DETERMINANTS AND PREVALENCE OF CHILDHOOD LEAD POISONING IN KAWAYE VILLAGE, ANKA LOCAL GOVERNMENT AREA, ZAMFARA STATE; 2013

BY

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FEBRUARY, 2014

ATTESTATION

I hereby declare that this work is carried out by me under the supervision of Dr S.H. Idris and has not been presented in part or whole to any other institution for the award of degree or diploma nor has it been submitted elsewhere for publication. The works of other investigators used in this research work have been duly acknowledged and referred to accordingly.

Kabiru Ibrahim Getso _____ _____

Signature Date

CERTIFICATION

This is to certify that this thesis work entitled "Determinants and prevalence of childhood lead poisoning in Kawaye village, Anka Local Government Area, Zamfara State; April 2013" by Kabiru Ibrahim Getso under our supervision meets the regulations governing the award of Master of Public Health degree (Field Epidemiology) of Ahmadu Bello University, Zaria and is approved for its contribution to science, knowledge and literary presentation.

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DEDICATION

This piece of work is dedicated to the loving memories of my late sister Rahmatu Ibrahim Getso, my late nephew Al-Amin Ahmed Ibrahim and all the children in Zamfara State who lost their lives or sustained disabilities as a result of the lead poisoning disaster.

It is my fervent hope that this research work (and all future researches arising from it) will help improve the lives of all the children at risk of and/or affected by lead poisoning!

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LIST OF ACRONYMS

ALA	Amino Levulinic Acid
ATSDR	Agency for Toxic Substances and Disease Registry
BAL	British Anti-Lewisite
BLL	Blood Lead Level
BLLs	Blood Lead Levels
CDC	Centers for Disease Control and Prevention
CHEWS	Community Health Extension Workers
CI	Confidence Interval
CPSC	Consumer Product Safety Commission
δ-ALAD	*delta*-Aminolevulinic Acid Dehydratase
DALY	Disability Adjusted Life Years
DMSA	2, 3 Dimercaptosuccinic Acid
DNA	Deoxyribo-Nucleic Acid
EBLLs	Elevated Blood Lead Levels
EDTA	Ethylene Diamine Tetra-Acetic acid
EPA	Environmental Protection Agency
EP/EPP	Erythropoietic (Erythrocyte) Protoporphyrin
ER	Electrokinetic Remediation
ESLL	Elevated Soil Lead Level
IgG	Immunoglobulin G
IgE	Immunoglobulin E
IgM	Immunoglobulin M
IL	Interleukin
IM	Intramuscular(ly)
IQ	Intelligence Quotient

IV	Intravenous(ly)
LGA	Local Government Area
LVH	Left Ventricular Hypertrophy
MCLG	Maximum Contaminant Level Goal
MP's	Malaria Parasites
MNCH	Maternal Newborn and Child Health
NAPEP	National Poverty Eradication Program
NHANES	National Health and Nutrition Examinations Survey
NIS	Nigerian Industrial Standard
NMDA	N-Methyl-D-Aspartic acid
NO	Nitric Oxide
OR	Odds ratio
OSHA	Occupational Safety and Health administration
PHC	Primary Health Center
PEL	Permissible Exposure Limit
PM_{10}	Particulate Matter ≤10 micrometers in diameter
PPE	Personal Protective Equipment
PRINN	Program for the Revival of Routine Immunization in Northern Nigeria
RBCs	Red Blood Cells
SAICM	Strategic Approach to International Chemicals Management
SD	Standard Deviation
SES	Socio-Economic Status
SLL	Soil Lead Level
SMEDAN	Small and Medium Enterprises Development Agency of Nigeria
UN	United Nations
UNEP	United Nations Environment Programme

USA	United States of America
VOCs	Volatile Organic Compounds
WHO	World Health Organization
ZESA	Zamfara Environmental Sanitation Agency
ZPP	Zinc ProtoPorphyrin

SUMMARY

Lead poisoning remains one of the most significant and prevalent diseases of environmental origin globally, accounting for 0.6% of the global burden of diseases. Children under the age of 6 years are especially vulnerable to lead poisoning, which can severely affect mental and physical development. This study was carried out in Kawaye village, Anka Local Government Area of Zamfara State to find out the determinants and prevalence of childhood lead poisoning in Zamfara State and specifically, to determine risk factors and prevalence of childhood lead poisoning and socio-demographic characteristics of children affected by lead poisoning in Kawaye village, Anka Local Government Area of Zamfara State.

In a descriptive cross sectional study design and using simple random sampling technique, 307 eligible children aged 6 years and below were recruited. Data were collected using interviewer-administered semi-structured questionnaire to obtain information on socio-demographics, risk factors for lead poisoning, history of exposure to lead, and signs and symptoms of toxicity. Blood specimens were collected for determination of blood lead levels, anemia and malaria parasitemia. In addition, household soils were tested for presence of lead. Data were entered into and analyzed using Epi info™ 7 software.

The study showed a very high prevalence of lead poisoning of 92.5% in Kawaye village. The mean age of studied children was 38.5 months ± 18.5 SD. Majority of the studied children belonged to age groups 48-59 and ≥ 60 months with slight male predominance. Children most affected by lead poisoning were males (p = 0.455), belonging to the age group 35 months and below (p = 0.422), and whose parents were miners (14.7%) and farmers (37.1%). More than half of the fathers and/or guardians of studied children had informal education (53.7%) with few having neither informal nor formal education (4.2%). In addition, nearly one-third of the fathers and/or guardians lived below poverty line and about the same proportion (30.3%) earned at least the minimum wage set by the Federal Government of Nigeria monthly.

Altogether, 44 of 307 (14.3%) households were found to have soils contaminated with lead of varying degrees. The median household soil lead level was 61 PPM, ranging from non-detectable to 24000 PPM. About 11% of the households had mild-moderate contamination, while 2.9% were severely contaminated with lead. Moreover, about 86% of the studied children had varying degrees of anemia with a mean PCV level of 26.9% (SD ± 5.5). On the other hand, 59% of the children had variable levels of malaria parasitemia. Low levels of education and socioeconomic status of father/guardian, use of kohl eye cosmetic on child, anemia and malaria parasitemia were found to be independent risk factors for childhood lead poisoning.

In conclusion, the study established a very high prevalence of lead poisoning in Kawaye village, with most of the affected children belonging to age group 35 months and below. It is therefore recommended that targeted interventions should be designed to address the identified risk factors in order to control and prevent childhood lead poisoning in Kawaye village and the State at large.

Key words: Lead poisoning, Pattern, Determinants, Zamfara State

CHAPTER 1

INTRODUCTION

1.1. Background

Lead - a heavy metal with symbol *Pb* (from Latin *plumbum)*- is a soft malleable metal that has bluish-white color when freshly cut, but soon tarnishes to dull-grayish color when exposed to air. It has a shiny chrome-silver luster when melted into liquid. It occurs naturally in the earth's crust and is used in building constructions, lead acid batteries, bullets, weights, as part of solders, plumbing materials, fusible alloys and as a radiation shield among others.

Lead poisoning on the other hand, is a medical condition that occurs when lead builds up in the body to at least 5 micrograms per deciliter (µg/dL) of blood, [1] often over a period of months or years. Even small amounts of lead can cause serious health problems. Children under the age of 6 years are especially vulnerable to lead poisoning, which can severely affect mental and physical development. Elevated blood lead levels can trigger serious health conditions, ranging from muscle weakness, gastro-intestinal symptoms, anemia, kidney inflammation, convulsions and brain damage. Lead exposure during childhood can have adverse health effects well into adulthood. For instance, some studies have linked childhood lead poisoning with hypertension and tooth decay later in life. Even low levels of lead are linked to lower intelligence quotient (IQ) scores and behavioral problems. Epidemiologic studies have convincingly demonstrated that low-level lead exposure in children less than 5 years of age (with blood levels less than 5-25µg/dL range) results in deficits of intellectual performance as manifested by lost IQ points. Because IQ is lower for children who have been exposed to lead, it is likely that they will complete lower levels of education and have lower paying jobs. Scientific research has also shown that lead-exposed children and adolescents are more likely to have behavioral problems which may require special education. These

children also often display delinquent behavior, which can lead to arrest and imprisonment.[2] For instance, for every incremental increase of 10µg/dL of lead in the blood, IQ scores decline between 2.5 and 3.0 points.[3] The U.S. Centers for Disease Control and Prevention (CDC) has set the standard elevated blood lead level for adults to be 25µg/dL of the whole blood and for children the level is set at a much lower level of 5µg/dL. It also recommended universal blood lead screening of all children between the ages of six months and five years.[1,4] At very high levels, lead poisoning can be fatal. These secondary impacts can then negatively affect the social and economic prospects of children far into their future.

Once lead enters the body it travels to soft tissues and organs such as liver, kidneys, lungs, brain, heart and spleen. In adults, once lead is absorbed, about 99% of it will be excreted within a couple of weeks via natural excretion. However, in children, much less amount, 32% leaves the child's body within the same period because of its immature systems. Continued exposure may therefore results in the accumulation of the lead within the body. Blood lead levels above 25µg/dL have been shown to lead to anemia, kidney malfunction, osteoporosis, hypertension and lead encephalopathy. While extremely high levels (70µg/dL and above) can cause brain swelling, convulsions and even death.

In two recent studies of heavy metals in the blood of paint factory workers and artisans, including mechanics, welders, spray painters and electricians in Southeastern Nigeria, a BLL of 17.00 ± 4.00µg/dL was found among samples from control subjects who were recruited from secondary schools and 35.00 ± 7.9µg/dL in samples of controls recruited from undergraduate medical students. The values obtained from the occupationally exposed workers in these studies were 59.6 ± 15.9µg/dL and 42.00 ± 4.00µg/dL respectively. These levels for both the controls and the exposed group exceeded the CDC's level of concern.

In another study, the mean BLL of Lagos traffic wardens was 18.1 ± 6.4μg/dL which was significantly higher than the level of 10.2 ± 2.7μg/dL in Ife traffic wardens and those in controls.[5] In a study conducted in Otukpo, south-western Nigeria; the BLL among children <6 years old ranged from 2.1 to 31.8 μg/dl (mean = 9.4 μg/dL; SD = 4.2), with 35% of the children exhibiting a BLL > 10 μg/dL.[6] A study on blood lead and tap water lead levels in Nigeria found mean BLL of 11.4 and 12.3 μg/dL for females and males respectively.[7]

Multiple health effects have been associated with lead exposure including systemic effects such as gastrointestinal effects, raised blood pressure, anemia, nephropathy, effects on vitamin D metabolism, decreased growth, immune system, nervous system and behavioral/cognitive/IQ effects (and, as a result, multiple social effects including increased risk of violence and substance abuse), nerve conductive effects, hearing loss, developmental and reproductive effects, genotoxicity and death from encephalopathy. A study carried out at Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife, Osun State, Nigeria demonstrated an association between the rising incidence of breast cancer among Nigerian women and widespread contamination of the soil and of the water supplies by lead and other industrial metals.[8,9]

Lead has had devastating consequences on the health of the world's children. At high levels of acute exposure, lead attacks the brain and central nervous system to cause coma, convulsions and even death. Lead poisoning is also known to cause mental retardation and behavioral disruption.[10,11] At lower levels of exposure that may not cause obvious symptoms and that previously were considered safe, lead is now known to produce a spectrum of injury that causes loss of cognition, shortening of attention span, alteration of behavior, dyslexia, attention deficit disorder, hypertension, renal impairment, immune-toxicity and toxicity to the reproductive organs.[12] For the most part, these effects are permanent. They are irreversible and untreatable by modern medicine. In a situation of widespread lead exposure, the

economic costs in terms of medical care and diminished opportunity amounted worldwide to hundreds of billions of dollars a year;[13] whereas, lead poisoning can be completely eliminated and prevented.

Lead exposure is estimated to account for 0.6% of the global burden of disease, with the highest burden in developing regions. Childhood lead exposure is estimated to contribute about 600,000 new cases of children with intellectual disabilities every year.[14] In a study to estimate the global burden of disease from environmental lead exposure, it was estimated that mild mental retardation and cardiovascular outcomes resulting from exposure to lead amount to almost 1% of the global burden of disease, with the highest burden in developing regions.[15] When revised estimates of the burden of disease were made in 2004, 16% of all children worldwide were estimated to have levels above 10µg/dL. Of children with elevated levels, an estimated 90% live in low-income regions.[16] Common sources of lead poisoning in developing countries include mining activities, battery recycling, leaded gasoline, lead paint, electronic wastes, traditional medicines and toys.[17]

In United States of America, estimates based on data from 1999 and 2000 indicated that about 2.2% of children aged 1-5 years (about 434,000 children) have Blood Lead Levels (BLL) elevated at or above 10µg/dL. *Healthy people 2010 goal* was established (objective 8-11) which calls for the eradication of lead poisoning as a public health problem by the year 2010 through the elimination of elevated BLLs in children.[18] In a study to assess the blood lead levels in children aged 24 to 36 months in Vancouver in 1995, 8.1% had BLL above 9.94µg/dL while 0.6% had a level higher than 14.9µg/dL.[19] A study conducted in South Africa showed that lead poisoning was estimated to cause 1,428 deaths of all deaths in South Africa in the year 2000. Burden of disease attributed to lead exposure which was dominated by mild mental disability in young children, accounting for 75% of the total 58 939 attributable DALYs.[8]

While the prevalence of lead poisoning is generally on the decline in most developed countries, for instance 1.6% in New York, USA in 2005,[20] the rate is still very high in developing countries especially Sub Saharan Africa. A study in South Africa showed that more than 50% of children in Cape Town, Durban, Johannesburg and probably other cities in the country have blood lead levels greater than 10µg/dL.[21] In a community survey carried out in Jos, Nigeria, 70% of children 6–35 months of age had blood lead concentrations of 10 µg/dL or greater.[22] In November 2007-March 2008, 18 children died in Thiaroye Sur Mer, Senegal, following encephalopathy caused by severe lead intoxication as a result of environmental contamination from informal used lead acid battery (ULAB) recycling. Blood lead levels of 50 children (3 months-19 years old) included in the investigation ranged from 39.8-613.9µg/dl, with a mean (SD) of 129.5 (92.4)µg/dL.[23]

In addition to the specific health and economic impacts on children and their families, lead poisoning results in huge fiscal impacts on the government and donor agencies. Elevated blood lead levels may necessitate need for health care, special education and/or criminal justice. Treatment of a single case of lead poisoning with just a course of chelation agent requires large sum of money; and a case may require up to 3 or more courses. Apart from the huge cost involved in treatment, the children may subsequently require special education.

On the other hand, Zamfara State Government has commenced remediation of contaminated villages. The exercise which commenced in November 2011 was officially flagged off by His Excellency the Executive Governor in December 2011. Ten contaminated compounds and environs have been fully remediated, though the State Government has suspended cleanup process in abid to redo the mapping exercise as a fall out of expert advice from TerraGraphics USA who had identified other pathways of re-contamination into already cleaned compounds. Financing has been strictly by the State Government with a total expenditure of 240 million naira in 2010. In addition, the State Ministry of Mines and Steel Development

has registered about seventeen Artisanal and Small-scale Mining Cooperatives in the State, while a total of 540 mineral titles have been issued in Zamfara State. Treatment of lead poisoned children is mainly provided by Medecins Sans Frontiers with some support from the Federal and State Ministries of Health and the Local Government Authorities. So far, over 2,500 children have been enrolled into chelation therapy out of which 500 have completed treatment.

1.2. Problem Statement

Lead poisoning has been a scourge to human health for millennia, it remains one of the most significant and prevalent diseases of environmental origin globally. Though many countries have initiated programs aimed at lowering the level of lead in the environment, human exposure to lead remains of concern to health care providers and public health officials worldwide. A study carried out to investigate an outbreak of acute lead poisoning among children under the age of 5 years in Zamfara State, Nigeria in 2010 showed that 97% had elevated blood lead levels above 45µg/dL, the CDC-recommended threshold for commencing chelation therapy. Eighty five percent of blood samples exceeded the maximum detection limit of Leadcare II blood lead analyzer (> 65 µg/dL). The study also showed that children are exposed to lead primarily through hand-to-mouth or the inhalation of lead dust; the main sources of lead exposure in the state being the artisanal gold ore mining and processing activities that are prevalent in many villages in the state.[24]

In a separate report from WHO, 200 children from Zamfara State, Nigeria have reportedly died from lead intoxication since March 2010 and an estimated 18,000 children and adults have been affected by widespread lead contamination resulting from the informal extraction of gold from lead-bearing ore. Children's BLLs in two acutely affected Zamfara villages averaged 119 µg/dL (range 33.3 - 445µg/dL), with 118 of the 463 resident children dying

during May 2009-May 2010. Parents reported that 82% of the children who died experienced convulsions before death, a sign of severe lead poisoning. As of July 2010, approximately 100 children from these villages were receiving chelation therapy while another 2,000 in five other villages were identified as needing it. More than 4,000 children are presently at risk of lead poisoning. Hospital records indicate that more villages may be affected, and lead poisoning may be more widespread than seen initially.[24-27]

1.3. Justification For The Study

Epidemiologic studies form the body of research most relevant to anticipating human health effects and developing guidelines for preventing and managing human exposures to lead. This research allows inference to be made in terms of issues critical to prevention and public health, such as potential causality, dose–response relationships, and susceptible subpopulations, among others. Further, a systematic study of this nature has not been carried out on lead poisoning in the state. This study will also serve as a baseline study which will provide a body of knowledge on childhood lead poisoning and reference for future researches.

Additionally, studies have not been carried out to identify risk factors for childhood lead poisoning. This study is undertaken to among other things, bring to fore an understanding of those factors that predisposes children to lead poisoning in the affected community so as to enable the state design targeted interventions to address the identified risk factors in order to control and prevent childhood lead poisoning in Kawaye village and the State at large.

In spite of presence of laws and guidelines on mining, there is as yet no specific policy on lead poisoning at both national and state levels. This project is therefore undertaken to identify determinants and prevalence of childhood lead poisoning in Zamfara State so as to

provide concrete and scientific information that will assist policy makers, at both state and national levels, in mapping out effective lead poisoning prevention and control strategies.

The high prevalence of lead poisoning, the morbidity and mortality it causes and its socio-economic consequences on the affected children and their families, the nation and donor agencies call for the need to investigate the condition and find measures to control and prevent it.

1.4. Research Questions

1. What is the prevalence of childhood lead poisoning in Kawaye village, Anka LGA of Zamfara State?

2. What are the socio-demographic characteristics of children affected by lead poisoning in Kawaye village, Anka LGA?

3. What are the risk factors for lead poisoning among children in Kawaye village, Anka LGA?

1.5. General And Specific Objectives

1.5.1. General objective

To determine the prevalence and identify determinants of childhood lead poisoning in Zamfara State

1.5.2. Specific objectives

1. To determine the socio-demographic characteristics of children affected by lead poisoning in Kawaye village, Anka Local Government Area of Zamfara State

2. To determine the prevalence of childhood lead poisoning in Kawaye village, Anka Local Government Area of Zamfara State

3. To identify risk factors associated with childhood lead poisoning in Kawaye village, Anka Local Government Area of Zamfara State

1.6. Scope Of The Study

- This study was set out to find the determinants and prevalence of lead poisoning in Kawaye village, Anka Local Government Area of Zamfara State and limited itself to studying children aged 6 years and below only,[6] who were, or whose mother's/caregiver's were resident in Kawaye village for at least 10 months duration.

