Objective

Mercury (Hg) is recognised as neurotoxin; nevertheless, the effect of prenatal mercury exposure on child behavior in fish eating population is still controversial. The benefit of nutrient element of fish may insufficient to explain it. Apolipoprotein (APOE) is a major protein transporter in brain, epsilon 4 (e4) allele is recognised with poorer neural repair function. We hypothesize that the APOE may modify the effect of prenatal mercury exposure on child behaviour.

Methods

The present study is a prospective cohort study. There were 166 subjects recruited at delivery and followed up at age of two years. The level of prenatal mercury exposure is determined in cord blood and the genotype of APOE is analysis by the methods Restriction Fragment Length Polymorphism Analysis of PCR-Amplified Fragments (PCR-RFLP). The Child Behavior Checklists version 1.5/5, a parent rating scale, is used to determine the child’s behaviour.

Results

The adverse effect is found in e4 carriers whose cord blood Hg level is greater than 12 μg/L. After controlling for the potential confounding factors, the total scale of internalising behavior (β = 8.4) and all symptoms of internalising problems is found statistically significant higher in this group. The symptoms and beta coefficients are emotional problem (β = 2.6), anxiety/depression (β = 2.4), somatic complaints (β = 1.68) and withdrawn (β = 1.7). In addition to internalising behaviour, the item of other problem (β = 6.7) from externalising behaviour and the total scale of CBCL (β = 20.7) are also found statistically significant higher in the group that e4 carriers with greater cord blood Hg.

Conclusion

APOE gene modifies the effect of prenatal mercury exposure on neurobehavior. The different frequency of gene susceptible across populations may be a reason of the controversial finding in previous study. The impact of genetic susceptibility should be considered in future study.
227 Cord Blood Mercury, APOE and Child's Behaviour

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*Occup Environ Med* 2013 70: A77
doi: 10.1136/oemed-2013-101717.227

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