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Running title: Acute Childhood Lead Poisoning in Nigeria

Keywords

Artisanal gold mining, childhood, environmental health, lead poisoning, nervous system

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Abbreviations

ASM: Artisanal and small-scale mining
BLL: Blood lead level
CDC: Centers for Disease Control and Prevention
EPA: U.S. Environmental Protection Agency
MSF: Médecins Sans Frontières
NFELTP: Nigeria Field Epidemiology and Laboratory Training Program
PPB: Parts per billion
PPM: Parts per million
U5MR: Under age 5 mortality rate
USGS: U.S. Geological Survey
XRF: X-Ray fluorescence spectrometer
WHO: World Health Organization

Abstract

Background: In May 2010, a team of national and international organizations was assembled to investigate children's deaths due to lead poisoning in villages in northwestern Nigeria. **Objectives:** To determine the cause of the childhood lead poisoning outbreak, investigate risk factors for child mortality, and identify children aged <5 years in need of emergency chelation therapy for lead poisoning.

Methods: We administered a cross-sectional, door-to-door questionnaire in two affected villages, collected blood from children aged 2–59 months, and soil samples from family compounds. Descriptive and bivariate analyses were performed with survey, blood-lead, and environmental data. Multivariate logistic regression techniques were used to determine risk factors for childhood mortality.

Results: We surveyed 119 family compounds. One hundred eighteen of 463 (25%) children aged <5 years had died in the last year. We tested 59% (204/345) of children, aged <5 years, and all were lead poisoned (≥10 µg/dL); 97% (198/204) of children had blood-lead levels ≥45 µg/dL, the threshold for initiating chelation therapy. Gold ore was processed inside two-thirds of the family compounds surveyed. In multivariate modeling significant risk factors for death in the previous year from suspected lead poisoning included: the child's age, the mother performing ore-processing activities, community well as primary water source, and the soil-lead concentration in the compound.

Conclusion: The high levels of environmental contamination, percentage of children aged <5 years with elevated blood-lead levels (97%, >45 µg/dL), and incidence of convulsions among children prior to death (82%) suggest that most of the recent childhood deaths in the two surveyed villages were caused by acute lead poisoning from gold ore-processing activities. Control measures included environmental remediation, chelation therapy, public health education, and control of mining activities.

Introduction

Childhood lead exposure results in lower intelligence and behavior problems, and negatively affects multiple body systems (Bellinger 2004; Canfield et al. 2003; Lamphear et al. 2005; Mendelsohn et al. 1998; Tellez-Rojo et al. 2006). Young children are particularly susceptible to lead exposure due to behavioral factors such as frequent hand-to-mouth activities, and biological factors including greater gastrointestinal absorption and developing neurological systems (Bellinger 2004; Henretig 2006; Lidsky and Schneider 2003). Encephalopathy typically occurs with blood-lead levels (BLLs) ≥100 µg/dL, but can occur with BLLs as low as 70 µg/dL (Henretig 2006; U.S. Centers for Disease Control and Prevention [CDC] 2002). Symptoms of acute lead encephalopathy include vomiting, changes in behavior, ataxia, convulsions, and coma (CDC 2002; Henretig 2006). Currently, childhood lead poisoning resulting in encephalopathy and death in developed countries is rarely reported, and the last documented child-fatality from lead poisoning in the United States (U.S.) was in 2006 (Berg et al. 2006). However, hundreds of U.S. pediatric acute lead encephalopathy deaths were recorded in the first half of the 20th century. For example, 202 deaths from childhood lead poisoning were recorded in U.S. cities from 1931 and 1940, with 25% occurring in Baltimore, Maryland (McDonald and Kaplan, 1942). Most lead poisoning in this period occurred in urban settings from deteriorating lead housepaint and lead painted cribs. Lead poisoned children in this era frequently had high BLLs. One study evaluating lead poisoning in 293 children from 1931—1970 found the mean BLL for children with mild, severe, and fatal acute lead encephalopathy to be 328 µg/dL, 336 µg/dL, and 327 µg/dL respectively (National Research Council 1972).

Sources of global lead poisoning include lead mining and smelting, battery recycling, leaded gasoline, paint, traditional medicines, toys, and discarded electronic devices (Meyer et al. 2008).