CHAPTER 2

LITERATURE REVIEW

2.1. Historical Background

Lead poisoning was first recognized as a pediatric disease in Australia over a century ago. A series of 10 cases in Queensland was reported in 1892; 12 years later, after extensive investigation, the source was found to be peeling, lead-based, residential paint on the verandas of the children's homes.[28] The toxic nature of lead has been known since at least 2000 BC. Lead poisoning was common in Roman times, due to the use of lead in water pipes, earthenware containers and wine storage vessels, and the use of a leaded syrup, called *sapa*, to sweeten wine.[29] Occupation-associated lead poisoning was first reported in 370 BC. In 1767, Benjamin Franklin the American statesman and philosopher obtained a list of patients in La Charité Hospital in Paris who had been admitted because of symptoms which, although not recognized as such, were evidently those of lead poisoning. All the patients were engaged in occupations that exposed them to lead. The French physician, Tanqueral des Planches described in 1839 the symptoms of acute lead poisoning on the basis of 1213 admissions to La Charité Hospital between 1830 and 1838. His study was so thorough that little has subsequently been added to the clinical picture of the symptoms and signs of acute lead poisoning in adults. [13, 30] Lead poisoning became common among industrial workers in the 19th and early 20th centuries, when workers were exposed to lead while engaged in trades involving smelting, painting, plumbing, printing and many other industrial activities.[31] In 1882, following the deaths of several employees in the lead industry in the United Kingdom, a parliamentary enquiry was initiated into working conditions in lead factories. This resulted in passage of the 1883 Factory and Workshop Act, which required lead factories in the United Kingdom to conform to certain minimum standards, such as the provision of

ventilation and protective clothing. From then on several Acts were passed to protect the health of workers in industries.

2.2. Lead And Its Types

The English name 'lead' is derived from the Celtic word 'luaide' meaning reddish, because of the color of red lead, or lead tetroxide. Red lead was prepared by heating white lead to a very high temperature. It was used as a pigment in the production of painting and in particular for illuminating manuscripts. The symbol for lead 'Pb' originates from the word 'plumbum' which is the Latin name for lead. A Roman worker in lead was called a plumbarius, hence the name plumber which started to be used in the 19th century when lead water pipes became the principal work of the trade. Plumbism is the medical term for lead poisoning. Elemental lead (*Pb*) is a soft, malleable, ductile, bluish-white, dense metallic element, extracted chiefly from galena and used in containers and pipes for corrosives, solder and type metal, bullets, radiation shielding, paints, and antiknock compounds. It belongs to group 14, period 6 and group “p” of the periodic table with atomic number 82; atomic weight 207.2; melting point 327.5°C; boiling point 1,744°C; specific gravity 11.35 and valence 2, 4. Metallic lead does occur in nature, but it is rare. Lead is usually found in ore with zinc, silver and (most abundantly) copper, and is extracted together with these metals. The main lead mineral is galena (PbS), which contains 86.6 % lead by weight. Other common varieties are cerussite ($PbCO_3$) and anglesite ($PbSO_4$). Most ores contain less than 10% lead, and ores containing as little as 3% lead can be economically exploited. Lead is a very toxic metal that has no any biological value to human body, and once extracted from the ores and processed into its pure elemental form, lead persists indefinitely in the environment.[32, 2]

Generally speaking, lead exists in 2 main forms: organic and inorganic lead. Organic lead comprises of compounds containing lead and carbon at the same time. The most well-known organic lead compounds are tetraethyl and triethyl lead, which were once used as octane

enhancers in gasoline. Tetra-ethyl lead was used extensively from the 1930s to the 1970s as a petrol additive to improve engine performance.[33] Tetra-ethyl lead has been eliminated from the petrol supplies of the majority of countries, but is still used in about 9 countries.[34] Today, organic lead compounds are used much less often - usually as drying agents for lacquers and similar materials. Organic forms of lead are extremely dangerous, as they are absorbed through the skin and are highly toxic to the brain and central nervous system, much more so than inorganic lead. The combustion of organic lead – when it is added to petrol as a fuel additive – results in the release of lead into the atmosphere.

Inorganic lead compounds include metallic lead, lead alloys, lead oxide, and lead sulphate. A major use of inorganic lead is in the manufacture of batteries, industrial paints (e.g., red lead oxide primer) or for use in assaying, soldering, radiator repair and metal reclaiming. Lead compounds are also used in jewellery making, stained glass making and in ceramic glazes. It is the form found in old paint, soil, dust and various consumer products. The color varies, depending on the chemical form, and the most common forms are white lead (a lead carbonate compound), yellow lead (lead chromate, lead monoxide) or red lead (lead tetraoxide). Lead acetate has a sweetish taste. Both organic and inorganic lead is very toxic to human body, and features of poisoning differ depending on whether the agent is an organic compound or an inorganic one.

2.3. Definition Of Lead Poisoning

Lead poisoning is also known as plumbism, colica Pictonum, saturnism, Devon colic, or painter's colic. Classically, "lead poisoning" or "lead intoxication" has been defined as exposure to high levels of lead typically associated with severe health effects. The condition occurs when lead builds up in the body to at least 5 and 10 micrograms per deciliter (µg/dL) of blood in children and adults respectively,[1, 35] usually over a period of months or years.

Poisoning is a pattern of symptoms that occur with toxic effects from moderate to high levels of exposure; toxicity on the other hand is a wider spectrum of effects, including subclinical ones.[36] However, the two terms "lead poisoning" and "lead toxicity" are often loosely used interchangeably.

2.4. Types Of Lead Poisoning

The amount of lead in blood and tissues determines the level of toxicity. Lead poisoning may be acute from intense exposure over a short period of time or chronic as a result of repeat low-level exposure over a prolonged period. It may also be organic or inorganic lead poisoning which occurs as a result of exposure to organic or inorganic lead respectively. Organic lead poisoning is now very rare, because countries across the world have phased out the use of organic lead compounds as gasoline additives, but such compounds are still used in industrial settings.[37] Organic lead compounds, which cross the skin and respiratory tract easily, are more toxic and affect the central nervous system predominantly. Furthermore, lead poisoning may be classified into subclinical and clinical lead poisoning. *Subclinical toxicity* is a terminology used to describe a relatively low-dose exposure to lead at blood lead levels previously thought to be *safe*.[21] Although they are not clinically obvious, the subclinical toxic effects of lead can be very damaging. Subclinical lead poisoning is defined by whole blood lead concentration of $\geq$ 10µg/dL and is usually as a result of exposure insufficient to cause frank lead poisoning.[38] In the clinical lead poisoning however, there is high-dose exposure to lead which manifest in overt signs and symptoms.

2.5. Epidemiology Of Lead Poisoning

The occurrence of lead poisoning is world-wide. Environmental lead is ubiquitous, and everyone has some measurable blood lead level. In the worlds of environmental health, lead exposure remains one of the most important problems in terms of prevalence of exposure and public health impact.[39] Although regulations reducing lead in products has greatly reduced exposure in the developed world since the 1970s, lead is still allowed in products in many developing countries.[40] Lead exposure is estimated to account for 0.6% of the global burden of disease, with the highest burden in developing regions. In all countries that have banned the use of leaded gasoline, average blood lead levels have fallen sharply. However, some developing countries (such as Algeria, Afghanistan, North Korea and Yemen) still allow leaded gasoline,[41,42] which is the primary source of lead exposure in most developing countries.[43] Generally, the widespread occurrence of lead in the environment is largely the result of human activity, such as mining, smelting, refining and informal recycling of lead; use of leaded petrol (gasoline); production of lead-acid batteries and paints; jewellery making, soldering, ceramics and leaded glass manufacture in informal and cottage (home-based) industries; electronic waste; and use in water pipes and solder. Other sources of lead in the environment include natural activities, such as volcanic activity, geochemical weathering and sea spray emissions, and remobilization of historic sources, such as lead in soil, sediment and water from mining areas. As lead is an element, once it is released into the environment, it persists. Because of lead's persistence and potential for global atmospheric transport, atmospheric emissions affect even the most remote regions of the world.[44] Lead poisoning is commoner in children, and poor children in developing countries are at especially high risk for lead poisoning.[45]

In 2000, an estimated 120 million people around the world had blood lead concentrations of between 5 and 10 μg/dL, and about the same number had concentrations greater than 10μg/dL, a level associated with considerable health risk. Children are particularly vulnerable and 40% of all children had blood lead concentrations above 5μg/dL and 20% had concentrations ≥ 10μg/dL, with the great majority (97%) of these living in the developing world. Of North American children, 7% have blood lead levels above 10μg/dL, whereas among Central and South American children, the percentage is 33 to 34%. About one fifth of the world's disease burden from lead poisoning occurs in the Western Pacific, and another fifth is in Southeast Asia.[8, 45] In contrast to risk reduction efforts in developed countries, lead contents of gasoline sold in African countries have gone up and are now among the highest in the world; typical concentrations of lead in regular fuel fall in the range of 0.5- >1.0g/L.[5] Studies in South Africa, for instance, showed that more than 50% of the children in Cape Town, Durban, Johannesburg, and possibly other cities have blood lead levels greater than 10μg/dL. Nigerian children are equally at risk of being poisoned by lead in their environment. A study in 1994 found average blood lead for non-occupationally exposed adults in Lagos and Ife to be 13μg/dL, which is above current level of concern. [8, 46] Another study on blood lead and tap water lead levels in Ibadan, Nigeria found mean BLL of 11.4 and 12.3μg/dl for females and males, respectively.[7] In a matched case-control study conducted in Port Harcourt, Nigeria, occupationally lead exposed subjects were found to have higher mean blood lead levels of 50.37±24.58 μg/dl than controls 41.40±26.85μg/dl.[47]

In developed countries, nonwhite people with low levels of education living in poorer areas are most at risk for elevated lead. In United States, the groups most at risk for lead exposure are the impoverished, city-dwellers, and immigrants.[40, 45] African-American children and those living in old housing have also been found to be at elevated risk for high blood lead levels in the US.[48, 49]

Risk factors for elevated lead blood levels include alcohol consumption and smoking. Adults with certain risk factors might be more susceptible to toxicity; these include calcium and iron deficiencies, malaria, old age, disease of organs targeted by lead (e.g. the brain, the kidneys), and possibly genetic susceptibility.[39,50,51] Socioeconomic factors are important predictors of exposure to lead. Poor families are more likely to live near industrial plants that handle lead, such as battery recyclers or smelters. Also, they are more likely to dwell on polluted lands, to work in polluting industries, or to live in older housing with lead-based paint. In addition, poor children are more likely to have iron or calcium deficient diets, iron and calcium deficiency states lead to increased body absorption of lead as lead competitively binds to ca^{2+} and Fe^{2+} receptors. Malaria, an infectious disease of humans and other animals caused by a parasite belonging to the genus Plasmodium and transmitted by female anopheles mosquitoes has also been associated with lead poisoning. Few studies have demonstrated a significant association between malaria and lead poisoning. According to a study conducted in three Nigerian cities, presence of malaria is a robust predictor of BLL.[51]

Culture and ethnicity are also strongly related to such risk factors for exposure to lead as the use of traditional cosmetics; herbal medicine products and pica during pregnancy have been associated with lead poisoning. Differences in vulnerability to lead-induced neurological damage between males and females have also been found, but some studies have found males to be at greater risk, while others have found females to be.[52] In adults, blood lead levels steadily increase with increasing age. In adults of all ages, men have higher blood lead levels than women do. Children are more sensitive to elevated blood lead levels than adults are. Children may also have a higher intake of lead than adults; they breathe faster and may be more likely to have contact with and ingest soil. Children aged one to three years tend to have the highest blood lead levels, possibly because at that age they begin to walk and explore

their environment, and they use their mouths in their exploration. Blood levels usually peak at about 18–24 months old.[52, 53, 54]

In a study aimed at identifying the socio-ecologic variables contributing to lead poisoning among children in suburban regions of Nigeria, mean BLL was significantly associated with household size, maternal literacy, parental occupation, home floor type, time spent outside the home, residential proximity to a ceramics shop, occupational circumstances and by parental employment in a print shop ($p < 0.05$), but not with age, years of parental education, housing situation, drinking water, and frequency of hand-to-mouth behavior. However, gasoline was found to be the major source of environmental lead contamination.[6]

2.6. Exposure Routes

An exposure pathway must, by definition, have five components:

- A source of contamination such as mining site, used car batteries and deteriorating lead-based paints on walls, etc.
- A mechanism for transport of a substance from the source to the air, surface water, groundwater and/or soil,
- A point of exposure (such as children's and adults' hands, clothing, working tools, etc;
- A route of entry into the body. Routes of entry can be eating or drinking contaminated materials, breathing contaminated air, or absorbing contaminants through the skin and
- An exposed population such as children in the home environment.

When all the five components are present, the pathway is termed a complete exposure pathway. Risks can be assessed when an exposure pathway is complete. If any part of an exposure pathway is absent, the pathway is said to be incomplete and no exposure or risk is possible. [55]

Exposure to lead occurs through three (3) main ways: ingestion, inhalation and dermal contact. Lead exposure in the general population (including children) occurs primarily through ingestion, although inhalation also contributes to lead body burden and may be the major contributor for workers in lead-related occupations.[56] Lead ingestion occurs as a result of children bringing lead containing or lead contaminated objects into their mouth. Once lead has been swallowed, it enters a child's body by absorption from the gastrointestinal tract. Inhalation is the second major pathway of exposure. Almost all inhaled lead is absorbed into the body, whereas from 20% to 70% of ingested lead is absorbed (with children generally absorbing a higher percentage than adults do). Inhalation usually occurs when people are exposed to lead in particulate matter less than 10μm in diameter (PM_{10}). Severe cases of lead poisoning following inhalation of contaminated dust have been documented.[57] Dermal exposure plays a role for exposure to organic lead among workers, but is not considered a significant pathway for the general population. Organic lead may be absorbed directly through the skin, organic lead (tetramethyllead) is more likely to be absorbed through the skin than inorganic lead and dermal exposure is most likely among people who work with lead. In addition to the above 3 main ways, exposure to lead can occur endogenously. Endogenous exposure to lead may also contribute significantly to an individual's current blood lead level, and of particular risk to the developing fetus. Once absorbed into the body, lead may be stored for long periods in mineralizing tissue (i.e., teeth and bones). The stored lead may be released again into the bloodstream, especially in times of calcium stress (e.g. pregnancy, lactation, osteoporosis), or calcium deficiency.

2.7. Sources Of Lead

Worldwide, the following sources and products account for most cases of childhood exposure to lead and lead poisoning:

- lead added to gasoline,

- lead-based paints and pigments,
- lead from an active industry, such as mining (especially in soils),
- drinking-water systems with lead solder and lead pipes,
- ceramic glazes,
- lead solder in food cans,
- lead in products, such as herbal and traditional medicines, folk remedies, cosmetics and toys,
- lead released by incineration of lead-containing waste,
- lead in electronic waste (e-waste) and
- lead in the food chain, via contaminated soil and water.

GASOLINE

Gasoline (Petrol) used to be the major source of exposure to lead in the environment. The phasing-out of lead from petrol has reduced the concentration of lead in blood worldwide and is considered a major international public health achievement.[58] Lead is added to petrol as an antiknock, however, with the discovery of the extent of environmental and health damage caused by lead and the incompatibility of lead with catalytic converters found on virtually all newly sold US automobiles since 1975, the practice of using lead as antiknock began to wane. In US, standards to phase out leaded gasoline were first implemented in 1973. From 1st January, 1995; the "Clean Air Act" banned the sales of leaded fuels for use in on-road vehicles in USA.[59]

In Sub-Saharan Africa, agreement to phase out the use of leaded petrol was reached in June 2001 at a regional conference held in Dakar, Senegal, with the theme "Phasing-out leaded gasoline in sub-Saharan Africa". This conference, organized by the World Bank in the

framework of the Clean Air Initiative in sub-Saharan Africa, was attended by delegates from 25 countries. One of the major outputs of the Dakar conference was a unanimous agreement to eliminate lead from gasoline as soon as possible and at the latest by 2005. Five sub-regional Actions Plans, framing national clean air implementation programs, were drafted by groups of countries. The sub-regional group led by Nigeria was the first to start the implementation process.[60] Nigeria phased out the use of leaded petrol in 2004.[61] Of the 49 African countries, only Algeria still uses leaded petrol to date. As at June 17, 2011, about six countries in the world are still possibly using leaded gasoline (Afghanistan, Algeria, Iraq, North Korea, Myanmar (Burma) and Yemen).[62]

In April 1994, the United Nations called for government action to eliminate lead from gasoline globally. Original phase-out targets were set at 2008 (by the United Nations in 2002), then 2010 (set in 2006 by SAICM (Strategic Approach to International Chemicals Management)). The target date has now been spun out to 2013.[63]

PAINT

After lead in petrol, lead in paint is one of the largest sources of exposure to lead. Leaded paint can remain a source of exposure to lead and lead poisoning for many years after the paint has been applied to surfaces. Childhood lead poisoning from residential lead-based paint was first described in 1892 in Brisbane, Australia, and the first published warnings of the risk to children from lead in residential paint were published more than a century ago. Lead was eventually banned from house paint in Australia in 1914, the same year that childhood lead poisoning was first reported in America. However, it was not until 1978 that the United States followed suit; by that time, 74% of dwellings in the United States contained some lead-based paint. As of 2009, lead-based residential paint was the main source of lead poisoning in children in the United States. [64, 6] A study to evaluate the lead content of paints

sold in Nigerian markets showed that paints sold in Nigeria contain substantial amount of lead with increased risk of exposure to children for whom domestic sources of lead exposure is more important than exposure through leaded petrol.[65] Deteriorating lead paint can produce dangerous lead levels in household dust and soil. Deteriorating lead paint and lead-containing household dust are the main causes of chronic lead poisoning. The lead breaks down into the dust and since children are more prone to crawling on the floor, it is easily ingested. Many young children display pica, eating things that are not food. Even a small amount of a lead-containing product such as a paint chip or a sip of glaze can contain tens or hundreds of milligrams of lead. Eating chips of lead paint presents a particular hazard to children, generally producing more severe poisoning than occurs from dust.[66]

The Second Session of the International Conference on Chemicals Management, held in Geneva in May 2009, endorsed a global initiative to promote phasing out the use of lead in paints (*WHO/UNEP initiative to remove lead from paints globally*).[67] This initiative was jointly put forward by Toxics Link, the Intergovernmental Forum on Chemical Safety and the United States Environment Protection Agency (EPA) to remove lead from paint. The International Conference on Chemicals Management, which is an intergovernmental body that consists of 162 countries, endorsed a global alliance to promote the phasing out of the lead in paints and invited all stakeholders to become members. WHO and United Nations Environment Programme (UNEP) within their respective mandates, serve as the Secretariat.

