Guideline value derivation	
• allocation to water	40% of TDI (because intake from other sources is low)
• body weight	60 kg adult
• consumption	2 litres/day
Additional comments	Because it will be difficult to achieve the guideline value of 2.4 mg/l in some desalinated supplies and in areas with high natural boron levels, local regulatory and health authorities should consider a value in excess of 2.4 mg/l by assessing exposure from other sources.
Assessment date	2009
Principal reference	WHO (2009) *Boron in drinking-water*

Short- and long-term oral exposures to boric acid or borax in laboratory animals have demonstrated that the male reproductive tract is a consistent target of toxicity. Testicular lesions have been observed in rats, mice and dogs given boric acid or borax in food or drinking-water. Developmental toxicity has been demonstrated experimentally in rats, mice and rabbits. Negative results in a large number of mutagenicity assays indicate that boric acid and borax are not genotoxic. In long-term studies in mice and rats, boric acid and borax caused no increase in tumour incidence.

Bromate

Sodium and potassium bromate are powerful oxidizers used mainly in permanent wave neutralizing solutions and the dyeing of textiles using sulfur dyes. Potassium bromate has also been used as an oxidizer to mature flour during milling, in treating barley in beer making and in fish paste products, although JECFA has concluded that the use of potassium bromate in food processing is not appropriate. Bromate is not normally found in water, but can occur as a result of pollution from industrial sources, sometimes as a consequence of its presence in contaminated soil. However, the main source in drinking-water is its formation during ozonation when the bromide ion is present in water. Bromate may also be formed in hypochlorite solutions produced by electrolysis of bromide-containing salt.

Provisional guideline value	0.01 mg/l (10 µg/l)
	The guideline value is provisional because of limitations in available analytical and treatment methods.
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 conclusion may be reached through several other methods of extrapolation, leading to values in the range 2–6 µg/l.

Limit of detection	0.2 µg/l by ion chromatography with UV/visible absorbance detection; 0.3 µg/l by ion chromatography with detection by ICP-MS; 1.5 µg/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.

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