Aluminum Toxicity of the Brain

BACKGROUND

The connection to metal toxicity in the brain is proven by aluminum’s presence in the neurons (brain cells) of autopsied patients who had suffered from Alzheimer’s disease. Aluminum is the most commonly found toxic metal that permeates such affected brains.

In 1897, aluminum was analyzed for pathological reaction in animals and reported to be a selective neurotoxin. Thereafter it was recognized as a human poison that caused loss of memory, jerking movements, and impaired coordination.

In 1980, Drs. D.P. Perl, and P.F. Good, two neuropathologists at Mt. Sinai Medical Center in New York City, discovered that not only did aluminum show up generally in the brains of Alzheimer’s disease victims, but it was also present in precisely those tangled brain cells that characterized the disease. Daniel Perl, M.D., found a baffling wave of degenerative brain disease in the Pacific Island of Guam. About 10 percent die of amyotrophic lateral sclerosis (ALS, or Lou Gehrig’s disease) and parkinsonism with dementia (PD).

There are high levels of aluminum in the drinking water and in nineteen common foods of Guam and Rota. Dr. Perl repeatedly has found the metal in the brains of deceased Mariana islanders who had been afflicted with ALS and PD.

Low calcium and high aluminum concentrations in the soils, waters, and native foods are strongly suspected. Amounts of elemental aluminum and calcium were measured in foods such as taro, yam, arrowroot, cassava, radish, turmeric, pepper, banana, coconut, melon, pumpkin, papaya, mango, breadfruit, pandanus, guava, achote, betel nut, and cycad. Taro contained the highest aluminum content and next were yam, arrowroot, radish, turmeric, peppers, and pandanu. They each had at least a concentration in excess of 100 micrograms aluminum per gram dry weight of the sample.

ALUMINUM INGESTION

Ingestion of aluminum in any form is dangerous for the brain. Aluminum is known to be a potent cross-linking agent that acts to immobilize reactive molecules within brain cells. It also causes free radical pathology inside the neurons.

Cross-linkage abnormally develops and brings on cellular damage.
Nucleic acid molecules that form part of the DNA become damaged. Such damage leads to mutations or to cellular death in the brain. As brain cells die, mental abilities of the affected person decrease.

Free-radical pathology occurring from bombardment of the brain by additional aluminum ions, penetrating the blood-brain barrier increases neuronal damage and advances dementia. Free-radical formation can be stopped by the neutralizing or quenching effect of antioxidants, such as selenium and vitamin E.

The stages of initiation and propagation are followed by the stage of termination, at which the free radicals are neutralized either by nutrient-derived antioxidants, by enzymatic mechanisms, or by recombination with each other.

One of the primary sources of free-radical activity in brain tissue is lipid peroxidation. Lipid peroxidation is pathology in tissues that takes place when lipids (fats) of the body actually turn rancid from oxidation. It requires the presence of abnormally located metal ions, such as iron, aluminum, copper, lead, etc. The brain is vulnerable because of its high concentration of biologically active lipids in the cellular membranes of neurons. An individual’s ongoing ingestion of the polyunsaturated fats increases lipid peroxidation to produce not only heart disease but also brain deterioration.

THE ALUMINUM / DEMENTIA CONNECTION

Donald R. McLaughlan, M.D., Professor of Physiology and Medicine, at the University of Toronto, and his co-workers, caused a replication of the entire sequence of symptoms of Alzheimer’s disease in cats. By a single injection of 100 nanomoles (one-billionth of a mole) of aluminum chloride into the hippocampic space, referred to as a brain ventricle, an area located in the hippocampus of the cat’s brain, they caused the cats to develop a dementia similar to Alzheimer’s.

Dr. McLaughlan and his coworkers watched the cats’ reactions to the aluminum chloride injections. No change was observed during the first week following treatment, but at the ninth day the cats began to fail to remember where they had seen food hidden. Approximately ten days after that the animals’ short range memory was completely gone.

Even more impressive study that Dr. McLaughlan performed involved his injecting aluminum chloride into a subcutaneous area of the cats’ skin covering the abdomen. After ten days the felines developed a progressive dementia.

The researcher proved that similar abnormal responses take place in laboratory cats no matter where the aluminum is placed in their bodies.

“Concentrations of aluminum that are toxic to many biochemical processes are found in:

a. senile and presenile dementia of the Alzheimer type
b. Down’s syndrome with Alzheimer disease

c. Guam parkinsonism-demential complex

d. Guam amyotrophic lateral sclerosis.

Each of these brain diseases is linked by two important markers:

1) neurons exhibit neurofibrillary degeneration composed of aggregates of 10 nm paired helical filaments

2) neurons with neurofibrillary degeneration contain elevated concentrations of aluminum.”

Dr. McLaughlan’s conclusions were confirmed and broadened by other investigators around the world.

Upon autopsy of the cats in both types of studies impartial investigators performed histochemical and microscopical studies. The aluminum was found to be preferentially absorbed by the chromatin in the pyramidal neurons in the hippocampic areas of the feline brains.

Rabbits injected in the brain ventricular system with aluminum salts or powder recover from the operation within an hour and develop clinical symptoms inside of two weeks after the injection. They show neurofibrillary degeneration with general motor inhibition and pronounced ataxia.

From ataxia brain studies on animals it has been established that aluminum is associated with the nuclei of neurons in neurofibrillary degeneration in senile human brains associated with Alzheimer’s disease.

Immunologic tests on animals have confirmed the chemical similarity between abnormal, tubular, intracellular growths in Alzheimer neurons and similar tubules induced in laboratory animals by the administration of aluminum in their daily diet and by injection into their skin.