OCCUPATIONAL EXPOSURE TO LEAD

In adults, occupational exposure is the main cause of lead poisoning. People can be exposed when working in facilities that produce a variety of lead-containing products; these include radiation shields, ammunition, plumbing, ore miners and smelters, circuit boards, jet engines,

and ceramic glazes.[68] In addition, plumbers and fitters, auto mechanics, glass manufacturers, construction workers, battery manufacturers and recyclers, firing range instructors, and plastic manufacturers are at risk for lead exposure. Other occupations that present lead exposure risks include welding, printing, zinc and copper smelting, processing of ore, combustion of solid waste, and production of paints and pigments. Parents who are exposed to lead in the workplace can bring lead dust home on clothes or skin and expose their children. The outbreak of lead poisoning in Zamfara State has been associated with unsafe artisanal gold ore processing that occurs within the villages in the State.[69, 70]

SOIL

Lead content in soil may be caused by broken-down lead paint, residues from lead-containing gasoline, used engine oil, or pesticides used in the past, contaminated landfills, or from nearby industries such as foundries or smelters. Leaded soil raises concern about the safety of people particularly children and agriculture; eating food grown in contaminated soil can present a lead hazard.[71]

WATER

Lead from the atmosphere or soil can end up in underground and/or surface water. Similarly, lead in water can result from plumbing and fixtures that are either made of lead or have lead solder which are dissolved by the water. Today the principal source of lead in drinking-water in most locales is lead solder. Lead solder used in the joints of pipes and water mains and as a component of brass fittings can leach into drinking-water, especially when the water has an acidic pH. The current WHO standard for the lead content of drinking-water is 10μg/l.[72, 73]

LEAD IN FOOD

Most of the daily intake of lead (>80%) is acquired through the ingestion of food, dirt and dust. The quantity of lead in food plants depends on soil concentrations and is highest around mines and smelters. Cereals can contain high levels of lead. Milk or formula is a significant source of exposure for infants. The use of lead-soldered food and beverage cans may considerably increase the lead content, especially in the case of acidic foods or drinks. Lead also comes to unintentionally contaminate food as the result of contamination with soil or from lead used in machinery to process items – for example, wheels for flour that are coated with lead. Since alcoholic drinks tend to be acidic, the use of any lead-containing products in their manufacture or distribution will raise lead levels. More so, smoking tobacco increases lead intake.

LEAD IN PRODUCTS

Lead is found in variety of products such as kohl eye cosmetic and toys. Lead is a problem in toys for two reasons: (1) the toy may be painted with leaded paint; and (2) the toy itself is made of lead. It is also found in vinyl mini-blinds and commonly incorporated into herbal remedies such as Indian Ayurvedic preparations and remedies of Chinese origin. Several recent episodes have been reported of lead in children's toys, and in 2007, millions of toys made in China were recalled from multiple countries owing to safety hazards including lead paint. Also in the United States in 2008 a large-scale recall of imported, lead-painted children's toys was instituted.[74] There have also been reports of cases of lead poisoning associated with Ayurvedic herbal products which raise serious health concerns.[75] Reported cases include fatal lead encephalopathy of a 9-month-old infant, severe developmental delay in a 5-year-old child, and congenital paralysis and deafness in a preterm infant.

LEAD IN ELECTRONIC WASTE

Lead from e-waste is an emerging hazard. With the global proliferation of computers, cellular telephones and other electronic equipment –as well as rapid cycles of replacement and obsolescence of these instruments – an enormous amount of electronic waste is now generated each year worldwide. A study carried out in 2005 to assess the blood lead level of children aged 6-15 years that scavenges wastes recorded elevated blood lead levels (mean blood lead levels of 28.4 μg/dL, standard deviation of 11.5).[76]

HUNTING

Some hunters use lead-based bullets in hunting game animals. It is argued that lead-based bullets offer greater accuracy and more humane kills of game animals than might copper-based bullets, which represent the most commonly available, though expensive, alternative to lead. Lead-based bullets are highly resistant to fragmentation, offering hunters the ability to clean game animals with negligible risk of including lead fragments in prepared meat. In a study conducted by the CDC, a cohort from North Dakota was enrolled and asked to self-report historical consumption of game meat, and participation in other activities that could cause lead exposure. The study found that participants' age, sex, housing age, current hobbies with potential for lead exposure, and game animal meat consumption were all associated with elevated blood lead level.[77] A similar study conducted in 2009 showed that human consumers of wildlife killed with lead ammunition are exposed to health risks associated with lead ingestion. [78]

2.8. Clinical Presentation

Lead poisoning causes a variety of signs and symptoms which differ based on an individual and the duration of exposure. Symptoms are nonspecific and may be subtle, and someone with elevated lead levels may have no symptoms. Symptoms usually develop over weeks to months as lead builds up in the body during a chronic exposure, but acute symptoms from brief, intense exposures also occur. Symptoms from exposure to organic lead, which is probably more toxic than inorganic lead due to its lipid solubility, occur rapidly, producing symptoms predominantly in the central nervous system such as insomnia, delirium, cognitive deficits, tremor, hallucinations, and convulsions. Symptoms also differ in both children and adults and depending on the nature of exposure: acute or chronic. Signs and symptoms of lead poisoning in adults some of which are nonspecific include loss of appetite, intermittent abdominal pain, an unusual taste in the mouth, nausea, diarrhea, constipation, malaise, weakness, muscle pain, fatigue, problems with sleep, depression and personality changes, headache, memory loss, kidney failure, decreased libido, infertility, and pain or tingling in the extremities.

Children are more at risk for lead poisoning because their smaller bodies are in a continuous state of growth and development. Lead is absorbed at a faster rate compared to adults, which causes more physical harm than to older people. Furthermore, children, especially as they are learning to crawl and walk, are constantly on the floor and therefore more prone to ingesting and inhaling dust that is contaminated with lead. Infants developing in the womb of a woman who has elevated blood lead level, are also susceptible to lead poisoning. There is also a risk as the baby comes closer to full term, as the woman is more likely to have a premature delivery and/or a low birth weight infant.[79, 80]

The common signs and symptoms of lead poisoning in children include loss of appetite, abdominal pain, vomiting, weight loss, constipation, anemia, kidney failure, lethargy,

irritability, convulsions, learning disabilities, delayed developmental milestone, behavioral abnormalities and permanent mental retardation.

However, in acute lead poisoning, the typical signs and symptoms are astringency, metallic taste, abdominal pain, nausea, vomiting, diarrhea, constipation, muscle pain, muscle weakness, paraesthesia and rarely, symptoms associated with encephalitis. Absorption of large amounts of lead over a short time can cause shock due to loss of water from the gastrointestinal tract, hemolysis and damage to kidneys (causing changes in urination such as decreased urine output and changes in urine color).

On the other hand, chronic poisoning usually presents with signs and symptoms affecting multiple systems. It typically present with three main types of signs and symptoms: gastrointestinal, neuromuscular and neurological. Features of chronic exposure include nausea, abdominal pain, fatigue, headaches, problems with sleep, numbness, tingling in the extremities, depression, loss of coordination, loss of short-term memory or concentration, slurred speech, stupor, anemia and a "lead hue" of the skin with pallor. Chronic poisoning may also manifest in children with refusal to play and hyperkinetic or aggressive behavior disorders. A blue line along the gum, with bluish black edging to the teeth, known as Burton's (lead) line is another indication of chronic lead poisoning.[81]

Physical examination may reveal signs of raised intracranial pressure, lead lines in teeth, gout, hypertension, signs of anemia and exaggerated deep tendon reflexes. And laboratory investigations may show the following:

- Elevated BLL
- Hematology :
    - Hypochromic anemia,
    - red blood cells with basophilic stippling,

- elevated protoporphyrin levels (erythropoietic protoporphyrin (EPP) or zinc protoporphyrin (ZPP))*
- Others: Hyperuricaemia and hypocalcaemia

- Urine: Proteinuria, glycosuria and aminoaciduria (acute poisoning)
- Hepatic injury:
  - Elevated transaminase levels (acute poisoning)
- Radiological: Lead lines in the metaphyses of long bones (chronic poisoning)

* = Protoporphyrin levels (EPP or ZPP) are usually not elevated until the BLL is ≥ 25μg/dL and may also be elevated in other conditions such as iron deficiency anemia.

2.9. Pathophysiology

2.9.1. Pharmacokinetics

ABSORPTION

Lead gain access into the body through inhalation, ingestion and occasionally skin absorption. It may also gain access to the body through direct contact with mouth, nose, and eyes, and through breaks in the skin. Tetraethyllead, which was a gasoline additive and is still used in fuels such as aviation fuel, passes through the skin easily; however inorganic lead found in paint, food, and most lead-containing consumer products is only minimally absorbed through the skin. The main sources of absorption of inorganic lead are from ingestion and inhalation. In adults, about 35–40% of inhaled lead dust is deposited in the lungs, and about 95% of that goes into the bloodstream.

Of ingested inorganic lead, about 15% is absorbed, but this percentage is higher in children, pregnant women, and people with deficiencies of calcium, zinc, or iron. Children and infants may absorb about 50% of ingested lead. Generally, lead absorption is dependent on several factors, including the type of lead (organic or inorganic), physical form of lead, the particle

size ingested, the GI transit time, and the nutritional status of the person ingesting. Lead absorption is inversely proportional to the particle size; the smaller the particle, the more completely the lead is absorbed. Thus, exposure to lead dust results in higher absorption than exposure to the equivalent amount of lead from chips of lead paint. Lead absorption is augmented in the presence of iron, zinc, and calcium deficiency. Lead absorption is also augmented by malnutrition, with lead absorption decreased if phosphorus, riboflavin, vitamin C, and vitamin E are in the diet. Low-energy (calorie) intake and high-fat intake have been associated with enhanced lead absorption. Furthermore, lead absorption is inversely proportional to chronologic age. In general, approximately 30-50% of lead ingested by children is absorbed, compared with approximately 10% of that ingested by adults.[82]

Cutaneous absorption of lead is limited (typically far less than 1%). The amount absorbed through the skin depends on the physical characteristics of the lead (i.e. organic vs. inorganic) and the integrity of the skin. Although inorganic lead is not absorbed through intact skin, organic lead compounds (i.e. tetraethyl lead) are absorbed.

If inhaled in a fine particulate state, lead may be absorbed directly through the lungs or may be carried by the mucociliary tree to the throat, where it is swallowed and absorbed via the gastrointestinal system. The amount of absorption of particulate lead that occurs through the respiratory system depends on the particle size, the patient's respiratory volume, the amount of deposition and the mucociliary clearance of the lead inhaled. The majority (nearly 100%) of lead inhaled as vapor or fumes is absorbed directly through the lungs.[82, 83]

DISTRIBUTION

Absorbed lead that is not excreted is exchanged primarily among 3 body compartments: blood, soft tissues (liver, kidneys, lungs, brain, spleen, muscles, and heart) and mineralizing tissues (bones and teeth).

Blood: following absorption, lead enters the blood. Lead in the blood is primarily found within red blood cells (RBCs). Although the blood generally carries only a small fraction of the total body lead burden, it serves as the initial receptacle of absorbed lead and distributes it throughout the body, making it available to other tissues or for excretion. The elimination half-life of lead in adult human blood has been estimated to be 1 month, whereas in children it may be as long as 10 months.

Approximately 99% of the lead in blood is associated with RBCs; the remaining 1% resides in blood plasma, which transfers lead between the different compartments. Blood lead is also important because the blood lead level (BLL) is the most widely used measure of lead exposure. The less sensitive erythrocyte protoporphyrin (EP) assay is also used as a measure of blood lead. These tests, however, do not measure total body burden; they are more reflective of recent or ongoing exposures.

Soft tissues: From the blood, lead enters into different body tissues and organs. Studies have indicated that the liver, kidneys, lungs and brain have the greatest soft-tissue lead concentrations following exposure.[84] Children retain more lead in soft tissue than adults do. Selective brain accumulation may occur in the hippocampus. Lead in soft tissues has an approximate half-life of 40 days.

Mineralizing tissues (Bones and teeth): The lead in blood is ultimately stored bones and teeth. About 75% and 90% of the total body lead in children and adults respectively is stored in bones and teeth. Lead in mineralizing tissues is not uniformly distributed, with accumulation in bone regions undergoing the most active calcification at the time of exposure. During periods of physiologic stress, the body can mobilize lead stores in bone, thereby increasing the level of lead in the blood. Bone-to-blood lead mobilization increases during periods of pregnancy, lactation, menopause, chronic disease, hyperthyroidism, kidney

disease, fractures, and advanced age, and is exacerbated by calcium deficiency. Consequently, the normally inert pool poses a special risk because it is a potential endogenous source of lead that can maintain BLLs long after exposure has ended.

EXCRETION

Lead is excreted primarily through the urine (65-75%) and feces (25-30%), with smaller amounts excreted in sweat, hair, nails and breast milk.[85, 86] About 99% of the lead in adults is excreted whereas, only about 32% of the lead in children is excreted.

2.9.2. Mechanisms of toxicity

Lead is associated with a wide range of toxicities across a very broad band of exposures, down to the lowest blood lead concentrations yet studied, both in animals and humans. Lead produces reactive radicals which damage cell structures including DNA and cell membranes. Lead also interferes with DNA transcription enzymes that help in the synthesis of vitamin D, and enzymes that maintain integrity of the cell membranes. It interferes with the normal metabolism of calcium in cells and causes it to build up within them. It also interferes with metabolism of bones and teeth, impair permeability of blood vessels and alters collagen synthesis. Thus, anemia may result when the cell membranes of red blood cells become more fragile as the result of damage to their membranes. Lead is also harmful to the developing immune system resulting in the liberation of excessive amount of inflammatory proteins which may be a risk factor for asthma particularly in children. In addition, it is also associated with decreased activity of immune cells such as polymorphonuclear leucocytes. Other effect of lead includes impairment male and female reproductive systems.

The principal mechanism of lead toxicity is its ability to interfere with variety of enzymatic processes. It binds to sulfhydryl group that is found on many enzymes. Another mechanism of lead toxicity at enzymatic level is its ability to mimic other metals that take part in

biological processes, which act as cofactors in many enzymatic reactions, thereby displacing them at the enzymes on which they act. Consequently, lead binds to and interacts with many of the same enzymes as these metals but, due to its differing chemistry, does not properly function as a cofactor, thus interfering with the enzyme's ability to catalyze its normal reaction(s). Some of the essential metals with which lead interacts are calcium, iron, and zinc. Some of the enzymes that lead interferes with include *delta-aminolevulinic acid dehydratase* (δ-ALAD) which is important in the biosynthesis of heme, the cofactor found in hemoglobin. It also inhibits the enzyme *ferrochelatase*, another enzyme involved in the formation of heme. Lead's interference with heme synthesis results in production of zinc protoporphyrin and the development of anemia. Furthermore, lead's interference with heme synthesis results in the buildup of heme precursors such as aminolevulinic acid, which are harmful to neuronal cells.

Neurotoxicity occurs due to ability of the lead to substitute for other polyvalent cations (particularly divalent cations, such as calcium (Ca^{2+}) and zinc (Zn^{2+})) in the molecular machinery of living organisms.[87] Lead also causes neurotoxicity by interfering with neurotransmitters, the chemicals used by neurons to send signals to other cells. It interferes with the release of glutamate, a neurotransmitter important in many functions including learning, by blocking N-Methyl-D-Aspartic acid (NMDA) receptors. The targeting of NMDA receptors is thought to be one of the main causes for lead's toxicity to neurons.

In children, whole blood lead levels as low as 1–3 μg/dL are associated with subclinical neurobehavioral toxicity.[88] Another study carried out on more than 4800 children aged 6-16 years in USA in found that there is an inverse relationship between blood lead level and math and reading scores in children at blood lead concentrations lower than 5 μg/dL. The relationship was still evident after adjustment for an extensive series of potential confounding factors. Indeed, the dose–response relationship between blood lead levels and loss of IQ was

stronger at blood lead levels lower than 10 µg/dL than at higher levels.[89] Increased blood lead level in children has been correlated with decreases in intelligence, nonverbal reasoning, short-term memory, attention, reading and arithmetic ability, fine motor skills, emotional regulation, and social engagement. High blood lead levels in adults are also associated with decreases in cognitive performance and with psychiatric symptoms such as depression and anxiety. It was found in a large group of current and former inorganic lead workers in Korea that blood lead levels in the range of 20–50 µg/dL were correlated with neuro-cognitive defects.[90] The neurobehavioral changes associated with early exposure to lead appear to be persistent and irreversible. These changes are not reversed or improved by chelation therapy.[91]

The effects of lead on the kidneys are the most insidious. Lead produces both acute and chronic nephropathies. Acute lead nephropathy is characterized by proximal tubular dysfunction with the development of a Fanconi-type syndrome, alterations in mitochondrial structure and the development of cytosolic and nuclear inclusion bodies. Chronic lead nephropathy on the other hand is irreversible and is typically accompanied by interstitial fibrosis, both hyperplasia and atrophy of the tubules, glomerulonephritis and ultimately, renal failure. In addition, lead produces renal neoplasms in experimental animals.[92] Lead also inhibits excretion of the waste product urate and causes a predisposition for gout, in which urate builds up. This condition is known as saturnine gout.

Chronic and acute lead poisoning cause overt clinical signs and symptoms of cardiac and vascular damage with potentially lethal consequences. Signs of vascular degeneration, abnormal vascular smooth muscle function and altered vessel compliance have been described in humans chronically and acutely exposed to toxic lead levels. Myocarditis, electrocardiographic disturbances, heightened catecholamine arrhythmogenicity, altered myocardial contractile responsiveness to inotropic stimulation, degenerative structural and

biochemical changes affecting the musculature of the heart and vasculature, hypertension, hypercholesterolemia, atherosclerosis and increased vascular reactivity to alpha-adrenergic agonists have been among the reported cardiovascular disturbances linked to lead poisoning.[93] In some studies these associations were observed at surprisingly low blood lead concentrations (< 5µg/dL). Schwartz published a study of the incidence of left ventricular hypertrophy (LVH) in a re-analysis of the NHANES II data set in individuals aged 25-74 yr. The odds ratio for LVH was 1.33 for each 10µg/dL increase in blood lead.[94]

In a nutshell, lead poisoning can affect virtually every organ system in the body. The principal organs affected are the central and peripheral nervous systems, cardiovascular, hematological, renal, immune, gastrointestinal and endocrine systems.

2.10. Diagnosis

The diagnosis of lead poisoning comprises of good clinical history, physical examination and laboratory testing.

LABORATORY TESTING

The laboratory tests carried out to diagnose lead poisoning include the following:

- **Blood Lead Level (BLL):** This is the single best diagnostic test for lead exposure. It reflects the amount of lead currently found in the blood and soft tissues (and hence key target organs). Because virtually 99% of the lead in blood is bound to red blood cells, whole blood (not serum or plasma) is required for its measurement. The major drawback of BLL is that, BLL alone is not a reliable indicator of prior or current exposure, or total body lead burden. BLLs reflect the contributions of recent external exposure to lead as well as the release of internal bone lead stores into the blood. As such, BLLs represent a mixture of both external exposure and internal lead stores. Periodic testing of BLL is called biological monitoring.

This provides valuable information to assess lead exposure for individuals as well as groups of workers.

- **Free Erythrocyte Protoporphyrin (FEP) or Erythrocyte Protoporphyrin (EP) testing:** The inhibition of heme synthesis (i.e. inhibition of the enzyme ferrochelatase) leads to the accumulation of excess porphyrins, particularly protoporphyrin IX in red cells. The fluorescence of these porphyrins has led to the development of methods to detect extracted porphyrins-free erythrocyte protoporphyrin (FEP) or erythrocyte porphyrin (EP) and to the observation of zinc protoporphyrin (ZPP) in red cells. Because the relationship between the results of these tests and BLLs is log-linear, these tests can be used to evaluate and follow children with very high BLLs. However, EP level alone is not sensitive enough to identify elevated blood lead levels below about 25µg/dL. Due to this poor correlation of EP with low blood lead levels and the fact that EP/ZPP levels also increase in iron deficiency and porphyria, EP tests should be used infrequently except in evaluating children with BLLs well above 25µg/dL whose BLLs do not show a steady decline in response to medical and environmental interventions.
- **Blood Film Examination:** this may reveal basophilic stippling of red blood cells (dots in red blood cells visible through a microscope) as well as the changes normally associated with iron-deficiency anemia (microcytosis and hypochromasia). However, basophilic stippling is also seen in megaloblastic anemia caused by vitamin B12 and folate deficiencies. The basophilic stippling of red cells is due to the presence of aggregated ribosomes, which may also include mitochondrial fragments. With staining, this appears as increased basophilic granulation.
- **X-Ray Fluorescence Of The Long Bones:** this may be the best measure of cumulative exposure and total body lead. However this method is not widely available and is mainly used for research rather than routine diagnosis. It reveals the presence of radiodense lines called

lead lines at the metaphysis of the long bones of growing children, especially around the knees. These lead lines, caused by increased calcification due to disrupted metabolism in the growing bones, become wider as the duration of lead exposure increases. Lead lines are usually not present unless BLLs exceed 50µg/dL and are indicative of chronic exposure.

