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Aluminium in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

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Preface

One of the primary goals of WHO and its member states is that "all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water." A major WHO function to achieve such goals is the responsibility "to propose ... regulations, and to make recommendations with respect to international health matters ...."

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as International Standards for Drinking-Water. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO Guidelines for drinking-water quality (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared/updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A "final task force" meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO internet site and in the current edition of the GDWQ.

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The work of the following coordinators was crucial in the development of this document and others in the Addendum:

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GENERAL DESCRIPTION

Identity

Aluminium is the most abundant metallic element and constitutes about 8% of the Earth's crust. It occurs naturally in the environment as silicates, oxides, and hydroxides, combined with other elements, such as sodium and fluoride, and as complexes with organic matter.

Compound	*CAS no.*	*Molecular formula*
Aluminium	7429-90-5	Al
Aluminium chloride	7446-70-0	$AlCl_3$
Aluminium hydroxide	21645-51-2	$Al(OH)_3$
Aluminium oxide	1344-28-1	Al_2O_3
Aluminium sulfate	10043-01-3	$Al_2(SO_4)_3$

Physicochemical properties *(Lide, 1993)*

Property	*Al*	*$AlCl_3$*	*$Al(OH)_3$*	*Al_2O_3*	*$Al_2(SO_4)_3$*
Melting point (°C)	660	190	300	2072	770 (d)
Boiling point (°C)	2467	262 (d)	–	2980	–
Density at 20°C (g/cm^3)	2.70	2.44	2.42	3.97	2.71
Water solubility (g/litre)	(i)	69.9	(i)	(i)	31.3 at 0°C

d, decomposes; i, insoluble

Organoleptic properties

Use of aluminium salts as coagulants in water treatment may lead to increased concentrations of aluminium in finished water. Where residual concentrations are high, aluminium may be deposited in the distribution system. Disturbance of the deposits by change in flow rate may increase aluminium levels at the tap and lead to undesirable colour and turbidity (WHO, 1996). 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.

Major uses

Aluminium metal is used as a structural material in the construction, automotive, and aircraft industries, in the production of metal alloys, in the electric industry, in cooking utensils, and in food packaging. Aluminium compounds are used as antacids, antiperspirants, and food additives (ATSDR, 1992). Aluminium salts are also widely used in water treatment as coagulants to reduce organic matter, colour, turbidity, and microorganism levels. The process usually consists of addition of an aluminium salt (often sulfate) at optimum pH and dosage, followed by flocculation, sedimentation, and filtration (Health Canada, 1993).

Environmental fate

Aluminium is released to the environment mainly by natural processes. Several factors influence aluminium mobility and subsequent transport within the environment. These include chemical speciation, hydrological flow paths, soil–water interactions, and the composition of the underlying geological materials. Acid environments caused by acid mine drainage or acid rain can cause an increase in the dissolved aluminium content of the surrounding waters (ATSDR, 1992; WHO, 1997).

Aluminium can occur in a number of different forms in water. It can form monomeric and polymeric hydroxy species, colloidal polymeric solutions and gels, and precipitates, all based on aquated positive ions or hydroxylated aluminates. In addition, it can form complexes with various organic compounds (e.g. humic or fulvic acids) and inorganic ligands (e.g. fluoride, chloride, and sulfate), most but not all of which are soluble. The chemistry of aluminium in water is complex, and many chemical parameters, including pH, determine which aluminium species are present in aqueous solutions. In pure water, aluminium has a minimum solubility in the pH range 5.5–6.0; concentrations of total dissolved aluminium increase at higher and lower pH values (CCME, 1988; ISO, 1994).

ANALYTICAL METHODS

Aluminium is reacted with pyrocatechol violet followed by spectrometric measurement of the resulting coloured complex. The method is restricted to the determination of the aquated cations and other forms of aluminium readily converted to that cationic form by acidification. The limit of detection is 2 µg/litre (ISO, 1994). The limit of detection for the determination of aluminium by inductively coupled plasma atomic emission spectroscopy ranges from 40 to 100 µg/litre (ISO, 1996).

