Selenium in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

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Preface

One of the primary goals of the World Health Organization (WHO) and its Member States is that "all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water." A major WHO function to achieve such goals is the responsibility "to propose ... regulations, and to make recommendations with respect to international health matters ...."

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as *International Standards for Drinking-water*. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO *Guidelines for Drinking-water Quality* (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002. The third edition of the GDWQ was published in 2004, the first addendum to the third edition was published in 2006 and the second addendum to the third edition was published in 2008. The fourth edition will be published in 2011.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared and updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants of potential health concern in drinking-water. In the first and second editions, these constituted Volume 2 of the GDWQ. Since publication of the third edition, they comprise a series of free-standing monographs, including this one.

For each chemical contaminant or substance considered, a lead institution prepared a background document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Japan, the United Kingdom and the United States of America (USA) prepared the documents for the fourth edition.

Under the oversight of a group of coordinators, each of whom was responsible for a group of chemicals considered in the GDWQ, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors. The draft documents were also released to the public domain for comment and submitted for final evaluation by expert meetings.

During the preparation of background documents and at expert meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the Joint FAO/WHO Meetings on Pesticide Residues and the Joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite, in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO Internet site and in the current edition of the GDWQ.

Acknowledgements

Acronyms and abbreviations used in the text

ALAT	alanine aminotransferase
DNA	deoxyribonucleic acid
FAO	Food and Agriculture Organization of the United Nations
LOAEL	lowest-observed-adverse effect level
NOAEL	no-observed-adverse-effect level
USA	United States of America
WHO	World Health Organization

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1. GENERAL DESCRIPTION

1.1 Identity

Selenium (Se) is present in the earth's crust, often in association with sulfur-containing minerals. It is normally found in concentrations of 50–90 µg/kg, but higher concentrations can be associated with some volcanic, sedimentary and carbonate rocks. Selenium concentrations in soils vary widely, from 5 to 1 200 000 µg/kg, being higher in soils of more recent volcanic origin. Selenium occurs in soils in several forms, according to its possible oxidation states: selenides (Se^{2-}), amorphous or polymeric elemental selenium (Se^0), selenites (Se^{4+}) and selenates (Se^{6+}) (IPCS, 1987; UK EGVM, 2002).

1.2 Environmental fate

Acidic and reducing conditions reduce inorganic selenites to elemental selenium, whereas alkaline and oxidizing conditions favour the formation of selenates. Because selenites and selenates are soluble in water, selenium is leached from well-aerated alkaline soils that favour its oxidation. In contrast, elemental selenium and selenides are insoluble in water; therefore, selenium tends to be retained in wet, poorly aerated soils, the reducing conditions of which favour those forms. Thus, selenium in alkaline soils is available for uptake by plants, whereas the availability of selenium in acidic soils tends to be limited by the adsorption of selenites and selenates to iron and aluminium oxide sols (NRC, 1983).

1.3 Organoleptic properties

While elemental selenium and many selenides have garlicky odours similar to their sulfur analogues, the dominant forms of selenium found in water, selenites and selenates, are not odiferous. Therefore, it is unlikely that concentrations of selenium normally encountered in drinking-water will be detectable by odour (IPCS, 1987).

2. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

2.1 Air

Selenium is released into the air as hydrogen selenide, produced metabolically by plants, and as elemental selenium, selenites and selenates in particulate form. The level of selenium in most urban air ranges from 0.1 to 10 ng/m^3, but higher levels may be found in certain areas, such as in the vicinity of copper smelters (Zoller & Reamer, 1976).

2.2 Water

The levels of selenium in groundwater and surface water range from 0.06 µg/l to about 400 µg/l (Smith & Westfall, 1937; Scott & Voegeli, 1961; Lindberg, 1968). In some areas, selenium levels in groundwater may approach 6000 µg/l (Cannon, 1964). Concentrations increase at high and low pH as a result of conversion into compounds of greater solubility in water. Levels of selenium in tap water samples from public water supplies around the world are usually much less than 10 µg/l but may exceed 50

µg/l (NAS, 1976, 1977; Gore, Fawell & Bartram, 2010). Drinking-water from a high soil selenium area in China was reported to contain 50–160 µg/l (IPCS, 1987).

