January 1990
(edited September 1990)

Barium

Guideline

The maximum acceptable concentration (MAC) for barium in drinking water is 1.0 mg/L (1000 μg/L).

Identity, Use and Sources in the Environment

Barium is present as a trace element in both igneous and sedimentary rocks. Although it is not found free in nature,[1] barium occurs in a number of compounds, most commonly barite ($BaSO_4$) and, to a lesser extent, witherite ($BaCO_3$).

Barium compounds have a wide variety of industrial applications. They are used in the plastics, rubber, electronics and textiles industries. Barium compounds are used in ceramic glazes and enamels, in glass making and paper making, as a lubricant additive and in pharmaceuticals and cosmetics.[2] Barite is used extensively in the oil and gas industry as a wetting agent for drilling mud.[2] Domestic consumption of barite in Canada in 1984 was 78 565 tonnes, 90% of which was used in oil and gas well drilling.[3] Witherite is used in the production of optical glasses, in brick making, in case-hardening of steel and as a rodenticide. Organometallic compounds containing barium have been used in diesel fuel to reduce black smoke emissions from diesel engines.[4]

The acetate, nitrate and halide salts of barium are soluble in water, but the carbonate, chromate, fluoride, oxalate, phosphate and sulphate salts are quite insoluble. The aqueous solubility of barium compounds increases as the pH decreases.[1] Organometallic barium compounds are ionic and are hydrolysed in water.[5] The concentration of barium ions in natural aquatic systems will be limited by naturally occurring anions, such as sulphates and carbonates, and by the possible adsorption of barium ions onto metal oxides and hydroxides.[6]

Exposure

Barium levels were determined in 122 municipalities in 10 provinces in samples of raw, treated and distributed drinking water serving approximately 36% of the Canadian population.[7] The provincial median concentrations of barium in distributed water ranged from not detectable (≤0.005 mg/L) in British Columbia, Newfoundland, Nova Scotia and Quebec to 0.084 mg/L in Alberta. The Canada-wide median concentration was 0.018 mg/L. The maximum level of barium in a sample of distributed water was 0.602 mg/L, measured at a site in Ontario. Barium levels in raw water were not significantly different from those in treated water.

Barium is generally present in air in particulate form, and its presence is attributed mainly to industrial emissions, specifically the combustion of coal and diesel oil and waste incineration. No data on concentrations of barium in ambient air in Canada have been found. In the United States, concentrations have been found to range from 0.0015 to 0.95 $\mu g/m^3$.[8]

Most foods contain less than 0.002 mg/g of barium.[9] The major dietary sources of barium in the United States are milk, potatoes and flour.[10] Some cereal products and nuts tend to contain high amounts of barium; for example, pecans contain 0.0067 mg/g, peanuts 0.0021 mg/g, bran flakes 0.0039 mg/g and Brazil nuts up to 4 mg/g.[11] Certain species of plants accumulate barium when grown in barium-rich soil.[11]

The International Commission on Radiological Protection (ICRP) reported that the long-term mean dietary barium intake for four individuals, including both food and fluids, was 0.9 mg/d, with a range of 0.44 to 1.8 mg/d.[12] In a survey by Schroeder *et al*. of the diets of five adults over 30 to 347 days, the mean daily intake of barium from food was 1.2 mg, with a range of 0.65 to 1.8 mg.[13] Owing to the lack of data on barium levels in Canadian foodstuffs, it is difficult to estimate daily intake of barium in the diet. However, on the basis of the data reported by the ICRP[12] and Schroeder *et al*.,[13] the mean intake of barium by Canadians from this source is estimated to be approximately 1 mg/d.

Assuming average daily water consumption to be 1.5 L and based on the median concentration of barium found in a national survey of Canadian drinking water (0.018 mg/L),[7] the barium intake from drinking water for most Canadians would be approximately 0.03 mg/d. For individuals consuming drinking water containing the highest barium concentration measured in the

national survey (0.6 mg/L),[7] the maximum daily intake of barium in drinking water would be 0.9 mg. At the levels of barium measured in U.S. air,[8] intake of barium through inhalation would be negligible compared with the amount ingested.

