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Prevalence of elevated blood lead levels in Nigerian children

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ABSTRACT

Objective To determine the prevalence and risk factors of elevated blood lead levels in young, urban Nigerian children.

Design A randomized cluster sample of children aged 6–35 months.

Setting Jos, a community in north central Nigeria.

Outcome measurement Blood lead level.

Results Of 218 children evaluated, 70% of the children had blood lead levels in excess of 10 μg/dL. Mean blood lead levels were 15.2 ± 1.4 μg/dL; median blood lead concentration was 12.0 (range 1– > 60 μg/dL). Mean lead concentrations were higher in those who professed the Islamic faith, used eye cosmetics, lived near a battery smelter, or lived in a certain geographical area. Pica was not associated with increased blood lead concentrations. Forward stepwise regression analysis revealed religion, area of residence, and proximity to a battery smelter as the variables which jointly predicted increased blood lead concentrations.

Conclusion A majority of the studied children in Jos, Nigeria have lead levels placing them at risk for intellectual impairment. The cause of lead intoxication appears to be multifactoral. Further studies should investigate the causality of these associations prior to the implementation of a primary preventive public health measure.

Keywords children, environmental health, lead toxicity, Nigeria

Introduction

Chronic exposure to lead has been associated with impaired cognitive development in children, which can persist into adulthood even at concentrations as low as 10 µg/dL.[1–4] Present American Academy of Pediatrics guidelines consider children with blood lead levels (BLLs) of 10 µg/dL or greater as having elevated BLLs and thus at risk for cognitive impairment and end organ damage.[5]

Children are at increased risk for lead toxicity due to their small size, their rapidly developing brain, and their behaviour. Compared with adults, the small body size of children results in higher blood concentrations for a given quantity of lead ingested. Additionally, lead is more neurotoxic to the developing brains of children, and hand-to-mouth behaviour facilitates the entry of lead into their bodies.[6]

> This study associates multiple factors with lead toxicity in Nigerian children: use of eye cosmetics, religion, proximity to a battery smelter, dwelling, and geographical area.

Little is known about the prevalence of lead toxicity in children or the sources of contamination in developing countries. Because the burden of malnutrition is much greater in developing countries, concurrent mineral deficiencies of calcium, iron and zinc, resulting in preferential lead absorption, may increase the risk of lead toxicity.[7] In Africa, most published studies have examined urban populations in South Africa. Mean BLLs in Cape children ranged from 8.2 to 12.7 µg/dL (range 2–28 µg/dL) in the three separate groups studied.[8] In another study, 13% of inner city children in South Africa had BLLs greater than 25 µg/dL.[9] However, in unpolluted rural areas in South Africa, researchers found a mean BLL of 3.4 µg/dL (range 0.5–7.5).[10] Among a convenience sample of children from affluent parents in Kaduna, Nigeria, researchers found a mean BLL of 10.6 µg/dL with 2% of children between 1 and 6 years having concentrations greater than 30 µg/dL.[11]

In view of lead's toxic potential, and the current dearth of medical literature describing the prevalence of lead toxicity in developing countries, this study was conducted to determine the prevalence and risk factors of elevated blood lead levels in young urban Nigerian children.

Methods

During a 4-week period in March/April of 1997, we conducted a community cross-sectional survey in Jos, Nigeria. Jos sits on a plateau in north-central Nigeria and has a tropical climate. The time of the study was near the end of the dry season, with the air being dusty except for a few rainy days during the study period. In the census year 1991, the area had a total population of 360 100 with children less than five years of age comprising 22% of the population. In 1995, the ratio of Christians to Muslims was 2:1.

Children aged between 6 and 35 months were targeted and were selected by means of a randomized cluster sample for this cross-sectional study.[12] Clusters for the study were the 12 administrative wards of Jos. From each ward, three streets were randomly selected. Beginning in the approximate centre of the street with direction determined by coin flip, we approached each place of residence to ask if any children were present in the age range of six to 35 months. If they were, verbal, informed consent for the child's participation was obtained. If children in this age range were not found, we continued to the next home until six children per street had been sampled. No more than two children per household were sampled. A household was defined as people who shared common sleeping quarters and food sources.