Lead poisoning is of specific concern for children in developing countries (Falk 2003). The World Health Organization (WHO) estimates that lead poisoning causes 0.6% of the global burden of disease and contributes to approximately 600,000 cases of intellectual disability in children annually (World Health Organization [WHO] 2010). During the past 20 years, moderate-to-high elevations of BLLs have been documented globally in clusters of children living in mining and smelting communities or areas where lead batteries are reclaimed (Brown et al. 2009; Garcia Vargas et al. 2001; Kaul et al. 1999; Lalor et al. 2006). However, only one report found in recent literature documents fatalities from childhood lead poisoning; in 2008 in Senegal, informal battery recycling was determined to be the likely cause of death of 18 children (Haefliger et al. 2009).

Description of the outbreak

During meningitis surveillance activities conducted February–April 2010, Médecins Sans Frontières (MSF) and local Nigerian public health officials observed higher-than-expected numbers of childhood illnesses and deaths in four villages in rural northwestern Nigeria. Most of the illnesses and deaths occurred in children aged <5 years. Symptoms included vomiting, abdominal pain, headache, and convulsions; ill children failed to respond to malaria treatment and empiric antibiotics. MSF and State Ministry of Health staff noted that all four villages participated in artisanal gold ore-processing activities, and heavy-metal poisoning was considered as a potential source of the illnesses. Venous blood samples, analyzed in Europe, from eight symptomatic children from an affected village indicated BLLs between 168–370 µg/dL. These concentrations exceed the United States Centers for Disease Control and Prevention (CDC) level of concern of 10 µg/dL and are well-known to be fatal (CDC 2002).

MSF notified Nigerian authorities of the results and a team of international experts was mobilized to assist in the investigation and response to this outbreak.

This report describes the response and investigation of an outbreak of childhood lead poisoning with child mortality unprecedented in modern times. On May 8, 2010, the Nigerian Federal Ministry of Health assembled representatives from federal, state, and international organizations, including the Nigeria Field Epidemiology and Laboratory Training Program (NFELTP), CDC, and WHO, to join in ongoing MSF efforts to investigate and respond to the outbreak. The purpose of this emergency response was to determine the cause and contributing factors to the outbreak, and identify and prioritize children aged <5 years with lead poisoning for emergency chelation therapy. The urgent nature of the response and limited resources necessitated quick investigation and intervention to stop the increasing rate of child fatalities in these villages. Although there were reports of adults in these villages with symptoms of lead poisoning, initial efforts focused almost entirely on children aged <5 years. Young children are most susceptible to lead poisoning and most of the fatalities in MSF clinics were among children aged <5 years. Investigators hypothesized the outbreak of lead poisoning was associated with artisanal gold ore-processing activities. Investigators used a three-pronged approach to investigate the outbreak that included a door-to-door survey, blood lead sampling, and environmental testing.

Methods

Cross-sectional survey

Stakeholders, including Nigerian state health officials and MSF, recommended the two most affected villages (A and B) for participation. The investigation protocol was reviewed and

approved by both the Nigerian government and the CDC. The investigation was conducted in accordance with the Declaration of Helsinki developed by the World Medical Association (World Medical Association 2008).

After obtaining consent of village leaders, investigators conducted a cross-sectional, door-to-door survey to interview parents, sample blood from children aged <5 years, and collect soil from households from May 23–June 4, 2010. Informed consent to administer the survey, draw blood, and collect environmental samples was obtained from the head of the household prior to survey initiation. Local health professionals and translators were trained in a four-hour session on survey administration, venous blood drawing, and environmental sampling.

Each village consisted of numerous family dwellings separated by low walls. Investigators defined a "compound" as several multigenerational and multifamily dwellings enclosed by a common wall. All compounds in Village A were eligible to participate in the survey. Because of time and logistical constraints, only compounds in the central area of Village B were eligible to participate. The central area of Village B included most of the compounds and common gathering places such as mosques, markets, and the head of the village's residence.

The survey collected information about children aged <5 years in each compound, including the number living in the compound, the number with a history of convulsions, the number who had died in the last 12 months, and their approximate date of death.

Prior to the investigation the team heard that artisanal gold ore-processing activities were occurring in both villages. These ore-processing activities included: 1) breaking rocks into small gravel-sized pieces (breaking); 2) grinding rocks into a fine powder with a flour mill or mortar and pestle (grinding); 3) washing ground ore powder with water to separate gold particles (washing); 4) drying ground ore after washing (drying); 5) using liquid mercury to amalgamate gold flakes (separating); and 6) using heat to vaporize mercury from the gold mixture after amalgamation (melting). Both men and women in the villages participated in ore-processing. Men typically processed ore in central locations around the village and women processed ore inside family compounds. The survey included questions about gold ore-processing activities inside and outside the compound, household member and maternal processing activities, history of animal deaths within the compound, and the family's primary water source. Prior to data collection investigators mapped each village and marked the location of each family compound. Global positioning system (GPS) coordinates were taken at the entrance of each compound to facilitate compound identification and follow-up on blood and environmental testing results. At the end of the investigation, MSF staff visited the compounds and provided parents with their child's blood lead test result and information on chelation therapy and the medical management of lead poisoning.

