ORIGINAL ARTICLE

Causes of lead toxicity in a Nigerian city

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Background: Up to 70% of young Nigerian children have been reported to have blood lead concentrations ⩾10 μg/dl.
Aims: To better elucidate risk factors for lead toxicity among Nigerian families with children at risk for lead toxicity.
Methods: Two geographic wards in Jos, Nigeria were selected for study, one previously reported to have a high mean blood lead level (37 (SD 13) μg/dl) and one with a lower mean blood lead level (17 (SD 10) μg/dl) in young children. Data pertaining to potential risk factors for lead exposure were collected from children and adults in 34 households.
Results: The mean (SD) blood lead concentration of 275 subjects, aged 3 weeks to 90 years, was 8.7 (5.7) μg/dl (range 1–34 μg/dl); 92 (34%) had concentrations ⩾10 μg/dl. In multivariate analysis, an age of 5 years and under, flaking house paint, residence near a gasoline seller, male gender, increasing maternal and paternal education, and use of a lead ore eye cosmetic were independently associated with greater blood lead concentration. Vehicle ownership was associated with reduced lead concentration. Compared with the low-lead ward, residence in the high-lead ward remained significantly associated with greater lead values, indicating that additional factors likely contribute to lead exposure.
Conclusion: Although the cause of increased lead levels in Jos appears to be multi-factorial, several remediable sources contribute to lead exposure in Nigeria.

Lead toxicity has historically been a paediatric concern. Lead is toxic to the developing nervous system, leading to decreased language, cognition, and fine motor skills.[1,2] Lead exposure has been linked to behavioural disturbances and increased high school dropout rates.[3,4] Other toxic effects of lead include delayed growth and pubertal development in females,[5] renal impairment,[6] dental caries,[7] and hypertension.[8] Though no threshold value for the toxic effects of lead is evident, the Centers for Disease Control and Prevention regard blood lead concentrations of 10 μg/dl or greater as raised.[9]

In the United States, the prevalence of lead toxicity in the general population has fallen due to reduced exposure to lead from chipping lead based paint, lead solder for canned foods, combustion of lead based gasoline, and drinking water flowing through lead pipes. In developing countries, lead exposure among young children remains a major problem. Furthermore, the low dietary calcium intake of children in many developing countries[10,11] increases lead absorption and the risk of lead toxicity.[12] In a previous community survey in Jos, Nigeria, 70% of children 6–35 months of age had blood lead concentrations of 10 μg/dl or greater.[13] Greater levels were found in Muslims, those living near areas where automobile batteries were melted, and in those using eye cosmetics.

Because the prevention of further exposure to sources of lead is the mainstay of managing increased blood lead levels, it is essential to identify potential sources of lead exposure. The aim of this study was to investigate exposures associated with increased blood lead levels in Nigerians.

METHODS

Two administrative wards with defined geographic boundaries of Jos, Nigeria (1991 population 622 873) were selected for study based on characteristics identified from a previous randomised cluster sample of young children (6–36 months of age).[13] To discern the practices accounting for the observed association of high blood lead levels with Muslim children in the previous study, we chose two predominantly Muslim wards, one of high mean blood lead levels (37 (SD 13) μg/dl), and the other with lower mean blood lead levels (17 (SD 10) μg/dl) among young children. These were designated the "high lead" and "low lead" wards, respectively. The intended effect of this design was to remove the confounding effect of religion and to identify practices that may result in lead exposure. Households of children in the previous survey were identified, and when possible, all individuals in the household were tested. A household was defined as a group of people who are related and share living quarters. When the family of the child in the earlier survey could not be located, another household on the same street was selected.

After informed consent, household data were collected pertaining to the use of vehicles, drinking water source and method of storage, type of eating and drinking utensils, education and occupations of the parents, housing material, flaking interior paint, and the proximity of the house to a car battery smelter, gasoline seller, welder, and a major road. Individual data were collected pertaining to age, gender, breast feeding status, pica, use of lead containing eye cosmetics, and practice of *rubutu*. *Rubutu* is the Muslim tradition of drinking ink washed off paper on which a Koranic passage has been written.

