



## Blood lead levels and potential environmental exposures among children under five years in Kibera slums, Nairobi

Tom M. Olewe<sup>1\*</sup>; Mutuku A. Mwanthi<sup>2</sup>; Joseph K. Wang'ombe<sup>2</sup> and Jeffrey K. Griffiths<sup>3</sup>

<sup>1</sup> Vision Integrity & Passion to Serve (VIPS) Health Services at Woodley, P.O. BOX 8238 – 00200, Nairobi, Kenya

<sup>2</sup> Department of Community Health, School of Medicine, University of Nairobi, Kenya

<sup>3</sup> Department of Public Health and Family Medicine, Tufts University, Boston, MA, USA

\*Corresponding author email: [tolewe@vipkenya.or.ke](mailto:tolewe@vipkenya.or.ke) ; Published online on January 5, 2009.

### ABSTRACT

**Objective:** Lead exposure has been associated with intellectual impairment in children in a number of international studies. Prevalence of elevated blood lead levels (eBLL  $\geq$  10ug/dL) of between 5 – 15% has been reported among children under 12 year old in Nairobi (UNEP, 2006). However, little is known about potential environmental exposure for eBLLs among children living in Kibera slums within Nairobi City, Kenya.

**Methodology and results:** A descriptive, cross-sectional study of children drawn from Kibera slums who presented at Yes to Kids (Y2K) program of VIPS Health Services at Woodley, Nairobi between June and August 2007 was carried out. The study assessed potential correlates of eBLLs in 387 children aged 6 to 59 months and had lived in Kibera slums since birth. Sampling was purposive. The factors examined were age, sex, breastfeeding history, respondent's education and occupation, type of house walls, sources of drinking water and kales, and awareness of lead poisoning among respondents. Potential risk factors such as exposure to paint, contaminated playgrounds, glazed pottery, cosmetics and para-occupational as well as living near lead industries and pica behavior were also examined. Potential environmental sources of lead such as drinking water, soil and kales were analyzed for lead levels. Seven percent (n = 27, N = 387) had BLLs above 10ug/dl. BLL  $\geq$  10ug/dl was associated with non-permanent housing (p = 0.812), playing on potentially lead contaminated grounds (p = 0.627) and pica behavior (p = 0.439). Low risk parental occupation (p = 0.001) and kales sourced from the market/kiosks (p = 0.001) were significantly associated with BLL  $\geq$  10ug/dl. Soil lead levels ranged from 3,000 to 90,000ug/kg, which was very high compared to WHO acceptable range of 100 – 200ug/kg. There was weak linear association ( $r^2 = 0.0160$ ) between soil Pb and mean BLLs for a given village. There were no detectable levels of lead in kales and tap water.

**Conclusion and application of findings:** The study found about 7% (N = 387) of the children tested had eBLL  $\geq$  10ug/dl in an area with very high soil lead levels (range in Kibera slums: 3,365 – 89,570 ug/kg compared to WHO allowable range: 100 – 120ug/kg). This finding raises a health concern that needs to be addressed through a multi-sectoral approach. Further studies are required since the study design and its inherent limitations could have masked the true picture of childhood lead poisoning in Kibera slums.

**Key words:** Blood lead levels, environmental exposure, GFAAS, LeadCare II analyzer

Citation: Olewe TM, Mwanthi MA, Wang'ombe JK. and Griffiths JK, 2009. Blood lead levels and potential environmental exposures among children under five years in Kibera slums, Nairobi. *Journal of Applied Biosciences* 13: 688 - 694.

## INTRODUCTION

Lead is a heavy metal that is mainly introduced to the environment through human activities. Excessive lead exposure through air, water, soil or food is harmful to the health and intellectual development of millions of children (Markowitz *et al.*, 2000). Widespread and potentially excessive lead exposure is evident in Nairobi. Mutuku (2006) reported lead levels in kales, maize, tap water and soil at 5,053ug/kg, 1,948ug/kg, 5.5ug/l and

44,350ug/kg, respectively. These were higher than the acceptable WHO lead levels in kales, maize, tap water and soil limited to 300ug/kg, 200ug/kg, 10ug/l and 100 – 120ug/kg, respectively. Based on these previous findings, the objective of the current study was to determine the impact of such high environmental lead levels on blood lead levels in children.

