



Published in final edited form as:

J Health Pollut. 2016 September ; 6(11): 2–8. doi:10.5696/2156-9614-6-11.2.

Prevalence and Risk Factors of Elevated Blood Lead in Children in Gold Ore Processing Communities, Zamfara, Nigeria, 2012

John A. Kaufman¹, Mary Jean Brown¹, Nasir T. Umar-Tsafe^{2,3}, Muhammad Bashir Adbullahi^{2,3}, Kabiru I. Getso^{2,4}, Ibrahim M. Kaita^{2,5}, Binta Bako Sule⁵, Ahmed Ba'aba², Lora Davis², Patrick M. Nguku², and Nasir Sani-Gwarzo⁶

¹National Center for Environmental Health, US Centers for Disease Control and Prevention, Atlanta, USA

²Nigeria Field Epidemiology and Laboratory Training Program, Abuja, Nigeria

³Ministry of Health, Zamfara State, Nigeria

⁴Ministry of Health, Kano State, Nigeria

⁵State Primary Health Care Development Agency, Katsina State, Nigeria

⁶Nigerian Federal Ministry of Health, Abuja, Nigeria

Abstract

Background—In March 2010, Medecins Sans Frontieres/Doctors Without Borders detected an outbreak of acute lead poisoning in Zamfara State, northwestern Nigeria, linked to low-technology gold ore processing. The outbreak killed more than 400 children 5 years of age in the first half of 2010 and has left more than 2,000 children with permanent disabilities.

Objectives—The aims of this study were to estimate the statewide prevalence of children 5 years old with elevated blood lead levels (BLLs) in gold ore processing and non-ore-processing communities, and to identify factors associated with elevated blood lead levels in children.

Methods—A representative, population-based study of ore processing and non-ore-processing villages was conducted throughout Zamfara in 2012. Blood samples from children, outdoor soil samples, indoor dust samples, and survey data on ore processing activities and other lead sources were collected from 383 children 5 years old in 383 family compounds across 56 villages.

Results—17.2% of compounds reported that at least one member had processed ore in the preceding 12 months (95% confidence intervals (CI): 9.7, 24.7). The prevalence of BLLs ≥ 10 $\mu\text{g/dL}$ in children 5 years old was 38.2% (95% CI: 26.5, 51.4) in compounds with members who processed ore and 22.3% (95% CI: 17.8, 27.7) in compounds where no one processed ore. Ore processing activities were associated with higher lead concentrations in soil, dust, and blood samples. Other factors associated with elevated BLL were a child's age and sex, breastfeeding, drinking water from a piped tap, and exposure to eye cosmetics.

Conclusions—Childhood lead poisoning is widespread in Zamfara State in both ore processing and non-ore-processing settings, although it is more prevalent in ore processing areas. Although most children’s BLLs were below the recommended level for chelation therapy, environmental remediation and use of safer ore processing practices are needed to prevent further exposures.

Patient consent—Obtained

Ethics approval—The study protocol was approved by the US Centers for Disease Control Institutional Review Board-A and the National Health Research Ethics Committee of Nigeria.

Competing Interests—The authors declare no competing financial interests.

Keywords

lead poisoning; mining; children’s environmental health

Introduction

A 2010 outbreak of acute lead poisoning in rural Zamfara State, northwestern Nigeria resulted in severe neurological morbidity and mortality in several hundred children, a result of lead-contaminated gold ore processed with low technology methods.¹ Gold ore processing became common in Zamfara after gold prices rose during the 2008–2012 global economic recession.² Lack of emission controls during processing, and the co-occurrence of gold ore with bioaccessible lead in the region’s underlying geology caused extensive contamination of villages. By March 2010, >1400 children had died and >2,000 children were left permanently disabled from inhalation and ingestion of lead particulates.³ An initial investigation found two mining villages where 118 of 463 (25%) children 5 years old had died in the previous 12 months, 82% of whom experienced symptoms consistent with lead poisoning.¹ Of the surviving children, 97% had blood lead levels (BLLs) 45 µg/dL, the level recommended by the US Centers for Disease Control and Prevention (US CDC) for chelation therapy, with measured BLLs as high as 700 µg/dL.⁴ Lead concentrations in residential soil exceeded the US Environmental Protection Agency’s (USEPA) 400 µg/g limit in 85% of samples, ranging to >100,000 µg/g, the limit of detection for the handheld x-ray fluorescence analyzer used. A follow-up investigation reported that the odds of lead poisoning or contamination were 3.5 times higher in ore processing villages versus non-ore-processing villages.⁵