Blood sampling

Venous blood was collected from children aged 2 months to 5 years. Phlebotomists attempted to draw blood from every available child in Village A and from children in every other surveyed compound in Village B due to time constraints. Samples of manufacturer lots of materials used for blood collection were pre-screened by CDC laboratories and determined to be free of lead.

To prevent sample contamination all blood collection supplies were kept in plastic gallon-size storage bags prior to sample collection. In addition, the venipuncture site was thoroughly cleaned with alcohol wipes prior to obtaining the specimen. One 1–3 mL blood sample was collected in a laboratory tube and analyzed for lead using a portable blood-lead analyzer, LeadCare II ® (Magellan Biosciences, Chelmsford, MA, USA) in an uncontaminated area away from the villages. This portable instrument can reliably determine BLLs from 3.3–65 µg/dL with an accuracy level of +/-3 µg/dL (Freeney and Zink 2007).

A second blood sample was collected from every third child and analyzed for lead, total mercury, and manganese, at the CDC National Center for Environmental Health's Inorganic and Radiation Analytical Toxicology laboratory in Atlanta, Georgia. Inductively coupled plasma mass spectroscopy was used to analyze lead and total mercury. Detailed explanations of CDC blood lead and total blood mercury laboratory methods have been published elsewhere (Jones RL et al. 2007; Caldwell et al. 2009). Inductively coupled dynamic reaction cell plasma mass spectrometry was used for whole blood manganese analysis as previously described (Jones DR et al. 2010). The limit of detection for blood lead is 0.25 µg/dL, total blood mercury 0.33 µg/L and manganese 0.8 µg/L. Precision was evaluated by monitoring the replicate results of internal quality control (QC) materials. QC tests, included at the beginning and end of each analytical run, help ensure the accuracy and precision of the analysis process. The low level QC should be in the low-normal range for blood levels in the U.S. population and the high level QC should be less than those found in the high normal population range of the U.S. population. The population ranges are taken from the Fourth National Report on Human Exposure in Environmental Chemicals (CDC 2010). The low level QC for mercury had an interday coefficient of variation

(CV) of 15.1 at 0.516 µg/L and a CV of 2.3 at 5.857 µg/L. For lead the low level QC had an interday CV of 1.7 at 2.876 µg/dL and a CV of 1.2 at 12.754 µg/dL. For manganese the low level QC, with a mean of 7.983, has a CV of 4.8% while the high level QC with a mean of 14.929 has a CV of 6.7%. Accuracy for blood lead and mercury was verified by analyzing standard reference material from the National Institute of Standards and Technology (NIST). Blood manganese accuracy was verified by participating in proficiency testing with the Wadsworth Center of New York Trace Elements in Whole Blood Program. The level of concern for blood lead is 10 µg/dL (CDC 2002). Although there is no blood level of concern for metallic mercury exposure, long-term effects, largely neurological, have been noted at total blood mercury levels <1.0 µg/L (ATSDR 1999). Investigators used the reference range of 7.7-12.1 µg/L from Tietz's *Textbook of Clinical Chemistry* (1999) for blood manganese (Milne 1999). Although some heavy metals, such as mercury, are better evaluated using urine biomarkers, logistical constraints precluded collection of urine specimens.

Environmental sampling

Places where children ate or slept, which were identified by the eldest mother in each compound, were targeted for environmental sampling in each surveyed compound. Samples of soil were swept, placed in a plastic bag, and analyzed for lead content using a portable, hand-held x-ray fluorescence spectrometer (XRF) (Innov-XSystems, Woburn, MA, USA and Thermo-Scientific Niton, Billerica, MA) in an uncontaminated area away from the villages. A certified industrial hygienist or environmental engineer also assessed each village and took XRF readings throughout the villages using U.S. Environmental Protection Agency (EPA) method 6200 to determine areas of high contamination (EPA 2007). Inside the villages priority for XRF

assessment and analysis was given to areas potentially affected by ore processing. The limit of detection for lead by XRF was approximately 40 ppm while the upper reporting limit was 100,000 ppm. Samples of processed ore were also collected and analyzed for lead using XRF. After XRF analysis, a subset of soil and ore samples were sent to the U.S. Geological Survey (USGS) for further analysis using inductively coupled plasma mass spectroscopy to verify XRF field results and to perform mineralogy analysis to determine the chemical composition of the samples.

