Facts and Myths About Aluminum and Human Health Including Selective Chelators

by
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Aluminum (Al) is the most abundant metal and the third most abundant element – it makes up about 8% of the Earth's crust by weight.

Health authorities would like you to believe that the human body poorly absorbs Al, though at least 99.9% of all Al ingested from exogenous source food and water or drug containing Al simply passes through the digestive tract and is easily excreted by the body. This is different than what is observed with other metals. Several studies have demonstrated no adverse side effects for those who consumed large amount of aluminum-containing antacids, aspirin, vaccines, and even flour. However, long-term exposure to Al does have accumulating side effects, which is not a myth.

So, although a neutral atom of aluminum has 13^{3+} electrons, the ion of aluminum, Al^{3+}, has lost three electrons and has only 10.

An investigational study reports that Al^{3+} is neurotoxic, and to the brain metabolism is of clinical relevance. The normal amount and lethal toxicity in brain levels of Al^{3+} are well documented and differ only by a factor of 3-10. The normal brain uptake of Al^{3+} is estimated from data on intestinal uptake of Al^{3+} and brain uptake of radionuclides of similar ions when administered intravenously. The uptake is very slow, 1 mg in 36 years, and is consistent with the assumption that Al^{3+} ***taken up by the brain cannot be eliminated*** and ***is, therefore, accumulative***. The possibility that with time Al^{3+} may cause or contribute to some specific diseases, most of them related to aging, is discussed with the proposed metabolic picture in mind. A hypothetical model is presented for the metabolism, based on documented direct observations of Al^{3+} and analogies from other ions. The main characteristics of Al are low intestinal absorption, with rapid urinary excretion, and ***slow tissue uptake***, which ***mostly occurs in skeleton*** and ***reticuloendothelial cells***. Intracellular Al^{3+} is probably first confined in the lysosomes but then slowly accumulates in the cell nucleus and chromatin. Large, long-lived cells, e.g., ***neurons, may be the most liable*** and ***susceptible to the noted accumulation within the brain***. In heterochromatin, Al^{3+} levels can be found comparable to those used in leather tannage. It is proposed that an accumulation ***may take place at a sub cellular level*** without any significant increase in the corresponding tissue concentration (Ganrot, 1986).

The regulatory agencies of the United States and Canada have placed aluminum on priority lists for research designed to fill data gaps relating to neurotoxicity. This is to create a factual basis for the establishment of health standards for drinking water. In this review, we consider evidence for a significant role for aluminum in Alzheimer's Disease (AD). Aluminum has been implicated as a potential risk factor in AD and for elderly cognitive impairment by epidemiology studies of drinking water and a food study. Most people experience aluminum brain overload in the aging process. Aluminum levels over 20 times higher than those of a middle-aged group were found in a brain autopsy study of elderly persons, roughly correlating over the age period with densities of senile plaques and neurofibrillary tangles. Persons with AD have been found to experience increased

absorption of aluminum and higher blood levels. More controversially, the majority of brain studies also show elevated aluminum levels, though there is disagreement over location of metal buildup. Clinical intervention to lower brain aluminum by chelation has slowed the progression of AD (Jansson, 2001).

Aluminium is the most widely distributed metal in the environment and is extensively used in daily life, which provides easy exposure to human beings. The exposure to this toxic metal occurs through air, food, and water. However, there is no known physiological role for aluminum within the body and hence this metal may produce adverse physiological effects. Chronic exposure of animals to aluminum is associated with behavioral, neuropathological, and neurochemical changes. Among them, deficits of learning and behavioral functions are most evident. Some epidemiological studies have shown poor performance in cognitive tests and a higher abundance of neurological symptoms for workers occupationally exposed to aluminum. However, in contrast to well established neurotoxic effects, neurobehavioral studies of aluminum in rodents have generally not produced consistent results. Current research shows that any impairment in mitochondrial functions may play a major role in many human disorders, including neurodegenerative disorders. Being involved in the production of reactive oxygen species (ROS) causing oxidative stress and damage, aluminum may cause impairments in mitochondrial bioenergetics and may lead to the generation of oxidative stress, which may lead to a gradual accumulation of oxidatively modified cellular proteins. In this review, the neuropathologist associated with aluminum exposure in terms of neurobehavioral changes have been discussed. In addition, the impact of aluminum on the mitochondrial functions has also been highlighted (Kumar & Gil, 2009).