## MATERIALS AND METHODS

**Methods:** Ethics and Research Committee approval was granted to conduct the study at Yes to Kids (Y2K) program, Vision Integrity & Passion to Serve (VIPS) health services at Woodley, Nairobi. The study was conducted between April and August 2007 and targeted children drawn from Kibera slums, approximately 7km South-western of Nairobi City centre. Kibera is an informal settlement that had a population of 159,083 male and 127,656 female children aged between 6 – 59 months in 1999 (Central Bureau of Statistics, 1999). It is heavily polluted, and has no infrastructure.

**Sampling and sample treatment:** The study purposively sampled 387 children from Kibera slums aged 6 – 59 months, who presented at the clinic. With parental consent, they were carefully screened by medical doctors for inclusion based on preset criteria. Pre-tested, coded and close-ended questionnaires were introduced by trained interviewers to children's surrogates and data on socio-demographic profiles, and residence characteristics, as well as potential risk factors for exposure to lead were documented.

**Blood samples:** Trained laboratory technologists collected capillary blood from the children using protocols described by Schonfeld *et al.* (1995). Capillary tubes, 50ul each, provided with LeadCare II blood lead analyzer kits were used to collect 387 blood samples, following instructor's protocols. Out of the 387 samples, 22 were collected in duplicates for analysis with both LeadCare II and Graphite furnace - Atomic Absorption Spectrometer (GFAAS). Capillary blood for analysis with GFAAS was collected using 50ul Sarstedt microvette CB300 tubes with EDTA anticoagulant. The tubes were filled to ¾ and standardized analytical methods (Flajnik *et al.*, 1994) were followed at the Massachusetts Public State Laboratory in Boston, USA.

**Environmental samples:** Pooled samples from selected potential sources of exposure (water, soil, kales) were purposively collected from the children's villages of residence since birth. Samples of water, soil and kales were collected according to the procedures of Jumba *et al.* (1995a, 1996a) and the Community Environmental Health Resource Center ([www.cehrc.org](http://www.cehrc.org), 2006). Samples were digested and extracted according to the procedures of Jumba *et al.* (2007). Lead levels were analyzed by flame atomic absorption spectrophotometer, using prescribed Shimadzu AA6300 standardized analytical methods (Shimadzu, 2002) at the University of Nairobi.

**Quality control:** The questionnaires were pre-tested and interviewers trained prior to commencement of research. Duplicate blood samples were collected for quality control comparing results obtained with LeadCare II against the gold standard, GFAAS. Certified controls and standards were used for both blood and environmental samples lead analyses.

**Data analysis:** Data management and analysis were done using Statistical Package for Social Sciences (SPSS) software, version 10. The Centre for Disease Control (CDC, 2001) permissible childhood blood lead levels of 10µg/dL was used as a cutoff point in the analysis. The socio-demographic characteristics, residence characteristics and potential risk factors for exposure to lead among children with blood lead levels  $\geq 10 \mu\text{g/dL}$  were compared to those of children with blood lead levels  $<10 \mu\text{g/dL}$ . Chi-square test for independence, Spearman's correlation, Eta correlation and analysis of variance (ANOVA) were used to determine measures of association and statistical significance at 5% level.

## RESULTS AND DISCUSSION

Three hundred and eighty seven (n = 387) children participated in the study, comprising of 52.8 and 47.2% boys and girls, respectively. Mean blood lead level (BLL) was 5.997ug/dL (median = 5.40ug/dL, SD=2.42, range 3.30 – 24.70ug/dL) (figure 1). There were 27 (7%, n = 386) children that had BLL  $\geq$  10 ug/dL. Low risk parental occupation (OR = 14.28; 95% CI: 3.05 – 66.75; p = 0.001) (Table 1) was significantly associated with BLL  $\geq$  10ug/dL among the children. The questionnaire was determined not to be the best surrogate for occupational lead exposure, implying that children classified as low risk for lead exposure could have been high risk.