Flame and graphite furnace atomic absorption spectrometric (AAS) methods are applicable for the determination of aluminium in water at concentrations of 5–100 mg/litre and 0.01–0.1 mg/litre, respectively. The working range of the graphite furnace AAS method can be shifted to higher concentrations either by dilution of the sample or by using a smaller sample volume (ISO, 1997).

ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Air

Aluminium enters the atmosphere as a major constituent of atmospheric particulates originating from natural soil erosion, mining or agricultural activities, volcanic eruptions, or coal combustion. Atmospheric aluminium concentrations show widespread temporal and spatial variations. Airborne aluminium levels range from 0.0005 µg/m^3 over Antarctica to more than 1 µg/m^3 in industrialized areas (WHO, 1997).

Water

The concentration of aluminium in natural waters can vary significantly depending on various physicochemical and mineralogical factors. Dissolved aluminium concentrations in waters with near-neutral pH values usually range from 0.001 to 0.05 mg/litre but rise to 0.5–1 mg/litre in more acidic waters or water rich in organic matter. At the extreme acidity of waters affected by acid mine drainage, dissolved aluminium concentrations of up to 90 mg/litre have been measured (WHO, 1997).

Aluminium levels in drinking-water vary according to the levels found in the source water and whether aluminium coagulants are used during water treatment. In Germany, levels of aluminium in public water supplies averaged 0.01 mg/litre in the western region, whereas levels in 2.7% of public supplies in the eastern region exceeded 0.2 mg/litre (Wilhelm & Idel, 1995). In a 1993–1994 survey of public water supplies in Ontario, Canada, 75% of all average levels were less than 0.1 mg/litre, with a range of 0.04–0.85 mg/litre (OMEE, 1995). In a large monitoring programme in 1991 in the United Kingdom, concentrations in 553 samples (0.7%) exceeded 0.2 mg/litre (MAFF, 1993). In a survey of 186 community water supplies in the USA, median aluminium concentrations for all finished drinking-water samples ranged from 0.03 to 0.1 mg/litre; for facilities using aluminium sulfate coagulation, the median level was 0.1 mg/litre, with a maximum of 2.7 mg/litre (Miller et al., 1984). In another US survey,

the average aluminium concentration in treated water at facilities using aluminium sulfate coagulation ranged from 0.01 to 1.3 mg/litre, with an overall average of 0.16 mg/litre (Letterman & Driscoll, 1988; ATSDR, 1992).

Food

Aluminium is present in foods naturally or from the use of aluminium-containing food additives. The use of aluminium cookware, utensils, and wrappings can increase the amount of aluminium in food; however, the magnitude of this increase is generally not of practical importance. Foods naturally high in aluminium include potatoes, spinach, and tea. Processed dairy products, flour, and infant formula may be high in aluminium if they contain aluminium-based food additives (FAO/WHO, 1989; Pennington & Schoen, 1995; WHO, 1997).

Adult dietary intakes of aluminium (mg/day) have been reported in several countries: Australia (1.9–2.4), Finland (6.7), Germany (8–11), Japan (4.5), Netherlands (3.1), Sweden (13), Switzerland (4.4), United Kingdom (3.9), and USA (7.1–8.2). Intake of children 5–8 years old was 0.8 mg/day in Germany and 6.5 mg/day in the USA. Infant intakes of aluminium in Canada, the United Kingdom, and the USA ranged from 0.03 to 0.7 mg/day (WHO, 1997).

Estimated total exposure and relative contribution of drinking-water

Aluminium intake from foods, particularly those containing aluminium compounds used as food additives, represents the major route of aluminium exposure for the general public, excluding persons who regularly ingest aluminium-containing antacids and buffered analgesics, for whom intakes may be as high as 5 g/day (WHO, 1997).

At an average adult intake of aluminium from food of 5 mg/day and a drinking-water aluminium concentration of 0.1 mg/litre, the contribution of drinking-water to the total oral exposure to aluminium will be about 4%. The contribution of air to the total exposure is generally negligible.

KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

In experimental animals, absorption of aluminium via the gastrointestinal tract is usually less than 1%. The main factors influencing absorption are solubility, pH, and chemical species. Organic complexing compounds, notably citrate, increase absorption. Aluminium absorption may interact with calcium and iron transport systems. Aluminium, once absorbed, is distributed in most organs within the body, with accumulation occurring mainly in bone at high dose levels. To a limited but as yet undetermined extent, aluminium passes the blood–brain barrier and is also distributed to the fetus. Aluminium is eliminated effectively in the urine (WHO, 1997).

In humans, aluminium and its compounds appear to be poorly absorbed, although the rate and extent of absorption have not been adequately studied. The mechanism of gastrointestinal absorption has not yet been fully elucidated. Variability results from the chemical properties of the element and the formation of various chemical species, which is dependent upon the pH, ionic strength, presence of competing elements (e.g. silicon), and presence of complexing agents within the gastrointestinal tract (e.g. citrate). The urine is the most important route of aluminium excretion (WHO, 1996, 1997).

EFFECTS ON EXPERIMENTAL ANIMALS AND IN VITRO TEST SYSTEMS

Acute exposure

The oral LD_{50} of aluminium nitrate, chloride, and sulfate in mice and rats ranges from 200 to 1000 mg of aluminium per kg of body weight (WHO, 1997).

Short-term exposure

Groups of 25 male Sprague-Dawley rats were fed diets containing basic sodium aluminium phosphate or aluminium hydroxide at 0, 5, 67, 141, or 288/302 mg of aluminium per kg of body weight per day for 28 days. No treatment-related effects on organ and body weights, haematology, clinical chemistry parameters, and histopathology were observed, and there was no evidence of deposition of aluminium in bones. The NOELs were 288 and 302 mg of aluminium per kg of body weight per day for sodium aluminium phosphate and aluminium hydroxide, respectively (Hicks et al., 1987).

In a study in which a wide range of end-points was examined, groups of 10 female Sprague-Dawley rats received drinking-water containing aluminium nitrate for 28 days at 0, 1, 26, 52, or 104 mg of aluminium per kg of body weight per day. The only effects noted were mild histopathological changes in the spleen and liver of the high-dose group. Although tissue aluminium concentrations were generally higher in treated animals, the increases were significant only for spleen, heart, and gastrointestinal tract of the high-dose group. The NOAEL was 52 mg of aluminium per kg of body weight per day (Gomez et al., 1986).

Groups of 10 female Sprague-Dawley rats received aluminium nitrate in their drinking-water at doses of 0, 26, 52, or 260 mg of aluminium per kg of body weight per day for 100 days. Organ and body weights, histopathology of the brain, heart, lungs, kidney, liver, and spleen, haematology, and plasma chemistry were examined. The only effect observed was a significant decrease in body-weight gain associated with a decrease in food consumption at 260 mg of aluminium per kg of body weight per day. Aluminium did not accumulate in a dose-dependent manner in the organs and tissues examined. The NOAEL in this study was 52 mg of aluminium per kg of body weight per day (Domingo et al., 1987a).

Sodium aluminium phosphate, a leavening acid, was administered to groups of six male and six female beagle dogs at dietary concentrations of 0, 0.3, 1.0, or 3.0% for 6 months. Statistically significant decreases in food consumption occurred sporadically in all treated groups of female dogs, but there was no associated decrease in body weight. No significant absolute or relative organ-weight differences were found between any of the treated groups and controls. Haematological, blood chemistry, and urinalysis data showed no toxicologically significant trend. The NOAEL was the highest dose tested, approximately 70 mg of aluminium per kg of body weight per day (Katz et al., 1984).

Beagle dogs (four per sex per dose) were fed diets containing basic sodium aluminium phosphate at 0, 10, 22–27, or 75–80 mg of aluminium per kg of body weight per day for 26 weeks. The only treatment-related effect was a sharp, transient decrease in food consumption and concomitant decrease in body weight in high-dose males. The LOAEL was 75–80 mg/kg of body weight per day (Pettersen et al., 1990)

Long-term exposure

No adverse effects on body weight or longevity were observed in Charles River mice (54 males and 54 females per group) receiving 0 or 5 mg of aluminium (as potassium aluminium sulfate) per kg of diet during their lifetime (Schroeder & Mitchener, 1975a; FAO/WHO, 1989).