2.3 Food

Most people obtain virtually all of their selenium from the foods they eat. In plant and animal tissues, selenium is found mostly bound to proteins. Therefore, the most important food sources of selenium are meats and seafood (0.3–0.5 mg/kg), because of their high protein contents, and cereals (0.1–10 mg/kg), because they tend to be consumed in large amounts. In contrast, foods with relatively low protein levels, such as vegetables and fruits, tend to have relatively low selenium contents (<0.01 mg/kg).

In all cases, the selenium content of foods reflects the available selenium content of the soils used to produce those foods (and the feedstuffs used to produce livestock). Accordingly, great variations in the selenium content of foods occur, with high-selenium foods produced in parts of the upper Great Plains of North America and isolated localities in Venezuela and China. In China, the selenium content of corn, rice and soy beans varies from 0.005 to 45 mg/kg (NRC, 1983; IPCS, 1987).

FAO/WHO (1998) noted that global selenium intakes vary significantly; average intakes were relatively high in North America (85–150 µg/day), moderate in Europe (40–90 µg/day) and low in parts of China (10–20 µg/day). In Europe, dietary selenium intakes have declined in recent decades: 29–39 µg/day in the United Kingdom and 30–80 µg/day in the Nordic countries in 1997 (UK EGVM, 2002), compared with earlier intakes of 40–90 µg/day (FAO/WHO, 1998). This decline has been attributed to reductions in the importation of higher-selenium wheat grown in North America.

2.4 Contribution of drinking-water to selenium intake

Most drinking-water contains concentrations of selenium that are much lower than 10 µg/l, except in certain seleniferous areas. Therefore, it would be unusual for drinking-water to make a significant contribution to total selenium intake. Even in high-selenium areas, the relative contribution of selenium from drinking-water is likely to be small in comparison with that from locally produced food.

2.5 Recommended intakes

Selenium is an essential element, and therefore various national and international organizations have established recommended daily intakes of selenium. The joint World Health Organization (WHO)/Food and Agriculture Organization of the United Nations (FAO) consultation on preparation and use of food-based dietary guidelines (FAO/WHO, 1998) listed recommended intakes of 6–21 µg of selenium per day for infants and children, according to age, 26 and 30 µg of selenium per day for adolescent females and males, respectively, and 26 and 35 µg of selenium per day for adult females and males, respectively. In 2000, the United States National Academy of Sciences Panel on Dietary Oxidants and Related Compounds revised the recommended intake of selenium to 55 µg/day for both men and women and 70 µg/day for women during pregnancy and lactation. Recommended selenium intakes for children are between 15 µg/day for infants 0–6 months of age and 30 µg/day for

children 4–8 years old (NAS, 2000). The United Kingdom Expert Group on Vitamins and Minerals recommended selenium intakes of 60 µg/day for women and 70 µg/day for men (UK EGVM, 2002). However, it is clear that the position with regard to selenium requirements is more complex than these recommendations would suggest, because some groups, such as New Zealanders and Swedish vegans, have very low intakes, comparable to those in selenium-deficient parts of China, with no apparent adverse effects. Therefore, other aspects of the diet would appear to be important in mitigating the effects of low selenium intakes (FAO/WHO, 1998).

Because of concern about the adverse effects resulting from exposure to excessive levels of selenium, various national and international organizations have established upper limits of exposure for selenium. The United States National Academy of Sciences Panel on Dietary Oxidants and Related Compounds set an upper tolerable limit for selenium at 400 µg/day (NAS, 2000). This level was also recommended by FAO/WHO (1998) and the United Kingdom Expert Group on Vitamins and Minerals (EGVM, 2002).

3. KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

Most water-soluble inorganic and organic selenium compounds in foods are relatively efficiently absorbed across the gastrointestinal tract (80–95%) (Bopp, Sonders & Kesterson, 1982), although elemental selenium (Medinsky, 1981) and selenium sulfide (Cummins & Kimura, 1971) are poorly absorbed. After absorption, selenium is cleared by the liver and then transported to peripheral tissues by a specific transporter, selenoprotein P. In this way, selenium is distributed to all organs, with the highest concentrations occurring in kidney, liver, spleen, testes and skeletal muscle (Brown & Burk, 1973; Thomassen & Aaseth, 1986).