The U.S. Environmental Protection Agency's (EPA) Toxic Substance Control Act defines a soil-lead hazard as bare soil containing total lead ≥400 parts per million (ppm) in a child's play area or 1,200 ppm in bare soil in other parts of a yard where a child lives (U.S. Environmental Protection Agency [EPA] 2008). EPA standards were used to categorize XRF soil-lead results from surveyed compounds into three groups: ≤400 ppm, 401–1200 ppm, and >1200 ppm. A limited number of water samples were collected by a certified industrial hygienist. Water was drawn up in a bucket and the sampling bottle was immediately submerged to obtain the sample. Water samples were stabilized with 1 milliliter of nitric acid. Unfortunately, no field blanks were taken. Water samples were sent to a commercial environmental testing laboratory in the U.S. and analyzed using the EPA method 200.8 for lead, arsenic, and manganese. The EPA action level of 15 parts per billion for drinking water was the reference standard for water results (EPA 1991).

Statistical analysis

Data from the household survey, blood-lead results, and environmental sample results were entered into Epi-Info version 3.5.1 (CDC, Atlanta, GA, USA). Statistical analyses were

performed using SAS software (SAS Institute Inc, Cary, NC, USA). Univariate analyses, including calculation of daily and overall under age 5 mortality rates (U5MR) were performed. The daily U5MR was calculated by taking the number of deaths per 10,000 children per day for the six month period of December 2009—May 2010. ,. This rate was compared to the United Nations High Commission on Refugees (UNHCR) threshold of > 2.0/10,000/day, which indicates deteriorating conditions in a refugee relief situation (UNHCR 2007). To calculate the overall U5MR, the number of deaths was divided by the number of children alive in the compounds in the 12 month period of May 2009—May 2010 and multiplied by 1,000. This was compared to the U5MR per 1,000 live births for this region of Northwestern Nigeria (217/1,000 live births) as a way to crudely estimate excess mortality in the villages (NPC 2009).

Bivariate and multivariate logistic regression was used to identify risk factors for child mortality from suspected lead poisoning. Using this outcome compounds without any children aged <5 years were excluded from bivariate and multivariate analyses. Risk factors were grouped into demographic, ore processing, and other environmental risk factors. Demographic risk factors included age, gender, and village of residence. Ore-processing risk factors included whether members of the compound or a child's mother participated in any of the six ore-processing activities. Investigators focused on maternal risk factors because children age <5 years in this population typically spend the majority of time with their mothers in the compound. Environmental risk factors included proximity to ore-grinding activities, presence of ground material inside the compound, soil-lead concentrations within the compound, primary water source, and history of animal death (as a proxy for environmental contamination) within the compound. The outcome of interest was a child's death. Multicollinearity, inter-variable

correlation, the Hosmer-Lemeshow goodness-of-fit assessment, and the effect of each risk factor on the outcome were considered. Variables from bivariate analysis with a p-value <0.1 were tested in the model using backward selection. Variables with a p-value of <0.05 remained in the final model.

Results

Survey results

All 54 compounds identified in Village A and 65 of 72 (90%) compounds in the central area of Village B participated in the survey (n = 119). Responses from one compound in Village A were excluded because data were incomplete. A total of 463 children aged <5 years living in the sampled areas of the villages from May 2009–May 2010 were included in this analysis. Data from all 118 surveyed compounds was used to calculate descriptive statistics regarding village demographics and ore-processing activities. To calculate bivariate and multivariate statistics only data from the 110 surveyed compounds with children aged <5 years was used.

The two villages were similar demographically (Table 1). An average of 4.2 children aged <5 years lived in each compound. From May 2009–May 2010, 118 of 463 (25%) children aged <5 years in the surveyed compounds were reported to have died and 82% (97/118) were reported to have had convulsions prior to death. A history of convulsions was reported in 17% (58 of 345) of the surviving children. Sixty-two (53%) of 110 surveyed compounds with children aged <5 years reported that from one to eight children aged <5 years had died from May 2009–May 2010. Eighty-two percent of deaths occurred from December 2009–May 2010 making the daily U5MR

12.1/10,000 children age <5 years/day for the period in the villages. The approximate overall U5MR for the one year period (May 2009–May 2010) was 255/1,000 live births.

