Blood lead in pregnant women in the urban slums of Lucknow, India

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Abstract

Objective—To determine the concentrations of blood lead (PbB) in pregnant women in the slums of Lucknow, north India.

Methods—Of the 203 designated municipal slums in Lucknow, 70 were randomly selected for study and a cohort of 500 pregnant women was enrolled. Each participant was interviewed with questions on possible sources of exposure to lead, surrogates of nutritional status were measured, and PbB was measured.

Results—The mean PbB was 14.3 μg/dl and 19.2% of women had PbB ≥ 20 μg/dl. PbB was not associated with age, height, weight, gestation, or history of abortions, although higher PbB was associated with higher parity. Women living in inner city neighbourhoods near heavy vehicular traffic had PbB 2-2 μg/dl higher (95% confidence interval (95% CI) 0.8 to 3.6) than those living in other neighbourhoods. The PbB was not associated with reported use of piped water or the presence of paint in homes, and increasing PbB was unexpectedly associated with decreasing use of the eye cosmetic “surma” and the duration of gestation.

Conclusions—The high PbB found in this population raises concerns about fetal development and points to the urgent need to reduce exposure to lead.

Keywords: lead; pregnancy; fetus; neurotoxicity; India

Although lead toxicity in children and adults is well recognised, exposure to lead is of special concern during pregnancy. Lead absorbed by the pregnant mother is readily transferred to the developing fetus.1 2 There is evidence from animal studies that intraterine exposure to lead may disrupt endocrine balance during pregnancy3 and lead to abnormalities of renal structure and function,4 5 abnormalities of the reproductive system,6 7 and neurodevelopmental toxicity11 13 in offspring. Human evidence corroborates these findings, linking prenatal exposure to lead with reduced birth weight and preterm delivery14 15 and with neurodevelopmental abnormalities in offspring.16 21 These concerns are especially salient for women and children in developing nations. Not only is exposure to lead common, but the toxicity of lead for pregnant women and their offspring may be amplified by nutritional deficiency22 23 and concomitant toxic exposures24 which often occur in poor nations.

The aim of this study was to find the distribution of PbB among pregnant women in Lucknow, India, a densely populated city of two million in Uttar Pradesh, northern India. The study was prompted by the fact that lead petrol is still used in Lucknow. Also, we were aware that a traditional Indian eye cosmetic “surma” contains lead, and might pose a hazard for women who use it through exposure to lead. This study was part of a larger ongoing study of the effect of maternal exposure to lead on neonatal birth weight and other outcomes of pregnancy.

Methods

This was a cross sectional study conducted in the urban slums of Lucknow. Under the national healthcare system, “slum” is an administrative designation that, in urban areas, means a population of about 1000 people, with incomes below the national poverty level and suboptimal living conditions. India’s integrated child development scheme provides primary care for children and pregnant women in each slum through an “aganwadi” centre. Pregnant women in each slum register at their local centre to receive prenatal care, and are therefore listed. Of Lucknow’s 203 slums, 70 were randomly selected and visited by a research assistant at least twice (in the same order) from June 1994 to July 1995. Pregnant women were identified by research assistants from the slum health workers’ registers, and were invited to participate in the study. This process continued until 500 subjects were enrolled. Potential subjects were excluded if they declined to participate or if they indicated a likelihood of moving out of the slum in the next six months.

Personal interviews were conducted to collect data on age, reproductive history, and date of last menstrual period, correct to the nearest week. Participants were also asked about other sources of potential exposure to lead, including the source of drinking water, use of surma, and the presence of lead paint at home. Maternal nutritional status was estimated by measuring weight (kg), height (cm), and haemoglobin (g/dl). For measurement of PbB, 5 ml of venous blood was taken after carefully cleaning the skin at the venepuncture site. Analysis of blood lead was performed at the laboratory of the Industrial Toxicology Department of Pediatrics and Clinical Epidemiology Unit, King George’s Medical College, Lucknow, India

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Research Centre (ITRC) in Lucknow, India. Five ml blood was digested in HNO₃:HClO₄ (6:1) for 24 hours and slowly evaporated to dryness. The process was repeated three times. The analyte was then brought to a volume of 4 ml in 1% HNO₃. Blood lead was analysed with graphite furnace atomic absorption spectroscopy (GF-AAS; SpectrAA-250 Plus, Varian, Palo Alto, California). To account for any matrix effects present, measurement was effected through the use of a four point standard addition calibration with concentrations developed from a traceable standard (National Physical Laboratory, CSIR, New Delhi). The mean of four replicate analyses were used to assess the concentration. The coefficient of variation of the four analyses was < 2% for all samples. The ITRC laboratory participates in Indian national quality control programmes, and has applied for ISO 9000 certification.

