Provisional guideline value	0.0004 mg/l (0.4 µg/l)
	The guideline value is provisional owing to serious limitations of the critical studies.
Occurrence	Detected in groundwater following its use as a soil fumigant at concentrations as high as 100 µg/l
Basis of guideline value derivation	Lower end of the range (and thus more conservative estimate) of lifetime low-dose cancer risks calculated by linearized multistage modelling of the incidences of haemangiosarcomas and tumours in the stomach, liver, lung and adrenal cortex (adjusted for the observed high early mortality, where appropriate, and corrected for the expected rate of increase in tumour formation in rodents in a standard bioassay of 104 weeks) of rats and mice exposed by gavage
Limit of detection	0.01 µg/l by microextraction GC-MS; 0.03 µg/l by purge-and-trap GC with halogen-specific detector; 0.8 µg/l by purge-and-trap capillary column GC with photoionization and electrolytic conductivity detectors in series
Treatment performance	0.1 µg/l should be achievable using GAC
Assessment date	2003
Principal references	IPCS (1995) *Report of the 1994 meeting of the Core Assessment Group* IPCS (1996) *1,2-Dibromoethane* WHO (2003) *1,2-Dibromoethane in drinking-water*

1,2-Dibromoethane has induced an increased incidence of tumours at several sites in all carcinogenicity bioassays identified in which rats or mice were exposed to the compound by gavage, ingestion in drinking-water, dermal application and inhalation. However, many of these studies were characterized by high early mortality, limited histopathological examination, small group sizes or use of only one exposure level. The substance acted as an initiator of liver foci in an initiation/promotion assay but did not initiate skin tumour development. 1,2-Dibromoethane was consistently genotoxic in in vitro assays, although results of in vivo assays were mixed. Biotransformation to active metabolites, which have been demonstrated to bind to DNA, is probably involved in the induction of tumours. Available data do not support the existence of a non-genotoxic mechanism of tumour induction. The available data thus indicate that 1,2-dibromoethane is a genotoxic carcinogen in rodents. Data on the potential carcinogenicity in humans are inadequate; however, it is likely that 1,2-dibromoethane is metabolized similarly in rodent species and in humans (although there may be varying potential for the production of active metabolites in humans, owing to genetic polymorphism). IARC classified 1,2-dibromoethane in Group 2A (probably carcinogenic to humans).

Dichloroacetic acid

Chlorinated acetic acids, including dichloroacetic acid (DCA), are formed from organic material during water chlorination. DCA has been used as a therapeutic agent to treat lactic acidosis, diabetes and familial hyperlipidaemia in humans.

Provisional guideline value	0.05 mg/l (50 µg/l)
	The guideline value is designated as provisional on the basis of technical achievability.
Occurrence	Found in groundwater and surface water distribution systems at concentrations up to about 100 µg/l, with mean concentrations below 20 µg/l
Basis of guideline value derivation	Linear multistage model applied to combined data for carcinomas and adenomas in male mice exposed to doses up to 429 mg/kg body weight per day for up to 2 years
Limit of detection	< 0.1–0.4 µg/l by GC with ECD; practical quantification limit 1 µg/l
Treatment performance	Concentrations may be reduced by installing or optimizing coagulation to remove precursors or by controlling the pH during chlorination.
Additional comments	The concentration associated with a 10^{-5} upper-bound excess lifetime cancer risk is 40 µg/l. In some circumstances, however, it may not be possible to adequately disinfect potable water and maintain DCA levels below 40 µg/l, so the provisional guideline value of 50 µg/l is retained.
Assessment date	2004
Principal reference	WHO (2005) *Dichloroacetic acid in drinking-water*

IARC reclassified DCA as Group 2B (possibly carcinogenic to humans) in 2002, based on the absence of data on human carcinogenicity and sufficient evidence of its carcinogenicity in experimental animals. This classification was based primarily on findings of liver tumours in rats and mice. Genotoxicity data are considered to be inconclusive, particularly at lower doses. Glycogen deposition, peroxisome proliferation, changes in signal transduction pathways and DNA hypomethylation have all been observed following DCA exposure and have been hypothesized to be involved in its carcinogenicity. However, the available data are not sufficient to establish a cancer mode of action with reasonable certainty, especially at the very low exposure levels expected to apply to humans ingesting chlorinated drinking-water. Recent data suggest that there may be more than one mechanism leading to tumours, as altered hepatic foci from treated mice were found to have three different types of cellular characteristics.

Dichlorobenzenes (1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,4-dichlorobenzene)

The dichlorobenzenes (DCBs) are widely used in industry and in domestic products such as odour-masking agents, chemical dyestuffs and pesticides. Sources of human exposure are predominantly air and food.

Guideline values	*1,2-Dichlorobenzene:* 1 mg/l (1000 µg/l)
	1,4-Dichlorobenzene: 0.3 mg/l (300 µg/l)
Occurrence	Have been found in raw water sources at levels as high as 10 µg/l and in drinking-water at concentrations up to 3 µg/l; much higher concentrations (up to 7 mg/l) present in contaminated groundwater