Kales sourced from the market/kiosks (OR = 14.24; 95% CI: 3.05 - 66.72; p = 0.001) (Table 1) were significantly associated with BLL  $\geq$  10ug/dL yet concentration of lead in analyzed kales were below detectable levels. It is possible that the kales analyzed for lead concentration were from sources different from those ingested by the children in the study. There was

weak and statistically insignificant association between BLL  $\geq$  10 ug/dL and non-permanent housing ( $X^2 = 0.0565$ , df = 1, p = 0.812), playing on potentially lead contaminated grounds (OR = 0.89; 95%CI: 0.25 – 2.34; p = 0.627) and pica behavior (OR = 0.72; 95%CI: 0.31 – 1.68; p = 0.439) (Table 2).

Soil lead levels (Soil Pb) ranged from 3,000 to 90,000ug/kg, which was very high compared to WHO acceptable range of 100 – 200ug/kg (Table 3). However, there was weak linear association ( $r^2 = 0.0160$ ) between Soil Pb and mean BLL for a given village. Soil Pb level could therefore not be used as a predictor of mean BLL for children in a given village. This could be attributed to inherent limitations of convenient sampling and the fact that the study did not ensure proportionate distribution of children per village. There were no detectable levels of lead in kales and tap water.

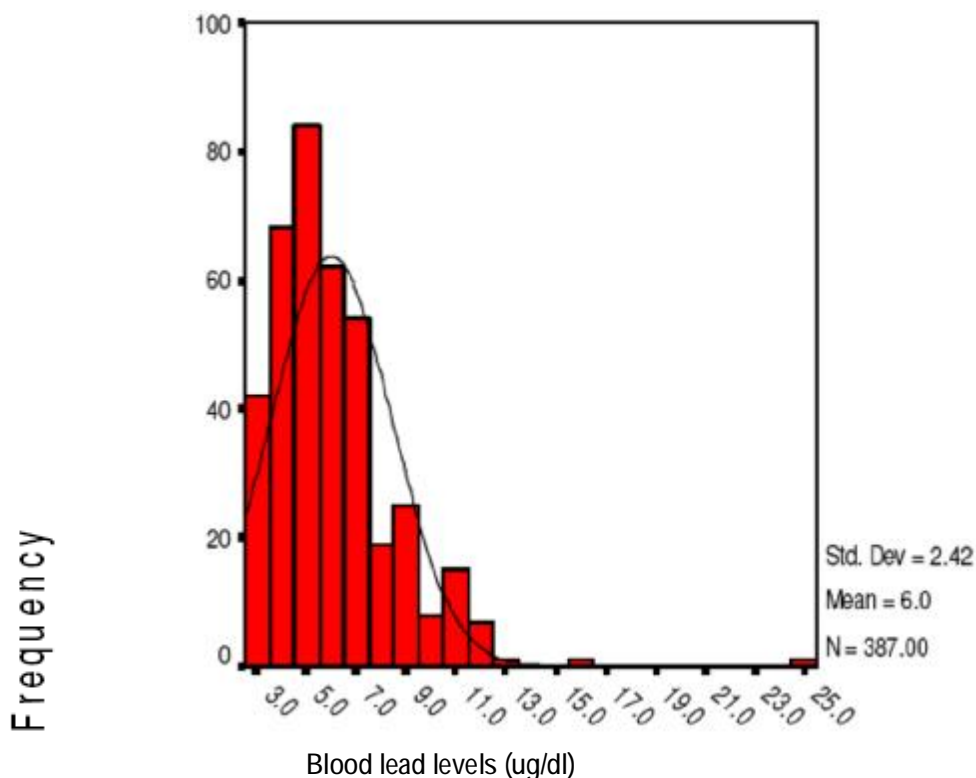


Figure 1: Frequency of blood lead levels among 6 – 59 months old children in Kibera slums, Nairobi, Kenya.



Table 1: Socio-demographic characteristics of children by blood lead concentration (n = 387) in Kibera slums in Nairobi, Kenya.

