

# Assessment of Lead Exposure in Schoolchildren from Jakarta

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Children attending schools in urban areas with high traffic density are a high risk group for lead poisoning. We assessed the magnitude of lead exposure in schoolchildren from Jakarta by analyzing blood lead concentrations and biomarkers of heme biosynthesis. A total of 131 children from four public elementary schools in Jakarta (two in the southern district and two in the central district) were enrolled in the study. To evaluate lead pollution in each area, soil samples and tap water were collected. The mean blood lead concentration was higher in the central district than in the southern district ( $8.3 \pm 2.8$  vs.  $6.9 \pm 3.5$   $\mu\text{g}/100$  ml;  $p < 0.05$ ); 26.7% of the children had lead levels greater than 10  $\mu\text{g}/100$  ml. In 24% of the children, zinc protoporphyrin concentrations were over 70  $\mu\text{mol}/\text{mol}$  hemoglobin; in 17% of the samples, hemoglobin was less than 11 g/100 ml. All other values were within the physiological range. Blood lead concentration and hematological biomarkers were not correlated. Analyses of tap water revealed lead values under 0.01 mg/l; lead contamination of soil ranged from 77 to 223 ppm. Our data indicate that Indonesian children living in urban areas are at increased risk for blood lead levels above the actual acceptable limit. Activities to reduce pollution (e.g., reduction of lead in gasoline) and continuous monitoring of lead exposure are strongly recommended. *Key words:* air pollution, blood levels, hematological biomarkers, Indonesia, lead exposure, schoolchildren, traffic density. *Environ Health Perspect* 106:499–501 (1998). [Online 13 July 1998] <http://ehpnet1.niehs.nih.gov/docs/1998/106p499-501heinze/abstract.html>

Several circumstances contribute to the high risk for lead poisoning in children. Children are often in contact with polluted dust, soil, or dirt; approximately 50% of the lead ingested comes from dirt and other lead-containing particles (1). During the period of rapid growth, enhanced absorption rates and an accelerated lead turnover (bone resorption/mobilization) resulted in higher blood lead levels than observed in adults living in the same area (2).

Even at low blood lead concentrations (10–25  $\mu\text{g}/100$  ml), adverse effects of lead on the neuropsychological development of children have been observed (3–6). At blood lead levels over 80  $\mu\text{g}/100$  ml, children generally show symptoms of an acute clinical lead intoxication with irreversible encephalopathy. Beside these neurotoxic effects, chronic lead exposure causes hematological dysfunctions (1,7,8). The reduction in the life span of erythrocytes observed at blood lead levels above 40  $\mu\text{g}/100$  ml is due to a direct toxic effect on the cell membrane and to a diminished hemoglobin (Hb) synthesis.

Most of the lead found in our environment derives from human activities. High lead concentrations in air and soil are measured in urban areas with high traffic density, especially when leaded gasoline is used. Consequently, lead pollution and poisoning are severe and acute problems in the rapidly developing cities in the Third World (9). In Jakarta, home of about 10 million people, the number of cars increases by about 10%

per year. Although the maximum lead concentration in gasoline is limited to 0.4 g/l, the traffic-derived lead pollution in Jakarta is estimated at 2 tons/day (10).

The aim of this study was to assess the magnitude of lead exposure in schoolchildren from Jakarta by analyzing blood lead concentrations and biomarkers of heme biosynthesis.

## Subjects and Methods

The study was carried out during the rainy season in January 1996. Based on recent measurements of lead concentrations in the surrounding atmosphere by Indonesian authorities, we defined two districts with different contamination: Pasar Minggu in the south and Menteng in central Jakarta. We selected two public elementary schools at random from each district. All schools were located less than 1 km from a main road.

In each school, all children of the specific age group selected (6–8 years) were enrolled in the study ( $n = 131$ ). Age, weight, and height of the participants were comparable between the schools (Table 1). The children predominantly were from families with lower to middle socioeconomic status; most of the fathers worked as civil servants or for other employers (information was from the questionnaire; data not shown). The purpose and risks of the study protocol were explained to subjects, parents, and teachers, and the parents gave their informed consent before blood sampling. The research protocol was approved by the Ethical Committee of SEAMEO-TROPED Center, Jakarta.

