated to be in the range of 600-6300 μg/day (EPA, 1976). Selenium is believed to be an essential trace element in man. Though no recommended daily intake has been established for humans, it has been determined that selenium performs certain nutritional functions.

The metabolic role of selenium deals with the production and maintenance of membranes (WHO, 1973). Selenium inhibits the oxidation of polyunsaturated fatty acids. This function curtails the production of "free radicals" which polymerize body proteins and diminish the elasticity of membrane tissues (Woolrich, 1973). Selenium may have a function in maintaining transmembrane cation gradients. Other studies indicate that selenium may influence the synthesis of glutathione and protein (WHO, 1973).

The toxicity of selenium depends on many factors, including the chemical form of the selenium compound and its solubility. The route of exposure is also important. Other factors include quantity consumed and the presence of modifying factors (e.g., other chemicals) in the diet.

Elemental selenium is relatively nontoxic. It is converted by the body into dimethyl selenide which is eliminated through the

breath and perspiration. The most toxic compounds are hydrogen selenide, methyl selenide and ethyl selenide. These compounds are retained in the tissues longer and in greater quantities (Waldbott, 1973).

Selenate and selenite are the forms which occur most often in water. Selenate compounds are of major concern due to their stability, solubility and ready availability to plants. Selenate is the form found in plants which accumulate selenium. Selenite, on the other hand, is less hazardous since it is likely to form insoluble compounds or be reduced to elemental selenium. Elemental selenium appears to be a major inert sink for selenium introduced into the environment. Though selenide forms are highly toxic, they represent an industrial hazard only due to rapid decomposition to elemental selenium in air (NAS, 1976).

Most toxicological data deal with acute exposure to selenium in animals. Acute exposures in man result in such symptoms as irritation of eyes and mucous membranes, sneezing, coughing, dizziness, dyspnea,* dermatitis, headaches, pulmonary edema,** nausea and garlic breath odor (NAS, 1977). Selenium is easily detected in fumes due to its unpleasant odor. Because of this, acute selenium poisoning is rare (Waldbott, 1973).

*Difficult or labored breathing.
**Abnormal accumulation of fluids in the pulmonary tissues.

Chronic intoxication is reported to produce the following symptoms: depression, marked pallor, coated tongue, languor, nervousness, occasional dermatitis, gastrointestinal disturbances, giddiness and garlic odor of breath and perspiration. Garlic odor is believed to be one of the earliest and most characteristic symptoms of exposure (Stahl, 1969).

The relationship between exposure to high levels of selenium in drinking water and human health effects was recently reported. Individuals drinking water containing selenium at concentrations between 50 and 125 μg/l were compared with controls consuming water containing 16 μg/l selenium or less. Even though urinary selenium levels were higher in the exposed group, there were no significant differences in the incidence or prevalence of any disease studied (Tsongas and Ferguson, 1977). Chronic selenosis has been reported in humans drinking water containing 9 ppm selenium. Lassitude, loss of hair and discoloration or loss of nails were symptoms noted (Cooper, 1967).

Epidemiological evidence indicates that selenium may increase the incidence of dental caries in children (Hadjimarkos, 1970).

Little informaton exists concerning the effects of long-term exposure to low levels of selenium. It does not appear that long-term systemic effects occur as a result of low-level, long-term exposure (Stahl, 1969).

Selenium toxicity is generally attributed to its interference with sulfur metabolism (Luckey and Venugopal, 1977). The harmful effects are believed to be caused by the organism's inability to distinguish between selenium and sulfur (Hausknecht and Ziskind, 1976).

Selenium interacts with many other compounds. Adequate dietary protein, organic sulfur, sulfate or arsenic decrease the toxicity of selenium by increasing its excretion rate (Luckey and Venugopal, 1977). Selenium has also been shown to stimulate gastrointestinal excretion of arsenic (Lee, 1977).

Selenium shows antagonistic properties with certain other elements. It has been shown to be highly effective in reducing the toxic effects of cadmium, mercury and arsenic (Diplock and Hoekstra, 1976).

Teratogenic effects have been reported in animals. Selenium has been shown to cross the placental barrier in several animal species. From the very limited data available, it has been suggested that selenium may be a teratogen in man (NAS, 1976).

Selenium has produced an increase in the incidence of liver tumors in rats but data are insufficient to evaluate the carcinogenicity of selenium compounds (IARC, 1975). Selenium has been suspected of being carcinogenic in man; however, there have been no direct reports of selenium carcinogenicity in humans. Moreover,

epidemiologic evidence suggests that selenium compounds may have therapeutic value against cancer in humans. These studies reveal an inverse relationship between human cancer mortality and environmental and blood selenium levels (Luckey and Venugopal, 1977). It is now believed that selenium can have an inhibitory effect on human cancer (Underwood, 1977). It is also possible that human breast cancer incidence and mortality could be lowered by appropriate dietary supplementation (Schrauzer and Ishmael, 1974).

