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EXPLORATORY REPORT ALUMINIUM AND ALUMINIUM COMPOUNDS

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Mailing list exploratory report Aluminium and Aluminium Compounds

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SUMMARY

This report contains general information on aluminium and aluminium compounds concerning the existing standards, emissions, exposure levels and effect levels. The document is to be considered as a first evaluation to be used for the national discussion during an exploratory meeting on integrated criteria documents.

In this report a difference is made between the risk of aluminium exposure related to acidification resulting into mobilization of (natural occurring) aluminium, and the risk related to the discharge of aluminium in our environment.

Acidification results, among others, into increased dissolved aluminium levels in surface water and soil. In most cases Dutch surface waters are sufficiently buffered and therefore acidification does not present a problem in terms of increased aluminium toxicity for aquatic ecosystems. In contrast to this, Dutch soils are vulnerable to acidification with regard to aluminium: in the process of leaching out first elevated dissolved aluminium levels at the root zone are harmful to terrestrial ecosystems and, subsequently, may reach levels in groundwater surpassing the current drinking water standards and guidelines.

As to emission of aluminium in the environment the figures show an increase of industrial emissions in the last decade. There are indications that current exposure levels may present a risk to both man and ecosystems.
Man may be at risk since a provisionally derived toxicological limit value of aluminium in air (0.05 μg Al.m^{-3}) is likely to be exceeded in ambient and indoor air in the Netherlands. However, exposure data are missing and the proposed toxicological limit value is rather conservative, introducing some uncertainty in the risk estimation. Based on the current provisional tolerable weekly intake as suggested by the JECFA and tentatively adopted by the RIVM, the risk of current exposure levels of aluminium to man is negligible. It must be emphasized that effects have been reported at lower doses, which was not included in the JECFA study implying the need for an evaluation update.
Aquatic life may be endangered in some surface waters, but effects on aquatic species are definitively expected to occur in the vicinity of emission sources. Bioaccumulation may lead to high aluminium content of macrophytes and insects and so, depending on the dietary intake of calcium and phosphoros, herbivorous and insectivorous species may be at risk. It is recommended to initiate activities to obtain further data needed for a more sound risk evaluation.

1. INTRODUCTION

This scoping report is part of the preparation for drawing up the integrated criteria document aluminium and aluminium compounds. The objective of this report is to bring the knowledge of the participants in the scoping meeting to the same level, and to put forward points for discussion and decision-making as to the contents of the integrated criteria document. It should be emphasized that the present report does not aim to be exhaustive: the actual standards and recommendations, the sources and exposure levels in the Netherlands are merely outlined, whereas on the other hand the principal effects and indicative (no) effect levels are described. Therefore all proposed toxicological limit values should be considered as provisional. Subsequently, problems will be pointed out and a proposal made as to the contents of the integrated criteria document aluminium and aluminium compounds to be drawn up.

Although aluminium phosphide is used as a pesticide in the Netherlands, it is not evaluated toxicologically in this document. Aluminium phosphide reacts with hydrogen, forming hydrogen phosphide, which is the toxic substance.

2. ACTUAL STANDARDS AND GUIDELINES

Table 2.1 gives an overview of the actual standards and guidelines in force in the Netherlands (Additievenboekje, 1988; Contaminantenboekje, 1991; NIA, 1991; Versteegh et al., 1992).

Table 2.1 Actual standards and guidelines for aluminium and aluminium compounds in the environment in the Netherlands

Environmental compartment/ type of standard	Concentration	Reference
AIR		
- indoor air (workspace)		
MAC aluminium chloride (free of water)	2 mg.m^{-3}	MAC (1989)
MAC aluminium nitrate (free of water)	2 mg.m^{-3}	MAC (1989)
MAC aluminium sulphate	2 mg.m^{-3}	MAC (1989)
MAC aluminium (pyrophorous)	5 mg.m^{-3}	MAC (1989)
MAC aluminium (powder)	10 mg.m^{-3}	MAC (1989)
MAC aluminium oxide	10 mg.m^{-3}	MAC (1989)
FOOD AND DRINKING WATER		
- food		
Provisional Tolerable Weekly Intake	7 mg.kg^{-1} b.w.	JEFCA (1988)
Total daily Intake (temporary)	0.6 mg.kg^{-1} b.w.	JEFCA (????)
- drinking water		
MAC	200 μg.l^{-1}	WLB (1984)
Signal value inspection*	30 μg.l^{-1}	????
Recommended value VEWIN	50 μg.l^{-1}	VEWIN (1985)
OTHER		
- chemical waste		
aluminium (powder/dust)** aluminium carbide (Al_4C_3)	50,000 mg.kg^{-1} d.w.	BACA (1991)

d.w. : dry weight
b.w. : body weight
* : for dialysis patients
** : metallic aluminium (not powder/dust) and aluminium oxides and hydroxides are not considered chemical waste

3. APPLICATIONS, SOURCES AND EMISSIONS

3.1 PRODUCTION

All current commercial aluminium manufacture is based on bauxite. This ore consists of several hydrous aluminium oxides. Bauxite contains 40-60% alumina (as Al_2O_3). The production of aluminium essentially proceeds in two steps: bauxite is refined using the Bayer process (a dissolving process) to yield gibbsite ($Al(OH)_3$), which is subsequently calcined and using the Hall-Héroult process (an electrolytical reduction process) turns into metallic aluminium (Quarles van Ufford and Ros, 1992; Smits et al., 1992).
Secondary aluminium production (from aluminium scrap) is relatively important because, amongst others, it takes approximately one tenth of the energy required for the production of the primary metal (Smits et al., 1992).

Data on the mining of bauxite and the production of aluminium in the Netherlands and world-wide are shown in table 3.1.

Table 3.1 Production of aluminium (in 1,000 ton.year^{-1}) (Smits et al., 1992)

Country/continent	Production			
	1985	1988	1990	Other
Netherlands				
Imported bauxite	157	142	154	-
Al production from imported bauxite	-	-	44	-
Primary Al production total	251	271	-	249 (1982)
Secondary Al production total	62	116	-	-
Total Al production	313	387	-	-
USA (1982)	-	-	-	3,248
USSR (1982)	-	-	-	2,381
Canada (1982)	-	-	-	1,056
Germany (1982)	-	-	-	780
World (1983)	-	-	-	13,908
World (2000) estimated	-	-	-	35,500

- : no data available

From table 3.1 it can be concluded that aluminium production in the Netherlands is increasing, especially the secondary production.

3.2 APPLICATIONS

Imports and exports of aluminium and aluminium products play an important role in the Netherlands. Data on imports and exports are shown in table 3.2.

Table 3.2 Imports and exports of aluminium and aluminium products for the Netherlands (1,000 ton.year^{-1}) (Smits et al., 1992)

Product	Import		Export	
	1988	1985	1988	1985
Bauxite	160	162	18	5
Old and new scrap	105	67	138	87
Semi-manufactures*	222	164	180	143
Chemicals**	55	43	-	-

- : no data available
* : including rods and sections, wires, plates, sheets, strips, foil (with or without coated with paper), tubes, cables bottle caps, powder and flakes, household articles and other manufactures
** : alumina hydrate and aluminium sulphate

Aluminium flows and aluminium available for consumption in the Netherlands can be calculated from production, import and export data. Aluminium flows are presented in table 3.3.

Table 3.3 Aluminium flows in the Netherlands in 1988 (1,000 ton.year^{-1}) (Smits et al., 1992)

Activity	Import	Export	Production	Total
Production (total)			387	+ 387
Unwrought aluminium	168	330		- 162
Semi-manufactures	223	180	0	+ 43
Available for consumption (1988)				+ 268
Available for consumption (1985)				+ 160

Table 3.4 gives an overview of the most important applications of aluminium and aluminium compounds in the Netherlands by end-users. Aluminium is used in the aircraft industry, building industry, chemical industry (paint pigments, cosmetics, medicines), food industry (additives), for packaging purposes and as flocculation agent in drinking water purification (Versteegh et al., 1992).

Table 3.4 Applications of aluminium and aluminium compounds in the Netherlands (end-uses) (Smits et al., 1992)

Application	1990 (1,000 ton.year^{-1})
Transport	48
Mechanical and electrical engineering	24
Building and construction	32
Chemical/agricultural use	10
Packaging	28
Household articles	8
Miscellaneous	24
Total consumption (1990)	189

3.3 EMISSIONS AND WASTE STREAMS

In the Netherlands aluminium and aluminium compounds are emitted to air and surface water. Interpreting the available data it must be kept in mind that aluminium is a major structural element in the earth's crust, and thus in the environmental compartments soil (7.1%) and water (see Chapter 4.1). The amounts of aluminium emitted are rather small compared with the amount of aluminium and aluminium compounds naturally present in the environment.

Table 3.5 presents an overview of the emissions of aluminium and aluminium compounds per industrial source to air and surface water in the Netherlands. No data on non-industrial emissions, like the use of aluminium compounds in pesticides, the aluminium emissions of traffic (and cigarettes), the use in sewage treatment plants, and the use for drinking water purification, are available.

Petroleum industry, chemical industry and metal/metallurgical industry are responsible for the major part of the emissions of aluminium and aluminium compounds. Most emission to water is directly to the surface water (approximately 85%). 15% of the aluminium is emitted indirectly via sewage systems.

The efficiency of sewage treatment plants in removing aluminium from the waste water varies between 30-85% (Van der Veer, pers. comm. 1992). If extra aluminium is added (for removal of phosphates), additional aluminium emissions to surface water will take place.

An overview of some emission factors and industrial emissions available from SPIN (Samenwerkingsproject Procesbeschrijvingen Industrie Nederland) is given in table 3.6. No emission factors on processes in the chemical industry are available. It must be noted, that data from SPIN and Emission Registration are difficult to compare. SPIN provides specific recent data on a few branches of industry, Emission Registration provides less specific emission data on all industry in the Netherlands.

Table 3.5 Industrial emissions of aluminium and aluminium compounds to air and water in the Netherlands (ton.year^{-1}) (Emission Registration, 1992)

Source	Emission to air 1985-1991	Emission to water 1985-1991
Food industry (starch, flour and beer)	0	11
Textile industry	0	5
Paper industry	0	64 (Al. Sil.)**
Petroleum industry	1,440 (Al. Sil.)**	0
Chemical industry	67	3,109
Rubber industry	0	0.2
Building materials and glass	6	0
Metal/metallurgical industry	1,098	1,212
Metal products industry	36	142
Machine industry	2	2
Electrotechnical industry	0.05	0
Automobile industry	16 (Al_2O_3)**	3
Glass trade	8	0
Wholesale trade	0.3	0
Public utilities	0	0.1
Trans shipment company	695 (Al_2O_3)**	0
All industries*	3,400	4,550

0 : emission 0 or negligible

* : measured as Al or Al-compounds (see table 3.7). This implicates that total industrial Al emission will be lower

** : predominant emitted Al compound

Table 3.6 Emission factors and industrial emissions of aluminium and aluminium compounds to air and water in the Netherlands (ton.year^{-1})

Source	Air/ Water	Emission factor	Emission
Iron foundries (Al. compounds)	a	40 g.ton^{-1} smelt	11 (1983)/ 5 (1985)[1]
Non ferrous foundries (Al_2O_3)	a	300 g.ton^{-1} product*	5 (1986)[2]
Blast furnaces (Hoogovens) (total)	a	1.3 g Al.ton^{-1} steel**	7 (1989)[2]
Blast furnaces (Hoogovens) (cinder)	a	0.75 g Al.ton^{-1} cinder***	3 (1989)[2]
Anodization ($AlPO_4$)	w	30 g.m^{-2}.yr^{-1}/10 ton.company^{-1}****	300 (1990)[3]
Secondary steel ind. (from scrap)	w	120 g.ton^{-1} steel	30 (1988)[2]
Electrolysis	w	261 g.ton^{-1}***** aluminium	71 (1988)[4]

\- : no data available

* : estimated 80 g.ton^{-1} product in future (1.3 ton.yr^{-1})

** : estimated 0.006 g.ton^{-1} steel in future (converted blast furnaces)

*** : estimated 0.19 g.ton^{-1} cinder in future

**** : estimated 1.5 g.m^{-2}.yr^{-1} or 0.5 ton.company^{-1} (in 2000) (15 ton.yr^{-1})

***** : estimated 210 g.ton^{-1} in future

1 : Eijssen et al., 1992

2 : SPIN, 1992

3 : Du Mortier and Ros, 1992

4 : Smits et al., 1992

In the provinces of Zuid-Holland (3,950 ton.year^{-1}), Zeeland (1,800 ton.year^{-1}) and Groningen (850 ton.year^{-1}) most aluminium and aluminium compounds are emitted (>80% of the total Al emissions in the Netherlands). This is caused by the presence of chemical industry and aluminium production industry (electrolysis) in these provinces.

