Published in final edited form as:

Int J Hyg Environ Health. 2008 July ; 211(3-4): 345–351. doi:10.1016/j.ijheh.2007.07.023.

Prenatal low-level lead exposure and developmental delay of infants at age 6 months (Krakow inner city study)

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Abstract

The purpose of the study was to assess the neurocognitive status of 6-month-old infants whose mothers were exposed to low but varying amounts of lead during pregnancy. Lead levels in the cord blood were used to assess environmental exposure and the Fagan Test of Infant Intelligence (FTII) assessed visual recognition memory (VRM). The cohort consisted of 452 infants of mothers who gave birth to babies at 33–42 weeks of gestation between January 2001 and March 2003. The overall mean lead level in the cord blood was 1.42 µg/dl (95% CI: 1.35–1.48). We found that VRM scores in 6 month olds were inversely related to lead cord blood levels (Spearman correlation coefficient -0.16 , $p = 0.007$). The infants scored lower by 1.5 points with an increase by one unit (1 µg/dl) of lead concentration in cord blood. In the lower exposed infants (≤ 1.67 µg/dl) the mean Fagan score was 61.0 (95% CI: 60.3–61.7) and that in the higher exposed group (> 1.67 µg/dl) was 58.4 (95% CI: 57.3–59.7). The difference of 2.5 points was significant at the $p = 0.0005$ level. The estimated risk of scoring the high-risk group of developmental delay (FTII classification 3) due to higher lead blood levels was two-fold greater (OR = 2.33, 95% CI: 1.32–4.11) than for lower lead blood levels after adjusting for potential confounders (gestational age, gender of the child and maternal education). As the risk of the deficit in VRM score (Fagan group 3) in exposed infants attributable to Pb prenatal exposure was about 50%, a large portion of cases with developmental delay could be prevented by reducing maternal blood lead level below 1.67 µg/dl. Although the negative predictive value of the chosen screening criterion (above 1.67 µg/dl) was relatively high (89%) its positive predictive value was too low (22%), so that the screening program based on the chosen cord blood lead criterion was recommended.

Keywords

Prenatal lead exposure; Biological markers; Infant visual recognition memory; Neurocognitive development

Introduction

Both endogenous and exogenous factors and their interaction regulate human child development. Among the exogenous factors affecting early neurobehavioral child development is the in utero exposure to toxic metals such as lead during the early stages of brain development. Because the fetal and infant brains are in a state of rapid growth, impairment of later cognitive function may result from relatively minor environmental toxic exposure. Humans begin to accumulate lead in their bodies already during prenatal development. Since the placenta is no effective biological barrier, pregnant women represent the group at increased risk because of maternal exposure of the fetus to lead (Dietrich et al., 1986, 1987; Fahim et al., 1976). Previous studies indicate that prenatal exposure to even low-level environmental contaminants to lead can have demonstrable effects on later cognitive and behavioral development (Bellinger et al., 1987; Emory et al., 1999, 2003; Needleman and Gatsonis, 1990; Pocock et al., 1994; Wigg et al., 1988). Current research was not able to determine accurately a threshold for many of lead health's effects and it was not possible to find a concentration of lead blood level below which no effect occurs. There is an opinion that the standard for interventions for kids or pregnant women should be within the range of 5–10 µg/dl of lead in the blood, but some scientists stress that to better define the risk more studies on the effect of the lead blood in the range of 1–10 µg/dl are needed and application of better tools to measure the smaller health effects are looked for.

The toxicity of lead may largely be explained by its interference with different enzyme systems: lead inactivates these enzymes by binding to SH-groups of its proteins or by displacing other essential metal ions. For this reason many organs or organ systems are potential targets for lead, and a wide range of biological effects of lead have been documented. These include effects on the hem biosynthesis, the nervous system, the kidneys and reproduction as well as on cardiovascular, hepatic, endocrinal and gastrointestinal functions. In conditions of low-level and long-term lead exposure, such as those found in the general population, the most critical effects are exerted on hem biosynthesis and the nervous system (Cookman et al., 1987; Freedman et al., 1990; Goyer, 1993; Juberg et al., 1997; Press, 1977). The cord blood concentration of lead represents the accumulated dose over the pregnancy period and to capture the early cognitive outcomes of lead exposure in the womb, we have chosen the Fagan test for measuring the intelligence of infants, which is a well recognized test of infant intelligence and has been found to be a predictor of later cognitive outcome (Fagan, 1992; Fagan et al., 1986; Fagan and Detterman, 1992).