Overall, 84 of 118 (71%) compounds reported processing gold ore within their compound (Table 2). Compounds participating in ore processing averaged four different processing activities. Ore drying was performed more frequently in compounds in Village A than in Village B (69% vs. 49% p = 0.002). Only 25 compounds reported grinding activities within their compound (Table 2), but 54 of 118 (46%) reported grinding activities nearby, and 32 of 118 (27%) compounds reported having ground ore present in their compound. Fifty-six of the 84 (67%) compounds processing ore started doing so in the 12 months preceding the investigation (May 2009–May 2010), however some compounds (33%) reported processing for as long as 18 years. Compounds in Village B were more likely to use a well inside their compound than those in Village A, who were more likely to use a community well as their main water source (94% vs. 60%, p <0.001).

Blood sampling

Venous blood-lead samples were obtained from 204 of 345 (59%) children aged <5 years. All blood samples indicated lead poisoning (BLL ≥10 µg/dL). For 198 of 204 (97%) children, BLLs were ≥45 µg/dL, the CDC-recommended threshold for initiating chelation therapy (CDC 2002). Eighty-five percent (173/204) of blood samples exceeded the maximum detection limit of the LeadCare II instrument (65 µg/dL).

Eighty-six blood samples were sent to CDC laboratories for blood lead, mercury, and manganese testing (Table 3). The mean blood-lead concentration for children in Village A was 153.3 µg/dL

(range 55.9–331.0 µg/dL) and 107.5 µg/dL (range 36.5–445.0 µg/dL) for children in Village B. The mean total blood mercury level for children in Village A was 2.4 µg/L (range 0.3–6.6 µg/L) and 1.4 µg/L (range 0.4–6.5 µg/L) in Village B. For comparison, the geometric mean value for total blood mercury reported for children ages 1–5 in the U.S. Fourth National Report on Human Exposure to Environmental Chemicals was 0.33 µg/L (95% CI 0.29–0.37) with the 95th percentile measuring 1.8 µg/L (95% CI 1.3–2.5) (CDC 2010). Mean blood manganese levels were 14.0 (range 4.0–39.0 µg/L) and 20.9 (range 4.9–40.8 µg/L) in Villages A and B respectively, and 66% (57/86) of samples were above the reference range of 7.7-12.1 µg/L for manganese.

Environmental sampling

Soil obtained from 116 of 118 compounds was analyzed in the field. Soil-lead concentrations ranged from 45 ppm to >100,000 ppm; 85% of samples exceeded the EPA threshold of 400 ppm. The average soil-lead concentration from household samples analyzed by XRF was 7,959 ppm (range 421–>100,000 ppm) in Village A and 3,298 ppm (range 45–>100,000 ppm) in Village B. Four water samples were collected and sent to a commercial environmental testing laboratory in the U.S. for analysis. Community wells in Villages A and B had 520 ppb and 1,300 ppb of lead respectively exceeding the EPA action level of 15 ppb of lead in drinking water. Two convenience water samples, one from pond and one from a private well in Village B were also tested and had lead concentrations of 250 and 37 ppb lead respectively. The private well was selected because no ore-processing impact was suspected, and the pond was selected because it had been used for sluicinggold ore and village livestock were observed using this water source. The lead concentrations of 5 ore samples from various stages of ore processing were measured at

USGS laboratories were 331, 9,150, 13,700 112,000, and 175,000 ppm. This confirmed that some gold ore processed by the villagers was rich in lead content.

Bivariate and multivariate analyses

Table 4 summarizes the results of bivariate analyses. Children who died were more likely than children who survived to have had a mother who participated in ore-processing activities (OR = 1.9, 95% CI 1.2, 2.9). Having a mother who participated in grinding, washing, drying, separating, or melting activities was significantly associated with a child's death ($p < 0.05$). Children who died were more likely than children who survived to have ground ore present in the compound (OR = 2.0, 95% CI 1.3, 3.2). Proximity to village grinding activities was not significantly associated with mortality in bivariate analysis.