Based on the above considerations, the mean daily intake of barium from food, water and air in Canada is estimated to be slightly more than 1 mg/d. Of this, food represents the primary source of barium for the non-occupationally exposed Canadian population. However, in cases where barium levels in drinking water are high (0.6 mg/L), drinking water may contribute significantly to barium intake (approximately 50%).

Analytical Methods and Treatment Technology

Barium concentrations in water may be determined by atomic absorption spectroscopy (AAS), either by direct aspiration into an air–acetylene flame or by atomization in a furnace. The detection limit for the furnace technique is much lower than that for the direct aspiration procedure (2 µg/L vs. 100 µg/L).[1] Barium in water may also be determined by inductively coupled plasma atomic emission spectrometry; detection limits for this method of analysis are reported to be equivalent or superior to those for flame AAS for most elements.[14]

Based upon data collected in a Canadian national survey, relatively little, if any, soluble or insoluble barium is removed by conventional water treatment processes.[7] Processes effective in removing barium from drinking water include ion exchange (93 to 98%), lime softening (>90%) and the reverse osmosis membrane technique (>90%); efficiency of removal varies, depending upon levels in the raw water.[1]

Health Effects

Barium is not considered to be an essential element for human nutrition.[13]

The degree of absorption of barium from the lungs and gastrointestinal tract depends on the solubility of the compound, the animal species, the contents of the gastrointestinal tract, diet and age. Soluble barium salts are absorbed most readily. Recent data from studies in rats and man indicate that insoluble barium compounds may also be absorbed to a significant extent.[15,16] Gastrointestinal absorption of barium in rats was found to decrease with age (85% in rats 14 to 18 days old vs. <10% in rats 60 to 70 weeks old) and was greater in fasted rats than in fed rats.[17]

Barium is rapidly distributed in blood plasma, principally to the bone.[18] The whole body content of barium in man is approximately 22 mg, of which 93% is found in the bone and connective tissues, with smaller amounts being present in the fat, skin and lungs.[13] In rats administered drinking water containing 0 to 250 mg/L barium as barium chloride for four, eight or 13 weeks, the concentration of barium in the bone was proportional to dose but not to duration of exposure.[19] It has also been reported that barium crosses the placental barrier in humans, based on the determination of barium content of infant and stillborn foetal tissues.[13]

Miller *et al*.[20] reported that the ratio of barium to calcium in the teeth of 34 children exposed in one community to drinking water containing high concentrations of barium (10 mg/L) was five times higher than that for 35 children from another community exposed to lower levels (0.2 mg/L). The two communities were similar with respect to population, ethnic composition and socioeconomic status.

The principal route of excretion of barium in humans and animals is faecal;[21] 20% is excreted in the faeces and 7% is excreted in the urine within 24 hours.[18]

Soluble barium salts are highly acutely toxic. At high concentrations, barium causes strong vasoconstriction by its direct stimulation of arterial muscle, peristalsis due to the violent stimulation of smooth muscle, and convulsions and paralysis following stimulation of the central nervous system.[22] Depending on the dose and solubility of the barium salt, death may occur in a few hours or a few days. The acute lethal oral dose of barium chloride for humans has been estimated to be between 3 and 4 g; the acute toxic dose is 0.2 to 0.5 g.[23] Repeated exposures to barium chloride in table salt are believed to have caused recurrent outbreaks of “Pa-Ping” disease (a transient paralysis resembling familial periodic paralysis) in the Szechwan province of China.[24]

The prevalence of dental caries was reported to be significantly lower in 39 children from one community ingesting drinking water with a barium concentration of 8 to 10 ppm than in 36 children from another community ingesting drinking water with much lower barium concentrations (<0.3 ppm).[25] There was no statistically significant relationship between the prevalence of dental caries and age, sex, presence of detectable mottling, use of water softener or income. The concentrations of other trace elements (i.e., magnesium, calcium, strontium and fluoride) in the water supplies of the two communities were not markedly different; however, the study population was small, and dental examinations were not conducted blindly.