The community study team consisted of two faculty paediatricians and local medical doctors in training. Each child's guardian was interviewed by one of the doctors in training using a structured questionnaire. Demographic information, presumed risk factors for lead intoxication, a medical history and a brief physical exam were recorded. Any child with abnormal physical findings or significant symptoms was referred to Jos University Teaching Hospital for evaluation. Blood was obtained via venipuncture; the skin was thoroughly cleaned and the blood used in the testing was that which was left in the butterfly tubing after the other samples were drawn. Two discrete areas of blood were placed on a filter paper taken from an individually sealed bag. The blood was briefly air-dried, then placed into the same bag to prevent contamination. Additionally, blood was placed in two heparinized capillary tubes for haematocrit determination. As a token of appreciation, parents of participating children over 1 years of age were provided with oral mebendazole, a medication commonly used in the community for periodic de-worming. Parents of participating children less than 1 years of age were given a small package of children's toiletries.

At the end of the study, the filter papers for lead analysis were transported to the LeadTech™ Corporation (North Bergen, NJ, USA) for analysis by atomic absorption spectrophotometry.[13,14] Any sample with a value greater than 10 μg/dL was repeated on the other area of blood on that filter paper, as well as having a non-bloodstained portion of the filter paper analysed to determine if contamination with lead had occurred. If the results of the second test were within 4 μg/dL of the first result, the results were averaged. No samples with values greater than 10 μg/dL had a variation of greater than 4 μg/dL between samples. This method is equivalent to traditional whole blood lead measurements.[15] This assay can measure BLLs up to and including 60 μg/dL; greater values are reported as > 60 μg/dL. For statistical calculations, 60 μg/dL was used for the one child with a BLL > 60 μg/dL.

Eye cosmetic and soil samples were analysed for lead content by the American Environmental Consultants Laboratory (Salt Lake City, Utah, USA) via flame atomic absorption using EPA SW 846, method 3050.

We entered data on Epi Info (v.6.04b, Centers for Disease Control, USA) statistical software. Additional analysis was performed using Statview for Windows Version 5 (SAS Institute). Means of normally distributed variables were compared using two sample *t*-tests for independent samples. ANOVA was used for the comparison of nominal data with the continuous dependent variable, BLL. Stepwise regression analysis was performed to develop a model to predict BLLs due to the multicollinearity of the variables. Findings were considered significant for values of $p < 0.05$.

Before the study, approval was obtained from local government officials. Additionally, the study was approved by the Institutional Review Board of the University of Utah and the Ethical Research Committee of the Jos University Teaching Hospital.

Results

The parents/guardians of 238 children were asked to participate in the study. Fifteen parents refused. Four children were excluded because a blood sample could not be readily obtained, and one child was later found to be older than 36 months. The remaining 218 were enrolled.

The families studied represented 48 different ethnic groups, with Hausa ($n = 50$, 23%), Ibo ($n = 41$, 19%), and Yoruba ($n = 24$, 11%) being the three most common tribes represented. The informant was the mother in 70% of cases ($n = 152$), the father in 15.4% ($n = 34$), and an immediate family member in the remainder of the contacts. One hundred and thirty-six (63%) described themselves as Christian, with the remainder being Muslim. The average household size was 6.4 individuals, and the average number of children under 15 years of age per household was 3.8. The average age of the study children was 22 months; 55% were female.

