

Principal component analysis and discrimination of variables associated with pre- and post-natal exposure to mercury

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Abstract

The variance of variables associated with neurodevelopment at 180 days, pre-natal variables (Hg in placenta, blood and hair) and post-natal Hg exposure (including Thimerosal-containing vaccines, TCV) were examined in 82 exclusively breastfed infants using principal component analysis (PCA). This multivariate method was applied to identify hierarchy and sets of interrelated variables. The PCA yielded a two-factor solution, explaining 92% of variance and summarizing most of the relevant information in the dataset matrix: the first component represented birth weight and vaccine (first doses of Hepatitis B and DTP) variability and explained 57% of variance; the second component represented a gradient of neurodevelopment (Gesell scores) and explained 35% of variance. The third component explained only 3% of the remaining 8% variance. Beside CNS priming by breastfeeding, infant development (birth weight) and time of immunization with TCV should be considered in epidemiological studies. PCA can classify sets of variables related to vaccination and neuromotor development schedules, clearly discriminating between earlier and later TCV exposures of exclusively breastfed infants. In conclusion, the incommensurable concept of the chance of toxic risk caused by TCV-EtHg exposure against the proven benefit of immunization is in no way disputed here. However, infant neurodevelopmental (ND) disorders linked to Thimerosal-Hg stands in need of proof, but PCA points to the possibility of identifying exposure risk variables associated with ND schedules.

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Introduction

Mercury's most widely recognized effects are neurological; the developing central nervous system (CNS) of fetus, infants and young children are vulnerable to these

effects. Fetal exposure to methylmercury (MeHg, from fish consumption) is thought to lower intelligence and alter behavior. The Harvard Center for Risk Analysis panel quantified the impact of chronic MeHg exposure on cognitive development (Cohen et al., 2005) and concluded that pre-natal MeHg exposure sufficient to increase maternal hair-Hg by 1 µg/g at parturition decreases intelligence quotient by 0.7 points (Cohen et al., 2005). However, neurological effects of post-natal

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