In several ecological epidemiological studies, associations between the barium content of drinking water and mortality from cardiovascular disease have been observed. For example, based on analysis of mortality rates in relation to variations in the amounts of numerous trace elements in drinking water in different geographical areas, Schroeder and Kramer[26] and Elwood *et al*.[27] reported significant negative correlations

between barium concentrations in drinking water and mortality from atherosclerotic heart disease and total cardiovascular disease, respectively. Conversely, Brenniman *et al*.[28] reported significantly higher sex- and age-adjusted death rates for "all cardiovascular diseases" and "heart disease" in an unspecified number of Illinois communities with high concentrations of barium in drinking water (≥2 to 10 mg/L) than in communities with low concentrations (0.0 to 0.2 mg/L) for the period 1971 to 1975. Although the communities were matched for similar demographic and socioeconomic status characteristics, population mobility varied between the communities with high and low barium levels (i.e., two of the communities with high barium concentrations in drinking water had a considerable increase in population size over the study period).

The lack of data on exposure of individuals in populations in the ecological studies described above and the resulting inability to adjust rigorously for confounding factors and population mobility limit their usefulness in assessing cause–effect relationships. Moreover, the results of the ecological study in Illinois conducted by Brenniman *et al*.[28] were not confirmed in a further cross-sectional study by Brenniman and Levy of cardiovascular disease prevalence in 1175 adult residents of West Dundee, Illinois (with a mean barium concentration in drinking water of 7.3 mg/L and a range of 2.0 to 10.0 mg/L) and in 1203 adult residents of McHenry (with a mean barium concentration in drinking water of 0.1 mg/L).[29] Blood pressures of all participants in the study were measured, and data on occurrence of cardiovascular, cerebrovascular and renal disease and possible confounding factors were determined by questionnaires administered by trained survey workers. (It was not specified whether or not the study was conducted blindly.) The socioeconomic status and demographic characteristics of the populations in the two towns were similar. There were no significant differences in the prevalence of hypertension, stroke and heart and kidney disease between the two populations, even when the use of water softeners, medication, duration of exposure, smoking and obesity were taken into account. The authors concluded that blood pressure in adults does not appear to be adversely affected even following prolonged ingestion of drinking water containing more than 7 mg/L barium.

In a recently completed but unpublished clinical study, 11 "healthy" men were exposed to barium (as barium chloride) in drinking water: 0 mg/L for two weeks, 5 mg/L for the next four weeks and 10 mg/L for the last four weeks.[30] Attempts were made to control several of the risk factors for cardiovascular disease, including diet, exercise, smoking and alcohol consumption throughout the study period (although study subjects were not continuously monitored in this regard). Based on twice daily monitoring of blood pressure, periodic monitoring of blood (for total cholesterol, triglycerides, HDL cholesterol, apolipoproteins A1, A2 and B and serum potassium, glucose, calcium and albumin) and urine (for vanillymandelic acid and total metanephrines), electrocardiographic monitoring at the end of each exposure period and liver function tests, there was no consistent indication of any adverse effects. There was, however, a trend towards increased serum calcium between 0 and 5 mg/L that persisted at 10 mg/L, which, for total calcium, normalized for differences in albumin level, was statistically significant. The authors suggested that this increase would not be expected to be clinically important. The lack of adverse effects observed in this study may be attributable to the small number of subjects included or the short period of exposure.

Tardiff *et al*.[19] exposed groups of 30 female and 30 male Charles River rats to 0, 10, 50 or 250 mg/L barium as barium chloride (equivalent to mean doses of 0, 1.7 and 2.1, 8.1 and 9.7, and 38.1 and 45.7 mg/kg bw per day for males and females, respectively) in drinking water for 13 weeks with interim kills (five of each sex) at four and eight weeks. No adverse histological or haematological effects were observed; there were also no effects on serum enzymes or ions, body weight gain or food consumption. Water consumption was slightly depressed in the highest dose group (38.1/45.7 mg/kg bw per day); there was also a slight decrease in adrenal weights in some dose groups, but it did not appear to be dose-related.

