Epichlorohydrin is rapidly and extensively absorbed following oral, inhalation or dermal exposure. It binds easily to cellular components. Major toxic effects are local irritation and damage to the central nervous system. It induces squamous cell carcinomas in the nasal cavity by inhalation and forestomach tumours by the oral route. It has been shown to be genotoxic in vitro and in vivo. IARC has placed epichlorohydrin in Group 2A (probably carcinogenic to humans).

Ethylbenzene

The primary sources of ethylbenzene in the environment are the petroleum industry and the use of petroleum products. Because of its physicochemical properties, more than 96% of ethylbenzene in the environment can be expected to be present in air. Values of up to 26 µg/m^3 in air have been reported. Ethylbenzene is found in trace amounts in surface water, groundwater, drinking-water and food.

Guideline value	0.3 mg/l (300 µg/l)
Occurrence	Concentrations in drinking-water generally below 1 µg/l; levels up to 300 µg/l have been reported in groundwater contaminated by point emissions
TDI	97.1 µg/kg body weight, based on a NOAEL of 136 mg/kg body weight per day for hepatotoxicity and nephrotoxicity observed in a limited 6-month study in rats, adjusting for daily dosing and using an uncertainty factor of 1000 (100 for interspecies and intraspecies variation and 10 for the limited database and short duration of the study)
Limit of detection	0.002–0.005 µg/l by GC with photoionization detector; 0.03–0.06 µg/l by 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 exceeds the lowest reported odour threshold for ethylbenzene in drinking-water (0.002 mg/l).
Assessment date	1993
Principal reference	WHO (2003) *Ethylbenzene in drinking-water*

Ethylbenzene is readily absorbed by the oral, inhalation or dermal route. In humans, storage in fat has been reported. Ethylbenzene is almost completely converted to soluble metabolites, which are excreted rapidly in urine. The acute oral toxicity is low. No definite conclusions can be drawn from limited teratogenicity data. No data on reproduction, long-term toxicity or carcinogenicity are available. Ethylbenzene has shown no evidence of genotoxicity in in vitro or in vivo systems.

Fenitrothion

Fenitrothion (CAS No. 122-14-5) is mainly used in agriculture for controlling insects on rice, cereals, fruits, vegetables, stored grains and cotton and in forest areas. It is also

used for the control of flies, mosquitoes and cockroaches in public health programmes and indoor use. Fenitrothion is stable in water only in the absence of sunlight or microbial contamination. In soil, biodegradation is the primary route of degradation, although photolysis may also play a role. Fenitrothion residues detected in water were low (maximum 1.30 µg/l) during the spruce budworm spray programme. Following the spraying of forests to control spruce budworm, water samples did not contain detectable amounts of fenitrothion; post-spray samples contained less than 0.01 µg/l. Levels of fenitrothion residues in fruits, vegetables and cereal grains decline rapidly after treatment, with a half-life of 1–2 days. Intake of fenitrothion appears to be primarily (95%) from food.

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	2003
Principal references	FAO/WHO (2001) *Pesticide residues in food—2000 evaluations* WHO (2004) *Fenitrothion in drinking-water*

On the basis of testing in an adequate range of studies in vitro and in vivo, JMPR concluded that fenitrothion is unlikely to be genotoxic. It also concluded that fenitrothion is unlikely to pose a carcinogenic risk to humans. In long-term studies of toxicity, inhibition of cholinesterase activity was the main toxicological finding in all species. A health-based value of 8 µg/l can be calculated for fenitrothion on the basis of an ADI of 0–0.005 mg/kg body weight, based on a NOAEL of 0.5 mg/kg body weight per day for inhibition of brain and erythrocyte cholinesterase activity in a 2-year study of toxicity in rats and supported by a NOAEL of 0.57 mg/kg body weight per day for inhibition of brain and erythrocyte cholinesterase activity in a 3-month study of ocular toxicity in rats and a NOAEL of 0.65 mg/kg body weight per day for reduced food consumption and body weight gain in a study of reproductive toxicity in rats, and allocating 5% of the upper limit of the ADI to drinking-water. However, because fenitrothion occurs at concentrations well below those of health concern, it is not considered necessary to derive a formal guideline value.

Fenoprop

The half-lives for degradation of chlorophenoxy herbicides, including fenoprop (CAS No. 93-72-1), also known as 2,4,5-trichlorophenoxy propionic acid or 2,4,5-TP, in the environment are in the order of several days. Chlorophenoxy herbicides are not often found in food.

Guideline value	0.009 mg/l (9 µg/l)
Occurrence	Chlorophenoxy herbicides not frequently found in drinking-water; when detected, concentrations usually no greater than a few micrograms per litre
TDI	3 µg/kg body weight, based on a NOAEL of 0.9 mg/kg body weight for adverse effects on the liver in a study in which dogs were administered fenoprop in the diet for 2 years, with an uncertainty factor of 300 (100 for interspecies and intraspecies variation and 3 for limitations of the database)