Most aluminium compounds emitted to air are aluminium oxides and silicates, while aluminium emitted to water mainly consists of soluble aluminium ions and the insoluble aluminium oxides. More specific data are given in table 3.7.

Table 3.7 Industrial emissions of aluminium and aluminium compounds to air and water in the Netherlands (ton.year^{-1}) (Emission Registration, 1990; Emission Registration, 1992)

Compound	Air 1985-1991	1981-1984	Surface water 1985-1991	1981-84
Aluminium ion	-	-	1,618**	1,406**
Aluminium oxides	1,605	1,685	1,304**	1,135**
Aluminium silicates	1,464	647	68**	235**
Sodium aluminium silicate	5	0.43	-	-
Aluminium hydroxide	1	1	319**	410**
Aluminium fluoride	275	233	-	-
Aluminium chlorides	-	-	39**	39**
Aluminium sulphates	-	-	20**	23**
Aluminates	-	-	106**	108**
Aluminium compounds (soluble)	0.44**	0.65**	-	-
Corrosion products and alloys (as Al_2O_3)	4	4	-	-
Aluminium compounds (pyr. as Al_2O_3)	5	16	-	-
Aluminium compounds (inorganic, insoluble as Al_2O_3)	7.5	1.5	-	-
Aluminium and Al compounds	-	-	1,075**	96**
Bauxite	16	15	-	-
Aluminium compounds (total)*	3,400	2,600	4,550**	3,500**

- : no data available

* : probably more aluminium compounds are emitted annually than stated here, because aluminium may be present in other emitted compounds

** : registered as Al

Both industrial emissions to air and water have increased the past 10 years, mainly because of increased productivity. No general emission standards have been issued for aluminium in the Netherlands, emission standards for aluminium are sometimes incorporated in licences for individual companies (based on the Pollution of Surface Waters Act and the Water Management Act). At this moment, aluminium concentrations up to 50 mg.l^{-1} (directly to surface water) are allowed. Other companies (without a emission licence) are still not restricted in concentrations of aluminium in the waste water. The State Council has determined that industries must use best feasible techniques to reduce the amount of aluminium in waste water.

In Germany, general emission standards to water range from 2-3 mg.l^{-1} in the metal and galvanic industries, in Belgium emission standards range from 2-10 mg.l^{-1} (LAE, pers. comm. 1992; RWS, pers. comm. 1992).

Aluminium is an important substance of several waste streams in the Netherlands. Table 3.8 gives an overview of aluminium concentrations in some waste streams. No data on the total amount of aluminium present in waste in the Netherlands are available.

Table 3.8 Aluminium concentrations in waste streams in the Netherlands

Type of waste	Al content (% d.w.)	Reference
Fly-ash and bottom ash (electrical power plant)	13-16	Meij and Te Winkel, 1991
Waste incineration slag	6 (as Al_2O_3)	Anthonissen, 1991
Waste incineration fly-ash	6	Anthonissen, 1991
Masonry aggregates	4.4-4.5	Kamphuis and Meiling, 1991
Concrete aggregates	2.2-2.5	Kamphuis and Meiling, 1991
Jarosite	1 (as Al_2O_3)	Meijer, 1990; Matthijsen and Meijer, 1992
Aluminium production waste	17-41	Kliest et al., 1987; Van de Beek et al., 1987
Auto wrecks	4 (metal)	Vos and Meiling, 1991

d.w. : dry weight

No data on transfrontier emissions of aluminium are available. These emissions are not expected to be important compared with the aluminium available in the natural environment. In general, the aluminium content of the waste streams is even lower than that of natural soil.

Intercompartmental flows of aluminium are important. For example, deposition can be estimated at about 1,000 ton yearly (see section 4.3.2). Another important flow of aluminium takes place from the environmental compartment soil to groundwater (see Chapter 4.1). This is enhanced by acidification caused by human activities (agriculture, industry and traffic). This change in equilibrium between soil and groundwater may be a much more important anthropogenic effect than direct aluminium emissions.

3.4 TRENDS

Aluminium production increases annually in the Netherlands. This increase mainly results from the increase of the secondary aluminium production (of scrap). The amount of imported bauxite remained stable the past years. There are no indications that this amount will increase in the near future.

Because of the import, export and secondary production the aluminium flows in the Netherlands are rather complicated. Both import and export of aluminium scrap, semi-manufactures and chemicals increased the past years. The amount of aluminium available for application for end-uses (consumption) increased from 160,000 ton in 1985 to 268,000 ton in 1988. Total consumption of aluminium also increased during these years, but the available data are difficult to compare. During the period 1981-1984 total industrial emissions of aluminium and aluminium compounds were approximately 2,600 ton.year^{-1} to air and 3,500 ton.year^{-1} to water (Emission Registration, 1990). This has increased to approximately 3,400 ton.year^{-1} to air and 4,550 ton.year^{-1} in the period 1985-1990

(Emission Registration, 1990 and 1992). According to SPIN (1992), many industrial processes will emit less aluminium in the near future. Although productivity is expected to increase during the coming years, industrial aluminium emissions are likely to decrease because of emission reducing measures (e.g. use of converted blast furnaces for steel production, use of other raw materials in metallurgical and anodization industries, "good house keeping").
No data are available on non-industrial aluminium emissions in the Netherlands (e.g. agriculture and traffic).

4. OCCURRENCE, CONCENTRATIONS AND EXPOSURE

Aluminium is the third most common element and accounts for about 7.45% of the lithosphere (8.8% according to Contaminantenboekje (1991)). It does not occur in the free state in nature, but is found in combination with oxygen, silicon and fluorine in minerals making up igneous, metamorphic and sedimentary rocks (IARC, 1984; Smits et al., 1992).

4.1 SOIL AND GROUNDWATER

4.1.1 Soil

The average aluminium content in soils in the Netherlands is 71,300 $mg.kg^{-1}$ d.w. (7.1%) (Stuyfzand, 1991).

Mobilization of aluminium is one of the major acid neutralization processes in sandy forest soils in the Netherlands impacted by acid deposition. This Al buffer (extractable from pyrophosphate or oxalate) in the soil is limited, however. Model results show that, at current deposition levels, the Al buffer in the top 10 cm of most sandy forest soils will be depleted by between 10 and 100 years. When there is no longer an Al buffer, the pH in the soil solution may decline even further to a value of 2.8 to 2.9. Moreover, there may be increasing P deficiency owing to the formation of iron phosphates and phosphorus-aluminium compounds (Heij and Schneider ed., 1991).
The acidification of the soil has led to increasing Al concentrations in the root zone, which has reduced the availability of cations such as K and Mg to the roots. The current composition of the soil solution in the top soil of Dutch forests is characterized by Al/Ca ratios just above the critical value of 1.0 and Al concentrations far above the critical value of 2,000 $\mu g.l^{-1}$ (in the soil solution). The above-mentioned changes in the soil chemistry will be difficult to reverse, because Al hydroxides dissolve in the top layer of the soil profile and (to a small extent) are deposited lower in the soil profile (Heij and Schneider, 1991).

4.1.2 Groundwater

Table 4.1 shows aluminium concentrations in groundwater in the Netherlands. Aluminium concentrations range from <0.5 to 39,000 $\mu g.l^{-1}$. They are strongly correlated to degree of acidity (LMG, 1991; Stuyfzand, 1991 and 1992) and correlated with soil use, soil type and depth.

Table 4.1 Aluminium concentrations in groundwater in the Netherlands (in μg.l^{-1})

Location	Aver.	Min	Max	Reference
Netherlands (pH$\geq$6.2)	-	<0.5	300	Stuyfzand, 1991 and 1992
Netherlands (pH<6.2)	-	20	39,000	Stuyfzand, 1991 and 1992
Natural groundwater	-	<1	240	Stuyfzand, 1991 and 1992
Groundwater polluted by:				
- polluted air (soil contains much lime)	-	1	89	Stuyfzand, 1991 and 1992
- polluted air (soil contains little lime)	-	130	7,200	Stuyfzand, 1991 and 1992
- Rhine water	-	<0.5	100	Stuyfzand, 1991 and 1992
- liquid manure on maize	-	15	39,000	Stuyfzand, 1991 and 1992

- : no data available

Table 4.2 presents an overview of aluminium in groundwater per soil type and land-use system in the Netherlands.

Table 4.2 Aluminium concentrations in groundwater per soil type and land-use in the Netherlands in 1991 (in μg.l^{-1}) (LMG, 1991)

Soil type/ Land-use	Al. concentration at 5-15 m below ground level			Al. concentration 15-30 m below ground level		
	mean	min	max	mean	min	max
Clayey and peaty soils						
- all land-use-systems	125 19.2***	12.5*	4,600**	32 18.8***	12.5*	424
- pastures (grassland)	56	-	-	27	-	-
- agricultural fields	104	-	-	78	-	-
Sandy soils						
- pastures (grassland)	153	13.5*	1,920	78	13.5*	2,010
- agricultural fields	1,235	13.5*	13,430	279	13.5*	6,980
- nature reserves	1,241	16.7	12,625	404	13.5*	13,850

- : no data available
* : below detection limit
** : 10,320 μg.l^{-1} in 1990
*** : median

It must be noted that aluminium concentrations are higher below agricultural fields and nature reserves, than below pastures. Acidity below agricultural fields and nature reserves is also lower. At this moment there is no scientific explanation for this phenomenon.

Solubility of aluminium in (ground)water depends on many factors, of which acidity (pH) is the most important one. Other important factors are the availability of other metals, organic material (humus), the oxidation state of mineral components, the redox potential of the system and the water temperature

(Versteegh et al., 1992; WHO, 1984).

There are signs that Al will become a problem for the drinking water supply, particularly in the case of shallow private wells. The Al content of the shallow groundwater at 150 sites (forests and heathlands) is often above the drinking water standard (200 $\mu g.l^{-1}$) (at Vierlingsbeek 1,450 $\mu g.l^{-1}$ (Versteegh et al., 1992)). This is true for almost 90% of the coniferous forest sites investigated and for 70% of the deciduous forest sites (80% in forest and heathland according to LMG (1992)). It should be mentioned, however, that these data provide an indication of the quality trend of the shallow groundwater under forest areas. The water that is withdrawn at a pumping site comes from a particular area around the well field, called the recharge area, where, in general, various forms of soil use are to be found, as a result of which there will be mixing of various types of water. Furthermore, Al retention in the deeper subsoil has an important effect here.
If the acidification of the soil continues, the groundwater quality will further deteriorate owing to increasing nitrate and aluminium contents (Heij and Schneider, 1991).