There were no effects on blood pressure in Sprague-Dawley rats exposed to 100 ppm barium in drinking water as barium chloride (equivalent to 15 mg/kg bw per day, based on the authors' calculations that 10 ppm was equal to 1.5 mg/kg bw per day) for up to 20 weeks.[31] In the same series of studies, there were no changes in blood pressure in hypertension-susceptible Dahl and uninephrectomized rats exposed for 16 weeks to up to 1000 ppm barium in distilled water or 0.9% saline. At the highest dose level (1000 ppm), however, there were ultrastructural changes in the glomeruli of the kidney discernible by electron microscopy. In addition, no significant electrocardiographic changes during L-norepinephrine challenge were observed in Sprague-Dawley rats ingesting drinking water containing 250 ppm barium for five months.[31]

In a limited study in which the survival, incidence of tumours upon gross examination at autopsy and serum levels of cholesterol, glucose and uric acids were determined in Long-Evans rats exposed for their lifetime to 5 ppm barium as barium acetate in drinking water, there was a slight enhancement in growth of females and a significant increase in proteinuria in males.[32] (It should be noted that mortality due to an epidemic of

pneumonia in this study was approximately 35% in males and 22% in females; there were 52 animals of each sex per group at the initiation of the study.) In a similar study in which 5 ppm barium as barium acetate was administered in drinking water to Charles River CD mice over their life span, there was a significant reduction in the survival of males but no effects on body weight gain, oedema, incidence of tumours or blanching of incisor teeth based on gross examination at autopsy.[33]

McCauley *et al*.[31] reported no histopathological effects upon examination of 34 tissues in male and female Sprague-Dawley rats exposed to 1, 10, 100 or 250 ppm barium as barium chloride in drinking water for up to 68 weeks.

Perry *et al*.[34] studied the effect of the ingestion of barium in drinking water on blood pressure in rats. Groups of female Long-Evans rats were exposed to 1, 10 or 100 ppm barium as barium chloride in drinking water for one, four or 16 months, equivalent to average daily barium doses of 0.051, 0.51 and 5.1 mg/kg bw per day.[35] There were no changes in mean systolic pressure in animals exposed to 1 ppm barium (0.051 mg/kg bw per day) for one to 16 months. At 10 ppm barium (0.51 mg/kg bw per day), there were mean increases in blood pressure of 4 to 7 mmHg by eight months, which persisted thereafter. In rats receiving 100 ppm barium (5.1 mg/kg bw per day), there were significant and persistent increases in mean systolic pressure (12 mmHg) after only one month, which gradually increased to a mean of 16 mmHg after 16 months of exposure. Rates of cardiac contraction, electrical excitability and high energy phosphate and phosphorylation potential were decreased. It has been suggested that the calcium content of the diet in this study may have been less than the minimum daily requirement; however, manifestations of calcium deficiency on growth patterns were not observed.

Increases in systolic pressure of 4 to 7 mmHg are deemed sufficiently small so as to not constitute an adverse effect; therefore, 5.1 mg/kg bw per day is considered to be the lowest-observed-adverse-effect level (LOAEL), and 0.51 mg/kg bw per day is considered to be the no-observed-adverse-effect level (NOAEL). This judgement is consistent with the opinion of the U.S. Environmental Protection Agency.[35] Perry *et al*.[34] estimated that a 0.1 to 1% increase in the clinical appearance of coronary heart disease in the U.S. population over a six-year period could result from an increase of 15 mmHg in mean systolic blood pressure. Wilkins and Calabrese[36] suggested that, whereas an increase in systolic blood pressure of 5 mmHg would have virtually no short-term clinical implications for persons 35 years old and younger, a shift of 5 mmHg may become a difference of nearly 10 mmHg by age 65. In the United States, an increase of 10 mmHg in systolic blood pressure at this age would increase the average risk of a heart attack by 14%.