Two groups of Long-Evans rats (52 of each sex) received 0 or 5 mg of aluminium (as potassium aluminium sulfate) per litre of drinking-water during their lifetime. No effects were found on body weight; average heart weight; glucose, cholesterol, and uric acid levels in serum; and protein and glucose content and pH of urine. The life span was not affected (Schroeder & Mitchener, 1975b; FAO/WHO, 1989).

Reproductive and developmental toxicity

Aluminium nitrate was administered by gavage to groups of pregnant Sprague-Dawley rats on day 14 of gestation through day 21 of lactation at doses of 0, 13, 26, or 52 mg of aluminium per kg of body weight per day. These doses did not produce overt fetotoxicity, but growth of offspring was significantly delayed (body weight, body length, and tail length) from birth to weaning in aluminium-treated groups (Domingo et al., 1987b).

Aluminium nitrate was administered by intubation to male Sprague-Dawley rats at 0, 13, 26, or 52 mg of aluminium per kg of body weight per day for 60 days prior to mating and to virgin females for 14 days prior to mating, with treatment continuing throughout mating, gestation, parturition, and weaning of the litters. No reproductive effects on fertility (number of litters produced), litter size, or intrauterine or postnatal offspring mortality were reported. There was a decrease in the numbers of corpus lutea in the highest dose group. However, a dose-dependent delay in the growth of the pups was observed in all treatment groups; female offspring were affected at 13 mg of aluminium per kg of body weight per day and males at 26 and 52 mg of aluminium per kg of body weight per day. Because of the design of this study, it is not clear whether the postnatal growth effects in offspring represented general toxicity to male or female parents or specific effects on reproduction or development. However, the reported LOAEL in females in this study was 13 mg of aluminium per kg of body weight per day (Domingo et al., 1987c).
The developmental toxicity of aluminium by the oral route is highly dependent on the form of aluminium and the presence of organic chelators that influence bioavailability. Aluminium hydroxide did not produce either maternal or developmental toxicity when it was administered by gavage during embryogenesis to mice at doses up to 92 mg of aluminium per kg of body weight per day (Domingo et al., 1989) or to rats at doses up to 265 mg of aluminium per kg of body weight per day (Gomez et al., 1990). When aluminium hydroxide at a dose of 104 mg of aluminium per kg of body weight per day was administered with ascorbic acid to mice, no maternal or developmental toxicity was seen, in spite of elevated maternal placenta and kidney concentrations of aluminium (Colomina et al., 1994); on the other hand, aluminium hydroxide at a dose of 133 mg of aluminium per kg of body weight per day administered with citric acid produced maternal and fetal toxicity in rats (Gomez et al., 1991). Aluminium hydroxide (57 mg of aluminium per kg of body weight) given with lactic acid (570 mg/kg of body weight) to mice by gavage was not toxic, but aluminium lactate (57 mg of aluminium per kg of body weight) produced developmental toxicity, including poor ossification, skeletal variations, and cleft palate (Colomina et al., 1992).

Mutagenicity and related end-points

Aluminium can form complexes with DNA and cross-link chromosomal proteins and DNA, but it has not been shown to be mutagenic in bacteria or induce mutation or transformation in mammalian cells *in vitro*. Chromosomal aberrations have been observed in bone marrow cells of exposed mice and rats (WHO, 1997).
Carcinogenicity

There is no indication that aluminium is carcinogenic. JECFA evaluated the limited studies of Schroeder and Mitchener (1975a,b; section 5.3) and concluded that there was no evidence of

an increase in tumour incidence related to the administration of potassium aluminium sulfate in mice or rats (FAO/WHO, 1989).

Neurotoxicity

Behavioural impairment has been reported in laboratory animals exposed to soluble aluminium salts (e.g. lactate, chloride) in the diet or drinking-water in the absence of overt encephalopathy or neurohistopathology. Both rats (Commissaris et al., 1982; Thorne et al., 1987; Connor et al., 1988) and mice (Yen-Koo, 1992) have demonstrated such impairments at doses exceeding 200 mg of aluminium per kg of body weight per day. Although significant alterations in acquisition and retention of learned behaviour were documented, the possible role of organ damage (kidney, liver, immunological) due to aluminium was incompletely evaluated in these studies (WHO, 1997).