This study was approved by the ethics committee of King George’s Medical College, and all subjects gave informed consent before their participation.

After the sampling was completed, we hypothesised that certain neighbourhood characteristics might be associated with variations in the concentration of PbB. We therefore divided the 70 slums into four categories, according to potential sources of environmental exposure to lead. These were (a) the inner city (with high population density and heavy go and vehicular traffic), (b) near industrial sources (factories producing batteries and paint, with medium population density and light vehicular traffic) that could potentially emit lead, (c) near an industry unlikely to emit lead (a large beer distillery, with medium population density and light vehicular traffic), and (d) near roads (with low population density and heavy, fast moving vehicular traffic). Univariate analyses were conducted to characterise the distribution of PbB by demographic, clinical, and exposure related variables.

**Results**

From the 70 participating slums, 500 pregnant women were enrolled, an average of 7.2 women per slum. The mean PbB was 14.3 (SD 7.9) μg/dl, and the median PbB was 12.9 μg/dl. One hundred and eighty one women (36-2%) had concentrations of PbB ≤ 10 μg/dl, 223 (44-6%) had concentrations 11 to 20 μg/dl, and 96 (19-2%) had concentrations > 20 μg/dl, of whom 25 women had concentrations of ≥ 30 μg/dl. The figure shows the distribution of PbB. Table 1 shows the concentration of PbB against the distribution of various demographic, clinical, and exposure related variables. The concentrations of PbB did not vary with age, weight, height, haemoglobin, gestation, or reproductive history, except that more of the women with high concentrations of lead were multiparous.

Of the 70 participating slums, 36 (51-4%) were inner city slums, nine (11-5%) were near industries that could potentially emit lead, five (6-4%) were near an industry that did not emit lead, and 20 (28-6%) were near highways. Table 2 shows the PbB results from these four categories of neighbourhood. Women in the inner city slums had a mean PbB that was 2.2 (95% confidence interval 95% CI 0.8 to 3.6) μg/dl higher than the combined concentration of women in the other categories.

As for other potential sources of exposure to lead, 193 subjects (38-6%) reported the use of surma, 376 subjects (75-2%) reported the use

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**Table 1** Distribution of demographic, clinical, and exposure related variables by concentration of lead blood

<table>
<thead>
<tr>
<th>PbB (μg/dl)</th>
<th>&lt; 10</th>
<th>11-20</th>
<th>&gt; 20</th>
<th>Total</th>
<th>P value of ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number (%)</td>
<td>181 (36-2)</td>
<td>223 (44-6)</td>
<td>96 (19-2)</td>
<td>500</td>
<td>0.77</td>
</tr>
<tr>
<td>Age (y, mean (SD))</td>
<td>26.4 (6-8)</td>
<td>26.3 (6-6)</td>
<td>26.9 (6-8)</td>
<td>26.5 (6-7)</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg, mean (SD))</td>
<td>42.9 (6-2)</td>
<td>42.5 (5-3)</td>
<td>41.3 (6-8)</td>
<td>42.79 (6-4)</td>
<td>NS</td>
</tr>
<tr>
<td>Height (cm, mean (SD))</td>
<td>149-8 (4-7)</td>
<td>149-9 (4-9)</td>
<td>150-1 (4-8)</td>
<td>149.9 (4-8)</td>
<td>NS</td>
</tr>
<tr>
<td>Gestation (weeks, mean (SD))</td>
<td>20-2 (6-6)</td>
<td>19-9 (6-6)</td>
<td>19-5 (6-6)</td>
<td>19-9 (6-6)</td>
<td>NS</td>
</tr>
<tr>
<td>Parity (n (%)):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>37 (20-4)</td>
<td>47 (19-7)</td>
<td>6 (6-7)</td>
<td>90 (18-0)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>144 (79-6)</td>
<td>176 (78-9)</td>
<td>90 (93-7)</td>
<td>410 (82-0)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>156 (86-2)</td>
<td>193 (86-5)</td>
<td>84 (87-5)</td>
<td>433 (86-6)</td>
<td></td>
</tr>
<tr>
<td>Trimester (n (%)):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>25 (13-8)</td>
<td>30 (13-5)</td>
<td>12 (12-5)</td>
<td>67 (13-4)</td>
<td>0.77</td>
</tr>
<tr>
<td>2</td>
<td>111 (61-3)</td>
<td>136 (60-9)</td>
<td>58 (60-4)</td>
<td>305</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>29 (16-2)</td>
<td>31 (15-4)</td>
<td>15 (15-2)</td>
<td>55 (11-4)</td>
<td>0.93</td>
</tr>
<tr>
<td>4</td>
<td>62 (34-3)</td>
<td>60 (28-6)</td>
<td>23 (23-9)</td>
<td>145</td>
<td>0.0005</td>
</tr>
<tr>
<td>Residence (n (%)):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>61 (31-7)</td>
<td>86 (38-6)</td>
<td>50 (52-1)</td>
<td>197</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>29 (16-0)</td>
<td>53 (23-8)</td>
<td>12 (12-5)</td>
<td>94</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>29 (16-0)</td>
<td>24 (10-8)</td>
<td>11 (11-5)</td>
<td>64</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>62 (34-3)</td>
<td>60 (28-6)</td>
<td>23 (23-9)</td>
<td>145</td>
<td>0.0005</td>
</tr>
<tr>
<td>Lead paint (n (%)):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>58 (32-0)</td>
<td>47 (21-1)</td>
<td>23 (23-9)</td>
<td>128</td>
<td>0.06</td>
</tr>
<tr>
<td>2</td>
<td>131 (72-4)</td>
<td>172 (77-1)</td>
<td>73 (76-0)</td>
<td>376</td>
<td>0.39</td>
</tr>
<tr>
<td>Surma (n (%)):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>78 (43-1)</td>
<td>89 (39-9)</td>
<td>26 (27-1)</td>
<td>193</td>
<td>0.015</td>
</tr>
</tbody>
</table>

