Beryllium in Drinking-water

Background document for development of
WHO Guidelines for Drinking-water Quality
1. GENERAL DESCRIPTION

1.1 Identity

Beryllium (Be; CAS No. 7440-41-7; glucinium) is a steel-grey, brittle metal with an atomic number of 4 and an atomic weight of 9.01 (Group IIA of the periodic table). Free beryllium metal is not found in nature.

1.2 Physicochemical properties

Beryllium has an oxidation state of +2. In addition to forming various types of ionic bonds, beryllium has a strong tendency for covalent bond formation. At pH 7.5 only a small amount of beryllium will be in a soluble form in water, although solubility is likely to increase to a small extent at lower and higher pH. Of the simple beryllium compounds only the chloride, fluoride, nitrate, phosphate and sulfate are soluble at neutral pH.

1.3 Major uses and sources in drinking-water

Beryllium metal, beryllium alloys, and beryllium oxide are the commercially important end products of beryllium processing, respectively representing 10%, 75%, and 15% of the total usage of beryllium. Beryllium metal is used primarily in the aerospace, weapons, and nuclear industries. Beryllium alloy, mostly beryllium–copper, is used in the aerospace, electronics, and mechanical industries due to its unique properties. It has been detected at low concentrations in discharges from weapons manufacturing. Beryllium oxide is used for some ceramic applications, principally in electronics and microelectronics. The primary source of beryllium compounds in water appears to be release from coal burning, and other industries using beryllium. Only a few beryllium compounds are soluble and beryllium is primarily detected in surface water but at very low concentrations.

1.4 Environmental fate

In most natural waters, the majority of beryllium will be adsorbed to suspended matter or in the sediment, rather than dissolved. For example, in the US Great Lakes, beryllium is present in sediment at concentrations several orders of magnitude higher than its concentration in water. Beryllium in sediment is primarily adsorbed to clay, but some beryllium may be in sediment as a result of the formation and precipitation of insoluble complexes. At neutral pH, most soluble beryllium salts dissolved in water will be hydrolysed to insoluble beryllium hydroxide, and only trace quantities of dissolved beryllium will remain. However, at high pH, water-soluble complexes with hydroxide ions may form, increasing the solubility and mobility of beryllium.
Solubility may also increase at low pH; detectable concentrations of dissolved beryllium have been found in acidified waters.

2. ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

2.1 Water

Beryllium is concentrated in silicate minerals relative to sulfides and in feldspar minerals relative to ferromagnesium minerals. The greatest known naturally occurring concentrations of beryllium are found in certain pegmatite bodies. Certain fossil fuels contain beryllium compounds, perhaps accounting for its presence in some community air samples. Beryllium is not likely to be found in natural water above trace levels due to the insolubility of oxides and hydroxides at the normal pH range.

There are only limited data on beryllium concentrations in water except from the USA where a specific survey was carried out to support possible regulation.

Beryllium Detections and Concentrations in Surface Water and Ground Water (USEPA 2002)

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<tr>
<th>Detection Frequency</th>
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<th>Concentrations</th>
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<tr>
<td>99th percentiles MDI</td>
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<td>All samples mg/L</td>
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<td>Samples</td>
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<tr>
<td>Surface Water</td>
<td>0.64%</td>
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<td>Ground Water</td>
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The Method Detection Limit (MDL) for beryllium in water is 0.001 mg/L and the Maximum Contaminant Level (MCL) is 0.004 mg/L.

Surface waters have been reported to contain beryllium at concentrations up to 1000 ng/litre. Beryllium concentrations ranged from <4 to 120 ng/litre in the US Great Lakes and from <10 to 120 ng/litre (10–30 ng/litre average) in Australian river waters. Based on the US EPA’s STORET database for the years 1960–1988, the geometric mean concentration of total beryllium in US surface waters was estimated to be 70 ng/litre. Sediments from lakes in Illinois, USA, contained 1.4–7.4 mg beryllium/kg. Groundwater in Germany contained an average beryllium concentration of 8 ng/litre. Beryllium concentrations in water and sediment will be higher in the vicinity of point sources; concentrations of 30–170 µg/litre have been reported in industrial effluents.

There may be higher concentrations locally related to specific discharges.

