DETERMINATION OF LEAD (Pb) LEVELS IN SELECTED MINING COMMUNITIES IN ZAMFARA STATE - NIGERIA

BY

ABDULJALAL DANBABA

PhD/PHARM SCI/3705/11-12

DEPARTMENT OF PHARMACEUTICAL AND MEDICINAL CHEMISTRY, FACULTY OF PHARMACEUTICAL SCIENCES, AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA

AUGUST, 2016

DETERMINATION OF LEAD (Pb) LEVELS IN SELECTED MINING COMMUNITIES IN ZAMFARA STATE - NIGERIA

By

Abduljalal DANBABA
B Sc Chemistry (ABU, 1987), M Sc. Pharm. Chem. (ABU, 2010),
PhD /Pharm. Sci./3705/ 11-12

A THESIS SUBMITTED TO THE SCHOOL OF POST GRADUATE STUDIES, AHMADU BELLO UNIVERSITY IN PARTIAL FULFILMENT FOR THE AWARD OF DOCTOR OF PHILOSOPHY DEGREE IN PHARMACEUTICAL CHEMISTRY.

DEPARTMENT OF PHARMACEUTICAL AND MEDICINAL CHEMISTRY
FACULTY OF PHARMACEUTICAL SCIENCES
AHMADU BELLO UNIVERSITY
ZARIA, NIGERIA.

AUGUST, 2016

DECLARATION

I declare that the work reported in this thesis entitled Determination of Lead (Pb) Levels in Selected Mining Communities in Zamfara State, Nigeria has been carried out by me in the Department of Pharmaceutical and Medicinal Chemistry under the supervision of Dr. Musa A. Usman, Professor Ibrahim A. Yakasai and Professor Magaji Garba, all of the Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, Ahmadu Bello University Zaria. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this thesis was previously presented for another degree or diploma at this or any other Institution.

Abduljalal DANBABA ---------- ----------

Signature Date

Department of Pharmaceutical and Medicinal Chemistry
Faculty of Pharmaceutical Sciences
Ahmadu Bello University, Zaria,
Nigeria

CERTIFICATION

This thesis entitled DETERMINATION OF LEAD (Pb) LEVELS IN SELECTED MINING COMMUNITIES IN ZAMFARA STATE, NIGERIA by Abduljalal DANBABA meets the regulations governing the award of the Degree of DOCTOR OF PHILOSOPHY of Ahmadu Bello University, Zaria and is approved for its contribution to knowledge and literary presentation.

Dr. Musa A. Usman Department of Pharmaceutical and Medicinal Chemistry, Ahmadu Bello University .Zaria	---------- (Signature)	---------- Date
Professor Ibrahim A. Yakasai Department of Pharmaceutical and Medicinal Chemistry, Ahmadu Bello University .Zaria	---------- (Signature)	---------- Date
Professor Magaji Garba. Department of Pharmaceutical and Medicinal Chemistry, Ahmadu Bello University .Zaria	---------- (Signature)	---------- Date
Dr. Aliyu M. Musa Head of Department, pharmaceutical and Medicinal Chemistry, Ahmadu Bello University, Zaria	---------- (Signature)	---------- Date
Professor Kabir Bala Dean, School of Post graduate Studies Ahmadu Bello. University, Zaria	---------- (Signature)	---------- Date

DEDICATION

I dedicate this work first to Almighty Allah (*Subhaanahu Wata'ala*) Who made everything possible. Then to my Late Father (Haruna Danbaba Sa'ad) and Mother (Karimatu Abdurrahman) for given me the basic training that prepared me for the challenges of this world, and their invaluable prayer remained my torchlight in this world and the hereafter; with the hope that this as my own little contribution that might save the lives of future generations from extinction as a result of pursuance of economic boom tied to its doom.