Lead toxicity can affect every organ system, often with irreversible effects.⁶ The World Health Organization estimates that 16% of children worldwide have BLLs 10 µg/dL, with 90% living in low-income countries.⁷ Children are especially vulnerable to lead toxicity due to their physiology, and are exposed primarily through hand-to-mouth behaviors and inhalation.⁸ Childhood lead poisoning is associated with impaired neurodevelopment and cognitive function, lower IQ, impaired hearing, and reduced stature.⁹ Symptoms of acute lead poisoning include convulsions, coma, and death.¹⁰ There is no known safe BLL in children.¹¹ The US CDC currently uses a BLL in children of 5 µg/dL for action to reduce exposures.¹² International efforts have successfully reduced lead in many parts of the world, although many sources remain.¹³ Subsistence gold mining using low-technology methods such as manual ore grinding and liquid mercury amalgamation accounts for an estimated

20–30% of global gold production and is an important potential source of environmental contamination, and Zamfara’s underlying geology makes subsistence gold mining in this region particularly hazardous.^{2,14}

Previous investigations in Zamfara focused on highly contaminated villages for priority environmental and medical interventions. In these investigations, background lead levels in soil in villages which did not process ore were <25 µg/g. In villages which did process ore, soil lead levels ranged from 1,560–69,700 µg/g, and soil lead was highly bioaccessible, with an estimated absorption through the gastrointestinal system of 39–66%.²

Our objectives were to estimate the statewide prevalence of elevated BLLs in children 5 years old in ore processing and non-ore-processing family compounds (clusters of proximate polygamous family dwellings) and villages (dispersed collections of compounds in a rural area), and to identify factors associated with elevated BLL.

Methods

The study protocol was granted approval by the US CDC Institutional Review Board-A and the National Health Research Ethics Committee of Nigeria. Field work was performed during June and July of 2012.

Sampling Approach

Zamfara has 14 local government areas (LGAs). Because census data were unavailable, we used LandScan imagery data (Oak Ridge National Laboratory, Oak Ridge, TN, USA) to produce population estimates. Each LGA was divided into 5 × 5 km² grids, and two villages were randomly selected from each grid, with the probability of selection relative to population estimates. Seven family compounds were randomly sampled within each selected village. One child was randomly selected within each compound.¹⁵ Eligible children were 5 years old and had lived in Zamfara for 10 months of the previous year. From the 14 LGAs, we randomly selected 112 villages (n=328 children) to complete a village-level survey, with ten additional villages selected as potential replacements if a village was inaccessible. To obtain population-based estimates of children’s BLLs and environmental lead levels, 56 villages were randomly selected from among all 122 selected villages (n=392 children).

Compound Surveys

A survey on demographic and risk factor data for enrolled children was administered to the head of the family compound. The survey asked about any compound member’s participation in six ore processing activities: breaking ore into gravel-sized pieces; grinding these pieces into a powder with flour grinders; washing the powder with water to separate gold particles; drying the powder after washing; separating gold from other particles through amalgamation using liquid mercury; and vaporizing mercury to remove it from the gold mixture.¹ Surveys also asked the following: whether the compound’s members processed ore and which activities they performed; whether compound members participated in lead-sulphate-based eyeliner manufacture, smelting, auto repair, activities on a firing range, painting, ceramics production, pottery, electrical work, or battery recycling; whether the

family used ceramic plates or cups at home; whether the child wore lead-sulphate-based eyeliner or had used home remedies in the past 30 days; whether the home was cleaned daily; whether the child was breastfed in the past 12 months; the child's primary and alternative drinking water sources; and the child's sickle cell status, age, and sex.

Blood Samples

Two venous blood samples (minimum 1.5 mL in a 5 mL Vacutainer®) were drawn from each enrolled child by a trained phlebotomist using equipment verified as lead free by the US CDC Division of Laboratory Sciences. One sample was analyzed using anodic stripping voltammetry with LeadCare II point of care instruments by trained members of the study team within 24 hours of collection. The other sample was sent unopened to the US CDC Division of Laboratory Sciences for analysis using inductively coupled plasma mass spectrometry (ICPMS). ICPMS has a detection limit of 0.25 µg/dL. These results were used in the analysis described here. Point of care results were shared with Medecins Sans Frontieres/Doctors Without Borders physicians accompanying field teams, Zamfara State Ministry of Health, the Nigerian CDC, and community representatives who informed parents.