Mean BLL in the population was 15.2 ± 1.4 μg/dL (1–> 60 μg/dL). No filter paper contamination was found. The distribution was skewed (Figure 1); the median lead value was 12.0 μg/dL. One child had a measured BLL of > 60 μg/dL. Seventy percent of the study population had BLLs > 10 μg/dL, and 18% had BLLs > 20 μg/dL. Of the other studied characteristics, area of residence (cluster), religion, use of eye cosmetics, residence near a battery smelter, type of housing, apartment floor and a history of remodelling were significantly related to increased BLLs (Table 1). Smelting of lead batteries was observed near the residences of six children in cluster 1, and two children in cluster 10. Those professing the Islamic faith were more likely to have a BLL greater than 10 μg/dL (OR 4.0, CI 1.94–8.26). Both male and female children used eye cosmetics, but the practice was significantly more common among Muslims than Christians (80% vs. 15%, respectively, $p < 0.0001$). Those children reported as using eye cosmetics were more likely to

Table 1: Mean blood lead level by the studied characteristics. *p*-values > 0.05 are not considered statistically significant (NS)

Characteristic (*n*)	Mean BLL (µg/dL) concentration (SEM)	*p*-value
Cluster		< 0.0001
1 (18)	36.7 (3.1)	
2 (19)	16.6 (2.3)	
3 (18)	10.3 (1.6)	
4 (18)	9.1 (0.7)	
5 (18)	14.8 (1.3)	
6 (19)	19.1 (2.9)	
7 (18)	11.6 (1.1)	
8 (18)	12.1 (1.3)	
9 (18)	15.4 (1.4)	
10 (18)	11.1 (1.1)	
11 (18)	13.3 (1.7)	
12 (18)	11.7 (0.8)	
Sex		
Male (97)	15.4 (1.0)	NS
Female (121)	15.0 (1.0)	
Ability to walk		
Yes (176)	14.5 (0.7)	NS
No (40)	16.3 (1.7)	
Breast fed		
Yes (61)	16.8 (1.6)	NS
No (157)	14.5 (0.7)	
Religion		
Islam (80)	21.0 (1.5)	< 0.0001
Christian (136)	11.8 (5.3)	
Eye cosmetic use		
Yes (87)	19.4 (0.6)	< 0.0001
No (131)	12.3 (1.4)	
History of pica		
Yes (119)	14.3 (0.9)	NS
No (99)	16.2 (1.1)	
Water supply		
Tap (176)	16.1 (0.8)	
Stream (1)	6.0 (–)	
Tap and well (23)	11.2 (0.8)	NS
Reservoir and well (6)	9.5 (2.2)	
Well (12)	12.9 (1.6)	
Water storage		
Plastic (135)	15.2 (0.8)	
Clay pot (49)	16.6 (1.8)	
Metallic container (15)	11.3 (1.3)	NS
Plastic and clay pot (12)	12.1 (1.6)	
Plastic and metallic (7)	17.1 (1.7)	
Residence near battery smelter		
Yes (8)	35.7 (7.3)	< 0.0001
No (210)	14.4 (0.6)	
Housing type		
Open air courtyard (187)	16.0 (0.8)	
Multilevel apartment (19)	9.4 (0.7)	0.2
Row houses (12)	12.1 (1.9)	
Apartment floor		
Ground (8)	11.9 (1.0)	
First (8)	8.0 (0.9)	0.008
Second (3)	6.7 (0.3)	
History of remodelling		
Yes (145)	16.2 (0.9)	0.04
No (71)	13.2 (1.0)	
Chipping paint present in home		
Yes (59)	13.5 (1.1)	NS
No (158)	15.9 (0.9)	
Type of road near housing		
Paved, traffic (44)	16.8 (1.5)	
Dirt/paved, some traffic (26)	11.9 (1.2)	NS
Dirt, occasional traffic (104)	14.6 (1.0)	
Dirt, no traffic (44)	11.7 (1.8)	

have a BLL greater than 10 µg/dL (OR 2.4, CI 1.26–4.51). Despite those with a positive history of remodelling having a higher BLL than those who did not ($p = 0.04$), the BLL did not vary by time since remodelling ($p = 0.14$). The mean time since remodelling was 5.7 ± 0.8 years, which is much greater than the mean age of children in the study (22 months). When analysed for recent remodelling (< 12 months), the BLL was not significantly higher between the groups ($p = 0.67$). Mean BLLs did not vary either by paternal or maternal occupation ($p = 0.21$, 0.16, respectively).