		Blood Lead Concentration (ug/dL)			
		< 10 (n = 359) n(%)	≥ 10 (n = 28) n (%)	Total (n = 387) n (%)	p value
Mean BLL (Range)		5.54 ( 3.3 - 9.8)	11.85 ( 10.0 - 24.7)	6.00 (3.3 - 24.7)	
Age ( months)	6 to 9	68 (18.9)	3 (11.1)	71 (18.4)	
	10 to 19	102 (28.4)	7 (25.9)	109 (28.2)	
	20 to 29	53 (14.5)	4 (14.8)	56 (14.5)	
	30 to 39	41 (11.4)	7 (25.9)	48 (12.4)	
	40 to 49	45 (12.5)	4 (14.8)	49 (12.7)	
	50 to 59	50 ( 13.9)	2 ( 7.4)	52 (13.5)	
	Missing	1 (0.3)	0	1 (0.3)	***0.339
Child's Sex	Male	169 (47.1)	13 (48.1)	182 (47.2)	
	Female	190 (52.9)	14 (51.9)	204 (52.8)	*0.914
Breastfeeding History	Never breast-fed	7 (1.9)	0	7 (1.8)	
	Stopped > 1yr	112 (31.2)	9 (33.3)	121 (31.3)	
	Stopped < 1yr	75 ( 20.9)	6 (22.2)	81 (20.9)	
	Currently breastfeeding	165 (46.0)	12 (44.4)	177 (45.9)	*0.897
Respondent's Sex	Male	34 (9.5)	1 (3.7)	35 (9.1)	
	Female	325 (90.5)	26 (96.3)	351 (90.9)	*0.314
Respondent's Education	Primary	167 (46.5)	13 (48.1)	180 (46.6)	
	Secondary	136 (37.9)	10 (37.0)	146 (37.8)	
	Tertiary	45 (12.5)	4 (14.8)	49 (12.7)	***0.993
	Others	11 (3.1)	0	11 (2.8)	
Respondent's Occupation	High Risk of Pb exposure at work	2 (0.6)	2 (7.4)	4 (1.0)	OR = 14.28 (95% CI: 3.05 – 66.75) p = 0.001
	Low Risk of lead exposure at work	357 (99.4)	25 (92.6)	382 (99.0)	
House wall type	Stone	87 (24.2)	6 (22.2)	93 (24.1)	***0.812
	Mud	228 (63.5)	17 (63.0)	245 (63.5)	
	Iron Sheet	40 (11.1)	3 (11.1)	43 (11.1)	
	Wood	4 (1.1)	0	4 (1.0)	
	Others	0	1 (3.7)	1 (0.3)	
Source of Drinking Water	Tap	355 (98.9)	27 (100)	382 (99.0)	****n
	Borehole	1 (0.3)	0	1 (0.3)	
	Others	3 (0.8)	0	3 (0.8)	
Source of Kales	Kitchen garden	2 (0.6)	2 (7.4)	4 (1.0)	OR = 14.24 (95% CI: 3.05, 66.45); p = 0.001
	Market	238 (66.5)	18 (66.7)	256 (66.4)	
	Others	118 (32.9)	7 (25.9)	125 (32.4)	
Awareness of Pb-	Yes	21 (5.8)	0	21 (5.4)	**0.230
	No	338 (93.9)	27(100)	366 (94.3)	
No. of Known sources of Lead	Nil	346 (96.4)	27 (100)	373 (96.6)	**0.395
	1 to 2	7 (1.4)	0	5 (1.3)	
	3 to 4	5 (1.9)	0	7 (1.8)	

\*Chi Square test for independence, X<sup>2</sup> for nominal data with all cells having expected count more than 5; \*\*Spearman's correlation, P, for ordinal by ordinal variables; \*\*\*Collapsed to determine Chi square test of independence; \*\*\*\*N – 100% of children with BLL ≥ 10ug/dL

Table 2: Potential risk factors for children exposure to lead by blood lead concentration (n = 387) at Kibera slums in Nairobi, Kenya.