Environmental Samples

At least 16 cm³ (1 in 3) of floor dust was collected by sweeping a 1 m² area in the child's sleeping area identified by the child's mother, using dedicated lead-free brushes and placed in a plastic bag. An outdoor soil sample was collected from the compound in the child's play area. Dust and soil samples were split, with one analyzed in Nigeria using handheld x-ray fluorescence and the other sent to the Department of Geosciences at Georgia State University. The latter were sieved at 2.0 µm with a nylon sieve, ground using ceramic mills, and handled in a high efficiency particulate arrestance-filtered hood. Powders were digested using a combination of hydrofluoric, perchloric, hydrochloric, and nitric acids in open Teflon beakers.^{16,17} All constituents were analyzed on a simultaneous inductively coupled plasma atomic emission spectrometer. Lead values <50 mg/kg were confirmed using an atomic absorption spectrophotometer. Standard reference materials were used for quality assurance and control. A common drinking water well was identified for each village, with samples taken from uncovered (n=51) and covered (n=29) wells where possible, for a total of 80 wells sampled in 54 villages. Water samples were analyzed using ICPMS at the University of Wisconsin.

Statistical Analysis

Statistical analyses were performed using SAS 9.3 (SAS Institute Inc., Cary, NC, USA) and Microsoft Excel (2010). When estimating prevalence and 95% confidence intervals (CI) for ore processing activities and elevated BLLs, we applied sampling weights to account for unequal probabilities of selection into the study due to the sampling design.^{18,19} Statistical analyses of blood and environmental data used lab results from samples sent to the US CDC Division of Laboratory Sciences, Georgia State University, and the University of Wisconsin. Due to right-skewed distributions, we used the natural log of blood, soil, and dust lead for tests requiring normal distributions. We performed multiple imputation using the expectation-maximization method to account for missing values for 19 dust samples and 18

soil samples (5.0% and 4.7% of observations, respectively), assumed to be missing at random. We used Pearson tests to assess correlations between continuous variables. We used linear regression to identify factors associated with increasing BLL, using a forward selection process with an entry criteria of $p < 0.10$. Soil and dust data were excluded from the final model as these were assumed to be an intermediate media for lead from ore to children's blood, as suggested by previous studies.²

Results

We collected data from 392 compounds. Blood samples were insufficient for nine children, leaving a final sample of 383 children with a mean age of 35.2 months (standard deviation 16.2), with 52% boys and 48% girls. Less than 6% of the 383 compounds reported being engaged in lead-related occupations, except for auto repair, which was reported by 12.8% of compounds. Of the 383 children, 28.9% (111) had breastfed in the previous 12 months, 94.5% (362) lived in homes cleaned daily, and 87.0% (333) had worn lead-sulphate-based eyeliner in the previous 30 days (79% of boys and 96% of girls).

The estimated prevalence of having a compound member who had processed ore in the previous 12 months was 17.2% (95% CI: 9.7, 24.7; $n=55$). The estimated prevalence of each processing activity was as follows: 12.8% for breaking (95% CI: 7.1, 18.6; $n=43$), 7.6% for grinding (95% CI: 2.9, 12.4; $n=31$), 16.3% for washing (95% CI: 9.3, 23.2; $n=50$), 13.1% for drying (95% CI: 6.7, 19.5; $n=45$), 7.3% for separating (95% CI: 2.3, 12.4; $n=30$), and 4.9% for vaporizing mercury (95% CI: 0.5, 9.2; $n=18$).

Elevated BLLs were widespread in all compounds, although were more prevalent in children whose compound members processed ore, as shown in Table 1.

The geometric mean (GM) BLL was 9.5 $\mu\text{g/dL}$ (95% CI: 7.9, 11.4) in compounds with members who processed ore (range: 2.6–61.3 $\mu\text{g/dL}$) and 6.5 $\mu\text{g/dL}$ (95% CI: 6.1, 6.8) in compounds with no members who processed ore (range: 1.6–44.7 $\mu\text{g/dL}$). The GM lead concentration in dust samples was 40.5 mg/kg (95% CI: 32.4, 50.5) in compounds with members who processed ore (range: 14.0–710.0 mg/kg) and 30.7 mg/kg (95% CI: 27.6, 34.1) in compounds with no members who processed ore (range: 6.8–20,000 mg/kg). The GM lead concentration in soil samples was 39.2 mg/kg (95% CI: 31.1, 49.3) in compounds with members who processed ore (range: 11.0–560.0 mg/kg) and 24.3 mg/kg (95% CI: 22.3, 26.4) in compounds with no members who processed ore (range: 5.3–1,100 mg/kg).