ACKNOWLEDGEMENT

My divine acknowledgement on this work goes to "Allah *Subhanahu Wataala"* for making the entire process of the study possible. My gratitude goes to my supervisors: Dr. Musa A. Usman, Professor Ibrahim A. Yakasai and Professor Magaji Garba, who have supported and encouraged me in view of the challenges involved, particularly the field trip. As a matter of fact, I have no qualifying words to express my appreciation except to pray that, may the Almighty reward all of you graciously. I also wish to acknowledge the support from my closest friends, my wives, Hadiza (Senior) Ja'afar Sambo who we started this struggle with and her invaluable commitment to see to the completion of this work. Also to Hadiza (Junior) Nasirudeen, I say a big thank you for understanding and moral support to the moving train. The financial and moral support of Halima A. Idris was a distinct catalyst to this research, and highly appreciated. To the young Muhammad Balarabe, I thank you for being asleep whenever I was on this work and lately for keeping me company on the computer even if not invited.

My sincere gratitude and invaluable appreciation goes to my research assistants, namely: Mal. Shehu Muhammed Anka (Environmentalist) of the Zamfara Environmental Sanitation Agency (ZESA). Mal. Bashir Abdullahi (Haematologist) of the Lead poisoning Specialist Centre Laboratory, Gusau, and Abdulmumini Ibrahim (Veterinary Officer) for their commitment, patience and public relations they demonstrated throughout our trips. Their wealth of experience that led to the collection of over 350 samples was highly commendable. May the Almighty Allah reward you in His most appreciated ways he rewards good deeds. I also wish to express my gratitude to the reception accorded us by the Traditional Rulers and people of the selected mining communities. We are also grateful for the time speared by the country Coordinator of

Medicines Sans Frontier (MSF) for our fruitful discussion with them on the research and activity of the mission, when we visited their office in Anka, Zamfara State.

My unalloyed appreciation is also extended to senior colleagues and staff of the Department of Pharmaceutical and Medicinal Chemistry ABU, Zaria, Dr. Aliyu Musa, the HOD, Pharmaceutical Chemistry for understanding and morale support. My sincere gratitude also goes to Dr. Aminu Musa, for his patience and guidance during corrections. To Prof. U.U. Pateh, the Dean Faculty of Pharmaceutical Sciences, and all his supportive staff. I appreciate the moral support and interest they showed in my progress, they include: Dr. Isah Mohammed Abbass, Prof. M. T. Odunola, Prof. J. O. Amupitan (of Chemistry Dept), and Prof. Kabir Y. Musa, Dr. U. H. Danmallam, Dr. Hadiza Nuhu and Prof. Hajara Ibrahim all of Pharmacognosy and Drug development department. I also wish to acknowledge the moral support of Prof. Nuhu Danjuma and Dr. Magaji of Dept of Pharmacology and Therapeutics. The invaluable support and contribution of the Hon. Commissioner of Health, Zamfara State, and the Special Adviser to his Excellency on Environment and in charge of ZESA cannot be over emphasised.

I also wish to acknowledge the tremendous support and assistance from Mal. Muhammad Bashir of Multi-User Science Research Laboratory, MUSRL/ABU, Zaria and Dr. Musa of National Research Institute of Chemical Technology (NARICT), Zaria and their supportive staff for jointly carrying out my analysis.

My sincere gratitude also goes to Alh. Suleiman Aliyu and Alh. Adamu Aliyu Shika, both former Rectors of Nuhu Bamalli Polytechnic, Zaria, for their moral support, encouragement and opportunity given to me to develop myself intellectually. I also wish to acknowledge the support from my former Director, Mal. Falalu Maska and former Head of Department Mal. Tukur Sidi, for their moral support. I equally wish to

recognize the support and interest shown by my colleagues in carrying out this research work, they include: Mal. Ahmed Garba, Mal Suleiman Musa Anchau, Mr. Jimmy Oshanimi, Mal. Mubarak Liman, Mal. Shafi'u Mustapha and Mal Abdulhamid and a host of others. Finally, my brothers and colleagues like Alh. Yusuf Danbaba, Abdurrahman Danbaba, Mal. Suleiman Ahmadu, Mal. Auwal Yakubu, Dr. Aminu Abdu, Mal. Lawal Pate, Honourable Dr. Abbas Tajudeen, the Provost, Shehu Idris School of Health Management Sciences, Makarfi, Dr. Musa Garba, and others too numerous to mention deserve a special mention and appreciation for their concern and interest in this work.