Blood was collected from all family members. The hands of each subject were washed with soap and water, wiped with an alcohol swab, and dried. After lancing the finger, two spots of blood were placed on filter paper kept in a sealed plastic bag. After being air dried, the filter paper was sealed in its individual plastic bag. The blood spots were sent to the LeadTech Corporation (North Bergen, NJ) for analysis by atomic absorption spectrophotometry. Each of the two blood spots and a non-bloodstained portion of each filter paper was analysed to assess variability and exclude contamination. This method has been validated and compares well with venous whole blood lead measurement.[14]

Participating households were given a small bag of toiletries, and individuals were given multivitamins. When

Table 1 Characteristics and blood lead concentrations of 275 Nigerian subjects

Characteristic	n	Mean blood lead (µg/dl)	95% CI
Ward			
High lead	137	9.7	7.5 to 12.0
Low lead	138	7.7	6.0 to 9.4
Sex			
Male	98	9.7	7.8 to 11.5
Female	177	8.2	7.0 to 9.4
Age (years)			
0–5	64	11.2	9.1 to 13.3
6–12	63	8.2	6.4 to 10.0
13–20	51	6.6	5.1 to 8.1
21–45	70	7.7	6.0 to 9.4
>45	27	10.4	8.3 to 12.5
Religion			
Christian	18	7.3	2.3 to 12.3
Moslem	257	8.8	7.5 to 10.1
Pica in children ages 0–5 years			
Yes	9	11.0	6.2 to 15.8
No	55	11.2	9.2 to 13.3
Sources of drinking water			
Any piped water			
Yes	249	8.1	6.9 to 9.4
No	26	14.2	13.6 to 14.9
Any well water			
Yes	200	9.2	7.7 to 10.7
No	75	7.3	4.5 to 10.1
Any rain water			
Yes	213	9.4	8.2 to 10.6
No	62	6.4	5.0 to 7.7
Container for water storage*			
Oil barrel			
Yes	44	6.4	5.3 to 7.6
No	231	9.1	7.8 to 10.5
Clay pot			
Yes	91	11.6	10.0 to 13.2
No	184	7.3	6.2 to 8.4
Metal bucket			
Yes	53	9.5	7.9 to 11.1
No	222	8.5	7.1 to 9.9
Plastic container			
Yes	160	8.4	6.9 to 9.9
No	115	9.1	6.9 to 11.4
Housing material			
Cement	250	8.3	7.0 to 9.6
Mud	25	12.8	10.3 to 15.3
Painted walls			
Yes	231	9.2	7.7 to 10.6
No	44	6.4	5.4 to 7.3
Flaking paint			
Yes	193	9.5	8.0 to 11.9
No	82	6.7	4.8 to 8.6
Home near welder			
Yes	121	9.7	7.6 to 11.9
No	154	7.9	6.7 to 9.1
Home near battery smelter			
Yes	40	12.9	10.7 to 15.0
No	235	8.0	6.7 to 9.3
Home near gasoline station			
Yes	76	9.3	7.0 to 11.5
No	199	8.5	7.0 to 10.0
Vehicle in the home			
Yes	99	8.1	6.0 to 10.1
No	176	9.1	7.7 to 10.5
Gasoline storage at home			
Yes	112	8.6	6.2 to 10.9
No	163	8.8	7.5 to 10.1
Use of eye cosmetics			
None	82	9.3	7.2 to 11.4
Crushed lead ore	51	9.3	7.1 to 11.2
Pencil	24	6.4	4.0 to 8.7
Imported paste (Kajal)	101	8.8	7.5 to 10.0
Local paste	16	7.4	†
Ink ingestion (rubutu)			
Yes	112	8.8	7.2 to 10.5
No	163	8.6	7.2 to 10.1
Type of plates			
Metal	96	8.4	6.3 to 10.5
Plastic	19	10.8	6.6 to 15.0
Both	160	8.7	7.0 to 10.3

