# SOURCES OF LEAD EXPOSURE IN URBAN SLUM SCHOOL CHILDREN

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**ABSTRACT**

*A school based study was undertaken to trace various sources of lead exposure in urban slum children. Two cases with elevated venous blood lead (PbB) levels were detected and confirmed in 100 consecutive Bombay school children, referred to a hospital school clinic for clinical pallor. A common source of lead exposure in one suburb, was implied by the significantly higher PbB levels here. Systematic family and environmental studies identified the source was a nearby factory manufacturing lead storage batteries. Similar studies for the other index case traced the source to the dust produced by small scale foundries and secondary lead smelters. This study indicates that large scale screening studies are necessary to establish the magnitude and epidemiology of this problem in Bombay.*

**Key words:** *Lead poisoning, Screening, Urban slum school children.*

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Childhood lead poisoning is a health problem faced by most of the industrialized world(1), due to combustion of leaded petrol, lead emitted by stationary sources and use of lead paints(2).

Bombay is one of the most densely populated and industrialized cities of India with over 450,000 registered vehicles. Automobile exhaust is a major cause of elevated blood lead levels (PbB) in children in Bombay(3). No screening programs for childhood lead poisoning exist in India and the epidemiology and magnitude of this problem is unknown. Information about the locally occurring health effects of existing environmental hazards is also scarce(4). This study attempts to trace the possible sources of lead exposure in urban school children.

## Material and Methods

Between July, 1988 and February, 1989, we studied 100 consecutive school children aged 4 to 15 years, referred to the School Clinic of a Municipal Hospital in Bombay, for clinical pallor by the attending school health physician. They were students of 14 Municipal public schools from Bandra to Goregaon (*Fig*). There were 38 boys and 62 girls, predominantly from Social Class IV(5), most were less than the 5th percentile of weight for age(6). A history of anorexia, vomiting, irritability, abdominal pain, seizures or ataxia was asked for.

After a clinical examination, blood was collected for complete blood count and venous blood lead (PbB) measurements and tested within one day of collection. It was not possible to screen for erythrocyte protoporphyrin (EP) or serum iron in this study. The criteria used for diagnosis of anemia and microcytosis were hemoglobin concentration and mean corpuscular

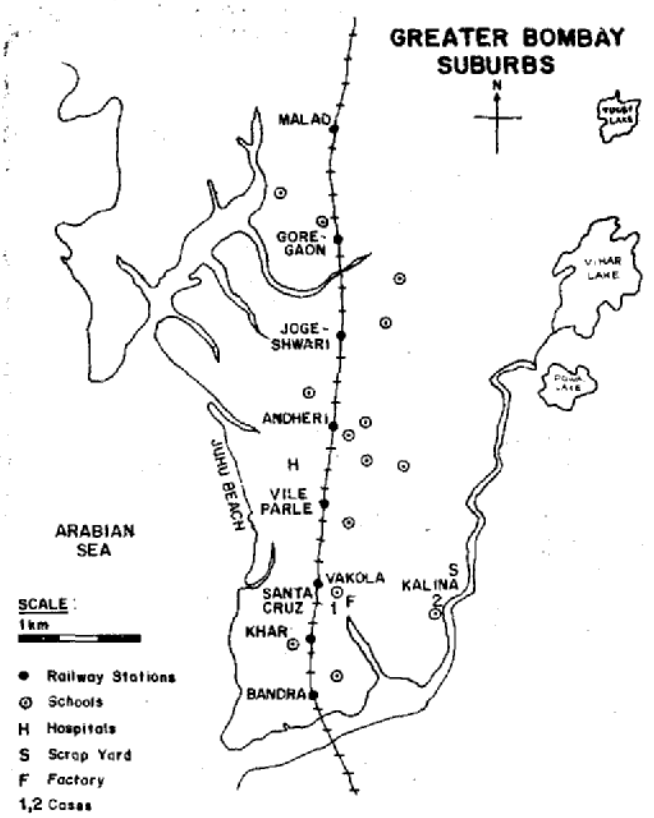


*Fig. Map showing Greater Bombay suburbs, location of Hospital (H), Scrapyard (S), and Factory (F) and Sites of detection of two cases with elevated blood lead levels.*