Selenium compounds are metabolized in three ways: to specific selenoproteins, to nonspecific proteins and to excretory products. The nutritionally essential functions of selenium appear to be discharged by some 25 selenoproteins, each of which contains selenium in the form of selenocysteine. This form is not found in any other protein, being produced by a unique co-translational modification of those specific proteins. The specific selenoproteins include glutathione peroxidases, thioredoxin reductases, 5-iodothyronine deiodinases, selenoprotein P and others. If selenomethionine is consumed, then that form of selenium can also be incorporated nonspecifically into proteins, as it can mimic methionine in protein synthesis.

Many forms of selenium (including selenite, selenate, selenocysteine and selenomethionine) are metabolized to hydrogen selenide. While the latter metabolite is the obligate precursor to the formation of selenocysteine in the specific selenoproteins, it can also be serially methylated (to methyl selenol, dimethylselenide and trimethylselenonium ion) or converted to a selenosugar and excreted.

4. EFFECTS ON LABORATORY ANIMALS AND IN VITRO TEST SYSTEMS

4.1 Selenium deficiency in animals

Deprivation of selenium can impair function and produce pathology in laboratory animals fed low levels of vitamin E or other antioxidants. Such pathologies include skeletal and cardiac myopathies, encephalopathies, transudative diathesis and infertility (Diplock, 1976; NRC, 1983). Conditions related to low concentrations in the environment include white muscle disease in cattle (Ellison, 2002).

4.2 Acute toxicity

Selenite, selenate, selenocysteine and selenomethionine are highly toxic and kill laboratory animals in single doses of 1.5–6 mg/kg of body weight (Högberg & Alexander, 1986; IPCS, 1987).

In rats, 5 mg of selenium per kilogram of diet may result in growth reduction (Halverson, Palmer & Guss, 1966; Ip, 1981). At a dietary level of 6.4 mg of selenium per kilogram (given as selenite), liver changes and splenomegaly occurred. At 8 mg of selenium per kilogram, anaemia, pancreatic enlargement and increased mortality were observed (Halverson, Palmer & Guss, 1966). Based on growth retardation, apparently caused by reduced secretion of growth hormone from the anterior pituitary gland as a result of local selenium accumulation (Thorlacius-Ussing, 1990), a no-observed-adverse-effect level (NOAEL) of about 0.4 mg of selenium per kilogram of body weight per day was suggested. Hepatotoxic effects have also been described following dietary administration of selenium (Harr et al., 1967; Harr & Muth, 1972). Based on both growth retardation and organ toxicity, a lowest-observed-adverse-effect level (LOAEL) of 0.03 mg/kg of body weight per day has been suggested.

The syndromes "blind staggers" and "alkali disease" have been described in livestock consuming selenium accumulator plants (Shamberger, 1983). The contributions of plant alkaloids to these conditions remain unclear.

4.3 Reproductive and developmental toxicity

Selenate, selenite, selenocysteine and selenomethionine are each teratogenic in avian species (Hoffman, Ohlendorf & Aldrich, 1988) and fish (Birget et al., 1983). Teratogenicity has also been observed in sheep (Rosenfeld & Beath, 1964) and pigs (Wahlström & Olson, 1959). In studies on monkeys (*Macaca fascicularis*) fed selenomethionine (25, 150 or 300 µg/kg of body weight per day) during organogenesis, no signs of teratogenicity were observed (Tarantal et al., 1991).

Adverse effects of selenate (3 mg of selenium per litre in drinking-water) on reproduction in mice and rats have been reported (Schroeder & Mitchener, 1971), but there are also two negative reports on the effects of selenite in hamsters and mice (Nobunaga, Satoh & Suzuki, 1979). Only at doses associated with overt maternal poisoning and nutritional deprivation was evidence of selenomethionine-induced embryonic or fetal toxicity observed in rabbits and hamsters (Berschneider et al., 1977; Ferm et al., 1990).

4.4 Mutagenicity and related end-points

A weak base pair substitution mutagenic activity has been demonstrated for both selenite and selenate in *Salmonella typhimurium* strain TA100 (Löfroth & Ames, 1978; Noda, Takano & Sakurai, 1979). Selenite, selenate and selenide induced unscheduled deoxyribonucleic acid (DNA) synthesis, sister chromatid exchange and chromosomal aberrations in cell cultures in vitro, often in the presence of glutathione (Ray & Altenburg, 1978; Whiting, Wei & Stich, 1980; Khalil, 1989). In one in vivo study, chromosomal aberrations and increased sister chromatid exchange were seen in hamster bone marrow cells after selenite treatment, but only at toxic doses (Norppa, Westermark & Knuutila, 1980).

