Toxicity Profiles

Formal Toxicity Summary for ALUMINUM

NOTE: Although the toxicity values presented in these toxicity profiles were correct at the time they were produced, these values are subject to change. Users should always refer to the Toxicity Value Database for the current toxicity values.

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September 1993

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Prepared for OAK RIDGE RESERVATION ENVIRONMENTAL RESTORATION PROGRAM.

*Managed by Martin Marietta Energy Systems, Inc., for the U.S. Department of Energy under Contract No. DE-AC05-84OR21400.

EXECUTIVE SUMMARY

Aluminum is a silver-white flexible metal with a vast number of uses. It is poorly absorbed and efficiently eliminated; however, when absorption does occur, aluminum is distributed mainly in bone, liver, testes, kidneys, and brain (ATSDR, 1990).

Aluminum may be involved in Alzheimer's disease (dialysis dementia) and in Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex) (ATSDR, 1990; Goyer, 1991). Aluminum content of brain, muscle, and bone increases in Alzheimer's patients. Neurofibrillary tangles (NFTs) are found in patients suffering from aluminum encephalopathy and Alzheimer's disease. Symptoms of "dialysis dementia" include speech disorders, dementia, convulsions, and myoclonus. People of Guam and Rota have an unusually high incidence of neurodegenerative diseases. The volcanic soil in the region of Guam where the high incidence of ALS-PD occurs contains high levels of aluminum and manganese. Neurological effects have also been observed in rats orally exposed to aluminum compounds.

The respiratory system appears to be the primary target following inhalation exposure to aluminum. Alveolar proteinosis has been observed in guinea pigs, rats, and hamsters exposed to aluminum powders (Gross et al., 1973). Rats and guinea pigs exposed to

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aluminum chlorohydrate exhibited an increase in alveolar macrophages, increased relative lung weight, and multifocal granulomatous pneumonia (Cavender et al., 1978).

No decrease in reproductive capacity, hormonal abnormalities, or testicular histopathology was observed in male rats exposed to aluminum in drinking water for 90 days (Dixon et al., 1979).

However, male rats exposed to aluminum (as aluminum chloride) via gavage for 6 months exhibited decreased spermatozoa counts and sperm motility, and testicular histological and histochemical changes (Krasovskii et al., 1979).

Subchronic and chronic reference doses and reference concentrations have not been derived for aluminum.

Male rats exposed to drinking water containing aluminum (as aluminum potassium sulfate) for a lifetime exhibited increases in unspecified malignant and nonmalignant tumors (Schroeder and Mitchener, 1975a), and similarly exposed female mice exhibited an increased incidence of leukemia (Schroeder and Mitchener, 1975b). Rats and guinea pigs exposed via inhalation to aluminum chlorohydrate developed lung granulomas (Cavender et al., 1978), while granulomatous foci developed in similarly exposed male hamsters (Drew et al., 1974).

The U.S. EPA has not evaluated aluminum or aluminum compounds for carcinogenicity, and a weight-of-evidence classification is currently not assigned.

1. INTRODUCTION

Aluminum (CAS registry number 7429-90-5) is a silver-white, flexible metal (ATSDR, 1990). It makes up about 8% of the earth's crust in undecomposed rock fragments and in secondary aluminosilicate clays. The aluminum content of seawater ranges from 3 to 2400 ppb (Venugopal and Luckey, 1978). Until recently, aluminum has existed in forms not available to humans and most other species. However, acid rain has increased the availability of aluminum to biological systems and has resulted in destructive effects on fish and plant species. It is unknown if humans are susceptible to this increased bioavailability (Goyer, 1991). Aluminum is not an essential element for mammals (Venugopal and Luckey, 1978).

Aluminum metal is used as a structural material in the construction, automotive, and aircraft industries, in the production of metal alloys, and in the electrical industry in power lines, insulated cables and wiring. Other uses of aluminum metal include cooking utensils, decorations, fencing, highway signs, cans, food packaging, foil, and dental crowns and dentures (ATSDR, 1990).