*1 = Inner city (with heavy vehicular traffic); 2 = industrial (with potential lead emissions); 3 = industrial (without potential lead emissions); 4 = near highway.
Table 2 Distribution of PbB by type of neighbourhood

<table>
<thead>
<tr>
<th>Type of neighbourhood</th>
<th>Slums n</th>
<th>Women n</th>
<th>Concentration of PbB (µg/dl) (mean (SD))</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inner city</td>
<td>36</td>
<td>197</td>
<td>15.65 (8.17)</td>
<td></td>
</tr>
<tr>
<td>Industrial (with potential Pb emissions)</td>
<td>9</td>
<td>94</td>
<td>13.57 (6.40)</td>
<td>0.03</td>
</tr>
<tr>
<td>Industrial (without potential Pb emissions)</td>
<td>5</td>
<td>64</td>
<td>12.99 (7.14)</td>
<td>0.01</td>
</tr>
<tr>
<td>Near highway</td>
<td>20</td>
<td>145</td>
<td>13.64 (8.40)</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Taiwan of piped water, and 128 (25.6%) reported the presence of paint on surfaces in their homes. The reported use of surma decreased with increasing PbB, and the reported presence of lead paint showed a trend in the same direction; both findings were unexpected.

Discussion

This study showed increased concentrations of PbB among pregnant women in the slums of Lucknow. In a sample of 500 women, the mean PbB was 14.3 µg/dl. Ninety-six women (19.2%) had PbB > 20 µg/dl, of whom 25 had PbB ≥ 30 µg/dl.

These results confirm previous data showing high concentrations of lead in the Indian population. In the early 1980s, mean (SD) concentrations of PbB among female teachers in various Indian cities were as follows: Ahmedabad 14.7 (7.26), Bangalore 19.8 (10.15), and Calcutta 10.8 (3.46) µg/dl. At the same time, a population study in Bombay found a mean concentration of PbB to be 35.6 µg/dl. Another recent study of pregnant women in Lucknow found a mean (SD) PbB of 19.4 (8.51) µg/dl among those with normal deliveries and 22.52 (8.76) µg/dl among those with various adverse outcomes such as preterm delivery, stillbirth, and spontaneous abortion. The higher concentrations found in the previous study in Lucknow may be explained by the fact that it was based in hospital, and involved a population selected for high obstetric risk.

These results suggest that Indian women of child bearing age have a higher biological lead burden than their counterparts in other developing countries. Table 3 shows that mean concentrations of PbB in pregnant women in developing nations ranged between 5.5 and 15.2 µg/dl, with the exception of a small study in Durban, South Africa with higher concentrations. There are several reasons why Indian women might have higher concentrations of PbB. The leading source implicated by our data was motor vehicle traffic as PbB was higher among women in inner city neighbourhoods, where traffic exposure is most intense. As our analysis was ecological, and individual women are not likely to spend all of their time in the neighbourhoods where they live, our results are probably biased toward the null, understimating the neighborhood effect, and underestimating the impact of lead in petrol. The lead content of Indian petrol remains high, ranging up to 0.56 g/l, and unleaded petrol only reached the Indian market in 1995. Lead petrol is well established as an important factor in concentrations of lead in the population.

