

The Effects of Dental Toxins

The Effects of Synergistic Toxicities and Genetic Susceptibilities on the Toxic Effects of Mercury Compounds and Toxins Produced by Oral Anaerobic Bacteria: The Relationship to Alzheimer's Disease, Autism and Related Disorders

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Data now exists that strongly indicates that there is a genetic subset of the human population that is unable to effectively excrete mercury from low level exposures. This leads to a retention toxicity in this subset at levels of exposure that is easily excreted by the bulk of the healthy population. Alzheimer's Disease (AD) patients and autistic children seem to fit into this subset. With AD subjects, mercury and oral toxins that react with sulfhydryl sensitive enzymes seem to mimic the biochemical abnormalities found in the AD brain. The observed low levels of mercury in the blood, urine and hair of autistics, when compared to the higher levels retained in their other body tissues, indicates that retention toxicity occurs. This susceptible subset of the population, due to the low frequency, is very likely to be overlooked or not be apparent in most epidemiological studies that consider general populations.

The retention and toxicity of mercury and mercury compounds is known to be enhanced by the presence of other synergistic factors that may or may not have toxicity by themselves. Such non-toxic compounds include antibiotics, a milk diet and male hormone. Toxic compounds such as other heavy metals (lead, aluminum) are well known to dramatically increase the toxicity of low levels of mercury exposures. Therefore, unless a total knowledge of the exposure to synergistic toxins are known it is impossible to define a safe level of mercury exposure for humans in the environment. In addition, the reaction of mercury from dental amalgams with methyl mercaptan ($\text{CH}_3\text{-SH}$) produces exceptionally toxic organic-thiol mercury compounds ($\text{CH}_3\text{-S-Hg}^+$ and $\text{CH}_3\text{-S-Hg-S-Hg}$) that are lipid soluble and dramatically cytotoxic. Other very toxic thiol containing compounds produced by oral anaerobic bacteria imbedded in teeth with root canals will be presented and their toxicity to enzymes demonstrated.

Further, an evaluation of the relative toxic effects of mercury and thimerosal (vaccines) show that the younger the infant the more toxic and lethal the effects can be. An evaluation of the considerations of synergistic toxicities, genetic susceptibility and infant age will be presented along with the basic biochemical and cellular level research that strongly supports the thimerosal causation of autism hypothesis. The biochemical processes that are adversely affected in autistic children are also those proven to be exceptionally sensitive to thiol reactive agents such as the ethylmercury produced by thimerosal and thiol reactive toxins produced by oral infections. Both of these categories of compounds react specifically with enzymes known to have reactive cysteines in their basic structures. The major protection against these toxins is the natural antioxidant called glutathione. Oxidative stress (low glutathione levels) is a correlating symptom of both AD and autism, indicating that the exposure to mercury compounds and oral toxins consume glutathione making the body more susceptible to other infections and toxicities.