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# Lead Levels in Ambient Air and Blood of Pregnant Mothers from the General Population of Lucknow (U.P.), India

P. P. Kaul,[1] S. Shyam,[1] R. Srivastava,[2] D. Misra,[1] P. R. Salve,[1] S. P. Srivastava[1]

[1] Industrial Toxicology Research Centre, Post Office Box 80, M.G. Marg, Lucknow, 226001 India
[2] Department of Sciences, Dr. Ram Manohar Lohia Awadh University, Faizabad, 224001 India

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Lead is a pollutant that emanates from use of leaded gasoline; a decade ago it was reported to account for nearly 90% of the air borne lead in the US (Rom 1992). Atmospheric automobile exhaust in India is still considered a significant contributor to total atmospheric lead (KGMC/ITRC Report 1977). Lead particles in air are estimated to have caused about 2 million hospital admissions and loss of 437,363 Intelligence quotient points in 1991 - 1992 across the major Indian cities (Carter and Kristnen 1995)

Lead (Pb) is toxic to humans when ingested or inhaled in high doses, causing encephalopathy, colic, renal diseases and anemia. Low blood lead levels can have subclinical but nonetheless harmful effects such as impairment of cognitive or neuropsychological development of children (Grandjean 1993; Bernard et al. 1995) and high blood pressure (Hense et al.1993). Deleterious effects of lead were described for heme synthesis at blood lead (PbB) levels of 100 - 200  g/l (Centre for Disease Control 1991).Lead is known to be an abortifacient (Satin et al. 1991) and is of special concern during pregnancy. The potency of lead increases as a risk factor for adverse pregnancy outcome due to its ability to reduce heme synthesis. Besides, absorption, retention, the toxicity of Pb is known to get aggravated in presence of iron deficiency. Lead absorbed by pregnant mother is readily transferred to the developing foetus (Roels 1978) at least during the third month of pregnancy until birth (Baetrop 1969).

Analysis of breast milk studies show positive linear relationship between PbB levels and breast milk Pb levels. Thus as PbB rises, the relative Pb amount in breast milk compared to PbB increases. This would make breast milk an especially toxic source of lead for the early infant. Lead exposure of the newborn through milk constitutes a hazard, especially in cases in which the initial body burden is already raised due to transplacental transfer and the absorption of Pb in infants is generally higher in milk diet probably due to binding to milk proteins which are readily absorbed.

In India, less attention has been paid on the effect of ambient air lead (PbA) levels, on blood lead (PbB) levels of pregnant mothers. Therefore, the present study was taken up with the aim of monitoring PbA levels and correlate the effects of PbA levels on PbB levels of pregnant mothers during the course of pregnancy.

*Correspondence to:* P. P. Kaul

## MATERIALS AND METHODS

Ambient air lead levels were monitored in 12 different localities divided into four zones of Lucknow, the capital city of Uttar Pradesh (India), on 24 hourly basis during the calendar years 1999 and 2000, on three monthly intervals. Suspended particulate matter samples were collected using high volume samplers (Envirotech model APM 415) from a location height of 2 to 4 meters at a rate of 12 –14 cu $m^3$/min for 24 hours on Whatman EPM - 2000 filter paper of 20 x 25 cm size. Samples were digested with nitric acid using microwave digestion system (MDS 200 CEM corporation NC USA). The digested samples were made upto 5.0 ml with double glass distilled water and read on a flameless atomic absorption spectrometer (Spectra Varian Australia) equipped with graphite tube atomizer (GTA) and with deuterium background correction. A hollow Cathode lamp (Varian) for lead was used at a working current of 5mA with 283.3 nm spectral line and 0.5 nm band width.

Pregnant mothers aged 15 to 32 years numbering 8 from each locality of Zone I, 5 from each locality of Zone II, 10 from each locality of Zone III and 20 from Zone IV, reporting at hospitals during the course of pregnancy, in the calender years 1999 and 2000, were screened for PbB at first term and at delivery. Blood was drawn by venipuncture and collected in heparinized tubes, which were evaluated for lead content prior to use. The samples were stored at 4°C.500 µl whole blood was hemolyzed by addition of 1.2 ml of 0.5% Triton X - 100 and 1%( $NH_4)_2HPO_4$ mixture solution in Polypropylene tube. 1.8 ml of trichloroacetic acid was added to the hemolyzed blood with vortex mixing. The samples were then centrifuged for 20 min. at 5000 rpm. and a volume of 10 µL supernatant was injected into the graphite tube. The instrument was calibrated using aqueous standards of 10, 20, 30, 40 µg/L .The detection limit was 2 µg/L. The calibration blank used was 0.2% nitric acid and 0.1% $(NH_4)_2HPO_4$ solution and reagent blank was diluent solution (0.5% Triton x-100 + 1% $(NH_4)_2HPO_4$. Accuracy and precision was checked by spiking samples with known amount of standard. The coefficients of variation were 5% and 4% at 10 µg/L and 40 µg/L respectively.

