FACT SHEETS

Aluminium

GUIDELINE

Based on aesthetic problems caused by post-flocculation, the concentration of acid-soluble aluminium in drinking water should not exceed 0.2 mg/L. Water authorities are strongly encouraged to keep acid-soluble aluminium concentrations as low as possible, preferably below 0.1 mg/L.

No health-based guideline is set for aluminium at this time but this issue will be kept under review.

GENERAL DESCRIPTION

Aluminium may be present in water through natural leaching from soil and rock, or from the use of aluminium salts as coagulants in water treatment.

Aluminium is used in many industrial and domestic products including antacids, antiperspirants and food additives, and in vaccines. It is commonly used by the food industry for food containers and packaging, and many cooking utensils are made from aluminium.

Surveys in the United States and the United Kingdom have reported aluminium concentrations in natural water sources of 0.014–1.2 mg/L. Concentrations in some Australian water sources can be considerably higher due to the presence of clay minerals (aluminosilicates); for example, up to 18 mg/L in the Murray River. Residual aluminium concentrations in treated water depend on the concentration in the water source, the alum dose used, the pH, and the filtration efficiency.

Where alum is used as a coagulant in water treatment, post-flocculation effects can occur if the soluble aluminium concentration in the treated water exceeds 0.2 mg/L. Depending on pH, a whitish gelatinous precipitate of aluminium hydroxide can be formed in the distribution system which may result in customer complaints about 'milky coloured' water. Aluminosilicates in source water are very insoluble and do not cause post-flocculation problems.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, the concentration of aluminium varies from 0.01 mg/L to 0.9 mg/L, with typical concentrations of approximately 0.1 mg/L for fully treated supplies.

TREATMENT OF DRINKING WATER

Aluminium concentrations in drinking water can be reduced using the conventional water treatment practices of flocculation and filtration. A well-operated water filtration plant (even using aluminium as a flocculant) can achieve aluminium concentrations in the finished water of less than 0.1 mg/L.

MEASUREMENT

The term 'soluble' should be taken to mean truly soluble, not 'filterable through a 0.45 µm pore size filter'. Finely suspended aluminosilicate clay particles can pass through a 0.45 µm filter but are not truly soluble and will not cause post-flocculation problems.

Acid-soluble aluminium is determined after acidifying the sample to pH 1.5-2, followed by filtration through a 0.45 µm membrane filter. If analysis of the filtrate by the normal method (e.g. graphite furnace atomic absorption spectroscopy, APHA Method 3500-Al Part B 1992) gives a result above the guideline value, the filtrate should be re-analysed using the catechol violet colorimetric method (APHA Method 3500-Al Part E 1992), which provides a better estimate of the reactive aluminium component. The limit of determination for the latter method is approximately 0.01 mg/L.

Based on experience with their water supplies, authorities may choose to monitor total aluminium concentration and perform specific assays for acid-soluble aluminium only if total aluminium concentration exceeds 0.1 mg/L.

HEALTH CONSIDERATIONS

It has been estimated that for Australian adults, the intake of aluminium from food and beverages is approximately 5-7 mg/day. Drinking water contributes less than 2% of the total daily intake, and only 0.3-0.4% of the aluminium in water is absorbed by the body. Recent studies have shown that the bioavailability (i.e. uptake into the bloodstream) of aluminium in drinking water is similar to that of food (Stauber et al. 1999).

The metabolism of aluminium in humans is poorly understood. Studies indicate that less than 1% of dietary aluminium is absorbed by the gastrointestinal tract, with the remainder excreted in faeces. The small amount absorbed passes into the blood stream. Some aluminium accumulates in bone, liver and brain tissue but most is removed from the blood stream by the kidneys and excreted. In healthy adults, the total accumulated body load of aluminium has been estimated at about 35 mg. Whether this remains constant with age has not been determined.

There is considerable evidence that aluminium is neurotoxic. Kidney dialysis patients, in whom the gut barrier is bypassed, can accumulate aluminium in their blood resulting in an encephalopathy known as dialysis dementia. Investigations have established a correlation between the concentration of aluminium in water used to prepare dialysis fluid and the incidence of dialysis dementia. If this condition is not too far advanced it responds to chelation therapy. It appears that dialysis patients are much more susceptible to aluminium in dialysis fluid than from other sources such as food and antacids. Aluminium has also been linked to other conditions associated with the use of dialysis units including osteomalacia (a softening of the bones) and anaemia. Reverse osmosis or deionisation units are now used to treat dialysis water before use, and aluminium concentrations are kept below 0.01 mg/L.