Our primary purpose of the study was to establish the possible association between low-level prenatal exposure to lead and visual recognition memory (VRM) score in infants. Secondary purpose of the study was to define the impact of the low-level lead prenatal exposure on the neurocognitive status of infants in the population sample and establish eventually a screening criterion for early detection of the developmental delay based on the lead cord blood level.

Material and methods

The cohort consisted of 452 infants who were born at 33–42 weeks of gestation between January 2001 and March 2003 to mothers participating in an ongoing prospective cohort study. The design of this cohort prospective study and the selection of the population have been described previously (Jedrychowski et al., 2003). Women attending ambulatory prenatal clinics in the first and second trimesters of pregnancy were eligible for the study. The enrollment included only non-smoking women with singleton pregnancies between the ages of 18 and 35 years, and who were free from chronic diseases such as diabetes and hypertension. Upon enrollment, a detailed questionnaire was administered to each subject to

elicit information on demographic data, date of the last menstrual period (LMP), medical and reproductive history, occupational exposures, alcohol consumption, and environmental tobacco smoke (ETS).

Blood sample collection and analysis

A cord blood sample (30–35 ml) was drawn into a vacutainer tube that had been treated with ethylene diamine tetra-acetate (EDTA). The tubes were inverted several times to mix the EDTA and the blood to prevent coagulation. Within 8 h of blood collection, the blood samples were transported to the clinical biochemistry laboratory at the University Hospital in Krakow for processing and storage. Packed red blood cells and plasma samples were separated and stored in liquid nitrogen in the laboratory prior to shipment to Columbia University. From Columbia University, portions of samples were then sent to the Centers for Disease Control and Prevention (CDC) for chemical analysis. Blood samples for lead analysis were refrigerated without any processing. Whole blood lead concentrations were determined using inductively coupled plasma mass spectrometry CLIA'88 method "Blood lead cadmium mercury ICPMS_ITB001A". This multi-element analytical technique is based on quadrupole ICP-MS technology (CDC, 2003).

Cognitive developmental tests

None of the children manifested obvious neurological abnormalities at the 6-month developmental check-up. The Fagan Test of Infant Intelligence (FTII) was assessed in 6-month-old infants (mean = 26.7 weeks, SD = 1.6) at the Chair of Epidemiology and Preventive Medicine by trained examiners, who were unaware of the child's exposure. The FTII measures VRM by calculating a novelty preference score based on comparison of 10 sets of photographs of faces. The infant's distribution of visual attention to new and old pictures reflects the operation of memory and abstraction processes. On most problems, infants are presented with one picture for study, and then on test, the familiar one is paired with a novel one. Infants typically devote a greater percentage of total fixations to the novel target. Preference for the novel picture shows that the two pictures are discriminable, and indicates that the infant recognizes one picture as familiar. The final test value is called the "novelty score" which is the length of fixation time during the test phase devoted to the novel picture divided by the total fixation time to both targets, multiplied by 100. According to the FTII manual, scaled novelty scores fall into three categories: Group 1-low risk, greater than 54.6, Group 2-suspected risk, greater than 52.2 and less than 54.4; and Group 3-high risk, less than 52.2.

Statistical methods

Linear regression analysis and Spearman rank correlation were used in the analysis of the association between Fagan ranked scores and lead levels in cord blood. Chi-square statistics and analysis of variance tested differences in characteristics between subgroups. The effect of lead exposure on novelty scores was assessed by multiple logistic regression models and adjusted for potential confounders (maternal education, parity, gender of child). In logistic regression analysis, exposure to lead was entered into models both continuously and as a dichotomized variable divided into two levels based on 75th percentile value of the lead distribution in the total sample. In addition, the ROC curve analysis was performed to establish a screening criterion of the lead in cord blood that could be valid for early detection programs of the developmental delay. Subsequently, the risk of low VRM score (FTII group 3) attributable to prenatal lead exposure was calculated. It estimates the proportion of impairment in exposed individuals due to the exposure in question. Statistical analyses were performed with BMDP software for Windows.