ABSTRACT

In 2010, the world's attention was drawn to Zamfara State, Nigeria following the 'unprecedented' death reported (particularly of children) as a result of lead poisoning in the process of small scale gold mining of lead-rich ore using rudimentary tools. This study was aimed at a wholistic assessment of lead levels in the affected communities. Lead (Pb) levels were determined from samples of residential compound soil, underground and surface water, common grains (sorghum, millet and maize), human and animal blood samples collected from ten selected villages (Bagega, Kadauri, Kawaye, Kwali, Magami, Sunke, Tsunami/Unguwan Danbaba, Tungar-Guru, Tungar-Kudaku and Yargalma) among the mining, communities for analysis. Samples collected were carefully handled, preserved, and digested using adopted and validated methods of analysis for lead using Atomic Absorption Spectrophotometry (AAS) technique. Results obtained from the analysis were subjected to statistical analysis using GraphPad Prism 6 analytical package. Results indicated that the mean lead concentration in residential soil ranged from 12.44 ± 3.23 to 757.69 ± 645.35 mg/Kg. Apart from Tungar-Guru, with the highest mean lead concentration in soil, the rest of the villages were below the US-EPA threshold level (400 mg/Kg) of concern. Mean lead concentration in common grains indicated that, apart from the millet sample from Kwali with the highest mean lead concentration of 72.07 ± 57.36mg/Kg, the mean lead concentration of all the grains from other locations ranged from 5.56 ± 2.02 to 28.18 ± 5.93 mg/Kg. The results indicated that the mean lead concentration of all the grain samples have exceeded the WHO threshold level of concern for the WHO (3.0 µg/Kg). Mean Blood lead levels (BLLs) for children within the age ranged of 2 – 6 years in the mining communities ranged from 32.20 ± 7.34 - 157.60 ± 9.11 µg/dL. With the exception of Kwali, Tungar-Kudaku and Yargalma with BLLs of 32.20 ± 7.34, 35.80 ±

8.10 and 41.60 ± 4.70µg/dL respectively, children in other villages have blood concentration exceeding the threshold level (45 µg/dL) for chelation therapy in children. The mean blood lead concentration in adults in the selected mining communities ranged from 37.80 ± 5.94 to 146.40 ± 27.40 µg/dL. Overall, children from Kwali have the lowest blood lead concentration and the highest from Bagega. Both children and adults blood lead levels have exceeded the threshold levels of concern for lead poisoning for both CDC (5µg/dL) and WHO (10µg/dL). The mean blood lead concentration of all the animals/livestock involved in the study ranged from 52.25 ± 5.19 to 101 ± 9.07µg/dL. Goats and chickens from Sunke and Tsunami have the lowest mean lead concentration (52.25 ± 5.19 µg/dL) while Yargalma has the highest (101 ± 9.07µg/dL). Blood lead concentration in all the animals were above the WHO acceptable level (10µg/dL). Underground (UW) and surface water (SW) were contaminated with lead. The mean lead concentration for underground water ranged from 187.67 ± 99.05 to 1273.00 ± 444.00 µg/L. For the surface water, the mean lead concentration ranged from 413.00 ± 202.43 to 4235.00 ± 121.75µg/L. These results indicated that, the mean lead concentration for both underground and surface water samples have exceeded the WHO and FAO/WHO acceptable guidelines of 10µg/L and 100µg/L for underground and surface water respectively. Statistical analysis indicated that there was no significant difference ($P < 0.05$) in lead concentrations in residential soil within each community but significantly different ($P < 0.05$) across communities. However, lead concentrations in common grains were not statistically significant ($P < 0.05$) within each community and across the communities, except for the millet from Kwali that was significantly higher. Results also indicated that blood lead levels in both children and adults within each community was significant ($P < 0.05$), however, comparison across communities indicated no statistical significance ($P < 0.05$) across

most of the communities. In animal blood samples, blood lead concentration was significant ($P < 0.05$) within community and across communities. Similarly, significant ($P < 0.05$) differences exist in underground water samples within community, while in surface water samples it was not significant. Apart from compound residential soil in few remediated villages, lead concentrations in all other samples were still significantly ($P < 0.05$) above internationally threshold levels of concern. Public education, remediation process, chelation therapy and safe mining practices must be fully implemented to bring the lead poisoning under control and concentration of lead levels in these communities below the threshold level of concern in order to save future generation of manpower from extinction or permanent incapacitation.

