Neurodevelopment of Amazonian children exposed to ethylmercury (from Thimerosal in vaccines) and methylmercury (from fish)

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1. Introduction

During developmental periods extending from prenatal stages, the brain is more vulnerable to the adverse effects of toxic insults than at more mature stages; however, experience-guided development drives neuro-cognition achievements. Early brain susceptibility to mercury toxicity (Clarkson et al., 2003) and adverse neurological outcomes have been reported in animal experiments (in vivo and in vitro) as well as in human epidemiological studies (Dórea, 2013; Grandjean et al., 2014). In fish-eating populations of the Amazon, during prenatal and postnatal life, not only methylmercury (MeHg) but also ethylmercury (EtHg) in Thimerosal-containing vaccines (TCVs) during infant’s neurodevelopment. We studied children (n=1139) from the Western Amazon based on combined (low, intermediate, and high) exposure to chronic MeHg from fish consumption and acute TCV- EtHg. Neurodevelopment outcomes were age of walking and age of talking, and the Bayley Scale of Infant Development (BSID). The Mental Developmental Index (MDI) and Psychomotor Developmental Index (PDI) were measured at six and 24 months of age. Median hair-Hg (HHg) at birth was 6.4 μg g⁻¹ in mothers, and 1.94 μg g⁻¹ in newborns; total (pregnancy and infancy) EtHg exposure ranged from 0 to 187.5 μg. The combined (MeHg + EtHg) exposure showed significant differences for MDI but not for PDI; however, there was a significant decrease in both MDI and PDI scores at 24 months. The increase in BSID delays (scores < 80) between six and 24 months was not discernible with regards to EtHg or MeHg exposure. We found a statistically significant increase in neurodevelopmental (BSID) delays related to the combined exposure to Hg (MeHg > EtHg). Neurodevelopment delays due to low-doses of organic mercury (albeit undiscernible) are not predictable but can be avoided by choosing low-Hg fish and providing Thimerosal-free vaccines.

Many studies have addressed co-occurring methylmercury (MeHg) from maternal origin and ethylmercury (EtHg) from Thimerosal-containing vaccines (TCVs) during infant’s neurodevelopment. We studied children (n=1139) from the Western Amazon based on combined (low, intermediate, and high) exposure to chronic MeHg from fish consumption and acute TCV- EtHg. Neurodevelopment outcomes were age of walking and age of talking, and the Bayley Scale of Infant Development (BSID). The Mental Developmental Index (MDI) and Psychomotor Developmental Index (PDI) were measured at six and 24 months of age. Median hair-Hg (HHg) at birth was 6.4 μg g⁻¹ in mothers, and 1.94 μg g⁻¹ in newborns; total (pregnancy and infancy) EtHg exposure ranged from 0 to 187.5 μg. The combined (MeHg + EtHg) exposure showed significant differences for MDI but not for PDI; however, there was a significant decrease in both MDI and PDI scores at 24 months. The increase in BSID delays (scores < 80) between six and 24 months was not discernible with regards to EtHg or MeHg exposure. We found a statistically significant increase in neurodevelopmental (BSID) delays related to the combined exposure to Hg (MeHg > EtHg). Neurodevelopment delays due to low-doses of organic mercury (albeit undiscernible) are not predictable but can be avoided by choosing low-Hg fish and providing Thimerosal-free vaccines.

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