Human exposure to EDTA arises directly from its use in food additives, medicines, and personal care and hygiene products. Exposure to EDTA from drinking-water is probably very small in comparison with that from other sources. Once EDTA is present in the aquatic environment, its speciation will depend on the water quality and the presence of trace metals with which it will combine. The removal of EDTA from communal wastewater by biodegradation in sewage purification plants is very limited.

**Guideline value** 0.6 mg/litre (for EDTA as the free acid)

**Occurrence** Present in surface waters generally at concentrations below 70 µg/litre, although higher concentrations (900 µg/litre) have been measured; detected in drinking-water prepared from surface waters at concentrations of 10–30 µg/litre

**ADI** 1.9 mg/kg of body weight as the free acid (ADI of 2.5 mg/kg of body weight proposed by JECFA for calcium disodium edetate as a food additive)

**Limit of detection** 1 µg/litre by potentiometric stripping analysis

**Treatment achievability** 0.01 mg/litre using GAC plus ozonation

**Guideline derivation**
- allocation to water: 1% of ADI
- weight: 60-kg adult
- consumption: 2 litres/day

**Additional comments** Concern has been expressed over the ability of EDTA to complex, and therefore reduce the availability of, zinc. However, this is of significance only at elevated doses substantially in excess of those encountered in the environment.

**Toxicological review**
Calcium disodium edetate is poorly absorbed from the gut. The long-term toxicity of EDTA is complicated by its ability to chelate essential and toxic metals. Those toxicological studies that are available indicate that the apparent toxicological effects of EDTA have in fact been due to zinc deficiency as a consequence of complexation. EDTA does not appear to be teratogenic or carcinogenic in animals. The vast clinical experience of the use of EDTA in the treatment of metal poisoning has demonstrated its safety in humans.

**History of guideline development**
refer to edetic acid. The 1993 Guidelines proposed a provisional health-based guideline value of 0.2 mg/litre for edetic acid, based on an ADI for calcium disodium edetate as a food additive proposed by JECFA in 1973 and assuming that a 10-kg child consumes 1 litre of water per day, in view of the possibility of zinc complexation. The value was considered provisional to reflect the fact that the JECFA ADI had not been considered since 1973. JECFA further evaluated the toxicological studies available on EDTA in 1993 and was unable to add any further important information regarding the toxicity of EDTA and its calcium and sodium salts to the 1973 evaluation. In the addendum to the second edition of the Guidelines, published in 1998, a guideline value of 0.6 mg/litre was derived for EDTA (free acid), using different assumptions from those used in the derivation of the provisional guideline value in the 1993 Guidelines. In particular, it was noted that the ability of EDTA to complex, and therefore reduce the availability of, zinc was of significance only at elevated doses substantially in excess of those encountered in the environment.

**Assessment date**
The risk assessment was originally conducted in 1998. 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**

**12.57 Endosulfan**
Endosulfan (CAS No. 115-29-7) is an insecticide used in countries throughout the world to control pests on fruit, vegetables and tea and on non-food crops such as tobacco and cotton. In addition to its agricultural use, it is used in the control of the tsetse fly, as a wood preservative and for the control of home garden pests. Endosulfan contamination does not appear to be widespread in the aquatic environment, but the chemical has been found in agricultural runoff and rivers in industrialized areas where it is manufactured or formulated, as well as in surface water and groundwater samples collected from hazardous waste sites in the USA. Surface water samples in the USA generally contain less than 1 mg/litre. The main source of exposure of the general population is food, but residues have generally been found to be well below the FAO/WHO maximum residue limits. Another important route of exposure to endosulfan for the general population is the use of tobacco products.

JMPR concluded that endosulfan is not genotoxic, and no carcinogenic effects were noted in long-term studies using mice and rats. The kidney is the target organ for toxicity. Several recent studies have shown that endosulfan, alone or in combination with other pesticides, may bind to estrogen receptors and perturb the endocrine system. A