Results

Table 1 presents the characteristics of the infants under study dichotomized by its median concentration. The maternal education and the VRM scores were significantly lower in newborns from the higher exposed group. The overall mean lead level in cord blood was 1.42 µg/dl (95% CI: 1.35–1.48). In total, 25% of newborns had lead cord concentrations above 1.67 µg/dl (95% CI: 1.60–1.80) and only 2.5% of them showed levels above 3 µg/dl (Fig. 1).

Fagan VRM scores ranged from 36.6 to 81.1 and were normally distributed with a mean of 60.3 (SD 6.8). There were 363 (80.3%) infants in the low risk group, 26 (5.8%) in the suspected risk group and 63 (13.9%) in the high-risk group. We found that VRM scores in 6 month olds were inversely related to lead cord blood levels (Spearman correlation coefficient -0.16 , $p = 0.007$). The mean lead cord blood level in the low risk group was 1.38 µg/dl (95% CI: 1.31–1.45) in the suspected risk group 1.46 (95% CI: 1.26–1.67) and in high risk it was 1.61 µg/dl (1.35–1.87). The infants scored lower by about 1.5 points with an increase by one unit (1 µg/dl) of lead concentration in cord blood (Fig. 2). While the regression of the VRM score on lead level was significant in the group with low or suspected risk, the relationship was not seen in the high-risk group (Fig. 3). In the lower exposed infants the mean Fagan score was 61.0 (95% CI: 60.3–61.7) and that in the higher exposed group was 58.4 (95% CI: 57.3–59.7). The difference of 2.5 points was significant at $p = 0.0005$.

The developmental delay (Fagan group 3) has been found in 21.6% of higher exposed infants and in 11.6% of lower exposed group (difference significant at $p = 0.008$). In order to estimate the risk of the developmental delay due to the prenatal lead exposure the multiple logistic regression analysis was performed for the high-risk category (FTII group 3) versus the rest (FTII groups 1 + 2) and OR estimates were adjusted for potential confounders (maternal education, parity, gender of child). The results of the latter analysis (Table 2) showed that the risk of the delayed neurocognitive function increased by about 50% with each unit (µg/dl) of lead blood concentration (OR = 1.47; 95% CI: 1.07–2.01). The similar statistical analysis carried out for the dichotomized lead blood level (dichotomized by 75th percentile) demonstrated that infants with a lead level above 1.67 µg/dl showed more than a twofold risk for developmental delay (OR = 2.33, 95% CI: 1.32–4.11) compared to those who had lead levels below 1.67 µg/dl (Table 3).

Finally we used the ROC curve analysis to establish a cut-off point for the lead cord blood level, which could eventually be useful in the screening programs for an early detection of delayed neurocognitive functions (Fig. 4). The analysis confirmed that the distribution of lead blood was significantly different in the infants classified as high-risk group (FTII 3 group), and the best screening criterion would be a lead concentration above 1.67 µg/dl. This cut-off point would have a sensitivity of 39.7% and specificity of 76.7% for the screening of early developmental delay. Since OR for the delayed function related to higher lead level was 2.3 we might assume that the estimated population attributable fraction of delayed neurocognitive function due to this criterion would amount to 21.3%, and that in the exposed 53.6%.

Discussion

It is noteworthy that the infants in our study were exposed to low prenatal lead exposure, which was within the range of 0.44–6.90 µg/dl and mean of 1.42 µg/dl (SD: 0.71). Among 75% of infants the lead level in the cord blood was ≤ 1.67 µg/dl. We found that the VRM score in 6 month olds, was inversely related to lead cord blood concentrations (Spearman

correlation coefficient -0.16 , $p = 0.007$) and the infants scored significantly lower by 1.5 points with an increase of lead concentration by one unit ($\mu\text{g/dl}$). Infants who had a lead cord blood level above $1.67 \mu\text{g/dl}$ fell two times more frequently within the high-risk category group (FTII group 3).