ALUMINUM IN THE HUMAN BODY AND BRAIN

When aluminum is inhaled or consumed by a person or absorbed through the skin and scalp, or ingested in some other way, it is metabolized in the body, transported through the blood stream, and either excreted in the feces and bile or absorbed into the brain cells. There is an age related increase in the level of aluminum absorption in several tissues.

In elderly people such blood brain barrier permeation takes place with certain amino acids, with peptides especially: the N-Tyr-delta-sleep-inducing peptide and the beta-endorphin. These two peptides combine with aluminum molecules placed in the body from the use of aluminum foil and cookware. Natural enzymes become body poisons by absorption of high technology aluminum.
Other studies indicate that aluminum and the ions of the heavy metals cadmium and manganese join to inhibit the certain brain enzymes that are the source of cellular energy. The result is that enzyme inhibition in a person causes brain cell energy and reasoning ability to diminish, and memory to fade.

Aluminum, cadmium, and manganese are potent inhibitors of the uptake of choline and dopamine by membrane bound sacs in the brain known as the synaptosomes. Choline and dopamine are vital brain chemicals released by nerve endings to transmit impulses across synapses to other nerves. The toxic metals bring about adverse effects for the brain’s neurotransmission of thoughts, reasoning, and short term memories.

There is strong evidence that alterations in the enzymes involved with acetylcholine metabolism are caused by the ingestion of aluminum. An associated motor lack takes place with reduced coordination, or non-transfer of the thought. There will be a reduction or prevention of the movement of nerve signals and ataxia is the disease symptom.

Increased levels of aluminum in the brain also affect the catecholamine neurotransmitters, noradrenaline and dopamine, in the frontal cortex, hippocampus, and cerebellum. The neurotoxicity of aluminum depends on the dietary intake of other metal nutrient ions such as copper, zinc, iron, and magnesium. Both noradrenaline and dopamine levels in the cortex and cerebellum decrease when there is a copper deficiency in the diet.

ALUMINUM NEUROENCEPHALOPATHY

Alzheimer’s disease is characterized by the presence of numerous senile plaques and neurofibrillary tangles within the neocortex and hippocampus. The neurofibrillary tangles comprise groups of pairs of twisted filaments 22 nanomoles in diameter with a constriction down to 10 nanomoles with every 80 nanomoles. These abnormal filaments have a substructure of six protofilaments. While the absolute causes of these twisted filaments, neurofibrillary tangles, and senile plaques remain unknown, toxic substances, and most especially aluminum, are strongly implicated.

In autopsy examinations of the brains, Dr. McLaughlan found concentrations of aluminum, which he had found to be cytotoxic in cats and rabbits during his earlier laboratory experiments. Similar amounts of the metal were not detected in the brains of neurologically normal patients. There was a significant elevation of aluminum in the brain with age.

Studies of human autopsy material by using a combination of X-rays and electron microscopes have led to claims by research pathologists of a very precise localization of aluminum in the nuclei of neurons containing tangles. The investigations affirm that aluminum is localized only in those brain cells that have undergone neurofibrillary degeneration.
THE NEUROTOXIC EFFECTS OF ALUMINUM

Neuropathological changes caused by high concentrations of aluminum show themselves in the central nervous system. The conclusion from animal experiments using powdered aluminum is that the neurotoxic effect produced is dose related.

The major lesion seen in the brain cells of animals that develop Alzheimer’s disease is the neurofibrillary tangle.

Aluminum does influence the properties of enzymes in the synaptosomal membranes and also affects enzymes involved in neurotransmitter metabolism. Neurofibrillary degeneration occurs in regions of the brain showing pronounced deficits in the activities of ChAT and AChe.

Aluminum lodged in the brain has an affinity for cancer causing agents. Extensive studies of aluminum on DNA reveal certain changes in the binding of carcinogenic complexes because of interactions between DNA and aluminum. The investigator saw DNA template damage, a precursor to brain cancer. The metal inhibits the coding reaction for genes that determine the formation of enzymes involved in the metabolism of neurofibrillary proteins.

ALUMINUM / ALZHEIMER’S CONNECTION

Dr. McLaughlan showed that concentrations of aluminum were localized in several areas of the brain. Another group of researchers led by William R. Markesberry, M.D., chairman of the medical and scientific advisory board of the Alzheimer’s Association, said that the aluminum content of demented brains was not focalized at all but generally was diversified in the tissues. Dr. Markesberry pointed out that the patients sampled by Dr. McLaughlan came from the Toronto region of Canada where alums are used by the majority of water treatment plants for producing a settling of sediment. Markesberry population samples taken from rural eastern Kentucky where the use of aluminum compounds in the water supply is rare. All of the scientific community did conclude that Alzheimer’s disease dementia is associated with several factors such as age, environmental effects, and the presence of toxic metals in the affected individual’s metabolism. Neurotoxicity of aluminum is increased by the dietary intake of various metallic ions such as insufficient concentrations of zinc and excessive intake of arsenic, cadmium, iron, lead, manganese, mercury, or additional toxic metals. There is an age related increase in the concentration of aluminum in many tissues. When the patients’ diet was supplemented with common, over-the-counter, aluminum containing antacids, the toxic metal was retained in the patients’ metabolism.
Much of the laboratory research for Alzheimer’s disease is carried out on three month old rabbits, rats, or mice. In human tissues there is this definite age related increase in the concentration of aluminum.

The general consensus among scientists is that aluminum intoxication is not a primary event in the Alzheimer’s syndrome, but most certainly it is a secondary feature. Dr. McLaughlan postulates that some pathological occurrence alters the blood brain barrier to be more receptive to the metal and allows aluminum deposits in the victim’s brain cells. The toxic metal gains access to neuronal chromatin and lethal effects result.

References