Railway Stations
⊙ Schools
H Hospital
S Scrapyard
F Factory
1,2 Children with elevated blood lead levels
Scale 1 cm = 1 km

volume (MCV), respectively(7). The normal values depend on age. The cut off points of Hb concentration were 11.5 g/dl; 12 g/dl and 12.5 g/dl for age groups of 4 to 7 years; 8 to 11 years and 12 to 14 years, respectively. The lower limits for MCV for these age groups were 75, 76 and 77 fl, respectively. Patients with elevated PbB (> 25 $\mu$g/dl) were recalled for follow-up an average of 2 weeks later by the School Health Nurse. At this time, sample for confirming high PbB was drawn and a detailed history for sources of lead exposure was taken from the accompanying parent. On confirmation of elevated PbB levels the children were admitted for a complete evaluation and treatment and subjected to more detailed study (home visits, screening of family members and a systematic examination for potential sources of lead exposure). The lead content of the school and house drinking water, dust, ambient air where required and nearby soil was estimated. In one case, we also analysed the lead content of a cosmetic 'Sindhur' used by the mother of a student. Diet was considered as a possible source of lead and investigated. Duplicate diet samples of children were collected and the lead content estimated. Lead levels in the drinking water samples were also analysed. Special care was taken during venipuncture to prevent contamination of blood samples. Blood was collected directly into heparinised 5 ml lead free polypropylene tubes(3). The hemoglobin concentration (Hb) and mean corpuscular volume (MCV) were determined by PC-604 Blood Cell Counter (Erma Inc., Tokyo).

The method of collection of air, dust, soil and water samples and analysis for lead content is mentioned elsewhere(8-11). The lead content of all samples were determined by Differential Pulse Anodic Stripping Voltametry (DPASV) using a Princeton Applied Research PARC-174-13 polarographic analyser system. The reliability of these procedures have been checked by analysis of reference materials obtained from International Atomic Energy Agency, Vienna and National Bureau of Standards, USA and inter-laboratory comparison samples. The results obtained were very close to the

certified/true values and the replicate analysis agreed within ±7%(12).

The test of significance used was the Student t-test.

## Result

Out of 100 school children, two cases had elevated PbB, both greater than 70 μg/dl confirmed by retesting. Clinical and laboratory data is shown in *Table I*. They were students of different schools. Both were asymptomatic but underweight (<5th percentile of weight for age) and anemic. On peripheral blood smear, there was microcytosis, but no basophilic stippling. The geometric mean PbB of the entire population was 9.84 μg/dl with a geometric standard deviation (GSD) of 1.81. *Table II* shows the area-wise distribution of the PbB values of the school children. There were significantly higher PbB levels ($p < 0.02$) in the school children of Vakola-Santacruz. The figure is a map showing greater Bombay suburbs, location of hospital scrapyard, factory and sites of detection of the two cases with elevated blood levels.

A survey around the school at Vakola-Santacruz revealed a factory manufacturing lead storage batteries 75m south-east of the school. Lead content of school dust (358.5 μg/g lead) and ambient air (0.99 μg/m³ lead) was elevated, with higher content on the higher floors. (Floors: ground: 271.6 μg/g; first 367.2; second 388.9; third 406 μg/g). The first child lived in a slum just adjacent to the factory in which her father had been a clerk for 20 years. High blood levels were found in the fathers (47.6 μg/dl) and brothers (28 μg/dl) blood, their house dust (350 μg/g) and ambient air (1.3 μg/m³). The girl's mother did not use Sindhur.

The second child was a student of Municipal School at Kalina-Santacruz, situated about 3 Km east of the factory. She lived with 5 other family members in a make-shift house in the midst of a scrapyard for industrial metallic and nonmetallic wastes, about 1.5 km from her school. There were several warehouses and a few small scale foundries and secondary lead smelters in the immediate vicinity. The whole environment around the house was dusty and dirty, with very high lead content in the house dust (18139 μg/g), ambient air (41.2 μg/m³), soil nearby (2351.5 μg/g) and the cosmetic "Sindhur" (43% lead by weight) used by the girl's mother. All family members showed elevated PbB levels (38.2, 63.6, 78.3, 60 and 49.6 μg/dl). The lead content of the ambient air near the school was elevated (1.22 μg/m³). A geometric mean of 12 μg lead with a geometric standard deviation of 1.30 was found in the duplicate diet samples. Drinking water lead content was 2.20 μg/litre (permissible limit of Pb in drinking water is 50 μg/litre).

## Discussion

In our target group (students of Municipal Schools) many were from the lower socio-economic class, residing in nearby slums. Several of them were poorly nourished and anemic and also predisposed to the effects of lead toxicity(13). We did not study pre-school children, in order to eliminate the variation in PbB due to pica or mouthing(14) and because we could not ensure follow-up. Childhood lead toxicity screening programmes rely on the initial estimation of erythrocyte protoporphyrin (EP) confirmed by PbB(15). As the facility for EP estimation was not available to us, we chose to measured PbB directly. Hence, we were able to recognize acute exposure to lead, but not cumulative toxicity.