In studies on brain development in mice and rats, grip strength was impaired in offspring of dams fed 100 mg of aluminium (as aluminium lactate) per kg of body weight per day in the diet, in the absence of maternal toxicity (WHO, 1997).

EFFECTS ON HUMANS

There is little indication that aluminium is acutely toxic by oral exposure despite its widespread occurrence in foods, drinking-water, and many antacid preparations (WHO, 1997).

In 1988, a population of about 20 000 individuals in Camelford, England, was exposed for at least 5 days to unknown but increased levels of aluminium accidentally distributed to the population from a water supply facility using aluminium sulfate for treatment. Symptoms including nausea, vomiting, diarrhoea, mouth ulcers, skin ulcers, skin rashes, and arthritic pain were noted. It was concluded that the symptoms were mostly mild and short-lived. No lasting effects on health could be attributed to the known exposures from aluminium in the drinking-water (Clayton, 1989).

It has been hypothesized that aluminium exposure is a risk factor for the development or acceleration of onset of Alzheimer disease (AD) in humans. WHO (1997) has evaluated some 20 epidemiological studies that have been carried out to test the hypothesis that aluminium in drinking-water is a risk factor for AD. Study designs ranged from ecological to case–control. Six studies on populations in Norway (Flaten, 1990), Canada (Neri & Hewitt, 1991), France (Michel et al., 1991; Jacqmin et al., 1994), Switzerland (Wettstein et al., 1991), and England (Martyn et al., 1989) were considered of sufficiently high quality to meet the general criteria for exposure and outcome assessment and the adjustment for at least some confounding variables. Of the six studies that examined the relationship between aluminium in drinking-water and dementia or AD, three found a positive relationship, but three did not. However, each of the studies had some deficiencies in the study design (e.g. ecological exposure assessment; failure to consider aluminium exposure from all sources and to control for important confounders, such as education, socioeconomic status, and family history; the use of surrogate outcome measures for AD; and selection bias). In general, the relative risks determined were less than 2, with large confidence intervals, when the total aluminium concentration in drinking-water was 0.1 mg/litre or higher. Based on current knowledge of the pathogenesis of AD and the totality of evidence from these epidemiological studies, it was concluded that the present epidemiological evidence does not support a causal association between AD and aluminium in drinking-water (WHO, 1997).

In addition to the epidemiological studies that examined the relationship between AD and aluminium in drinking-water, two studies examined cognitive dysfunction in elderly populations in relation to the levels of aluminium in drinking-water. The results were again

conflicting. One study of 800 male octogenarians consuming drinking-water with aluminium concentrations up to 98 µg/litre found no relationship (Wettstein et al., 1991). The second study used "any evidence of mental impairment" as an outcome measure and found a relative risk of 1.72 at aluminium drinking-water concentrations above 85 µg/litre in 250 males (Forbes et al., 1994). Such data are insufficient to show that aluminium is a cause of cognitive impairment in the elderly.

CONCLUSIONS

The Environmental Health Criteria document for aluminium (WHO, 1997) concluded that:

> On the whole, the positive relationship between aluminium in drinking-water and AD, 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/litre, 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.

The degree of aluminium absorption depends on a number of parameters, such as the aluminium salt administered, pH (for aluminium speciation and solubility), bioavailability, and dietary factors. These should be taken into consideration during tissue dosimetry and response assessment. Thus, the use of currently available animal studies to develop a guideline value is not appropriate because of these specific toxicokinetic/dynamic considerations.

Owing to the uncertainty surrounding the human data and the limitations of the animal data as a model for humans, a health-based guideline value for aluminium cannot be derived at this time.

The beneficial effects of the use of aluminium as a coagulant in water treatment are recognized. Taking this into account and considering the potential health concerns (i.e. neurotoxicity) of aluminium, 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.

A number of 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 (Letterman & Driscoll, 1988; WRc, 1997). Under good operating conditions, concentrations of aluminium of 0.1 mg/litre 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, and small facilities often have limited resources and access to expertise to solve specific operational problems. For these small facilities, 0.2 mg/litre or less is a practicable level for aluminium in finished water (WRc, 1997). (see Acceptability aspects)

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