The data obtained from our study may suggest that a subtle neurotoxic impact of low-level prenatal lead exposure occurs in infants from the Krakow inner city area. Assuming the causal relationship between VRM score and lead exposure, we estimate that lowering the prenatal lead cord blood level below $1.67 \mu\text{g/dl}$ could result in a decrease of developmental delay in infants by about 20%. We tried to explore the results of our study to establish the screening criterion for the early detection of the developmental delay of infants at 6 months of age based on the lead cord blood level above $1.67 \mu\text{g/dl}$. Although, the negative predictive value of this criterion was relatively high (89%) its positive predictive value was too low (22%), so that a screening program based on the chosen lead cord blood criterion could have been recommended.

The question of low-level lead exposure has been studied widely over the past two decades, but up to now; there has been considerable uncertainty about the exact dose–response relationships and threshold values for low-level lead toxicity in humans. Different methodological approaches may contribute to the controversy, among which one has to mention selecting adequate marker of exposure, measuring health outcome with sensitive tools, considering adequate set of potentially important confounders/modifiers, and recruiting the unbiased and adequate sample size to detect the small effects.

Blood lead levels determined at the time of gestation are probably the most accurate marker of fetal exposure, since toxicokinetic studies in rodents and primates demonstrate that lead readily moves across the placenta. However, it is not known which blood lead measure is most appropriate – blood leads during a specified gestational period, peak blood lead, or integrated blood lead measured over gestation. In our study we used the cord blood concentration of lead, which represents the accumulated dose over the pregnancy period.

The most sensitive endpoints for low-level lead toxicity are not yet known. Some investigators have focused on motor development and some on visual/ motor performance based upon the effects of lead observed in occupational settings. In order to capture the early cognitive outcomes of lead exposure in the womb, we have chosen the Fagan test for measuring the intelligence of infants, which was designed to diagnose infants at high risk for later intellectual deficits (Fagan, 1992; Fagan et al., 1986; McCall and Carriger, 1993; Rose and Wallace, 1985).

Our study population may be not be representative for the general urban population of infants within the country as the enrollment covered only pregnant non-smoking women with singleton pregnancies between the ages of 18 and 35 years, and who were free from chronic diseases such as diabetes and hypertension. However, these inclusion criteria helped us to eliminate from the study those infants that were at a higher risk of neurocognitive disorders due to maternal chronic diseases or active smoking. The strength of the study results from the very high power of the study (0.99) to estimate precisely the prevalence of the developmental delay in the study sample. Moreover, the power of the study was also adequate (0.74) for detecting relatively small effects across the groups of infants with lower and higher lead exposure level. The strength of the study results also from the fact that in the analysis of neurodevelopmental outcomes, we have considered a set of important confounders potentially affecting child development such as maternal education, parity, and gender of child.

The results of the study are consistent with evidence of heightened susceptibility of the fetal nervous system to prenatal toxic exposure and that VRM scores are a sensitive end-point for very low level lead toxicity. Needleman and Gatsonis (1990) in a meta-analysis of 12 modern studies of childhood exposures to lead in relation to IQ where lead blood was measured had found a strong support for the hypothesis that lead impairs children's IQ. The negative partial Spearman correlation coefficient for lead ranged from -0.27 to -0.003 . However, the power of the studies to find an effect was limited, below 0.6 in 7 of 12 studies. Our results would also be consistent with the data reported by Bellinger et al. (1987) who analyzed longitudinally prenatal and postnatal lead exposure on early cognitive development of children (Bayley Scales of Mental development) examined semiannually over 2 years. The estimated difference between the adjusted performance at the prenatal low exposure (<3.0 $\mu\text{g/dl}$) and high exposure group (>10 $\mu\text{g/dl}$) was 4.8 points. Scores were not related to infants' postnatal blood lead levels.