TABLE I—*Clinical and Laboratory Data of 2 Children with Elevated Blood Lead Levels*

| Case | Residence | Age/Sex | Weight (kg) | Symptoms | Blood lead levels | | Follow up (days) | Hb/MCV (g/$\mu$l) | IQ |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | Initial | Follow-up | | | |
| 1 | Vakola | 11/F | 19 | Nil | 69.02 | 70.36 | 18 | 6.2/64 | 78 |
| 2 | Kalina | 9/F | 20 | Nil | 91.36 | 89.10 | 9 | 9.0/65 | 95 |

TABLE II—*Geographical Distribution of Blood Lead Levels of Bombay School Children*

| Area | Bandrakhar | Santa Cruz-Vakola | Vile-Parle | Andheri | Jogeshwari | Goregaon | Total |
|---|---|---|---|---|---|---|---|
| No. of school children (n) | 9 | 13 | 9 | 38 | 20 | 11 | 100 |
| Geometric Mean ($\bar{X}g$) $\mu$g/dl | 9.0 | 17.1 | 9.1 | 9.8 | 7.5 | 7.8 | 9.84 |
| Geometric SD ($\sigma g$) | 1.6 | 2.3 | 1.7 | 1.7 | 1.4 | 1.4 | 1.81 |

Contribution of blank to $\bar{X}$ g was 2.4 $\mu$g/dl and to $\sigma g$ was 1.02

School based screening of children for lead toxicity led to the recognition of important sources of lead pollution in the suburbs of Bombay. In the first case, the source of lead pollution was a factory manufacturing lead storage batteries near her school and home. Exposure of all the school children of Vakola-Santacruz, was implied by their significantly higher PbB values and the increased lead levels in the school ambient air and dust. The higher content of lead on the upper floors of the school building, suggested airborne spread. Further, her brother and father (employed in the factory) had elevated PbB levels, with high lead in the air and dust of their home. Lead poisoning in the vicinity of smelters and other stationary sources, owing to the high ambient air lead concentrations are well recognised(14,16). In a long term study of Belgian children attending schools 1 and 2.5 km from a lead smelter, there was a high correlation of PbB with playground dust and air lead. Although air borne lead is the starting point of environmental contamination, the enhanced daily and intake is the primary identifiable factor predicting the PbB of school children(17).

The second girl lived about 3 km away from the factory, making it less important as a source of lead exposure. More important, this family lived in a scrap-yard for industrial wastes with warehouses and secondary lead smelters in the vicinity and the entire family had elevated PbB levels.

Each increase of 100 ppm in the lead content of surface soil above a level of 500 ppm is associated with a mean increase in children's whole blood lead level of 1 to 2 $\mu$g/dl(18). The elevated lead levels in dust, soil and ambient air observed in the second case are higher than those reported in other studies in Bombay(8,10,11). The ambient air lead concentration was above the maximum permissible level of 1.5 $\mu$g/mg set by the Environmental Protection Agency (USA)(19).

In the second case, the girl's mother used "Sindhur" with a very high content of lead. "Sindhur" has been earlier implicated in causing undue lead absorption(20). Inspite of the very high PbB in these cases, there were no symptoms or signs except for anemia. When dust and soil are the only sources of lead, symptoms are rare, though toxicity occurs(18).

The goal of prevention is to reduce children's exposure to lead to the maximum extent. Lead hazards must be identified and removed from the environment. The community and especially parents of young children should be informed about the sources (*e.g.,* lead based paint, Sindhur) and basic preventive measures emphasised (*e.g.,* wet mopping and removal of accessible paint flakes and dust to reduce potential hazards in the child's environment). Emissions from industrial sources should be reduced and factories as part of their licensing specifications should have minimal lead emission.

As a sequel to this study, a large scale program is under way to establish the magnitude, prevalence and epidemiology of lead poisoning in children of Bombay.

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## NOTES AND NEWS

### PAKISTAN PEDIATRIC ASSOCIATION

#### XIth International Pediatric Conference

The XIth Biennial International Pediatric Conference is scheduled to be held in Karachi from *February 4-7, 1992* at the College of Physicians and Surgeons, Pakistan, Karachi. The conference is expected to host more than 700 national and international delegates. In addition to leading Pediatricians of Pakistan and the SAARC region, we expect participation by over a dozen international authorities.

An interesting comprehensive programme of a wide ranging scientific, content is being drawn up. In addition there will be a full social and cultural programme for participants and accompanying spouses. Overseas visitors may also be able to avail a wide variety of breathtaking sightseeing tours that Pakistan offers.

Registration Fee:
(including Banquet and variety programme)

| | *by 30.11.1991* | *Late fee* |
|---|---|---|
| Consultants | Rs. 750/- | 900/- |
| Overseas Registrants | US$ 150/- | 200/- |
| (Accompanying spouses) | US$ 75/- | |

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