WHO/HSE/WSH/09.01/5
English only

Beryllium in drinking-water

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

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Printed by the WHO Document Production Services, Geneva, Switzerland

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

The first draft of Beryllium in Drinking-water, Background document for development of WHO *Guidelines for Drinking-water Quality* (GDWQ), was prepared by Mr J.K. Fawell, United Kingdom, to whom special thanks are due.

The work of the following working group coordinators was crucial in the development of this document and others contributing to the fourth edition:

Dr J. Cotruvo, J. Cotruvo Associates, USA (*Materials and chemicals*)
Mr J.K. Fawell, United Kingdom (*Naturally occurring and industrial contaminants* and *Pesticides*)
Ms M. Giddings, Health Canada (*Disinfectants and disinfection by-products*)
Mr P. Jackson, WRc-NSF, United Kingdom (*Chemicals – practical aspects*)
Professor Y. Magara, Hokkaido University, Japan (*Analytical achievability*)
Dr Aiwerasia Vera Festo Ngowi, Muhimbili University of Health and Allied Sciences, United Republic of Tanzania (*Pesticides*)
Dr E. Ohanian, Environmental Protection Agency, USA (*Disinfectants and disinfection by-products*)

The draft text was discussed at the Expert Consultation for the fourth edition of the GDWQ, held on 19–23 June 2008. The final version of the document takes into consideration comments from both peer reviewers and the public. The input of those who provided comments and of participants at the meeting is gratefully acknowledged.

The WHO coordinators were Mr R. Bos and Mr B. Gordon, WHO Headquarters. Ms C. Vickers provided a liaison with the International Programme on Chemical Safety, WHO Headquarters. Mr M. Zaim, Public Health and the Environment Programme, WHO Headquarters, provided input on pesticides added to drinking-water for public health purposes.

Ms P. Ward provided invaluable administrative support at the Expert Consultation and throughout the review and publication process. Ms M. Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

Many individuals from various countries contributed to the development of the GDWQ. The efforts of all who contributed to the preparation of this document and in particular those who provided peer or public domain review comments are greatly appreciated.

Acronyms and abbreviations used in the text

BMD	benchmark dose
BMD_{10}	benchmark dose for a 10% response
LOAEL	lowest-observed-adverse-effect level
MCL	maximum contaminant level
MDL	method detection limit
MTD	maximum tolerable dose
NOAEL	no-observed-adverse-effect level
TDI	tolerable daily intake
USA	United States of America
USEPA	United States Environmental Protection Agency

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This background document is based on and largely taken from Concise International Chemical Assessment Document No. 32, entitled *Beryllium and beryllium compounds*. This document should be consulted for more detailed information and for primary references (IPCS, 2001).

1. GENERAL DESCRIPTION

1.1 Identity

Beryllium (Be; Chemical Abstracts Service Registry 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 values. 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 because of 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.

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.

The primary source of beryllium compounds in water appears to be release from coal burning and other industries using beryllium. Other sources of beryllium in surface water include deposition of atmospheric beryllium and weathering of rocks and soils containing beryllium.

1.4 Environmental fate

In most natural waters, the majority of beryllium will be adsorbed to suspended matter or in sediment, rather than dissolved. For example, in the Great Lakes in the United States of America (USA), 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 Air

Atmospheric beryllium concentrations at rural sites in the USA ranged from 0.03 to 0.06 ng/m^3. Lower levels may be found in less industrialized countries. Also in the USA, concentrations of 0.04–0.07 ng/m^3 have been reported at suburban sites and 0.1–0.2 ng/m^3 at urban industrial sites. A survey of beryllium concentrations in Japanese cities reported an average concentration of 0.042 ng/m^3 and a maximum concentration of 0.222 ng/m^3. Urban areas in Germany had beryllium concentrations in air ranging from 0.06 to 0.33 ng/m^3.

2.2 Water

Beryllium is not likely to be found in natural water above trace levels as a result of 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 (Table 1).

Table 1. Beryllium detection[1] and concentrations in surface water and groundwater in the USA (USEPA, 2002)

	Detection frequency >MDL		Detection frequency >MCL		Median concentration, all samples (mg/l)
	% samples	% sites	% samples	% sites	
Surface water	0.64	3.8	0.06	0.55	0.001
Groundwater	0.00	0.00	0.00	0.00	N/A

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

Data reported from the Czech Republic (F. Kozicek, personal communication, 2009) show that the average beryllium concentration in 19 173 water samples taken in

2004–2008 was 0.19 µg/l, with a median of 0.1 µg/l. In 11.29% of samples, the concentration was below the limit of determination, whereas the concentrations of 101 samples (0.53%) were above 2 µg/l, with a maximum of 35 µg/l.

2.3 Food

Beryllium is generally found in plant samples at concentrations below 1 mg/kg dry weight. Concentrations up to 100 µg/kg fresh weight have been reported in various fish and other marine organisms.

