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Research Article

# THE GENERAL EFFECT OF ALUMINUM ON THE BODY

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## ABSTRACT

Aluminum is ubiquitous; the third most common element of the earth's crust. It is naturally released to the environment from the weathering of rocks and volcanic activity. Human activities such as mining also result in the release of aluminum to the environment. Aluminum levels in environmental media vary widely depending upon the location and sampling site. In general, background levels of aluminum in the atmosphere are low, typically ranging from about 0.005 to 0.18 µg/m3. Much higher levels are routinely observed in urban and industrial locations. Aluminum levels in surface water is usually very low (<0.1 mg/L); however, in acidic waters or water high in humic or fulvic acid content, the concentration of soluble aluminum increases due to the increased solubility of aluminum oxide and aluminum salts. Its concentration in soils varies widely, ranging from about 7 to over 100 g/kg.

## KEYWORDS

Aluminum, Aluminum salts, Aluminum oxide.

## INTRODUCTION

In the environment, aluminum exists in only one oxidation state (+3), and does not undergo oxidationreduction reactions. It can react with other matter in the environment to form various complexes. The fate and transport of aluminum is largely controlled by environmental factors such as pH, salinity, and the presence of various species with which it may form complexes. In general, the solubility and mobility of aluminum in soil is greatest when the soil is rich in organic matter capable of forming aluminum-

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organic complexes and when the pH is low, such as in areas prone to acid rain or in acidic mine tailings. The general population is primarily exposed to aluminum through the consumption of food items, although minor exposures may occur through ingestion of aluminum in drinking water and inhalation of ambient air. Aluminum found in over-the-counter medicinals, such as antacids and buffered aspirin, is used as a food additive, and is found in a number of topically applied consumer products such as antiperspirants, and first aid antibiotic and antiseptics, diaper rash and prickly heat, insect sting and bite, sunscreen and suntan, and dry skin products. The concentration of aluminum in foods and beverages varies widely, depending upon the food product, the type of processing used, and the geographical areas in which food crops are grown. Based on the FDA's 1993 Total Diet Study dietary exposure model and the 1987–1988 U.S. Department of Agriculture (USDA) Nationwide Food Consumption Survey, the authors estimated daily aluminum intakes of 0.10 mg Al/kg/day for 6–11-month-old infants; 0.30–0.35 mg Al/kg/day for 2–6-year-old children; 0.11 mg Al/kg/day for 10-yearold children; 0.15–0.18 mg Al/kg/day for 14–16-year-old males and females; and 0.10–0.12 mg Al/kg/day for adult (25–30- and 70+-year-old) males and females. Users of aluminum containing medications who are healthy (i.e., have normal renal function) can ingest much larger amounts of aluminum than in the diet, possibly as high as 12–71 mg Al/kg/day from antacid/anti-ulcer products and 2–10 mg Al/kg/day from buffered analgesics when taken at recommended dosages [1,2].

Gastrointestinal absorption of aluminum is low, generally in the range of 0.1–0.4% in humans, although absorption of particularly bioavailable forms such as aluminum citrate may be on the order of 0.5–5%. Although large bolus doses of as much as half a gram of aluminum as aluminum hydroxide throughout the day can be ingested during antacid therapy, absorption of aluminum hydroxide is usually ≤0.01% of the intake amount. Bioavailability of aluminum varies depending mainly on the chemical form of the ingested compound (i.e., type of anion) and the concurrent exposure to dietary chelators such as citric acid, ascorbic acid, or lactic acid. The total body burden of aluminum in healthy human subjects is approximately 30–50 mg. Normal levels of aluminum in serum are approximately 1–3 µg/L. Of the total body burden of aluminum, about one-half is in the skeleton, and about one-fourth is in the lungs.

There are numerous studies that have examined aluminum's potential to induce toxic effects in humans exposed via inhalation, oral, or dermal exposure. Most of these findings are supported by a large number of studies in laboratory animals. Occupational exposure studies and animal studies suggest that the lungs and nervous system may be the most sensitive targets of toxicity following inhalation exposure.

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Respiratory effects, in particular impaired lung function and fibrosis, have been observed in workers exposed to aluminum dust or fumes; however, this has not been consistently observed across studies and it is possible that co-exposure to other compounds contributed to observed effects. Respiratory effects (granulomatous lesions) have also been observed in rats, hamsters, and guinea pigs. There is concern that these effects are due to dust overload rather than a direct effect of aluminum in lung tissue. Occupational studies in workers exposed to aluminum dust in the form of McIntyre powder, aluminum dust and fumes in potrooms, and aluminum fumes during welding provide suggestive evidence that there may be a relationship between chronic aluminum exposure and subclinical neurological effects such as impairment on neurobehavioral tests for psychomotor and cognitive performance and an increased incidence of subjective neurological symptoms. With the exception of some isolated cases, inhalation exposure has not been associated with overt symptoms of neurotoxicity. A common limitation of these occupational exposure studies is that aluminum exposure has not been well characterized. The available animal inhalation studies are inadequate for assessing the potential for aluminum-induced neurotoxicity because the only neurological end points examined were brain weight and histology of the brain; no function tests were performed.

