Interview with Dr. Boyd E. Haley: Biomarkers supporting mercury toxicity as the major exacerbator of neurological illness, recent evidence via the urinary porphyrin tests

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Abstract

In the recent past, several biological finds have supported the hypothesis that early exposure of infants to Thimerosal was the major exacerbation factor in the increase in autism-related disorders since the advent of the mandated vaccine program. These initially included the observations of a genetic susceptibility impairing the excretion of mercury and the increased retention of mercury by autistic children. This was followed by data indicating that autistics have low levels of the natural compound glutathione that is necessary for the bilary excretion of mercury, possibly explaining the genetic susceptibility. Other observations clearly point out that various biochemical processes are inhibited at exceptionally low nanomolar levels of Thimerosal, including the killing of neurons in culture, the inhibition of the enzyme that makes methyl-B_{12}, the inhibition of phagocytosis (the first step in the innate and acquired immune system), the inhibition of nerve growth factor function at levels not cytotoxic, and the negative effect on brain dendritic cells. It is also now quite clear from primate studies that Thimerosal, or more correctly, the ethylmercury from Thimerosal delivers mercury to the brain, and causes brain inorganic mercury levels higher than equal levels of methylmercury.

Most recently, one study showed that 53% of autistic children had aberrant prophyrin profiles similar to mercury toxic individuals. Treatment of these children with a mercury chelator brought these porphyrins back towards normal levels indicating mercury toxicity was the cause, not genetic impairment. Porphyrin profiles are one of the most sensitive methods of measuring toxic mercury exposures. Recently, in a major advance it was shown that about 15% of individuals in one population displayed a marked sensitivity to mercury exposure in their porphyrin physiology, again supporting the concept of a genetically susceptible population that is more sensitive to mercury than the general population.

This observation on porphyrin aberrancies brings into consideration other possible effects of mercury toxicity that are secondary to porphyrin depletion. Porphyrins are the precursors to heme synthesis. Heme is the oxygen binding prosthetic group in hemoglobin and depletion of heme would affect oxygen delivery to the mitochondria and decrease energy production. Also, heme is a component of the electron transport system of mitochondria and a prosthetic group in the P450 enzymes which are fundamental in the detox of the body from many organic toxicants including pesticides and PCBs. Just recently, a report was released implying that lack of heme was the major reason why ß-amyloid plaques build up in the brains of Alzheimer's diseased subjects. It seems that heme attaches to ß-amyloid helping it remain soluble and excretable. Without adequate heme one of the major pathological diagnostic hallmarks of Alzheimer's disease appears. It is well known that mercury rapidly disrupts the normal polymerization of tubulin into microtubulin in brain tissue and aberrant tubulin polymerization is a consistent factor observed in Alzheimer's diseased brain. Therefore, it is the multiple inhibitions of mercury that can cause various neurological and systemic problems and many of these are secondary to the primary site of mercury binding.

Keywords: mercury toxicity, porphyrin, heme, tubulin, autism