4.5 Carcinogenicity

Early studies in which tumours were seen in test animals (Volgarev & Tscherkes, 1967; Inne et al., 1969) have been seriously questioned because of study limitations (IARC, 1975), and several evaluators have found the data to be inconclusive (e.g. Gore, Fawell & Bartram, 2010). In two studies on mice, there was either no increase or a decrease in the incidence of tumours after the administration of selenite or selenate (3 mg of selenium per litre of drinking-water) (Schroeder & Mitchener, 1972) or selenium oxide (2 mg of selenium per litre of drinking-water) (Schrauzer & Ishmael, 1974). Further data indicate an anticarcinogenic effect of selected selenium compounds. Viewed collectively, these data seem to show that the compounds studied will not act as carcinogens at low or moderate doses (Högberg & Alexander, 1986).

Selenium sulfide given by gavage resulted in hepatocellular carcinomas in rats and mice (NCI, 1980a) but caused no increased incidence in tumours when applied to the skin of mice (NCI, 1980b).

On the other hand, selenite, selenomethionine and some high-selenium foods have been shown to prevent or reduce carcinogenesis in every animal tumour model studied to date (e.g. Jackson & Combs, 2008). Animal studies have shown anticarcinogenic effects for both inorganic and organic selenium compounds at doses greater than needed for maximal selenoprotein expression (Combs & Lü, 2006) (see also section 5.1 below).

5. EFFECTS ON HUMANS

5.1 Inadequate levels of intake

Very low selenium status in humans has been associated with a juvenile, multifocal myocarditis called Keshan disease and a chondrodystrophy called Kaschin-Beck disease (Högberg & Alexander, 1986; IPCS, 1987; FAO/WHO, 2004). While Kaschin-Beck disease has not been well characterized, it is clear that selenium supplementation can prevent Keshan disease. Still, the etiology of Keshan disease has been difficult to understand. Studies by Levander & Beck (1999) and Beck, Levander & Handy (2003) have shed light, however; they suggest a role of a cardiophilic virus, the virulence of which increases in selenium-deficient hosts.

A major health focus of selenium has been its putative role in anticarcinogenesis. That selenium can be anticarcinogenic was suggested in the 1960s based on an inverse relationship of cancer mortality rates in the United States of America (USA) and crop selenium contents (Shamberger & Frost, 1969). Subsequent studies found blood selenium levels to be inversely associated with the prevalence of several types of cancer (Combs & Gray, 1998; Rayman, 2005; Combs & Lü, 2006; Gromadzińsk et al., 2008). The Nutritional Prevention of Cancer Trial (Clark et al., 1996) showed that supplemental selenium (200 µg/day as high-selenium yeast) reduced risks for total cancers and prostate and colorectal carcinomas. Although only a few other clinical trials have addressed this issue, most have indicated reduced cancer risk associated with selenium treatment (Raman, 2005; Combs & Lü, 2006; Gromadzińsk et al., 2008), although a recent one (Peters et al., 2008) found self-reported selenium supplement use to be unrelated to prostate cancer risk.

Evidence indicates several mechanisms for selenium anticarcinogenesis: altered carcinogen metabolism, cell cycle regulation, immune surveillance, cell death programming, cancer cell migration and angiogenesis (Combs & Gray, 1998; Rayman, 2005; Combs & Lü, 2006; Jackson & Combs, 2008). As these effects occur at supranutritional selenium doses, their molecular bases would appear to involve an increase in selenium metabolite(s) under such conditions (Combs & Gray, 1998; Combs & Lü, 2006; Jackson & Combs, 2008). Studies point to hydrogen selenide (Jiang et al., 2002) and its methylated metabolite methylselenol (Jackson & Combs, 2008) as active species, but selenomethionine may also play a role. Selenoproteins may also be involved, as differential cancer risk has been associated with allelic variants of some selenoproteins.

5.2 Excessive levels of intake

High dietary intakes of selenium have been identified in parts of Venezuela, China and South Dakota, USA (Smith & Westfall, 1937). Symptoms in people with high urinary selenium levels included gastrointestinal disturbances, discoloration of the skin and decayed teeth (Smith & Westfall, 1937). Children living in a seleniferous area in Venezuela exhibited more pathological nail changes, loss of hair and dermatitis than those living in Caracas (Jaffe, 1976).

