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Assessment of blood lead levels among children in Owino Ouru Settlement in Mombasa County, Kenya, 2015

By
Lead Poisoning Investigation Team

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Executive Summary

Background: Concerns of possible lead exposure from fly ash emissions of a battery recycling factory in an informal settlement in Mombasa was brought up by the community members following high lead levels found in blood samples from three children. This study aimed at determining blood lead concentrations, lead levels in the environment and factors associated with elevated blood lead level among children aged 12 to 59 months from the settlement.

Methods: This is a cross sectional study of children aged 12-59 randomly selected from households in two informal neighbouring settlements (Owino Ouru and Bangladesh). Structured questionnaires on social, demographic, child's behaviour and household characteristics were administered to caregivers. Venous blood (1-3ml) drawn from each child was tested for lead using LeadCare®II portable analyzer. Household dust, drinking water and soil from compound in half of the sampled households was collected and tested for lead using graphite furnace atomic spectrometry at the Government chemist.

Results: We obtained blood samples from 161 children in 161 households, 83 soil samples, 76 dust and 73 water samples. Of these, 31 blood samples were not included in the analysis because they were obtained during pretesting and upon parents' request. We randomly selected 130 children, 65 from each settlement, of these 59 (45%) were males and median age was 39 months (Interquartile Range (IQR): 30 - 52). Blood lead levels ranged from 1μg/dL to 31μg/dL with 20 (31%) children from Owino Ouru and 5 (8%) children from Bangladesh having blood lead levels of ≥ 10μg/dL. Forty five (69%) children from Owino Ouru and 18 (28%) children from Bangladesh had blood lead levels of ≥ 5μg/dL. None of the children had blood lead levels above 45μg/dL. The mean blood lead level (9μg/dL; Standard deviation (SD): 6) of children from Owino Ouru was significantly higher than the mean blood lead level (4.4μg/dL; SD: 3) of children from Bangladesh, t= 5.47; 95% CI: 2.9 - 6.2; (p = < 0.0001). Nine (30%) of environmental samples from Owino Ouru and 15 (47%) of those from Bangladesh had lead concentrations in either soil >400mg/Kg, dust >40μg/f^2 or drinking water (>10μg/L or 0.01mg/L).The mean lead concentration of soil (387 mg/Kg; SD: 625) from Owino Ouru was also significantly higher than the mean lead concentration of soil (74mg/Kg; SD: 127) from Bangladesh, t= 2.77; 95% CI: 87 – 539; (p = 0.007). The presence of soil lead > 400 mg/Kg was associated with higher blood lead levels (p=0.01).

Conclusions: There were significantly high blood lead levels in children from Owino Ouru indicating that the children are being exposed to lead in their living environment. This requires medical care, blood lead screening, community sensitization, health education, nutritional counselling, surveillance and environmental lead reduction interventions.

Key words: Childhood, Blood, Lead, Environmental Exposure, Kenya

Introduction

It is estimated that high blood lead levels contribute to approximately 600,000 cases of intellectual disability in children annually (WHO, 2010) and was responsible for 143,000 deaths in 2004. There is no known safe threshold of blood exposure since adverse effects have been shown to occur at levels of below 5µg/dL (1). Low lead levels in young children results in mental retardation, impaired intellectual and cognitive function, learning disabilities, poor attention, hearing disorders and decreased growth (2). Acute lead poisoning causes gastrointestinal disturbances (anorexia, nausea, vomiting, abdominal pain), hepatic and renal damage and neurological effects (malaise, drowsiness, encephalopathy) that may lead to convulsions, coma and death (3). Lead exposure is often asymptomatic at low levels hence remains undiagnosed and untreated.

Children <5 years old are at increased risk of lead poisoning because of their innate curiosity and hand to mouth behaviour (4). Children have higher lead intake per unit body weight and body systems not fully developed leading to increased gastrointestinal absorption and distribution to the brain through the blood brain barrier (5). Their developing central nervous system is also more vulnerable to lead. Absorption of lead into the body is affected by many factors, including age, nutritional status and lead particulate size. Lead is poorly excreted, and most lead is sequestered in bone. As a result, elevated BLLs take months to years to decrease, even in cases where external exposures have been well controlled and chelation therapy has been instituted, because bone stores are mobilized into blood (6).