All participants were visited at their schools (in general between 800 and 1100 hr). Blood samples (1 ml and 5 ml in EDTA tubes) were drawn by a nurse or a medical assistant.

To evaluate the magnitude of lead pollution in each specific area, six soil samples (7–10 g) were taken (dry weather conditions) near the school buildings and at the nearest two-lane street. In addition, 1 liter of cold tap water was collected in three of the schools after flushing the pipes.

**Sample treatment and analysis.** Blood samples were immediately stored in a cooling box at 4°C. Hb, hematocrit (HCT), red blood cells (RBCs), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and MCH concentration (MCHC) were determined with a Coulter counter on the day of harvest in the 1-ml sample.

Whole blood lead was analyzed using electrothermal atomic absorption spectroscopy (AAS; Hitachi Z-8270 Polarized Zeeman AAS; Hitachi, Tokyo, Japan) at 283.3 nm in a commercial analytical laboratory. The detection limit was 1  $\mu\text{g}/100$  ml, with a sensitivity of 0.5  $\mu\text{g}/100$  ml. Repeated analyses of standard solutions confirmed the reliability of the method. Zinc protoporphyrin (ZPP) was measured fluorimetrically (excitation 415 nm, emission 595 nm) in whole blood (11,12). The coefficient of variation for repeated analyses was less than 3%.

Aliquots of the tap water collected were mixed with  $\text{HNO}_3$  (65%; 0.1 ml/100 ml), reduced in volume by boiling, and then analyzed using AAS. Soil samples (~7 g) were dried at 100°C, pulverized, and boiled for 3 hr in 200 ml  $\text{HNO}_3$  (12%). The clear supernatant was diluted with water to 100 ml in a graduated flask. Precipitates were heated and melted in presence of  $\text{Na}_2\text{CO}_3/\text{Na}_2\text{B}_4\text{O}_7$ , dissolved in  $\text{HNO}_3$ , and diluted to 100 ml. Both final solutions were analyzed by AAS, and the total lead content of soil was expressed in parts per million.

**Statistics.** Data are presented as mean  $\pm$  standard deviation (SD). SPSS statistical software (SPSS Inc., Chicago, IL) was used for statistical analysis. Normal distribution was tested according to Kolmogorov-Smirnow.

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**Table 1.** Characteristics of study population

Characteristic	Southern district		Central district				All (n = 131)
	School 1 (n = 28)	School 2 (n = 34)	School 3 (n = 37)	School 4 (n = 32)	South (n = 62)	Central (n = 69)	
Age (months)	83.8 ± 7.9	86.0 ± 10.8	85.8 ± 8.8	82.5 ± 9.7	85.0 ± 9.6	84.3 ± 9.3	84.6 ± 9.4
Boys	86.2 ± 9.0	82.9 ± 10.9	85.9 ± 9.8	82.8 ± 11.5	87.2 ± 10.0	84.7 ± 10.4	85.9 ± 10.2
Girls	81.7 ± 6.5	84.3 ± 10.7	85.7 ± 7.9	82.3 ± 8.7	83.1 ± 9.0	83.9 ± 8.4	83.5 ± 8.6
Weight (kg)	20.2 ± 3.5	19.3 ± 2.9	18.6 ± 2.6	18.5 ± 3.2	19.7 ± 3.2	18.6 ± 2.9	19.1 ± 3.0
Boys	20.7 ± 4.0	18.7 ± 1.9	18.0 ± 2.8	20.1 ± 3.5	19.6 ± 3.1	18.8 ± 3.3	19.2 ± 3.2
Girls	19.9 ± 3.1	19.8 ± 3.5	19.2 ± 2.2	17.4 ± 2.4	19.8 ± 3.2	18.3 ± 2.5	19.0 ± 2.9
Height (cm)	116.5 ± 6.2	115.5 ± 6.9	113.8 ± 6.2	113.7 ± 6.5	115.9 ± 6.6	113.8 ± 6.3	114.8 ± 6.5
Boys	119.3 ± 6.2	115.0 ± 5.7	113.3 ± 7.4	115.3 ± 7.4	116.9 ± 6.2	114.1 ± 7.3	115.4 ± 6.9
Girls	114.9 ± 5.3	116.0 ± 8.0	114.4 ± 4.7	112.6 ± 5.7	115.0 ± 6.8	113.5 ± 5.3	114.2 ± 6.1

The differences between the groups were analyzed using one-way analysis of variance (ANOVA) and the Student's *t*-test. Correlation between two normally distributed variables was tested according to Pearson. The nonparametric Kruskal-Wallis H-test was employed for data that were not normally distributed. Differences between groups were then identified by the Mann-Whitney U-test. Differences were considered significant at  $p < 0.05$ .