2.2 Estimated total exposure and relative contribution of drinking-water

The general population may be exposed to trace amounts of beryllium by inhalation of air, consumption of drinking-water and food, and inadvertent ingestion of dust. The estimated total daily beryllium intake in the USA was 423 ng, with the largest contributions from food (120 ng/day, based on daily consumption of 1200 g of food containing 0.1 ng beryllium/g fresh weight) and drinking-water (300 ng/day, based on
daily intake of 1500 g of water containing 0.2 ng beryllium/g), and smaller contributions from air (1.6 ng/day, based on daily inhalation of 20 m$^3$ of air containing 0.08 ng beryllium/m$^3$) and dust (1.2 ng/day, based on daily intake of 0.02 g/day of dust containing 60 ng beryllium/g). The concentration used for beryllium in food was the midpoint of a range of values reported for a variety of foods in an Australian survey. The concentration used for beryllium in drinking-water was based on a survey of 1577 drinking-water samples throughout the USA, where beryllium was detected in 5.4% of samples with mean and maximum concentrations of 190 and 1220 ng/litre, respectively. The concentration used for beryllium in air was taken as a likely average concentration in a residential area based on air sampling results reported above. The concentration used for beryllium in household dust was estimated by assuming an indoor air concentration of 0.1 ng/m$^3$ and an air/dust ratio of 600. Although intake from air and dust are minor under background conditions, these can be important pathways of exposure in the vicinity of a point source. Beryllium intake through air and dust can be increased 2–3 orders of magnitude in the vicinity of a point source, such as a coal-fired power plant.

Tobacco smoke is another potential source of exposure to beryllium in the general population. Beryllium levels of 0.47, 0.68, and 0.74 µg/cigarette were found in three brands of cigarettes. Between 1.6 and 10% of the beryllium content, or 0.011–0.074 µg/cigarette, was reported to pass into the smoke during smoking. Assuming the smoke is entirely inhaled, an average smoker (20 cigarettes per day) might take in approximately 1.5 µg beryllium/day (3 times the combined total of the other routes). Other potential exposures to beryllium in the general population from consumer products are limited but may include leaching of beryllium from beryllium–nickel dental alloys and emission of beryllium from the mantle of gas lanterns.

3. Evaluation of health effects

3.1 Hazard identification and dose–response assessment

3.1.1 Non-cancer — oral

Beryllium is poorly absorbed from the gastrointestinal tract, probably because as soluble beryllium compounds pass into the intestine, which has a higher pH, the beryllium is precipitated as the insoluble phosphate and thus is no longer available for absorption.

There are no reliable data on the oral toxicity of beryllium in humans. Morgareidge et al. (1976) conducted a long-term feeding study in which groups of five male and five female beagle dogs (aged 8–12 months) were fed diets containing 0, 5, 50, or 500 ppm (mg/kg) of beryllium as beryllium sulfate tetrahydrate for 172 weeks. Dogs fed 500 ppm (mg/kg) beryllium as beryllium sulfate tetrahydrate (12.2 and 17.4 mg beryllium/kg body weight per day for males and females, respectively) developed gastrointestinal lesions. Similar, but less severe, gastrointestinal tract lesions were observed in one female given 50 ppm (1.3 mg/kg body weight per day) beryllium, which died during week 70. The remaining animals at this dose showed no histopathological alterations in the gastrointestinal tract related to treatment. A NOAEL of approximately 0.1 mg beryllium/kg body weight per day and a clear effect level of 12 mg beryllium/kg body weight per day for gastrointestinal tract lesions, anorexia, and weight loss in moribund dogs can be derived from this study. The LOAEL is not clear, as the findings are limited to one animal. To decrease reliance on findings on individual animals, the benchmark dose (BMD) approach was used to derive a BMD$_{10}$. The average of the male and female doses and the combined male
and female incidence for small intestinal lesions were modelled by the exponential polynomial, THRESH, and Weibull models; a 10% change (extra risk), BMD$^{10}$, was calculated to be 0.46 mg beryllium/kg body weight per day.

Gastrointestinal effects were not observed in rats or mice exposed to dietary beryllium sulfate, but the gastrointestinal tract was not examined in studies on beryllium carbonate.

"Beryllium rickets" was observed in young rats fed a "normal" stock diet for 3–4 weeks containing 0.125–3.0% beryllium carbonate (13–300 mg beryllium/kg body weight per day using a food factor of 0.05 with an estimate that the beryllium carbonate used in the study contained 20% beryllium). It is not known if exposure to beryllium compounds other than beryllium carbonate will result in rickets, because the available studies on beryllium sulfate (the only other beryllium compound with available oral toxicity data) did not examine the skeletal system or measure serum phosphate levels. It was noted that rickets was not observed in studies on beryllium-exposed rats, but the criteria used to assess potential rachitic effects were not reported. There was no mention of the occurrence of rickets in dogs that were observed daily and that underwent histological examination of the bone.

The potential of beryllium to induce developmental and/or reproductive effects has not been adequately assessed. In the only oral exposure study examining reproductive or developmental end-points, beryllium did not affect fertility or pup survival, weight, or skeletal formation in dogs. However, only small numbers of animals were evaluated, visceral examinations of pups and examination of dying pups were not conducted, and postnatal development was not evaluated. Measures of immune response or dysfunction have not been evaluated in oral exposure studies in animals.