Aluminum compounds and materials also have a number of uses. Aluminum powder is used in paints and fireworks, and natural aluminum minerals are used in water purification, sugar refining, and in the brewing and paper industries. Aluminum borate is used in the production of glass and ceramics, and aluminum chloride is used to make rubber, lubricants, wood preservatives, and cosmetics. Aluminum chlorohydrate is the active ingredient in antiperspirants and deodorants, while aluminum hydroxide is used as a pharmaceutical to lower plasma phosphorus levels of patients with kidney failure. Aluminum silicate is a component of dental cement, and various other aluminum compounds are used as tanning agents in the leather industry, and as components of veterinary medicines, glues, and disinfectants (ATSDR, 1990).

2. METABOLISM AND DISPOSITION

2.1. ABSORPTION

In mammals, gastrointestinal absorption of ingested aluminum is poor due to the conversion of aluminum salts into insoluble aluminum phosphate in the digestive tract. This is the result of pH changes and the presence of phosphate in the diet (Venugopal and Luckey, 1978). Absorption from oral exposure is also affected by the chemical form of the aluminum, vitamin D, parathyroid hormone, and other ions (ATSDR, 1990). At high doses, such as 200 mg aluminum/kg as $Al_2(SO_4)_3$, intestinal absorption in rats has been shown to be 10% (Kortus, 1967). Aluminum compounds can affect absorption of other elements in the gastrointestinal tract; aluminum inhibits fluoride absorption and may decrease the absorption of calcium and

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iron compounds. It may possibly decrease the absorption of cholesterol by forming an aluminum pectin complex that binds fats to nondigestable vegetable fibers (Nagyvary and Bradbury, 1977). Absorption from intramuscular, subcutaneous, and peritoneal cavity injection of aluminum compounds is slow, occurring mainly through phagocytosis by macrophages (Venugopal and Luckey, 1978). When aluminum compounds are intravenously injected, a small portion of ionic aluminum is bound to albumin and is transported out of the blood into soft tissues, including the brain. Following inhalation, most insoluble aluminum salts are retained in the lung, while soluble compounds are slowly absorbed into the blood. Aluminum measured in the lung tissue of aluminum refinery workers increases with duration of exposure (Venugopal and Luckey, 1978). Aluminum is not readily absorbed through the skin (Venugopal and Luckey, 1978).

2.2. DISTRIBUTION

Following absorption, aluminum is distributed mainly in the skeleton, liver, testes, kidneys, and brain, and in smaller amounts in other soft tissues (Venugopal and Luckey, 1978). Retention of aluminum in bone is prolonged; however, it is transient in soft tissues. Renal failure increases aluminum deposition in the bone (Thurston et al., 1972). Rats fed diets containing aluminum hydroxide exhibited increased aluminum levels in bone, muscle, and kidneys. Aluminum concentrations in these tissues decreased 3 days after withdrawal of aluminum hydroxide from the diet (Greger and Donnaubauer, 1986). There is limited evidence to suggest that aluminum administered orally to rabbits can cross the placenta, accumulate in the fetus, and be transferred to the neonate via milk (Cranmer et al., 1986; Yokel and McNamara, 1985). When aluminum hydroxide was injected intraperitoneally in rats, the highest aluminum concentration was observed in the liver. High aluminum levels were also observed in muscle, brain, bone, and heart (Berlyne et al., 1972). Following inhalation exposure, aluminum accumulates in the lungs as particulates of poorly soluble compounds (Ganrot, 1986).

2.3. METABOLISM

Aluminum, itself, does not undergo metabolism in that it is absorbed and excreted unchanged. However, it is found attached to other chemicals, and these moieties can change within the body. The aluminum ion is easily bound to many substances and structures in the organism, and its fate is determined by its affinity to each of the ligands and their metabolism (ATSDR, 1990).