Table of Contents

Cover Page **.....i**

Approval Page.....ii

Title Page.....iii

Declaration iv

Certification**.....v**

Dedication **.....vi**

Acknowledgement**.....vii**

Abstract**.....x**

Table of Contents..... **.....xiii**

List of Maps.....xviii

List of Tables**.....xix**

List of Figures **.....xx**

List of Appendices**.....xxi**

Abbreviations and Symbols **.....xxiii**

CHAPTER ONE

1.0 INTRODUCTION.....1

1.1 Lead……...1

1.2 History of Lead Poisoning in Zamfara State……...5

1.2.1 Findings of the joint mission……...7

1.3 Environmental Lead Pollution……....13

1.3.1 Soil pollution……..13

1.3.2 Water pollution……...15

1.3.3 Causes of water pollution.…….…….....16

1.3.4 Air pollution.....18

1.3.5 Food and Lead poisoning.....25

1.3.6 Lead dietary intake.....26

1.4 Statement of the Problem.....28

1.5 Justification of the Study.....29

1.6 Aim and Objectives of the Study.....29

1.6.1 Aim of the study 29

1.6.2 Objectives of the study.....29

1.6.3 Scope of the study.....30

1.7 Research Hypothesis.....30

CHAPTER TWO

2.0 LITERATURE REVIEW……31

2.1 Heavy Metals and their Health Effects...……31

2.2 Lead…….....32

2.2.1 Health effects of lead.…….....33

2.2.2 Sources of exposure to lead…….....34

2.2.3 Global Dimension of Lead Exposure…….....39

2.2.4 Lead contamination before and after industrial revolution…….....40

2.2.5 Occupational lead exposure..……..……41

2.2.6 Environmental lead exposure …….....42

2.2.7 Toxic effects of lead in children and adults……47

2.2.8 Reproductive and developmental effects ……..……48

2.2.9 Treatment of lead poisoning……...50

2.2.10 International standards for lead levels……56

2.3 Methods of Lead Determination in Different Samples…….....58

2.3.1 Analysis of biological samples ……..58

2.3.2 Analysis of environmental samples ….……..59

2.4 Atomic Absorption Spectroscopy (AAS)…….....60

CHAPTER THREE

3.0 MATERIALS AND METHOD…….66

3.1 Materials……...66

3.1.1 Equipments and glassware.…….....66

3.1.2 Chemicals and reagents…….....67

3.1.3 Instrumentation…….....67

3.2 Methods……..……..69

3.2.1 Geographical location of the study areas.....69

3.2.2 Advocacy and community relations……..73

3.2.3 Sample and sampling site selection ….……..75

3.2.4 Samples collection and preservation……..75

3.2.5 Preparation of lead stock solution for calibration curve……77

3.2.6 Calibration curve for lead in soil samples……..77

3.2.7 Calibration curve for lead in grain samples…….....77

3.2.8 Calibration curve for lead in blood samples ……..78

3.2.9 Calibration curve for lead in underground and surface water samples …….78

3.2.10 Preparation and analysis of samples……...78

3.2.11 Validation of the adopted methods……81

3.2.12 Demographic distribution of socio-economic opportunities in the selecting mining communities……...82

3.2.13 Statistical analysis …….....82

CHAPTER FOUR

4.0 RESULTS…….83

4.1 Geographical Position System (GPS) location of the Sampling Compounds……....83

4.2 Calibration curve and Validation of the adopted methods……...83

4.2.1 Calibration curves of the adopted methods…….83

4.2.2 Validation results of the adopted methods…….88

4.3 Lead Concentration of Residential Compound Soil……..89

4.4 Lead Concentration in Sorghum, Millet, and Maize ……91

4.5 Comparison of Mean Lead Concentration of the three common grains….95

4.6 Lead Concentration in Human (Children and Adults) Blood Samples…..97

4.7 Comparison of Lead Concentration between Children and Adults …….100

4.8 Lead Concentration in Animals Blood Samples ……102

4.9 Lead Concentration in Underground and Surface Water Sample……104

