

Additional comments	Aldrin and dieldrin are listed under the Stockholm Convention on Persistent Organic Pollutants. Hence, monitoring may occur in addition to that required by drinking-water guidelines.
Assessment date	2003
Principal references	FAO/WHO (1995) <i>Pesticide residues in food—1994 evaluations</i> WHO (2003) <i>Aldrin and dieldrin in drinking-water</i>

Both compounds are highly toxic in experimental animals, and cases of poisoning in humans have occurred. Aldrin and dieldrin have more than one mechanism of toxicity. The target organs are the central nervous system and the liver. In long-term studies, dieldrin was shown to produce liver tumours in both sexes of two strains of mice. It did not produce an increase in tumours in rats and does not appear to be genotoxic. IARC has classified aldrin and dieldrin in Group 3 (not classifiable as to its carcinogenicity to humans). Exposure through food has decreased significantly with the dramatic reduction in use.

### **Aluminium**

Aluminium is the most abundant metallic element and constitutes about 8% of Earth's crust. Aluminium salts are widely used in water treatment as coagulants to reduce organic matter, colour, turbidity and microorganism levels. Such use may lead to increased concentrations of aluminium in finished water. Where residual concentrations are high, undesirable colour and turbidity may ensue. Concentrations of aluminium at which such problems may occur are highly dependent on a number of water quality parameters and operational factors at the water treatment plant. Aluminium intake from foods, particularly those containing aluminium compounds used as food additives, represents the major route of aluminium exposure for the general public. The contribution of drinking-water to the total oral exposure to aluminium is usually less than 5% of the total intake.

Reason for not establishing a guideline value	A health-based value of 0.9 mg/l could be derived from the JECFA provisional tolerable weekly intake (PTWI), but this value exceeds practicable levels based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants: 0.1 mg/l or less in large water treatment facilities and 0.2 mg/l or less in small facilities
Assessment date	2009
Principal references	FAO/WHO (2007) Aluminium (from all sources, including food additives) IPCS (1997) <i>Aluminium</i> WHO (2010) <i>Aluminium in drinking-water</i>

There is little indication that orally ingested aluminium is acutely toxic to humans despite the widespread occurrence of the element in foods, drinking-water and many antacid preparations. It has been hypothesized that aluminium exposure is a risk factor for the development or acceleration of onset of Alzheimer disease in humans. The 1997 WHO Environmental Health Criteria document for aluminium concludes that:

On the whole, the positive relationship between aluminium in drinking-water and AD [Alzheimer disease], which was demonstrated in several epidemiological studies, cannot be totally dismissed. However, strong reservations about inferring a causal relationship are warranted in view of the failure of these studies to account for demonstrated confounding factors and for total aluminium intake from all sources.

Taken together, the relative risks for AD from exposure to aluminium in drinking-water above 100 µg/l, as determined in these studies, are low (less than 2.0). But, because the risk estimates are imprecise for a variety of methodological reasons, a population-attributable risk cannot be calculated with precision. Such imprecise predictions may, however, be useful in making decisions about the need to control exposures to aluminium in the general population.

In 2007, JECFA developed a PTWI for aluminium from all sources of 1 mg/kg body weight. JECFA concluded the following:

... the available studies have many limitations and are not adequate for defining the dose-response relationships. The Committee therefore based its evaluation on the combined evidence from several studies. The relevance of studies involving administration of aluminium compounds by gavage was unclear because the toxicokinetics after gavage were expected to differ from toxicokinetics after dietary administration, and the gavage studies generally did not report total aluminium exposure including basal levels in the feed. The studies conducted with dietary administration of aluminium compounds were considered most appropriate for the evaluation. The lowest LOELs [lowest-observed-effect levels] for aluminium in a range of different dietary studies in mice, rats and dogs were in the region of 50–75 mg/kg bw [body weight] per day expressed as Al.