## Results

In the three tap water samples analyzed, the concentration of lead was less than 0.01 mg/l. Contamination of soil ranged from 77 to 223 ppm, with higher values for the central area (Table 2).

As shown in Figure 1, blood lead concentrations were normally distributed; 73.3% of the children had blood levels below 10  $\mu\text{g}/100\text{ ml}$ . The mean blood lead

concentration analyzed was 7.7  $\mu\text{g}/100\text{ ml}$  and was not influenced by sex. The average value measured in children attending schools in the central area was significantly higher than in those in southern Jakarta (Table 3).

All hematological parameters showed a normal distribution; mean values are summarized in Table 3. There were no statistical differences between the schools. In 24% of the children (7.5% from the south and 16.5% from the central district), ZPP levels were higher than the upper reference value (70  $\mu\text{mol}/\text{mol Hb}$ ). Only 5.7% had an average concentration of RBCs below  $4.1 \times 10^6/\text{mm}^3$ , and about 17% (4.9% from the south and 12.2% from the central district) had Hb levels under 11 g/100 ml. All other values were within the physiological range.

A highly significant correlation was found between the number of RBCs and Hb ( $r = 0.50$ ;  $p < 0.001$  between RBCs and HCT ( $r = 0.68$ ;  $p < 0.001$ ), and between Hb

**Table 2.** Lead concentration in tap water and soil

	Pb in soil ( $\mu\text{g}/\text{g}$ )		Pb in drinking- water (mg/l)
Southern district			
Jl. Bacang	77	School 1	<0.01
Jl. Salihara	143	School 2	NA
Central district			
Jl. Cidurian	196	School 3	<0.01
Jl. Amfion	223	School 4	<0.01
Jl. Cikini	119		
Jl. Salemba	200		

NA, not applicable.

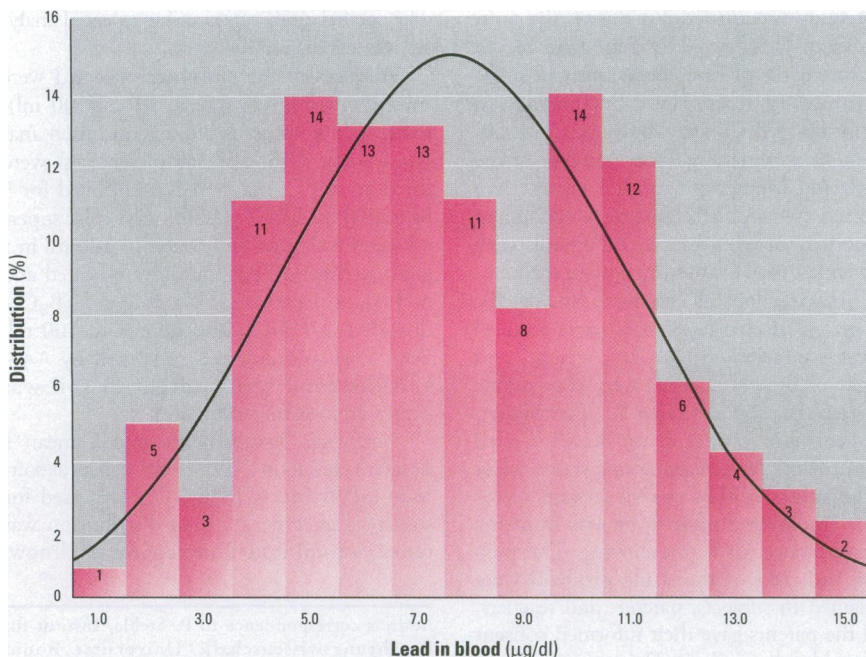
and HCT ( $r = -0.26$ ;  $p < 0.05$ ). Blood lead concentrations and hematological biomarkers were not correlated.