4.10 Demographic Data of the Selected Mining Communities…….....108

CHAPTER FIVE

5.0 DISCUSSION…….110

5.1 Lead Concentration in Residential Compound Soil Samples……110

5.2 Lead Concentration in Sorghum, Maize and Millet Samples…….112

5.3 Lead Concentration in Children and Adults Blood Samples…….113

5.4 Lead Concentration in Animal Blood Samples……115

5.5 Lead Concentration in Underground and Surface Water…….116

5.6 Demographic distribution of socio-economic opportunities……...118

CHAPTER SIX

6.0 SUMMARY, CONCLUSION AND RECOMMENDATIONS……....120

6.1 Summary…….120

6.2 Conclusion……...123

6.3 Recommendations……...124

References……...126

Appendices……..xxv

LIST OF MAPS

Map of Nigeria showing Zamfara State of Nigeria.....71

Map of Zamfara State of Nigeria showing the Local Govt. Areas.....72

LIST OF TABLES

1.1 Top Ten Sources of Toxic Pollution Hotspot.....12

1.2 The list of the top six toxic health threats……...13

2.1 International Standards for Lead Levels…….57

2.2 Standard Atomic Absorption Conditions for Lead……....65

4.1 Validation results of the adopted methods……..88

4.2 Demographic Distribution of Socio-Economic Opportunities.....109

4.3 Geographical Position System (GPS) Coordinates of the Sampling Compounds (as Appendix I).....xxv

4.4 Lead Levels in Residential Compound Soil Samples (as Appendix II).....xxvi

4.5 Lead Levels in Sorghum (GS1) Sample (as Appendix III).....xxvii

4.6 Lead Levels in Millet (GS2) Sample (as Appendix IV) xxviii

4.7 Lead Levels in Maize (GS3) Sample (as Appendix V).....xxix

4.8 Comparison (Mean± SEM) of Lead Concentration (mg/Kg) in Common Grains (as Appendix VI).....xxx

4.9 Human Blood Lead Levels (BLLs) in Children (as Appendix VII).....xxxi

4.10 Human Blood Lead Levels (BLLs) in Adults (as Appendix VIII).....xxxii

4.11 Comparison (Mean ± SEM) for Children & Adults Blood Lead Levels (BLLs) (µg/dL) Samples (as Appendix IX).....xxxiii

4.12 Animals Mean Blood Lead Levels (BLLs) (as Appendix X).....xxxiv

4.13 Lead Levels in Underground Water (UW) Sample (as Appendix XI).....xxxv

4.14 Lead Levels in Surface Water (SW) Sample (as Appendix XII).....xxxvi

4.15 Comparison (Mean ± SEM) of Lead Concentration in Underground and Surface Water (as Appendix XIII).....xxxvii

LIST OF FIGURES

2.1 2,3-Dimercaptosuccinic Acid (DMSA, Succimer).....53

2.2 Newly synthesized monoesters of DMSA.....54

2.3 Map of Nigeria indicting Zamfara State.....71

3.2 Map of Zamfara State and the 14 LGAs.....72

3.3 Advocacy and Community Relations 74

4.1 Calibration Curve for Residential Soil Samples.....84

4.2 Calibration Curve for Grain samples……..85

4.3 Calibration Curve for Lead Levels in Human and Animal Blood samples……86

4.4 Calibration curve for Lead Levels in Underground and Surface water…….87

4.5 Mean Lead Concentration in Residential Soil Samples (SLLs).....90

4.6 Mean Lead Levels Concentration in Sorghum (GS1).....92

4.7 Mean Lead Levels Concentration in Millet (GS2).....93

4.8 Mean Lead Levels Concentration in Maize (GS3).....94

4.9 Comparison of Mean Lead Levels in Common Grains.....96

4.10 Blood Lead Levels (BLLs) in Children.....98

4.11 Blood Lead Levels (BLLs) in Adults.....99

4.12 Comparison of Blood Lead Levels (BLLs) in Children & Adults…….....101

4.13 Comparison of Animals Mean Blood Lead Levels……..103

4.14 Lead Levels in Underground Water (UW)……..105

4.15 Lead Levels in Surface Water (SW)…….....106

4.16 Comparison of Lead Levels in Underground Water & Surface Water.....107

LIST OF APPENDICES

Appendix I: Geographical Position System (GPS) Coordinates of the Sampling Compounds.....xxv