Environmental and demographic variables tested in the logistic regression model are illustrated in Table 4. Although we collected data regarding whether any household member performed ore-processing activities as well as if the child's mother performed ore-processing activities the two responses were not mutually exclusive and were highly correlated in bivariate analysis., As previous studies have found that a child's BLL is most related to the mother's activities and BLL (CDC 2002) we only evaluated risk factors related to the mother's activities in the logistic regression model. Child mortality was significantly related to five risk factors in the final model: the child's age, whether the mother performed ore breaking or washing activities, whether a community well was used as a primary water source, and the soil-lead level of the child's compound (Table 5). No potential interaction terms were included in the final model as they did not improve the model's explanatory power.

After controlling for the maternal ore-processing activities of breaking and washing, and environmental factors including water source and soil-lead level within the compound, children ≤24 months were 2.7 times as likely to have died as children 25–49 months of age (95% CI 1.6, 4.4). In addition, children living in a compound with a soil-lead level of >1200 ppm were 3.6 times as likely to have died as children in a compound with a soil level of <400 ppm (95% CI 1.5, 8.5) and children whose primary water source was a community well outside their compound were 3.7 times as likely to have died as children whose primary source was a well inside the compound (95% CI 2.0, 6.8).

Discussion

The United Nations High Commission on Refugees (UNHCR) considers a daily U5MR of >2.0/10,000/day a key indicator that conditions in a refugee situation are deteriorating (UNHCR 2007). The daily U5MR of this outbreak was more than six times the UNHCR's benchmark and well above the overall U5MR in the region (217/1000) and the nation (157/1000). Children aged ≤24 months had 2.6 times the odds of death compared to older children, consistent with previous research that indicates that younger children are at greater risk for lead poisoning.

This investigation was subject to at least four limitations. First, post-mortem evaluations and blood-lead samples were not available for deceased children. Thus, not all of these deaths can be reliably attributed to lead poisoning and BLLs in children who died cannot be compared to those who lived. Second, we were unable to determine whether other unmeasured cultural or nutritional factors exist that may explain some of the high mortality rates. Third, the survey and

environmental sampling were conducted by local staff with limited training and no field blanks were obtained to validate sampling methods. Variation could have occurred in the way survey questions were asked or how environmental samples were collected. Finally, we were only able to determine an approximate U5MR for the villages prior to the outbreak based on regional data.

Despite these limitations, it is reasonable and prudent to conclude that most of the recent childhood deaths in these villages were caused by acute lead poisoning and take steps to stop the exposure. This conclusion is based on high environmental lead concentrations, the percentage of children with elevated BLLs (97% >45 µg/dL), the high incidence of convulsions before death (82%), and the significant reduction in mortality once treatment for lead poisoning was initiated and children were removed from the source of contamination. Although mortality was reduced it will take some time to fully characterize the extent of neurological damage that has occurred in these children. In this investigation 85% of surveyed compounds had soil-lead levels that exceeded 400 ppm and some had levels 250 times the EPA threshold. Most compounds in this rural, impoverished setting were constructed of dirt walls and floors. Children came in direct contact large amounts of soil through daily activities and this provides a constant and direct route of exposure for children. We also demonstrated a relationship of increasing exposure related to increasing odds of dying — as children whose compounds contained ≤1200 ppm lead were less likely to die than children whose compounds contained >1200 ppm lead. Moreover, environmental contamination may not be limited to soil, and contamination of water sources is also possible. In our multivariate model, the odds of death was 3.7 times greater for children who had a community well as their main source of water compared to children whose main water source was a private well in their compound. Four water samples, including two from

community wells, had lead concentrations that greatly exceeded the US EPA's action level for drinking water of 15 ppb.

Most of the compounds engaged in ore-processing activities (67%) began them in the 12 months preceding this investigation, when child mortality also increased. Although not captured in our survey, anecdotal evidence indicates that the increase in child mortality was related to the increased use of flour-grinding machines located within the villages to grind ore. Prior to November 2009 at least one grinder was reported by villagers to be in each of the surveyed villages, but after November 2009 villagers reported that there were up to 10 grinding machines in each village. The increase in the number of grinding machines would likely amplify the amount of ground ore around the village and in individual compounds, contributing to widespread contamination. The increase in lead contamination and continued use of mercury also warrant investigation into the health effects from multiple metals in children and adults. Further research on multiple metal exposures in this population is needed.

