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 (1996) *Pesticide residues in food—1995 evaluations.* IPCS (1992) *Methyl parathion* WHO (2004) *Methyl parathion in drinking-water*

A NOAEL of 0.3 mg/kg body weight per day was derived from the combined results of several studies conducted in humans, based on the depression of erythrocyte and plasma cholinesterase activities. Methyl parathion decreased cholinesterase activities in long-term studies in mice and rats, but did not induce carcinogenic effects. Methyl parathion was mutagenic in bacteria, but there was no evidence of genotoxicity in a limited range of studies in mammalian systems.

A health-based value of 9 µg/l can be calculated for methyl parathion on the basis of an ADI of 0–0.003 mg/kg body weight, based on a NOAEL of 0.25 mg/kg body weight per day in a 2-year study in rats for retinal degeneration, sciatic nerve demyelination, reduced body weight, anaemia and decreased brain acetylcholinesterase activity, using an uncertainty factor of 100 for interspecies and intraspecies variation. As the toxicological end-points seen in experimental animals were other than acetylcholinesterase inhibition, it was considered more appropriate to use these data rather than the NOAEL derived for cholinesterase inhibition in humans.

Intake of methyl parathion from all sources is generally low and well below the upper limit of the ADI. As the health-based value is much higher than concentrations of methyl parathion likely to be found in drinking-water, the presence of methyl parathion in drinking-water under usual conditions is unlikely to represent a hazard to human health. For this reason, the establishment of a formal guideline value for methyl parathion is not deemed necessary.

Methyl tertiary-butyl ether

The major use of methyl *tert*-butyl ether, or MTBE, is as a gasoline additive. Surface water can be contaminated by gasoline spills; however, owing to the high volatility of MTBE, most is lost to evaporation. Spills and leaking storage tanks can cause more serious problems in groundwater, where MTBE is more persistent. MTBE has been detected in groundwater and drinking-water at concentrations in the nanogram to microgram per litre range.

Reason for not establishing a guideline value	Any guideline that would be derived would be significantly higher than concentrations at which MTBE would be detected by odour
Assessment date	2004
Principal references	IPCS (1998) *Methyl tertiary-butyl ether* WHO (2005) *Methyl tertiary-butyl ether (MTBE) in drinking-water*

No human cancer studies have been published for either the general population or occupationally exposed cohorts. There have been a number of human studies of

neurological and clinical effects of exposure to MTBE by inhalation, with mixed results. In general, no objective changes could be seen at levels of MTBE normally found, even in such microenvironments as gasoline filling stations.

The weight of evidence suggests that MTBE is not genotoxic. A large number of studies using in vitro and in vivo mammalian and non-mammalian systems have been conducted to assess the mutagenicity of MTBE, almost all of which have produced negative results. These results suggest that the mechanism of action of MTBE is more likely to be non-genotoxic than genotoxic, although no one mechanism appears to explain all of the observed effects.

It has been concluded that MTBE should be considered a rodent carcinogen but that it is not genotoxic, and the carcinogenic response is evident only at high levels of exposure that also induce other adverse effects. The available data are therefore considered inconclusive and prohibit their use for human carcinogenic risk assessment. A health-based guideline value has not been derived for MTBE, owing to the fact that any guideline value that would be derived would be significantly higher than the concentration at which it would be detected by odour (15 µg/l is the lowest level eliciting a response in a study using taste- and odour-sensitive participants).

Metolachlor

Metolachlor (CAS No. 51218-45-2) is a selective pre-emergence herbicide used on a number of crops. It can be lost from the soil through biodegradation, photodegradation and volatilization. It is fairly mobile and under certain conditions can contaminate groundwater, but it is mostly found in surface water.

Guideline value	0.01 mg/l (10 µg/l)
Occurrence	Detected in surface water and groundwater at concentrations that can exceed 10 µg/l
TDI	3.5 µg/kg body weight, based on a NOAEL of 3.5 mg/kg body weight for an apparent decrease in kidney weight at the two highest dose levels in a 1-year dog study, with an uncertainty factor of 1000 (100 for interspecies and intraspecies variation and 10 reflecting some concern regarding carcinogenicity)
Limit of detection	0.75–0.01 µg/l by GC with nitrogen–phosphorus 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	1993
Principal reference	WHO (2003) *Metolachlor in drinking-water*

In a 1-year study in dogs, administration of metolachlor resulted in decreased kidney weight at the two highest dose levels. In 2-year studies with rodents fed metolachlor in the diet, the only toxicological effects observed in mice were decreased body