During the last decade measures have been taken to reduce lead exposure globally. Following the removal of lead from gasoline, dramatic reductions in blood lead concentrations have been observed (7) (8) however, leaded paint in houses built before 1950 remains the major source of lead exposure for American children (9). Children are exposed to lead through ingestion of weathering paint and inhaling dust or soil contaminated with such paint. In low income countries children are at increased risk of exposure to lead from other multiple sources (10). Studies have documented elevated blood lead levels in children living in communities involved in mining (11) smelting (12) and used acid lead battery recycling (13) where the children get exposed to lead by eating the contaminated soil or inhaling polluted air from the industrial emissions. Additionally, outbreaks of lead intoxication have been associated with drinking water from pipes, solders and fittings containing lead, use of ceramic utensils glazed with lead, eating food stored in lead soldered cans, playing with toys

containing lead, use of traditional remedies such as *kohl* and cosmetics containing lead (10). Adults working in jobs or hobbies such as smelting, auto repair, firing ranges, painting, ceramics, pottery, electrical, wire and cable works, battery manufacturing and recycling and stained glass making, carry the generated lead dust on clothes and contribute to the lead dust in the child's home. Children living in these homes are exposed to lead by licking dust laden fingers or inhaling lead particles in the air.

In 2007 a battery recycling and smelting factory was established in an informal settlement in Mombasa County along coastal Kenya. In 2010 an investigation was conducted in the Owino Ouru and three children were tested for blood lead levels. They had blood lead levels as high as 23µg/dl, 17µg/dl and 12µg/dl (Okeyo, 2012). Following these findings community members complained and the factory was closed in July 2012 by the local health authorities. Despite this action, concerns remained regarding appropriate closure of the factory and possible ongoing lead exposures in the community.

On 21st July 2014, the Ministry of Health (MOH) began an investigation to determine blood lead levels among children aged 12 to 59 months in the informal settlement, lead levels in the environment and identify risk factors for elevated blood lead level in this population. Findings from the investigation will enable public health authorities to make evidence based actions to safeguard public health in the affected community.

Methods

Site

We conducted the investigation in two informal settlements located in Mombasa County along coastal Kenya (Figure 1). Owino Ouru borders the northern side of the battery recycling factory, and consists of 450 households with a population of 1,700 persons. Bangladesh, a community located on the south western side and more than 2km upwind from the factory was included as the comparison site. Bangladesh consists of 1,500 households with a population of 5,000 persons. Bangladesh has similar demographics (such as traffic patterns, housing conditions) but does not share water sources with Owino Ouru and has no known battery recycling factories.

Investigation

We enrolled children aged 12 to 59 months who resided in either of the settlements since January 2014. We investigated children because at this age they are vulnerable to relatively low lead levels and develop irreversible health effects. We excluded children from the survey whose parents or guardians did not provide consent and households that were vacant at time of household listing. Village elders from the two settlements filled in a household list with details of household membership and ensured that all eligible households with children 12-59 months of age were included in the sampling frame.

When we assumed blood lead level variance in this population to be 25 and 10% non-response rate, a sample size of 65 children from each settlement provided us 99% power to detect a difference in mean blood lead levels of 4μg/dL at 95% confidence. We randomly selected 65 households from each sampling frame using table of random numbers. Upon reaching a house we administered four screening questions to whoever was found in the house. These questions included whether the household has a child aged 12 – 59 months, duration of stay in the settlement ≥12 months, presence of parent or guardian and whether they will provide a written consent. If the answers to all the four questions were 'yes' we proceeded with the interview. One child was randomly selected to participate in the study. If a house was unoccupied or the sampled child was not present at time of visit, the house was visited later that day or at different time on another day. If a house was permanently vacant, if the caregiver declined to participate or was not available for interview after multiple attempts

then the next household in the sampling frame was visited. We recorded this information on a household visitation log sheet.

A standard explanation describing the purpose of the study, the procedures to be followed, the risks and benefits of participation was read to all caregivers and a signed written consent obtained. Data was collected using a pretested structured questionnaire translated to Kiswahili. We collected demographic information including sex and age, clinical and treatment history, factors known to increase risk for elevated blood lead levels such as children's behaviour habits (frequency of outdoor playing, washing hands before eating, eating soil or sand), housing type, sources of drinking or cooking water, the presence of a household member with known exposure to lead, proximity to a battery recycling factory, and whether waste from factory stand around the house. Global positioning system (GPS) coordinates of place of residence was taken at the entrance of the house and used to map and calculate distances from the smelter factory.