## Discussion

Since the early 1970s, soil has been considered to be noncontaminated when the lead concentration is  $\leq 70\text{ }\mu\text{g}/\text{g}$ . This value was derived from measurements in the United States when lead in gasoline was near its peak usage (13). Recent studies revealed median lead concentrations below 10  $\mu\text{g}/\text{g}$  in soil samples from rural areas (14). Thus, the reference limit should probably be lowered by a factor of nearly 10. Based on these considerations, all soil samples should be ranked as contaminated. Indeed, analyses of six soil samples are not representative for the whole area. Nevertheless, these measurements provide a hint of the trend.

Lead contamination of soil is proportional to the lead concentration in the surrounding atmosphere. Measurements performed by Indonesian authorities in various locations of Jakarta in 1993 revealed values between 0.84 and 1.72  $\mu\text{g Pb}/\text{m}^3$ , the average contamination being higher in central districts (1.47  $\mu\text{g Pb}/\text{m}^3$ ) compared to south Jakarta (0.93  $\mu\text{g}/\text{m}^3$ ) (unpublished data, Environmental Impact Management Agency, Jakarta, Indonesia). This observation is in line with our results that soil in central areas is more contaminated. In urban and rural areas in African countries, average lead concentrations are considerably higher ( $>1,000\text{ }\mu\text{g}/\text{g}$  dust and soils) (9), presumably due to the higher lead content of gasoline.

Whether contaminated soil or dust plays a quantitative role in the lead exposure among schoolchildren is still controversial. In a recent study, no association between blood lead levels and lead in soil could be found in Uruguayan children (14). In contrast, Mielke et al. (15) stated that blood lead in children is closely associated to soil lead and that primary lead prevention should also consider this factor. As recently pointed out, mouthing behavior is an important mechanism of lead exposure



**Figure 1.** Distribution (% of total) of blood lead concentrations in the study population. Mean  $\pm$  standard deviation =  $7.7 \pm 3.21$ ;  $n = 120$ .

**Table 3.** Blood lead concentrations and hematological biomarkers in schoolchildren from Jakarta (mean  $\pm$  standard deviation)

Biomarkers	School 1 (n = 22)	School 2 (n = 31)	School 3 (n = 36)	School 4 (n = 31)	Southern district (n = 53)	Central district (n = 67)	All (n = 120)
Pb ( $\mu\text{g}/100\text{ ml}$ )	5.9 $\pm$ 2.6	7.6 $\pm$ 3.9	8.5 $\pm$ 2.5 <sup>a</sup>	8.1 $\pm$ 3.2 <sup>a</sup>	6.9 $\pm$ 3.5	8.3 $\pm$ 2.8 <sup>b</sup>	7.7 $\pm$ 3.2
ZPP ( $\mu\text{mol}/\text{mol Hb}$ )	58.4 $\pm$ 13.0	57.3 $\pm$ 13.3	62.6 $\pm$ 21.0	59.4 $\pm$ 23.1	57.7 $\pm$ 13.0	61.1 $\pm$ 21.9	59.6 $\pm$ 18.5
RBCs ( $10^9/\text{nm}^3$ )	4.51 $\pm$ 0.42	4.53 $\pm$ 0.39	4.65 $\pm$ 0.36	4.56 $\pm$ 0.32	4.52 $\pm$ 0.40	4.61 $\pm$ 0.34	4.57 $\pm$ 0.37
Hb (g/l)	120 $\pm$ 9	117 $\pm$ 7	117 $\pm$ 6	116 $\pm$ 8	118 $\pm$ 8	117 $\pm$ 7	118 $\pm$ 7
HTC (%)	34.4 $\pm$ 2.8	34.5 $\pm$ 2.2	34.6 $\pm$ 1.8	34.5 $\pm$ 2.3	34.4 $\pm$ 2.4	34.6 $\pm$ 2.0	34.5 $\pm$ 2.2
MCH (pg/cell)	26.7 $\pm$ 1.7	26.0 $\pm$ 2.1	25.3 $\pm$ 1.6	25.4 $\pm$ 1.6	26.3 $\pm$ 1.9	25.4 $\pm$ 1.6	25.7 $\pm$ 1.8
MCHC (g/100 ml)	34.9 $\pm$ 1.1	34.0 $\pm$ 1.0	33.9 $\pm$ 0.7	33.5 $\pm$ 0.7	34.4 $\pm$ 1.1	33.7 $\pm$ 0.7	34.0 $\pm$ 1.0
MCV ( $\mu\text{m}^3$ )	76.4 $\pm$ 3.7	76.4 $\pm$ 5.0	74.6 $\pm$ 3.9	75.9 $\pm$ 4.0	76.4 $\pm$ 4.5	75.2 $\pm$ 4.0	75.7 $\pm$ 4.2