The mean haematocrit was 37.1 ± 0.6% ($n = 192$; 26 samples were lost due to handling errors) and this did not vary by BLL ($p = 0.75$). Only 12 individuals had haematocrits ≤ 30% (6.3%); BLLs in these individuals were not significantly different than those with higher haematocrits ($p = 0.47$).

Forward stepwise regression was performed with the following variables: religion, age, sex, breastfeeding, ability to walk, use of eye cosmetics, history of pica, history of home remodelling, type of dwelling, presence of chipping paint in the home, residence near a battery smelter, and residence in cluster 1. In Step 1, religion was placed in the model ($R^2 = 0.195$, $F = 50.8$); residence in cluster 1 was added in Step 2 ($R^2 = 0.275$, $F = 22.9$); and presence of a battery smelter near the primary residence was added in

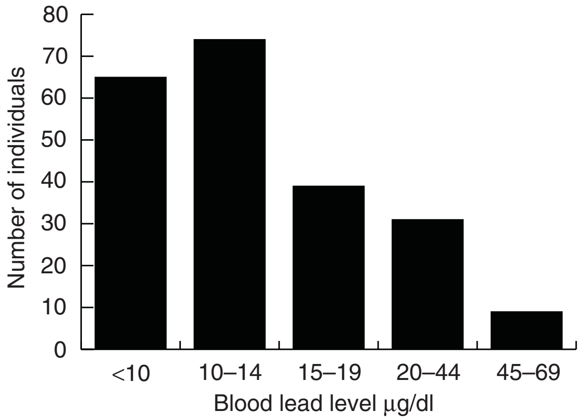

Figure 1: Frequency of blood lead concentrations in the study population as categorized by the American Academy of Paediatrics guidelines. Any value above 10 µg/dL is considered elevated.[5]

Step 3 (R^2 = 0.318, F = 13.3). These three variables predicted 32% of the variability in the BLL.

In an attempt to explain why cluster 1 might have had such a higher mean BLL, two roadside soil samples were collected from only that cluster. The two samples contained 71 and 809 µg of lead per gm of soil. Additionally, samples of eye cosmetics used by eight children from cluster 1 with the highest BLL (at least 45 µg/dL) were obtained. The samples consisted of either a thick black paste in a metallic tube or a black powdered material. Six samples of the paste type were tested for lead; they averaged 0.11% lead (range 0.01–0.24%). Two samples were powdered; they were 43.8 and 68.8% lead, respectively.

Discussion

Environmental sources of lead can include chipping lead-based paint, ingestion of soil containing lead, and inhalation of aerosolized lead dust from the combustion of lead-based fuels.[16] Parental occupations such as mining may bring lead dust into the house. Reports from the Middle East and the United States have implicated kohl or surma, lead-based eye cosmetics used by people of certain religions, in lead toxicity.[17–20] Exposure is thought to be due to children rubbing their eyes, placing their hands in their mouths, and ingesting the lead rather than from direct conjunctival absorption.[21]

Seventy per cent of children in this study had lead concentrations >10 µg/dL placing them at risk for cognitive impairment. This represents a significant health problem in this population not only for the present generation, but for future generations as well.

No single factor was responsible for all lead toxicity in this population. This study associates multiple factors with lead toxicity in Nigerian children: use of eye cosmetics, religion, proximity to a battery smelter, dwelling, and geographical area. It is unclear how religion influences BLLs except through the area of residence, proximity to a battery smelter, or the use of eye cosmetics. Yet the use of eye cosmetics was not associated with lead toxicity in the regression model, even though its use is thoroughly connected to the Islamic faith, which was significant in the regression model. More importantly, most of these factors could potentially be modified, either through community education or government action and regulation.

