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Potential Risk to Human from Environmental Aluminum Exposure

Antoine Al-Achi*
College of Pharmacy & Health Sciences, Campbell University, USA

Editorial

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***For Correspondence**

Antoine Al-Achi, College of Pharmacy & Health Sciences, Campbell University P.O. Box 1090, Buies Creek, NC 27506, USA

E-mail: alachi@campbell.edu

The element Aluminum (Al) is found in relatively high amounts in the earth's crust (about 7%) despite being non-essential for life. [1] However, Al is present mostly in its water insoluble form unless the pH of the soil is highly acidic (pH<5.0). Under acidic conditions, Al converts to its soluble form [1] and is mostly found in soluble fluoride or organic complexes.[2] Drinking water also contains an appreciable amount of Al, however its quantity in water varies with the region **(Figure 1).** [3] The presence of Al in drinking water is perhaps due to acid rain in industrial regions that permits insoluble Al to become soluble and enter the underground water reservoir.[4] Moir et al. proposed an empirical formula for estimating the concentration of exchangeable soil Al as a function of soil pH (this was based on soil found in Lees Valley, North Canterbury, New Zealand.) This relationship describes the amount of Al available for exchange with plants grown in soil, and it can be used as an indirect measure to quantify the amount of soluble Al in soil: [5]

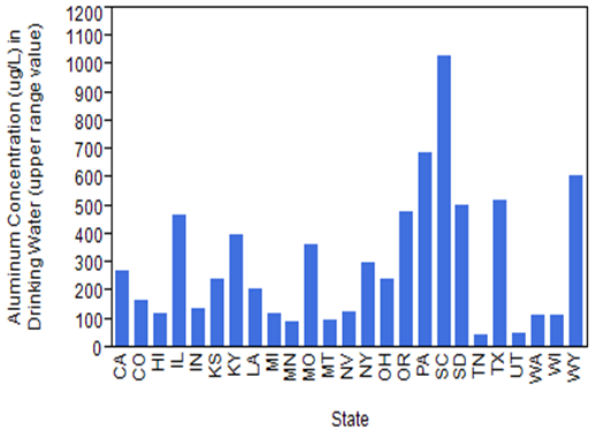

Figure 1. Estimated average aluminum content (µg/L) in drinking water in various states (USA). (Adopted from [3]).

Exchangeable Soil Al (me/100 g soil) =0.014+ {2.37/[1+ $(pH/5.5)^{23.5}$]}

Higher values for the exchangeable soil Al indicate greater availability of *soluble* Al in soil and in plants utilizing the soil. Similarly, ingested aluminum in food gets solubilized in acidic pH of the stomach, but later converts back to its insoluble form in the duodenum under relatively close to neutral pH values. [6] The rate of intestinal absorption of Al was reported to be around 0.2%.[7] Thus, it is expected that the amount of Al that reaches the blood compartment via intestinal absorption increases with the amount ingested. Once Al reaches the systemic circulation, it complexes with transferrin, and in turn this complex reaches the nervous system and the brain by endocytosis. [7]

Aluminum is considered to be a toxic metal in human. It causes a variety of health issues related to inflammatory reactions, [8-9] immune system toxicity, [10-13] and even increasing the risk for cancer,[14] among others. But, perhaps the most disturbing aspect of Al toxicity is the one related to the nervous system,[15] including the possible link to Alzheimer's disease (AD). [16-20],

experimental animal models have shown that Al administration resulted in a significant accumulation of amyloid-β protein in the brain tissues (commonly detected in the brain of AD patients). [21] It should be noted that 70% of AD cases are due to genetic predisposition, and only 30% are linked to environmental factors such as exposure to Al. [19] (Other environmental factors linked to AD include pesticides exposure, unhealthy air quality, and various industrial pollutants). [19] And, there is evidence to suggest that Al intake does accelerate the rate of AD in genetically predisposed individuals (experimental animal data) [8] Aluminum in drinking water has been suspected to be a risk for AD. For example, Rondeau et al. concluded that the presence of Al in drinking water in concentration greater than 100 µg/L was a significant risk factor for AD development (RR=2.20;95% CI=1.24–3.89) [16]**Figure 1** shows that the majority of states in the United States unfortunately meet that level [3].

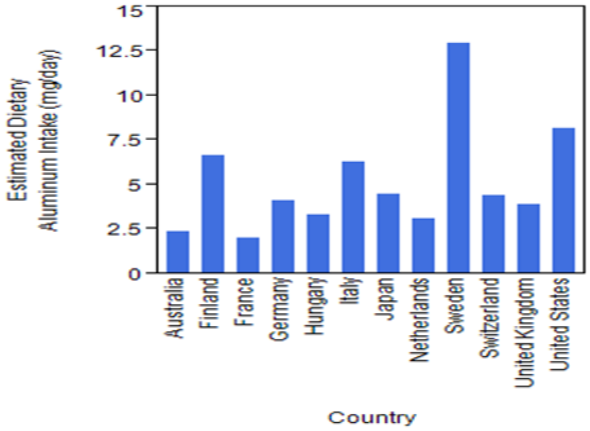

Figure 2. Estimated average dietary aluminum intake (mg/day) in different countries. (Adopted from [22,23]).

A recent meta-analysis on the exposure of Al indicated that there was an increased risk for AD by 71% in individuals who were *chronically* exposed to Al (OR=1.71;95% CI=1.35-2.18) [20]. In this meta-analysis study, chronic exposure was defined as concentration of Al in drinking water greater than 100 µg/L, a significant daily consumption of Al, or occupational exposure to Al. [20] It is important to note that the amount of Al consumed in drinking water is only approximately 4% of the total Al dietary intake. [22] **Figure 2** shows the dietary intake of Al in various countries.[22,23] Repeated exposure to Al in diet over time has been implicated in neurological damages similar to those prevailing in AD and other neurological degenerative diseases. [4,7,15] Aluminum exposure can also be the result of medications consumption (antacids) or occupational contact. (Vaccines also contain Al, however, unlike food and water the exposure to Al from vaccines is considered to be limited.) [10-13] In this context, meta-analysis studies could not find any association between *occupational exposure* to Al or chronic consumption of Al in *antacid formulations* with the development of AD. [24-25] On a positive note, various agents have been shown to limit the deleterious effect of *acute* administration Al on the cognitive functions. Among these agents that were shown to have a *protective* effect against the *acute* administration of a soluble aluminum salt (aluminum chloride) in animal models were black tea (*Camellia sinensis*), the antioxidant selenium, and the benzothiazepine calcium channel blocker drug, diltiazem. [26-28]

Although a cause-effect relationship has *not* been clearly established between Al exposure and the development of AD, all available evidence suggests that such an association might exist. Until a definitive answer to this relationship is found, it is recommended that Al exposure be minimized. To that end, drinking water that is low in Al content (<100 µg/L), limiting daily dietary Al intake to a minimum, avoiding aluminum-containing medications if possible (using non-Al-containing antacids), reducing occupational exposure to Al, and the inclusion of protective agents (selenium and black tea) in the diet might all be warranted to limit Al damaging effects on health.

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