Fifty (62.5%) water samples tested below the limit of detection for lead (1 $\mu\text{g/L}$). Wells in non-ore-processing villages ($n=22$) had a similar mean lead concentration to wells in ore processing villages ($n=58$) [2.2 $\mu\text{g/L}$ (95% CI: 0.0, 5.3) vs 2.4 $\mu\text{g/L}$ (95% CI: 1.1, 3.3)].

There were statistically significant positive Pearson correlations between natural-log transformed lead concentrations in soil and blood ($r=0.3$), dust and blood ($r=0.3$), and soil and dust ($r=0.6$), as well as between the number of ore processing activities performed by the child's compound members and BLL ($r=0.3$). Children in compounds with members performing all six processing activities had GM BLLs of 14.1 (95% CI: 9.0, 21.9), higher than children in compounds performing fewer activities. The GM BLL was highest among

children whose compound members participated in vaporizing mercury to remove it from the ore (Figure 1).

Differences in GM BLL were statistically significant ($p < 0.05$) for children living in villages where any village resident participated in any ore processing activity, washing, drying, or vaporizing mercury compared to children in villages where it was reported that no one processed ore, regardless of whether children's family compound members processed ore (Table 2). Although not statistically significant, breaking and grinding showed a similar trend.

The covariates identified as risk factors for natural-log transformed elevated BLL in the final linear regression model included living in a compound in which family members participated in vaporizing mercury (standardized coefficient ($\beta = 0.45$, 95% CI: 0.10, 0.81), ore grinding ($\beta = 0.30$, 95% CI: 0.06, 0.55), and separating ($\beta = 0.16$, 95% CI: -0.20, 0.51) in the last 12 months, using a piped tap as the primary drinking water source ($\beta = 0.63$, 95% CI: 0.33, 0.93), lead-sulphate-based eyeliner exposure in the past 30 days ($\beta = 0.15$, 95% CI: -0.02, 0.31), breastfeeding in the past 12 months ($\beta = 0.14$, 95% CI: 0.002, 0.28), male sex ($\beta = 0.12$, 95% CI: 0.01, 0.23), and child's age ($\beta = -0.004$, 95% CI: -0.008, -0.0004).

Discussion

We found elevated BLL rates lower than those reported in previous studies of highly contaminated villages conducted before widespread education on the dangerous but then-common practice of processing ore within compounds.^{1,5} Nevertheless, we found that children with family members involved in ore processing were more likely to have elevated BLLs, and that BLLs tended to increase with the number of processing activities.

Our estimates for the GM BLL in children in non-ore-processing compounds (6.5 $\mu\text{g}/\text{dL}$) approximate those reported for children in urban environments in Nigeria.^{20,21} Elevated BLLs in non-processing compounds might result from exposure to mine tailings generated by other village residents, as suggested by data in Table 2, or from using contaminated soil or water to make house bricks, processing prior to the 12 months in question, exposure to lead-sulphate-based eyeliner, or drinking water from leaded pipes.

Activities most strongly associated with higher BLL were grinding, separating, drying, and vaporizing mercury. Vaporizing was the least prevalent, but most strongly associated, possibly due to highly respirable aerosolized particulates produced from heating lead. Our finding that having compound members engaged in more activities was associated with higher BLLs may be explained by generation of more lead tailings, leading to environmental contamination, and to take-home lead on workers' clothing and skin.

The USEPA's recommended 400 $\mu\text{g}/\text{g}$ limit for play area soil was exceeded by four sampled compounds, and indoor sleep area dust samples exceeded this level in 10 sampled compounds. By comparison, 85% of compounds sampled in 2010 had soil concentrations $> 400 \mu\text{g}/\text{g}$.¹ While GM lead concentrations in soil and dust were higher in compounds where ore processing workers lived in our study, the highest concentrations were found in compounds that reported no processing in the previous 12 months. These compounds may

have processed ore prior to the previous 12 months, or used soil or water contaminated from nearby processing to make bricks for their homes.

Our findings that elevated BLLs were associated with being male, younger age, lead-sulphate-based eyeliner exposure, and breastfeeding are consistent with the current literature.^{22–24} Our observed association between piped tap water and elevated BLL could be due to lead pipes or solder. Data on lead in pipes were not collected.