Clearly the Islamic custom of using eye cosmetics, which may contain lead, provides one mechanism by which children can ingest lead. Lead ore (galena) is sold for crushing into a fine powder and used as an eye cosmetic. This lead ore is not imported, but found and sold locally by traditional vendors, and is thus not easily regulated. Healy, in a review of lead-containing traditional medicines in Nigeria, describes one form of this eye cosmetic as a powdered mixture of ground lead ore and carbon.[22] It is typically applied with a finger three to four times per day from about 4 months of age on, regardless of gender, about the eyes in the manner of eyeliner until a dark stain is formed. The two powdered samples we obtained were predominately lead. The paste type of this cosmetic is virtually lead-free. Although it is more costly than the powdered type, anecdotal evidence from one of the local authors reveals that this does not seem to be an impediment to its use. Since lead-free alternatives are available, educating the public regarding these materials could be done without disrupting a custom that is clearly important to this religious group.

Aerosolization of lead, through lead-containing dust or automotive exhaust, represents another source of exposure. The housing data in which children living in ground floor apartments had higher BLLs than those living in upper floor apartments clearly suggests particulate transmission, either by direct tracking of contaminated soil into the dwelling or through roadside dust from vehicular and pedestrian traffic. Occupations that place workers in areas with a large amount of automotive exhausts have been shown to increase BLLs in those workers; for example, traffic officers in Cairo had mean BLLs of 29 μg/dL compared to controls of 18 μg/dL.[23] The *Harmattan* (the dusty winds that blow over parts of sub-Saharan Africa during the dry season, when particulate matter in the atmosphere quintuples compared to the rainy season) increases the lead particulate matter in the air. This study was carried out at the end of the dry season; thus lead in the air possibly played a greater role in the prevalence of lead toxicity than it would during the rainy season.

Contaminated food and water can represent another source of lead exposure. This study did find an association with the water supply. Those using tap water had higher mean BLLs; yet as most of the families used tap or a mixture of tap and another source, the water supply cannot be indicted until formal testing has been done. In Nigeria, contaminated food, including plant materials, canned goods, and dried fish have all been shown to be lead-contaminated.[24–26] These were not investigated in this study.

Malnutrition may also contribute to lead intoxication. Deficiencies of both calcium and iron enhance lead absorption.[7] One marker for iron deficiency is anaemia, but only a few children in the study population were anaemic, and those children were no more likely to be lead toxic than those who were not anaemic. The diet in Nigeria is lacking in calcium,[27] and children may be preferentially absorbing another divalent cation—lead—in lieu of calcium.[28] Lead has been found in breast milk in Nigeria.[29]

A more thorough analysis of the factors associated with lead toxicity needs to be performed to assess religious practices and geographical areas for additional sources of lead contamination. Intervention needs to be divided between education, community/government involvement, and treatment through chelation. Education as a primary, preventive public health measure would be the easiest to implement and the least likely to cause political resistance. Eventually, dangerous practices must be discontinued and environmental sources removed. Lead screening is not available to these children at present; thus education is the only alternative.

Clearly there are limitations to this study. The results may not be completely generalizable to paediatric populations in rural parts of Nigeria or in other developing countries. Relevant environmental factors such as proximity to a lead smelter were based upon a superficial visual inspection; hazards not readily apparent would be missed. The sampling may be biased toward a less affluent population. The more affluent neighbourhoods were much more difficult to sample, as parents were typically at work and no one was available to give consent.

Implications for practice

Though malnutrition and infectious disease represent the most significant health problems for children in the developing world, environmental hazards are understudied. Because environmental hazards are often invisible and their symptomatology non-specific, they have largely been ignored. Children in developing countries may not reach their intellectual potential due to the presence of lead, resulting in a 'dumbing down' of the population. However, future generations need not be affected by environmental hazard if efforts are made now to correct the problem. Further work is necessary to better identify the exact causes of lead toxicity in Nigeria and to develop methods to decrease the lead burden currently imposed on many young children.