Table 1 continued

Characteristic	n	Mean blood lead (μg/dl)	95% CI
Type of cups			
Metal	11	12.2	8.5 to 15.9
Plastic	101	8.1	6.4 to 9.9
Both	163	8.8	7.1 to 10.5
Type of eating utensils			
Metal	111	8.8	7.0 to 10.7
Plastic	10	7.7	4.7 to 10.7
Both	145	8.9	7.1 to 10.7

*Some households used more than one container for water storage.
†Sample size insufficient to calculate confidence interval.

results were obtained, subjects were informed of their blood lead level. Those with levels 10 μg/dl or greater were advised to avoid potential sources of lead exposure, particularly eye cosmetics and battery melting. The Ethical Review Committee of the Jos University Teaching Hospital and the Institutional Review Board of the University of Utah approved this study.

The complex sample programs of Epi Info 3.01 (CDC, Atlanta, GA) were used in the data analysis to account for the cluster design. Confidence intervals of mean lead values were calculated using the ward as the stratification variable and the household as the primary sampling unit. Logarithmic transformation to normalise the moderately skewed distribution of lead values did not materially alter the results, so we did not use transformed lead values in the analysis, unless noted. Multiple linear regression with the TRANSREG procedure of SAS 6.12 (SAS Institute, Cary, NC) and logistic regression with Epi Info 3.01 were performed to identify variables independently predictive of blood lead values. For highly correlated variables, the variable explaining the most variance was retained in the model.

RESULTS

Of 283 subjects tested for blood lead, eight results could not be verified, possibly due to contamination. Mean blood lead was 8.7 μg/dl; 92 subjects (34%) had blood lead levels of 10 μg/dl or greater. Table 1 displays mean lead values with confidence intervals that account for the stratified, cluster design. As expected, subjects residing in the ''high lead'' ward had greater mean blood lead levels than those residing in the ''low lead'' ward, but the confidence intervals overlap.

Children aged 5 years and under had higher mean lead values (11.2 μg/dl) than other age groups, and 35 (55%) had blood lead levels of 10 μg/dl or greater. However, age itself was not a predictor of blood lead ($r = 0.03$). Pica in young children was not associated with increased blood lead values. Persons who did not use any pipe borne water had greater blood lead levels than those who did. Water storage in clay pots was a risk factor for increased lead values, whereas those who used oil barrels for water storage had lower lead values than those who did not. Those living in mud dwellings had significantly greater lead values than those in cement houses. Most homes with painted walls also had flaking paint. However, the greater lead values among those in painted dwellings were not statistically significant. Residence near a local battery smelter was a risk factor for increased blood lead levels. Although blood lead levels decreased the closer the home was to the nearest road ($r = 0.17$, $p = 0.005$), the number of vehicles per minute using the road was associated with an increase in blood lead levels ($r = 0.15$, $p = 0.01$).

All of the potential household level and individual risk factors for lead exposure were entered into a multiple linear regression model with blood lead concentration as the dependent variable. Variables were removed using a backwards stepwise elimination procedure until all remaining variables had significant ($p < 0.05$) regression coefficients (table 2). None of the variables in the final model were highly correlated (all r values <0.45). Water source and form of water storage, although significant in the univariate analysis, were no longer significant predictors of blood lead values in the multivariate model. Family vehicle ownership was the only factor significantly associated with lower blood lead values. An age of 5 years and under, flaking paint, residence near a battery smelter or a gasoline seller, male sex, parental education, and use of lead ore eye pencil were significantly related to increasing blood lead values. Persons residing in the ''high lead'' ward had greater blood lead values than those in the ''low lead'' ward, indicating that other unmeasured variables explain the difference in blood lead values between the two communities. The model including all of the variables in table 2 accounted for 38% of the total variance in blood lead values. The same variables, except

Table 2 Results of multivariate linear regression analysis of independent predictors of blood lead

