Limit of detection	0.1 µg/l by HPLC with UV detection
Treatment performance	0.1 µg/l should be achievable using GAC
Guideline value derivation	
• allocation to water	10% of TDI
• weight	60 kg adult
• consumption	2 litres/day
Assessment date	1998
Principal reference	WHO (2003) *Terbuthylazine in drinking-water*

There is no evidence that TBA is carcinogenic or mutagenic. In long-term dietary studies in rats, effects on red blood cell parameters in females, an increased incidence of non-neoplastic lesions in the liver, lung, thyroid and testis and a slight decrease in body weight gain were observed.

Tetrachloroethene

Tetrachloroethene (PCE) has been used primarily as a solvent in dry-cleaning industries, and to a lesser extent as a degreasing solvent. Since the 1980s, as a result of regulations in North America, Europe and elsewhere, its use has substantially decreased. PCE is widespread in the environment and is found in trace amounts in water, aquatic organisms, air, foodstuffs and human tissue. The most relevant routes of exposure are considered to be inhalation of contaminated air and ingestion of contaminated drinking-water, particularly from groundwater sources. Poor handling and improper disposal of PCE in landfills have been the main causes of water contamination. Higher levels of PCE are expected in groundwater than in surface water because of the lack of volatilization that occursfrom groundwater. However, drinking-water is not a major source of exposure, unless in the vicinity of a contaminated site.

Guideline value	0.1 mg/l (100 µg/l)
Occurrence	Concentrations in drinking-water are generally below 10 µg/l, although much higher concentrations have been detected in well water (23 mg/l) and in contaminated groundwater (1.5 mg/l)
TDI	16 µg/kg bw, based on a BMDL10 of 4.7 mg/kg bw per day for neurological effects (decreased colour vision) observed in an occupational study and applying an uncertainty factor of 300 (10 each for inter- and intra-species variability and 3 for extrapolation from an occupational study with intermittent exposure)
Limit of detection	0.002–0.008 µg/l by GC with ECD after liquid–liquid extraction; 0.02–0.05 µg/l by purge-and-trap capillary GC with PD and ECD in series; 0.036 µg/l by volatile organic compound analysis with GC-MS; and 0.05–0.14 µg/l by purge-and-trap capillary GC-MS

Prevention and treatment	Source control, by improving handling and disposal practices, should be the priority action since PCE can persist in waters where volatilization cannot occur. Treatment of surface water sources is not needed because PCE volatilizes to the atmosphere. GAC, packed tower aeration and diffused aeration are effective central treatment technologies, although diffused aeration achieves lower removal efficiencies than packed tower aeration systems. Advanced oxidation processes may also be effective, but effectiveness depends on the physical and chemical properties of the water.
Guideline value derivation	
• allocation to water	20% of TDI
• weight	60 kg adult
• consumption	2 litres/day
Additional comments	The guideline value is considered protective against both cancer and noncancer effects.
	In developing national standards, authorities may take into consideration the additional exposures through the dermal and inhalation routes from showering and bathing, especially in countries with low rates of ventilation in houses.Authorities may also consider overall exposure in developing national standards, noting the continued decline in human exposure to PCE by all probable exposure routes.
	Requirements for monitoring PCE in drinking-water regulations and standards should be limited to groundwater sources, where a possibility of PCE contamination is indicated. Monitoring is not needed for surface water sources because PCE volatilizes to the atmosphere.
Assessment date	2020
Principal reference	WHO (2020) *Tetrachloroethene in drinking-water*

IARC has classified PCE in Group 2A (probably carcinogenic to humans). PCE has been reported to produce liver tumours in male and female mice following inhalation, and there is some evidence of mononuclear cell leukaemia in male and female rats and kidney tumours in male rats. However, absorption, metabolic pathways, excretion and pattern of effects are similar for inhalation and oral exposure. The weight of evidence for PCE suggests that a nonlinear, nonmutagenic mode of action is predominant for liver tumours, and this effect is considered the most relevant end-point for cancer risk assessment of PCE exposure. Additionally, noncancer effects resulting from inhalation exposure, including evidence of neurotoxicity, were observed in human occupational studies and in laboratory animal studies. The nervous system is the most sensitive organ for noncancer effects. To protect against both cancer and noncancer effects, comparative points of departure were identified using benchmark dose modelling and physiologically based pharmacokinetic modelling (to account for the first-pass effects and inhalation-to-ingestion extrapolation). The most sensitive human-relevant effects were determined to be reductions in colour vision, and changes in cognitive function and reaction time in exposed workers; neurological effects were therefore the basis for the TDI derivation.