There has been no evidence of the carcinogenicity of barium in extremely limited lifetime bioassays of rats and mice exposed to 5 mg/L in drinking water, based on gross examination only of tumours at autopsy.[32,33]

Barium chloride did not increase the frequency of mutation in repair-deficient strains of *Bacillus subtilis*.[37] Results were also negative for the induction of errors in viral DNA transcription *in vitro*.[38]

It has been reported that the oestrous cycle was shortened and that the morphological structure of the ovaries was disturbed in female rats exposed to 13.4 ± 0.7 mg/m^3 and 3.1 ± 0.16 mg/m^3 barium carbonate dust for four months.[39] Mortality increased and body weight gain decreased in the offspring of the dams in the high dose group; the lability of the peripheral nervous system decreased, and blood disorders (erythropenia, leukocytosis, eosinophilia, neutrophilia) related to the irritant effect of barium on the bone marrow in two-month-old offspring were also reported.[39] In a similar inhalation study in male rats exposed to 22.6 ± 0.6 mg/m^3 barium carbonate dust,[39] deleterious effects on spermatogenesis were reported. However, data presented in the published account were insufficient to permit evaluation of the protocols of these studies.

Classification and Assessment

Because there are few data available regarding the carcinogenicity of barium, it has been classified in Group VA (inadequate data for evaluation). For compounds classified in Group VA, the maximum acceptable concentration (MAC) is derived on the basis of division of the NOAEL or LOAEL in an animal species by an uncertainty factor.

In studies in which Long-Evans rats ingested drinking water containing 100 mg/L (5.1 mg/kg bw per day) for up to 16 months, there were significant increases in mean systolic blood pressure (12 mmHg after one month, 16 mmHg after 16 months), depressed rates of cardiac contraction, depressed electrical excitability and decreased high energy phosphate and phosphorylation potential.[34,40] Increases of 4 to 7 mmHg in mean systolic blood pressure were observed in rats exposed to a barium concentration of 10 mg/L (0.51 mg/kg bw per day) in drinking water after eight months. In a study with Sprague-Dawley rats, however, there were no effects on blood pressure following exposure to barium at 100 ppm in drinking water (approximately 15 mg/kg bw per day) for 20 weeks.[31]

It has been suggested by some authors that small increases in mean systolic blood pressure in the human population similar to those observed in rats by Perry *et al*.[34,40] (5 to 15 mmHg) could result in significant increases in clinical cases of coronary heart disease. However, in the most sensitive epidemiological study conducted to date, there were no significant differences in blood pressure or the prevalence of cardiovascular disease between a population drinking water containing 7.3 mg/L barium and one ingesting water containing 0.1 mg/L barium.[29]

On the basis of the results of the epidemiological study by Brenniman and Levy,[29] in which adverse effects on blood pressure and increases in the prevalence of cardiovascular disease were not observed in a population ingesting water containing a mean concentration of 7.3 mg/L barium, the MAC is derived as follows:

$$\text{MAC} = \frac{7.3 \text{ mg/L}}{10} = 0.73 \text{ mg/L}$$

where:

- 7.3 mg/L is the NOAEL in the most sensitive epidemiological study conducted to date[29]
- 10 is the uncertainty factor (to account for intraspecies variation).

This value is not substantially different from that derived on the basis of the results of toxicological studies in animals, based on the NOAEL of 0.51 mg/kg bw per day for effects on blood pressure in rats[34,40] and an uncertainty factor of 10 (×10 for intraspecies variation; ×1 for interspecies variation, as the results of a well-conducted epidemiological study indicate that humans are less sensitive than rats to barium in drinking water).

Because the MAC derived above does not differ greatly from the MAC of 1.0 mg/L included in the 1978 Guidelines, a MAC for barium of 1.0 mg/L has been retained.

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