Abbreviations: ZPP, Zinc protoporphyrin; RBCs, red blood cells; Hb, hemoglobin; HTC, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume.

<sup>a</sup>Significantly different from school 1 ( $p < 0.05$ ).

<sup>b</sup>Significantly different from the Central district ( $p < 0.05$ ).

in urban children (16). With increasing age, however, the children learn to wash their hands and to clean fruits and vegetables before consumption. Moreover, children in better socioeconomic situations regularly take showers. Thus, it can be assumed that contaminated soil contributes only in minor quantities to the total lead exposure of the schoolchildren in our study.

Lead content in tap water is only a problem when lead pipes are used (9,17). This is generally not the case in Jakarta; thus, lead content of drinking water was expectedly low (Table 2).

Because adverse effects of lead are already seen at very low blood lead concentrations (18) and because lead is ubiquitous in our environment, the definition of an acceptable no-effect level in blood is very crucial. In a remote Himalayan population, average blood lead concentrations of 2.7  $\mu\text{g}/100\text{ ml}$  were measured (7); in Yanomami Indians, lead in blood was not higher than 0.84  $\mu\text{g}/100\text{ ml}$  (19). Thus, the physiological concentration of lead in humans is speculated to be considerably lower than the actual average level generally observed. In 1982, the World Health Organization defined 20  $\mu\text{g}/100\text{ ml}$  as the upper acceptable limit. Based on recent reports (20), the U.S. EPA and the Centers for Disease Control lowered this level in 1991 to 10  $\mu\text{g}/100\text{ ml}$  (21). In our study, more than 26% of the schoolchildren investigated had lead blood levels above this acceptable limit and must be considered to be at an increased risk for neurological dysfunction, e.g. lower IQ and decreased learning ability (6).

Blood lead levels in a comparable range have been observed in children in Mexico City (22) and in South African cities (23). In both studies, traffic was identified as a quantitative factor that influenced blood lead concentrations. Our observation that blood levels are higher in the central than in the southern district (Table 3) is in line

with this interpretation. In Germany, the ban on adding lead to gasoline was associated with a significant decline in blood lead levels; recently, concentrations of about 5–6  $\mu\text{g}/100\text{ ml}$  have been reported (24).

As previously published, lead-induced alterations of heme biosynthesis should not occur until blood levels are higher than 15  $\mu\text{g}/100\text{ ml}$  (25). In our study, only 1.7% of the children exhibited lead levels above this range. The incidence for nonphysiologically high ZPP levels and decreased concentrations of hematological biomarkers was, however, considerably higher. Thus, we concluded that these effects were not only caused by lead poisoning but most likely by nutritional deficiencies. This interpretation is further supported by the fact that blood lead levels and hematological measures were not correlated. In addition, our data confirmed recent reports that ZPP cannot be used as indicator of low-level lead exposure (26).

Although our data should be further strengthened by a cross-sectional study including rural schools, it can be attentively estimated that more than 20% of the 1.8 million Indonesian children under 10 years of age live at high risk for lead poisoning. We expect that the number of motor vehicles and the extent of air pollution will considerably increase during the next decade. Consequently, activities to reduce pollution and to monitor lead exposure are needed. One important step should be the reduction of lead in gasoline, a measure that has proven to be effective in lowering blood lead in Western countries.

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