The most relevant neurotoxic metals – aluminum, lead, manganese, mercury, cadmium, arsenic, bismuth, and tin, transport of metals across the neural barriers, and their potential role in diseased human brains is documented. The role of metal exposure and disturbance of metal homeostasis in the brain can be a root cause of neurodegenerative diseases like Alzheimer's or Parkinson's (Michalke et al., 2009). Aluminum may mediate Alzheimer's disease through liver toxicity, with aberrant hepatic synthesis of ceruloplasmin and ATPase7B, the resultant excess of free copper causing brain oxidation, beta-amyloid aggregation, and Alzheimer’s disease (Brenner, 2013).

Evidence for the neurotoxicity of extended exposure to low levels of aluminum salts is described using an animal model treated with aluminum at low levels reflecting those found in some water supplies. Emphasis is given to the potential role of aluminum in acceleration and promotion of some indices characteristic of brain aging. These hallmarks include the appearance of excess levels of inflammation in specific brain areas. Aluminum salts can increase levels of glial activation, inflammatory cytokines, and amyloid precursor protein within the brain. Both normal brain aging and Alzheimer's disease is associated with elevated basal levels of markers for inflammation. These are not attributable to obvious exogenous stimuli and may reflect the lifespan history of the organism's immune responses. It is possible that aluminum salts can act as a subtle promoter of such apparently unprovoked responses. Yet to date, the neurotoxicity of environmental aluminum is still an issue (Bonde, 2010).

Several individual metals, including aluminum, arsenic, cadmium, lead, manganese, and mercury were demonstrated to affect the neurological system. Metals are ubiquitous in

the environment. Environmental and occupational exposure to one metal is likely to be accompanied by exposure to other metals, as well. It is, therefore, expected that interactions or "joint toxic actions" may occur in populations exposed to mixtures of metals or to mixtures of metals with other chemicals. Some metals seem to have a protective role against neurotoxicity of other metals, yet other interactions may result in increased neurotoxicity. For example, zinc and copper provided a protective role in cases of lead-induced neurotoxicity. In contrast, arsenic and lead co-exposure resulted in synergistic effects. Similarly, information is available in the current literature on interactions of metals with some organic chemicals such as ethanol, polychlorinated biphenyls, and pesticides. In depth understanding of the toxicity and the mechanism of action (including toxicokinetics and toxicodynamics) of individual chemicals is important for predicting the outcomes of interactions in mixtures (Pohl et al., 2011).

Medicinal Embryonic Phytotherapy (MEP™) for the selective chelation of Al leaving unscathed the necessary essential minerals. It's bad enough we are being sufficiently polluted from exogenous environmental sources – can we afford to further strip our body from these most essential minerals using non-selective chelators?

Embryonic Plant Extracts (EPEs) Selective Chelator of Al includes:

- Ash – Fraxinus Excelsior (buds) adult dose 25 drops 3 times daily.
- Beech – Fagus Sylvatica (buds), the ***sulfur*** plant, adult dose 15-25 drops 3 times daily.
- Bilberry – Vaccinium Myrtillus (young shoots and embryonic fruits) adult dose 15-25 drops 3 times daily.
- Black Currant – Ribes Nigrum (buds) adult dose 15-25 drops 3 times daily.
- Black Elder – Sambucus Nigra (buds) adult dose 15-25 drops 3 times daily.
- Black Poplar – Populus Nigra (buds) *Polycrest* adult dose 15-25 drops 3 times daily.
- Bramble – Rubus Fruticosus (young shoots) adult dose 15-25 drops 3 times daily.
- California Poppy – Eschscholzia Californica (buds) *Polycrest* adult dose 15-25 drops 3 times daily.
- Crab Apple – Malus Sylvestris (buds) *Polycrest* adult dose 15-25 drops 3 times daily.
- Propolis Blend 15 drops 3 times daily.
- Sour Cherries Montmorency – Prunus Cerasus (buds) *Polycrest* adult dose 15-25 drops 3 times daily.
- Virginia Creeper – Ampelopsis Veitchii (young shoots) *Polycrest* adult dose 15-25 drops 3 times daily.
- Wheatgrass – Triticum Aestivum (germinating seeds) adult dose 15-25 drops 3 times daily.

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