Laboratory

A venous blood specimen was collected from every child. The venepuncture site was thoroughly cleaned with alcohol wipes, dried with dry gauze before the specimen was obtained. Approximately 1 -3ml of blood sample was collected in a vacutainer containing an anticoagulant, ethylenediamine tetra acetic acid (EDTA). In 10% of the children a second sample was obtained for quality control. Lead free blood collection supplies were provided by the Inorganic Chemistry Laboratory at US CDC. All blood samples were stored at 4°C and venous samples were shipped on frozen packs to be analyzed at the National Public Health Laboratory in Nairobi accompanied by laboratory request form.

The blood samples were tested for lead using LeadCare ® II, a portable blood lead analyzer. The LeadCare ® II instrument quantifies BLLs from 3.3- 65 µg/dL. These levels are measured with a level of detection (LOD) accuracy level of ± 3 µg/dL. We used the actual value of levels below the LOD for statistical analyses. However, parents and clinical health care providers were only notified that the child's BLL is below the LOD.

Environmental Sampling

We collected dust, water and soil samples from fifty percent of randomly visited households as per the US Housing and Urban Development (HUD) protocol sampling procedures

recognized by EPA (HUD, 1996). We collected five composite soil samples from bare soil outside the households where the child normally plays. A subsample was obtained from the center and one subsample from each of four different directions one meter from the center. The subsamples were collected by scooping the top surface soil using a four oz plastic scoop and poured into a labelled zip-lock bag. We collected water used by the household for drinking into a 125ml sample bottle. Dust was collected from the floor of the house by multidirectional wipe sampling method using wet wipes over an area laid using reusable template measuring 6 by 6 inches. The folded wipe was placed in a zip lock bag. All the used materials were discarded in trash bags. To prevent contamination or exporting of lead dust, hands were cleaned with baby wipes after each sample collection and clean gloves worn before collecting samples in the next area. All environmental samples were placed separately in properly labeled clean, plastic bags. Sample forms were filled with details that included unique sample number, gprs location, sampling method used, date of collection, and the name of the person who collected each sample. The samples together with laboratory form were sent to Government chemist laboratory for analyses. The environmental samples were analyzed for lead levels using graphite furnace atomic absorption spectrometry at the Government chemist laboratory.

Data management and analysis

All data was entered and validated in database using Epi Info™ 7.1.4 (CDC, Atlanta, GA, USA). We performed descriptive analysis and used the two-sample t-test in the continuous variables. An elevated blood lead level was defined as blood lead concentration $\geq$10μg/dL based on WHO guidelines. Contaminated environment was defined as lead concentration in either soil >400mg/Kg, in house dust >40μg/f^2 or in drinking water >50μg/L (0.05mg/L). The results were presented in tables and figures.

Ethical Considerations

Written informed consent to administer the questionnaire and draw blood was obtained from the caregivers. All the questionnaires, laboratory specimens and other records were identified by unique identity number to maintain participant confidentiality. We obtained additional blood samples from other children in the sampled household when the parent requested but their data was not included in this analysis. All the children who had elevated blood lead levels ($\geq$ 10μg/dL) were linked to follow up care as per the CDC guidelines of managing elevated BLL in children (ACCLPP, 2012). Questionnaires were stored under lock and key.

Electronic data was stored in a password protected database. The scientific approval for the study was obtained from the Ministry of Health, lead poisoning Technical Working Group (TWG). Expedited ethical approval was sought from the Kenya Medical Research Institute (KEMRI) since this investigation was considered a public health response. Prior to commencement of the investigation, permission was sought from the Mombasa County Department of Health Services.

Results

We visited 161 households and obtained blood samples from 161 children in both settlements. Responses from 31 participants were excluded from data analysis because data and blood samples were obtained during pretesting or from the children whose parents requested but were not in the sampling list. A total of 130 children aged 12 – 59 months: 65 from each settlement were included in the analysis. Median age was 39 months (IQR: 30 - 52), and 59 (45%) males. The median number of people living in households of sampled children was five (range 2 -13) and the median number of children 12 – 59 months in each sampled household was one child (range: 1 – 4). Of the 65 children from Owino Ouru, 31 (48%) lived within 200m radii from factory and 16 (25%) lived in a household with at least one adult working in any job involving lead for last five years. Nine (14%) of the children from Bangladesh were also living in a household with at least one adult working in any job involving lead for last 5 years (Table 1).