4.2 SURFACE WATER AND SEDIMENT

4.2.1 Surface water

Aluminium concentrations in surface water are not monitored in the Netherlands. Table 4.3 gives an overview of some aluminium concentrations in surface water. The average concentration in fresh surface waters is a few hundreds $\mu g.l^{-1}$.

Table 4.3 Aluminium concentrations in surface water in the Netherlands (total, in $\mu g.l^{-1}$)

Location	Aver.	Min	Max	Reference
Meuse (Keizersveer, 1990)	600	190	2600	RIWA, 1990
Rhine (Lobith, 1991)	190	50	360	RIWA, 1991
Rhine (1990)	230	100	590	RWS, 1992
Rhine (1989)	270	170	500	Versteegh et al., 1992
Rhine (1988)	190	70	450	Versteegh et al., 1992
Hoogheemraadschap Rijnland (1977-1992)*	120- 1,200	30- 360	270- ('92) 18,000	Van der Veer, pers. comm. 1992
Sea	5	-	-	Stuyfzand, 1991 and 1992

- : no data available
* : highest concentrations are measured in inlet water, lowest in lakes

According to WHO (1984), industrial wastes, erosion, leaching of minerals and soils, contamination from atmospheric dust, and precipitation are the main pathways by which aluminium enters the aquatic environment. The natural aluminium concentrations in surface water vary between <1 to 3,500 $\mu g.l^{-1}$. Speciation of aluminium in surface water depends on acidity, temperature, the availability of other substances (like phosphates) and the oxidation state of the substances available (Van der Veer, pers. comm. 1992). So far no relationship has been found between decrease in phosphate emissions and aluminium concentrations. In acid circumstances ($pH<5$), Al^{3+} ions is the predominant form, in more alkaline circumstances ($pH>6$), the predominant form is $Al(OH)_4^-$. Aluminium compounds are precipitated out of solution or adsorbed on sediments (US FDA, 1973).
The level of aluminium in water varies considerably and may exceed 10,000 $\mu g.l^{-1}$ in the vicinity of aluminium processing plants (US FDA, 1973). Aluminium concentrations in the influent of sewage treatment plants in the Province Zuid-Holland are approximately 4,000 $\mu g.l^{-1}$, concentrations in the effluent vary between 280 and 1,120 $\mu g.l^{-1}$. Industrial effluents may contain much higher aluminium levels, for example those discharged into the Eems-Dollard estuary, ranging from 11 up to 865 mg $Al.l^{-1}$ (Van der Veer, pers. comm. 1992).

4.2.2 Sediment

No data on aluminium concentrations in sediment in the Netherlands are available. There are no indications that concentrations of aluminium and aluminium compounds in sediments will differ much from concentrations found in soils (approximately 7.1%).

4.3 AIR

4.3.1 Air (outdoor)

The aluminium content of outdoor air is not monitored in the Netherlands. In England, concentrations have been estimated at 0.05-0.5 $\mu g.m^{-3}$ in rural areas, and at 0.1 and 5 $\mu g.m^{-3}$ in urban areas (Versteegh et al., 1992). According to WHO (1989), in industrial areas concentrations up to 6.2 $mg.m^{-3}$ have been reported.

4.3.2 Rainwater

Few data on aluminium levels in rainwater in the Netherlands are available. Stuyfzand (1991) found an average aluminium concentration of about 100 $\mu g.l^{-1}$ in the Dutch coastal provinces. Hoogheemraadschap Rijnland has also reported average aluminium concentrations in rainwater of approximately 100 $\mu g.l^{-1}$ (Van der Veer, pers. comm. 1992). These concentrations are lower than average concentrations present in surface water and groundwater.
A combined RIVM/ECN study from 1983 showed average aluminium depositions in the Netherlands of 23 $kg.km^{-2}$ per annum (Van der Veer, pers. comm. 1992), resulting in a total annual aluminium deposition in the Netherlands of approximately 900 tonnes.

4.3.3 Air (indoor)

Except for some data on occupational exposure, no data are available on the aluminium content of indoor air in the Netherlands and world-wide. An important source of aluminium in indoor air could be cigarette smoke.

In "potrooms" in aluminium reduction plants (in Poland) alumina (Al_2O_3) concentrations in air of 1 to 148 $mg.m^{-3}$ (personal samples) have been found. Air samples led to total aluminium concentrations between non-detectable to 2.85 $mg.m^{-3}$ (IARC, 1984).

4.4 FOOD AND DRINKING WATER

4.4.1 Food

Table 4.4 gives an overview of aluminium concentrations in some foodstuffs and beverages.

Table 4.4 Aluminium concentrations in food and beverages.

Food/beverage	Concentration ($mg.kg^{-1}$)	Reference
24-hour diet	3.8-42.2 d.w. (median 9.1)	Ellen and Van Loon, 1986
24-hour diet	0.6-7.8 w.w. (median 1.7)	Ellen and Van Loon, 1986
Cereals	0.03-42 w.w. (up to 68 d.w.)	RIKILT, 1985
Vegetables	0.01-32 (up to 250 d.w.)	RIKILT, 1985
Fruit	0.01-35 w.w.	RIKILT, 1985
Spices and herbs	0.59-700 d.w.	RIKILT, 1985
Nuts	<2 w.w.	RIKILT, 1985
Sugar	0.85-5.29 w.w.	RIKILT, 1985
Honey	4.76-59 w.w.	RIKILT, 1985
Coffee	<0.4 w.w.	RIKILT, 1985
Coffee	11 d.w.	RIKILT, 1985
Tea	2.8 w.w.	RIKILT, 1985
Tea leaves	2,800 d.w. (100-17,000)	RIKILT, 1985; US FDA, 1973
Chocolate syrup	<2 w.w.	RIKILT, 1985
Cacao	45 d.w.	RIKILT, 1985
Mineral water	0-1.3 w.w. (median 0.06)	Contaminantenboekje, 1991
Beer and wine	0.06-29 (in al. tin) w.w.	RIKILT, 1985
Milk and diary products	0.03-19 w.w.	RIKILT, 1985
Milk	73-81 d.w.	RIKILT, 1985
Cheese	up to 695 w.w.	RIKILT, 1985
Eggs	0.2-1.4 w.w.	RIKILT, 1985
Meat and poultry	0.4-11.2 w.w. (up to 68 d.w.)	RIKILT, 1985
Fishery products	0.7-60 w.w.	RIKILT, 1985

w.w. : wet weight or fresh weight
d.w. : dry weight

Aluminium (E 173; Cl 77000: aluminium ammonium sulphate, aluminium potassium sulphate, aluminium sodium sulphate and aluminium sulphate) is permitted as an additive (preservatives, fillers, colouring agents, anti-caking agents, emulsifiers and baking powders). In the Netherlands permission is granted by the Warenwet for use of aluminium sodium sulphate in pasteurized egg-white (maximum concentration 30 mg $Al.kg^{-1}$ d.w.) (Additievenboekje, 1988). Furthermore, a number of aluminium containing minerals, like bentonite, are used in the preparation of beers, cider, wine and animal fodder (RIKILT, 1985).

Analysis of 101 duplicates of 24-hour diet, collected in 1978, led to aluminium intakes varying between 1.4 and 33.3 $mg.day^{-1}$ (average 4.6 $mg.day^{-1}$, median 4.0 $mg.day^{-1}$). In a similar study with 110 duplicates in 1984/1985) daily intakes of 0.6-12.9 mg were found (average 3.1 mg, median 2.8 mg). These values are similar to those found in Switzerland, England and the USA. Other, much higher, values (up to 240 $mg.day^{-1}$) are probably influenced by analytical problems.

In the Dutch studies, preparation of food in aluminium containing cooking utensils did not have a major influence on the results (Ellen and Van Loon, 1986; Ellen et al., 1988). Greger et al. (1985: cited in Ellen and Van Loon, 1986) found that the aluminium content of products (especially sour products like spinach, tomatoes and tomato puree, rhubarb, citrus fruits and applesauce) increases if prepared in aluminium pans (especially new ones). Other possible aluminium sources in food are coffee-percolators and aluminium foil. It is concluded that the daily intake of aluminium by the use of aluminium containing cooking utensils and food packaging material is a few additional mg per day (RIKILT, 1985; Versteegh et al., 1992).

4.4.2 Drinking water

Table 4.5 gives an overview of aluminium concentrations in drinking water in the Netherlands and world-wide.

Elevated aluminium concentrations in drinking water may occur because of the application of aluminium salts as a coagulant in the treatment of drinking water. Another additional source may be the addition of lime (contaminated with aluminium). In shallow groundwater, high levels have been found (see Chapter 4.1.2). It must be noted that no relationship between the aluminium content of the drinking water and that of groundwater as a raw water source has been found in the Netherlands (Versteegh et al., 1992).
Using aluminium as a coagulant aluminium concentrations in drinking water are higher than those where other coagulants are used. In the Netherlands mostly iron salts are used as coagulants (Versteegh et al, 1992).

Table 4.5 Aluminium concentrations in drinking water in the Netherlands and world-wide (in $\mu g.l^{-1}$)

Location	Average	Min	Max	Reference
Netherlands (1991) (n=41)*	1.9 (median) 14.3 (average)	<0.8	156.9	Versteegh et al., 1992
Netherlands (1983) (n=37)*	4.6 (median) 16.0 (average)	2.2	107.1	Versteegh et al., 1992
Netherlands (1977-1978)	5-6	<2	11	RIKILT, 1985
England and Wales				
* Al salts as coagulant	20-730	<10	>730	Versteegh et al., 1992
* Other coagulants	10-230	<10	340	
Other European countries				
* Al salts as coagulant	15-170	<1	330	Versteegh et al., 1992
* Other coagulants	4-16	<4	40	
Europe and USA	-	<1	760	RIKILT, 1985
Maximum Permissible Concentration	200			WLB, 1984

- : no data available

* : non-representative selection. Only pumping stations with more than 2 μg Al.l^{-1} are selected

4.5 HUMAN EXPOSURE LEVELS

Man takes in aluminium and aluminium compounds from exposure to different environmental compartments. Intake by inhalation seems to be negligible compared to dietary intake by food and drinking water (approximately 95% respectively 5% of total intake (Versteegh et al., 1992)).

The average dietary intake is approximately 4 mg.day^{-1} in the Netherlands (Ellen and Van Loon, 1986; Ellen et al., 1988). Assuming a concentration of 15 $\mu g.l^{-1}$ (see Chapter 4.2.2), a daily consumption of drinking water would result in a daily aluminium intake of 0.03 mg. Aluminium uptake is expected to be much lower (see Chapter 5.1.1). However, the relationship between intake and uptake is not completely clear yet, depending on which chemical form is taken in. There might also be differences in aluminium uptake between food and drinking water.

Although the daily intake of aluminium by inhalation is small (e.g. 0.0008-0.0031 mg in Great-Britain and 0.0042 mg in Ontario Canada; Versteegh et al., 1992), in industrial areas intakes could reach 0.125 mg.day^{-1} (WHO, 1989).

Aluminium is present in cigarette smoke, but no data on aluminium concentrations and daily intake by smoking are available.

Aluminium containing medications and cosmetics may be a significant source of exposure for some individuals.

5. EFFECTS

There are no indications that aluminium is an essential element for plants and animals (including humans) (Ellen and Van Loon, 1986).