- **Abdominal Radiographs:** may be useful in determining whether children are currently ingesting lead-contaminated non-food items, including paint chips.
- **Urinary delta-aminolevulinic acid:** inhibition of heme synthesis through the inhibition of δ-aminolevulinic acid dehydratase by lead results in the accumulation of delta-aminolevulinic acid in the blood which is subsequently excreted in the urine.
- **Urinary Coproporphyrin:** Pb inhibits coproporphyrinogen decarboxylase and this leads to excretion of coproporphyrin in urine.
- **Fecal Lead Assessment:** Fecal lead content measured over the course of a few days may also be an accurate way to estimate the overall amount of childhood lead intake. This form of measurement may serve as a useful way to see the extent of oral lead exposure from all the diet and environmental sources of lead.
- **Hair, Fingernail and Tooth (dentin) Lead Measurements:** these are of no documented utility in the management of children with EBLLs.[95] Further, hair and fingernails are prone to external contamination, which makes the results of lead tests on them unreliable.
- **Chelation challenge testing or provocation testing:** this test is used to indicate an elevated and mobilizable body burden of lead. It involves collecting urine before and after administering a one-off dose of chelating agent to mobilize lead into the urine. The urine is then analyzed for level of lead; from this analysis overall body burden is inferred. The test mainly measures the burden of lead in soft tissues, and may not accurately reflect long-term exposure or the amount of lead stored in bone. Another limitation of this test is that the single

chelation dose could redistribute the lead to more sensitive areas such as central nervous system tissue.

- **Other supportive investigations:**
- **Renal Function Tests:** Though very high BLLs have been associated with serious to severe renal tubular dysfunction, there is no evidence to support routinely evaluating the renal status of children with pre-symptomatic BLLs. However, if potentially nephrotoxic chelating agents such as EDTA are to be used in treatment, renal function testing is appropriate prior to and during therapy.
- **Hemoglobin/Packed Cell Volume (PCV):** in chronic, moderately severe lead poisoning, anemia is commonly found. A decrease in hemoglobin is reported to occur in iron-sufficient children when blood lead concentration exceeds 60μg/dL. Anemia in lead poisoning results from impairment of hemoglobin production and changes in the red blood cell membrane. While anemia may not be seen until blood lead concentrations are markedly elevated, the effect on hemoglobin synthesis occurs at lower levels. The anemia is a normocytic, normochromic, well-compensated hemolytic anemia. In children with iron deficiency, the decrease in hemoglobin may occur at lower blood lead levels and present as a microcytic anemia.

2.11. Differential Diagnoses

The differential diagnoses of lead poisoning include iron deficiency, renal colic, appendicitis, carpal tunnel syndrome, encephalitis, Guillain-Barré syndrome, abdominal colic, viral gastroenteritis (in children), emotional and behavioral disorders, mental retardation, neoplasms of the central nervous system and subdural hematoma.

2.12. Treatment/Prevention

2.12.1. Treatment

Treatment of lead poisoning involves the following:

DECREASING EXPOSURE

By far, the most successful management occurs due to the removal of the lead risk from the environment and ultimately, the child.[96] Once the source of lead is found in the home, soil, or workplace every effort should be made to remove this source. This may be accomplished by home lead paint abatement, home dust reduction techniques and decreasing bare soil available to children.

BALANCED DIET

Nutritional evaluation and counseling should be conducted. Those children with iron, calcium, and zinc deficiencies should be treated as these conditions are associated with increased lead absorption. In addition, diet containing adequate amounts of vitamins and minerals such as iron, calcium, and vitamin C should be given. A person who eats a balanced, nutritious diet may absorb less lead than a person whose diet is inadequate.

REMOVAL OF LEAD-CONTAINING MATERIALS FROM THE BODY

When lead-containing materials are present in the gastrointestinal tract (as evidenced by abdominal X-rays), whole bowel irrigation, cathartics, endoscopy, or even surgical removal may be used to eliminate it from the gut and prevent further exposure. Lead-containing bullets and shrapnel may also present a threat of further exposure and may need to be surgically removed.

CHELATION

Usually, the measures above are sufficient to limit exposure to lead and reduce lead levels in the body. However, if blood lead levels remain persistently high, chelation therapy may be indicated. Chelation is not a decision that should be taken lightly. It represents the final component of treatment in lead poisoning. And it is a dangerous process that is not devoid of complications such hypocalcemia, hypozincaemia and nephrotoxicity. Chelation therapy refers to a process that lowers the amount of lead stored in the body through the use of drugs called chelating agents or chelators which cause metals like lead to bind to them at two or more sites, and then they are eliminated from the body through excretion (urinary and biliary).[97] Ideally, the chelated metal should be excreted; however, the lead:chelate complex may persist in tissues where the binding occurred or be redistributed to other tissues. More so, chelating agents may increase the absorption of lead and other metals, it is therefore essential that sources of lead exposure be removed before a person is treated. Generally speaking, chelation is indicated in cases with severe lead poisoning (when blood lead level reaches $\geq 45\mu g/dL$)[98] and/or encephalopathy.

There are several chelating agents used in the treatment of lead poisoning, but the chelator of choice depends on the blood lead concentration, the patient's age, presenting signs and symptoms and the environmental lead burden. Chelating agents that are used in treating lead poisoning are:

- Calcium Di-Sodium Edetate (CaNa2EDTA),
- British Anti-Lewisite (BAL), also called Dimercaprol,
- 2,3 Dimercaptosuccinic Acid (DMSA or Succimer),
- D-Penicillamine and
- Dimercapto-propane sulfonate (DMPS, Unithiol)

Calcium Di-Sodium Edetate ($CaNa_2EDTA$):

Calcium Disodium Edetate ($CaNa_2EDTA$) belongs to the family of polyaminocarboxylic acids. Although it is capable of chelating many metals, its current use is almost exclusively restricted to the management of lead poisoning. It can be used intramuscularly (IM) or intravenously (IV); in many centers, the IM route has been abandoned in favor of a continuous IV drip that appears to improve outcome and decrease the adverse effects associated with the IM route. Oral administration of the drug is not practicable owing to its bioavailability of < 5% when given through this route. It is given at a dose of 1000-1500 mg/m^2/day as an IV infusion. A major limitation of $CaNa_2EDTA$ is the enormous diuresis of zinc that it produces and nephrotoxicity. Also, when calcium disodium edetate is used alone, it may aggravate symptoms in patients with very high blood lead levels. When clinical symptoms consistent with lead poisoning or when blood lead levels are greater than 70µg/dL, it is recommended that calcium disodium edetate be used in conjunction with BAL. Administration of BAL prior to calcium $CaNa_2EDTA$ is necessary to prevent the redistribution of lead into the central nervous system. $CaNa_2EDTA$ is the only agent recommended for intravenous chelation therapy for children.[97]

British Anti- Lewisite (BAL, Dimercaprol):

BAL is the prototype chelator. A bisulfide, lipid-soluble drug must be administered intramuscularly at a dose of 300 - 450 mg/m^2/day. It has the typical sulfide odor, and patients often complain of the taste and bad feeling when the drug is administered.

2, 3 Dimercaptosuccinic Acid (DMSA or Succimer):

Succimer is a white crystalline powder with a molecular weight of 182 daltons and a characteristic sulfur taste and odour. Succimer is the meso form of 2, 3 dimercaptosuccinic acid. It is a water soluble analog of dimercaprol. However, it has a wider therapeutic index and has advantages over dimercaprol and $CaNa_2EDTA$. Succimer is a four carbon molecule

with two carboxyl groups and two sulfur groups. Lead binds to adjoining sulfur and oxygen atoms to form a complex that is excreted from the body. It is administered orally and is the most commonly used chelating agent in the USA. Succimer has few side effects and has replaced D-Penicillamine as an oral agent since 1991. It is the preferred agent in mild lead poisoning cases. Succimer is generally well tolerated. Its common side effects are gastrointestinal in nature including nausea, vomiting, flatus, diarrhea and a metallic taste in about 10-20% of patients.

D-Penicillamine:

Penicillamine is a D-B, B-dimethylcysteine, a penicillin degradation product. It has a sulfhydryl group which combines with lead to form a chelate that is readily excreted. It is a potent lead chelator that is administered orally. D-penicillamine is also used in the treatment of poisoning due to mercury, gold, zinc and copper and is the drug of choice for treating Wilson's disease. In addition, it is used in the treatment of cystenuria and rheumatic disorders. It has been used since 1957 for the treatment of lead poisoning and was the only oral chelator for lead until the availability of DMSA.

Penicillamine is absorbed rapidly, but has an oral bioavailability of 40 to 70%. It is not dose dependent. Food, antacids, and iron decrease absorption. It is given 4 times daily at a dose of 25 to 30 mg/kg/dose for several months. Since the introduction of penicillamine, its use has been limited due to the significant adverse effects that result. This has led to the development of the thiol chelators (DMSA and DMPS) which are considered safer alternatives. Side effects of D-penicillamine include adverse reactions that are attributable to zinc deficiency such as skin lesions on pressure points, desquamations, delayed wound healing, alopecia and sometimes glossitis, and stomatitis.

OTHER SUPPORTIVE MANAGEMENT

Other supportive management may be indicated depending on the presentation. If lead encephalopathy is present, anticonvulsants may be given to control seizures, and treatments to control swelling of the brain may include corticosteroids and mannitol. In case of severe dehydration, IV fluids are given and if the child presents with severe anemia, blood transfusion should be given.

REMEDIATION

Remediation implies the application of a remedy, and the ultimate objective of this remedy is hazard reduction. It refers to the restoration of a contaminated environmental site to a condition that is not a threat to human health or other forms of life. In some cases, a great deal of effort is spent to clean up contaminated sites. In other cases, sites are so contaminated that they can only be fenced off and isolated as much as possible from the rest of the environment. The remedy is expected to minimize the hazard to the extent that the environment is safe for human habitation and ecological habitability.[99] Environmental remediation has been included as part of case management program for children with blood lead levels ≥15 µg/dL from 1995.[100] In lead poisoning, the use of the term 'remediation' applies to surface water, underground water, air, soil, sediments and other parts of the environment that have become contaminated through any of the sources of lead in the environment such as mining, transport and processing of ore, battery manufacturing and recycling, paints, glass manufacturing, among others. Before environmental remediation is conducted, sampling of the environment is carried out so as to eliminate bias.

USA Environmental Protection Agency (EPA) has set standards for lead in drinking water, air, paint and soil. EPA's action level for lead in water delivered to users of public drinking water systems is 15µg/L. Its Maximum Contaminant Level Goal (MCLG) is zero. This is the

level determined to be safe by toxicological and biomedical considerations. The OSHA Lead Standard specifies the permissible exposure limit (PEL) of lead in the workplace, the frequency and extent of medical monitoring, and other responsibilities of the employer. OSHA has set a PEL (enforceable) of lead in workplace air at 50µg/m^3 averaged over an 8hour workday for workers in general industry. For those exposed to air concentrations at or above 30µg/m3 for more than 30 days per year, OSHA mandates periodic determination of BLLs. If a BLL is found to be greater than 40µg/dL, the worker must be notified in writing and provided with a medical examination. If a worker's one-time BLL reaches 60µg/dL (or averages 50 µg/dL or more on three or more tests), the employer is obligated to remove the employee from excessive exposure, with maintenance of seniority and pay, until the employee's BLL falls below 40µg/dL.

Lead contaminated soil can pose a risk through direct ingestion, uptake in vegetable gardens, or tracking into homes. Uncontaminated soil contains lead concentrations less than 50 ppm (50mg/kg or 0.005%) but soil lead levels in many urban areas exceed 400 ppm.[101] The EPA's standard for lead in bare soil in residential/play areas is 400 ppm by weight and 1200 ppm for no-residential/non-play areas. For paints, the CPSC has limited since 1977 the lead in most paints to 0.06% (600 ppm by dry weight). Paint for bridges and marine use may contain greater amounts of lead. For food items, various levels of concern have been set based on the calculation of the amount of lead a person can consume without ill effects, for instance, FDA has set an action level of 0.5 µg/mL for lead in products intended for use by infants and children and has banned the use of lead-soldered food cans.[102]

There are many techniques for environmental remediation. Generally speaking, there are 3 major types of remediation: biological (bioremediation) which involves the use of biological agents, physical remediation which involves the use of energy and that which does not change the physico-chemical properties of the lead accumulated in the soil to be cleaned and

chemical remediation which involves the use of chemical agents. These methods may be used singly or in conjunction with one another. Remediation can be further classified into *in situ* and *ex situ* remediation. *In situ* remediation involves treating the contaminated material at the site, e.g. solidification and stabilization, etc. On the other hand, *ex situ* remediation method involves the removal of the contaminated material to be treated elsewhere[103] e.g. excavation, underground water pump and treat, etc. *Ex situ* remediation represents the future of remediation process.

Summary of recommendations for children with confirmed (venous) elevated blood lead levels[104]

BLOOD LEAD LEVEL (µg/dL)				
10-14	15-19	20-44	45-69	≥70
Lead education -Dietary -Environmental **Follow-up blood** **Lead monitoring**	**Lead education** -Dietary -Environmental **Follow-up blood lead monitoring** **Proceed according to actions for 20-44µg/dL if**: -A follow-up BLL is in this range at least 3 months after initial venous test or -BLLs increase	**Lead education** -Dietary -Environmental **Follow-up blood lead monitoring** **Complete history and physical exam** **Lab work:** -Hemoglobin or hematocrit -Iron status **Environmental investigation** **Lead hazard reduction** **Neurodevelop-mental monitoring** **Abdominal X-ray** (if particulate lead ingestion is suspected) **with bowel decontamination if indicated**	**Lead education** -Dietary -Environmental **Follow-up blood lead monitoring** **Complete history and physical exam** **Lab work:** -Hemoglobin or hematocrit -Iron status -FEP or ZPP **Environmental investigation** **Lead hazard reduction** **Neurodevelop-mental monitoring** **Abdominal X-ray with bowel decontamination if indicated** **Chelation therapy**	**Hospitalize and commence chelation therapy** **Proceed according to actions for 45-69µg/dL**

The following actions are NOT recommended at any blood lead level:
Searching for gingival lead lines , Testing of neurophysiologic function, Evaluation of renal function (except during chelation with EDTA), Testing of hair, teeth, or fingernails for lead, Radiographic imaging of long bones and X-ray fluorescence of long bones

SCHEDULE FOR FOLLOW-UP BLOOD LEAD TESTING[105]

Venous blood lead level (µg/dL)	Early follow-up (first 2-4 tests after identification)	Late follow-up (after BLL begins to decline)
10-14	3 months	6-9 months
15-19	1-3 months	3-6 months
20-24	1-3 months	1-3 months
25-44	2 weeks-1 month	1 month
> 45	As soon as possible	Chelation with subsequent follow-up

2.12.2. Prevention of lead poisoning

Lead poisoning is entirely preventable. Generally speaking, there are three major preventive strategies of lead poisoning:

Primary prevention: By far, this is the most important lead poisoning prevention strategy which entails preventing children from becoming exposed to lead. It consists of environmental inspection and abatement, and lead removal from soil before a child develops lead poisoning. However, because of the prohibitive cost of primary prevention, it is very difficult to implement. Prevention has therefore focused mainly on secondary efforts.

Secondary Prevention: Under this principle, because lead poisoning is asymptomatic in its early phase the goal is to identify children who have developed lead poisoning in order to prevent their lead from rising further or causing more harm and to prevent other siblings and their play mates from getting exposed to lead. In addition, it aims at removing the child from the source of exposure and/or removing the source from the individual. These can be achieved through the following:

- Screening: this is the most effective means of reducing or eliminating the effects of lead poisoning aimed at identifying the disease at its early stage. It involves the search of

disease in apparently healthy subjects. It can be "universal screening" which comprises of BLL screening of all children in an area or "targeted screening" in which BLL screening is carried out on children selected based on individual and/or environmental risk assessment.

- Early diagnosis and prompt treatment: this is aimed at removing the lead from the child's body and stopping further damage. It is achieved through removal of or from the source of contamination, improved nutrition and chelation therapy.

Tertiary Prevention: This is aimed at prevention of complications and limiting disabilities as a result of lead poisoning such as deafness and mental retardation.

Other preventive strategies:

- **Health education**: The educational component of the program is aimed at enhancing primary and secondary prevention of childhood lead poisoning. It works towards increasing awareness and knowledge of the community on childhood lead poisoning and facilitates the adoption of preventive behaviors.
- **Monitoring/surveillance:** these activities provide information that forms the basis for planning, evaluation, and public support of policies and programs. Activities include development of systems to monitor children's BLLs, sources of exposure, lead hazards reduction among others.
- **Safer Mining Practices:** the mining industry is among the largest and lucrative industries and has contributed tremendously to economic and commercial development, but it is one of those industries which has created significant damage and degradation to the environment. Today, it is widely recognized and acknowledged and has been universally accepted that there is a need for balancing the benefits of mining with that of preventing damage and harm to the environment and humans. In this regard, introducing low-tech safe mining practices like grinding

rock outside the villages, wet-method of grinding rocks to contain the dust, use of PPE and making sure that workers have a change of clothes or showers after work so that they don't go home and contaminate their children could save lives.

- **Legislation:** laws and regulations governing mining activities have been developed to safeguard the environment and health of the people. These laws should be enforced and appropriate sanctions be applied to defaulters.

2.13. Complications

Lead affects all the organ systems of the body. Its complications include encephalopathy, mental retardation, behavioral problems, lowered IQ, learning disabilities, cataract, tooth decay, deafness, hypertension, abdominal colic, nephropathy, Fanconi syndrome, male and female reproductive disorders such as oligospermia, miscarriages and low birth weight, anemia, stunted growth, impaired immune system among others.

2.14. Prognosis

The outcome of lead poisoning depends on the extent and duration of lead exposure. When lead exposure ceases, the effects of lead on the physiology, blood and kidneys are reversible, while its effects on the nervous system are in most cases irreversible particularly in children.[106] About 70-80% of children that had lead encephalopathy develop permanent brain damage, including those that receive the best of treatment.

Lead poisoning has been associated with decreased life span. Mortality from variety of other causes such as cancers, stroke and heart disease has been found to be higher among those with elevated BLL. The mortality rate for people who develop cerebral involvement is about 25%, and of those that survived about 40% have permanent neurological problems such as cerebral palsy.

CHAPTER 3

METHODOLOGY

3.1. Study Area

The study was conducted in Kawaye village, Anka Local Government Area of Zamfara State. Anka LGA is one of the 14 LGAs of the state which is located about 120km from the State capital, Gusau at 12°06′30″N and 5°56′00″E. The LGA has an area of 2,746km^2, 10 political wards, a total population of about 142,280[107] and a total under 5 years population of about 33,310. It is bounded by Bakura and Talata Mafara on the north, Maru on the south-east, and Bukkuyum on the west.