Variable	B	95% CI	p value
Family owns a vehicle	−5.4	−7.0 to −3.8	0.0001
High lead ward	4.8	3.1 to 6.4	0.0001
Chipping paint	4.5	2.9 to 6.2	0.0001
Child 5 years and under	3.0	1.7 to 4.3	0.0001
Near gasoline seller	3.2	1.5 to 5.0	0.0003
Male gender	2.0	0.81 to 3.2	0.0009
Maternal education (per year)	0.28	0.11 to 0.45	0.001
Paternal education (per year)	0.24	0.08 to 0.39	0.0025
Use of lead ore eye cosmetic	1.8	0.27 to 3.4	0.02
Near battery smelter	2.1	0.13 to 4.2	0.035

What is already known on this topic

- Lead toxicity poses a significant risk to young children, but data regarding lead toxicity and sources of lead exposure in developing countries are sparse
- Low dietary calcium intake of children in many developing countries increases lead absorption and the risk of lead toxicity
- In Nigeria, greater blood lead values have been found in Muslim children, those living near areas where automobile batteries are melted, and in those using eye cosmetics

What this study adds

- This study confirms the previously identified association of lead containing eye cosmetics with greater blood lead values
- Children had higher blood lead values than adult family members, and chipping paint in the home, male sex, residence near a gasoline seller, and increasing parental education were positively associated with increasing blood lead concentration
- Family vehicle ownership was associated with reduced lead values, possibly because it reflects improved living conditions

residence near a battery smelter, remained significant predictors of blood lead concentration after logarithmic transformation of the lead values.

To control for the effect of household on individual level risk factors, we created dummy variables representing the 34 households studied. Individual risk factors that were significant predictors of blood lead values in this analysis were male sex, age of 5 years and under, pica, and ink ingestion. However, using a logarithmic transformation of lead values, pica no longer significantly predicted lead values.

A logistic regression analysis was performed using a blood lead value of 10 µg/dl or greater as the dependent variable. Except for maternal education and residence near a battery smelter, all significant predictors of blood lead levels in the multiple linear regression were also significant predictors of blood lead values ⩾10 µg/dl in the logistic regression.

DISCUSSION

We found multiple risk factors for lead toxicity in an urban area of central Nigeria. Blood lead levels of 10 µg/dl or greater were found in 34% of individuals of all ages, and children ages 5 years and under had the greatest risk of increased blood lead levels. Some factors associated with increased blood lead levels such as mud housing, clay pot water storage, and lack of pipe borne water may actually be proxy indicators of lower socioeconomic status, rather than risk factors themselves. A protective effect of higher socioeconomic status could account for the negative association of vehicle ownership with lead toxicity. Another explanation might be that those who do not own cars are exposed to fumes from vehicles using leaded fuel while walking along polluted roads. Unexpectedly, lead values increased with both paternal and maternal education. As observed in our previous study, we confirmed an association between lead ore eye cosmetics and increased blood lead levels. In Nigeria, powdered eye cosmetics are frequently placed around the eyelids of children, particularly among Muslims. There have been other reports of increased blood lead levels among children using lead containing eye cosmetics.[15 16] Presumably, lead ingestion occurs when the child rubs his eyes with his hands and then engages in hand-to-mouth behaviours rather than from conjunctival penetration.[17]

While government regulation has contributed to the decline in the prevalence of lead toxicity among children in developed nations, the problem in developing countries remains unacceptably high. Blood lead levels of 10 µg/dl or greater were found in 78% of young children in Johannesburg[18] and in 80% of Pakistani children,[19] figures similar to our previously reported prevalence of 70% in young Nigerian children.[13] Lower socioeconomic indicators were found to be risk factors for lead toxicity in both South Africa and Pakistan, and use of lead containing eye cosmetics was an important source of lead exposure in Pakistani children.

Despite examination of multiple risk factors, our model still failed to account for the majority of the observed variance of blood lead levels. Because the ward of residence remained predictive of blood lead values, other unidentified sources of lead exposure remain important contributors to lead toxicity in Nigerians. The observational design of the study makes it impossible to control for all potentially confounding variables. Interior and exterior dust have been identified as important sources of environmental lead,[9] but we did not attempt to measure the lead content of dust in this study.