3.1.2 Non-cancer — inhalation
In humans, the lung is the primary target of inhalation exposure to beryllium. Exposure to beryllium may result in the development of chronic beryllium disease (CBD), characterized by the formation of granulomas. These granulomas result from an immune reaction, primarily based on cell-mediated immunity. A genetic component to CBD susceptibility has also been identified. The toxicity of beryllium compounds increases with increasing water solubility.

3.1.3 Cancer weight-of-evidence
Studies regarding the potential carcinogenicity of ingested beryllium to humans are not available. Increases in lung cancer mortality were observed in cohort mortality studies of beryllium processing workers and in studies of entrants on the British Cancer Registry. No increases in other types of cancer were found, but increases in deaths from non-malignant respiratory disease were also observed. These studies are considered to provide evidence of carcinogenicity in humans exposed by inhalation; the evidence is limited because of relatively small increases in lung cancer risks, poorly defined estimates of beryllium exposure, incomplete smoking data, and lack of control for potential exposure to other carcinogens, including co-exposure to sulfuric or hydrofluoric acid mists during employment in the beryllium industry. Regardless of the shortcomings of the epidemiological studies of beryllium exposure, the results of all the follow-up mortality studies on the same cohort and of the BCR cohort studies are suggestive of a causal relationship between beryllium exposure and an increased
risk of lung cancer. This conclusion is strengthened by the increased incidences of lung cancers among workers with ABD (presumably these workers were exposed to very high concentrations of beryllium), the higher incidences of lung cancers among workers first employed when exposure levels were very high, a consistent finding of lung cancer excesses in six of seven beryllium processing facilities, and the occurrence of the highest risks for lung cancer in plants where the risk for non-malignant respiratory disease is the highest. Studies of beryllium carcinogenicity in animals are available by inhalation, intratracheal, oral, and parenteral exposure. Inhalation exposure to beryllium (metal, ores, and sulfate compounds) produced significant increases in lung cancer in rats and monkeys. These observations support the possible causal association noted in the occupational studies. Beryllium (metal, alloys, and compounds) has also been shown to produce lung cancer in rats by intratracheal instillation and osteosarcomas in rabbits by intravenous and intramedullary injection. Oral exposure studies using the sulfate tetrahydrate in rats and mice did not find significant increases in tumour incidences, but were inadequate for assessment of carcinogenicity due to the use of doses below the MTD. Overall, the animal data are considered to provide sufficient evidence of beryllium carcinogenicity in animals.

Genotoxicity data for beryllium are mixed. Beryllium did not produce gene mutations in the majority of bacterial assays, with or without metabolic activation. However, gene mutations were observed in mammalian cells cultured with beryllium chloride, and clastogenic alterations were found in mammalian cells cultured with beryllium chloride, beryllium sulfate, and beryllium nitrate.

3.1.2 Criteria for setting tolerable intakes/concentrations or guidance values for beryllium

3.1.2.1 Non-cancer — oral

An oral tolerable intake of 0.002 mg/kg body weight per day was estimated from the BMD_{10} (0.46 mg/kg body weight per day, dose calculated at the lower 95% confidence interval for a 10% incidence [response] of small intestinal lesions in the study by Morgareidge et al., (1976); assumed to be equal to a NOAEL) in dogs chronically exposed to beryllium sulfate tetrahydrate using the benchmark dose approach and an uncertainty factor of 300. The uncertainty factor of 300 was composed of 10-fold factors each for intra- and interspecies variation and a 3-fold factor for database deficiencies (no studies available on developmental effects and no mechanistic/mode of action data to suggest this may be an issue). Although there are several chronic oral animal studies, there is a lack of human toxicity data by the oral route, reproductive/developmental end-points have not been adequately assessed, and oral studies examining immunological end-points, the most sensitive end-point by the inhalation route, are lacking. Since the principal study is of chronic duration and a benchmark dose was used, there are no uncertainty factors for duration or NOAEL/LOAEL extrapolation.

3.1.2.2 Cancer

The oral carcinogenicity database is considered inadequate for assessing the carcinogenic potential of ingested beryllium. No human data are available, and the
animal studies produced only negative results and were limited by failure to achieve the MTD. Derivation of a quantitative cancer risk estimate for oral exposure is therefore precluded.

The inhalation carcinogenicity database includes both animal studies and epidemiology studies in exposed humans.

The inhalation tolerable concentration of 0.02 µg beryllium/m$^3$ is an estimate of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious non-cancer effects during a lifetime. For beryllium, therefore, an average lifetime exposure to 0.02 µg beryllium/m$^3$ (20 ng beryllium/m$^3$) is likely to be without appreciable risk of deleterious effects.