Kawaye village where the study was conducted is a rural community which has an estimated total population of about 8,608 and under 5 population of 1,522 (2006 Census). The people are predominantly Muslims of Hausa/Fulani tribes with few other tribes such as Gwari, Kambari, Zabarmawa and Igbo. Their main occupation is farming, however, a proportion of the people are engaged in mining of gold ore; few are civil servants and traders. The type of family structure in the community is mainly monogamy, but a few have polygamous family structure. The community has one dispensary, one primary school and a market that holds on Fridays. The village has neither tarred roads nor electricity supply; there is no mobile telecommunication network in the area as well. The major sources of drinking water in the community are bore-holes and wells.

3.2. Study Design

A descriptive cross sectional study design was employed.

3.3. Study Population

The study population comprised of children aged 6 years and below from Kawaye village in Anka LGA of Zamfara State. Subjects and their parents were recruited into the study based on the criteria stated below.

3.3.1. Inclusion criteria

- Willingness of parent to give written or oral consent for the study,
- Child aged 6 years and below,
- Child must be resident in Kawaye village for at least 10 months (child younger than 10 months of age whose mother/caregiver has been resident in the village for at least 10 months will be included in the study).

3.3.2. Exclusion criteria

- Subject on chelation therapy,
- Subject critically ill,
- Age > 6 years,

3.4. Study Period

The study was conducted between December 2012 and April 2013.

3.5. Sample Size Determination

The sample size was determined using the formula:

$n_o = z^2 pq/d^2$ [110]

Where:

n_o = minimum sample size

Z = Standard normal deviate at α probability (1.96)

P = Prevalence of lead poisoning in children in Nigeria (25%) [51]

q = 1-p (1-0.25 = 0.75)

d = precision (0.05)

Therefore, $n_o = \frac{(1.96)^2 \times 0.25 \times 0.75}{(0.05)^2} = 288.12 \approx 288$

Finite population correction for proportions:

Since the population of Kawaye village is small (<10,000 people), the sample size (n_o) was adjusted using the formula:

$$n = \frac{n_o}{1 + \frac{(n_o - 1)}{N}}$$ 111

Where n = corrected sample size

n_o = calculated sample size above = 288

N= Population of the study site (Kawaye village) = 8,608

Therefore, $n = \frac{288}{1 + \frac{(288 - 1)}{8,608}} = 288/1.033 = 278.8 \approx 279$

The sample size was inflated by 10% to account for potential non response:

n = 279 + 27.9 = 306.9 ≈ 307

3.6. Sampling

Simple random sampling technique was used to recruit participants into the study. First and foremost, a list of all the households in entire community in which the study was conducted was

obtained from Kawaye PHC (Source: PRINN MNCH Enumeration Exercise, 2011). The sampling units were the households. A household was defined as a domestic unit consisting of at least a husband and wife living under the same roof and sharing at least a single meal per day. One mother-child pair was recruited into the study per house-hold (including polygamous setting). In a household where there were many eligible mothers/children, balloting was used to select one mother/child. Computer generated random number table was used to select the desired sample size, the direction for reading the table was determined before looking at the table.

3.7. Study Instruments/Data Collection Tools

The instruments used in carrying out this study are enumerated below:

- Questionnaires: the questionnaire was semi-structured, having 5 sections as follows:
    - Section A- Identification: comprised of questionnaire ID, date, compound ID, interviewer's name, etc.
    - Section B- Socio-demographic information: comprised of socio-demographic information of the child's father/guardian such as age, sex, religion, occupation, socioeconomic status and educational status.
    - Section C- Mother and child information: this sections comprised of child's and his mother's/caregiver's socio-demographic information and signs and symptoms of lead poisoning.
    - Section D- Exposure history: this section assessed history of child's exposure to lead poisoning.
- Proforma/Spread sheet: used for line listing of laboratory findings

3.8. Data Collection Methods

Data were collected using interviewer-administered pretested semi-structured questionnaires. Four interviewers who are Community Health Extension Workers, two phlebotomists who work

at the State's Blood Lead and Inorganic Metals Laboratory as laboratory technicians and two environmentalists from Zamfara State Environmental Sanitation Agency (ZESA) were recruited and trained for a duration of 3 days. The training started with a pretest to assess the research assistants' baseline knowledge on lead poisoning, phlebotomy and questionnaire administration. The training covered areas like etiology, epidemiology, clinical presentations, complications, treatment and prevention of childhood lead poisoning, questionnaire/questionnaire administration, phlebotomy and safety precautions among others.

Questionnaires were then pre-tested and validated. The pretested questionnaires were administered on the study subjects that have consented to participate in the study. Socio-demographic information as well as information on the subjects' knowledge, practice and beliefs with regards to lead poisoning, risk factors for lead poisoning, history of exposure to lead, signs and symptoms of toxicity, history of possible complications from lead poisoning and history of participation in ore mining and/or ore processing were obtained. Subsequently, blood samples were collected for determination of blood lead levels using Lead Care II analyzer, while soils were tested for lead contamination, directly, using X Ray Fluorescence (XRF) machine. Packed Cell Volume (PCV) and Malaria Parasites (MP) tests were carried out on the collected blood samples to determine anemia and malaria parasitemia respectively.

3.9. Data Quality

To ensure data quality, all filled questionnaires were checked for errors. The data were cleaned using filters to check for missing and logically impossible findings/records. Frequency tables, bar charts and histograms were also used to check for unlikely values and outliers. Resort was made to original questionnaires and records to verify unlikely and problematic entries.

3.10. Data Management

3.10.1. Measurement of variables

Exposure variables

The following exposure variables were obtained from the study- age, sex, occupation, educational status, socio-economic status, household floor type, flaky house paint, house-hold soil lead level (SLL), use of kohl eye cosmetic, malaria parasitemia and anemia.

Outcome variables

The outcome variable was elevated blood lead level (EBLL) of ≥ 5µg/dL (lead poisoning).

Possible confounders

Age, sex, use of kohl eye cosmetic, living in painted house/flaky house paint, pottery and battery recycling.

3.10.2. Statistical analyses

The data obtained was entered into Microsoft Excel and Epi info (version 3.5.3, CDC Atlanta) and analyzed. Statistical tests of Chi-square and regression analyses were performed as appropriate. The findings were presented and discussed appropriately in chapters 4 and 5 respectively.

3.11. Venous Blood Specimen Collection

Blood collection criteria: blood specimen were collected from children that satisfied the following criteria

- Sick children/history of convulsion and/or
- Children ≤ 6 years old,

Tools for determining BLL, SLL, Anemia and Malaria parasitemia

1. LeadCare II Blood Lead Analyzer (to be obtained from Zamfara State Ministry of Health)
2. LeadCare II Blood test Strip
3. Control sample
4. Diluent
5. EDTA-Vacutainer bottle
6. Reagent tubes (pre-containing 250 µL of 0.34M HCl in de-ionized water)
7. Plunger
8. Plain non-heparinized capillary tubes
9. Clean glass slide
10. Slide box
11. Lead Pencil
12. Surgical gloves
13. Face masks
14. Alcohol swipes
15. Syringes
16. Cotton wool
17. Gauze
18. Adhesive plaster
19. Tourniquet
20. X-Ray Fluorescence (XRF) Spectrophotometer (to be obtained from Zamfara State Ministry of Health/Environment)
21. Brush (to be obtained from Zamfara State Ministry of Health/Environment)
22. Scoop (to be obtained from Zamfara State Ministry of Health/Environment)
23. Ziploc bags
24. Safety boxes

25. First aid box

Sample collection procedures

1. Vacutainer/blood collection tubes were labeled with unique identification details: sample ID, child's name, child's age, mother's name and sample collection date,
2. Gloves will be put on,
3. A tourniquet was placed above the venipuncture site.
4. The vein was palpated and located,
5. The venipuncture site was meticulously disinfected with 70% alcohol by swabbing the skin starting from the center of the venipuncture site and moving outwards. The disinfectant was allowed to evaporate. The vein was not re-palpated,
6. Venipuncture was performed with a vacuum venipuncture system,
7. The desired volume of fresh whole blood was drawn directly into EDTA Vacutainer tube (bottle). At least 2-3ml of blood was drawn (otherwise this may lead to obtaining falsely lower blood lead results),
8. The tourniquet was removed. Clean and dry cotton was used to apply pressure to the venipuncture site until bleeding stopped and then plaster was applied,
9. The tubes were mixed thoroughly to make sure the blood sample does not contain clots,
10. Used sharps were never be recapped. They were discarded directly into sharps container,
11. It was rechecked to ensure that the tubes used for sampling have been correctly labeled,
12. After collecting all the samples, the laboratory line list form was completed using the same identification details used on the tubes,
13. Only fresh whole blood was used. Samples were NOT refrigerated throughout, they were maintained at 10 - 32^{o} C,
14. It was ensured that the blood specimens were processed within 24 hours of collection (otherwise falsely lower results may be obtained),
15. **Gloves were changed after each blood collection.**

3.12. Lead Care II Blood Test Protocol

1. The treatment reagent tubes (pre-containing 250 µL of 0.34M HCl in de-ionized water) were labeled as appropriate - Compound ID, father's or mother's number, father's or mother's name, child's name, child's age (months), sample collection date and a new Lab No.

2. The Lead Care II analyzer was turned ON and the machine was allowed to finish self-test (until 'PREPARE SAMPLE' appeared).

3. The machine was calibrated using the magnetic button provided. The magnetic calibration button was placed on the calibration reader and kept in place until "CALIBRATION SUCCESSFUL" appeared.

4. The LOW control sample from the vials in the kit was prepared. Using the capillary tubes provided, 50 µL of the control sample was removed, ensuring that there were no gaps or bubbles. Gauze pads were used to clean/remove any excess sample and the control sample was transferred it into a pre-labeled reagent tube using a plunger and mixed very well.

5. The microsensor strip was inserted into LC II machine first halfway, until the sample was ready for immediate placement into the microsensor.

6. Transfer dropper was squeezed and inserted into the bottle containing the prepared sample to collect a reasonable amount of the prepared sample.

7. At this stage, the microsensor strip was inserted completely into the LC II machine until a BEEP sound appeared, which meant the sample MUST now be dropped into the sensor immediately.

8. A FULL drop (about 50 µL) of the control sample (in 6 above) was placed on the X position of microsensor. Another BEEP sound appeared indicating that the sample placement on the strip and the volume applied was correct.

9. The analyzer will now start reading, showing a countdown of 180 seconds, after which the blood lead result will be displayed in µg/dL.

10. Steps 4 - 9 were repeated with the HIGH control sample and ensuring that CONTROL results fell within the acceptable ranges (LOW = 5.2 - 11.2 µg/dL and HIGH = 20.3 - 28.7 µg/dL).

11. All control results were recorded on the blood lead control results form.

12. Steps 4 - 9 were repeated with all the subsequent blood samples and all results were recorded on the Lab. line list form.

13. It was ensured that the work was organized properly such that the NEXT sample preparation was sorted while waiting for the analyzer to read the current sample within 3 minutes.

14. For samples that were higher than 65 µg/dL, they were DILUTED at **1 in 3** dilution ratio, using an original LOW sample (having a BLL reading of <3.3 µg/dL) as diluent. Final dilution contained **125 µl** of freshly prepared sample found to be HIGH (>65 µg/dL) **+ 250 µl** of the diluent - giving a total volume of **375 µl.** The dilution factor is **3**, thus, results obtained were multiplied by **3** in order to get the actual value of blood lead level (concentration in µg/dL).

15. For maintenance, the sensor area and all its surroundings were cleaned using alcohol wipes before and after each run and following any accidental spillage(s) and/or when the machine was dirty.

16. FINALLY, the LOW and HIGH controls were run again (steps 4 - 11) to ensure that the machine was giving precise results.

<u>Reference:</u> Adapted from Lead Care II Blood Test Kit. ESA Biosciences, Inc., Chelmsford, MS, USA.

Laboratory line list form for recording BLL results:

Laboratory Line List Form

Lab No.	Date	Compound ID	Mother's Name	Mother's Number	Child's Name	Child's Age (month)	Child's Sex	Initial Sample Results (µg/dL)	Diluted Sample Results (µg/dL)	Final Sample Results (µg/dL)

3.13. Environmental Sampling

Altogether, 307 households were tested for soil lead levels. Sites where soils were tested for presence of lead in the households included the main entrance, children playing area, children sleeping area, cooking area, washing area, common area and other areas such as under the tree, drinking water pot area, wall plaster, animals area, etc. At least four of the mentioned areas above were tested. The mentioned areas were tested for lead directly using a portable, hand-held X-Ray Fluorescence spectrometer. Findings were recorded in the laboratory line list form provided in the questionnaire for the household in question (appendix VI B).

3.14. Ethical Considerations

The study involved collection of blood specimens from selected children. There was little risk associated with collection of the blood specimen. The child might feel a slight pinch, slight pain, possible dizziness or may faint when the blood was being drawn from an arm. Children may also develop slight bruise or infection following the venipuncture. However, these risks were mitigated by careful selection of vein to be used for collection of specimen, skin disinfection prior to specimen collection and new sterile instruments that were clean and completely safe were used to collect blood samples. In addition, first aid box containing adequate resuscitation materials were always made available during specimen collection procedures.

3.14.1. Ethical clearance

Ethical approval to conduct this research was obtained from the Scientific and Health Research Ethics committee of Ahmadu Bello University, Zaria and Zamfara State Ministry of Health.

3.14.2. Informed consent

Written informed consent was obtained from the parents/guardians of each study participant.

3.15. Limitations

Some of the limitations of this study were:

1. The study did not determine the type of anemia due to financial/logistical reasons.
2. The causes of anemia were not elucidated.
3. The study only assessed for malaria parasitemia but did not ascertain those children actually having malaria fever.

CHAPTER 4

RESULTS

This study carried out in Kawaye village, Anka LGA of Zamfara State focused on finding out the determinants and prevalence of childhood lead poisoning in the village.

The results obtained from the study are given below:

4.1. Baseline Socio-demographic Data

Table 1: Socio-demographic characteristics of children and their fathers/guardians

This is a table showing socio-demographic characteristics of studied children and their fathers and/or guardians

Characteristic	Frequency	Percent
Gender of children		
Male	157	51.1
Age group of children (months)		
0-11	16	5.2
12- 23	39	12.7
24-35	61	19.9
36-47	51	16.6
48-59	66	21.5
≥ 60	74	24.1
Father's/guardian's occupation		
Farmer	114	37.1
Miner	45	14.7
Cattle rearer	39	12.7
Businessman/ Businesswoman	32	10.4
Civil servant	30	9.8
Petty trader	7	2.3
Others: Tailor, driver, mechanic, student, etc	40	13.0
Father's/guardian's educational status		
None	13	4.2
Informal	165	53.7
Primary	47	15.3
Secondary	53	17.3
Tertiary	29	9.4

There was preponderance of male gender among the study subjects. Mean age of children = 38.5 months ± 18.5 SD. Most of the children belonged to age groups 48-59 and ≥ 60 months. Parents/guardians of the studied children were predominantly farmers and miners, with more than half of them having informal education; few (4.2%) had no education at all.

The figure below depicts the distribution of socio-economic status of fathers/guardians of studied children based on poverty level and minimum wage of Federal Government of Nigeria.

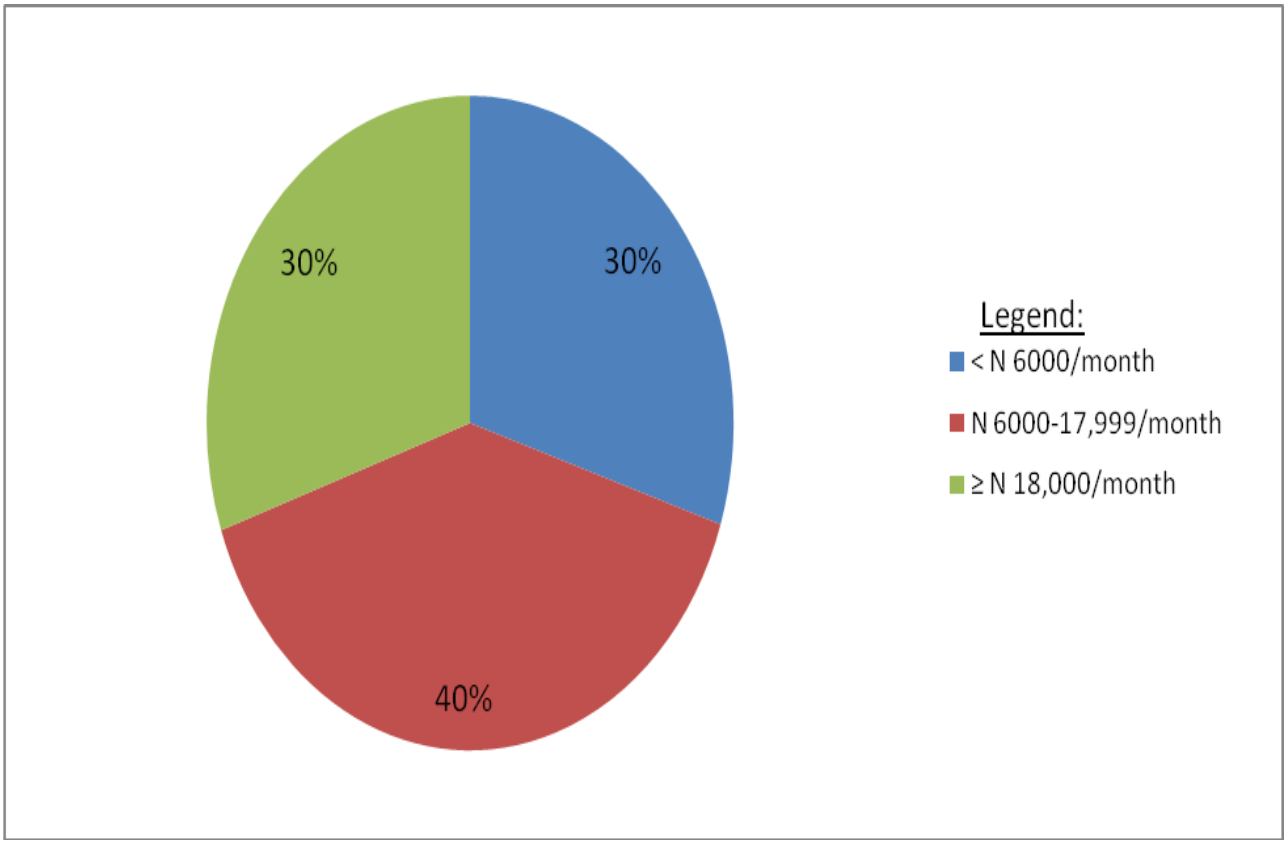


Figure 1: Distribution of children according to father's/guardian's socio-economic status

Nearly one-third of the parents/guardians of recruited children lived below poverty line, more than one-third (40%) earned at least the amount set as minimum wage by the Federal Government of Nigeria. About one-third earned above the minimum wage.

4.2. Prevalence Of Lead Poisoning And Associated Factors

Table 2: Prevalence of childhood lead poisoning

The table below shows the prevalence of childhood lead poisoning in Kawaye village.

BLL	Count	Percent
< 5 µg/dL	23	7.5
≥ 5 µg/dL	284	92.5
Total	**307**	**100**

Median BLL= 19.7 µg/dL, Range= <3.3- 372.3

The prevalence of lead poisoning among children in the community was very high (92.5%).

Table 3: Distribution of Households according to Soil Lead Levels (SLL)

The table below shows the prevalence of soil contamination with lead in the sampled households.

