

Letters

Aluminum Toxicity

The hazards of aluminum ingestion have become apparent primarily in studies with dialysis patients having impaired kidney function. Recently a report in the German literature has come to our attention which presents convincing direct evidence that, even in individuals with normal kidney function, aluminum accumulates in tissues. This study by Zumkey, et al, involved a group of 40 patients with normal kidney function from whom brain tissue samples could be obtained because they required brain surgery. Ten patients received preoperatively an antacid with a high aluminum content, a second group of ten received an antacid with a low aluminum content. Both groups received the antacid for 10 days prior to surgery. Twenty patients did not receive any antacid and served as a control group. Brain Al levels for the three groups were as follows:

Al rich $1.05 \pm 0.31 \text{ ug Al/g}$, Alpoor $0.412 \pm 0.100 \text{ ug Al/g}$ and control $0.583 \pm 0.087 \text{ ug Al/g}$.

A further study was carried out by these investigators in which aluminum levels were measured in bone samples from patients undergoing bone biopsies. Patients were divided into a group of twenty who received an Al rich antacid and another group which received an Al poor antacid for a period of four weeks prior to biopsy. The bone Al for the Al rich group was $4.09 \pm 0.531 \text{ ug Al/g}$ while the level for the Al poor group was $1.71 \pm 0.271 \text{ ug Al/g}$. A control group which had no antacid gave a mean $1.71 \pm 0.134 \text{ ug Al/g}$.

The data clearly shows that even in patients with normal renal function, tissue aluminum concentration increases in a relatively short period of time upon ingestion of aluminum rich antacids.

The ubiquitous nature of aluminum is evident in preparations such as antacids, phosphate binders, anti-diarrhea medication, deodorants, as well as drinking water, beer, wine and juices where Al salts such as bentonite are used as

clarifying agents. In the light of this study, it is advisable to minimize the use of aluminum containing preparations. Aluminum is a far greater toxic metal than has previously been assumed.

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References

Von H. Zumkley, H.P. Bertram, M. Brandt, M. Roedig, S. Spieker and Kisters. Aluminum concentration in bone and brain after giving antacids. *Fortschritte der Meclizine*, 105:S19., 15-17, 1987.

BioMembrane Engineering with AL 721

A Nontoxic Membrane Based Therapy for AIDS, Addiction, Cystic Fibrosis and other Chronic Degenerative Diseases

Many cellular functions are inhibited when the fluidity of cellular membranes is reduced. This usually occurs with an increase in cholesterol content, and a decrease of phospholipid content of cellular membranes^{1,2}.

One pathological condition that has been associated with membrane rigidity is drug addiction and withdrawal³. Drug tolerance and physical dependence are believed to be strongly associated with adaptation processes of nerve cell membranes to the drug. It is now generally accepted that the adapting mechanism for drugs such as ethanol and barbiturates is the lipid matrix of the cell membrane. It has been shown by several investigators^{4,5} that the acute affect of ethanol or barbiturates is the fluidization of brain membranes, namely melting and expanding of the lipid constituents thereby disordering the lipid structure of the membrane. When given chronically, these drugs, by a process of homeoviscous adaptation, increase the cholesterol content of erythrocytes and of brain neuronal membranes — thus counterbalancing the fluidizing of the drug.

A similar course of events occurs in the brain neurons of rats chronically treated with morphine. When the drug is withdrawn the brain membranes remain hyperviscous, probably resulting in the unmasking of latent receptors and other proteins^{6 7} which are embedded in the neural membranes. Supersensitivity results and is expressed in the abstinence syndrome.

When animals are treated experimentally to reduce membrane cholesterol levels, withdrawal symptoms are markedly reduced. Such treatment has been achieved with a complex mixture of phospholipids and neutral lipids as naturally occurring from a simple solvent extract of egg yolk^{3 8}.

The membrane fluidizing mixture derived from egg yolks had been developed and tested *in vitro* and *in vivo* in both animals and humans and found to be valuable in rehabilitation of drug addicts⁸, and in the treatment of AIDS (acquired immune deficiency syndrome)⁹.

Just recently an open clinical trial with this egg lipid extract (AL 721 - standing for active lipids in ratio of 7 parts neutral lipids, 2 parts phosphatidyl choline and 1 part phosphatidyl ethanolamine) finds substantial clinical improvement in AIDS and ARC patients¹⁰. The formulation is considered nontoxic and similar formulations are being utilized by AIDS patients all over the world.

1. I suspect that this process of hyperrigidification of neuronal membranes associated with drug addiction is also associated with certain types of chemical allergy-addictions especially with those fat soluble pesticide compounds which will come to reside in cellular membranes.
2. As in drug addiction, the condition is treatable and reversible with nontoxic natural therapies: in this case a phospholipid mixture as outlined and discussed above.
3. I have prepared such a mixture and am supplying to People with Aids (PWA) groups all over the country and we are now starting to get preliminary data concerning its effects in AIDS.
4. I propose a clinical study with chemical-sensitive patients (recalcitrant to other therapies) using a suitable egg lecithin

material (i.e. equivalent to the AL721 used in the referenced studies).

5. I have reproduced this material and had it qualified by independent laboratory analysis (Adams Lab in New York City) and wish to work with physicians in testing my hypothesis.

I have already presented this material to The Society for Biochemical Intolerances and to the Well Mind Association in Seattle and to the American Academy for Medical Preventives.

Sincerely,

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