In China, endemic selenium intoxication has been studied by Yang et al. (1983). Morbidity was 49% among 248 inhabitants of five villages where the daily intake was about 5 mg selenium. The main symptoms were brittle hair with intact follicles, lack of pigment in new hair, thickened and brittle nails and skin lesions. Symptoms of neurological disturbances were observed in 18 of the 22 inhabitants of one heavily affected village only. Those affected recovered once diets were changed following evacuation from the areas concerned.

In a follow-up study, Yang et al. (1989a,b) studied a population of about 400 individuals with average daily intakes ranging from 62 to 1438 µg. Clinical signs of selenosis (hair or nail loss, nail abnormalities, mottled teeth, skin lesions and changes in peripheral nerves) were observed in 5 of 439 adults having a mean blood selenium level of 1346 µg/l, corresponding to a daily selenium intake of 1260 µg. Decreases in prothrombin time and in the concentration of glutathione in blood were seen at dietary intakes exceeding 750–850 µg/day.

In a study in which 142 subjects from geographical areas where the average selenium intake was 239 µg/day (68–724 µg/day) were examined over 2 years (Longnecker et al., 1991), an association between selenium intake and increased alanine aminotransferase (ALAT) levels in serum was observed but considered to be clinically insignificant. None of the effects, including nail abnormalities, were related to selenium intake.

One case of selenium toxicity directly attributable to a water source has been reported. A family was exposed for about 3 months to well water containing 9 mg of selenium per litre. They suffered from loss of hair, weakened nails and mental symptoms, but they recovered when they stopped using the water from the well concerned (Rosenfeld & Beath, 1964).

Two individuals received about 350 and 600 µg of selenium per day via diet and selenium-containing yeast for 18 months. Marginal haematological changes and a borderline increase in ALAT levels were seen (Schrauzer & White, 1978). Levels of selenium in serum and erythrocytes were increased considerably in a small group of patients with rheumatoid arthritis who received daily supplements of 250 µg of selenium in selenium-enriched yeast in addition to selenium from food for 6 months, in comparison with patients receiving placebo (Tarp et al., 1985).

The average dietary intake that is associated with selenosis is in excess of 900 µg/day (Yang et al., 1989b; ATSDR, 2003). The studies by Yang et al. (1983) in China indicated that people living in areas with elevated selenium intake of 750 µg/day showed no overt signs of selenosis; in those areas in which selenosis was apparent, intake was approximately 3–6 mg/day. In other regions, such as South Dakota, USA, where consumption was similar to that in areas of China affected by selenosis, there were no apparent adverse effects (Longnecker et al., 1991; Levander, 1997). This may reflect differences in nutritional status, but a study by Reid et al. (2004) of individuals taking supplements failed to find adverse effects at intakes of 1.6 mg/day; adverse effects were reported by a group taking 3.2 mg of selenium per day, but these effects did not coincide with peaks of blood selenium.

6. PRACTICAL ASPECTS

6.1 Analytical methods and achievability

Atomic absorption spectrometry with hydride generation is the most convenient method for determining selenium in drinking-water. If 100 ml samples are used for routine analysis, the detection limit is about 0.5 µg/l. Inductively coupled plasma/mass spectrometry is also used, with a similar detection limit.

6.2 Treatment and control methods and performance

The most common forms of selenium in water are selenite (Se(IV), SeO_3^{2-}) and selenate (Se(VI), SeO_4^{2-}). The formation of selenate from selenite is slow, and both forms exist together in solution. Neither can be oxidized or reduced easily (Sorg & Logsdon, 1978). Selenate is more difficult to remove from water by processes such as

coagulation compared with selenite; therefore, oxidation of selenite to selenate would be undesirable in this context.

It has been reported that chemical clarification with lime, ferric sulfate or aluminium sulfate and activated carbon adsorption are moderately effective in removing selenite from water and ineffective at removing selenate. Tests have shown that the greatest removal was achieved by clarification with ferric sulfate at a pH below 7 (Culp/Wesner/Culp, 1986).