2.4. EXCRETION

In orally exposed humans, absorbed aluminum is eliminated through the urine, and unabsorbed aluminum is excreted in the feces (ATSDR, 1990), while in dogs and pigs injected with aluminum, the major excretion route is the kidney (Kolvalchik et al, 1978; Monteagudo et al, 1988). After inhalation exposure to aluminum, the urine is the major route of excretion (ATSDR, 1991). Small amounts of aluminum may also be excreted through sweat (Venugopal and Luckey, 1978).

3. NONCARCINOGENIC HEALTH EFFECTS

3.1. ORAL EXPOSURES

3.1.1. Acute Toxicity

3.1.1.1. Human

No information was available regarding the acute toxicity of aluminum in humans.

3.1.1.2. Animal

Due to the poor absorption and efficient excretion of aluminum, acute oral toxicity is observed only after relatively large doses. The LD_{50} for aluminum nitrate in rats is 261 mg aluminum/kg (Llobet et al., 1987), and the LD_{50} for aluminum chloride in mice is 770 mg aluminum/kg (Ondreicka et al., 1966).

3.1.2. Subchronic Toxicity

3.1.2.1. Human

Greger and Baier (1983) gave 4 healthy men a control diet containing 4.6 mg aluminum/day for 20 days, while 4 other men received a test diet of 125 mg aluminum/day as aluminum lactate. The diets were then exchanged for an additional 20 days, and each subject acted as his own control. Fecal, urine, and serum albumin measurements indicated that absorption and rapid excretion occurred. No adverse effects were observed.

3.1.2.2. Animal

Male rats and male guinea pigs were given 0, 6, 17, or 50 mg aluminum/kg/day as aluminum chloride in water by gavage for 20-30 days. Male rabbits were similarly exposed to 0, 3, 9, or 27 mg aluminum/kg/day (Krasovskii et al., 1979). Decreased serum alkaline phosphatase activity was observed at $\geq$ 17 mg/kg/day in the rats and guinea pigs, and $\geq$ 9 mg/kg/day in the rabbits. Serum ATP, ADP, and AMP levels were decreased at $\geq$ 17 mg/kg/day in the rats and guinea pigs, and $\geq$ 27 mg/kg/day in the rabbits. Rats were also similarly exposed to 0.0025, 0.25, or 2.5 mg aluminum/kg/day for 6-12 months (Krasovskii et al., 1979). Decreased serum alkaline phosphatase and decreased motor reflexes were observed at the high dose. Alkaline phosphatase activity was also decreased at 0.25 mg/kg, but only during the first month of exposure. Eight male Fischer rats and eight female Sprague-Dawley rats were fed a diet containing 0.2% aluminum (as aluminum chloride) for 12 weeks (Commissaris et al., 1982). Significantly decreased locomotor activity was observed in the female rats, while the males exhibited a trend in this effect. Sprague-Dawley rats administered 0.1% aluminum (as aluminum chloride) for 11 months exhibited decreased locomotor activity and learning (Commissaris et al., 1982).

3.1.3. Chronic Toxicity

3.1.3.1. Human

Aluminum may be involved in Alzheimer's disease, "dialysis dementia", and Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex) but the causal link between aluminum and these diseases is tenuous at best (ATSDR, 1990; Goyer, 1991). Increased amounts of aluminum have been observed in the brains of persons dying of Alzheimer's disease, however aluminum content varies greatly in these patients. Also, neurofibrillary tangles (NFTs) are found in patients suffering from aluminum encephalopathy and Alzheimer's disease. The formation of NFTs is associated with loss of synapsis and atrophy of the dendritic tree (Goyer, 1991). Although Alzheimer's patients often have more aluminum than usual in the NFTs, there are no significant differences between Alzheimer's patients and controls in the aluminum content of hair, serum or spinal fluid (Shore and Wyatt, 1986). This may indicate that Alzheimer's patients have a decreased blood -brain barrier for aluminum that may be the result of genetic factors, viral, or immune mediated damage (Goyer, 1991). "Dialysis dementia" occurs in patients on renal dialysis who receive large amounts of aluminum orally or intravenously (ATSDR, 1990; Goyer, 1991). Symptoms include speech disorders, dementia, convulsions, and myoclonus. These symptoms usually occur after 3 to 7 years of dialysis treatment and may be due to aluminum intoxication. Aluminum content of brain, muscle, and bone increases in these patients (Goyer, 1991).