Blood lead levels among sampled children ranged from 1μg/dL to 31μg/dL. In overall, 63(48%) children had blood lead levels of ≥5μg/dL and 20 (19%) children had blood lead levels of ≥ 10μg/dL. There were 45 (69%) children from Owino Ouru and 18 (28%) children from Bangladesh with blood lead levels of ≥ 5μg/dL. Twenty (31%) children from Owino Ouru and five (8%) children from Bangladesh had blood lead levels of ≥ 10μg/dL. None of the children had blood lead levels above 45μg/dL. The mean blood lead level (9μg/dL; Standard deviation (SD): 6) of children from Owino Ouru was significantly higher than the mean blood lead level (4.4μg/dL; SD: 3) of children from Bangladesh, t= 5.47; 95% CI: 2.9 - 6.2; ($p = < 0.0001$).

We collected 83 soil samples, 76 dust and 73 water samples. Among these, 62 of each sample type: 30 from Owino and 32 from Bangladesh were included in the analysis. The remainder were obtained from households that recorded high blood lead in children and from the factory and its environs. The mean lead concentration of soil from Owino Ouru (387 mg/Kg; SD: 625) was significantly higher than the mean lead concentration of soil from Bangladesh (74mg/Kg; SD: 127); t= 2.77; 95% CI: 87 – 539; ($p = 0.007$). Soil samples obtained in the factory compound had the highest lead concentration (26,837 mg/Kg) followed by the one collected at the factory gate (2,381 mg/Kg). The mean lead loading of house dust from Owino Ouru (15.6μg/f^2; SD: 29) was higher than the mean lead loading of house dust samples from Bangladesh (4.7μg/f^2; SD: 14) but this difference was not statistically

significant t= 1.93; 95% CI: 0.4 – 22; (p = 0.058) and both levels were below the action levels. The lead concentration of water samples from Owino Ouru was below the action level (50µg/L).

In the analysis of children from Owino Ouru only, more males 24 (60%) as compared to females had blood lead levels ≥ 10µg/dL (OR: 5 (95% CI:2-15). Of the 20 children with blood lead level ≥ 10µg/dL, 19(90%) reside <200m radii from factory. There was no significant difference in blood lead among children living in households that had adults who worked for the last 5 years in jobs that involved lead. The main source of drinking or cooking water in 100% the households was from tanks supplied by the municipality. Children from houses that used other sources of water such as wells 8(40%), rain water 2(10%) and boreholes in 6(30%) did not show significant differences in the blood lead levels. Mud floor 5(25%), mud wall 15(60%), growing own or obtaining vegetables grown by neighbour for consumption 1(5%), eating soil/sand 13(65%), not washing hands all times before eating 9(45%), ever breastfed 65(100%), playing outside >6hrs 13(65%) and spending most time in school 3(15%), in compound 2(10%) or in the house 2(10%) also did not reveal any differences in blood lead (Table 2).

Discussion

Findings from our study demonstrate significant elevations in blood lead levels among children aged 12 – 59 months in Owino Ouru in comparison to similar children from Bangladesh. There were also significant elevations in soil lead concentration in Owino Ouru. Lead content in soil, close proximity of the household to the smelter and sex of child were identified as risk factors that expose children living in Owino Ouru to higher blood lead levels. Our findings support the hypothesis that blood lead levels among children in Owino Ouru are different from the blood lead levels of children from Bangladesh and the difference is associated with increased lead concentrations in the Owino Ouru children's environment. Urgent interventions are required to reduce lead exposure in the affected community.

Although none of the children had blood lead level 45μg/dL or above, the level at which CDC recommends chelating therapy, the resulting study showed that 70% of the tested children in Owino Ouru had levels above the reference value of 5μg/dL and 30% had blood lead levels at or above the WHO maximum acceptable level ≥10μg/dL, a rate 4 times higher than that found in children in a demographically similar community of Bangladesh that does not have a battery factory. This prevalence is higher than the prevalence of 1.4% reported in bio-monitoring studies of U.S. children 1-5 years old in NHANES 1999-2004 (7). Blood lead elevations have similarly been documented in other studies of children exposed to industrial emissions from used acid battery recycling informal establishments. Fifty nine percent of children in Jamaica (12), 80% of Nicaragua children (14) and 91% of children from Haina, Dominican (12) had blood lead level ≥10μg/dL. Three of the children in Owino Ouru had levels of between 20 and 45μg/dL. At those levels, there could be clinical evidence of lead poisoning (interference with vitamin D metabolism), but the health consequences of the prevailing elevations of blood lead are usually demonstrable only on a population basis as cognitive and developmental deficits. Further studies on neuropsychological and developmental evaluations are necessary. The public health response to such findings should be termination of toxic emissions and abatement of existing contamination; removal of children from the identified sources is usually not an option.