This study benefited from a large sample of compounds with and without members engaged in ore processing, survey data, and blood, soil, and dust samples. These data are likely free from effects of seasonal variation in lead exposures, as all field work was performed during the beginning of the rainy season, when tailings are less likely to be spread by dry, windy conditions.²⁵

Drinking water is a possible lead ingestion route, although we did not observe an association between processing and lead in water. Most families use multiple seasonal water sources, including rainwater, so a single sample would not reflect exposures. Additionally, village-level water samples are a more crude exposure measure than compound-level soil, dust, and blood samples.

Study limitations include a lack of data on the actual locations of ore processing, and a lack of ability to distinguish processing-related take-home lead exposures from direct exposures to tailings. Additionally, we did not collect data on children's behaviors that could impact exposure, such as hand-to-mouth behaviors or time spent near ore processing or tailings. Our inclusion of child's age and sex in regression models may have controlled for some potential effect of behaviors on lead exposure. Another limitation is the lack of detailed dietary data on factors that could reduce lead uptake, such as iron supplements, or that could increase exposures, such as eating animals hunted with lead ammunition.²⁶

There are a number of opportunities for further study. Little is known about the synergistic effects of multiple metal exposures, and exploring this relationship in Zamfara could be informative. We did not find alarmingly high mercury levels in children, although they were elevated by US standards. Blood manganese and cadmium levels were also elevated for some children (data not shown). It would also be informative to perform follow-up cognitive studies of the children in the Zamfara area, because little is known about the impact of such high blood lead levels in children who survived the earlier disaster.

Conclusion

The results of this study demonstrate that elevated BLLs are widespread in Zamfara, and are higher among children living in compounds with members who processed lead-containing gold ore. The proportion of children with BLLs $\geq 45 \mu\text{g/dL}$ appears lower at the statewide level than previously reported for highly contaminated villages. Although elevated BLLs $<45 \mu\text{g/dL}$ are not associated with mortality, they can have life-long health impacts. These children, and those who continue to be exposed, can suffer intelligence loss, learning and behavioral disorders, anemia, reduced stature, hearing loss, peripheral neuropathy, hypertension, and reproductive and renal effects. The societal and economic effects of

aggregate intelligence loss in areas with widespread lead exposures can be substantial. Continued remediation, education, BLL surveillance, emission controls, and occupational hygiene measures are needed to prevent childhood lead exposures. The lead poisoning deaths in May 2015 of 28 children related to ore processing in Zamfara's neighbor state of Niger demonstrate that these measures are needed throughout the region.²⁸

Abbreviations

β	Standardized coefficient
BLL	Blood lead level
CI	Confidence intervals
GM	Geometric mean
ICPMS	Inductively coupled plasma mass spectrometry
LGA	Local government area
US CDC	US Centers for Disease Control and Prevention
USEPA	US Environmental Protection Agency

References

1. Dooyema CA, Neri A, Lo YC, Durant J, Dargan PI, Swarthout T, Biya O, Gidado SO, Haladu S, Sani-Gwarzo N, Nguku PM, Akpan H, Idris S, Bashir AM, Brown MJ. Outbreak of fatal childhood lead poisoning related to artisanal gold mining in northwestern Nigeria, 2010. *Environ Health Perspect* [Internet]. 2012 Apr; 120(4):601–7. [cited 2016 Jul 12] Available from: <http://ehp.niehs.nih.gov/1103965/>.
2. Plumlee GS, Durant JT, Morman SA, Neri A, Wolf RE, Dooyema CA, Hageman PL, Lowers HA, Fernette GL, Meeker GP, Benzel WM, Driscoll RL, Berry CJ, Crock JG, Goldstein HL, Adams M, Bartrem CL, Tirima S, Behbod B, von Lindern I, Brown MJ. Linking geological and health sciences to assess childhood lead poisoning from artisanal gold mining in Nigeria. *Environ Health Perspect* [Internet]. 2013 Jun; 121(6):744–50. [cited 2016 Jul 12] Available from: <http://ehp.niehs.nih.gov/1206051/>.
3. von Lindern, IH., von Braun, MC., Tirima, S., Bartrem, C. Zamfara, Nigeria lead poisoning epidemic emergency response: May 2010 – March 2011 [Internet]. Moscow, Idaho: TerraGraphics Environmental Engineering, Inc.; 2011. p. 126 Available from: http://www.terragraphics.com/Docs/Zamfara_Emergency_Response_UNICEF_Final_Report.pdf [cited 2016 Jul 12]
4. AO, Ashagre T, Ayela A, Chukwumalu K, Criado-Perez A, Gomez-Restrepo C, Meredith C, Neri A, Stellmach D, Sani-Gwarzo N, Nasidi A, Shanks L, Dargan PI. Association of blood lead level with neurological features in 972 children affected by an acute severe lead poisoning outbreak in Zamfara State, northern Nigeria. *PLoS One* [Internet]. 2014 Apr 16.9(4):e93716. [cited 2016 Jul 12] Available from: <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0093716>.
5. Lo YC, Dooyema CA, Neri A, Durant J, Jefferies T, Medina-Marino A, de Ravello L, Thoroughman D, Davis L, Dankoli RS, Samson MY, Ibrahim LM, Okechukwu O, Umar-Tsafe NT, Dama AH, Brown MJ. Childhood lead poisoning associated with gold ore processing: a village-level investigation-Zamfara State, Nigeria, October-November 2010. *Environ Health Perspect* [Internet]. 2012 Oct; 120(10):1450–5. [cited 2016 Jul 12] Available from: <http://ehp.niehs.nih.gov/1104793/>.
6. Sanders T, Liu Y, Buchner V, Tchounwou PB. Neurotoxic effects and biomarkers of lead exposure: a review. *Rev Environ Health* [Internet]. 2009 Jan-Mar;24(1):15–45. [cited 2016 Jul 12] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2858639/>.