Appendix II: Lead Levels in Residential Soil Compound Sample.....xxvi

Appendix III: Lead (Pb) Levels in Sorghum (GS1) Sample.....xxvii

Appendix IV: Lead (Pb) Levels in Millet (GS2) Sample.....xxviii

Appendix V: Lead (Pb) Levels in Maize (GS3) Sample.....xxix

Appendix VI: Comparison (Mean± SEM) of Lead Concentration (mg/Kg) in Common Grains.....xxx

Appendix VII: Human Blood Lead Levels (BLLs) in Children.....xxxi

Appendix VIII: Human Blood Lead Levels (BLLs) in Adults.....xxxii

Appendix IX: Comparison (Mean ± SEM) for Children & Adults Blood Lead Levels (BLLs) (µg/dL) Samples.....xxiii

Appendix X: Mean Blood Lead Levels (BLLs) for Goat.....xxxiv

Appendix XI: Mean Blood Lead Levels (BLLs) for Sheep and Chicken.....xxxv

Appendix XII: Comparison of Mean Blood Lead Levels (BLLs) in Goat, Sheep and Chicken.....xxxvi

Appendix XIII: Lead Levels in Underground Water (UW) Sample.....xxxvii

Appendix XIV: Lead Levels in Surface Water (SW) Sample.....xxxviii

Appendix XV: Comparison (Mean ± SEM) of Lead Concentration in Underground and Surface Water.....xxxix

Appendix XVI: Advocacy, field trips & collection of samples xl

Appendix XVII: AAS Absorbance of Lead in Compound Soil Samples of Selected Mining Communities in Zamfara State Nigeria.....xli

Appendix XVIII: AAS Absorbance of Lead in Sorghum (GS1) Samples of Selected Mining Communities in Zamfara State Nigeria.....xlii

Appendix XIX: AAS Absorbance of Lead in Millet (GS2) Samples of Selected Mining Communities in Zamfara State Nigeria.....xliii

Appendix XX: AAS Absorbance of Lead in Maize (GS3) Samples of Selected Mining Communities in Zamfara State Nigeria.....xliv

Appendix XXI: AAS Absorbance of Lead in Children Blood Samples of Selected Mining Communities in Zamfara State Nigeria.....xlv

Appendix XXII: AAS Absorbance of Lead in Adults Blood Samples of Selected Mining Communities in Zamfara State Nigeria.....xlvi

Appendix XXIII: AAS Absorbance of Lead in Animals Blood Samples of Selected Mining Communities in Zamfara State Nigeria.....xlvii

Appendix XXIV: AAS Absorbance of Lead in Underground and Surface Water Samples of Selected Mining Communities in Zamfara State xlviii

Appendix XXV:

Danbaba A [1], Yakasai IA [2], Usman MA [2], Garba M [2], Abdullahi MG [3] (2012).Health and Environmental Effects of Lead Nigeria. *International Journal of Scientific Innovations and Sustainable Development,* Vol. 2, No1, 2012: 47-55.....xlix

Danbaba A [1], Usman MA [2] ,Yakasai IA [2], Garba M [2], (2014). of Blood Lead Levels (BLLs) in Children and Adults of Selected Mining Communities of Zamfara State – Nigeria. *International Journal of Medical, Biological and Pharmaceutical Sciences* (IJMBPS) Vol. 2, No.3: 89- xlix

Danbaba A [1], Usman MA [2] ,Yakasai IA [2] (2015). Assessment of Lead Levels Contamination of Common Grains (Sorguhm, Millet and Maize) Grown in Selected Mining Communities of Zamfara State – Nigeria. *International Journal of Pure and Applied Sciences* Vol.2 No.2:12-21.....xlix

Danbaba A[1], Liman M. L [2], Garba M.A [3] (2015). Solid Mineral Exploration: The Effects of Lead and other Heavy Metals Challenges to Future manpower in Zamfara State – Nigeria. *1st Multidisciplinary Journal (with Press)*.....l