The finding that 8% of the children from Bangladesh had blood lead level >10μg/dL indicates that sources of lead exposure other than battery activities in these communities exist. This finding is however lower than the prevalence of 14% reported among 1-3 year old well Lebanese children presenting to paediatric clinics (15) and 30% of the controls in the

Nicaraguan children (14). Higher lead concentration observed in water samples could be a possible reason for this elevation. There is need to screen for blood lead, monitor water quality and establish the other common causes of lead exposure among the children not necessarily living near battery factory.

Our study had two limitations. First, the study sample size focused on determining difference in blood lead levels in the two settlements. We did not have enough statistical power to study the potential risk factors between the children with different blood lead levels. We also had few adults working in jobs known to involve lead and therefore we could not determine effect of take home lead. Secondly because this cross sectional investigation carried out two years after closure of factory, the magnitude of poisoning may have been different among the children who were in this age category two years ago. The difference in blood lead levels obtained in this study cannot be generalised to that of children living in the settlement when the factory was operational. Despite these limitations it appears that significant childhood lead exposure from the environment still persists, however follow-up assessments are needed to identify all potential sources.

Conclusions

There was significantly high blood lead levels in children from Owino Ouru associated with high soil lead concentrations. The most immediate priority is to reduce exposure to lead and other contaminants. This is best accomplished by developing and implementing a comprehensive and integrated intervention plan. Specifically, the process should ensure that the factory remains closed so as to reduce air lead emissions; Implement interventions that have been demonstrated scientifically to reduce lead exposure from historical soil contamination and develop a scientifically robust plan to monitor the impact of lead reduction efforts. To strengthen the overall process and plan, and to improve credibility and ensure that monitoring and other needs of affected parties are met, stakeholders should participate in reduction planning, implementation, and monitoring of lead and other contaminants.

Laboratory results should be made available to study participants immediately for appropriate action. In 2012 CDC adopted an upper reference range value for blood lead of 5µg/dL based on the 97.5% of the distribution of BLLs in children. We recommend that children with blood lead levels $\geq$ 5µg/dL have venous blood lead tests performed three months after this investigation and follow up of the children over time until the environmental investigations

and subsequent responses are complete. Children with blood lead levels < 5 μg/dL require retesting in not less than one year until the lead sources have been controlled or eliminated or they turn 6 years old. In this community irreversible adverse effects of lead poisoning will be prevented if children undergo monitoring of blood lead levels.

Certain vitamins and minerals, especially Calcium, Iron, Zinc and vitamin C, play a specific role in minimizing lead absorption. A well-balanced diet is essential to meeting the child's recommended daily allowance of essential vitamins and minerals and to provide adequate calories for growth. Regular assessment of the child's nutritional status during follow up care can identify children with inadequate intake of these and other nutrients, and allow the clinician to proactively recommend supplementation. Children exposed to lead should be evaluated for anaemia and any iron deficiency corrected using iron supplements (AAP guidelines).

Training of clinicians on high index of suspicion, lead screening questions, blood testing of lead, and treatment is essential in management of lead exposure. Clinicians should emphasize on healthy nutrition and/or dietary supplements. They should also be involved in outreach and community health education, overseeing ongoing monitoring of children with elevated blood lead levels, defined as levels above the reference value, coordinating efforts with parents and county authorities to minimize risks to individual children and to assist communities in their primary prevention efforts.

Primary prevention is a strategy that emphasizes the prevention of lead exposure, rather than a response to exposure after it has taken place. Primary prevention is necessary because the effects of lead appear to be irreversible. In the U.S., this strategy largely requires that children not live in older housing with lead-based paint hazards. Screening children for elevated BLLs and dealing with their housing only when their BLL is already elevated is no longer advisable (Centers for Disease Control and Prevention (CDC) Advisory Committee on Childhood Lead Poisoning Prevention (ACCLP, 2012). The goal of primary prevention is to ensure that all homes become lead-safe and do not contribute to childhood lead exposure. Prevention requires that we reduce environmental exposures before children are exposed to these hazards. It is important to carry out regular environmental testing of dust, soil and water testing and evaluation of homes for lead sources. Termination of toxic emissions and abatement of existing contamination is necessary to reduce exposures. This may require the

removal of children from the identified sources until the environment is confirmed to be safe for the children.