5.1. HUMAN TOXICITY

According to ECETOC (1991), effects on humans of aluminium and aluminium compounds have been evaluated by the following groups:

- ACGIH (American Conference of Governmental Industrial Hygienists) (1986);
- ATSDR SARA (Agency for Toxic Substances and Disease Registry, Superfund Amendments and Reauthorization Act) (1990);
- BG (Berufsgenossenschaft der Chemischen Industrie (listed);
- EEC (Commission of the European Communities);
- EPA CHIPS (Environmental Protection Agency, Chemical Hazard Information Profiles);
- IARC (International Agency for Research on Cancer) (1987);
- MAK (Senatskommission zur Prüfung Gesundheidschädlicher Arbeitsstoffe der Deutschen Forschungsgemeinschaft) (1986);
- WHO IPCS/EHC (World Health Organization, International Programme on Chemical Safety, Environmental Health Criteria (listed);

This Chapter is mainly based on ATSDR SARA (1992), CEC (1991), SDWC (1982), US FDA (1973), WHO (1977), WHO (1984) and WHO (1989).

5.1.1 Chemobiokinetics and metabolism

The dynamics of absorption, distribution, and excretion of aluminium are poorly understood. Furthermore, little is known about its metabolism or the factor that determines burdens of aluminium in specific tissues. This is particularly due to a lack of detection methodology and the universal contamination of laboratory reagents and chemicals with the metal (SDWC, 1982).

Absorption

* Inhalation
No quantitative data are available on inhalatory absorption. However, in an occupational study it was shown that workers in the electrolytic production of aluminium had higher concentrations of aluminium in urine than referents (IARC, 1984). Inhalatory absorption seems to be of minor importance because of relatively low aluminium concentrations in air. However, the relatively high aluminium concentrations in the lungs (see distribution) indicate a possible retention of aluminium in the lungs.

* Ingestion
As a result of the formation of insoluble aluminium phosphate ($AlPO_4$) in the gastrointestinal tract, only a minor amount of orally administered aluminium salts

is absorbed (Jones, 1938 and Kirsner, 1943 (both cited in WHO 1989)). According to Brusewitz (1984: cited in WHO, 1989), the percentage of net absorption of dietary aluminium may be in the order of 1% of the administered dose and at very high doses the uptake mechanisms may become saturated.
Other studies (Kaehny et al., 1977 (cited in SDWC, 1982)) however have shown that aluminium is readily absorbed from the gastrointestinal tract by normal persons who consume one of several aluminium salts (e.g. hydroxide or carbonate) or dihydroxy aluminium aminoacetate, but not aluminium phosphate. This can, however, be doubted, because phosphate is ubiquitous present in the body and other salts will ultimately result in $AlPO_4$.
In dialysis patients taking antacids (orally), net gastrointestinal absorption of aluminium of approximately 10 % (3%-28%) was found. Maximum absorption is higher in humans with renal failure, compared with controls (corrected for lower elimination) (Clarkson et al., 1972 and Cam et al., 1975 (cited in SDWC, 1982)). Indications are found that increasing levels of parathyroid hormone enhances the absorption of aluminium. Absorption is also influenced by phosphate and fluoride content, acidity, solubility, vitamin D and aluminium chelators (WHO, 1989).

* Skin absorption
No data are available on skin absorption of aluminium and aluminium compounds. Because of the extensive use of aluminium compounds in dermal medications and cosmetics, opportunities for skin exposure to humans cannot be excluded.

Distribution
The human body burden of aluminium is estimated to range from 50-150 mg. Aluminium appears to bind to serum proteins. In man, 95% of aluminium in blood is bound to plasma proteins (50% molecular weight greater than 8,000) (SDWC, 1982).
Following absorption, aluminium distributes to nearly all the organs including the brain (SDWC, 1982). In healthy human tissues from the United Kingdom, Al concentrations were usually below 0.5 $mg.kg^{-1}$ wet weight. Higher levels were observed in liver (2.6 $mg.kg^{-1}$ w.w.), lung (18.2 $mg.kg^{-1}$ w.w.), lymph nodes (32.5 $mg.kg^{-1}$ w.w.) and bone (73.4 $mg.kg^{-1}$ of ash) (Hamilton et al., 1972 (cited in WHO, 1989)). Concentrations in the brain are lower, this because of the blood-brain barrier. People suffering from Alzheimer's disease, dialysis encephalopathy syndrome and progressive encephalopathy were found to have high aluminium concentrations in brain tissue 0.4-107 $mg.kg^{-1}$ d.w. (SDWC, 1982; WHO, 1989). It must be noted however, that McDermott et al. (1978: cited in SDWC, 1982) did not find significant differences in aluminium content of brain samples between patients suffering from Alzheimer's disease and healthy, age-matched controls.
The Al concentration in human tissues from different geographic regions was found to be widely scattered, and probably reflected the geochemical environment of the individuals and of locally grown food products (Tipton and Cook, 1965 (cited in WHO, 1989)).
Increasing levels of parathyroid hormone may exert specific effects on the distribution of aluminium (SDWC, 1982; WHO, 1989).

Biotransformation

No data on biotransformation of aluminium and aluminium compounds in animals and humans are available.

Elimination

In both animals and humans, excretion of ingested aluminium mainly takes place via faeces, this probably because of the poor absorption. However, urinary aluminium concentration is also increased after aluminium treatment. Skalsky and Carchman (1983: cited in WHO, 1989) found that although 75-90% of an oral dose of aluminium was reported to be excreted in the faeces, urinary excretion is the major excretion route once aluminium has entered the blood stream.

5.1.2 Toxicity

Acute toxicity (single and short-term exposure): animal data

No data on lethal concentrations in case of inhalatory exposure of aluminium and aluminium compounds are available. Intratracheal instillation of aluminium salts or metallic aluminium powder has produced pulmonary fibroses (Stacey et al. (cited in SDWC, 1982)).

Lethal concentrations of aluminium compounds in case of oral and intraperitoneal application are shown in table 5.1. LD_{50}s vary between 6.3 and 980 mg $Al.kg^{-1}$ body weight, depending on compound, test species and route of entry. Severe aluminium intoxication following parenteral or oral administration of aluminium hydroxide, chloride, or sulphate is characterized by lethargy, anorexia, or death (Berlyne et al., 1972 (cited in SDWC, 1982)). Other symptoms are serious lesions of the digestive tract, ovarian lesions and reproductive failure (WHO, 1987 and 1989). After intraperitoneal administration, fibrotic peritonitis was found (SDWC, 1982).

Acute toxicity (single and short-term exposure): human data

No quantitative data are available on lethal concentrations of aluminium and aluminium compounds in case of inhalatory, oral and dermal exposure.

Following large oral doses of aluminium, toxic syndromes involve gastrointestinal tract irritation and, eventually, interference with phosphate absorption, which results in rickets (Casarett and Doull, 1977 (cited in SDWC, 1982)).

Table 5.1 Acute toxicity studies: oral and intraperitoneal exposure (SDWC, 1982; US FDA, 1973; WHO, 1989)

Compound	LD_{50} (average) ($mg.kg^{-1}$ bw)	LD_{50} (average) expressed as Al ($mg.kg^{-1}$ bw)	References
Oral exposure:			
Mouse			
$AlCl_3$	3,800	710^{*} or 780^{****}	Ondreicka et al., 1966
$Al_2(SO_4)_3$	6,200	980^{*}	Ondreicka et al., 1966
$Al_2(SO_4)_3$	-	970	Christensen, 1971
Rat			
$AlCl_3$	1,100	210^{*}	Berlyne et al., 1972
$AlCl_3$	-	380	Krasovskii et al., 1979
$AlCl_3$	3,700	690^{*} or 757^{****}	Spector, 1956
$Al(NO_3)_3(9H_2O)$	4,280	310^{*} or 543^{****}	Spector, 1956
$Al_2(SO_4)_{3*}^{*}$	1,500	240^{*}	Berlyne et al., 1972
Guinea pig			
$AlCl_3$	-	400	Krasovskii et al., 1979
Rabbit			
$AlCl_3$	-	400	Krasovskii et al., 1979
Intraperitoneal application:			
Mouse			
$Al(NO_3)_3(9H_2O)$	320	23^{*} or 37^{****}	Hart and Adamson, 1971
$Al_2(SO_4)_3$	-	6.3	Bienvenu et al., 1963
Rat			
$AlCl_3$	1,500	280^{*}	Berlyne et al., 1972
$Al(NO_3)_3(9H_2O)$	320	23^{*} or 37^{****}	Hart and Adamson, 1971
$Al(OH)_3^{***}$	1,100	380^{*}	Berlyne et al., 1972
$Al_2(SO_4)_3^{**}$	1,100	170	Berlyne et al., 1972

- : no data available
* : calculated
** : speciation according to WHO (1989) as $AlSO_4$
*** : speciation according to WHO (1989) as AlOH
**** : data cited from SDWC (1982)

Subacute and (sub)chronic toxicity (long-term exposure): animal data
An overview of NO(A)ECs and NO(A)ELs of aluminium and aluminium compounds is given in table 5.2.

Exposure by inhalation
Few data are available on subacute and (sub)chronic toxicity effects of inhalatory exposure to aluminium compounds (ATSDR SARA, 1992). NO(A)EC-values for local irritative effects were found of 0.05 mg $Al.m^{-3}$. Lowest LO(A)EL-values vary between 0.36 and 0.5 mg $Al.m^{-3}$. The effects reported by Finelli et al (1981: cited in ATSDR SARA, 1992), increased lysozyme levels resulting from damaged pulmonary alveolar macrophages, increase in protein levels in the lavage fluid and increase in alkaline phosphatase activity are often considered to be an adaptive response to many types of dusts.

Oral exposure

Few data on the long-term toxicity effects of oral exposure to aluminium and aluminium compounds are available. Lowest NO(A)EL-values for toxicity effects vary between 0.25-110 mg Al.kg^{-1} body weight per day. Lowest LO(A)EL-values vary between 0.5-190 mg Al.kg^{-1} body weight per day. In other studies (than given in table 5.2) much higher NO(A)EL- and LO(A)EL-values have been found.

Sodium aluminium phosphate (acidic) was administered to beagle dogs for 189 consecutive days at dietary levels of 0, 0.3, 1.0 or 3% (110 mg Al.kg^{-1} body weight per day). Each dose group consisted of 6 males and 6 females. The food intake of all female test groups were sporadically lower than the control group, but no statistically significant differences in weekly mean body weights were evident between male and female test groups and their respective controls. There were no treatment-related effects seen in blood chemistry, haematology, urinalyis, or in ophtalmic and physical examinations. Gross necropsy and micropathological findings did not show any significant toxicological effects. There was very mild renal tubular mineralization in both test and control groups with no significant difference in the frequency of occurrence of the degree of mineralization in the various groups (Katz, 1981 (cited in WHO, 1989)).

In a study in which dogs (4/sex/dose) were fed diets containing basic sodium aluminium phosphate (4, 10, 27, and 75 mg Al.kg^{-1} body weight per day for males and 3, 10, 22, and 80 mg.kg^{-1} for females) for 26 weeks, mild histopathological changes were observed in the liver, kidney, and testes of highest-dose males, whereas brain aluminium concentrations were slightly elevated in highest-dose females. No effects were noted at the lower dose levels (Pettersen et al., 1988 (cited in WHO, 1993 in press)).

Schroeder and Mitchener (1975a: cited in SDWC, 1982; WHO, 1977 and 1989) exposed weanling male and female Long-Evans rats (52 of each sex) to aluminium as aluminium potassium sulphate salt in concentrations of 5 mg.l^{-1} (0.5 mg.kg^{-1} body weight) (single dose study) in drinking water over the lifetime (1064±20 days) of the animals. According to SDWC (1982) males fed aluminium grew significantly heavier, but WHO (1977 and 1989) states that no effects were found on body weight. The weight of the females were similar to those of the controls. No effects were found on average heart weight, glucose, cholesterol and uric acid level in serum, protein and glucose content and pH of urine. The life span was not affected.

