trace elements in medicine

An International Journal

Volume 8 – 1991

Dustri-Verlag
Dr. Karl Feistle
München-
Deisenhofen

LETTER TO THE EDITOR

Mercury as a potential source for the etiology of Alzheimer's disease

Sir, – The pathogenesis of Alzheimer's disease (AD) is unknown. Heredity, infections with slow virus, aluminum poisoning etc. have been discussed as possible sources for the disease. Recent studies suggest that certain complexes forms of Hg^{2+} must be considered as a potential source for the etiology of Alzheimer's disease.

An American research team at the University of Kentucky has done extensive work on Alzheimer's disease [Editorial 1991, Wenstrup et al. 1990]. They published their first studies in 1986 and 1987. Utilizing Instrumental Neutron Activation Analysis (INAA), the research team determined quantities of 18 elements in brains from patients with Alzheimer's disease and age-matched controls. The most consistent alteration in the patient group were elevations of mercury and bromine, and depletions of rubidium.

The American research team have recently published their last findings [Wenstrup et al. 1990]. They have in this study determined quantities of 13 trace elements (Ag, Br, Co, Cr, Cs, Fe, Hg, K, Na, Rb, Sc, Se, Zn) in brains from 10 autopsied patients with Alzheimer's disease (ages 59 – 93) and 12 controls (ages 59 – 83). Analysis from the patient group show the following: elevated bromine and reduced rubidium in whole brain fractions; diminished selenium, elevated mercury and reduced rubidium in microsomal fractions; reduced zink and rubidium in nuclear fractions. The analysis showed also increased Hg/Se and Hg/Zn ratios in AD microsomal fractions; increased Hg/Se ratio in AD nuclear fractions; and increased Zn/Se ratio in AD mitocondrial fractions.

Most important in the study [Wenstrup et al. 1990] is the elevation of mercury. There was a significant increase of mercury in AD bulk brain samples, particularly in the cerebral cortex compared to controls (31.4 vs. 17.5 ng/g, fresh weight basis). Important is also the elevation of mercury in the nucleus basalis of Meynert (nbM) of AD victims compared to controls (39.3 vs. 8.9 ng/g, fresh weight basis). The nucleus basalis of Meynert is the major cholinergic projection to cerebral cortex [Editorial 1991, Wenstrup et al. 1990]. The nbM is severely degenerated in patients with Alzheimer's disease. The altered Hg/Se and Hg/Zn ratios are of great interest since selenium and zinc have a protective role against mercury toxicity [Ziff 1987].

The tubulin synthesis is impaired in human AD brains. Researchers [Duhr et al. 1991] have shown that low micromolar levels of Hg^{2+} blocked interactions of tubulin-GTP formula in central human brain homogenates. Aluminum had no effect compared to mercury. Duhr and co-workers [Duhr et al. 1991] have recently registered that brain samples from mercury-fed rats display an abolished GTP-tubulin interaction similar to AD brain samples. Less tubulin was found in the areas of the AD brain with highest accumulation of mercury. The authors concluded: "These results suggest that certain complexes of Hg^{2+} must be considered as a potential source for the etiology of Alzheimer's disease.

The release of mercury from dental amalgams makes the predominant contribution to human exposure to inorganic mercury and vapor in the general population [Clarkson et al. 1988]. Professor Lars Friberg and co-workers [Friberg et al. 1986] revealed a direct correlation between the amount of inorganic mercury in the brain and the number and surfaces of amalgam fillings. The above named researchers of Alzheimer's disease noted dental amalgams as a primary potential source of the high levels of mercury in AD brains. Mercury from dental amalgams passes rapidly and directly into body tissues and accumulates in the patients bodies with time.

No exposure to mercury vapor can be considered totally harmless, since mercury vapor has no toxic threshold [Bjørklund 1991]. Dental amalgams cannot be excluded as a primary potential source of Alzheimer's disease.