There is need for coordination of care with the local authorities and organizations to plan a response strategy. Efforts to increase awareness of lead hazards and ameliorative nutritional interventions are also key components of a successful prevention policy. Finally the establishment of a screening program and effective screening policies and practices by the Kenya Ministry of Health will ensure that the children of high-risk families are screened, and that lead-exposed children or children with elevated blood lead levels receive key environmental interventions and case management services.

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References

1. Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, et al. Low-Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis. Environ Health Perspect. 2005 Jul;113(7):894–9.

2. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual Impairment in Children with Blood Lead Concentrations below 10 ug per Deciliter. N Engl J Med. 2003 Apr 17;348(16):1517–26.

3. Bellinger DC. Lead. Pediatrics. 2004 Apr;113(4 Suppl):1016–22.

4. Abadin H, Ashizawa A, Stevens Y-W, Llados F, Diamond G, Sage G, et al. Toxicological Profile for Lead [Internet]. Atlanta (GA): Agency for Toxic Substances and Disease Registry (US); 2007 [cited 2015 Apr 29]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK158766/

5. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain. 2003 Jan 1;126(1):5–19.

6. Kaul B, Sandhu RS, Depratt C, Reyes F. Follow-up screening of lead-poisoned children near an auto battery recycling plant, Haina, Dominican Republic. Environ Health Perspect. 1999 Nov;107(11):917–20.

7. Jones RL, Homa DM, Meyer PA, Brody DJ, Caldwell KL, Pirkle JL, et al. Trends in Blood Lead Levels and Blood Lead Testing Among US Children Aged 1 to 5 Years, 1988–2004. Pediatrics. 2009 Mar 1;123(3):e376–85.

8. Strömberg U, Schütz A, Skerfving S. Substantial decrease of blood lead in Swedish children, 1978-94, associated with petrol lead. Occup Environ Med. 1995 Nov;52(11):764.

9. Needleman H. Lead poisoning. Annu Rev Med. 2004;55:209–22.

10. Falk H. International Environmental Health for the Pediatrician: Case Study of Lead Poisoning. Pediatrics. 2003 Jul 1;112(Supplement 1):259–64.

11. Dooyema CA, Neri A, Lo Y-C, Durant J, Dargan PI, Swarthout T, et al. Outbreak of Fatal Childhood Lead Poisoning Related to Artisanal Gold Mining in Northwestern Nigeria, 2010. Environ Health Perspect. 2011 Dec 20;120(4):601–7.

12. Lalor GC, Vutchkov MK, Bryan ST, Christie CDC, Donaldson D, Young J, et al. Acute lead poisoning associated with backyard lead smelting in Jamaica. West Indian Med J. 2006 Jan;55(6):394–8.

13. Haefliger P, Mathieu-Nolf M, Lociciro S, Ndiaye C, Coly M, Diouf A, et al. Mass Lead Intoxication from Informal Used Lead-Acid Battery Recycling in Dakar, Senegal. Environ Health Perspect. 2009 Oct;117(10):1535–40.

14. Bonilla CM, Mauss EA. A community-initiated study of blood lead levels of Nicaraguan children living near a battery factory. Am J Public Health. 1998 Dec;88(12):1843.

15. Nuwayhid I, Nabulsi M, Muwakkit S, Kouzi S, Salem G, Mikati M, et al. Blood Lead Concentrations in 1–3 Year Old Lebanese Children: A Cross-sectional study. Environ Health. 2003 Apr 15;2(1):5.

ACCLPP (2012). Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention. Atlanta: Centers for Disease Control and Prevention.

Brown, M.J., McWeeney, G., Kim, R., Tahirukaj, A., Bulat, P., Syla, S., Savic, Z., Amitai, Y., Dignam, T and Kaluski, D.N. (2010). Lead poisoning among internally displaced Roma, Ashkali and Egyptian children in the United Nations-Administered Province of Kosovo. *The European Journal of Public Health 20, 288–292.*

HUD. (1996). HUD guidelines for the Evaluation and Control of Lead based paint hazards in housing. Chapter 5 Lead based paint risk assessment. Retrieved from *http://www.hud.gov/offices/lead/lbp/hudguidelines/Ch05.pdf*

Okeyo, D. (2012). Lead Poisoning in Owino Uhuru Slums in Mombasa- Kenya. Integrated SAICM Implementation Project (ISIP).

