The relationship of the toxic effects of mercury to exacerbation of the medical condition classified as Alzheimer's disease

Boyd E. Haley, PhD
Professor, Department of Chemistry
University of Kentucky
Lexington, KY 40506-0055
Email: behaley@uky.edu

Abstract

Mercury(II) or Hg^{2+}, is neurotoxic. When exposed to normal brain tissue homogenates, neurons in culture, Hg^{2+} (mercury(II) or mercuric mercury) is capable of causing many of the same biochemical aberrancies found in Alzheimer's diseased (AD) brain. Also, rats exposed to mercury(0), metallic mercury, vapor show some of these same abnormalities in their brain tissue. Specifically, the rapid inactivation of the brain thiol-sensitive enzymes (tubulin, creatine kinase and glutamine synthetase) occurs after: (a) the addition of low micromolar levels of Hg^{2+}, (b) exposure to mercury vapor (Hg^0) or (c) the addition of Thimerosal (ethylmercurythiosalicylate sodium salt). Moreover, these same enzymes are significantly inhibited in the AD brain. Further, exposure of neurons in culture to nanomolar levels of Hg^{2+} has been shown to produce three of the widely accepted pathological diagnostic hallmarks of AD. These AD hallmarks are elevated amyloid protein, hyper-phosphorylation of Tau, and formation of neurofibillary tangles (NFTs).

This paper proposes the hypothesis that elemental mercury, organic mercury compounds, and other blood-brain permeable toxicants, which have enhanced specificity for thiol-sensitive enzymes, are the etiological source of AD. Included in this category are other heavy metals such as lead and cadmium that act synergistically to enhance, by many-fold, the toxicity of metallic mercury and organic-mercury(II) compounds, like Thimerosal. This hypothesis also explains the genetic susceptibility to AD that is expressed through the APO-E geneotype. Specifically, a reduction of APO-E gene type that contains two cysteines decreases the one of the innate detoxification capabilities of APO-E, the removal of mercury and other thiol-reactive toxicants from the central nervous system. This increases brain exposure to thiol-reactive toxicants and elevates the risk of AD. Also, the increased exposure to mercury through breathing the mercury vapor emanating from mercury amalgam dental fillings can have a deleterious effect on olfactory capability. This effect may explain the high correlation between the loss of sense of smell and the subsequent development of AD.

Keywords: Alzheimer's Disease (AD), mercury, mercury toxicity, neurotoxicants