Potential Risk Factors	Blood Lead Concentration (ug/dL)		Total (n = 387) n (%)	p value
	< 10 (n = 359) n (%)	≥ 10 (n = 28) n (%)		
<b>Paint Exposure</b>				
Yes	108 (30.1)	7 (25.9)	115 (29.8)	OR = 1.23 (95%CI: 0.51 – 2.98), p = 0.649
No	251 (69.9)	20 (74.1)	271 (70.2)	
<b>Contaminated Playgrounds</b>				
Yes	272 (75.8)	21 (77.8)	293 (75.9)	OR = 0.89 (95%CI: 0.25 – 2.34), p = 0.627
No	87 (24.2)	6 (22.2)	93 (24.2)	
<b>Knows Poisoned playmate</b>				
Yes	2 (0.6)	1 (3.7)	3 (0.8)	OR = 0.073 (95%CI:0.004 - 0.75), p = 0.069
No	352 (99.4)	26 (96.3)	379 (99.2)	
<b>Occupationally exposed parent</b>				
Yes	105 (29.2)	10 (37.0)	115 (29.8)	OR = 0.70 (95%CI:0.302 – 1.58), p = 0.356
No	254 (70.8)	17 (63.0)	271 (70.2)	
<b>Living near Lead industry</b>				
Yes	89 (24.8)	4 (14.8)	93 (24.1)	OR = 1.90 (95%CI:1.54 – 5.54), p = 0.279
No	270 (75.2)	23 (85.2)	293 (75.9)	
<b>Pica Behavior</b>				
Yes	226 (63.0)	19 (70.4)	245 (63.5)	OR = 0.72 (95%CI: 0.31 – 1.68), p = 0.439
No	133 (37.1)	8 (29.6)	141 (36.6)	
<b>Signs of Lead Poisoning</b>				
Yes	34 (9.5)	2 (7.4)	36 (9.3)	OR = 1.31 (95%CI:0.30 - 5.76), p = 0.722
No	325 (90.5)	25 (92.6)	350 (90.7)	
<b>Use glazed pottery</b>				
Yes	64 (17.8)	5 (18.5)	69 (17.9)	OR = 0.10 (95%CI:0.35 - 2.62), p = 0.928
No	295 (82.2)	22 (81.5)	319 (82.1)	
<b>Cosmetic Exposure</b>				
Yes	52 (14.5)	5 (18.5)	57 (14.8)	OR = 0.75 (95%CI:0.27 - 2.06), p = 0.569
No	307 (85.5)	22 (81.5)	329 (85.2)	
<b>Mental Development Concerns</b>				
Yes	8 (2.2)	0	8 (2.1)	**N
No	351 (97.8)	27 (100)	380 (97.9)	

Table 3: Soil lead concentration and mean blood lead level by village in Kibera slums, in Nairobi, Kenya.

Village name	Soil Pb (ug/kg)	Mean Blood Pb level (ug/dL)
Ayany	11,365.00	5.285
D.C	15,270.00	6.180
Darajani	16,653.00	6.400
Fort Jesus	4,585.00	5.919
Kambi muru	18,905.00	7.275
Karanja	13,560.00	5.991
Katwekera	6,040.00	6.804
Kianda	89,570.00	5.353
Kichijio	5,209.00	6.353
Laini Saba	26,925.00	5.808
Lindi	33,450.00	7.011
Makina	25,220.00	6.261
Mashimoni	11,870.00	5.947
Mbobolulu	8,220.00	5.650
Olympic	12,520.00	6.241
Raila	3,365.00	4.100
Silanga	32,745.00	6.400
Soweto	7,250.00	6.944

Note: Villages with no figures for SoilPb have been omitted.

About 7% (n = 387) of the children tested had childhood lead poisoning (BLL  $\geq$  10ug/dL), which is higher prevalence than in more economically developed countries (CDC, 1997). The range of Soil Pb levels found in Kibera slums was 3,365 – 89,570 ug/kg (WHO allowable range: 100 – 120ug/kg). With such high soil lead levels coupled with documented pica behavior of children within the age group tested, the prevalence of childhood lead poisoning in Kibera could be higher than that detected using the more convenient, clinic – based sampling procedure used in this study. The knowledge on lead poisoning (5.4%, n = 387) and potential sources of exposure (3.1%, n = 387) among parents, guardians or caretakers in Kibera, were very low. Intervention strategies at the community would therefore require advocacy and education on childhood lead poisoning. The 7% prevalence of elevated BLL found in the study is of public health concern and needs to be addressed.

**ACKNOWLEDGEMENTS:** We thank all the participants and acknowledge funding provided in part by US NIH grants R25 TW007498 and K07 GM072954 through Dr. J.K. Griffiths of Tufts University, Boston, Massachusetts, USA. We would also like to thank J. Nassif and Massachusetts Public Health Laboratory for their assistance.

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***Journal of Applied Biosciences (2009), Vol. 13: 688 - 694.***

**ISSN 1997 – 5902: [www.biosciences.elewa.org](http://www.biosciences.elewa.org)**



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