Aluminum and disease: Another indictment

Aluminum, one of the most abundant elements in the earth's crust, was long thought to be innocuous to the human body. Then two studies linked abnormally high levels of it with senile dementia. And now a third, reported in the Sept. 10 SCIENCE, ties excessive amounts of aluminum not just to senile dementia but also to another degenerative nerve disease called amyotrophic lateral sclerosis.

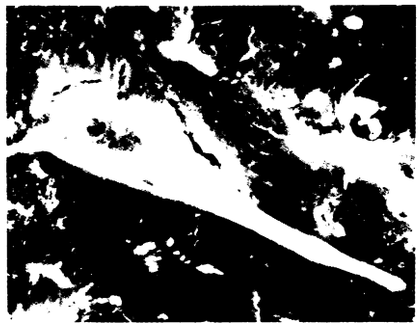
The latest study was conducted by Daniel P. Perl at the University of Vermont College of Medicine in Burlington, in conjunction with D. Carleton Gajdusek, Ralph M. Garruto, Richard T. Yanagihara and Clarence J. Gibbs Jr. of the National Institute of Neurological and Communicative Diseases and Stroke in Bethesda, Md. They used an especially sensitive analytical method — scanning electron microscopy with X-ray spectrometry — to analyze levels of aluminum in brain neurons from one victim of amyotrophic lateral sclerosis, from two victims of severe dementia associated with Parkinson's disease and from five neurologically normal patients who had died at a relatively young age from causes other than neuronal diseases. All eight subjects had been lifelong residents of Guam, one of three areas in the world known to have an unusually high incidence of both amyotrophic lateral sclerosis and parkinsonism-dementia and also known to contain high levels of aluminum in gardening soil and drinking water. (The other two areas are the Kii Peninsula of Japan and southern West New Guinea.) In addition, nearly all of the neurons from the amyotrophic lateral sclerosis patient and from the parkinsonism-dementia patients contained neurofibrillary tangles, whereas four out of five control subjects had neurons free from such tangles. Neurofibrillary tangles in brain neurons are hallmarks of amyotrophic lateral sclerosis and parkinsonism-dementia. The fifth control subject showed extensive tangles in his neurons despite the absence of neuronal disease; the tangles might have led to neuronal disease had the subject lived longer.

Three to four times more aluminum was found in the neurons of the amyotrophic lateral sclerosis patient and in the parkinsonism-dementia patients than in the neurons of the first four control cases, Perl and his team found. In contrast, there was about twice as much aluminum in the neurons of the fifth control case that contained lots of neurofibrillary tangles as there was in the neurons of the four cases that did not. These findings, they conclude, "further extend the association between intraneuronal aluminum and neurofibrillary tangle formation" and suggest that such tangles in turn can lead to neuronal diseases such as senile dementia and amyotrophic lateral sclerosis.

The first connection between aluminum and senile dementia was made in the 1970s by Donald R. McLachlin of the University of Toronto and colleagues. They used atomic absorption spectrometry to analyze the content of aluminum in brain samples from eight healthy persons and from 16 senile ones. Four times as much aluminum was found in the neurons of senile brains as in the neurons of healthy brains, and the aluminum was especially prevalent in areas of the senile brain rich in neurofibrillary tangles (SN: 10/1/77, p. 219).

The second link between aluminum and senile dementia was made by Perl in 1980, along with Arnold R. Brody of the National Institute of Environmental Health Science in Research Triangle Park, N.C. They used scanning electron microscopy in conjunction with X-ray spectrometry to examine aluminum levels in both senile brains and normal aged brains. They detected an abnormally high amount of aluminum in neurons bearing neurofibrillary tangles in both the senile and normal aged brains, yet virtually none in normal neurons from both types of brains. These findings, they contended, further implicated aluminum in senile dementia (SN: 4/19/80, p. 246).

—*J. A. Treichel*



Perl et al.

A brain neuron containing a neurofibrillary tangle (pictured) also contains aluminum.

Progress in paralysis treatment

Clonidine, a drug previously prescribed for high blood pressure, appears to lessen paralysis that occurs after spinal cord injury, according to a newly released study.

While reports in the past (SN: 11/7/81, p. 293) have shown that other drugs help paralysis symptoms, these drugs have to be given immediately after spinal cord injury. But clonidine, according to a report in the Sept. 10 SCIENCE, appears to work even when given up to seven weeks after the injury has occurred.

"We have one cat that was treated immediately after injury — it's walking and jumping," says N. Eric Naftchi, professor of rehabilitation medicine at New York University. "And I have others that were treated later that are doing the same thing." Naftchi says that although clonidine works when given weeks after spinal cord injury, the best results were obtained when cats were treated right away. But, he adds, treated cats all showed some motor and sensory return.

Naftchi gave clonidine at different time intervals to 17 mongrel cats with spinal cord injuries. Thirty cats with similar injuries served as a control group and were not treated. To determine the level of injury, Naftchi stimulated a specific nerve and measured the response, called a somatosensory evoked potential (SEP). Before clonidine treatment, cats showed a total absence of SEP, which means that spinal cord nerves were completely disrupted. After treatment, all clonidine-treated cats showed a complete return of SEPs and Naftchi reports six cats treated with clonidine from two to seven weeks after injury, "walked with complete return of sensory and motor functions, including those of bladder and bowel." The untreated control cats tested up to four months after injury never showed SEP return, and Naftchi reports all 30 controls eventually deteriorated to a chronic stage of paralysis.

According to recent estimates, 10,000 Americans are paralyzed each year after automobile or sporting accidents in which they damage the spinal cord. Naftchi says that preliminary clinical trials with clonidine are encouraging; paralyzed patients given the drug apparently feel some heat and pain sensation.

"The findings of this report are potentially of great importance, [and] if confirmed by other labs, they could significantly affect the treatment of spinal cord injury as well as our concept of the mechanism of spinal cord injury," says Alan I. Faden, a neurologist at the Uniformed Services Medical Center in Bethesda, Md.

Spinal cord injuries set off a poorly understood chain reaction in which blood flow to the injured area is reduced causing cell death and scar formation. Research in the past has focused on ways of stopping this progressive reaction before much damage occurs. Thus, drugs, such as naloxone, an opiate antagonist, were given immediately — usually within a few hours after injury. Most researchers believed that damage to the spinal cord could not be reversed once it had occurred. But Naftchi's results seem to challenge this notion because clonidine seems to alleviate paralysis even when administered weeks after the injury.

Naftchi says he doesn't know how the drug works but says clonidine might cause severed nerve fibers to grow through scar tissue that blocks nerve regrowth.

Scientists reached for comment say Naftchi's findings need to be confirmed.

—*K. A. Fackelmann*