WHO. (2010). Global health risks: Mortality and burden of disease attributable to selected major risks. Geneva, Switzerland: *World Health Organization.*

Lead in Drinking-water

WHO (2011). Background document for development of WHO Guidelines for Drinking-water Quality

Tables and figures

Table 1: Demographic, clinical and environmental characteristics of children in Lead study in two informal Settlements, Mombasa County, Kenya, January 2015

Characteristics	Owino Ouru (N = 65) n (%)	Bangladesh (N = 65) n (%)	Total (N = 130) n (%)
Male	13 (52)	58 (55)	71 (55)
Median age in months (IQR)	42 (31 – 50)	36 (27 – 52)	39 (30 – 52)
Age Groups (Months)			
12 – 23	10 (15)	14 (22)	24 (19)
24 – 35	15 (23)	18 (28)	33 (25)
36 – 47	18 (28)	6 (9)	24 (19)
48 – 59	22 (34)	27 (42)	49 (38)
Living < 200m from Battery recycling Factory	31 (48)	0 0)	31 (24)
Living in HH* with adults working in job involving lead for last 5 yrs	16 (25)	9 (14)	25 (19)
Blood lead Levels (µg/dL)			
< 5.0	20 (31)	47 (72)	67 (52)
5 – 9.9	25 (39)	13 (20)	38 (29)
10 – 19.9	16 (25)	5 (8)	21 (16)
20 – 45.0	4 (6)	0 (0)	4 (3)
Clinical Symptoms			
Recurrent abdominal Pain	20 (31)	18 (28)	38 (29)
Speech/language delay	11 (17)	2 (3)	13 (10)
Recurrent constipation	6 (9)	4 (6)	10 (8)
Joint pains	4 (6)	2 (3)	6 (5)
Poor concentration	2 (3)	2 (3)	4 (3)
Muscle aches	2 (3)	1 (2)	3 (2)
Seizures	0 (0)	1 (2)	1 (1)
Lead content of environmental samples			
(n)	30	32	62
Soil (>400mg/Kg)	6(20)	1(3)	7(11)
House dust (>40µg/f^2)	4(13)	3(9)	7(11)
Drinking water (>10µg/L or 0.01mg/L)	1(3)	14(44)	15(24)

*Households

Table 2: Analysis of child's behaviour, household characteristics and environment by blood lead level among children aged 12 – 59 months in an informal Settlement, Mombasa County, Kenya, January 2015

	Blood Lead Level (µg/dL)			
	>10 (N=20) n (%)	< 10 (N=45) n (%)	OR (95% CI)	p- Value
Male	24 (60)	12 (53)	5 (2 -15)	0.002
Age Groups (Months)				
12 – 23	3 (15)	7 (16)	1 (0.2 - 4)	0.950
24 – 35	7 (35)	8 (18)	3 (1 - 8)	0.131
36 – 47	4 (20)	14 (31)	1 (0.2 - 2)	0.359
48 – 59	6 (30)	16 (36)	1 (0.2 - 2)	0.665
Living < 200m from Battery Recycling factory	18 (90)	13 (29)	22 (5 - 109)	< 0.0001
Adult worked in job that involved lead	6 (30)	10 (22.2)	_2 (1 - 5)	0.5
Households with adults working in following jobs for last 5 yrs.				
Smelting	2 (10)	2 (4)	2 (0.3 - 18)	0.393
Auto Repair	1 (5)	5 (11)	1 (0.1 - 4)	0.436
Firing Ranges	0 (0)	2 (4)		
Painting	2 (10)	6 (13)	1 (0.1 - 4)	0.708
Ceramics	2 (10)	2 (4)	2 (0.3 -18)	1.393
Electrical	1 (5)	2 (4)	1 (0.1 -14)	0.922
Battery Manufacturing	2 (10)	0 (0)		
Recycling Batteries	5 (25)	0 (0)		
Wire and Cable	1 (5)	0 (0)		
Pottery	2 (10)	1 (2)	5 (0.4 - 57)	0.171
Stained glass making	2 (10)	2 (4)	2 (0.3 - 18)	1.393
Primary Drinking & Cooking Water Source				
Public Water	65 (100)	65 (100)		
Well	8 (40)	11 (24)	2 (1 - 6)	0.207
Rain Water	2 (10)	7 (16)	1 (0.1 - 3)	
Bore hole	6 (30)	10 (22)	2 (1 - 5)	0.505
Water Vendors	0 (0)	2 (4)		
Floor Type				
Mud	5 (25)	17 (38)	1 (0.2 - 2)	0.318
Concrete	13 (65)	26 (58)	1 (1 - 4)	0.586
Wall type				
Mud	15 (60)	76 (72)	1 (0.2 – 1)	0.227
Bricks/Stone	15 (24)	18 (27)	1 (0.4 – 2)	0.690
Sources of Vegetables/Fruits consumed in household				
Grow Own	1 (5)	12 (27)	0.1 (0.02 - 1)	0.04
Neighbour grows	0 (0)	4 (9)		
Vendors	15 (75)	35 (78)	1 (0.3 - 3)	0.808
Types of Vegetables consumed that are grown by household or from neighbour who grows				
Tomatoes	0 (0)	8 (18)		
Kales	0 (0)	11 (24)		