No adverse effects on body weight and longevity were observed in mice (54 of each sex per group, Charles River CD strain) receiving additionally 0 or 5 mg.l^{-1} (0.5 mg.kg^{-1}) body weight aluminium (as aluminium potassium sulphate during lifetime (936±49 days: single dose study). No details on histopathology are available (Schroeder and Mitchener, 1975b (cited in WHO 1977 and 1989)).

The results of both Schroeder and Mitchener studies cannot be used in establishing limit values, because of the fact that only one dose was tested.

Rats given oral doses of 0.0025, 0.25 and 2.5 Al mg.kg^{-1} body weight for 6 months (application not stated) exhibited some effects on behaviour and mild changes in the biochemistry of the testes at the highest dose level but there were no significant effects at the lower doses (Krasovskii et al., 1979). The reliability of this russian study is considered uncertain.

Neurotoxic effects have been demonstrated in rats, rabbits, cats and monkeys following systemic administration (subcutaneously, per os or directly into the central nervous system). The major pathological lesion is neurofibrillar degeneration, which is to some extent similar but not identical to that observed in Alzheimer's disease (SDWC, 1982). Other neurological syndromes produced by aluminium include that of epilepsy.

Subacute and (sub)chronic toxicity (long-term exposure): human data

Exposure by inhalation

Few data on subacute and (sub)chronic inhalatory exposure in humans are available from occupational studies.

Industrial exposure to high concentrations of aluminium-containing airborne dusts has resulted in a number of cases of occupational pneumoconiosis (Norseth, 1979 and Sorenson et al., 1974 (both cited in SDWC, 1982)). Most of these exposures were chronic, but other substances were involved in nearly all instances. Silicosis, aluminosis, aluminium lung, and bauxite pneumoconiosis are the result of pulmonary fibrotic reactions to silica and aluminium-containing compounds, which have been observed in the lung tissue in humans (Sorenson et al., 1974 (cited in SDWC, 1982)). Paradoxally, aluminium powder has been used in the prevention and therapy of silicosis (Casarett and Doull, 1977 (cited in SDWC, 1982)). There is however no unequivocable evidence that the procedure is clinically effective (Sorenson et al., 1974 (cited in SDWC, 1982)).

An aluminium-ball-mill worker died with encephalopathy and pulmonary fibrosis. After having been exposed to aluminium-containing compounds for more than 13 years, the concentration of aluminium in his brain was 20 times greater than that in the brain of controls (McLaughlin et al., 1962 (cited in SDWC, 1982)). This worker has also been exposed occupationally to other substances (particular matter from aluminium production plants only consists for a small part of aluminium (compounds).

Table 5.2 Subacute, subchronic and chronic toxicity no-observed-(adverse)-effect-concentrations (mg Al.m^{-3}) and no-observed-(adverse)-effect-levels (in mg Al.kg^{-1} body weight.day^{-1}) of aluminium and aluminium compounds (ATDSR SARA, 1992; SDWC, 1982; US FDA, 1973; WHO, 1977; WHO, 1989; WHO, 1993 in press)

Test species	Exposure time	NO(A)EL/LO(A)EL		Effects	References
inhalatory exposure					
AlF_3					
Rat	5m (dust)		0.42	Increased lysozyme levels	1
$AlCl_3$ (hydrated)					
Rat	6m, 5d.w^{-1}, 6h.d^{-1}	0.05	0.5	Lung nodules, no hepatic, renal, cardio, haemato, gastro and musc/skelet. effects	2
Rat	5m (dust)		0.36	Increased lysozyme levels, increase in in levels in the lavage fluid, and in alkaline phosphatase activity	1
Guinea pig	6m, 5d.w^{-1}, 6h.d^{-1}	0.05	0.5	Lung nodules, no hepatic, renal, cardio, haemato, gastro and musc/skelet. effects	2
oral exposure					
Al					
Rat	6m (?)	0.25	2.5	Some effects on behaviour, mild changes in biochemistry of testes	3
$AlCl_3$					
Mouse	6-12m (dw)		19	No changes in growth, reproduction, retarded growth in 2nd and 3rd generation litter. No pathological changes in liver, spleen and kidney (see developmental toxicity)	4
Mouse	long-term (?)		19*	Retarded growth and disturbances of phosphate and carbohydrate metabolism	5
Rat	18d (?)		190*	Decrease in liver glycogen and coenzyme A	6
Rat	6-12m (?)		28*	Negative phosphorus balance; decreased incorporation of ^{32}P into phospholipids; decrease in ATP	4
$Al(NO_3)_3(9H_2O)$					
Rat	1m (dw)	54*	108*	Less urine excretion, higher tissue Al concentration than controls	7
Rat	3m (dw)		41*	Significant Al accumulation in tissues; No toxic effects	8
$AlK(SO_4)_2(12H_2O)$					
Mouse	life-time (dw)	0.5		No adverse effects on body weight and longevity. No details on histopathology	9
Rat	life-time (dw)		0.5	Increased bodyweight (males)	9
Sodium aluminium phosphate (acidic)					
Dog	189d (d)	110		No adverse effects on body weight, blood chemistry, haematology, urinalysis, ophtalmic and physics. No histopathologic effects	10
Sodium aluminium phosphate (basic)					
Dog	26w (d)	27 (m)	75 (m)	Mild histopath. changes liver/kidney/testes	11
		22 (f)	80 (f)	Elevated brain Al concentrations	11

*:calculated; (dw):drinking water; (d):diet; (?): application not stated; (m):male; (f):female
ref: 1:Finelli et al.,1881; 2:Steinhagen et al.,1978; 3:Krasovski et al.,1979; 4:Ondreicka et al.,1966; 5:Sorenson et al.,1984; 6:Kortus,1967; 7:Gomez et al.,1986; 8:Llobet and Domingo,1985; 9:Schroeder and Mitchener,1975a,b; 10:Katz,1981; 11:Petterson et al.,1988)

Oral exposure

Aluminium deposition in the brain has been implicated as an etiologic factor in two neurologic disorders: Alzheimer's disease and chronic renal failure accompanied by senile dementia (SDWC, 1982).

Alzheimer's disease usually occurs in humans after the age of 40. It is a slowly progressive, fatal encephalopathy associated with behavioral alterations, memory disturbances, spacial disorientation, agnosia, dysphasia, and seizures. Numerous "causes" have been proposed for Alzheimer's disease. The disease is mainly genetically (autosomal dominant) determined (Farrar et al., 1989 (cited in Versteegh et al., 1992)). A causal role of aluminium in the onset of Alzheimer's disease is one of the theories.

In Norway and Great-Britain three epidemiological studies have been performed on the relationship between Alzheimer's disease and the exposure to aluminium. A weak relationship between the occurrence of Alzheimer's disease and the aluminium content has been found. In Great-Britain a geographical relation was found between Alzheimer's disease and aluminium in drinking water. In areas with an drinking water aluminium content $> 10\ \mu g.l^{-1}$, the incidence of Alzheimer's disease was approximately 50% higher than in areas with $< 10\ \mu g.l^{-1}$ (a relative risk of 1.5). No dose-effect relationship was established within the concentration intervals (0.02-0.04; 0.05-0.07; 0.08-0.11; $> 0.11\ mg.l^{-1}$) (Martyn et al., 1989 (cited in Versteegh et al., 1992)). Confounding factors have not been taken into account. Still the intake of aluminium by drinking water is relatively small compared with the intake of food. Furthermore, many types of dementia have been classified as Alzheimer's disease, not always correct. This leads to many doubts on the validity of these studies.

Another encephalopatic syndrome in which aluminium has been suggested as an etiologic agent has been described as "dialysis encephalopathy" or "dialysis dementia", which is a relentlessly progressive form of dementia observed in chronic dialysis patients. This disorder is characterized by an insidious onset of altered behaviour, speech disturbances, dyspraxia, tremor, myoclonus, convulsions, personality changes, psychoses, and effects on bones and muscles, resulting in death within approximately 6 to 7 months. The majority of the patients in whom this syndrome developed had been on intermittent haemodialysis for 3 to 7 years before the onset of symptoms. All had routinely received aluminium-containing antacids for the purpose of binding gastrointestinal phosphates for at least 2 years. Patients dying of the syndrome had significantly higher tissue concentrations of aluminium in their bones, skeletal muscles and grey matter of the brain (SDWC, 1982). A major etiologic factor associated with this syndrome is untreated aluminium containing tap water that is used to prepare the dialysis fluid.

One outbreak of "dialysis encephalopathy" occurring in Chicago between September 1972 and January 1976 affected 20 patients who had been maintained on long-term haemodialysis (Dunea et al., 1978 (cited in SDWC, 1982)). The cases of dementia coincided with peaks in the aluminium content of tap water (300-400 $\mu g.l^{-1}$ instead of 0-150 $\mu g.l^{-1}$) caused by a change in drinking water treatment. The other constituents of the water were not significantly altered.

Reproductive (and developmental) toxicity: animal data

Exposure by inhalation

No animal data on reproductive (and developmental) toxicity of aluminium and aluminium compounds in case of inhalatory exposure are available.

Oral exposure

Ondreicka et al. (1966: cited in US FDA, 1973) studied the effects of aluminium chloride on the growth rate of three generations of mice. Ten white mice were treated with an average 19.3 mg $Al.kg^{-1}$ body weight per day via drinking water for 180-390 days. There were 10 controls. The 4-week old weanlings were treated in the same way as their parents. Two generations litters (2nd and 3rd) showed growth impairments. No influence on the number of litters or offspring was reported. No histopathological abnormalities were observed in liver, spleen and kidneys. There was no significant difference in the erythrocyte count or haemoglobin level between the first and last generation and the controls (US FDA, 1973).

A study in which mice were fed diets containing aluminium treated bread (1.3-4.4% Al in the bread: considered as a very large dose) for a period of 4 months led to a decreased number of offspring and an increased mortality of offspring during the first week of life. Ovaries of these animals contained a large number of arthritic follicles, and were greatly reduced in size (Schaeffer et al., 1928 (cited in US FDA, 1973; WHO, 1989)). Furthermore, at these concentrations serious lesions on the digestive tract developed.

Cramer et al. (1986: cited in WHO, 1989) found that in mice the number of resorptions was increased in all dams given aluminium (oral gavage: 200 or 300 mg $AlCl_3.kg^{-1}$ body weight per day during days 7-16 of gestation) in a dose dependent manner (no statistics were performed). No effects on aluminium content of maternal liver or fetal or placental weight were found.

Pregnant rats were administered 40-200 $mg.kg^{-1}$ body weight aluminium chloride intraperitoneally on either gestational days 9, 13, 9-13 or 14-18. Treatment with 100-200 mg $AlCl_3.kg^{-1}$ body weight caused a dose-related increased incidence of maternal deaths; doses of 75-200 $mg.kg^{-1}$ resulted in extensive liver damage. The incidence of congenital abnormalities (poor ossification and skeletal defects) (further details not available) was higher in treated animals (Benett et al., 1974 (cited in WHO, 1977)).

Domingo et al. (1987: cited in WHO, 1989) dosed rats aluminium nitrate (0, 180, 360 and 720 $mg.kg^{-1}$ bw for 60 days (males) or 14 days (females)) intragastrically. Only effects were seen in the highest dose group (decrease in number corpea lutea, higher number of dead young/litter on days 1, 4 and 21 of lactation, decreased initial body weight).

In other studies (Lymann and Scott, 1930; Scott and Helz, 1932; McCollum et al. (all cited in FDA, 1973 and WHO, 1989); McCormack et al., 1979 (cited in SDWC, 1982)), no reproduction effects were found.
According to Léonard et al. (review: 1988), studies performed on mammals have found that aluminium compounds may be embryotoxic at concentrations likely to cause maternal toxicity (dependant on route of administration and stage of gestation).

