carbon tetrachloride, manufacture and use have dropped and will continue to drop. Carbon tetrachloride is released mostly into the atmosphere but also into industrial wastewater. Although it readily migrates from surface water to the atmosphere, levels in anaerobic groundwater may remain elevated for months or even years. Although available data on concentrations in food are limited, the intake from air is expected to be much greater than that from food or drinking-water.

Guideline value	0.004 mg/l (4 µg/l)
Occurrence	Concentrations in drinking-water generally less than 5 µg/l
TDI	1.4 µg/kg body weight, based on a NOAEL of 1 mg/kg body weight per day for hepatotoxic effects in a 12-week oral gavage study in rats, adjusting for daily dosing and applying an uncertainty factor of 500 (100 for interspecies and intraspecies variation, 10 for the duration of the study and a modifying factor of 0.5 because it was a bolus study)
Limit of detection	0.1–0.3 µg/l by GC-ECD or GC-MS
Treatment performance	0.001 mg/l should be achievable using air stripping
Guideline value derivation	
• allocation to water	10% of TDI
• weight	60 kg adult
• consumption	2 litres/day
Additional comments	The guideline value is lower than the range of values associated with upper-bound lifetime excess cancer risks of 10^{-4}, 10^{-5} and 10^{-6} calculated by linear extrapolation.
Assessment date	2003
Principal references	IPCS (1999) *Carbon tetrachloride* WHO (2004) *Carbon tetrachloride in drinking-water*

The primary targets for carbon tetrachloride toxicity are liver and kidney. In experiments with mice and rats, carbon tetrachloride proved to be capable of inducing hepatomas and hepatocellular carcinomas. The doses inducing hepatic tumours were higher than those inducing cell toxicity. It is likely that the carcinogenicity of carbon tetrachloride is secondary to its hepatotoxic effects. On the basis of available data, carbon tetrachloride can be considered to be a non-genotoxic compound. Carbon tetrachloride is classified by IARC as being possibly carcinogenic to humans (Group 2B): there is sufficient evidence that carbon tetrachloride is carcinogenic in laboratory animals, but inadequate evidence in humans.

Chloral hydrate

Chloral hydrate, or trichloroacetaldehyde, can be formed as a by-product of the chlorination of water containing organic precursor material, such as fulvic and humic acids. It has been found in drinking-water at concentrations of up to 100 µg/l, but concentrations are usually below 10 µg/l. Concentrations are generally higher in surface water than in groundwater, and concentrations appear to increase during distribution.

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	2004
Principal references	IPCS (2000) *Chloral hydrate* IPCS (2000) *Disinfectants and disinfectant by-products* WHO (2005) *Chloral hydrate in drinking-water*

Chloral hydrate is used as an intermediate in the production of insecticides, herbicides and hypnotic drugs. It has also been widely used as a sedative or hypnotic drug in humans at oral doses of up to about 750–1000 mg/day. Although intake from clinical use is considerably higher than intake from drinking-water, clinical exposure is of shorter-term duration.

No epidemiological or carcinogenic studies were found in humans that associated exposure to chloral hydrate with cancer, despite the fact that chloral hydrate has been used for many decades (and still is used) as a sedative and hypnotic drug in adults and children (specifically for dental procedures). IARC classified chloral hydrate as not classifiable as to its carcinogenicity to humans (Group 3), based on inadequate evidence in humans and limited evidence in experimental animals. There is equivocal evidence for the genotoxicity of chloral hydrate.

A health-based value of 0.1 mg/l (rounded figure) can be calculated on the basis of a TDI of 0.0045 mg/kg body weight derived based on an increased incidence of liver histopathology observed in mice in a 2-year drinking-water study, allocating 80% of the TDI to drinking-water (because most exposure to chloral hydrate is from drinking-water) and assuming a 60 kg adult consuming 2 litres of water per day. However, because chloral hydrate usually occurs in drinking-water at concentrations well below those of health concern, it is not considered necessary to derive a guideline value.

Chloral hydrate levels in drinking-water can be controlled by changes to disinfection practice (e.g. enhanced coagulation and softening to remove organic precursor compounds, moving the point of disinfection to reduce the reaction between chlorine and precursor compounds and using chloramines for residual disinfection instead of chlorine) and by GAC treatment.

Chloramines (monochloramine, dichloramine, trichloramine)

Monochloramine, dichloramines and trichloramines are considered by-products of drinking-water chlorination, being formed when chlorine and ammonia are added to water. Monochloramine may also be added to maintain residual disinfection activity in potable water distribution systems. Because higher chloramines are formed only occasionally and cause taste and odour problems at concentrations lower than those at which monochloramine causes taste and odour problems, only monochloramine has been considered for development of a health-based guideline value. Chloramine is rapidly decomposed in the stomach by gastric juice. The use of chloramines for disinfection instead of chlorine reduces the formation of THMs in drinking-water supplies. However, formation of other by-products, such as haloketones, chloropicrin, cyanogen chloride, HAAs, haloacetonitriles, aldehydes and chlorophenols, has been