The International Agency for Research on Cancer evaluated the carcinogenicity of beryllium and assigned beryllium and beryllium compounds to Group 1, concluding that they are carcinogenic to humans. The assessment was based on sufficient evidence for carcinogenicity in humans and sufficient evidence for carcinogenicity in animals.

3.1.4 Uncertainties and degree of confidence in human health risk characterization

Although a number of subchronic studies in laboratory animals have been conducted with beryllium compounds, none has been done using modern criteria for high-quality toxicology studies.

Gastrointestinal effects have been observed in dogs, but not — in limited studies — in rodents. It is not clear if these effects are relevant to humans. There is an important uncertainty concerning the LOAELs of the gastrointestinal effects; this uncertainty was diminished by using the benchmark dose approach. Rickets was induced by beryllium carbonate in rats; again, it is not clear whether this effect, the mechanism of which apparently was an indirect one, is relevant to humans.

4. PRACTICAL ASPECTS

4.1 Analytical methods and analytical achievability

Beryllium can be analyzed with ICP/MS. The minimum quantification limit ranges from 0.000005mg/L to 0.001mg/L. It can also be analysed using ICP-AES for which the minimum quantification limit ranges from 0.002mg/L to 0.2mg/L. In some official methods for water analysis beryllium is recommended as an internal standard at 0.000005 mg/L because it is not normally measured in routine drinking-water analysis (Standard Methods for Water Supply, JWWA 2001, USEPA 2003). Analysis using an atomic absorption-platform appears to give a slightly better limit of detection over EPA Method 200.7 (USEPA 2003). All of these methods provide a means of achieving a level of detection of less than one tenth of the guideline value.
4.2 Treatment and control methods and technical achievability

Jar tests followed by centrifugation were used to evaluate beryllium removal by coagulation and lime softening. With an initial beryllium concentration of 18 µg/litre in river water, removals of 85% and 80% were achieved using aluminium sulfate (2.5 mg/litre Al) and ferric chloride (10 mg/litre Fe) respectively at a final pH of 6.5. Using the same procedure but without coagulant, 28% removal was obtained. Removal increased with increasing raw water pH in the range pH 6 to 9 but percentage removal was unaffected by initial beryllium concentration in the range 5 to 50 µg/litre. Removal of 20 µg/litre beryllium spiked into groundwater increased with lime dose in the range 75 to 450 mg/litre; at the maximum dose 99% removal occurred (Lytle et al. 1992).

Physico-chemical treatment of a domestic wastewater containing trace metals showed that clarification and filtration using lime (415 mg/litre lime; pH 11.5) as a coagulant removed 99.4% beryllium from an initial concentration of 100 µg/litre. When aluminium sulfate (18 mg/litre Al; pH 6.4) was used the removal was 98.1% and with ferric chloride (40 mg/litre as Fe; pH 6.2) the removal was 94%. Activated carbon only slightly increased removals with the lime and aluminium sulfate systems, but increased cumulative removal to 98.7% with the ferric chloride system (Hannah et al. 1977).

Laboratory tests were used to assess the removal of beryllium by adsorption on a range of natural and synthetic aluminium silicate zeolites, including greensand. With groundwater containing 1.6 µg/litre beryllium and tap water spiked at around 2 µg/litre removals in the range 50 to 80% were obtained with zeolite doses of 0.1 and 0.2 g/litre. Equilibrium was attained within about one hour. It was concluded that zeolites could be used in normal filter beds for beryllium removal (Strnadova et al. 2000).

No information was found on removal of beryllium by point of use or point of entry water treatment systems.

5. GUIDELINE VALUE

Beryllium is rarely, if ever, found in drinking-water at concentrations of concern, therefore, it is not considered necessary to set a formal guideline value. A health-based value for beryllium in drinking-water would be 12 µg/L based on an allocation of 20% of the TDI of 0.2 µg/kg body weight to drinking water and assuming a 60 kg adult drinking 2 litres of water per day. This allocation is probably conservative since the limited data on food indicate that exposure from this source is likely to be well below the TDI.

Although beryllium appears to be found in drinking-water sources and drinking-water at low concentrations, the database on occurrence is limited and there may be specific circumstances in which concentrations can be elevated due to natural sources where the pH is either below 5 or above 8 or there is high turbidity.
6. REFERENCES


[http://www.inchem.org/documents/cicads/cicads/cicad32.htm](http://www.inchem.org/documents/cicads/cicads/cicad32.htm)


USEPA (2003) Analytical feasibility support document for the six year review of existing National Primary Drinking Water Regulations (Reassessment of feasibility for chemical contaminants. (beryllium) EPA 815-R-03-003