SLL	Count	Percent
< 400PPM	263	85.7
≥ 400PPM	44	14.3
Total	**307**	**100**

Median household SLL = 61PPM, range= 0-24000PPM

About 14% of the households in the community had soil lead levels exceeding the limit set by EPA for residential areas (<400PPM).

Table 4: Pattern of Household Soil contamination by lead

This table depicts the pattern of soil contamination with lead in the sampled households in Kawaye village.

SLL	Count	Percent
< 400PPM	263	85.7
400-1200PPM	35	11.4
>1200PPM	9	2.9
Total	**307**	**100**

Eighty six percent of the households had normal lead levels, while about 11% had mild-moderate contamination, whereas 2.9% were severely contaminated.

The figure below shows the prevalence of anemia amongst the studied children in Kawaye village.

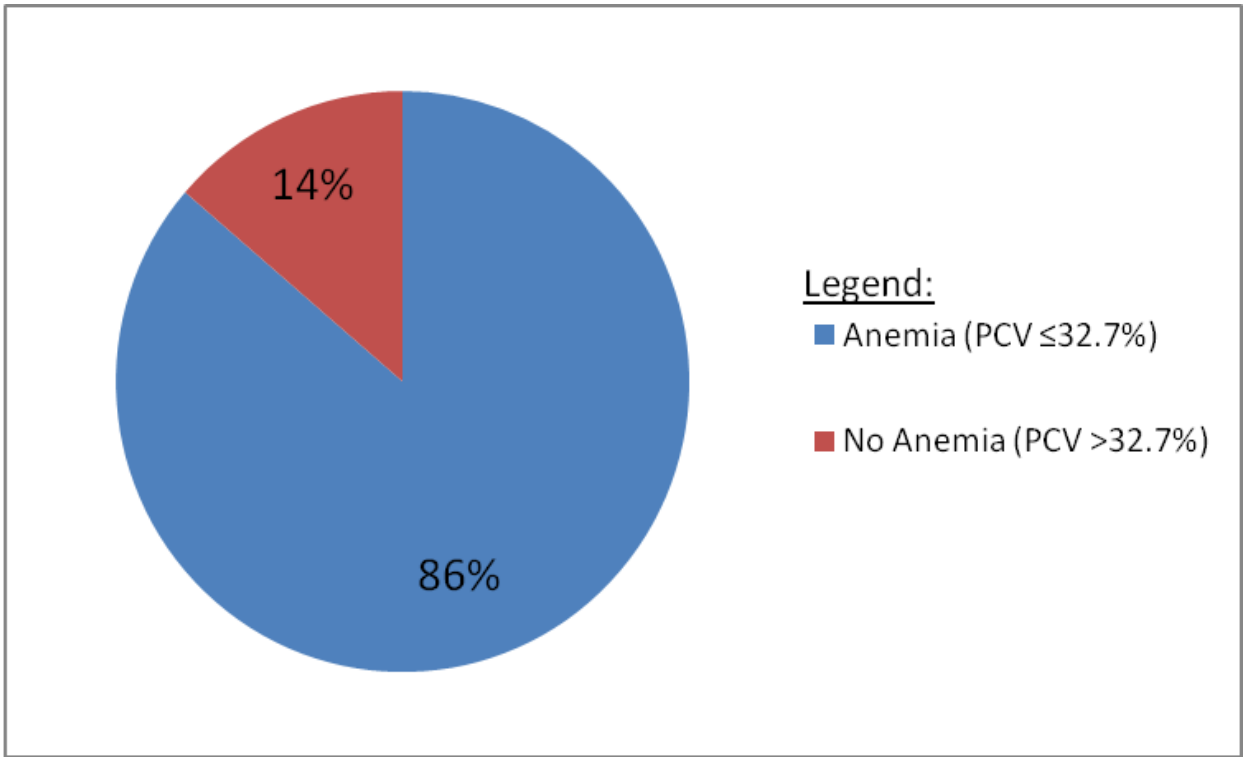


Figure 2: Prevalence of anemia among studied children

Mean PCV level= 26.9% ± 5.5SD. About 86% of the children had varying degrees of anemia.

This figure depicts the prevalence of malaria parasitemia among children in Kawaye village.

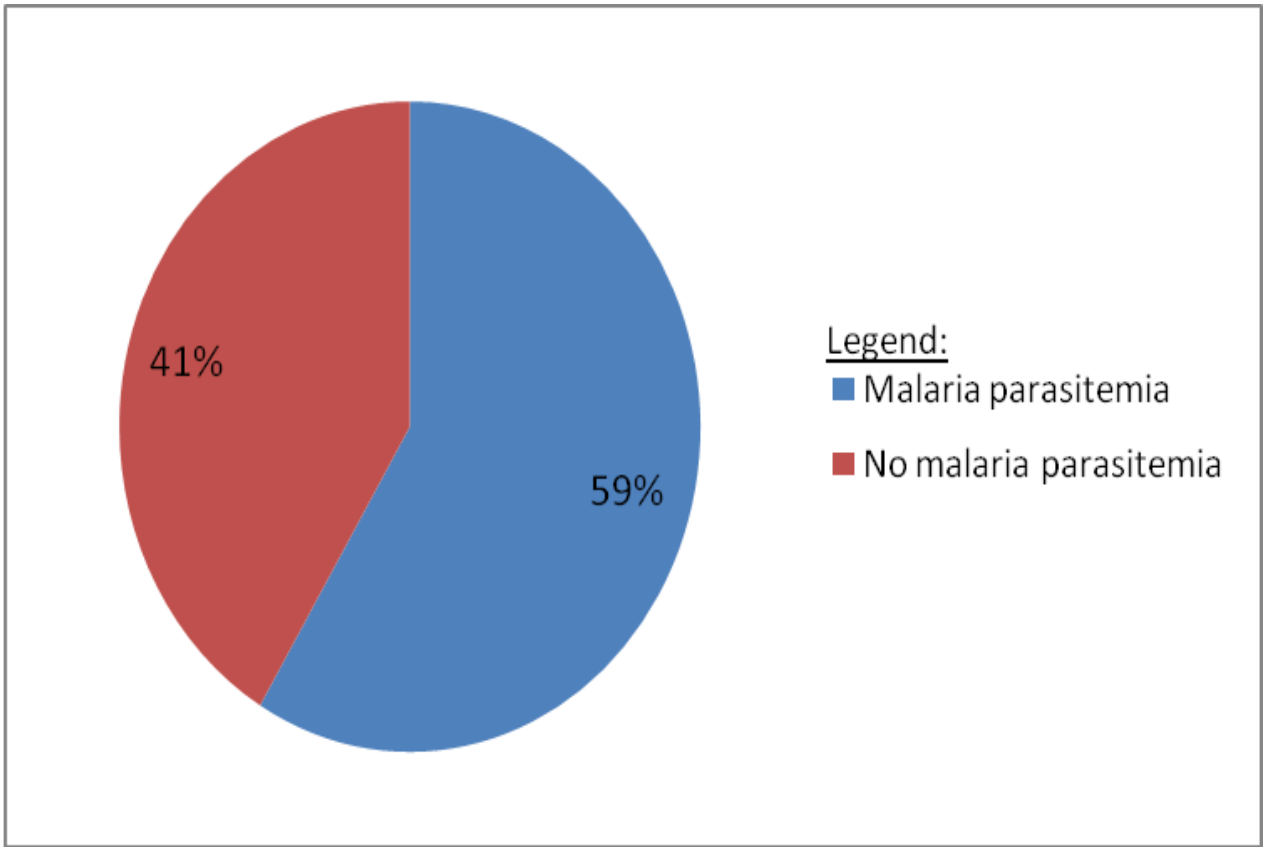


Figure 3: Prevalence of malaria parasitemia among studied children

Median parasite density = 40 parasites/µL, range = 0-2800 parasites/µL

About 59% of the children had variable levels of malaria parasitemia ranging from mild to moderate parasitemia.

Table 5: Age and lead poisoning

The table below shows the relationship between age of child and lead poisoning in Kawaye village.

Age group (months)	Diseased :Blood lead level (BLL) ≥ 5 µg/dL Count (%)	Not Diseased: Blood lead level (BLL) <5µg/dL Count (%)	Total
0-11	16 (100)	0 (0)	**16**
12- 23	36 (92.3)	3 (7.7)	**39**
24-35	59 (96.7)	2 (3.3)	**61**
36-47	47 (92.2)	4 (7.8)	**51**
48-59	58 (87.9)	8 (12.1)	**66**
≥ 60	68 (91.9)	6 (8.1)	**74**
Total			**307**

$\chi 2 = 4.95$, $p = 0.4219$

Lead poisoning was higher among children aged 35 months and below. However, this association between age and lead poisoning is not significant statistically.

Table 6: Age and severity of lead poisoning

This table shows the relationship between the age studied children and severity of lead poisoning in Kawaye village.

Age group (months)	Degree of lead poisoning (BLL) in µg/dL				Total
	5-19.9 Count (%)	20-24.9 Count (%)	25-44.9 Count (%)	≥45 Count (%)	
0-11	11 (68.8)	0 (0)	4 (25)	1 (6.3)	**16**
12- 23	12 (33.3)	5 (13.9)	12 (33.3)	7 (19.4)	**36**
24-35	21 (35.6)	10 (16.9)	17 (28.8)	11 (18.6)	**59**
36-47	24 (51.1)	2 (4.3)	15 (31.9)	6 (12.8)	**47**
48-59	27 (46.6)	11 (19)	18 (31)	2 (3.4)	**58**
≥ 60	38 (55.9)	7 (10.3)	14 (20.6)	9 (13.2)	**68**
Total					**284**

$\chi 2 = 23.45$, $p = 0.0751$

Prevalence of sub-clinical lead poisoning appeared to increase with increasing age; however, this trend was not demonstrated with mild, moderate and severe lead poisoning. The association is not significant statistically.

Table 7: Gender and lead poisoning

The table below shows the relationship between gender of a child and lead poisoning in Kawaye village.

Gender	Diseased :Blood lead level (BLL) ≥ 5µg/dL Count (%)	Not Diseased :Blood lead level (BLL) <5µg/dL Count (%)
Male	145 (51.1)	12 (52.2)
Female	139 (48.9)	11 (47.8)
Total	**284 (100)**	**23 (100)**

$\chi 2 = 0.0129$, $p = 0.4547$

The prevalence of lead poisoning was higher among males; however, this association is not statistically significant.

Table 8: Gender and severity of lead poisoning

This table depicts the relationship between gender of a child and severity of lead poisoning in Kawaye village.

Gender	**Degree of lead poisoning (BLL) in µg/dL**				**Total**
	5-19.9 Count (%)	20-24.9 Count (%)	25-44.9 Count (%)	≥45 Count (%)	
Male	67 (46.2)	11 (7.6)	40 (27.6)	27 (4.8)	**145**
Female	66 (47.5)	24 (17.3)	40 (28.8)	9 (6.5)	**139**
Total					**284**

$\chi 2 = 13.72$, ***p* = 0.0033**

All forms of lead poisoning were higher among females. This association is statistically significant.

Table 9: Educational status of father/guardian and childhood lead poisoning

The table below shows the relationship between educational status of the child's father/guardian and childhood lead poisoning in Kawaye village.

Educational status of father/Guardian	**Diseased :Blood lead level (BLL) ≥ 5 µg/dL Count (%)**	**Not Diseased: Blood lead level (BLL) <5µg/dL Count (%)**	**Total**
None/ Informal	169 (94.9%)	9 (5.1%)	**178**
Formal	115 (89.1%)	14 (10.9%)	**129**
Total			**307**

$\chi 2 = 2.84$, ***p* = 0.046**

The prevalence of lead poisoning appeared to be higher among those children whose parents/guardians had low educational level (none/informal). This association is statistically significant.

Table 10: Occupation of father/guardian and childhood lead poisoning

This table shows the relationship between occupation of the child's father/guardian and prevalence of childhood lead poisoning in Kawaye village.

Occupation of father/Guardian	**Diseased: Blood lead level (BLL) ≥ 5 µg/dL** **Count (%)**	**Not Diseased: Blood lead level (BLL) <5µg/dL** **Count (%)**	**Total**
Mining	41 (97.6%)	1 (2.4%)	**42**
Farming	113 (96.6%)	4 (3.4%)	**117**
Business	30 (93.8%)	2 (6.2%)	**32**
Civil servant	28 (93.3%)	2 (6.7%)	**30**
Petty trading	6 (85.7%)	1 (14.3%)	**7**
Cattle rearing	28 (71.8%)	11 (28.2%)	**39**
Other occupations: mechanic, driving, tailoring, student, butcher, etc	38 (95.0%)	2 (5%)	**40**
Total			**307**

$\chi 2 = 29.45$, ***p* <0.0001**

Prevalence of lead poisoning was highest among children whose parents/guardians were miners and farmers, while the prevalence was lowest among children whose parents were cattle rearers. The association between occupation and prevalence of childhood lead poisoning is statistically significant.

Table 11: Socioeconomic status of father/guardian and childhood lead poisoning

The table below shows the relationship between socioeconomic status of the child's father/guardian and prevalence of childhood lead poisoning in Kawaye village.

Socioeconomic status of father/Guardian	**Diseased :Blood lead level (BLL) ≥ 5 µg/dL Count (%)**	**Not Diseased :Blood lead level (BLL) <5µg/dL Count (%)**	**Total**
< ₦ 6000/month	88 (95.7)	4 (4.3)	**92**
₦ 6000-17,999/month	116 (95.1)	6 (4.9)	**122**
≥ ₦ 18,000/month	80 (86)	13 (14)	**93**
Total			**307**

$\chi 2 = 8.12$, $\boldsymbol{p = 0.0172}$

Children whose fathers/guardians lived below poverty line were found to be more affected by lead poisoning compared to those whose fathers/guardians lived above poverty line or earned at least the minimum wage approved by the Federal Government of Nigeria. These associations are statistically significant.

Table 12: Household ESLL and childhood lead poisoning

This table depicts the relationship between household soil contamination with lead and prevalence of childhood lead poisoning in Kawaye village.

SLL	**Diseased: Blood lead level (BLL) ≥ 5 µg/dL Count (%)**	**Not Diseased: Blood lead level (BLL) <5µg/dL Count (%)**	**Total**
Elevated SLL	44 (100)	0(0)	44 (100)
Not elevated SLL	240 (91.3)	23 (8.7)	263 (100)
Total			**307**

$\chi 2 = 0.0246$, ***p* = 0.0493**

All the children whose households had elevated SLL had lead poisoning of varying severity. This association is found to be statistically significant.

Table 13: Occupation of father/guardian and ESLL in households

The table depicts the relationship between occupation of the father/guardian and household soil contamination with lead in Kawaye village.

Occupation of father/Guardian	**Contaminated: SLL ≥400PPM Count (%)**	**Not contaminated: SLL <400PPM Count (%)**	**Total**
Mining	15 (36.6%)	26 (63.4%)	41
Petty trading	2 (28.6%)	5 (71.4%)	7
Business	5 (15.6%)	27 (84.4%)	32
Farming	14 (11.9%)	104 (88.1%)	118
Civil servant	3 (10.0%)	27 (90.0%)	30
Cattle rearing	1 (2.6%)	38 (97.4%)	39
Other occupations: mechanic, driving, tailoring, student, butcher, etc	4 (10.0%)	36 (90.0%)	40
Total			**307**

$\chi 2 = 23.79$, ***p* = 0.0006**

The households where the fathers/guardians were miners and petty traders appeared to have higher soil lead contamination, while households where the fathers/guardians were cattle rearers had lowest levels of soil lead contamination. This association is statistically significant.

Table 14: Malaria parasitemia and lead poisoning

The table below shows the relationship between malaria parasitemia among studied children and prevalence of childhood lead poisoning in Kawaye village.

Malaria parasitemia status	**Diseased: Blood lead level (BLL) ≥ 5 µg/dL Count (%)**	**Not Diseased: Blood lead level (BLL) <5µg/dL Count (%)**	**Total**
Malaria parasitemia	175 (97.2)	5 (2.8)	180
No malaria parasitemia	109 (85.8)	18 (14.2)	127
Total			**307**

$\chi 2 = 12.36$, ***p* = 0.0002**

Prevalence of lead poisoning was higher among children having malaria parasitemia compared to those without parasitemia. This association is found to be statistically significant.

Table 15: Malaria parasitemia and current history of fever

The table depicts the prevalence of fever among children with malaria parasitemia in Kawaye village.

Malaria parasitemia status	**Fever Count (%)**	**No fever Count (%)**	**Total**
Malaria parasitemia	24 (51.1)	156 (60)	
No malaria parasitemia	23 (48.9)	104 (40)	
Total	47 (100)	260 (100)	**307**

$\chi 2 = 0.97$, ***p* = 0.1626**

There was a preponderance of fever among children having malaria parasitemia. However, this association is not statistically significant.

Table 16: Degree of malaria parasitemia and severity of lead poisoning

The table depicts the relationship between the level of malaria parasitemia in the sampled children and severity of lead poisoning in Kawaye village.

Degree of Parasitemia	**Degree of lead poisoning (BLL) in µg/dL**				**Total (%)**
	5-19.9	20-24.9	25-44.9	≥45	
	Count (%)	Count (%)	Count (%)	Count (%)	
10-100/µL	78 (49.4)	20 (12.7)	40 (25.3)	20 (12.7)	158 (100)
101-10,000/µL	6 (27.3)	5 (22.7)	7 (31.8)	4 (18.2)	22 (100)
TOTAL					**180**

$\chi 2 = 4.18$, $p = 0.2422$

None of the sampled children had severe malaria parasitemia (parasite density >10,000/µL). Mild to severe lead poisoning were higher among children with moderate parasitemia. This association is not significant statistically.

Table 17: Anemia and lead poisoning

The table below shows the relationship between anemia and prevalence of childhood lead poisoning in Kawaye village.

Co-morbidity status	**Diseased :Blood lead level (BLL) ≥ 5 µg/dL Count (%)**	**Not Diseased :Blood lead level (BLL) <5µg/dL Count (%)**	**Total**
Anemia	249 (87.7)	16 (69.6)	
No anemia	35 (12.3)	7 (30.4)	
Total	**284 (100)**	**23 (100)**	**307**

$\chi 2 = 4.48$, ***p* = 0.0172**

The prevalence of lead poisoning was higher among children with anemia compared to those without. This association is statistically significant.

Table 18: Degree of anemia and severity of lead poisoning

The table depicts the relationship between degree of anemia in the sampled children and severity of lead poisoning in Kawaye village.

Degree of Anemia(PCV)[112]	**Degree of lead poisoning (BLL) in µg/dL**				**Total**
	5-19.9 Count (%)	20-24.9 Count (%)	25-44.9 Count (%)	≥45 Count (%)	
<21%	10 (8.8)	3 (9.4)	13 (18.3)	7 (21.9)	
21- 29.9%	77 (67.5)	15 (46.9)	47 (66.2)	23 (71.9)	
30- 32.7%	27 (23.7)	14 (43.8)	11 (15.5)	2 (6.3)	
TOTAL	**114 (100)**	**32 (100)**	**71 (100)**	**32 (100)**	**249**

$\chi 2 = 19.4$, ***p* = 0.0035**

All degrees of severity of lead poisoning were found to be higher among children with moderate anemia. This association is statistically significant.

Table 19: Co-morbidity status of children and lead poisoning

The table below shows the association between child's co-morbidity status and prevalence of lead poisoning in Kawaye village.