People of Guam and Rota have an unusually high incidence of neurodegenerative diseases associated with nerve cell loss and neurofibrillary degeneration of the Alzheimer type termed Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex). The volcanic soil in the region of Guam where the high incidence of ALS-PD occurs, contains high levels of aluminum and manganese and low levels of calcium and magnesium. It is hypothesized that low calcium and magnesium intake induce secondary hyperparathyroidism resulting in an increase in aluminum and other toxic metals. It is unknown how aluminum enters the brains of the ALS-PD patients (Goyer, 1991).

3.1.3.2. Animal

Aluminum chloride was administered to groups of 10 mice at concentrations of 0 or 19.3 mg aluminum/kg/day in drinking water in a 3 generation study (Ondreicka et al.,1966). The parental generation was treated for 180-390 days, and weanlings were similarly treated from 4 weeks of age. Decreased body weight was observed in the second and third generations; however, the significance of this effect is difficult to assess since food consumption was not reported. Zero or 5 ppm aluminum (as aluminum chloride) was administered to 52 Long-Evans rats/sex (Schroeder and Mitchener, 1975a) and 54 Swiss mice/sex (Schroeder and Mitchener, 1975b) for life. No adverse effects were observed in either species.

3.1.4. Developmental and Reproductive Toxicity

3.1.4.1. Human

No information was available regarding the developmental and reproductive toxicity of aluminum in humans.

3.1.4.2. Animal

Dixon et al. (1979) exposed male Sprague-Dawley rats (31/group) to 0, 5, 50, or 500 mg aluminum/L (as aluminum chloride) in drinking water for 30, 60, or 90 days. Seven rats from each group were sacrificed at each time point for hormone determination and histological examination of the testes. The remaining males from each group were mated after 90 days of treatment; a different female was paired with each male every 7 days for a total of 70 days. No adverse effects were observed. Krasovskii et al. (1979) exposed male rats to 0, 0.025, 0.25 or 2.5 mg aluminum/kg/day (as aluminum chloride) by gavage for 6 months. Decreased spermatozoa counts and sperm motility, and testicular histological and histochemical changes were observed at 2.5 mg/kg/day. As described in section 3.1.3.2, Ondreicka et al. (1966) administered aluminum chloride in drinking water to groups of 10 mice at concentrations of 0 or 19.3 mg aluminum/kg/day for 3 generations. Decreased body weight was observed in the second and third generations; however, food consumption was not reported so this effect may not be significant.

3.1.5. Reference Dose

3.1.5.1. Subchronic

An oral, subchronic reference dose has not been calculated for aluminum.

3.1.5.2. Chronic

An oral, chronic reference dose has not been calculated for aluminum.

3.2. INHALATION EXPOSURES

3.2.1. Acute Toxicity

3.2.1.1. Human

No information was available regarding the acute toxicity of aluminum in humans.

3.2.1.2. Animal

No information was available regarding the acute toxicity of aluminum in animals.

3.2.2. Subchronic Toxicity

3.2.2.1. Human

No information was available regarding the subchronic toxicity of aluminum in humans.