During and after this investigation, accounts of similar ore-processing activities and increased rates of child mortality were reported in other nearby villages. Characterizing the full extent of the outbreak remains an urgent and ongoing matter. As more affected villages are identified, medical treatment and environmental remediation will require trained personnel and increased funding. Initiating medical treatment in the absence of environmental remediation is not only ineffective in reducing children's lead exposure and body burden, but may cause more harm than good (Chisholm 1992). CDC recommendations for follow-up and rehabilitation of lead-poisoned children in the U.S. prioritize control or elimination of sources of exposure as the most essential

intervention, and great progress has been made to reduce lead exposure (CDC 2002). In these villages in Nigeria, contamination of water systems, crops, and animals, as well as the risk of recurrent contamination from villages who temporarily cease and then resume ore processing, remain enormous issues to be addressed. Community-based education campaigns are ongoing. Messages include the need for blood-lead testing and environmental remediation and the dangers of ore-processing, particularly within the confines of the village.

Beginning in June 2010, chelation therapy was provided free of charge in hospitals supported by MSF and Nigerian staff for all children aged <5 in the surveyed villages. Hundreds of children have undergone chelation therapy and unpublished data from MSF clinics in these villages indicate that child mortality has been reduced from ~43% immediately prior to the investigation to <1% post investigation. Each compound in the villages has been remediated to decrease the risk for lead poisoning. Environmental remediation efforts spearheaded by TerraGraphics and the Blacksmith Institute with the support of Nigerian state and local officials began in early June 2010. TerraGraphics/Blacksmith Institute provided technical assistance and training for the remediation and the manual labor was largely performed by Nigerian state, local, and village members. Remediation included removing contaminated surface soil and replacing it with clean uncontaminated soil and disposing of contaminated soil in secure landfills. Remediation drastically brought down soil-lead levels inside compounds and reduced child's exposure to lead. Remediation efforts also included education messages emphasizing the importance of prohibiting ore-processing activities and ore-processing materials inside the compound. However, post-remediation follow-up is necessary to determine the long-term efficacy of the remediation.

Unfortunately, the type of artisanal and small-scale mining (ASM) demonstrated in this article is occurring on a global scale. According to the Global Mercury Project (GMP), an initiative of the United Nations, ASM occurs in over 55 countries and 10-15 million miners work globally, primarily in Africa, Asia, and South America. An estimated 100 million people globally rely directly or indirectly on ASM for their livelihood (Spiegal and Veiga, 2007). Participation in mining for many individuals is driven by poverty and a lack of economic opportunities, especially in rural communities, as seen in this outbreak. ASM is the source of 20-30% of the world's gold (Spiegal and Veiga, 2007). The price of gold continues to rise on the global market and during the last decade gold has increased 360% (Swenson et al. 2011).

ASM has long been recognized as having negative health and environmental impacts. Mercury exposure in mining communities has been well-established in the literature in places such as Brazil, Indonesia, Zimbabwe, and Ghana, (GMP 2006; Kwaansa-Ansah, Basu, Nriagu, 2010) but much less is known about other toxic chemicals, such as lead. Findings from this investigation should be included into existing global initiatives to address other heavy metals in addition to mercury.

Conclusion

In this article we describe an outbreak of childhood lead poisoning related to artisanal mining that resulted in child mortality rates which were unprecedented in modern times. To our knowledge this is the first documentation of an outbreak of childhood lead poisoning associated with artisanal gold mining. Twenty five percent of children aged <5 years in the two villages died from May 2009–May 2010 and 97% of surviving children had blood lead levels that

required chelation therapy. Extensive environmental contamination was found in both of the villages and inside individual family compounds. The response to this outbreak required extensive coordination with federal and state ministries, international non-governmental organizations, international organizations, and U.S. government agencies.

The need for technological assistance and transfer in countries with developing economies and healthcare infrastructure is critical. In resource-limited areas such as northern Nigeria, utilization of natural resources is an economic necessity. However, mining has exposed the communities to high lead concentrations with devastating effects on the population. Affected children in these villages may suffer long-term consequences, such as intellectual deficits and blindness. Safer mining practices, including moving the processing away from village, techniques to reduce dust generation, and basic hygiene could prevent lead exposure and subsequent lead poisoning in persons participating in ore-processing activities and their families.