7. Global health risks: mortality and burden of disease attributable to selected major risks [Internet]. Geneva, Switzerland: World Health Organization; 2009. p. 70 Available from: http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf [cited 2016 Jul 12]
8. Lidsky TI, Schneider JS. Lead neurotoxicity in children: basic mechanisms and clinical correlates. *Brain* [Internet]. 2003 Jan; 126(Pt 1):5–19. [cited 2016 Jul 12] Available from: <http://brain.oxfordjournals.org/content/126/1/5.long>.
9. Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *N Engl J Med* [Internet]. 2003 Apr 17; 348(16):1517–26. [cited 2016 Jul 12] Available from: <http://www.nejm.org/doi/full/10.1056/NEJMoa022848#t=articleTop>.
10. Needleman H. Lead poisoning. *Annu Rev Med* [Internet]. 2004; 55:209–22. [cited 2016 Jul 12] Available from: <http://www.annualreviews.org/doi/pdf/10.1146/annurev.med.55.091902.103653> Subscription required to view.
11. Jusko TA, Henderson CR, Lanphear BP, Cory-Selecta DA, Parsons JPG, Canfield RL. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. *Environ Health Perspex* [Internet]. 2008 Feb; 116(2):243–8. [cited 2016 Jul 12] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2235210/>.
12. Low level lead exposure harms children: a renewed call for primary prevention [Internet]. Atlanta, Georgia: US Centers for Disease Control and Prevention; 2012 Jan 4. p. 65 Available from: http://www.cdc.gov/nceh/lead/acclpp/final_document_030712.pdf [cited 2016 Jul 12]
13. Meyer PA, Brown MY, Falk H. Global approach to reducing lead exposure and poisoning. *Mutate Res* [Internet]. 2008 Jul-Aug; 659(1–2):166–75. [cited 2016 Jul 12] Available from: <http://www.sciencedirect.com/science/article/pii/S1383574208000367> Subscription required to view.
14. Mercury use in artisanal and small scale gold mining [Internet]. Nairobi, Kenya: United Nations Environment Programme; 2008. p. 20 Available from: http://www.unep.org/hazardoussubstances/Portals/9/Mercury/AwarenessPack/English/UNEP_Mod3_UK_Web.pdf [cited 2016 Jul 12]
15. Bashir M, Umar-Tsafe N, Getso K, Kaita IM, Nasidi A, Sani-Gwarzo N, Nguku P, Davis L, Brown MJ. Assessment of blood lead levels among children aged 5 years - Zamfara State, Nigeria, June–July 2012. *Morb Mortal Wkly Rep* [Internet]. 2014 Apr 18; 63(15):325–7. [cited 2016 Jul 12] Available from: <https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6315a2.htm>.
16. Horowitz AJ, Elrick KA, Smith JJ. Estimating suspended sediment and trace element fluxes in large river basins: methodological considerations as applied to the NASQAN programme. *Hydrol Process* [Internet]. 2001 May; 15(7):1107–32. [cited 2016 Jul 12] Available from: <http://onlinelibrary.wiley.com/doi/10.1002/hyp.206/abstract> Subscription required to view.
17. Horowitz AJ, Stephens VC, Elrick KA, Smith JJ. Concentrations and annual fluxes of sediment-associated chemical constituents from conterminous US coastal rivers using bed sediment data. *Hydrol Process* [Internet]. 2012 Mar 30; 26(7):1090–114. [cited 2016 Jul 12] Available from: <http://onlinelibrary.wiley.com/doi/10.1002/hyp.8437/abstract> Subscription required to view.
18. Rothman KJ, Gallacher JE, Hatch EE. Why representativeness should be avoided. *Int J Epidemiol* [Internet]. 2013 Aug; 42(4):1012–4. [cited 2016 Jul 12] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3888189/>.
19. Elwood JM. Commentary: on representativeness. *Int J Epidemiol* [Internet]. 2013 Aug; 42(4):1014–5. [cited 2016 Jul 12] Available from: <http://ije.oxfordjournals.org/content/42/4/1014.long>.
20. Mirage J, Afeiche M, Linder A, Arowolo T, Ana G, Sridhar MK, Oloruntoba EO, Obi E, Ebenebe JC, Orisakwe OE, Adesina A. Lead poisoning associated with malaria in children of urban areas of Nigeria. *Int J Hyg Environ Health* [Internet]. 2008 Oct; 211(5–6):591–605. [cited 2016 Jul 12] Available from: [http://linkinghub.elsevier.com/retrieve/pii/S1438-4639\(08\)00032-1](http://linkinghub.elsevier.com/retrieve/pii/S1438-4639(08)00032-1) Subscription required to view.
21. Wright NJ, Thacher TD, Pfitzner MA, Fischer PR, Pettifor JM. Causes of lead toxicity in a Nigerian city. *Arch Dis Child* [Internet]. 2005 Mar; 90(3):262–6. [cited 2016 Jul 12] Available from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1720320/pdf/v090p00262.pdf>.
22. Llop S, Lopez-Espinosa MJ, Rebagliato M, Ballester F. Gender differences in the neurotoxicity of metals in children. *Toxicology* [Internet]. 2013 Sep 6; 311(1–2):3–12. [cited 2016 Jul 12] Available from: <http://www.sciencedirect.com/science/article/pii/S0300483X13001194> Subscription required to view.