3.2.2.2. Animal

Gross et al. (1973) exposed groups of 14-30 guinea pigs, rats, and hamsters to metallic aluminum powders at air concentrations of 15, 30, 50, or 100 mg/m^3, 6 hours/day, 5 days/week for 6 months. Alveolar proteinosis was observed in all 3 species. Groups of 35 Fischer rats/sex and 35 Hartley guinea pigs/sex were exposed to 0.25, 2.5, or 25 mg/m^3 aluminum chlorohydrate, 6 hours/day, 5 days/week, for 6 or 12 months (Cavender et al., 1978). At 6 months (in both species), alveolar macrophages were increased at all dose levels. Decreased body weight, increased relative lung weight, and multifocal granulomatous pneumonia were observed at 25 mg/m^3. At 12 months granulomas were observed in the lungs of both species exposed to 25 mg/m^3.

3.2.3. Chronic Toxicity

3.2.3.1. Human

Pulmonary fibrosis has been associated with occupational exposure to aluminum powder and dust (ATSDR, 1990; U.S. EPA, 1987). However, this association is inconclusive because of concurrent exposure to other irritants, cigarette smoking, or previous occupational exposures. The U.S. EPA reports that there is no evidence of fibrogenic activity of aluminum at exposure levels recommended by the ACGIH (10 mg/m^3 for dust and 5 mg/m^3 for powder) and classifies aluminum dust and powder as inert particles (U.S. EPA, 1987). Workers were treated with 350 mg/m^3 of respirable alumina powder for 10 minutes/day as a treatment for silicosis (Stokinger, 1981). Over 42,000,000 treatments were given over a 27-year period, and no adverse effects were observed.

3.2.3.2. Animal

Pigott et al. (1981) exposed a group of 50 Aderly Park rats to 2.18 mg/m^3 aluminum fibers, 6 hours/day, 5 days/week for 86 weeks. Slight increases in alveolar macrophages and irritation of the nasal passages were observed. However, these responses are typical of exposure to inert, irritant particles, and thus may be due to physical irritation rather than the composition of the aluminum itself.

3.2.4. Developmental and Reproductive Toxicity

3.2.4.1. Human

No information was available regarding the developmental and reproductive toxicity of aluminum in humans.

3.2.4.2. Animal

No information was available regarding the acute developmental and reproductive toxicity of aluminum in animals.

3.2.5. Reference Concentration

3.2.5.1. Subchronic

An inhalation, subchronic reference concentration has not been calculated for aluminum.

3.2.5.2. Chronic

An inhalation, chronic reference concentration has not been calculated for aluminum.

3.3. OTHER ROUTES OF EXPOSURE

3.3.1. Acute Toxicity

3.3.1.1. Human

Aluminum compounds are used in antiperspirant products without harmful effects to the skin or other organs. However, some people are unusually sensitive to these products and may develop skin rashes (ATSDR, 1990). Children who had injections of vaccines or allergens in an aluminum-based vehicle showed hypersensitivity to aluminum chloride in a patch test (ATSDR, 1990).

3.3.1.2. Animal

Mice, pigs, and rabbits exhibited skin hyperplasia, microabscess formation, dermal inflammatory cell infiltration, and ulceration from the dermal application of 10% aluminum chloride and aluminum nitrate applied for 5 days. No effects were observed with similarly applied aluminum sulfate, hydroxide, chlorohydrate, or acetate (Lansdown, 1973).

3.3.2. Subchronic Toxicity

3.3.2.1. Human

No information was available regarding the subchronic toxicity of aluminum by other routes of exposure in humans.

3.3.2.2. Animal

No information was available regarding the subchronic toxicity of aluminum by other routes of exposure in animals.

3.3.3. Chronic toxicity

3.3.3.1. Human

No information was available regarding the chronic toxicity of aluminum by other routes of exposure in humans.

3.3.3.2. Animal

No information was available regarding the chronic toxicity of aluminum by other routes of exposure in animals.

3.3.4. Developmental and Reproductive Toxicity

3.3.4.1. Human

No information was available regarding the developmental and reproductive toxicity of aluminum by other routes of exposure in humans.

3.3.4.2. Animal

No information was available regarding the developmental and reproductive toxicity of aluminum by other routes of exposure in animals.