Lead exposure, particularly in developing countries, further exacerbates the already compromised cognitive development of children due to malnutrition and infectious disease. We identified potentially remediable risk factors, including the use of lead containing eye cosmetics, chipping paint, and proximity to battery smelters and gasoline sellers. In addition to other health benefits, nutritional interventions to provide adequate calcium, iron, and vitamin C may potentially lower blood lead levels.[9] Identifying the sources of environmental health hazards in developing countries is an essential first step toward their remediation.

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REFERENCES

1 **Needleman HL**, Schell A, Bellinger D, *et al*. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *N Engl J Med* 1990;**322**:83–8.
2 **Canfield RL**, Henderson CR, Cory-Slechta DA, *et al*. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *N Engl J Med* 2003;**348**:1517–26.

3 **Sciarillo WG**, Alexander G, Farrell KP. Lead exposure and child behavior. *Am J Public Health* 1992;**82**:1356–60.
4 **Needleman HL**, Riess JA, Tobin MJ, *et al.* Bone lead levels and delinquent behavior. *JAMA* 1996;**275**:363–9.
5 **Selevan SG**, Rice DC, Hogan KA, *et al.* Blood lead concentration and delayed puberty in girls. *N Engl J Med* 2003;**348**:1527–36.
6 **Lin J-L**, Lin-Tan D-T, Hsu K-H, *et al.* Environmental lead exposure and progression of chronic renal diseases in patients without diabetes. *N Engl J Med* 2003;**348**:277–86.
7 **Moss ME**, Lanphear BP, Auinger P. Association of dental caries and blood lead levels. *JAMA* 1999;**281**:2294–8.
8 **Cheng Y**, Schwartz J, Sparrow D, *et al.* Bone lead and blood lead levels in relation to baseline blood pressure and the prospective development of hypertension: the Normative Aging Study. *Am J Epidemiol* 2001;**153**:164–71.
9 **Harvey B**, ed. *Managing elevated blood lead levels among young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention.* Atlanta, GA: Centers for Disease Control and Prevention, 2002.
10 **Thacher TD**, Fischer PR, Pettifor JM, *et al.* Case-control study of factors associated with nutritional rickets in Nigerian children. *J Pediatr* 2000;**137**:367–73.
11 **Pettifor JM**, Ross P, Moodley G, *et al.* Calcium deficiency in rural black children in South Africa: a comparison between rural and urban communities. *Am J Clin Nutr* 1979;**32**:2477–83.
12 **Bruening K**, Kemp FW, Simone N, *et al.* Dietary calcium intakes of urban children at risk of lead poisoning. *Environ Health Perspect* 1999;**107**:431–5.
13 **Pfitzner MA**, Thacher TD, Pettifor JM, *et al.* Prevalence of elevated blood lead in Nigerian children. *Ambulatory Child Health* 2000;**6**:115–23.
14 **Schlenker TL**, Fritz CJ, Mark D, *et al.* Screening for pediatric lead poisoning. *JAMA* 1994;**271**:1346–8.
15 **Sprinkle R**. Lead eye cosmetics: a cultural cause of elevated lead levels in children. *J Fam Pract* 1995;**40**:358–62.
16 **Al-Saleh I**, Nester M, DeVol E, *et al.* Determinants of blood lead levels in Saudi Arabian schoolgirls. *Int J Occup Environ Health* 1999;**5**:107–14.
17 **Parry C**, Eaton J. Kohl: a lead-hazardous eye makeup from the third world to the first world. *Environ Health Perspect* 1991;**94**:121–3.
18 **Mathee A**, von Schirnding YE, Levin J, *et al.* A survey of blood lead levels among young Johannesburg school children. *Environ Res* 2002;**90**:181–4.
19 **Rahbar MH**, White F, Agboatwalla M, *et al.* Factors associated with elevated blood lead concentrations in children in Karachi, Pakistan. *Bull World Health Organ* 2002;**80**:768.