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Table 1: Demographics of participating family compounds by village

Demographic	Village A (%) (n = 53)	Village B (%) (n = 65)	Both villages (%) (n = 118)
Mean number of married men per compound	3.1	2.1	2.6
Mean number of mothers per compound	3.7	3.0	3.4
Mean number of children aged <5 years per compound	4.8	3.1	4.2
Total number of children aged <5 years in participating compounds, as of May 2009	259	204	463
Number of children aged <5 years living at time of survey (%)	181 (70)	164 (80)	345 (75)
Number of children aged <5 years who had died within last 12 months before survey (%)	78 (30)	40 (20)	118 (25)
Number compounds with ≥1 pregnant women (%)	26 (49)	24 (37)	50 (42)

n = number of compounds

Table 2: Participation in ore-processing within family compounds by village

Activity	Village A (%) (n = 53)	Village B (%) (n = 65)	Both villages (%) (n = 118)
Participate in ≥1 activities	42 (79)	42 (65)	84 (71)
Rock breaking	29 (55)	39 (60)	68 (58)
Rock grinding	10 (19)	15 (23)	25 (21)
Rock washing	27 (51)	27 (42)	54 (46)
Rock drying	33 (62)*	29 (45)*	62 (53)
Rock separating	22 (42)	30 (46)	52 (44)
Rock melting	22 (42)	32 (49)	54 (46)
Mean number of activities	3.4	4.1	3.8

* Denotes a significant difference (p-value <0.05).

n = number of compounds

Table 3: Blood lead (Pb), manganese (Mn), and mercury (Hg) results by village

Village	n	Mean Pb	Median Pb	Range Pb	Mean Mn	Median Mn	Range Mn	Mean Hg	Median Hg	Range Hg
Village A	44	153.3	143.8	55.9–331	14.0	11.6	4.0–39.0	2.4	2.1	0.3–6.6
Village B	42	107.5	85.7	36.5–445	20.9	20.1	4.9–40.8	1.4	0.99	0.4–6.5

Results are lead µg /dL, manganese and mercury µg /L. Reference range for lead is <10 µg/dL; manganese is 7.7–12.1 µg/L; health effects of Hg can be seen at >1 µg/L.

Table 4: Potential risk factors for child mortality

Potential risk factor	Outcome		Crude Odds Ratio (95% CI)	p-value
	Deceased	Alive		
Maternal Activities				
Breaks ore			1.5 (0.96, 2.2)	0.08
Yes	56	132		
No	62	213		
Grinds ore			2.6 (1.4, 5.0)	0.002
Yes	20	25		
No	98	320		
Washes ore			2.6 (1.4, 4.7)	0.002
Yes	22	28		
No	96	317		
Dries ore			2.9 (1.8, 4.8)	<0.0001
Yes	38	48		
No	80	297		
Separates ore			1.7 (1.0, 3.1)	0.05
Yes	23	42		
No	95	303		
Other environmental risk factors				
Mother performs ≥1 ore-processing activity	74	163	1.9 (1.2, 2.9)	0.0037
Mother performs 0 ore-processing activities	44	182		
Soil-lead level in compound				
<400 ppm	8	53	Ref	Ref
400-1200 ppm	20	111	1.2 (0.5, 2.9)	0.7
>1200 ppm	90	178	3.4 (1.5, 7.4)	0.0026
Dried ore in compound			2.0 (1.3, 3.2)	0.0041
Yes	46	82		
No	72	263		
			3.4 (2.0, 5.7)	<0.0001
Main water source	35	38		
Community well				
Private well	83	307		
History of animal death in compound			2.4 (1.5, 3.8)	0.0003
Yes animal death	89	195		
No animal death	29	150		
Demographic risk factors				
Age Birth-24 mos)	88	181	2.6 (1.6, 4.2)	<0.0001
25-49 mos	30	161		
Village of residence			1.8 (1.1, 2.7)	0.01
Village A	78	181		
Village B	40	164		

Table 5: Significant risk factors retained in final model

Risk factors	Crude Odds Ratio (95% CI)	p-value	Adjusted Odds Ratios (95% CI)
Maternal Activities			
Breaks ore	1.5 (0.96, 2.2)	0.08	1.8 (1.1, 3.0)
Washes ore	2.6 (1.4, 4.7)	0.002	3.4 (1.7, 6.7)
Other environmental risk factors			
Lead level in compound			
<400 ppm	Ref	Ref	Ref
400-1200 ppm	1.2 (0.5, 2.9)	0.7	1.5 (0.6, 3.8)
>1200 ppm	3.4 (1.5, 7.4)	0.0026	3.6 (1.5, 8.5)
Main water source	3.4 (2.0, 5.7)	<0.0001	3.7 (2.0, 6.8)
Demographic risk factors			
Age (birth-24 mos vs. 25-49 mos)	2.6 (1.6, 4.2)	<0.0001	2.6 (1.6, 4.4)