The Committee applied an uncertainty factor of 100 to the lower end of this range of LOELs (50 mg/kg bw per day expressed as Al) to allow for inter- and intraspecies differences. There are deficiencies in the database, notably the absence of NOELs [no-observed-effect levels] in the majority of the studies evaluated and the absence of long-term studies on the relevant toxicological end-points. The deficiencies are counterbalanced by the probable lower bioavailability of the less soluble aluminium species present in food. Overall, an additional uncertainty factor of three was considered to be appropriate. The Committee confirmed that the resulting health-based guidance value should be expressed as a PTWI, because of the potential for bioaccumulation. The Committee established a PTWI of 1 mg/kg bw for Al, which applies to all aluminium compounds in food, including additives.

A health-based value derived from the JECFA PTWI would be 0.9 mg/l (rounded value), based on an allocation of 20% of the PTWI to drinking-water and assuming a 60 kg adult drinking 2 litres of water per day. However, there remain uncertainties as to the extent of aluminium absorption from drinking-water, which depends on a number of parameters, such as the aluminium salt administered, pH (for aluminium speciation and solubility), bioavailability and dietary factors.

The beneficial effects of the use of aluminium as a coagulant in water treatment are recognized. Taking this into account, and considering the health concerns about aluminium (i.e. its potential neurotoxicity), a practicable level is derived, based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants, to minimize aluminium levels in finished water.

Several approaches are available for minimizing residual aluminium concentrations in treated water. These include use of optimum pH in the coagulation process, avoiding excessive aluminium dosage, good mixing at the point of application of the coagulant, optimum paddle speeds for flocculation and efficient filtration of the aluminium floc. Under good operating conditions, concentrations of aluminium of 0.1 mg/l or less are achievable in large water treatment facilities. Small facilities (e.g. those serving fewer than 10 000 people) might experience some difficulties in attaining this level, because the small size of the plant provides little buffering for fluctuation in operation; moreover, such facilities often have limited resources and limited access to the expertise needed to solve specific operational problems. For these small facilities, 0.2 mg/l or less is a practicable level for aluminium in finished water.

As indicated above, a health-based value derived from the JECFA PTWI would be 0.9 mg/l (rounded value) based on an allocation of 20% of the PTWI to drinking-water and assuming a 60 kg adult drinking 2 litres of water per day. However, as also noted above, practicable levels based on optimization of the coagulation process in drinking-water plants using aluminium-based coagulants are less than 0.1 mg/l in large water treatment facilities and less than 0.2 mg/l in small facilities. In view of the importance of optimizing coagulation to prevent microbial contamination and the need to minimize deposition of aluminium floc in distribution systems, it is important to ensure that average residuals do not exceed these values.

### **Ammonia**

The term ammonia includes the non-ionized ( $\text{NH}_3$ ) and ionized ( $\text{NH}_4^+$ ) species. Ammonia in the environment originates from metabolic, agricultural and industrial processes and from disinfection with chloramine. Natural levels in groundwater and surface water are usually below 0.2 mg/l. Anaerobic groundwaters may contain up to 3 mg/l. Intensive rearing of farm animals can give rise to much higher levels in surface water. Ammonia contamination can also arise from cement mortar pipe linings. Ammonia in water is an indicator of possible bacterial, sewage and animal waste pollution.

Reason for not establishing a guideline value	Occurs in drinking-water at concentrations well below those of health concern
Assessment date	1993
Principal reference	WHO (2003) <i>Ammonia in drinking-water</i>

Ammonia is a major component of the metabolism of mammals. Exposure from environmental sources is insignificant in comparison with endogenous synthesis of ammonia. Toxicological effects are observed only at exposures above about 200 mg/kg body weight.

Ammonia in drinking-water is not of immediate health relevance, and therefore no health-based guideline value is proposed. However, ammonia can compromise disinfection efficiency, result in nitrite formation in distribution systems, cause the failure of filters for the removal of manganese and cause taste and odour problems (see also chapter 10).