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) *Evaluation of certain food additives and contaminants* WHO (2003) *Cadmium in drinking-water*

Absorption of cadmium compounds is dependent on the solubility of the compounds. Cadmium accumulates primarily in the kidneys and has a long biological half-life in humans of 10–35 years. There is evidence that cadmium is carcinogenic by the inhalation route, and IARC has classified cadmium and cadmium compounds in Group 2A (probably carcinogenic to humans). However, there is no evidence of carcinogenicity by the oral route and no clear evidence for the genotoxicity of cadmium. The kidney is the main target organ for cadmium toxicity.

In its recent evaluation of cadmium, JECFA found that data relating excretion of the biomarker β_2-microglobulin in urine to cadmium excretion in urine for individuals who are 50 years of age and older provided the most reliable basis on which to determine a critical concentration of cadmium in the urine. Urinary excretion of less than 5.24 µg of cadmium per gram creatinine was not associated with an increased excretion of β_2-microglobulin, and the dietary exposure that would result in a urinary cadmium concentration at the breakpoint of 5.24 µg/g creatinine was estimated to be 0.8 µg/kg body weight per day or about 25 µg/kg body weight per month. Because of cadmium's exceptionally long half-life, the previous PTWI of 7 µg/kg body weight was withdrawn, and a PTMI of 25 µg/kg body weight was established.

Carbaryl

Carbaryl (CAS No. 63-25-2) is a broad-spectrum carbamate insecticide that is used to control insect pests in crops, trees and ornamental plants. It also has some uses in public health and veterinary practice. Carbaryl has not been reported in drinking-water; however, it could occur following overspraying or spillage into surface water. Exposure through drinking-water is therefore considered to be low unless in exceptional circumstances. The major route of carbaryl intake for the general population is food, but residues are considered to be relatively low.

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	2006
Principal references	FAO/WHO (2002) *Pesticide residues in food—2001 evaluations* WHO (2008) *Carbaryl in drinking-water*

Carbaryl acts through inhibition of brain cholinesterase, and this is also its primary mode of toxicity. However, carbaryl is also considered to be a non-genotoxic carcinogen in mice, in which it causes vascular tumours in males. On this basis, JMPR established an ADI of 0–0.008 mg/kg body weight. This was based on a lowest-observed-adverse-effect level (LOAEL) of 15 mg/kg body weight per day and application of a safety factor of 2000 (10 for interspecies variation, 10 for intraspecies variation and 20 to reflect the occurrence of the rare and malignant tumour for which a no-effect level could not be identified).

A health-based value of 50 µg/l (rounded value) can be determined from the JMPR ADI of 0–0.008 mg/kg body weight, assuming a 60 kg adult drinking 2 litres of water per day and allowing 20% of the upper limit of the ADI from drinking-