Onions	0 (0)	9 (20)		
Traditional Vegetables	1 (5)	17 (38)	0.1 (0.01-1)	0.007
Carrots	0 (0)	6 (13)		
Spends most of day				
In School	3 (15)	9 (20)	3 (1 -13)	0.245
Inside compound	2 (10)	4 (9)	2 (0.2 - 7)	0.887
In the house	2 (10)	4 (9)	2 (0.2 - 7)	0.887
Eat Soil or Sand	13 (65)	23 (51)	2 (1 – 5)	0.302
Doesn't wash hands all times	9 (45)	15 (33)	2 (1 – 5)	0.372

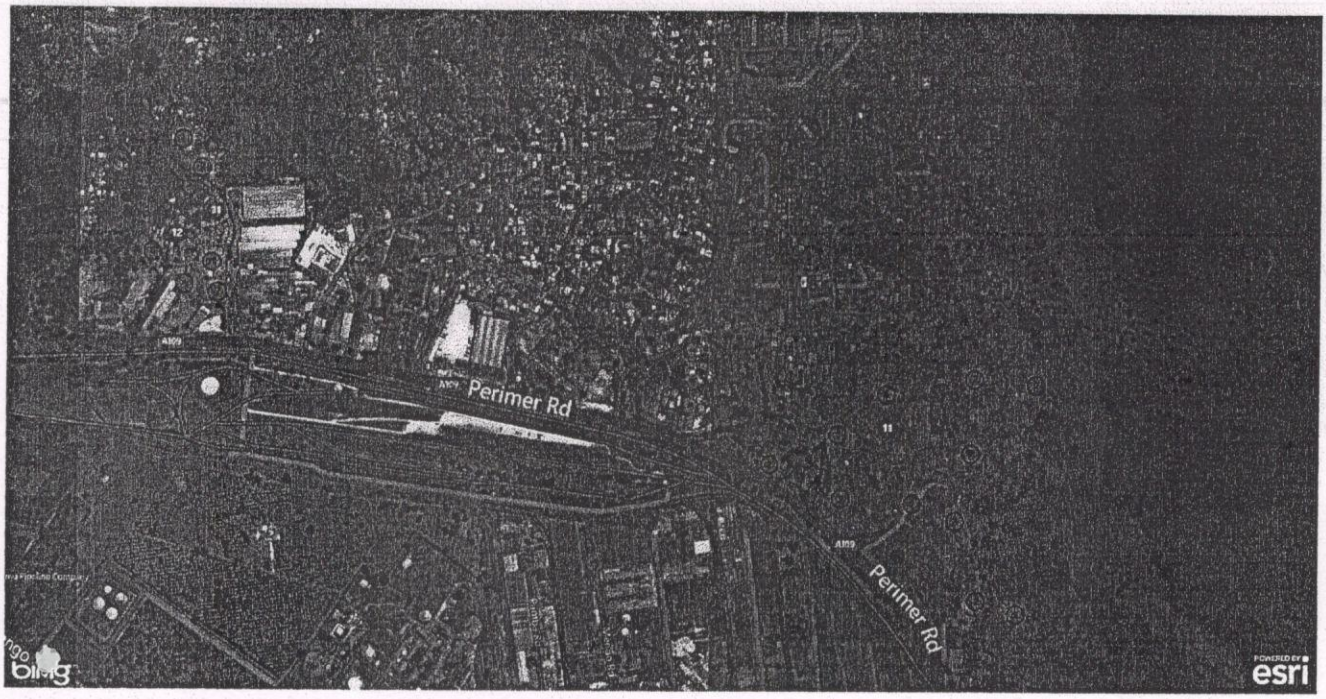

Figure 1: Map showing distribution of children aged 12 – 59 months in two informal settlements