Several other environmental sources of lead may affect Indian women, including surma, contaminated water supplies, and lead paints. However, our results did not point to any of these sources, and if anything the results for lead paint and surma showed an unexpected trend away from an association. Before concluding that these sources are harmless in India, further consideration is necessary.

Surma and a related but more finely ground cosmetic kohl have been found to contain lead at concentrations as high as 92%, although specific data on surma from Lucknow are not available. Studies of children in Israel, Saudi Arabia, Kuwait, the United Kingdom, and the United States have linked the use of these cosmetics with increased PbB, although studies of children in Delhi and Glasgow did not find such an association. The presumed routes of absorption in children are conjunctival (the cosmetics are applied directly to the palpebral conjunctiva) and gastrointestinal (when children rub their eyes and lick their fingers). There is also evidence that adults who use surma may develop increased PbB, based on a study of pregnant women in Saudi Arabia. The negative findings for surma in our study may be explained in several ways: the lead content of local surma may be negligibly low, the imprecise subjects in this study may use surma sparingly or infrequently due to its cost, there may have been exposure misclassification due to misreporting of use of surma, or absorption of lead from local surma may be low. More detailed study of surma in India would help clarify the risk from this common cosmetic.

The negative findings for water supplies may be due to exposure misclassification by subjects unaware of their household water source. It may also indicate low consumption of tap water, or low concentrations of lead in tap water due to limited lead in Lucknow's water distribution system or water that does not dissolve lead due to hardness or alkalinity. Finally, the negative findings for paint are perhaps least surprising. Most homes in Lucknow slums, if they have any painted surfaces at all, only have paint on limited areas such as door frames. Moreover, as leaded paint is more expensive than alternatives, much of the paint used in homes is probably lead free. Therefore, the high prevalence of reported lead paint (25.6%) probably reflects misclassification.

The toxicokinetics of lead in pregnancy are complex. Previous reports have noted a fall

Table 3 Previously reported PbB among pregnant women in developing nations

<table>
<thead>
<tr>
<th>Location</th>
<th>Subjects</th>
<th>PbB (µg/dl) (mean (SD))</th>
<th>Reference (year)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Riyadh</td>
<td>124</td>
<td>5.5 (2.6)</td>
<td>28 (1995)</td>
</tr>
<tr>
<td>Bangkok</td>
<td>500</td>
<td>6.2 (2.0)</td>
<td>29 (1994)</td>
</tr>
<tr>
<td>Mexico City:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At term</td>
<td>250</td>
<td>9.0 (6.0)</td>
<td>30 (1993)</td>
</tr>
<tr>
<td>Durban</td>
<td>21</td>
<td>8.5 (6.2)</td>
<td>30 (1993)</td>
</tr>
<tr>
<td>Shanghai</td>
<td>165</td>
<td>14.1 (6.8)</td>
<td>32 (1990)</td>
</tr>
<tr>
<td>Taiwan</td>
<td>147</td>
<td>6.5 (4.2)</td>
<td>33 (1991)</td>
</tr>
<tr>
<td>Kuala Lumpur</td>
<td>114</td>
<td>15.2 (4.2)</td>
<td>34 (1985)</td>
</tr>
</tbody>
</table>
Blood lead in pregnant women in the urban slums of Lucknow, India

in PbB during the second trimester of pregnancy, attributed to haemodilution caused by the expansion of blood volume. Others have found an increase in PbB during pregnancy, possibly due to the release of lead from bone stores. In our population, the PbB did not vary with the trimester of pregnancy. This may reflect the unusually high concentrations of PbB, or it may be related to deficiencies of iron, calcium, or zinc, which facilitate intestinal absorption of lead and mobilisation of bone lead during pregnancy. The effect of pregnancy on lead toxicokinetics needs further investigation.

A weakness of this study is that we did not attempt to ascertain exposures to other potential sources of lead. The most important of these are direct or secondary occupational exposures. Consumer goods with paint containing lead, contaminated food, and contaminated beverages are other potential sources. Future studies should attempt to ascertain the importance of these exposures.

The blood lead concentrations in the women in this study were increased to an alarming extent. In combination with poor nutrition, infectious diseases, and other disadvantages associated with poverty, the exposure to lead threatens the normal development of children. Aggressive approaches to primary prevention are needed, including the removal of lead from petrol, an intervention known to decrease concentrations of lead in the population.

Further research is necessary to identify other sources of exposure to lead, such as in consumer products and cosmetics, and to eliminate these as well.

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