G. Bjørklund
Toften, 24 N-8610 Grubhei, Norway

REFERENCES

Bjørklund G 1991 Kvikksolv i tannhelsepersonalets arbeidsmiljo. Tidsskr Nor Lageforen *111:* 948-951

Clarkson TW, Friberg L, Nordberg GF, Sager P 1988 Biological monitoring of metals. Plenum Press, New York

Duhr E, Pendergrass C, Kasarskis E, Slevin J, Haley B 1991 Hg^{2+} induces GTP-tubulin interactions in rat brain similar to those observed in Alzheimer's Disease. Federation of American Societies for Experimental Biology (FASEB). 75th Annual Meeting. Atlanta, Georgia 21-25 April 1991. Abstract 493

Editorial 1991 Mercury and Alzheimer's Disease (AD) "These results suggest that certain complexed forms of Hg^{2+} must be considered as a potential source for the etiology of AD." Bio-Probe Newsletter *7:* 3-5

Friberg L, Kullman L, Lind B, Nylander M 1986 Kvicksilver i centrala nervystemet i relation til amalgamfyllingar. Läkartidningen *83:* 519-522

Wenstrup D, Ehmann WD, Markesbery WR 1990 Trace Element Imbalances in Isolated Subcellular Fractions of Alzheimer's Disease Brains. Brain Research *553:* 125-131

Ziff S, Ziff M 1987 Infertility & Birth Defects. Bio-Probe Inc, Orlando

Trace Elements in Medicine 1991; 8: 208

CP*CURRENTS*

1992

March
Volume 2, Number 3

Published by ITServices
3301 Alta Arden #3
Sacramento, CA 95825 USA
(916)489-4400 (CA)
(800)422-9887 (outside CA)

ISSN# 1055-4483
(printed on recycled paper)

usually very simple. 3. If additives fall below 500 parts per million, they do not have to be put on the label so labels can be deceptive. 4. The first 3 ingredients make up 90% of the diet. Be cautious of food that starts off with grain products. 5. Where did the vital ingredients of the food come from. Are they from condemned, contaminated food that is not fit for human consumption? 6. Beware of state laws. In California it is against the law to use condemned or contaminated products in pet foods. 7. Byproducts may or may not be good for the animal. For example, organs not sold for human consumption such as spleen or lung may be acceptable in pet food. 8. Avoid the additives BHA, BHT and ethoxyquin. **15006**

"The Allergy Connection: Is Junk Food The Source?", Plechner, Alfred J., DVM and Rugg, Leslie, Veterinary Forum, January 1992;36. (Address: Alfred J. Plechner, DVM, California Animal Hospital, 1736 S. Sepulveda, West Wood, CA 90025/(310)473-0969, U.S.A.)

Psyllium Side Effects

Psyllium is a hydrophilic muciloid which comes from the seeds or husk of the plant *Plantago Ovato,* also known as ispaghul. Adverse side effects to psyllium are generally uncommon. There are known IgE mediated hypersensitivity reactions to susceptible people. Symptoms can include rhinoconjunctivitis, skin reactions, asthma, gastrointestinal symptoms and anaphylaxis. When psyllium is mixed into powdered form, it can be inhaled and cause sensitization. Pharmaceutical workers handling psyllium are at particular risk and in 1 study 42% were found to have allergic symptoms. Oral ingested pysllium seems less likely to induce sensitization. To reduce the risk of psyllium allergy in health care workers the authors recommend: 1. That these health care workers who are suspected of allergies to psyllium be identified. 2. Workers with known allergies to psyllium should avoid mixing and dispensing the products, especially the dry material. 3. Measures to reduce psyllium dust particles including face masks and fumed hoods. 4. The use of granulated as opposed to finely powdered formulations may make it less likely to induce sensitization. **15016**

"Adverse Effects of Psyllium", Gillespie, Brian F., M.D. and Rathbun, Flora, MSc, M.D., Canadian Medical Association Journal, January 1, 1991;16-17. (Address: Brian F. Gillespie, M.D./Flora J. Rathbun, MSc, M.D., Drug Evaluation Division, Bureau of Nonprescription Drugs, Department of National Health and Welfare, Ottawa, Ontario, Canada)

AIDS

Sports Risk

Even though there have been cases, such as an English soccer player becoming HIV positive after colliding with a Dutch player, experts suggest that athletes with AIDS are at a very low risk for spreading the disease. No traces of the AIDS virus have been collected in sweat from 50 AIDS individuals during workouts. The AIDS virus has never been found in sweat or saliva. One expert states that "wrestlers, boxers, hockey players and all other athletes, while playing are not at high risk" despite hard contact and some bleeding. This was the opinion of Dr. Leonard Calabrese, D.O., a rheumatologist at the Cleveland Clinic. **14930**

"No Sports AIDS Risk", Medical Tribune, January 16, 1992;33(1):1. (Address: Medical Tribune, 257 Park Avenue South, New York, NY 10010, U.S.A.)