2.4 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 a beryllium concentration of 0.1 ng/g fresh weight) and drinking-water (300 ng/day, based on daily intake of 1500 g of water containing beryllium at 0.2 ng/g), with smaller contributions from air (1.6 ng/day, based on daily inhalation of 20 m^3 of air containing a beryllium concentration of 0.08 ng/m^3) and dust (1.2 ng/day, based on daily intake of 0.02 g/day of dust containing beryllium at 60 ng/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/l, 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 to dust ratio of 600. Although intakes 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 0.008–0.074 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 of beryllium per 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, Cox & Gallo (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 mg beryllium (as beryllium sulfate tetrahydrate) per kilogram for 172 weeks. Dogs in the high-dose group (beryllium doses of 12.2 and 17.4 mg/kg body weight per day for males and females, respectively) developed gastrointestinal tract lesions. Similar, but less severe, gastrointestinal tract lesions were observed in one female in the middle dose group (1.3 mg/kg body weight per day), which died during week 70. The remaining animals at this dose showed no histopathological alterations in the gastrointestinal tract related to treatment. A no-observed-adverse-effect level (NOAEL) for beryllium of approximately 0.1 mg/kg body weight per day (the low-dose group) and a clear beryllium effect level of 12 mg/kg body weight per day (the high-dose group) for gastrointestinal tract lesions, anorexia and weight loss in moribund dogs can be derived from this study. The lowest-observed-adverse-effect level (LOAEL) is not clear, as the findings are limited to one animal. To decrease reliance on findings in individual animals, the benchmark dose (BMD) approach was used to derive a benchmark dose for a 10% change (extra risk), or 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; the BMD_{10} for beryllium was calculated to be 0.46 mg/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 containing 0.125–3.0% beryllium carbonate (13–300 mg/kg body weight per day as beryllium using a food factor of 0.05 with an estimate that the beryllium carbonate used in the study contained 20% beryllium) for 3–4 weeks. 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 beryllium-exposed rats in another study, 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, characterized by the formation of granulomas. These granulomas result from an immune reaction, primarily based on cell-mediated immunity. A genetic component to chronic beryllium disease 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 British Cancer Registry 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 acute beryllium disease (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 experimental animals are available for the inhalation, intratracheal, oral and parenteral routes of 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 maximum tolerable dose (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. Limited data suggest that beryllium is not genotoxic in vivo.

3.2 Criteria for setting tolerable intakes or tolerable concentrations for beryllium

3.2.1 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 limit for a 10% incidence [response] of lesions in the small intestine in the study by Morgareidge, Cox & Gallo, 1976; assumed to be equal to a NOAEL) in dogs chronically exposed to beryllium sulfate tetrahydrate using the BMD approach and an uncertainty factor of 300. The uncertainty factor of 300 was composed of 10-fold factors each for intraspecies and interspecies variation and a 3-fold factor for database deficiencies (no studies available on developmental effects and no mechanistic/mode of action data). 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. As the principal study is of chronic duration and a BMD was used, there are no uncertainty factors for duration or NOAEL/LOAEL extrapolation.

3.2.2 Cancer

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. However, this relates to its carcinogenicity through the inhalation route and not the oral route.

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.

3.3 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 for the gastrointestinal effects; this uncertainty was diminished by using the BMD 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 analysed with inductively coupled plasma mass spectrometry. The minimum quantification limit ranges from 0.000 005 to 0.001 mg/l. Beryllium can also be analysed using inductively coupled plasma atomic emission spectrometry, for which the minimum quantification limit ranges from 0.002 to 0.2 mg/l. In some official methods for water analysis, beryllium is recommended as an internal standard at 0.000 005 mg/l, because it is not normally measured in routine drinking-water analysis (JWWA, 2001; USEPA, 2003). Analysis using an atomic absorption platform appears to give a slightly better limit of detection over USEPA Method 200.7 (USEPA, 2003). All of these methods provide a means of achieving a level of detection of less than 1 µg/l.

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/l in river water, removals of 85% and 80% were achieved using aluminium sulfate (2.5 mg/l as aluminium) and iron(III) chloride (10 mg/l as iron), 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 6–9, but the percentage removal was unaffected by the initial beryllium concentration in the range 5–50 µg/l. Removal of beryllium at 20 µg/l spiked into groundwater increased with lime dose in the range 75–450 mg/l; at the maximum dose, 99% removal occurred (Lytle, Summers & Sorg, 1992).

Physicochemical treatment of domestic wastewater containing trace metals showed that clarification and filtration using lime (415 mg/l; pH 11.5) as a coagulant removed 99.4% beryllium from an initial concentration of 100 µg/l. When aluminium sulfate (18 mg/l as aluminium; pH 6.4) was used, the removal was 98.1%; with iron(III) chloride (40 mg/l as iron; 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 iron(III) chloride system (Hannah, Jelus & Cohen, 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 beryllium at 1.6 µg/l and tap water spiked at around 2 µg/l, removals in the range 50–80% were obtained with zeolite doses of 0.1 and 0.2 g/l. Equilibrium was attained within about 1 h. It was concluded that zeolites could be used in normal filter beds for beryllium removal (Strnadova, Halasova & Holocek, 2000).

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

5. CONCLUSIONS

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 tolerable daily intake (TDI) of 2 µg/kg body weight, derived from the dog study by Morgareidge, Cox & Gallo (1976) (see section 3.2.2), to drinking-water and assuming a 60 kg adult drinking 2 litres of water per day. This allocation is probably conservative, as 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.

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