Selenium can be adsorbed onto iron oxide–coated sand. Practically complete removal of Se(IV) from a 10 mg/l solution in contact with 100 g/l coated sand was achieved within 10 min, whereas Se(VI) removal required about 90 min. The adsorption capacity was approximately 1 mg/g of coated sand (Lo & Chen, 1997). Similar results were reported using aluminium oxide–coated sand, although the adsorption capacities were lower—approximately 0.5 mg/g for Se(IV) and 0.25 mg/g for Se(VI) (Kuan et al., 1998). Treatment of natural water containing 4 μg/l selenium with iron(II) hydroxide at pH 8.8 reduced the concentration to below 1 μg/l (Zingaro et al., 1997). Selenium can also be removed using zero-valent iron, which forms iron oxyhydroxides as corrosion products (Zhang, Amrhein & Frankenberger, 2005).

Less than 4% removal of either Se(IV) or Se(VI) could be obtained using 100 mg/l powdered activated carbon to treat well water spiked with selenium at 0.03 and 0.1 mg/l. The pH did not affect the results (Sorg & Logsdon, 1978).

Pilot plant trials have shown that adsorption by soil has the ability to remove selenium from water. Selenium removal of 95% was achieved in the absence of nitrate (Weres et al., 1990). The presence of nitrate interferes with the adsorption of selenium.

It is reported that activated alumina adsorption using Alcoa F-1 can lead to selenium removals of 98% at pH 5 (Flemming, 1986).

Other studies have confirmed that ion exchange using synthetic resins is capable of removing Se(VI) anions from groundwater (Baes et al., 1990). This study compared the ability of the synthetic resins with an amine-modified coconut coir (MCC-AE) and reported that MCC-AE may be used as a low-cost adsorbent/ion exchanger for the treatment of anion-contaminated groundwater.

Laboratory research suggested that both selenite and selenate may be removed by ion exchange and reverse osmosis (Culp/Wesner/Culp, 1986). Cellulose acetate and cellulose triacetate membranes were effective at removing both selenite and selenate; removals in excess of 95% were achieved (Huxstep & Sorg, 1987). A 95% removal of selenium was achieved by nanofiltration of highly contaminated agricultural drainage water during laboratory studies (Kharaka et al., 1996).

There are few, if any, reported data on the chlorination of selenium in water, and the following guidance is therefore general in nature. Under relatively harsh conditions, chlorine will oxidize selenite to selenate (Krivan et al., 1985), but this seems unlikely to be the case under water treatment conditions. Selenium is unlikely to react with ozone, chlorine dioxide or chloramines.

7. PROVISIONAL GUIDELINE VALUE

Selenium is an essential element for humans, and there are indications that selenium status may be marginal in many parts of the world, including western Europe. The potential for adverse effects from selenium deficiency appears to be dependent on a number of factors, including overall health and nutritional status. High intakes of selenium are associated with a number of specific diseases and the potential for adverse effects, but, again, this seems to be strongly influenced by other factors. The potential for subtle biochemical effects that may affect the incidence of diseases such as cancer and cardiovascular disease remains uncertain, for both low (i.e. increased incidence) and high intakes (i.e. increased or decreased incidence).

Water is not normally a major source of selenium intake, but it is important that a proper balance be achieved between recommended intakes and undesirable intakes in determining an appropriate guideline value for selenium in drinking-water. While for most parts of the world, the concentration of selenium in drinking-water will not exceed 10 µg/l, there are circumstances in which selenium may be elevated above normal concentrations, and guidance may be required. Where selenium intake from the diet is known, this should be used in determining a concentration that ensures that intake is safe and sufficient. Where selenium intake from the diet is not known, guidance may be required. In determining a guideline value, an allocation of 20% of the upper tolerable intake of 400 µg/day to drinking-water provides a sensible balance that will assist regulators and suppliers in making decisions about whether further action is needed. This gives a guideline value of 40 µg/l. The choice of an allocation of 20% provides a significant margin to allow for those with a high intake. The guideline value is designated as provisional because of the uncertainties inherent in the scientific database.

For most Member States, a drinking-water guideline for selenium is unnecessary. Where there are regions of high intake from a number of sources, of which drinking-water may be one, then Member States should take into consideration exposure from all sources in determining actions to reduce exposure. For drinking-water, this may include using alternative sources, blending low-selenium sources with high-selenium sources as well as considering selenium removal.

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