occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. DBCP was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines calculated a guideline value of 0.001 mg/litre for DBCP in drinking-water, corresponding to an upper-bound excess lifetime cancer risk of 10^{-5} and sufficiently protective for the reproductive toxicity of the pesticide. It was noted that for a contaminated water supply, extensive treatment would be required to reduce the level of DBCP to the guideline value.

Assessment date

The risk assessment was originally conducted in 1993. The Final Task Force Meeting in 2003 agreed that this risk assessment be brought forward to this edition of the *Guidelines for Drinking-water Quality*.

Principal reference

WHO (2003) *1,2-Dibromo-3-chloropropane in drinking-water. Background document for preparation of WHO Guidelines for drinking-water quality*. Geneva, World Health Organization (WHO/SDE/WSH/03.04/34).

12.40 1,2-Dibromoethane (ethylene dibromide)

1,2-Dibromoethane (CAS No. 106-93-4) is used as a lead scavenger in tetra-alkyl lead petrol and antiknock preparations and as a fumigant for soils, grains and fruits. However, with the phasing out of leaded petrol and of the use of 1,2-dibromoethane in agricultural applications in many countries, use of this substance has declined significantly. In addition to its continued use as a petrol additive in some countries, 1,2-dibromoethane is currently used principally as a solvent and as an intermediate in the chemical industry.

Provisional guideline value	0.0004 mg/litre (0.4 µg/litre) The guideline value is provisional due 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/litre
Basis of guideline 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/or mice exposed to 1,2-dibromoethane by gavage

Limit of detection	0.01 µg/litre by microextraction GC/MS; 0.03 µg/litre by purge and trap GC with halogen-specific detector; 0.8 µg/litre by purge-and-trap capillary column GC with photoionization and electrolytic conductivity detectors in series
Treatment achievability	0.1 µg/litre should be achievable using GAC

Toxicological review

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 (the agent is probably carcinogenic to humans).

History of guideline development

The 1958 and 1963 WHO *International Standards for Drinking-water* did not refer to 1,2-dibromoethane, but the 1971 International Standards suggested that pesticide residues that may occur in community water supplies make only a minimal contribution to the total daily intake of pesticides for the population served. 1,2-Dibromoethane was not evaluated in the first edition of the *Guidelines for Drinking-water Quality*, published in 1984, but the 1993 Guidelines noted that 1,2-dibromoethane appears to be a genotoxic carcinogen. However, as the studies to date were inadequate for mathematical risk extrapolation, a guideline value for 1,2-dibromoethane was not derived. The Guidelines recommended that 1,2-dibromoethane be re-evaluated as soon as new data became available. In the addendum to these Guidelines, published in 1998, the guideline value that corresponds to an upper-bound excess lifetime cancer risk for various tumour types of 10^{-5} was calculated to be in the range 0.0004–0.015 mg/litre. This guideline value was considered to be provisional because of the serious limitations of the critical studies.

Assessment date

The risk assessment was conducted in 2003.

Principal references

IPCS (1995) *Report of the 1994 meeting of the Core Assessment Group*. Geneva, World Health Organization, International Programme on Chemical Safety, Joint Meeting on Pesticides (WHO/PCS/95.7).

IPCS (1996) *1,2-Dibromoethane*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 177).

WHO (2003) *1,2-Dibromoethane in drinking-water. Background document for preparation of WHO Guidelines for drinking-water quality*. Geneva, World Health Organization (WHO/SDE/WSH/03.04/66).

12.41 Dichloroacetic acid

Chlorinated acetic acids are formed from organic material during water chlorination.

Provisional guideline value	0.05 mg/litre The guideline value is designated as provisional because the data are insufficient to ensure that the value is technically achievable. Difficulties in meeting a guideline value must never be a reason to compromise adequate disinfection.
Occurrence	Found in finished chlorinated water at concentrations up to about 100 µg/litre, but in most cases at concentrations less than 50 µg/litre
TDI	7.6 µg/kg of body weight, based on a study in which no effects were seen in the livers of mice exposed to dichloroacetate at 7.6 mg/kg of body weight per day for 75 weeks and incorporating an uncertainty factor of 1000 (100 for intra- and interspecies variation and 10 for possible carcinogenicity)
Limit of detection	1 µg/litre by GC with ECD; 2 µg/litre by GC/MS
Treatment achievability	Concentrations may be reduced by installing or optimizing coagulation to remove precursors and/or by controlling the pH during chlorination.
Guideline derivation	
• allocation to water	20% of TDI
• weight	60-kg adult
• consumption	2 litres/day

Toxicological review

In several bioassays, dichloroacetate has been shown to induce hepatic tumours in mice. However, the evidence for the carcinogenicity of dichloroacetate is insufficient to derive a guideline value based on carcinogenicity. No adequate data on genotoxicity are available.