Administration of aluminium lactate (oral, subcutaneous) to lactating animals (mice, rabbits) did not result in significant effects on pups (except for effects directly resulting from maternal toxicity) (several studies cited in WHO, 1989). Less than 2% of injected aluminium was found in the milk.

Food and Drug Research laboratories (1973: cited in WHO, 1989) found that orally administered sodium aluminium sulphate was not teratogenic in mice and rabbits at levels up to 352 mg.kg^{-1} respectively 191 mg.kg^{-1} bw.day^{-1}. Sodium silicoaluminate showed no evidence of teratogenicity in mice, rats and rabbits after oral administration at levels up to 1,600 mg.kg^{-1} bw.day^{-1}. On the basis of these studies there was no evidence for teratogenicity.

Reproductive (and developmental) toxicity: human data
Observations on a small number (22) of women using aluminium-containing antacids during pregnancy suggest that such treatment does not cause hyperaluminiumemia in newborns (Weberg et al., 1985 (cited in Léonard et al., 1988)).
No human data on reproductive (and developmental) toxicity of aluminium and aluminium compounds in case of inhalatory or dermal exposure are available.

5.1.3 Genotoxicity and carcinogenicity

Genotoxicity
Negative results were obtained in the *Salmonella typhimurium* reverse mutation assay (Marzin et al., 1985 (cited in Royal Society of Chemistry, 1989)) and in various mammalian cell systems (no further details available) (Royal Society of Chemistry, 1989). Positive results were obtained in a mouse bone marrow assay, however several factors make the results of this test uncertain (Manna et al., 1972 (cited in Royal Society of Chemistry, 1989)). In experiments of 20 to 30 days, aluminium chloride was given orally to rats, guinea pigs, and rabbits in doses ranging from 3 to 50 mg.kg^{-1} body weight per day, and in chronic experiments (6 months) it was given to rats in oral doses ranging from 0.025 to 2.5 mg.kg^{-1}. No increase of chromosomal aberrations were found in bone marrow cells as a result of these exposures (Krasovskii et al., 1979).
Furthermore, negative results were obtained in *in vitro* DNA-synthesis (Sirover et al., 1976 (cited in Royal Society of Chemistry, 1989)), the SOS chromotest with *Escherichia coli* (Olivier et al., cited in Royal Society of Chemistry, 1989)) and the rec assay with *Bacillus subtilis* (Nishioka, 1975; Kada et al., 1980; Kanematsu et al., 1980 (cited in Royal Society of Chemistry, 1989 and WHO, 1977)). Positive results were obtained in some plant assays, but it must be remembered that so far almost all metal salts have given positive results in these tests (Léonard et al., 1988). Positive results were also obtained in a *Drosophila* assay. CA were produced in spermatocytes of grasshoppers, feeded with a

Drosophila food mix with aluminium chloride (Manna et al., 1965 (cited in Royal Society of Chemistry)).
DiPaolo and Casto (1979: cited in SDWC, 1982) studied the effect of various metals on the *in-vitro* morphological transformation of Syrian hamster embryo cells. The results for aluminium chloride administered in concentrations up to 20 $\mu g.ml^{-1}$ were negative.

According to IARC (1984 and 1987a), air samples from various locations in two aluminium production facilities were mutagenic to *Salmonella typhimurium*.It must be noted, that particular matter from aluminium production plants only consist for a small part of aluminium and aluminium compounds. Other compounds present are for instance polynuclear aromatic hydrocarbons and fluorides. There are no data available on the genotoxicity of the aluminium compounds in the mixture.

Léonard et al. (review: 1988) concluded that aluminium and its salts are non-mutagenic.

Carcinogenicity

Animal data

Exposure by inhalation

No animal data on carcinogenicity of aluminium and aluminium compounds in case of inhalatory exposure are available.

Oral application

Lifetime (936 ± 49 days) administration of 5 $mg.l^{-1}$ Al (as aluminium potassium sulphate) in drinking water of mice (Charles River CD strain, two groups of 38-47 males and females) increased the number of lymphoma leukaemia tumours in females, but not the number of animals with tumours (Schroeder and Mitchener, 1975b (cited in WHO, 1977)). No effects on body weight and longevity were observed, and no details on histopathology are available. Further evaluation of a more lengthy final progress report (obtained by US FDA (1979: cited in WHO, 1989) indicates that there was no evidence for an increase in tumour incidence related to the administration of potassium aluminium sulphate.
A significant increase in (non malignant) tumour incidence was reported to be found in male rats (Long Evans Blu: LE strain, two groups with 19-26 males and females) after treatment with 5 $mg.l^{-1}$ Al (as aluminium potassium sulphate) in drinking water during lifetime (1,064 ± 20 days) (Schroeder and Mitchener, 1975a (cited in SDWC, 1982; WHO, 1989)). Further evaluation of a more lengthy final progress report (obtained by US FDA (1979: cited in WHO, 1989) indicates that there was no evidence for an increase in tumour incidence related to the administration of potassium aluminium sulphate.
No carcinogenic activity was demonstrated in studies using mice, rats, rabbits and guinea pigs after administration (intraperitoneal, intravenous) of Al-powder, $Al(OH)_3$, Al_2O_3, $AlPO_4$ and Al dextran (Furst, 1969 and 1971; Shubik and Hartwell, 1969 (all cited in SDWC, 1982; WHO, 1977)).
Aluminium hydroxide was not carcinogenic after daily intraperitoneal administration to mice for 4 months at dosages up to about 200 mg $Al.kg^{-1}$ body weight per day (Kay and Thorton, 1955 (cited in WHO, 1989)).

Skin application

No animal data on carcinogenicity of aluminium and aluminium compounds in case of skin application are available.
According to IARC (1984), there is sufficient evidence that samples of particulate polynuclear organic matter from one aluminium production plant were carcinogenic to experimental animals.
It must be noted, that particular matter from aluminium production plants, only consist for a small part of aluminium and aluminium compounds. A number of individual polynuclear aromatic compounds for which there is sufficient evidence of carcinogenicity in experimental animals have been measured at high levels in air samples taken from certain areas in aluminium production plants. Therefore no evaluation of the role of the aluminium compounds in the mixture can be made.

It is generally considered that aluminium and its salts are non-carcinogenic in experimental animals (Léonard et al., review 1988).

Human data

No human data on carcinogenicity of aluminium and aluminium compounds (not in complex mixtures) in case of inhalatory, oral and dermal exposure are available.
According to IARC (1987b), "the available epidemiological studies provide sufficient evidence that certain exposures in the aluminium production industry are carcinogenic to humans". A possible causative agent is pitch-fume. Dose-response relationships have been clarified and confounding by smoking controlled for.
It must be noted, that particular matter from aluminium production plants, only consist for a small part of aluminium and aluminium compounds. There are no data available on the carcinogenicity of the inhalatory exposure to the aluminium compounds in the mixture.

5.2 ECOTOXICITY

5.2.1 Bioaccumulation and biomagnification

Aluminium occurs in biota, but Spry and Wiener (1991) concluded that aluminium should not be considered to bioaccumulate. However, certain aquatic macrophytes, especially submergent species, can accumulate very high concentrations of Al (>4,000 mg.kg^{-1} dry weight) in some acidified waters (Sprenger and McIntosh, 1989). Also aquatic insects exhibit high Al concentrations in the range of 1,000 - 3,000 mg.kg^{-1} dry weight (Scheuhammer, 1991).
Biomagnification does not occur; the flesh of fish, birds and mammals contains low Al concentrations (< 50 mg.kg^{-1} dry weight) as most of the Al intake is excreted with the faeces.

5.2.2 Aquatic species

Aquatic toxicity data on aluminium are scarce. In table 5.3 a summary is given of the available short- and long-term toxicity data.

In situations where ionic aluminium is present, aluminium may have considerably greater toxicity than has been assumed. Of the aluminium forms inorganic monomeric (labile) aluminium has been reported to be the most toxic one (Driscoll et al., 1980). As indicated in table 5.3 most toxicity experiments are related to acidification. Usually the proportion of the labile aluminium in acidified lakes is about 50% in the summer (Vuorinen et al., 1992). The toxicity of aluminium is lowest at circumneutral pH values; both under acidic (Vuorinen et al., 1992) and alkaline conditions (Freeman and Everhart, 1971) the toxicity increases. Other abiotic factors that influence the toxicity of aluminium are humic substances and calcium, both influencing its bioavailability.
Differences in species susceptibility have been observed; certain strains of fish exhibit tolerance to the stress of acidic waters (see table 5.3: Vuorinen et al., 1992; pike may tolerate a pH of 4.0-5.3 whereas roach will be affected at pH below 5). This variation in tolerance may be related to differences in the reaction to aluminium.

No data are available on the toxicity to species living in the sediment.

Table 5.3 Toxicity data on aluminium in $mg.l^{-1}$ derived from laboratory experiments with various freshwater species

Species	Life stage	Exposure time	pH	Criterion	Result	Ref
Plants						
M. spicatum			7	EC50 (grwth)	2.5	1
S. capricornutum		4 d	8.2	EC50 (grwth)	0.46	2
Crustaceans						
C. reticulata		2 d	5.5-6.0	LC50	0.3-0.5	3
D. magna	<24 h	21 d	6.5-7.5	EC50 (repr)	0.68	4
		21 d	6.5-7.5	NOEC (repr)	0.16	5
Insects						
T. dissimilis	2-3 inst	55 d	6.8	LC37	0.8	6
C. punctipennis	4 instar		3.5-6.5	LC10	>1	7
C. anthrocinus	4 instar		3.5-6.5	LC10	>1	7
Fish						
S. gairdneri	fry		4.5	LC27	<0.02	8
		4-8 d	4.5	LC90	0.075	9
	alevins	4 d	4.5	LC50	0.12	8
		45 d	9	LC44	0.52	10
C. commersoni			5	LC50	0.05	11
			4.5-4.8	LC10	0.01	12
S. namaycush	fry		4.6-5.6	LC25	0.07	13
E. lucius	fry	10 d	4.0	LC50	0.1	14
			4.5	LC50	0.6	14
			5.0	LC50	>1	14
C. pallasi	fry	10 d	4.0	LC50	0.2	14
			4.5	LC50	0.25	14
			5.0	LC50	0.6	14
S. lucioperca	fry	10 d	4.0	LC50	<0.1	14
			4.5	LC50	<0.2	14
			5.0	LC50	0.3	14
R. rutilus	fry	10 d	4.0	LC50	<0.1	14
			4.5	LC50	<0.1	14
			5.0	LC50	0.1	14
B. rerio		2 d	7.6	LC50	80	15
M. saxatilis		7 d	6.0	NOEC (mort)	0.022	16
			6.5	MOEC (mort)	0.087	16
			7.2	NOEC (mort)	0.17	16
S. fontinalis		30 d	6.5	NOEC (repr)	0.057	17
Amphibians						
B. americanus	fry		4.3	NOEC(hatch)	0.005	18

1:Stanley (1974); 2:Gostomski (1990); 3:Shepard (1983); 4:Schofield and Trojnar (1980); 5:Biesinger and Christensen (1972); 6:Lamb and Baily (1981); 7:Havas and Likens (1985); 8:Holtze (1983); 9: Neville (1985); 10:Freeman and Everhart (1971); 11:Driscoll et al. (1980); 12:Baker and Schofield (1982); 13:Gunn and Keller (1984); 14:Vuorinen et al.(1992); 15:Dave (1985); 16:Buckler et al.(1987); 17:Cleveland et al.(1987); 18:Clark and LaZerte (1985)

5.2.3 Terrestrial species

Aluminium toxicity in soil ecosystems has been investigated in association with acidification only. Soil acidification results into increased aluminium concentration in the soil solution, limiting root development. Also, in acid soils ($pH<4.1$) competition for uptake sites may occur between Al^{3+} and Ca^{2+}, indicated by the $[Al^{3+}]/[Ca^{2+}]$ ratio (Heij and Schneider, 1991). In this section attention will be paid to the effects of aluminium only.

