GUIDELINES FOR DRINKING-WATER QUALITY: FOURTH EDITION INCORPORATING THE FIRST AND SECOND ADDENDA

Occurrence	Has been reported in drinking-water with a variety of source water characteristics after ozonation at concentrations ranging from less than 2 to 293 µg/l, depending on bromide ion concentration, ozone dosage, pH, alkalinity and dissolved organic carbon; can also be formed in the electrolytic generation of chlorine and hypochlorite from brine with a high level of bromide contamination
Basis of guideline value derivation	Upper-bound estimate of cancer potency for bromate is 0.19 per mg/ kg body weight per day, based on low-dose linear extrapolation (a one-stage Weibull time-to-tumour model was applied to the incidence of mesotheliomas, renal tubule tumours and thyroid follicular tumours in male rats given potassium bromate in drinking-water, using the 12-, 26-, 52- and 77-week interim kill data). A health-based value of 2 µg/l is associated with the upper-bound excess cancer risk of 10 ⁻⁵ . A similar con- clusion may be reached through several other methods of extrapolation, leading to values in the range 2–6 µg/l.
Limit of detection	0.2 μ g/l by ion chromatography with UV/visible absorbance detection; 0.3 μ g/l by ion chromatography with detection by ICP-MS; 1.5 μ g/l by ion chromatography with suppressed conductivity detection
Treatment performance	Bromate is difficult to remove once formed. By appropriate control of disinfection conditions, it is possible to achieve bromate concentrations below 0.01 mg/l.
Assessment date	2003
Principal reference	WHO (2003) Bromate in drinking-water

IARC has concluded that although there is inadequate evidence of carcinogenicity in humans, there is sufficient evidence for the carcinogenicity of bromate from high-dose studies in experimental animals; IARC has classified bromate in Group 2B (possibly carcinogenic to humans). Bromate is mutagenic both in vitro and in vivo. At this time, there is not sufficient evidence to conclude as to the mode of carcinogenic action for bromate. Observation of tumours at a relatively early time and the positive response of bromate in a variety of genotoxicity assays suggest that the predominant mode of action at low doses is due to oxidative deoxyribonucleic acid (DNA) damage. Although there is evidence to suggest that the DNA reactivity in kidney tumours may have a non-linear dose-response relationship, there is no evidence to suggest that this same dose-response relationship operates in the development of mesotheliomas or thyroid tumours. Oxidative stress may play a role in the formation of kidney tumours, but the evidence is insufficient to establish lipid peroxidation and free radical production as key events responsible for the induction of kidney tumours. However, emerging evidence points to rapid decomposition of bromate in the gastrointestinal tract, blood and liver, which supports a non-linear dose-response relationship at low doses.

Bromide

Bromide is commonly found in nature along with sodium chloride, owing to their similar physical and chemical properties, but in smaller quantities. Bromide concentrations in seawater range from 65 mg/l to well over 80 mg/l, in fresh water from trace amounts to about 0.5 mg/l and in desalinated waters up to 1 mg/l.

12. CHEMICAL FACT SHEETS

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	2009
Principal reference	WHO (2009) Bromide in drinking-water

Inorganic bromide was evaluated in 1966 by JMPR, which recommended an ADI of 0–1 mg/kg body weight, based on a minimum pharmacologically effective dosage in humans of about 900 mg of potassium bromide, equivalent to 600 mg of bromide ion. The JMPR ADI was reaffirmed with new data in 1988.

The results of human studies suggest a conservative no-observed-effect level (NOEL) (for marginal effect within normal limits of electroencephalograms in females) of 4 mg/kg body weight per day, giving an ADI of 0–0.4 mg/kg body weight, including a safety factor of 10 for population diversity.

The upper limit of the ADI of 0-0.4 mg/kg body weight yields an acceptable total daily intake of 24 mg/person for a 60 kg person. Assuming a relative source contribution of 50%, the drinking-water value for a 60 kg adult consuming 2 litres/day would be up to 6 mg/l; for a 10 kg child consuming 1 litre/day, the value would be up to 2 mg/l. However, the dietary bromide contribution for a 10 kg child would probably be less than that for an adult. These are reasonably conservative values, and they are unlikely to be encountered in drinking-water supplies.

Bromide can be involved in the reaction between chlorine and naturally occurring organic matter in drinking-water, forming brominated and mixed chloro-bromo by-products, such as trihalomethanes (THMs) and halogenated acetic acids (HAAs), or it can react with ozone to form bromate. The levels of bromide that can result in the formation of these substances are well below the health-based values suggested above. This guidance applies specifically to inorganic bromide ion and not to bromate or organohalogen compounds, for which individual health-based guideline values have been developed.

Brominated acetic acids

Brominated acetic acids are formed during disinfection of water that contains bromide ions and organic matter. Bromide ions occur naturally in surface water and groundwater and exhibit seasonal fluctuations in levels. Bromide ion levels can increase as a result of either saltwater intrusion resulting from drought conditions or pollution. Brominated acetates are generally present in surface water and groundwater distribution systems at mean concentrations below 5 µg/l.

Reason for not establishing guideline values	Available data inadequate to permit derivation of health-based guideline values
Assessment date	2003
Principal references	IPCS (2000) Disinfectants and disinfectant by-products WHO (2004) Brominated acetic acids in drinking-water