Objectives Lead is one of the oldest known toxic metals. For decades, its effects on child development has been remained a topic of concern with an increased interest in ‘what prenatal blood lead levels should be considered toxic’. Many recent studies have shown the impacts of increased lead on different aspects of infants’ development at ‘acceptable’ levels (≤100 μg/L).

Methods To investigate the effects of prenatal lead exposure on children mental development, we conducted a longitudinal study. Pregnant women (n = 364) who referred to hospitals for prenatal care at the first trimester of pregnancy were asked to participate in the survey. Maternal whole blood (MWB) samples, one for each pregnancy trimesters (3 times), and the umbilical cord blood samples, at the time of delivery, were collected and subjected to ICP-MS analysis for measurement of lead concentrations. We invited the mothers and their children to the research hospitals when the children were between 20 and 36 months of age and assessed mental development using Early Child Development Inventory (ECDI). The inventory included 60 items, which cover seven different development areas.

Results MWB lead followed a U-shaped pattern over the course of pregnancy with lowest level during the second trimester. The ECDI score was inversely related to the first trimester blood lead concentrations (r = -0.15, p < 0.05). The logistic regression analysis demonstrated significant relationships between increasing the first trimester lead concentrations (log), with low score of ECDI, adjusting for multiple covariates (Unit risk: 5.7, 95% CI: 1.1-30.7, p < 0.001).

Conclusions Increased prenatal lead concentrations, even at “acceptable” level, adversely affects ECDI scores. Therefore, a reappraisal of lead exposure standards for female workers is a critical public health concern.

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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 behaviour (b = 8.4) and all symptoms of internalising problems is found statistically significant higher in this group. The symptoms and beta coefficients are emotional problem (b = 2.6), anxiety/depression (b = 2.4), somatic complaints (b = 1.68) and withdrawal (b = 1.7). In additional to internalising behaviour, the item of other problem (b = 6.7) from externalising behaviour and the total scale of CBCL (b = 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.