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References

1 Baghurst P A, McMichael A J, Wigg N R, Vimpani G V, Robertson E F, Roberts R J and Tong S (1992) Environmental exposure to lead and children's intelligence at the age of seven years: the Port Pirie cohort study. *New England Journal of Medicine*, **327:** 1279–1284.

2 Tong S, Baghurst P, McMichael A, Sawyer M and Mudge J (1996) Lifetime exposure to environmental lead and children's intelligence at 11–13 years: the Port Pirie cohort study. *British Medical Journal*, **312:** 1569–1575.

3 Needleman H L, Schell A, Bellinger D, Leviton A and Allred E N (1990) The long-term effects of exposure to low doses of lead in childhood. *New England Journal of Medicine*, **322:** 83–88.

4 Mendelsohn A L, Dreyer B P, Fierman A H, Rosen C M, Legano L A, Kruger H A, Lim S W *et al.* (1998) Low-level lead exposure and behavior in early childhood *Pediatrics*, **101** (3). URL:
http://www.pediatrics.org/cgi/content/full/101/3/e10

5 American Academy of Pediatrics Committee on Environmental Health (1998) Screening for elevated blood lead levels. *Pediatrics*, **101** (6)**:** 1072–1078.

6 Lin-Fu J S (1973) Vulnerability of children to lead exposure and toxicity. *New England Journal of Medicine*, **289** (24)**:** 1289–1293.

7 Goyer R A (1993) Lead toxicity: current concerns. *Environmental Health Perspectives*, **100:** 177–187.

8 White I W, Dempster W S, Pocock F and Kibel M A (1982) Lead absorption in Cape Children. *South African Medical Journal*, **62:** 799–802.

9 Von Schirnding Y, Bradshaw D, Fuggle R and Stokol M (1991) Blood lead levels in South African Inner-city children. *Environmental Health Perspectives*, **94:** 125–130.

10 Grobler S R, Rossouw R J and Maresky L S (1985) Blood lead levels in a remote, unpolluted rural area in South Africa. *South African Medicine Journal*, **68:** 323–324.

11 Nriagu J, Oleru N T, Cudjoe C and Chine A (1997) Lead poisoning of children in Africa, III Kaduna, Nigeria. *The Science and the Total Environment*, **197:** 13–19.

12 Henderson R H and Sundaresan T (1982) Cluster sampling to assess immunization coverage: a review of experience with a simplified method. *Bulletin of the World Health Organisation*, **60** (2)**:** 253–260.

13 Verebey K, Eng Y M, Davidow B and Ramon A (1991) Rapid sensitive micro blood lead analysis: a mass screening technique for lead. *Poisoning Journal of Analytical Toxicology*, **15:** 237–240.

14 Verebey K, Rosen J F, Schonfeld D J, Carriero D, Eng Y M, Deutsch J, Reimer S *et al.* (1995) Blood collection and analytical considerations in blood lead screening in children. *Clinical Chemistry*, **41** (3)**:** 469–470.

15 Schlenker T L, Fritz C J, Mark D, Layde M, Linke G, Murphy A and Matte T (1994) Screening for pediatric lead poisoning: comparability of simultaneously drawn capillary and venous blood samples. *Journal of the American Medical Association*, **271** (17)**:** 1346–1348.

16 Roper W L, Houk V N, Falk H and Binder S (1991) *Preventing lead poisoning in young children*. Centers for Disease Control, Washington D.C., October.

17 Parry C and Eaton J (1991) Kohl: a lead-hazardous eye makeup from the third world to the first world. *Environmental Health Perspectives*, **94:** 121–123.

18 Alkhawajah A M (1992) Alcohol use in Saudi Arabia: extent of use and possible lead toxicity. *Tropical Geographical Medicine*, **44** (4)**:** 373–377.

19 Nir A, Tamir A, Zelnik N and Iancu T C (1992) Is eye cosmetic a source of lead poisoning? *Israeli Journal of Medical Science*, **28** (7)**:** 417–421.