ALZHEIMER'S DISEASE

Mercury

This letter to the editor reports that certain complex forms of mercury may be involved in the etiology of Alzheimer's disease. In recent work by Wenstrup, et al (Brain Research 1990;53:125-131), the brains of 10 autopsied Alzheimer's disease patients between 59 and 93 years of age and 12 controls between 59 and 83 years of age were evaluated for 13 trace elements. Most remarkable were increases in the mercury/selenium and mercury/zinc ratios in Alzheimer's disease microsomal subfractions; increases in mercury/selenium ratio in Alzheimer's disease nuclear fractions; and increased zinc/selenium ratio in Alzheimer's disease mitochondrial fractions. There were elevations of mercury in Alzheimer's disease bulk brain samples, particularly in the cerebral cortex as well as the nucleus basalis of Meynert compared to controls. It is noted that the nucleus basilis of Meynert is the major cholinergic projection to the cerebral cortex. This anatomical site is severely degenerated in Alzheimer's disease patients. The author notes that the imbalances between mercury and selenium, and mercury and zinc, are of importance since selenium and zinc are protective against mercury toxicity. Tubulin, which can be inhibited by mercury, was found to be low in the areas of Alzheimer's disease brains with the highest accumulation of mercury. The researchers Duhr, et al,(FASEB, April 1991, Abstract 493) states that "These results suggest that certain complexes of mercury or HG^{2+} must be considered as a potential source for the etiology of Alzheimer's disease". The author concludes by stating that the release of mercury from dental amalgams is a major contributor to human exposure from inorganic mercury. There have been studies which show a correlation between inorganic mercury in the brain and the number and surfaces of amalgam fillings. Mercury from dental amalgams is rapidly and directly passed into body tissues and it accumulates with time. The author concludes that no amount of exposure to mercury vapor is safe, and suggests that dental amalgams are possibly a primary source of mercury which may affect Alzheimer's disease. **14937**

"Mercury as a Potential Source for The Etiology of Alzheimer's Disease", Bjorklund, G., Trace Elements in Medicine, 1991;8(4):208. (Address: G. Bjorklund, Toften, 24 N-8610 Grubbei, Norway)

ANIMAL RESEARCH

Automobile Testing

Since 1981 over 20,000 animals have been killed in crash-related experiments by General Motors. These studies have been done on dogs, cats, ferrets, pigs, mice, rats, guinea pigs, rabbits, hamsters, crayfish and frogs at the General Motors Research Laboratories in Warren, Michigan, Brigham Young and Wayne State Universities. It is noted that Ford, Chrysler, Volvo, Mercedes-Benz, SAAB, Nissan, Volkswagen, Toyota, Mazda, Hyundai and Subaru all avoid these animal experiments. Both Volvo and Mazda officials have been quoted that they use human dummies and do not use animals in their testing. Neurologist Carol Van Petten, M.D., states, "There are simply no new findings here either at the physiologic, macroscopic, or microscopic level. General Motors is not a biomedical research institution. Might not GM better direct its attention toward more socially productive approaches to the problem of head trauma such as funding efforts directed at prevention of the accidents that cause it." One reported use of animals was in a pig trauma model. Twenty-nine anesthetized pigs were hung horizontally in a sling and hit in the chest by a pneumatically driven piston and shaft assembly. Usually the pigs died immediately from heart tamponade and hemothoraces. Those pigs that did not die immediately were kept anesthetized for up to 1 hour and then killed by electrocution or overdose of halothane. Other examples are given. **14975**

"Twenty Thousand Animals Killed in Tests by General Motors", Thacher, Wendy, DVM, Physician Committee For Responsible Medicine Update, Winter, 1992;4-5,13. (Address: Physicians Committee For Responsible Medicine, P.O. Box 6322, Washington, D.C. 20015, U.S.A.)

ARRHYTHMIAS

Sunflower Seed Oil

In a rat model myocardial ischemia followed by reperfusion was induced. Feeding the animals a linoleic rich diet by supplying 12% sunflower seed oil in rat food pellet for 4 weeks decreased the incidence of reperfusion-induced ventricular fibrillation, both after 6 minutes and 12 minutes of