5.0 SOURCE CONTRIBUTIONS TO DAILY SELENIUM UPTAKE IN HUMANS

To determine whether the current drinking water standard is adequate, an evaluation of selenium content from the different sources is necessary. An appreciation of the percent contributed via drinking water is needed to determine whether the dose contributed by drinking water is significant. An evaluation of the exposure from the other routes is also important in determining the overall hazards of exposure.

5.1 Approach

The method employed in this study to estimate the degree to which each major environmental source of selenium exposure contributes to an individual's total daily uptake is based on probable exposure conditions (i.e., ambient selenium levels) as well as absorption rates for each exposure route. The method consists of a five-step process:

- definition of ambient concentrations of selenium in the major exposure sources (i.e., air, food, and drinking water);
- determination of daily selenium intake from each exposure source according to the relationship:

$$I_i = C_i \cdot [Se]_i$$

where I_i is the daily selenium intake from each source i, C_i is the consumption per day of each source (i.e., air, food, drinking water), and $[Se]_i$ is the concentration of selenium in each source i;

- calculation of the amount of selenium absorbed from each exposure source:

$$U_i = I_i \cdot A_j$$

where U_i is selenium uptake for each exposure source i, I_i is daily selenium intake from each source i, and A_j is the fraction of selenium absorbed for each particular exposure route j (i.e., inhalation or ingestion);

- calculation of the total daily selenium uptake (U_t):

$$U_t = \Sigma(I_i \cdot A_j) = \Sigma U_i$$

for all appropriate pairs of i and j;

- determination of percent (P_i) of total daily uptake provided by each of the three exposure sources (i.e., source contribution factors):

$$P_i = \frac{U_i}{U_t} \cdot 100$$

5.2 Basic Assumptions

Several assumptions were made in defining the amount of each source material consumed each day. When possible, Reference Man* Values were utilized for daily air and food consumption rates (see Table 5-1). Daily consumption of drinking water is that value suggested by NAS and EPA (NAS, 1977).

Pulmonary and gastrointestinal absorption rates utilized in the calculations are also specified in Table 5-1. These figures represent reasonable absorption values for inhaled or ingested selenium, as reported in the scientific literature.

*From the ICRP Reference Man Tables (ICRP, 1975).

TABLE 5-1

BASIC ASSUMPTIONS EMPLOYED IN THE
CALCULATION OF INDIVIDUAL SOURCE CONTRIBUTION FACTORS

Basic Assumptions		Remarks
• Reference Man:		
Adult consumes:	2.0ℓ H_2O/day	- Daily intake as suggested by NAS (1977); conservative estimate, since all beverages assumed to be water, which has higher [Se] than generic beverages
	~2200g food/day	- Approximate daily intake for 18-yr. old in FDA total diet studies; comparable to Reference Man (ICRP report 23); however, since daily Se intake from the total diet will be assumed, this figure is not used in the calculations
	22.8 m^3 air/day	- Assumes 8 hrs. light work, 8 hrs. non-occupational, and 8 hrs. resting
• Absorption Characteristics:		
Gastrointestinal		
Adult	90%	- Approximation based on limited data
Pulmonary		
Adult	70%	- From ICRP (1959) data
Dermal	Insignificant	- Relatively unimportant, except in rare circumstances

5.3 Estimated Daily Selenium Uptake From All Sources

The relative contribution to an individual's daily selenium uptake from each of the three exposure routes was determined by using average environmental selenium occurrence data in the calculation sequence previously described. Several concentrations of selenium in drinking water and several values of daily intake from food are utilized to represent the range of values reported. The maximum reported value from Table 2-1 was used for ambient air concentration. Even at this level the contribution to an individual's daily uptake is negligible. Table 5-2 provides the exposure values used in the calculations.

It should be noted that selenium levels in the diet reflect daily selenium intake excluding any contribution by beverages. The selenium intake reported in beverages by FDA studies was much lower than that calculated for drinking water using the basic assumptions as previously outlined. Therefore, any error due to this manipulation would be a conservative one. Water is assumed to be the only beverage intake.

Table 5-3 provides an example of the actual calculation sequence employed. The source contribution factors for air, food and drinking water are summarized in Table 5-4.