3.4. TARGET ORGANS/CRITICAL EFFECTS

3.4.1. Oral Exposures

3.4.1.1. Primary Target Organs

Central Nervous System: Aluminum may be involved in Alzheimer's disease, "dialysis dementia", and Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex). Increased amounts of aluminum have been observed in the brains of persons dying of Alzheimer's disease and neurofibrillary tangles (NFTs) are found in patients suffering from aluminum encephalopathy and Alzheimer's disease. Significantly decreased locomotor activity was observed in female rats fed aluminum in the diet for 12 weeks, and Sprague-Dawley rats administered aluminum for 11 months exhibited decreased locomotor activity and learning.

3.4.1.2. Other Target Organs

Reproductive System: Male rats exposed to aluminum chloride by gavage for 6 months exhibited decreased spermatozoa counts and sperm motility, and testicular histological and histochemical changes.

3.4.2. Inhalation Exposures

3.4.2.1. Primary Target Organs

Respiratory System: Alveolar proteinosis was observed in guinea pigs, rats, and hamsters exposed to aluminum powders for 6 months. Rats and guinea pigs exposed to aluminum chlorohydrate for 6 months exhibited an increase in alveolar macrophages, increased relative lung weight, and multifocal granulomatous pneumonia. After 12 months exposure, granulomas were observed in the lungs of both rats and guinea pigs.

4. CARCINOGENICITY

4.1. ORAL EXPOSURES

4.1.1. Human

No information was available regarding the carcinogenicity of aluminum by the oral route in humans.

4.1.2. Animal

Schroeder and Mitchener (1975a) exposed Long-Evans weanling rats (52/sex) to drinking water containing 5 mg/L aluminum (as aluminum potassium sulfate) for life. Specific tumor types were not specified; the incidence of total tumors was 13/25 in treated males and 4/26 in control males. The incidence of malignant tumors was 6/25 in treated males and 2/26 in control males. In another study, groups of 54 Swiss mice/sex were given 5 mg aluminum/L (as aluminum potassium sulfate) in the drinking water for life (Schroeder and Mitchener, 1975b). The incidence of leukemia in treated females was 10/41, while the incidence in control females was 3/47.

4.2. INHALATION EXPOSURES

4.2.1. Human

No information was available regarding the carcinogenicity of aluminum by the inhalation route in humans.

4.2.2. Animal

Rats and guinea pigs (35/sex/group) were exposed to 0.25, 2.5, or 25 mg/m^3 of aluminum chlorohydrate, 6 hours/day, 5 days/week for 6-12 months (Cavender et al., 1978). Lung granulomas were observed in both species following exposure to 25 mg/m^3 for 6 months and

2.5 mg/m^3 for 12 months. Drew et al. (1974) exposed male hamsters to 52 mg/m^3 aluminum chlorohydrate, 6 hours/day, 5 days/week for 20 or 30 exposures. Granulomatous foci developed at the bifurcation of the bronchioalveolar ducts.

4.3. OTHER ROUTES OF EXPOSURE

4.3.1. Human

No information was available regarding the carcinogenicity of aluminum by the other routes of exposure in humans.

4.3.2. Animal

No information was available regarding the carcinogenicity of aluminum by other routes of exposure in animals.

4.4. EPA WEIGHT-OF-EVIDENCE

4.4.1. Oral

No weight-of-evidence classification has been assigned.

4.4.2. Inhalation

No weight-of-evidence classification has been assigned.

4.5. CARCINOGENICITY SLOPE FACTORS

4.5.1. Oral

An oral carcinogenicity slope factor has not been derived for aluminum.

4.5.2. Inhalation

An oral carcinogenicity slope factor has not been derived for aluminum.

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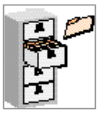
Yokel, R.A. and P.J. McNamara. 1985. Aluminum bioavailability and disposition in adult and immature rabbits. Toxicology and Applied Pharmacology. 77: 344-352.  **Retrieve Toxicity Profiles** **Condensed Version**

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