

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) <i>Bromide in drinking-water</i>

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) <i>Disinfectants and disinfectant by-products</i> WHO (2004) <i>Brominated acetic acids in drinking-water</i>

The database for dibromoacetic acid is considered inadequate for the derivation of a guideline value. There are no systemic toxicity studies of subchronic duration or longer. The database also lacks suitable toxicokinetic studies, a carcinogenicity study, a developmental study in a second species and a multigeneration reproductive toxicity study. Available mutagenicity data suggest that dibromoacetate is genotoxic.

Data are also limited on the oral toxicity of monobromoacetic acid and bromochloroacetic acid. Limited mutagenicity and genotoxicity data give mixed results for monobromoacetic acid and generally positive results for bromochloroacetic acid. Data gaps include subchronic or chronic toxicity studies, multigeneration reproductive toxicity studies, standard developmental toxicity studies and carcinogenicity studies. The available data are considered inadequate to establish guideline values for these chemicals.

Cadmium

Cadmium metal is used in the steel industry and in plastics. Cadmium compounds are widely used in batteries. Cadmium is released to the environment in wastewater, and diffuse pollution is caused by contamination from fertilizers and local air pollution. Contamination in drinking-water may also be caused by impurities in the zinc of galvanized pipes and solders and some metal fittings. Food is the main source of daily exposure to cadmium. The daily oral intake is 10–35 µg. Smoking is a significant additional source of cadmium exposure.

Guideline value	0.003 mg/l (3 µg/l)
Occurrence	Levels in drinking-water usually less than 1 µg/l
PTMI	25 µg/kg body weight, based on the relationship between β_2 -microglobulin excretion in urine and cadmium excretion in urine for individuals who are 50 years of age and older
Limit of detection	0.01 µg/l by ICP-MS; 2 µg/l by flame AAS
Treatment performance	0.002 mg/l should be achievable using coagulation or precipitation softening
Guideline value derivation	
• allocation to water	10% of provisional tolerable monthly intake (PTMI) because of high intake from food
• weight	60 kg adult
• consumption	2 litres/day
Additional comments	Although new information indicates that a proportion of the general population may be at increased risk for tubular dysfunction when exposed at the current PTMI, the risk estimates that can be made at present are imprecise.
	It is recognized that the margin between the PTMI and the actual monthly intake of cadmium by the general population is small and that this margin may be even smaller in smokers.
Assessment date	2011
Principal references	FAO/WHO (2011) <i>Evaluation of certain food additives and contaminants</i> WHO (2003) <i>Cadmium in drinking-water</i>