23. Pfitzner MA, Thacher TD, Pettifor JM, Zoakah AI, Lawson JO, Fischer PR. Prevalence of elevated blood lead levels in Nigerian children. *Ambul Child Health* [Internet]. 2000 Jun; 6(2):115–23. [cited 2016 Jul 12] Available from: <http://onlinelibrary.wiley.com/doi/10.1046/j.1467-0658.2000.00069.x/abstract> Subscription required to view.
24. Marques RC, Bernardi JV, Dorea JG, de Fatima R, Moreira M, Malm O. Perinatal multiple exposure to neurotoxic (lead, methylmercury, ethylmercury, and aluminum) substances and neurodevelopment at six and 24 months of age. *Environ Pollut* [Internet]. 2014 Apr. 187:130–5. [cited 2016 Jul 12] Available from: <http://www.sciencedirect.com/science/article/pii/S0269749114000104> Subscription required to view.
25. Nriagu JO. Toxic metal pollution in Africa. *Sci Total Environ* [Internet]. 1992 Jun 30. 121:1–37. [cited 2016 Jul 12] Available from: <http://www.sciencedirect.com/science/article/pii/004896979290304B> Subscription required to view.
26. Khan DA, Ansari WM, Khan FA. Synergistic effects of iron deficiency and lead exposure on blood lead levels in children. *World J Pediatr* [Internet]. 2011 May; 7(2):150–4. [cited 2016 Jul 12] Available from: <http://link.springer.com/article/10.1007%2Fs12519-011-0257-9> Subscription required to view.
27. Attina TM, Trasande L. Economic costs of childhood lead exposure in low- and middle-income countries. *Environ Health Perspect* [Internet]. 2013 Sep; 121(9):1097–102. [cited 2016 Jul 12] Available from: <http://ehp.niehs.nih.gov/1206424/>.
28. Umar-Tsafe NT. Personal communication to Brown MJ. 2015 Sep 21.