There is limited information on aluminum toxicity following dermal exposure. Application of aluminum compounds to the skin, such as aluminum chloride in ethanol or alum, may cause rashes in some people. Skin damage has been observed in mice, rabbits, and pigs exposed to aluminum chloride or aluminum nitrate, but not following exposure to aluminum sulfate, aluminum hydroxide, aluminum acetate, or aluminum chlorhydrate. There is a fair amount of human data on the toxicity of aluminum following oral exposure. However, the preponderance of human studies are in patients with reduced renal function who accumulated aluminum as a result of long-term intravenous hemodialysis therapy with aluminum-contaminated dialysis fluid and, in many cases, concurrent administration of high oral doses of aluminum to regulate phosphate levels (i.e., reduce uptake of phosphate by binding it in the gut) and have limited usefulness in predicting toxicity in the general population because the very large aluminum exposure levels and impaired renal function results in aluminum accumulation. Dialysis encephalopathy syndrome (also referred to as dialysis dementia) can result from this accumulation of aluminum in the brain. Dialysis encephalopathy is a degenerative neurological syndrome, characterized by the gradual loss of motor, speech, and cognitive functions. Another neurological effect that has been proposed to be associated with aluminum exposure is Alzheimer's disease. Although a possible association was proposed over 40 years ago,

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this association is still highly controversial and there is little consensus regarding current evidence. A number of studies have found weak associations between living in areas with elevated aluminum levels in drinking water and an increased risk (or prevalence) of Alzheimer's disease; other studies have not found significant associations. In contrast, no significant associations have been found between tea consumption or antacid use and the risk of Alzheimer's disease; although the levels of aluminum in tea and antacids are very high compared to drinking water, aluminum from these sources is poorly absorbed [3,4].

The available data do not suggest that aluminum is a causative agent of Alzheimer's disease; however, it is possible that it may play a role in the disease development. Aluminum is found in several ingested over-the-counter products such as antacids and buffered aspirin; clinical studies on health effects of aluminum medicinals in people with normal renal function have been identified. These aluminum-containing products are assumed to be safe in healthy individuals at recommended doses based on historical use. The assumed safety of aluminum is also partly due to the generally regarded as safe (GRAS) status of aluminum-containing food additives. However, there is some indication that adverse effects can result from long-term use of aluminum-containing medications in some healthy individuals. There are a number of case reports of skeletal changes (e.g., osteomalacia) in adults and children with normal kidney function due to long-term antacid use for the treatment of gastrointestinal disorders. These skeletal effects are secondary to hypophosphatemia and phosphate depletion caused by aluminum impairing phosphorus absorption by binding with dietary phosphorus [5,6].

There is a rather extensive database on the oral toxicity of aluminum in animals. These studies clearly identify the nervous system as the most sensitive target of aluminum toxicity and most of the animal studies have focused on neurotoxicity and neurodevelopmental toxicity. Other adverse effects that have been observed in animals orally exposed to aluminum include impaired erythropoiesis in rats exposed to 230 mg Al/kg/day and higher, erythrocyte damage (as evidenced by decreases in hemoglobin, hematocrit, and erythrocyte osmotic fragility, and altered erythrocyte morphology) in rats exposed to 230 mg Al/kg/day and higher, increased susceptibility to infection in mouse dams exposed to 155 mg Al/kg/day, delays in pup maturation following exposure of rats to 53 mg Al/kg/day, and decreases in pup body weight gain in rats and mice exposed to 103 mg Al/kg/day and higher. Neurodegenerative changes in the brain, manifested as intraneuronal hyperphosphorylated neurofilamentous aggregates, is a characteristic response to aluminum in certain species and non-natural exposure situations generally involving direct application to brain tissue, particularly intracerebral

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and intracisternal administration and in vitro incubation in rabbits, cats, ferrets, and nonhuman primates. Oral studies in rats and mice have not found significant histopathological changes in the brain under typical exposure conditions; however, altered myelination was found in the spinal cord of mouse pups exposed to 330 mg Al/kg/day on gestation day 1 through postnatal day 35. Overt signs of neurotoxicity are rarely reported at the doses tested in the available animal studies ( ≤330mg Al/kg/day for bioavailable aluminum compounds); rather, exposure to these doses is associated with subtle neurological effects detected with neurobehavioral performance tests. Significant alterations in motor function, sensory function, and cognitive function have been detected following exposure to adult or weanling rats and mice or following gestation and/or lactation exposure of rats and mice to aluminum lactate, aluminum nitrate, and aluminum chloride. The most consistently affected performance tests were forelimb and/or hind limb grip strength, spontaneous motor activity, thermal sensitivity, and startle responsiveness. Significant impairments in cognitive function have been observed in some studies, although this has not been found in other studies even at higher doses. Adverse neurological effects have been observed in rats and mice at doses of 100–200 mg Al/kg/day and neurodevelopmental effects have been observed in rats and mice at doses of 103–330 mg Al/kg/day [1,2,7,8].

A number of human studies have examined the occurrence of cancer among aluminum industry workers and found a higher-than-expected cancer mortality rate, but this is probably due to the other potent carcinogens to which they are exposed, such as polycyclic aromatic hydrocarbons (PAHs) and tobacco smoke. Available cancer studies in animals have not found biologically relevant increases in malignant tumors. The International Agency for Research on Cancer (IARC) concluded that aluminum production was carcinogenic to humans and that pitch volatiles have fairly consistently been suggested in epidemiological studies as being possible causative agents. The Department of Health and Human Services and EPA have not evaluated the human carcinogenic potential of aluminum.

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