Co-morbidity status of child	Diseased :Blood lead level (BLL) ≥5µg/dL Count (%)	Not Diseased: Blood lead level (BLL) <5µg/dL Count (%)	OR (95% CI)
No co-morbidity	13 (4.6)	5 (21.7)	Ref. = 1
Malaria parasitemia	22 (7.7)	2 (8.7)	4.2 (0.72-25.02)
Anemia	96 (33.8)	13 (56.5)	2.84 (0.87-9.27)
Malaria and anemia	153 (53.9)	3 (13)	**19.62 (4.21-91.42)**
Total	**284**	**23**	

Children who had a combination of malaria and anemia had about 20 times increased risk of lead poisoning compared to children without co-morbidity or having any of anemia or malaria parasitemia. This association is statistically significant.

Table 20: Use of kohl eye cosmetic on child and lead poisoning

The table below shows the relationship between use of kohl eye cosmetic on child and prevalence of childhood lead poisoning in Kawaye village.

Use of kohl eye cosmetic on child	Diseased :Blood lead level (BLL) ≥ 5 µg/dL Count (%)	Not Diseased :Blood lead level (BLL) <5µg/dL Count (%)	Total
Yes	268 (94.4)	19 (82.6)	
No	16 (5.6)	4 (17.4)	
Total	**284 (100)**	**23 (100)**	**307**

$p = 0.0517$

The prevalence of lead poisoning was higher among children with anemia compared to those without. This association is statistically significant.

4.3. Risk Factors For Lead Poisoning

Table 21: Unconditional logistic regression to assess factors associated with childhood lead poisoning

The table below shows the factors associated with childhood lead poisoning in Kawaye village using unconditional logistic regression model.

Variable	Odds Ratio	95% C.I.		*p*-Value
Anemia	**5.5825**	**1.5813**	**19.7078**	**0.0075**
Low level of Father's/guardian's education (None/Informal)	**9.0979**	**2.2797**	**36.3075**	**0.0018**
Flaky house paint	444753.4514	0.0000	>1.0E12	0.9695
Floor type (Mud)	**0.1177**	**0.0324**	**0.4272**	**0.0011**
Low socioeconomic status (Father/guardian)	**5.2846**	**1.7141**	**16.2924**	**0.0038**
Malaria parasitemia	**3.7720**	**1.1648**	**12.2157**	**0.0268**
Mining occupation	3.6549	0.3798	35.1735	0.2619
Use of kohl eye cosmetic	**6.9830**	**1.1555**	**42.1989**	**0.0342**

Low level of father's/guardian's education, use of kohl eye cosmetic, anemia, low socioeconomic status and malaria parasitemia were found to be independently associated with increased risk of childhood lead poisoning. On the other hand, mud floor type was protective for lead poisoning.

CHAPTER 5

DISCUSSIONS

Since the confirmation of the outbreak of lead poisoning in Zamfara State in March, 2010; which experts have described as the largest ever seen in the history of mankind, in terms of its scope and magnitude, its containment kept resurfacing as a public health and environmental issue of global concerns. Different researches have focused their attention on the issue with a view to finding ways to tame the problem and this form the basis of undertaking this research.

The prevalence of childhood lead poisoning in Kawaye village was found to be very high (92.5%). This finding is similar to results of a study conducted in the State in 2010 to determine risk factors for mortality among lead poisoned children, which reported a 100% "prevalence" of lead poisoning among children tested for lead.[24] The mean age of studied children was 38.5 months ± 18.5. Males were slightly more affected by lead poisoning than females with most of the children belonging to age group 35 months and below. Though a similar study conducted in Otukpo, Nigeria among children aged 1-6 years showed preponderance of lead poisoning among the males, it however found blood lead levels to be evenly distributed across the different age groups.[6] According to Bellinger in 2004, blood lead levels usually peak around the age range of 12-36 months.[52]

The parents and/or guardians of children mostly affected by lead poisoning were farmers (37.1%) and miners (14.7%). This may not be unconnected with the observation that the major source of lead contamination in Zamfara environments was artisanal gold mining as demonstrated by previous studies.[24] The mining activities contaminate soil and water bodies in the environs thereby posing an obvious risk to children, adults and even the animals living in these environments. It is worthy of note that more than half of the parents and/or guardians of studied children had informal education (53.7%) with few of them having neither informal nor formal

education (4.2%). In terms of socioeconomic status of the male parents and/or guardians of the children, nearly one-third of them live below poverty line ($1.25 per day) and about the same proportion (30.3%) earn at least the minimum wage set by the Federal Government of Nigeria monthly.

A total of 307 households were assessed for contamination with lead. At least four of children playing area, main entrance, children sleeping area, cooking area, washing area, common area and other areas such as under the tree, drinking water pot area, wall plaster, animals area, etc were directly tested for presence of lead contamination in the soil. The median household soil lead level was 61PPM, ranging from non-detectable to 24000PPM. Forty four of the households (14.3%) had lead levels reaching at least 400PPM which is the cut-off point for environmental lead contamination in residential areas recommended by United State EPA.[24, 102] The pattern of house hold soil contamination with lead was such that about 11% had mild-moderate contamination, whereas 2.9% were severely contaminated. However, this finding is much lower than that of a similar study in which 85% of soil samples obtained from 116 compounds had soil lead levels exceeding 400PPM threshold set by United States EPA.[24]

Furthermore, about 86% of the studied children had varying degrees of anemia with a mean PCV level of 26.9% (SD± 5.5). On the other hand, 59% of the children had variable levels of malaria parasitemia ranging from mild to moderate parasitemia; median parasite density was 40 parasites/µL; with a range of 0-2800 parasites/µL. A study done in Jos, Nigeria showed a prevalence of malaria parasitemia among febrile U5s to be 56.9%.[113]

Generally speaking, lead poisoning was found to be highest among children aged 35 months and below. Prevalence of sub-clinical lead poisoning appeared to increase with increasing age; however, this trend was not demonstrated with mild, moderate and severe lead poisoning. This is in contrast to findings of a similar study conducted in Kaduna, Nigeria to determine prevalence of elevated blood lead levels among children 1-6 years which showed that highest average blood

lead levels are found in children aged 60 months in both sexes with significant decline at the age of 72 months.[114] Our study did not show any statistically significant association between gender of child and prevalence of lead poisoning. However, severity of lead poisoning was significantly higher in the female gender. In the same vein, the study cited above, by Nriagu J et al, did not show any variation in the prevalence of lead poisoning with gender, similarly; another study conducted to identify socio-ecologic variables contributing to lead poisoning among children in suburban regions of Nigeria also showed that the mean blood lead value for males was not significantly higher than for females.[6, 22]

With regards to educational status of parents and/or guardians of studied children, the prevalence of lead poisoning appeared to be higher among children whose parents/guardians had low educational levels (no education and/or informal education). This finding is similar to that of a study conducted in Ottawa County, Oklahoma to determine lead sources, behaviors and socioeconomic factors in relation to blood lead of native American and White children which demonstrated strong association between caregivers' lesser education and elevated blood lead levels (OR 7.3; 95% CI 1.4–38.4).[115] However, a study conducted in Otukpo, Nigeria showed a significant relationship between maternal literacy and mean children blood lead level. Literate mothers had children with an average BLL that was approximately 1.1 mg/dL lower than illiterate mothers.[6] Mining and farming occupations of parents/guardians were found to be significantly associated with higher prevalence of childhood lead poisoning. However, findings of this study are in contrast to those of Ogunseitan et al which showed no significant association between parental occupation and mean blood lead level of children.[6]

Furthermore, this study found a significant association between poverty and elevated blood lead levels in children. Children whose parents/guardians were below poverty line were found to be more affected by lead poisoning compared to those above poverty line or earning at least the minimum wage approved by the Federal Government of Nigeria. This finding is corroborated by findings of the study by Malcoe L H et al which showed living below the Federal poverty level

by the caregiver was associated with elevated blood lead levels in children in (unadjusted model).[115]

It is worthy of note that all the children that came from a household whose soil was contaminated by lead had variable degrees of lead poisoning. This association was found to be statistically significantly associated with lead poisoning in an unadjusted model. Several studies carried out locally and internationally, have demonstrated a strong positive correlation between level of soil contamination with lead and elevated blood lead levels in children.[22,115,116] This study found out that, the households where the heads were miners and petty traders appeared to have higher soil lead contamination, while households where the heads were cattle rearers had lowest levels of soil lead contamination. This may be due, in part, to the fact that the cattle rearers were living in an area which, though part of the village, but is detached from the main settlement.

The prevalence of lead poisoning was found to be higher among those children with malaria parasitemia. However, there was no significant difference in current history of fever between those children with malaria parasitemia and those without. A study conducted to explore the relationship between lead poisoning and malaria among children in urban areas in Nigeria found negative association between BLL and malaria among pediatric population.[51] There was no significant association between degree of malaria parasitemia and severity of lead poisoning. None of the studied children had severe malaria parasitemia (parasite density of >10,000/µL). However, this study found a significant association between anemia and childhood lead poisoning. All degrees of lead poisoning were found to be significantly higher among children with moderate anemia. In a case-control study to determine Synergistic effects of iron deficiency and lead exposure on blood lead levels in children, children with anemia were found to be at a significantly increased risk of lead poisoning.[117] On the other hand, children with lead poisoning are also more likely to develop anemia compared to their non-lead poisoned counterparts. In study carried out in India to determine the relation between blood lead levels and childhood anemia, children with lead levels of ≥10 µg/dl were 1.3 times as likely to have moderate anemia

as children with lead levels <10 µg/dl (95% confidence interval: 1.0-1.7).[118] Children who had a combination of malaria and anemia had 19.6 times increased risk of lead poisoning compared to children without co-morbidity or having either of anemia or malaria (95% CI: 4.21-91.42).

Finally, logistic regression model was built and all the significant variables were entered into the model. Low level of father's/guardian's education, use of kohl eye cosmetic on child, anemia, low socioeconomic status of father/guardian and malaria parasitemia were found to be independently associated with increased risk of childhood lead poisoning. On the other hand, children living in houses with mud floor type were less likely to develop lead poisoning. Findings of this study are consistent with findings of many similar studies that assessed the risk factors for childhood lead poisoning.[6,19,24,109,118]

CHAPTER 6

CONCLUSIONS AND RECOMMENDATIONS

6.1. Conclusions

This study found a very high prevalence of lead poisoning among children in Kawaye village of 92.5%. Majority of the affected children were males belonging to the age group 35 months and below, whose parents/guardians were predominantly farmers and miners having informal education. Low socioeconomic status and low level of education of father/guardian, use of kohl eye cosmetic on child, anemia and malaria parasitemia were identified significant risk factors for childhood lead poisoning in the village.

6.2. Recommendations

Based on findings from this study, the following general and specific recommendations are made:

6.2.1. General recommendations

1. The Federal Government should support the State with adequate funding to enable it tackle the problem of lead poisoning in the State.
2. The Federal Government through federal ministry of health should promptly provide adequate supply of chelation agents to the State to enable/support it to begin treatment of cases of lead poisoning that meet the criteria for treatment.
3. The Federal Government through the Federal ministry of mines and steel development should enforce legislation on illegal mining. It should ensure that mining is done in accordance with the Nigerian Minerals and Mining Act, 2007.
4. Zamfara State ministry of health should operationalize its Center of Excellence on Lead and other heavy metals poisoning to strengthen management of cases of lead poisoning and support scientific researches on heavy metals poisoning.

6.2.2. Specific recommendations

1. Zamfara State ministry of health should commence immediate treatment of all cases of severe lead poisoning identified by this study.

2. The State ministry of mines and solid minerals should ban all mining activities within the village. The industrial and artisanal miners should be organized into cooperative associations and registered, and their activities regulated and properly supervised.

3. The State Ministry of Environment should commence plans for complete characterization of Kawaye village with a view to remediate the entire community.

4. The State ministry of health should monitor all children with elevated blood lead levels identified by this study.

5. The State ministry of mines and solid minerals should promote low-tech safer mining practices in the community.

6. The State ministry of health should educate people of Kawaye village about the dangers of use of kohl eye cosmetic on their children.

7. In view of the high prevalence of anemia and malaria parasitemia among children in the community and their strong relationship with childhood lead poisoning, further researches on malaria and causes of anemia in children in the community should be undertaken.

8. The State ministry of rural development in collaboration with National Poverty Eradication Program (NAPEP), Small and Medium Enterprises Development Agency of Nigeria (SMEDAN), State ministries of Works and Agriculture and other major stakeholders should improve the socioeconomic status of the people through improvement of agricultural practices, microcredit disbursement, jobs creation and provision of social amenities, among others.

9. The State ministry of education and agency for mass education should collaborate to improve the literacy level of the people particularly in the areas of adult education and secondary and tertiary education.

10. The State ministry of health in collaboration with partners should establish sustainable public health programs aimed at control and prevention of lead poisoning in the State. These programs should, among others, include the following:

    - ✓ Active surveillance,
    - ✓ Treatment of cases,
    - ✓ Continuous health education of miners and parents/guardians on lead poisoning and
    - ✓ Enforcement of legislation and ban on illegal mining and mining within the communities respectively.

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APPENDICES

Appendix 1: Study Instruments

Appendix 1(a): Participant information (English version)

DEPARTMENT OF COMMUNITY MEDICINE,

AHMADU BELLO UNIVERSITY, ZARIA

RESEARCH ON DETERMINANTS AND PREVALENCE OF CHILDHOOD LEAD POISONING IN KAWAYE VILLAGE, ANKA LOCAL GOVERNMENT AREA, ZAMFARA STATE

APPENDIX 1A

PARTICIPANT INFORMATION

Dear Parent/Guardian,

Introduction:

My name is Kabiru Ibrahim Getso from Ahmadu Bello University, Zaria/Nigeria Field Epidemiology and Laboratory Training Programme, Federal Ministry of Health, Abuja. We are undertaking a research to find out about lead poisoning among children in this community. Childhood lead poisoning is a health problem which is said to occur when lead builds up in the body of children beyond certain limits (≥ 5 micrograms per deciliter of blood). Children under 6 years and pregnant women are at the greatest risk for lead poisoning. Lead is a metal that occurs naturally in the earth's crust. It is very toxic to human body, even a small amount of lead in the body can cause serious problems such as anemia, kidney disease, inhibition of proper physical and cognitive development, learning disabilities, lowered IQ, speech delay and hearing impairment. High levels of lead can cause severe mental disabilities, convulsions, coma or even death. However, lead poisoning is completely preventable and children can be kept safe by

taking steps to decrease the amount of lead in the environment. Those children that happened to have lead poisoning can be treated using medical treatments. We expect to have 307 children join this study.

We are undertaking this study to learn whether or not your child has been exposed to harmful levels lead. In the end, we will be able to find out how common lead poisoning is in this community, its distribution among children, whether activities like artisanal gold ore mining and processing contribute to the problem and presence of other co-morbidities (malaria and anemia) among children in this community. This will enable us to learn how severe the problem is and what can be done to address the situation. To do this, we will visit compounds that are randomly selected in the community to select a single child from a household and ask some questions and test the child and compound for the presence of lead.

Is participation in this study compulsory?

Participation in this study is voluntary. You are free to or no to participate in this study or to leave the study at any stage. However, we will greatly appreciate it if you will be part of this study as your participation will help in understanding the problem of childhood lead poisoning better and provide useful information that will assist in designing targeted public health interventions to control and prevent lead poisoning in Zamfara State. If you choose not to join, you will not be denied any of the services that you normally receive. If you do not want to participate in this study and would still like to know your child's blood lead level, you can contact doctors in the treatment center in Anka.

Testing the children/compound:

Study workers will collect about a teaspoon full of blood samples from a vein on the selected child's arm. This sample will be about a teaspoonful in volume. The sample will be taken to Lead poisoning Center of Excellence and Farida General Hospital laboratories in Gusau to test

for presence of lead, malaria and anemia. In addition, the child's height and weight will be measured and household soil samples will be collected/ checked for presence of lead. New sterile instruments that are clean and completely safe will be used to collect blood samples. After analysis, the remaining blood samples will be destroyed. You will not be charged any money for the tests. You will be informed the results of your child's tests by Zamfara State Ministry of Health. In a situation where the child's blood lead level is very high requiring medical attention, he will be immediately referred to the treatment centre in Anka LGA where treatment is provided free of charge.

Risks to your child for participating in this study:

There is little risk to your child from the blood test. Your child will feel a slight pinch, slight pain, or possible dizziness when the blood is drawn from an arm. Children may develop a slight bruise or infection following the venous blood test.

Will you or your child receive any compensation for participating in this study?

This study is entirely voluntary. You or your child will not receive compensation for participating in this study. However, you will know the results of your child's blood test. And if your child has elevated blood lead level requiring medical attention, he/she will be immediately referred to lead poisoning treatment centre in Anka where free treatment is provided.

Benefits of participating in the study:

The benefit of your child/ward participating in this study is that you will be able to know whether or not your child has been exposed to harmful levels of lead or has other co-morbidities (malaria or anemia). Also, your participation will provide useful information on how to tackle the problem. Those children that have very high levels of blood lead that requires attention will be immediately referred to lead poisoning treatment center in Anka LGA where treatment is

provided free of charge. Participation in this study is completely free: you will not be charged anything for the tests or any other procedure in this study. You will also learn about the results of your child's test from Zamfara State Ministry of Health.

How is your child's privacy going to be protected?

The results of your child's test will be kept confidential to the extent permitted by law in the country. Your answers to our questions and your child's blood sample will be identified by using numbers instead of their names. However, if your child needs urgent medical care, the Zamfara State Ministry of Health / Nigerian FELTP/MSF will keep a master list of names to ensure that all affected children receive appropriate medical care from MSF/SMoH. After the study, this master list of names will be destroyed.

If you have further questions:

If you have further questions, you contact:

- Dr Kabiru Ibrahim Getso - Tel: 08037032586, Email: kabirgetso@yahoo.com
- Dr Ahmed Na'abba Keku- Tel: 08032442140, Email: knaabbanah@ymail.com

Thank you very much.

Appendix 1(b): Participant information (Hausa version)

SASHEN KULA DA LAFIYAR AL'UMMA

JAMI'AR AHMADU BELLO, ZARIA

BINCIKE AKAN SANADIYYAR DA KUMA ADADIN YADUWAR CUTAR GUBAR DALMA GA YARA A GARIN KAWAYE, KARAMAR HUKUMAR ANKA, JIHAR ZAMFARA

KARI NA IB

BAYANAI GA IYAYEN YARA

Zuwa ga iyayen yara,

Gabatarwa:

Sunana Kabiru Ibrahim Getso daga Jamiar Ahmadu Bello, Zaria/Cibiyar nazari da binciken cututtuka ta Najeriya, Ma'aikatar lafiya ta tarayya, Abuja. Muna yin bincike ne akan cutar gubar dalma a kananan yara na wannan gari. Ita cutar gubar dalma wata cut ace wadda takan afku sakamakon taruwar gubar dalma a cikin jikin yara fiya yadda hukumar lafiya ta kaiyade (≥5 µg/dL). Yara 'yan kasa da shekara 6 da mata masu juna biyu su wannan matsala tafi shafa. Dalma wani ma'adani ne wanda ake samun sa acikin kasa/dutse. Yana da illa matuka ga jikin dan Adam, komai kankantar sa ajiki yana iya haifar da matsala kamar karancin jinni, cutar koda, matsalar kaifin hankali, matsalar karatu, makara wajen fara magana da kuma matsala wajen ji. Yawan gubar dalma a cikin jini na iya haifar da matsananciyar matsalar kwakwalwa, fizge fizge, suma ko kuma mutuwa. Amma ita cutar gubar cut ace wadda za a iya hana afkuwar ta gaba daya, kuma kuma za a iya kare yara daga kamuwa da ita ta hanyar bin matakan da zasu rage gurbatar muhalli da ita wannan guba. Yaran da su ka kasance suna dauke da wannan guba za a iya warkar das u ta hanyar basu magun guna. Muna fatan samun yara dari uku da bakwai (307) a cikin wannan bincike da za muyi.