Statistical significances were analyzed by ANOVA (Armitage 1971). Chemicals namely Nitric acid 60% ultrapure, Triton X-100 USP grade, Ammonium hydrogen phosphate dibasic purity 99% Analar grade, Heparin-sodium salt grade 1-A approx. 170 USP units/mg were purchased from Merck India and Lead atomic absorption standard solution concentration 1000 µg/Pb/ml in 1% nitric acid Lot 36H3588 was purchased from Sigma.

## RESULTS AND DISCUSSION

The ambient air lead content in four zones covering 12 localities of Lucknow, in the calendar years 1999 and 2000 have been summarized in (Table 1). EPA while setting air quality criteria for lead suggested National Ambient Air Quality (NAAQ) standards and recommended 1.5 µg/ $m^3$ as a 3 month average, and has been followed in the present study. Blood lead levels of pregnant mothers during pregnancy have been summarized in (Table 2).

Lead content in ambient air, in 5 localities of zone 1, classified as residential areas was significantly less than the NAAQ standard limits in both calendar years 1999 and 2000.

**Table 1.** Ambient air lead concentration in Lucknow city during the calendar years 1999-2000.

| Calendar Years | Lead content in air μg/m$^3$ 1999 | Lead content in air μg/m$^3$ 2000 | Height of the sampling point in meters | NAAQ Standards (24 hrs) |
|---|---|---|---|---|
| Zone 1 Residential Area | | | | |
| Indira Nagar | 0.11 | 0.09 | 2.0 | 1.5 μg/m$^3$ |
| Gomti Nagar | 0.04 | 0.05 | 3.0 | |
| Hussainganj | 0.13 | 0.18 | 2.5 | |
| Aminabad | 0.26 | 0.10 | 3.0 | |
| Alambagh | 0.22 | 0.09 | 2.5 | |
| Zone II Heavy traffic area and railway station | | | | |
| Aliganj | 2.45 | 2.05 | 2.5 | |
| Vikas Nagar | 2.24 | 2.15 | 3.5 | |
| Charbagh | 2.90 | 2.00 | 3.0 | |
| Chowk | 2.27 | 2.12 | 3.5 | |
| Zone III Airport and Industrial Area | | | | |
| Amausi | 3.16 | 2.86 | 3.5 | 1.5 μg/m$^3$ |
| Talkatora | 3.27 | 2.90 | 3.0 | |
| Zone IV Main Marketing Centre | | | | |
| Hazratganj | 3.91 | 3.00 | 4.0 | |

The PbB level of pregnant mothers from Zone 1 in the first trimester ranged from 2-8 μg/dL with a mean of 4.7 μg/dL while at delivery the PbB levels of pregnant mothers ranged from 2 - 10 μg/dL with a mean of 6.2 μg/dL.

The 4 localities of zone II representing heavy traffic area and railway station had significantly high PbA levels than the recommended NAAQ standard limits in both the calendar years 1999 and 2000.The PbB levels of Pregnant mothers from this zone, in first trimester ranged from 9 -15 μg/dL with a mean of 11 μg/dL, while at delivery the PbB levels ranged from 11- 20 μg/dL with a mean of 18.9 μg/dL. In the 2 localities of zone III representing airport and industrial area the PbA levels were 2 times higher than the NAAQ standard limits. The PbB levels of pregnant mothers from this zone at first trimester ranged from 16 -20 μg/dL with a mean of 17.5 μg/dL while at delivery the PbB levels ranged from 21- 30 μg/dL with a mean of 25.4 μg/dL.

Zone IV, representing the main marketing centre had maximum Pb in air 3.19 μg/m$^3$, in comparison to zone II and zone III. The PbA levels were more than double the NAAQ standard limits. The PbB levels in pregnant mothers from this zone in

Furthermore, the rate of release of bone lead is accelerated during pregnancy and the accelerated bone lead release continues into lactation and lactation release can infact exceed that occurring in pregnancy. The accelerated bone lead releases during lactation extend the developmental lead exposures to early infancy via breast milk lead intake.

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