Alkyl Mercury-Induced Toxicity: Multiple Mechanisms of Action

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Abstract There are a number of mechanisms by which alkylmercury compounds cause toxic action in the body. Collectively, published studies reveal that there are some similarities between the mechanisms of the toxic action of the mono-alkyl mercury compounds methylmercury (MeHg) and ethylmercury (EtHg). This paper represents a summary of some of the studies regarding these mechanisms of action in order to facilitate the understanding of the many varied effects of alkylmercurials in the human body. The similarities in mechanisms of toxicity for MeHg and EtHg are presented and compared. The difference in manifested toxicity of MeHg and EtHg are likely the result of the differences in exposure, metabolism, and elimination from the body, rather than differences in mechanisms of action between the two.

Keywords Arachidonic acid • Calcium homeostasis • Cell cycle/division • Ethylmercury • Glial cells • Glutamate • Glutamine • Glutathione (GSH) • Leukotriene synthesis mechanism of toxicity • Membrane permeability/integrity • Methylmercury • Mitochondria • Neurotransmitter release nitric oxide • Oxidative stress • Reactive oxygen species • Receptor binding • ROS • Thimerosal

1 Preface

The alkyl mercury compounds methylmercury (MeHg) and ethylmercury (EtHg) have been shown to be toxic to humans and non-human animals as well (Driscoll et al. 2013). Both of those compounds have similar chemical properties, and both have been shown to disrupt the normal function of the CNS in a variety of animal species.

In mass poisonings from the consumption of MeHg-contaminated seafood in Japan (Kutsuna 1968) and MeHg-treated grain in Iraq (Bakir et al. 1973), frank developmental/birth effects and/or severe brain damage were observed in the children of some mothers who consumed large quantities of mercury-contaminated fish, bread, or grain products during pregnancy. In addition, a variety of neurologic

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