Za muyi wannan bincike ne don mu tabbatar cewa akwai ko babu wannan guba a jikin yaronka. Da ga karshe zamu iya yin kiyasin yawan wannan matsala a wannan gari, rarrabuwarta a cikin yara, abubuwan dake habakata, nasabarta da wasu sana'oi kamar aikin gina ko sarrafa zinari, sannan mu kuma gano wasu cututtuka dakan addabi kananan yara (zazabin cizon sauro da karancin jinni) da nasabar su da ita wannan cuta ta gubar dalma. Wannan zai taimaka mana wajen fahimtar girman matsalar da kuma hanyoyin da za'a iya bi don shawo kan matsalar. Don gudanar da hakan, zamu ziyarci gidaje wadanda aka zaba ta hanyar kacici kacici. Kuma a kowanne gida, daga bangaren kowanne magidanci zamu yi tambayoyi, mu dauki kasar gidan sannan mu canki yaro guda daya don mu gwada jininsa don ganeda matsalar gubar dalma.

Shin dole ne yaronka ya shiga wannan binciken?

Shiga wannan bincike ganin dama ne. Kana da dammar ka shiga ko ka ki shiga cikin wannan bincike, kuma koda ka shiga zaka iya fita a kowanne lokaci. Amma zai kyautu, matukar yaronka na bukatar kyakkyawan tsaron lafiyar jikinsa ka aminta ya shiga cikin wannan yekuwar/binciken; domin kuwa shigar ka zai bamu dammar fahimtar wannan matsala kwarai ya kuma samar da bayani mai muhimmanci wanda zai taimaka wajen samar da hanyoyin shawo kan wannan matsala a wannan jiha. Idan baka shiga wannan bincike ba babu abinda zai sameka, kuma baza a tauye maka wata dama dakake samu dab a. Idan baka son shiga wannan bincike kuma ka damu da ka san adadin gubar dalma dake cikin jinin yaronka, zaka iya tuntubar likitoci a asibitin da aka tanadar don wannan cuta dake garin Anka.

yadda ake yiwa yara awon cutar gubar dalma:

Ma'aikatan da aka zaba za su zo gidanku domin su auna yaranku. Za su]ebi jinin yaronka daga jijiyar hannunsa. Jinin da za a diba zai kai kimanin karamin cokali daya. Za a tafi da jinin cibiyar binciken akan cutar gubar dalma da kuma babban asibitin Farida na Garin Gusau don gwaji akan cutar gubar dalma, zazzabin cizon sauro da kuma karancin jinni. Har ila yau, za a gwada tsawo da nauyin yaro sannan kuma za a dauki kasar gida a gwada don a gano ko ta gurbata da gubar

dalma. A wajen diban jini za mu sa allura sabuwa mai tsafta kubutacciya daga kowace irin cuta. Idan aka kare awon, za a watsar da sauran jinin da aka gama aiki da shi.

Wannan aiki kyauta ne, ba za a karbi kwabonka ba wajen wa]annan gwaje gwaje. Za a sanar dakai sakamakon awon yaron ka ta hannun ma'aikatar lafiya ta jihar Zamfara. Sa'ilin da aka samu cewa akwai guba mai matukar yawa acikin jinin yaronka wadda ke bukatar kulawa cikin gaggawa, to za a tura shi cikin hanzari zuwa asibitin da aka tanada don kulawa da cutar gubar dalma wadda ke karamar hukumar Anka a inda ake bada maganin wannan cuta kyauta.

Mene ne hadarin wannan ga yaranka?

Babu shakka duk abin da ya taba hanci ido zai yi ruwa. Yaronka zai ji ciwo ka]an daga sukar allura ko ya iya jin 'yar juwa kadan ta dibar jinin. Za a iya samu dan kwarzane kadan na sukan allura da aka yi ko shigar kwayoyin cuta.

Ko za a biya wata diyya gare ka da yaranka ?

Wannan aikin taimako ne kuma shigarsa ganin dama ne, ba za a biya ka ko yaron ka ba kowace irin diyya ba don kun shiga wannan bincike. Duk da haka, za a ba ka sakamakon awon jinin yaronka da aka yi. Idan aka samu yaronka na da cutar wadda ke bu}atar kulawar gaggawa, to za a tura shi cikin hanzari zuwa asibitin da aka tanada don kulawa da masu cutar gubar dalma wadda ke karamar hukumar Anka a inda ake bada maganin cutar kyauta.

Mene ne amfanin sa yaronka cikin wannan bincike?

Babban amfanin da za ka samu shi ne, gano ko yaronka ya kamu da cutar gubar dalma ko bai kamu da ita ba kokuma yana dauke cutar zazzabin cizon sauro ko }arancin jinni. Binciken wani mataki ne na gano irin hanyoyin da za a bi wajen kawar da matsalar daga jiha gaba daya. Har ila yau, idan aka samu cewa yaronka na da cutar wadda ke bu}atar kulawar gaggawa, to za a tura shi cikin hanzari zuwa asibitin da aka tanada don kulawa da masu cutar gubar dalma wadda ke

karamar hukumar Anka a inda ake bada maganin cutar kyauta tun kafin cutar tayi lahani ga jikinsa da lafiyarsa. Ka kula, ba za a nemi ka ba da ko kwabo ba ga awon da za a yiwa yaronka. Kai tsaye za a ba ka sakamakon binciken da aka yi. Ma'aikatar lafiya ta jiha zata labarta maka sakamakon binciken jinin yaronka da aka yi.

Yaya za a tsare sirrin yara?

Sakamakon awon jinin yaronka da aka yi za a tsare shi sosai a kan amana ba tare da yin terere da shi ga kowa ba gwargwadon yadda dokar kasa ta tanada. Amsoshin tambayoyin da muka yi maka a kanka da na yaranka za mu adana su ta fuskar amfani da lambobi ba da sunan yaronka ko kai ko garinku ko unguwarku ba. Duk da haka, idan yaronka na bukatar cetonmu na gaggawa ma'aikatar lafiya ta Zamfara da kungiyar lafiya ta MSF za su zagaya da kundayen da aka adana sunayen yaranku domin a ba su magani. Bayan an gama ba su magani bincike ya kare sai a watsar da diwanin sunayen yaran ta yadda ba wanda zai gan su balle ya yi wani abu da su.

To, idan kana da tambaya fa?

Idan kana da kowace irin tambaya game da wannan bincike ka iya tuntubar mu ta wadannan lambobi kamar haka:

- Dr Kabiru Ibrahim Getso – lambar waya: 08037032586, ko ta adireshin akwatin yanar gizo: kabirgetso@yahoo.com

- Dr Ahmed Na'abba Keku- lambar waya: 08032442140, ko ta adireshin akwatin yanar gizo: knaabbanah@ymail.com

Mun gode kwarai.

Appendix 1(c): Consent form (English version)

DEPARTMENT OF COMMUNITY MEDICINE,

AHMADU BELLO UNIVERSITY, ZARIA

RESEARCH ON DETERMINANTS AND PREVALENCE OF CHILDHOOD LEAD POISONING IN KAWAYE VILLAGE, ANKA LOCAL GOVERNMENT AREA, ZAMFARA STATE

APPENDIX 1C

CONSENT FORM FOR THE CHILD'S PARENT/GUARDIAN

I am undertaking a study to find out the determinants and prevalence of lead poisoning among children ≤ 6 years in this community. Lead is a metal that is very toxic to human beings particularly children < 6 years. It is toxic to their brain, hearing, kidneys, blood, immune system, growth, intelligence and virtually every organ/system in the body. At very high levels, lead can cause seizures, coma and death. It is found in the environment, and one of its common sources is gold mining and processing.

You are free to or not to participate in the study, but we will greatly appreciate it if you will be part of this study as your participation will help in understanding the problem better and provide useful information that will assist in designing targeted public health interventions to control and prevent lead poisoning in Zamfara State.

If you agree, I will ask you some questions which will last for about 30 minutes and afterwards, a small amount of blood, about a tea spoonful will be taken from the arm of your child ≤ 6 years old. We are offering blood tests for lead (BLL), malaria parasites (MP's) and Packed Cell Volume (PCV). This is to see if your child has been exposed to harmful levels of lead or has other co-morbidities.

Drawing blood from the arm is usually very safe and only causes mild discomfort for a short time. Other less common risks are bruising and swelling around the puncture site, infection or

fainting from the procedure. You will be communicated the results of these tests through the State Ministry of Health. Since lead can be anywhere in the environment, soil samples will be collected from your compound to check for the presence of lead.

Is it okay if I ask you the questions and subsequently collect blood specimen from your child and soil specimen from your compound?

Consent obtained? Yes ☐ No ☐

If yes,

Name……

Signature or right thumb print of parent/guardian and date**.....**……..

WITNESS (If parent/guardian cannot read and write):

Name……

Signature/Date …….

Appendix 1(d): Consent form (Hausa version)

SASHEN KULA DA LAFIYAR AL'UMMA

JAMI'AR AHMADU BELLO, ZARIA

BINCIKE AKAN SANADIYYAR DA KUMA ADADIN YADUWAR CUTAR GUBAR DALMA GA YARA A GARIN KAWAYE, KARAMAR HUKUMAR ANKA, JIHAR ZAMFARA

KARI NA ID

ZUWA GA IYAYEN YARA,

Ina yin wannan bincike ne don gano sanadiyyar da kuma adadin yaduwar cutar gubar dalma a yara yan shekara shida zuwa kasa a wannan gari. Dalma wani karfe ne wanda yake da matukar guba musamman ga yara yan kasa da shekara 6. Ya na da illa ga kwakwalwa, ji, jini, garkuwar jiki, girma, hazaka da kusan kowanne bangare na jiki. Idan gubar tayi yawa a jiki tana iya haifar da fizge fizge, suma ko mutuwa. Ana samun wannan guba a cikin muhalli, musamman sakamon aikin gina ko sarrafa zinari.

Kana da dammar ka shiga ko ka ki shiga wannan bincike, amma zamu yi matukar farinciki idan kai da yaron ka kuka shiga cikin wannan bincike saboda bada hadin kan ku zai taimaka wajen fahintar wannan matsala da samar da bayanai masu muhimmanci wadanda zasu taimaka gaya wajen samar da tsari wanda zai taimaka wajen shawo kan wannan matsala ta gubar dalma a jihar Zamfara.

Idan ka amince, zan yi maka wasu 'yan tambayoyi wanda za su iya kaiwa tsawon mintota talatin, sannan bayan haka zan dauki jini daga hannun yaron ka guda dan shekara shida zuwa kasa. Za a yi masa gwajin jinin ne don gano ko ya na da cutar gubar dalma, zazzabin cizon sauro ko karancin jini a jiki.

Daukan jini daga daya daga cikin hannaye biyu abu ne mara hadari amma yaro zai ji zafi yayin shigar allurar jikinsa. A mafi karancin lokuta hakan na iya sa gurin da aka dauki jinin ya dan kumbura ko yayi kuraje, ko yasa yaro yaji jiri ko suma. Za a sanar dakai sakamakon wadannan gwaje gwaje ta hannun ma'aikatar lafiya ta jihar Zamfara. Tun da ita wannan guba an iya samun ta a ko ina acikin muhalli, za a dauki kasar gidanka don a auna a tabbatar bata gurbata da wannan guba ba.

Idan ka aminta ba da tilastawa ba za ka ba da bayanai ga rangadin binciken da muke yi domin neman bayani a gidanka, ka ba mu dama mu auna yaronka mu gano ko jininsa na cushe da gubar dalma; ka ba mu dama mu taya juna sani a kan wannan cuta ta gubar dalma to ka sa hannu a kasa:

Eh na amince ☐ **Aa ban amince ba** ☐

Idan ka amince:

Suna……

Ka sa hannu ko ka dangwala babban dan yatsa na dama anan/kwanan wata……

SHAIDA:

Suna……

Sa hannu da kwanan wata……..

Appendix 1(e): Questionnaire

DEPARTMENT OF COMMUNITY MEDICINE,

AHMADU BELLO UNIVERSITY, ZARIA

QUESTIONNAIRE ON DETERMINANTS AND PREVALENCE OF CHILDHOOD LEAD POISONING IN KAWAYE VILLAGE, ANKA LOCAL GOVERNMENT AREA, ZAMFARA STATE

(Adopted/modified from NFELTP questionnaire for the investigation of outbreak of lead poisoning in Zamfara State, May 2010)

APPENDIX 1E

(A) IDENTIFICATION

1. Questionnaire number.....
2. Compound ID.....
3. Date of interview (dd/mm/yyyy): _____/_____/2012
4. Interviewer's name: .....
5. Village name.....
6. Name of Head of Compound……Age.....Sex.....

(B) SOCIO-DEMOGRAPHIC CHARACTERISTICS OF THE CHILD'S PARENT/GUARDIAN

7. How many married men are living in the Compound? _____
8. How many mothers are living in the Compound? _____
9. Age (yrs).....
10. Sex: ☐ Female ☐ Male

11. Tribe/ethnicity: ☐ Hausa ☐ Fulani ☐ Others (Please specify).....

12. Religion: ☐Islam ☐Christianity ☐Others (Please specify) .....

13. Father's/guardian's occupation:

☐ Farmer ☐ Petty trader ☐ Cattle rearer ☐ Miner ☐ Civil servant

☐ Businessman ☐ Others (Please specify) .....

14. Father's/guardian's educational status:

☐ None ☐ Koranic/Islamiyya ☐ Primary ☐ Secondary ☐ Tertiary

15. Father's/guardian's socio-economic status:

How much do you earn in a month (in Naira)? ₦.....

(C) INFORMATION ON SELECTED MOTHER/CHILD PAIR

30. **Mother**

(a) Mother's name.....

(b) Age.....

(c)Tribe: ☐ Hausa ☐ Fulani ☐ Others (Please specify).....

(d)Mother's occupation: ☐House-wife ☐Petty trader ☐Civil servant ☐ Businesswoman ☐Others (Please specify).....

(e) Mother's educational status: ☐ None ☐ Koranic/Islamiyya ☐ Primary ☐ Secondary ☐ Tertiary

(f) Are you currently pregnant? Yes ☐ No☐ Don't know☐ (g) If yes, how many months pregnant? _____ months

(h) Have you had miscarriage in the last 12 months? Yes ☐ No☐ Don't know☐

(i) Are you participating in any of the ore proceesing activities? Yes ☐ No☐ Refused ☐

(j) How many children do you have? .....

(k) Do you use kohl eye cosmetic on your child(ren)? ☐ Yes ☐ No ☐ Refused

31. **NOW, I WILL RANDOMLY SELECT ONE OF YOUR CHILDREN ≤ 6 YEARS OLD AND ASK THE FOLLOWING QUESTIONS ABOUT HIM/HER:**

Called name	**Sex**	**Age (Year or month)**	**Weight (Kg)**	**Height (Cm)**	**Did the child Convulse?**	**If the child had convulsions, when was the last time he/she convulsed? (in months or years)**	**Does the child has other symptoms?**
	☐ Male ☐ Female				☐ Yes ☐ No		☐ Fever ☐ Head ache ☐ Vomiting ☐ Diarrhea ☐ Abdominal pains ☐ Body Weakness ☐ Others.....

(D) EXPOSURE HISTORY

32. **In the last 12 months, has there been any of the following gold extraction activities <u>WITHIN your compound</u>?**

S/No.	Activities	Answer	If Yes, please answer the following questions	
			When did it start?	Has it stopped? When?
1	Breaking down ore rocks	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
2	Pounding of ore gravels	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
3	Grinding of ore gravel	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
4	Washing of grinded ore gravel	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
5	Drying of the grinded materials	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
6	Gold amalgamation with Mercury	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
7	Separating mercury from gold	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____
8	Melting/burning mercury	Yes☐ No☐		**Stopped?** Yes☐ No☐ **If yes, when?**_____

33. If there is breaking down of ore rocks within the compound, where did you get your ore rock from? (Check all that apply)

☐ Dareta If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Yargalma If checked, how long have you been getting rocks from this source? _____ (in years or months)

☐ Kwali If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Sunke If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Abare If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Tamani If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Dogon Dajin

Sarkin Noma If checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Others _____; if checked, how long have you been getting rocks from this source? _____(in years or months)

☐ Don't know

34. Are there dried grinded materials within your compound? [Observe and tick as appropriate]

☐Yes ☐ No

35. Type of housing [Observe and tick as appropriate]: ☐ Mud ☐ Brick ☐ Thatch

☐ Others (Please specify).....

36. Is your house painted? [Observe and tick as appropriate]: ☐ Yes ☐ No

37. If yes to above, is the paint flaking? [Observe and tick as appropriate] ☐ Yes ☐ No

38. What is the type of floor in the rooms in your compound? [Observe and tick as appropriate]

☐ Sandy ☐ Mud ☐ Cemented ☐ Others (Please specify).....

39. In the last 12 months, have there been grinding of ore gravel JUST OUTSIDE your compound? ☐Yes ☐ No ☐ Don't know

40. In the past 12 months, what has been your MAIN source of drinking water?

☐Bore hole ☐ Well within the compound ☐ Well outside the compound

☐ Stream ☐ Pond ☐ Other _____

40a. If source is well, is the well is open or closed? ☐ Open well ☐ Closed well

40b. Does washing of grinded ore gravels take place in or near your main source of drinking water? ☐ Yes ☐ No

41. Do you or your spouse engage in battery recycling? ☐Yes ☐ No

42. Do you or your spouse engage in pottery? ☐Yes ☐ No

43. What is your source of information on lead poisoning? *(Indicate all that apply)*

☐No information ☐Radio ☐Television ☐Colleague ☐Health worker

☐Newspapers ☐Others (Please, specify) _____

44. **Environmental sampling:**

Environmental Sample ID#	**Location**	**Type**	**Results**

Appendix 1(f): Laboratory line list form

A. Blood samples test results:

LAB. NO./ SAMPLE ID	NAME	AGE	SEX	TEST TYPE		
				BLL	MP's	PCV

B. Soil specimens results:

S/NO.	SITE ID	DESCRIPTION	SOIL LEAD LEVELS IN PARTS PER MILLION (PPM)			
			1ST READING	2ND READING	3RD READING	AVERAGE
1						
2						
3						
4						
5						

Appendix 2: Ethical approval

Ahmadu Bello University Teaching Hospital

P.M.B. 06, Shika - Zaria, Kaduna State, Nigeria. 069-876305

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Chairman of Board:

Chief Medical Director: DR. LAWAL KHALID, *MBBS, FMCS, FWACS, FRCS(ED) mni*

Chairman, Medical Advisory Committee: DR. ABDULLAHI MOHAMMED, *MBBS, FWACP FICS*

Director of Administration: BARR. ISHAK BELLO, *LL.B, BL, LL.M, PGDM, AHAN, FCAI*

Our Ref:

ABUTH/HREC/TRG/36

26th November, 2012

Dr Kabiru Ibrahim Getso
Dept of Community Medicine
A.B.U
Zaria.

ETHICAL CLEARANCE

Your application for ethical clearance on the research proposal titled "**Pattern and Determinants of Childhood Lead Poisoning in Anka Local Government Area Zamfara State** "refers.

This is to convey ethical approval for you to commence the study. The ABUTH Scientific and Health Research Ethics Committee require an annual update from the Principal investigator.

Prof J U Okpapi
Chairman, ABUTH HREC