Nevertheless, there are some studies that raise suspicion and reserve about the effect of low-level lead exposure on human fetus. In fact, in two independent studies on low level prenatal exposure, Ernhart et al. (1986) and Rothenberg et al. (1989) did not find evidence of behavioral effects. Despite these inconsistencies, which may to some extent be the results of different methodological approaches in measuring exposure and health end-points, various recruitment schemes or insufficient sample sizes, the more robust results obtained with older school-aged children (David et al., 1972, 1977; Dietrich et al., 1993; Gittelman and Eskenazi, 1983; Fergusson et al., 1993; Needleman et al., 1979; Ruff et al., 1996) render the necessity to continue research on the impact of low-level prenatal lead exposure and early deficit in neurocognitive development. Confirmation of our initial results in the course of ongoing follow-up of this cohort would strengthen the argument in the debate on lead toxicity eventually in early infancy.

Acknowledgments

This is part of an ongoing comparative longitudinal investigation on the health impact of prenatal exposure to outdoor/indoor air pollution in infants and children being conducted in New York City and Krakow. The study received funding from an RO1 Grant entitled, "Vulnerability of the Fetus/Infant to PAH, PM_{2.5} and ETS" (5 RO1 ES10165 NIEHS; 02/01/00–01/31/04) and from the NIEHS (RO1 ES010165-0451) and the Gladys T. and Roland Harriman Foundation. Principal investigator: Prof. FP Perera.

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Fig. 1.
Cumulative frequency distribution of the lead cord blood level in the Krakow study population.

Fig. 2.
Scatterplot of the Fagan score and the lead cord blood level in $\mu\text{g}/\text{dl}$.

Fig. 3.
Scatterplot of the Fagan score and lead cord blood level by the risk groups.

Fig. 4.
The ROC curve for the cord blood lead levels and the Fagan high-risk group.

Table 1

Characteristics of the material in the total study sample and in subgroups dichotomized by the median cord blood lead level

	Total n = 452	Pb (µg/dl) – cord blood		
		≤1.23 µg/dl n = 227	>1.23 µg/dl n = 225	p-value
Maternal age:				
Mean	27.57	27.53	27.60	
SD	3.482	3.345	3.621	0.8277
Maternal education (years):				
Mean	15.59	15.95	15.22	
SD	2.733	2.601	2.820	0.0044
Gestational age (weeks):				
Mean	39.42	39.34	39.50	
SD	1.344	1.387	1.296	0.1975
Fagan score:				
Mean	60.36	61.09	59.62	
SD	6.736	6.907	6.490	0.0199
Birth weight (g):				
Mean	3424.1	3420.0	3428.3	
SD	459.4	450.3	469.4	0.8494
Length at birth (cm):				
Mean	54.67	54.63	54.70	
SD	2.729	2.722	2.741	0.8052
Fagan score:				
Mean	60.36	61.09	59.62	
SD	6.736	6.907	6.490	0.0199
Age at Fagan testing (weeks):				
Mean	26.70	26.70	26.70	
SD	1.564	1.672	1.450	0.9904
Pb (µg/dl)—cord blood:				
Mean	1.415	0.957	1.877	
SD	0.708	0.177	0.743	0.0000

Table 2

The results of the multiple logistic regression of FTII (higher risk score) by the level of cord lead blood (continuous variable) and adjusted for potential confounders ($n = 452$)

	Coefficient	Standard error	OR	95% CI
Gender of child	−0.369	0.278	0.69	0.40–1.19
Maternal education	0.095	0.100	1.10	0.91–1.34
Parity	0.287	0.207	1.33	0.89–2.00
Cord lead level (in $\mu\text{g}/\text{dl}$)	0.383	0.161	1.47	1.07–2.01

Table 3

The results of the multiple logistic regression of FTII (higher risk score) by the level cord blood lead level (dichotomized by the screening criterion level 1.67 $\mu\text{g/dl}$) and adjusted for potential confounders ($n = 452$)

	Coefficient	Standard error	OR	95% CI
Gender of child	−0.398	0.279	0.67	0.39–1.16
Maternal education	0.105	0.101	1.11	0.91–1.35
Parity	0.296	0.208	1.34	0.89–2.00
Cord lead level	0.848	0.289	2.33	1.32–4.11