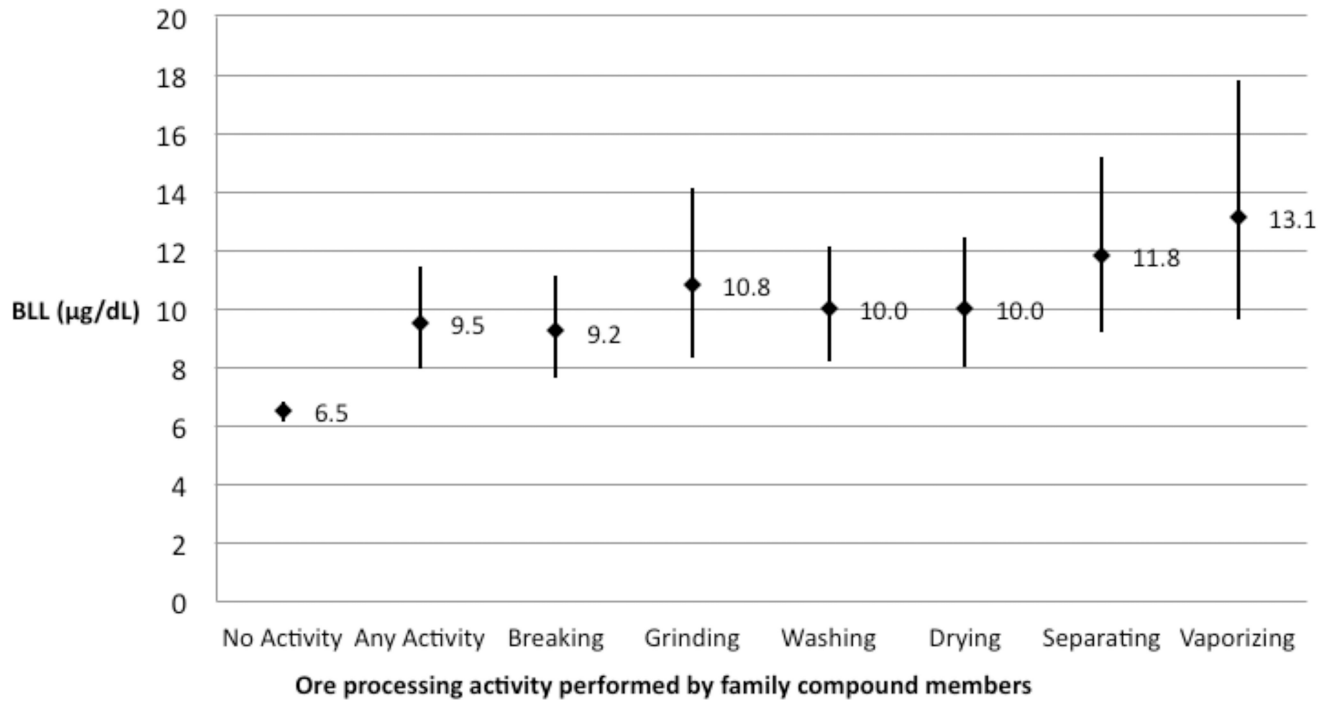


Figure 1. Geometric mean blood lead level (µg/dL) and 95% confidence interval of children by ore processing activity performed by family compound members

Estimated Prevalence of Elevated BLLs in Ore Processing and Non-ore-processing Compounds

Table 1

	Ore Processing Compounds (n=55)			Non-Ore-Processing Compounds (n=273)		
	n	Prevalence	95% CI	n	Prevalence	95% CI
BLL						
BLL 5 µg/dL	45	81.8	69.7, 89.9	188	68.9	63.1, 74.1
BLL 10 µg/dL	21	38.2	26.5, 51.4	61	22.3	17.8, 27.7
BLL 20 µg/dL	11	20.0	11.6, 32.4	5	1.8	0.8, 4.2
BLL 30 µg/dL	7	12.7	6.3, 24.0	3	1.1	0.4, 3.2
BLL 40 µg/dL	4	7.3	2.9, 17.3	1	0.4	0.1, 2.0

Table 2
Geometric Mean Blood Lead Levels ($\mu\text{g/dL}$) in Children Stratified by Ore Processing Activity of Village Residents

Activity	Village Participated in Activity			Village Did Not Participate in Activity		
	n	Geometric Mean	95% CI	n	Geometric Mean	95% CI
Any activity	173	8.1	6.6, 9.6	210	6.1	5.5, 6.6
Breaking	153	8.2	6.5, 9.8	230	6.2	5.7, 6.7
Grinding	111	8.6	6.5, 10.8	272	6.3	5.8, 6.8
Washing	159	8.3	6.7, 9.9	224	6.1	5.6, 6.6
Drying	131	8.8	6.9, 10.7	252	6.1	5.7, 6.6
Separating	83	9.3	6.7, 12.0	300	6.4	5.8, 6.9
Vaporizing	55	11.9	8.6, 15.3	328	6.3	5.7, 6.9