Food is the major source of selenium under normal conditions, accounting for more than 90% of the total daily selenium uptake. At the level of the current interim drinking water standard (10 μg/l),

TABLE 5-2

REPRESENTATIVE ENVIRONMENTAL SELENIUM EXPOSURE LEVELS

Exposure Routes	Exposure Levels	Remarks
Diet	150 μg/day 200 μg/day	Selenium concentration in food is highly variable; dietary preferences are a major factor
Ambient Air	0.006 $\mu g/m^3$	Highest reported concentration (see Table 2-1)
Drinking Water	1 μg/ℓ	---
	10 μg/ℓ	National Interim Primary Drinking Water Standard
	30 μg/ℓ	Highest concentration reported in large National Survey (See Table 2-3)
	12,000 μg/ℓ	Highest concentration reported in isolated case (see Table 2-3)

TABLE 5-3

CALCULATION SEQUENCE IN DETERMINING SOURCE CONTRIBUTION FACTORS

Source	Ambient Concentration	x	Consumption Rate	x	Absorption Rate	=	Daily Uptake	Percent of Total Uptake
Drinking Water	10 µg/ℓ		2ℓ		0.9		18 µg/day	9.1%
Food	200 µg/day				0.9		180 µg/day	90.9%
Air	0.006 µg/m^3		22.8 m^3/day		0.7		0.096 µg/day	-----
					TOTAL		198.1 µg/day	100.0%

TABLE 5-4

ESTIMATED DAILY SELENIUM UPTAKE*

Selenium Concentration			Total Daily Selenium Uptake	Source Contribution Factors (Percent of Total Uptake)		
Drinking Water	Air	Food		Drinking Water	Air	Food
1 µg/ℓ	0.006 µg/m^3	150 µg/day	137 µg/day	1.4%	----**	98.6%
1 µg/ℓ	0.006 µg/m^3	200 µg/day	182 µg/day	1.1%	----	98.9%
10 µg/ℓ	0.006 µg/m^3	150 µg/day	153 µg/day	11.8%	----	88.2%
10 µg/ℓ	0.006 µg/m^3	200 µg/day	198 µg/day	9.1%	----	90.9%
50 µg/ℓ	0.006 µg/m^3	150 µg/day	226 µg/day	40.0%	----	60.0%
50 µg/ℓ	0.006 µg/m^3	200 µg/day	270 µg/day	33.3%	----	66.7%
12,000 µg/ℓ	0.006 µg/m^3	150 µg/day	21,735 µg/day	99.4%	----	.6%
12,000 µg/ℓ	0.006 µg/m^3	200 µg/day	21,780 µg/day	99.2%	----	.8%

* Selenium uptake = selenium absorbed

** Contribution < 0.1%

water represents approximately 10% of the daily selenium uptake. Since most of the values reported for water were less than 10 μg/l, the contribution is actually less than this figure. A level of 12,000 μg/l was reported for one drinking water source. It can be seen from the table that at this level water would be the source of over 99% of the selenium uptake. This represents an isolated case, however, and is not a common occurrence. Toxic effects were reported for people drinking this water which shows that water can be the source of selenium toxicity.

5.4 Significance of Current Standard

The current uptake of selenium under normal conditions appears to be approximately 200 μg/day. Since toxic symptoms have been reported in man at daily uptake levels of 600-6300 μg/day, the current levels represent a safety factor of only three. However, no data have been found which indicate toxicity at the current levels. There is not a very broad range between toxicity and nutritional requirements. The current standard appears to be in the range where neither deficiency diseases nor symptoms of toxicity occur.

There do not appear to be any groups within the general population who are at great risk from selenium exposure. However, since infants can be exposed *in utero* as well as through breast feeding, they may represent a sensitive population. Human milk contains an average of 0.021 ppm selenium. This means that a breast-fed infant

at 6 months might consume about 17 μg Se/day (ICRP, 1975). Available information does not permit an accurate assessment of the risk such an exposed infant may be assuming.

As more information becomes available concerning these points, the data will need to be taken into account when developing any new standard for selenium in drinking water.

5.5 Information Needs

In this preliminary review, there are several areas where data are limited or lacking:

- National ambient levels of selenium - more complete data are needed on levels, chemical forms and solubility of selenium in the environment.
- Human nutritional requirements - it is not definitively known at what levels selenium is required by man, although values averaging 150 μg/day have been suggested.
- Acute/chronic human toxicity levels - there is no clear differentiation between selenium exposure resulting in acute or chronic toxicity.
- Human absorption/retention/excretion rates and biological half-life - no human studies are currently available that provide definitive absorption/retention/excretion rates.
- Metabolic interactions with other elements - the interactions of selenium with mercury, cadmium and arsenic, among others, need to be more clearly defined.

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