20 Sprinkle R V (1995) Leaded eye cosmetics: a cultural cause of elevated lead levels in children. *Journal of Family Practice*, **40** (4)**:** 358–362.

21 Smart A and Madan N (1990) Surma: a cause for concern. *Health Visitor*, **63** (11)**:** 379–380.

22 Healy M A, Aslam M and Bamboye O A (1984) Traditional medicines and lead-containing preparations in Nigeria. *Public Health*, **98:** 26–32.

23 Kamal A A M, Eldamaty S E and Faris R (1991) Blood lead level of Cairo traffic policeman. *The Science of the Total Environment*, **105:** 165–170.

24 Nriagu J O (1992) Toxic metal pollution in Africa. *The Science of the Total Environment*, **121:** 1–37.

25 Onasanya L O and Ajewole K (1993) Lead content in roadside vegetation as indicators of atmospheric pollution. *Environment International*, **19:** 615–618.

26 Okoye C O B (1994) Lead and other metals in dried fish from Nigerian markets. *Bulletin of Environmental Contamination and Toxicology*, **52:** 825–832.

27 Thacher T, Ighogboja S I and Fischer P R (1997) Rickets without vitamin D deficiency in Nigerian children. *Ambulatory Child Health*, **3:** 56–64.

28 Pounds J G, Long G J and Rosen J F (1991) Cellular and molecular toxicity of lead in bone. *Environmental Health Perspectives*, **91:** 17–32.

29 Parr R M, DeMaeyer E M, Iyengar V G, Byrne A R, Kirkbright G F, Schoch G, Niinisto L *et al.* (1991) Minor and trace elements in human milk from Guatamala, Hungary, Nigeria, Phillipines, Sweden and Zaire. *Biological Trace Element Research*, **29:** 51–75.

Biosketches

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Commentary

Elevated blood lead is an important cause of intellectual impairment. Data from less developed countries based on methodologically sound studies are sparse. This well designed and executed study is an important contribution to the literature, raising concerns about environmental and other factors that represent further potential threats to the already compromised health of children in less developed countries.

The authors have used a cluster sampling methodology, which is ideally suited to research in urban settings in less developed countries. There was a high participant rate in the study. However, selection bias may have been increased by under-representation of more affluent families. This was due to nonavailability of main carers to give consent. In addition, it is likely that the urban population studied would have higher levels of blood lead levels than rural children due to increased traffic fumes and environmental pollution associated with industry. For these reasons, the reported prevalence of these elevated levels is likely to be an overestimate of the levels in the whole population. The extent of the overestimate is difficult to judge, as the authors present no socio-economic data for children studied and give no indication of the extent of failed data collection in more affluent areas. Nonetheless, the reported prevalence rate suggests that urban children living in poorer areas are at high risk of elevated blood lead levels, with potentially serious consequences for their intellectual potential.

The finding that elevation of lead level is associated with more than one factor is unsurprising. The apparent importance of religion, however, is difficult to explain, as the authors acknowledge. A possible explanation could be confounding by socio-economic status. It is possible that Christian religion is associated with higher socio-economic status. The absence of socio-economic data on individual families makes it difficult to test this explanation. The lack of an independent association of eye cosmetic use with elevated blood lead levels after regression analysis does not rule out a causal role. Again, it is possible that the relationship between religion and eye cosmetic use is confounded by maternal education and socio-economic status, and the failure to measure these variables limits the value of the regression models.

These limitations notwithstanding, this is a very valuable study that highlights a further potential threat to the well-being of children in poor urban areas of less developed countries whose health and educational potential are already seriously compromised by their increased risk of malnutrition and other exposures associated with adverse health outcomes.

Biosketch: Nick Spencer is Professor of Child Health at the School of Postgraduate Medical Education, University of Warwick and Consultant Community Paediatrician in the Coventry Healthcare NHS Trust. His research interests are poverty and child health and the global determinants of child health. He is editor of the *Child Health across the Globe* section of the journal.