Aluminium has been associated with two severe neurodegenerative diseases: Parkinsonism dementia (PD) and amyotrophic lateral sclerosis (ALS). Both conditions have a high incidence amongst the Chamorro people of Guam, an area where aluminium is naturally present in food and drinking water. ALS is common in the Pacific, Western New Guinea and the Kii peninsula of Japan. Both PD and ALS are characterised by loss of motor function and the presence of neurofibrillary tangles in the brain. One hypothesis suggests that chronic nutritional deficiencies of calcium and magnesium lead to increased absorption of aluminium, resulting in its deposition in neurons of the brain (Garruto and Yase 1986, Garruto et al. 1990). There was an appreciable decrease in the incidence of these conditions when the areas became westernised, with associated changes in dietary habits, importing of food and improvements to the water supply.

Elevated concentrations of aluminium have been found in the autopsied brains of people who had suffered Alzheimer's disease, in regions of the brain containing large numbers of the neurofibrillary tangles which are characteristic of the disease, and aluminium has been proposed as one of a number of causal agents (Perl and Brody 1980). There have been a number of epidemiological studies to determine if aluminium in drinking water plays a role in Alzheimer's disease. Although some studies indicated that a tentative link may exist, more recent evidence (Martyn et al. 1997) suggests that aluminium in drinking water is not associated with increased risk of Alzheimer's disease.

A number of animal studies of aluminium toxicity have been undertaken although there has been very little research done using aged animals. Most studies have used rats fed or injected with large amounts of aluminium and have reported only minor changes to bodyweight, with some behavioural changes and locomotor effects. Elevated concentrations of aluminium have been reported in the brain, liver and kidneys. The studies are not adequate to set a reliable no observable effect level (NOEL).

Aluminium is not generally thought to be mutagenic or genotoxic, although aluminium has been shown to bind to DNA of a number of animal species and has displayed mutagenic activity in some, but not all, tests using bacteria. Ingestion of aluminium is not known to cause cancer in humans or animals.

The NHMRC Standing Committee on Toxicity has reviewed the toxicological data for aluminium and concluded that there are insufficient data to set a NOEL.

DERIVATION OF GUIDELINE

Post-flocculation problems (described above) associated with the use of alum as a coagulant may occur if acid-soluble aluminium exceeds 0.2 mg/L. As the alum floc is soluble in dilute acid (pH 1.5-2), postflocculation problems will generally be avoided if the acid-soluble concentration of aluminium is below 0.2 mg/L. Water authorities are strongly encouraged to keep acid-soluble aluminium concentrations as low as possible, preferably below 0.1 mg/L. Well operated water filtration plants, even those using aluminium salts as flocculants, should have little difficulty in achieving this.

A guideline value lower than 0.2 mg/L may need to be adopted by some water authorities, depending on the amount of naturally occurring organic material in the water.

Although data are insufficient to set a guideline value based on health considerations, there is public concern over the possible health effects of aluminium. This issue should be reviewed when further studies are undertaken.

REFERENCES

APHA Method 3500-Al Part B (1992). Aluminium: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Al Part E (1992). Aluminium: Automated Pyrocatechol violet method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Garruto RM, Yase Y (1986). Neurodegenerative disorders of the Western Pacific: the search for mechanisms of pathogenesis. Trends in Neurosciences, 9:368–374.

Garruto RM, Yanagihara R, Gajdusek DC (1990). Models of environmentally induced neurological disease: epidemiology and etiology of amyotrophic lateral sclerosis and parkinsonism-dementia in the Western Pacific. Environmental Geochemistry and Health, 12:137-151.

Martyn CN, Osmond C, Edwardson JA, Barker DJP, Harris EC, Lacey RF (1989). Geographical relation between Alzheimer's disease and aluminium in drinking water. Lancet (January):59-62.

Perl DP, Brody AR (1980). Alzheimer's disease: X-ray spectrometric evidence of aluminium accumulation in neurofibrillary tangle-bearing neurons. Science, 208:297-299.

Stauber JL, Florence TM, Davies CM, Adams MS, Buchanan SJ (1999). Bioavailability of aluminium in alum-treated drinking water. Journal American Water Works Association, 91:84–93.