As to plants aluminium toxicity thresholds (using survival and root and/or shoot growth as parameters) have been reported varying between < 1.5 to > 30 $mg.l^{-1}$, based on experiments with seedlings growing either in solution or in greenhouse pots (De Vries, 1991). The red spruce has been identified as the most sensitive tree species, with biomass reductions starting to occur near 5 $mg.l^{-1}$ of total aluminium or 2 $mg.l^{-1}$ of labile inorganic aluminium.
Both laboratory and field studies indicated the important influence of the Al/Ca ratio on root development. Several studies showed that at an Al/Ca ratio of 1 in the soil solution, the uptake of bivalent cations is seriously inhibited and the roots are effected (Heij and Schneider, 1991).
In addition to damage due to changes in the soil, soil acidification and root damage as a result of aluminium are considered predisposing factors that increase susceptibility of a stand to gaseous air pollutants and extreme climatic conditions.

Data on effects of aluminium on soil organisms are limited to range finding values for two nematode species: *Rhabditis spec.* and *Plectus parietinus*, the 96 hr LC_{50}s being 75 and > 1,000 $mg.l^{-1}$ respectively (Schouten and Van der Brugge, 1989).

Data on effects of aluminium on various bird species have been evaluated by Scheuhammer (1991). The toxicity of dietary aluminium is primarily a function of the ability of aluminium to disrupt the absorption and metabolism of Ca and P. Experiments with chickens, ring doves, black and mallard ducklings indicate that the effect levels of aluminium are largely dependent upon the levels of dietary P (and to a less extent to dietary Ca levels). Scheuhammer (1991) concludes that dietary concentrations of soluble Al must be at least 50% of dietary P in order for adverse effects to occur.
The discussion on Al toxicity in insectivorous birds was started by a Swedish research group (Nyholm and Myhrberg, 1977; Nyholm, 1981). The Swedish group reported severe eggshell defects, reduced clutch sizes, and mortality in pied flycatchers and other insectivorous passerines nesting by the shore of an acid-stressed Swedish lake. The results indicated that high dietary Al intake may have played a causative role. However, emergent insects used as a food source were found to contain 100 mg $Al.kg^{-1}$ body weight (Nyholm, 1982), which level is not high enough to pose a significant threat to reproductive succes, even at a normal or reduced P content (Scheuhammer, 1991). Studies on the reproductive succes of kingbirds in acidified habitats in Canada (Glooschenko et al., 1986) and dippers in Britain (Ormerod et al., 1988) failed to demonstrate direct effects due to dietary Al, even though these birds prey on invertebrate taxa containing comparable concentrations of Al. Whereas the Swedish research group did not measure the P content in the insects, two British studies indicate that phosphorus concentrations in aquatic insects averaged at least 5-10 times the Al content and

did not yield strong relationships between Al concentrations in the insects and environmental pH over a range of about 5-8 (Sadler and Lynam, 1985; Ormerod et al., 1988). Hence, it is highly improbable that the reproductive impairments observed by Nyholm and Myhrberg (1977) can be attributed directly to Al toxicity (Scheuhammer, 1991).

5.3 TOXICOLOGICAL LIMIT VALUES

5.3.1. Humans

Orally administered aluminium is poorly absorbed through the gastrointestinal tract, and the small amount absorbed is almost completely excreted in the urine. Aluminium is of low toxicity to experimental animals. Aluminium compounds may be embryotoxic at concentrations causing maternal toxicity. There was no evidence for teratogenicity. There is also no evidence for carcinogenicity of aluminium compounds in experimental animals after oral exposure. Animal data on effects after inhalatory exposure to aluminium compounds are limited; a few studies describe local irritation in the respiratory system.

Aluminium in drinking water has been implicated in the etiology of some neurodiseases in humans; Alzheimer's disease and "dialysis dementia" (or "dialysis encephalopathy"), which is a relentlessly progressive form of dementia observed in chronic dialysis patients. The importance of aluminium in the development of these diseases has, however, not yet been established. Large oral doses cause gastrointestinal tract irritation and interference with phosphate metabolism. No data were available on carcinogenic, embryotoxic or teratogenic effects of aluminium compounds to humans. No adequate data were available on effects after inhalatory exposure.

There is no evidence for genotoxicity of aluminium and aluminium compounds. Therefore, a threshold extrapolation method is used for effect assessment. The assessment of toxicological limit values will be based on experimental studies.

Oral exposure

The oral (sub)chronic toxicity has been studied in rats, mice and dogs. According to the JECFA (1989) the "level causing no toxicological effect" is 110 $mg.kg^{-1}$ bw, derived from a study with dogs exposed to aluminium phosphate (acidic) in the diet for 189 days. On the basis of this study a "provisional tolerable weekly intake" (PTWI) of 7,0 $mg.kg^{-1}$ bw was estimated. The PTWI includes intake of aluminium from food additives (JECFA, 1989).

This PTWI is accepted by the RIVM temporarily. It must be emphasized that there are a number of studies in which (mild) effects were reported at lower doses (for example the study of Pettersen et al., 1988, which was not included in the JECFA evaluation). This implies that a more thorough evaluation of the data might be necessary.

Inhalatory exposure

A subchronic inhalation study in rats and guinea pigs exposed to aluminium chlorohydrate for six months resulted in a NO(A)EL of 0.05 $mg\ Al.m^{-3}$. In establishing a provisional limit value a safety factor of 1,000 instead of 100 is used, because the NO(A)EL resulted from a subchronic study in which the animals were exposed for only 5 days a week for 6 hours a day. This results in a provisional toxicological limit value of 0.05 μg Al.m^{-3}.

5.3.2. Ecosystems

Surface water

Literature data on the effects of aluminium at pH levels commonly occurring in Dutch surface waters (pH 7.8 - 8.4) are scarce. In this range a 4-d EC_{50} of 0.46 mg.l^{-1} for an algae species and a 2-d LC_{50} of 80 mg.l^{-1} for a fish species have been reported; long-term toxicity data are missing and no information is given on the no-effect levels. Previously the EPA (1973) tentatively recommended a limit value of 0.1 mg.l^{-1} for waters with a pH equal to or greater than 6.5. The Ontario Ministry of the Environment (1984) stated however that this level would be deleterious to growth and survival of fish. The Canadian guideline (1991) for dissolved inorganic Al is set on 0.1 mg.l^{-1} for waters with a pH > 6.5, $[Ca^{2+}]$ > 4.0 mg.l^{-1} and a Dissolved Organic Carbon (DOC) > 2.0 mg.l^{-1}. EIFAC (Howells et al., 1990) proposed the same criterion for European freshwater fish for waters with a pH > 6, $[Ca^{2+}]$ > 5 mg.l^{-1} and a DOC > 10 mg.l^{-1} or a Si/Al ratio > 13. In the Netherlands the pH is usually higher than 6.5, the average Ca^{2+}-concentration being greater than 2 mg.l^{-1}, whereas the DOC levels usually exceed the 10 mg.l^{-1} (CUVWO, 1988; RIWA, 1989). At the present stage of knowledge it is assumed that a toxicological limit value of 0.1 mg Al.l^{-1} (dissolved) is applicable to most Dutch surface waters. At a pH of 7 this corresponds with 1 mg Al.l^{-1} in total (Spry and Wiener, 1991).

In fens, however, much lower pH levels and Ca^{2+}-concentrations are found; the pH ranging from 4-6 and Ca^{2+}-concentrations from 0-10 mg.l^{-1}, respectively. For these areas the aluminium concentrations in water should be well below the 0.1 mg.l^{-1} . The NOEC-values reported in this pH-range are 0.005 mg.l^{-1} (at pH 4.3 for amphibians) and 0.022 mg.l^{-1} (at pH 6.0 for fish). The first value is mentioned in The Canadian guideline (1991): 0.005 mg.l^{-1} for waters with a pH<6.5, $[Ca^{2+}]<4.0$ mg.l^{-1} and a DOC<2.0 mg.l^{-1}. This value may be applicable to the fens in the Netherlands. It should be noted, however, that this value seems to be impractically low and, moreover, that under very acidic conditions the toxic effects of the high hydrogen ion concentrations are more important than is the presence of low (natural?) concentrations of aluminium. In fact aluminium has been shown to mitigate the effects of low pH (4.2-4.8) (Schofield and Trojnar, 1980; Baker and Schofield, 1982).

The above-mentioned limit values are based on the effects of aluminium on aquatic species only. Bioaccumulation in aquatic insects and macrophytes and possible effects on insectivorous and herbivorous species are not accounted for. The available information is not sufficient to make an effect assessment of this route of exposure.

No information is available on natural background concentrations in order to propose a tentative target value.

Sediment

There is no information to derive a tentative maximum permissible concentration in sediments: data on both toxicity and partitioning are lacking.

Soil

Based on the observation that effects on biomass of the most sensitive tree species (red spruce) start to occur near 2 $mg.l^{-1}$ of labile inorganic aluminium, Heij and Schneider (1991) considered the critical value for aluminium in the soil solution to be 2 $mg.l^{-1}$. According to the procedure described by Slooff (1992), however, at least a safety factor of 10 should be applied to derive a maximum permissible concentration if there are no indications for essentiality of aluminium and if the result is greater than the naturally occurring concentration levels. There are no indications for essentiality but there is no information on background levels either. For the Al/Ca ratio a provisional critical value of 1 has been set (Heij and Schneider, 1991). Yet the same final report of the Dutch priority programme on Acidification reports on observations of effects (not specified) below this level. Other factors that should be considered are DOC, pH and Fe-content. Current information is insufficient to derive a maximum permissible concentration in the soil (solution); tentatively it is suggested to apply a safety factor of 10 to the critical value: 1/10 x 2,000 $\mu g.l^{-1}$ = 200 $\mu g.l^{-1}$. This concentration corresponds with the maximum acceptable concentration of aluminium in drinking water (WLB, 1984; cited in Versteegh et al., 1992).

6. EVALUATION

6.1 EXCEEDING OF STANDARDS AND GUIDELINES

6.1.1. Soil and groundwater

The EC directive for aluminium in drinking water is 50 $\mu g.l^{-1}$, and maximum allowed is 200 $\mu g.l^{-1}$ (see chapter 2). In groundwater (5-15 m below land surface) aluminium concentrations are found which significantly surpass these values. In figure 6.1. the percentage of observations on exceeding the 50 and 200 $\mu g.l^{-1}$ levels are presented.

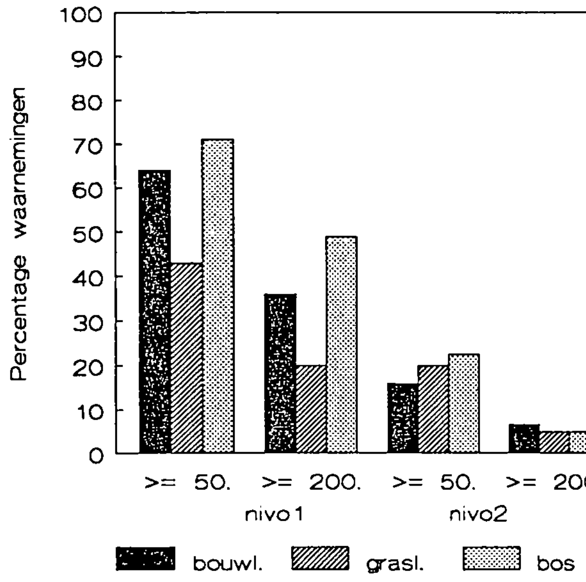

Figure 6.1 Percentage of observations on exceeding the 50 and 200 $\mu g.l^{-1}$ in groundwater underneath agricultural land, grassland and woodland at 5 - 15 m below surface land (data from the national groundwater monitoring network

6.1.2 Others

There are no guidelines for surface water or outdoor air. The current aluminium concentrations are below the maximum allowable and the average values are below the EC-directive. In contrast to Germany and Belgium there are no general emission standards to surface water in the Netherlands. Those in Germany and Belgium are likely to be exceeded in the Netherlands.

6.2 RISKS TO MAN

There is no evidence for genotoxicity of aluminium and aluminium compounds. As to oral exposure the provisional tolerable weekly intake of 7 $mg.kg^{-1}$ body weight as suggested by the JECFA (1989) is tentatively adopted. Two 24-hour diet studies performed in 1978 and 1984/1985 in the Netherlands indicate weekly total intakes of 9.8 - 230 (average 32; median 28) mg and 4.2 - 90 (average 22; median 20) mg, respectively. Based on an average body weight of 70 kg for adults this corresponds with 0.14 -3.2 (average 0.44; median 0.39) and 0.06 - 1.25 (average 0.30; median 0.28) $mg.kg^{-1}$ body weight. Based on these data one may conclude that the risk of current oral exposure levels of aluminium to man is negligible. It is emphasized, however, that there are studies in which effects were reported at lower doses (a factor of 5), which were not included in the JECFA evaluation. Taking into account this factor of five, the risk of oral exposure level is still to be considered low.
As to inhalatory exposure provisionally a toxicological limit value of 0.05 μg $Al.m^{-3}$ is proposed, applying a safety factor of 1,000 to a NO(A)EL determined for rats and guinea pigs exposed for six months, five days a week for six hours a day. This limit value may be considered as conservative since the observed effects are often considered to be an adaptive response to many types of dusts. The aluminium content of outdoor and indoor air in the Netherlands is unknown. However, one may assume that this content will not deviate significantly from that in the UK. In England the outdoor concentrations have been estimated at 0.05 - 0.5 $\mu g.m^{-3}$ in rural areas, up to 0.1 - 5 $\mu g.m^{-3}$ in urban areas. In indoor air the Al concentrations may be higher as a result of smoking. Hence, comparing the provisional (conservative) toxicological limit value with possible exposure levels, one may conclude that a certain risk can not be ruled out.
Medications and cosmetics may significantly contribute to the aluminium exposure for some individuals. This route of exposure will not be discussed in this report.

6.3 RISKS TO ECOSYSTEMS

Aquatic ecosystems

Based on the available aquatic toxicity data it is assumed that a tentative toxicological limit value of 0.1 mg inorganic $Al.l^{-1}$ (as "dissolved" Al, < 0.45 μm; corresponding to approx. 1 mg $Al.l^{-1}$ in total) is applicable to protect aquatic species in most Dutch surface waters. In the river Rhine the average total Al concentrations range from 0.19 to 0.27 $mg.l^{-1}$ in the period 1988-1992, the maximum concentrations were below 1 mg $Al.l^{-1}$ during this period as well. Higher levels have been recorded in the river Meuse, although the critical concentration of 1 mg $Al.l^{-1}$ in total is seldom exceeded (1990: average, 90% and maximum concentration being 0.60, 0.71 and 2.6 mg $Al.l^{-1}$, respectively). In surface waters of Hoogheemraadschap Rijnland the yearly average concentrations exceeded the tentative toxicological limit value in 1981, 1982 and 1989 during the period of 1977 - 1992, the highest concentration measured being 18 $mg.l^{-1}$ at Gouda. Aluminium concentrations in effluent of domestic sewage treatment plants are generally below the toxicological limit value, but industrial effluents may contain several hundreds of mg $Al.l^{-1}$. From this one may conclude that in some water bodies the current aluminium concentrations are close to, or even exceed, the critical level. Definitively in the vicinity of point sources aquatic life will be at risk.

Although aluminium is considered not to bioaccumulate, high levels have been observed in aquatic insects (chironomid midges, caddisflies, stoneflies and mayflies) and aquatic macrophytes (especially submergent species). Therefore certain insectivores and herbivores may be at risk, depending on the dietary P levels.

The discussion on Al toxicity in insectivorous birds was started by a Swedish research group (Nyholm and Myhrberg, 1977; Nyholm, 1981) and resulted in anxiety in The Netherlands (Graveland, 1991; LAC, 1991). The Swedish group reported severe effects in birds nesting by the shore of an acid-stressed Swedish lake. The results indicated that high dietary Al intake may have played a causative role. However, as discussed in section 5.2.3, it is highly improbable that the reproductive impairments observed by Nyholm and Myhrberg (1977) can be attributed directly to Al toxicity (Scheuhammer, 1991).

The discussion on Al toxicity in herbivores was initiated by Scheuhammer (1991), stating that the Al levels found in submergent macrophytes in some acidified waters are high enough to be of toxicological concern for aquatic herbivores even assuming an adequate dietary intake of Ca and P. This also may be true for terrestric herbivores if the high levels found in tea (2,800 on the average and up to 17,000 mg $Al.kg^{-1}$ dry weight) is not an exceptional case. The current information however is too limited to draw any conclusions.

Terrestrial ecosystems

Due to acidification aluminium has been mobilized in the soil system, especially in sandy forest soils. As a result concentrations of dissolved Al in the top soil are elevated and subsequently are lowered by leaching out to deeper groundwater levels. As such the Al buffer capacity will be lost and so the pH in the root zone may decline to values below pH 3. These (irreversable) effects result from acidification and therefore will not be discussed as such. The only aspect taken into account is the (temporarily) increase in aluminium concentrations and its effects on terrestrial ecosystems. In sandy forest soil systems the aluminium concentrations are far above the critical value of 2,000 $\mu g.l^{-1}$ and therefore these ecosystems are at risk.

7. POINTS FOR DISCUSSION AND DECISION-MAKING

In discussing the risks of aluminium one should distinguish the risks related to acidification resulting into mobilization of (natural occurring) aluminium, and those resulting from the discharge of aluminium in our environment.
Acidification results, among others, into increased dissolved aluminium levels in surface water and soil. In most cases Dutch surface waters are sufficiently buffered and therefore acidification does not present a problem in terms of increased aluminium toxicity for aquatic ecosystems. In contrast to this, Dutch soils are vulnerable to acidification with regard to aluminium: in the process of leaching out first elevated dissolved aluminium levels at the root zone are harmful to terrestrial ecosystems and, subsequently, reach levels in groundwater surpassing the current drinking water standars and guidelines.

As to emission of aluminium in the environment the figures show an increase of industrial emissions in the last decade. However, although productivity is expected to increase during the coming years the industrial emissions are likely to decrease because of emission reducing measures. One may expect an increase of free aluminium, resulting from the decreasing phosphate emissions, but this has not been found so far. If extra aluminium is added for removal of phosphates, additional aluminium emissions to surface water will take place. Further it should be noted that emission data of various non-industrial sources (e.g. agriculture and traffic) were not available.

There are indications that current exposure levels may present a risk to both man and ecosystems. Man may be at risk since a provisionally derived toxicological limit value of aluminium in air (0.05 μg Al.m^{-3}) is likely to be exceeded in ambient and indoor air in the Netherlands. However, exposure data are missing and the proposed toxicological limit value is rather conservative, introducing some uncertainties in the risk estimation. Aquatic life may be endangered in some surface waters, but definitively in the vicinity of emission sources effects on aquatic species are expected to occur. Bioaccumulation may lead to high aluminium content of macrophytes and insects and so, depending on the dietary intake of calcium and phosphoros, herbivorous and insectivorous species may be at risk.

Hence, the following items may deserve further attention:

- Acidification leads to leaching out of aluminium. Apart from the toxic effects of high dissolved aluminium concentrationsand the subsequent pH-effects on soil ecosystems, this will lead to increased aluminium concentrations in groundwater that may be used as a raw water source for drinking water supply. The PTWI is 7 mg.kg^{-1} b.w. Based on a body weight of 70 kg, the average and maximum weekly intake is 0.30 - 0.44 and 1.25 - 3.2 mg.kg^{-1} b.w., respectively. The question may rise what is the risk of the increased aluminium concentrations in drinking water for human health. Consumption of drinking water containing aluminium levels ten times (!) the maximum acceptable concentration result in an additional exposure of less than 0.06 mg.kg^{-1}. On the other hand a geographical relation was found between Alzheimer's disease and aluminium in drinking water: in areas with a drinking water Al content >0.01 mg.l^{-1} (approx. >0.14 mg per week), the incidence of the disease was about 50% higher than in areas with a drinking water Al content <0.01 mg.l^{-1}. However, no dose relationship was found nor confounding factors were taken into consideration. Therefore the ecological impact of consequent increased aluminium levels in tape water and effluents may be of more concern.

- In contrast to Germany and Belgium in the Netherlands there are no general emission standards, though high aluminium levels in surface water are deleterious to aquatic life. In some cases emission standards are incorporated in licences for individual companies and so companies without a emission licence are not restricted in their aluminium emissions other than that they should apply best feasible techniques to reduce the amount of aluminium in waste water.
- This report presents just a superficial evaluation of the risks of aluminium; an in depth study is needed for a sound evaluation of the current and future risks of aluminium for both man and ecosystems.
  As to man the provisional tolerable weekly intake of 7 mg.kg^{-1} body weight as suggested by the JECFA (1989) needs updating, since there are studies in which effects were reported at lower doses (a factor of 5), which were not included in the JECFA evaluation. Taking into account this factor of five, the risk of oral exposure level is still to be considered low and therefore this may have a low priority. Another aspect of concern is inhalatory exposure: a certain risk cannot be ruled out. In order to quantify this risk exposure data are needed for the situation in the Netherlands.
  Both aquatic and terrestrial ecosystems may be at risk. It is suggested to initiate a study into the exposure levels in water systems near sources and the dietary levels of Al and P for species at risk.

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APPENDIX A

PHYSICAL/CHEMICAL CHARACTERISTICS OF ALUMINIUM AND SOME ALUMINIUM COMPOUNDS

Aluminium (Lide, 1990; NIA, 1990)

chemical formula:	Al
atomic number:	13
atomic weight:	26.981539
physical form:	ductile metal, cubic
colour:	silver white
melting point:	660.37°C
boiling point:	2467°C
autoignition temperature:	590°C
density:	2.702
vapour pressure:	0 mbar at 20°C
solubility:	insoluble in H_2O; soluble in alkali, HCl, H_2SO_4; insoluble in concentrated HNO_3, hot acetic acid
CAS registry number:	7429-90-5

Aluminium chloride (Lide, 1990; NIA, 1990)

chemical formula:	$AlCl_3$
molecular weight:	133.34
physical form:	hexagonal chrystals or powder, very deliquescent
colour:	white to colourless
odour:	HCl
melting point:	190°C at 2.5 atmospheres
sublimation point:	177.8°C; decomposes at 262°C
density:	fused 2.44 at 25°C
vapour pressure:	0.004 mbar at 50°C
solubility:	699 $g.l^{-1}$ H_2O at 15°C (reactive); 1,000 $g.l^{-1}$ absolute alcohol; 0.72 $g.l^{-1}$ chloroform at 25 °C; soluble in CCl_4, ether, slightly soluble in benzene
CAS registry number:	7446-70-0

Aluminium oxide (Lide, 1990)

chemical formula:	Al_2O_3
molecular weight:	101.96
physical form:	hexagonal chrystals (α-Alumina rhombic chrystals; Γ-Alumina microscopic chrystals)
colour:	colourless (Γ-Alumina white)
melting point:	2,072
boiling point:	2,980
density:	3.965 at 25°C
solubility:	insoluble in H_2O; very slightly soluble in acid, alkali
CAS registry number:	1344-28-1