Appendix XXVI: Conference papers.....l

Appendix XXVII: Questionnaire for demographic data.....li

Appendix XXVIII: Research activity in pictures.....lxii - lxix

ABBREVIATIONS AND SYMBOLS

µg/dl	=	Micrograms per decilitre
µg/g	=	Microgram. Per gram
µg/l	=	Microgram per litre
AAS	=	Atomic Absorption Spectrophotometry.
ABU	=	Ahmadu Bello University
AES	=	Atomic Emission Spectroscopy
ALA	=	Aminolevulinic Acid
AOAC	=	Association of Official Analytical Chemists
APHA	=	American Public Health Association
ASV	=	Anode Stripping Voltammetry
ATSDR	=	Agency of Toxic Substances and Disease Registry
BDL	=	Below Detection Limit
BLL	=	Blood Lead Level
Cd	=	Cadmium
CDC	=	Centres for Disease Control and Prevention
CNS	=	Central Nervous System
CO	=	Carbon monoxide
COHb	=	Carboxy Haemoglobin
Concentration	=.	Concentration
CONTAM	=	EFSA Panel on Contaminants in the Food Chain
DMSA	=	Dimacapto Succinic Acid
EC	=	European Commission
EFSA	=	European Food Safety Association
EPA	=	Environmental Protection Agency

ESADDI	=	Estimated Safe and Adequate Daily Dietary Intake.
EU	=	European Union
F.D.A	=	Food Drug Administration
FAAS	=	Flame Atomic Absorption Spectrophotometry
FAO	=	Food and Agricultural Organization
FDA``	=	Food and Drug Administration
g	=	Gram
GAR	=	Global Alert Response
GFAAS	=	Graphite Flame Atomic Absorption Spectrophotometry
GFR	=	Glomerular Filtration Rate
GIT	=	Gastro Intestinal Track
GPS	=	Geographical Position System
HCl	=	Hydrochloric Acid/Hydrogen Chloride
HPLC	=	High Pressure Liquid Chromatography
IAEA	=	International Atomic Energy Agency
IARC	=	International Agency for Research on Cancer
ICH	=	International Committee on Harmonization
ICP	=	Inductively Coupled Plasma
ICP-AES	=	Inductively Coupled Plasma Atomic Emission
ICP-MS	=	Inductively Coupled Plasma Mass Spectrometry
IDMS	=	Isotope Dilution Mass Spectroscopy
IPCS	=	International Programme on Chemical Safety
JECFA	=	Joint FAO/WHO Expert Committee on Food Additives
JEU	=	Joint Environmental Unit
Kg	=	Kilogram

LGA	=	Local Government Area
LoD	=	Level of Detection
LoQ	=	Level of Quantitation
mg/Kg	=	Milligram per Kilogram
ml	=	Millilitre.
MS	=	Mass Spectroscopy
MSF	=	Medicines Sans Frontier
MUSRL	=	Multi-User Science Research Laboratory
NARICT	=	National Research Institute for Chemical Technology
ND	=	Not Detected.
NFELTP	=	Nigeria Field Epidemiology and Laboratory Training Program
NIOSH	=	National Institute for Occupational Safety and Health
NO	=	Nitrogen (II) oxide
NO_2	=	Nitrogen dioxide
NWRI	=	National Water Research Institute
OCHA	=	Office for the Coordinator of Humanitarian Affairs
OSHA	=	Occupational Safety and Health Administration
P	=	Probability
Pb	=	Lead (element)
PBT	=	Persistent Bioaccumulative and Toxic
PEL	=	Permissible Exposure Limit
ppm	=	Parts per million.
REL	=	Recommended Exposure Limit
ROS	=	Reactive Oxygen Species
S.E.M	=	Standard Error of the Mean.

SCF	=	Scientific Committee on Food
SW	=	Surface Water
T½	=	Half life
UNECE	=	United Nations Emergency Committee on Environment?
UNEP	=	United Nations Environmental Protection
US-ATSDR	=	US- Agency of Toxic Substances and Disease Registry
US-EPA	=	United State- Environmental Protection Agency
UV	=	Ultra-Violet
UW	=	Underground Water
VDRs	=	Vitamin D Receptors
WHO	=	World Health Organization
WWPP	=	World Worst Pollution Problem
XRF	=	X-Ray Fluorescence

CHAPTER ONE

1.0 INTRODUCTION

1.1 LEAD (Pb)

Lead is a naturally occurring element that belongs to Group IVA of the periodic table and has an atomic number of 82 and atomic mass of 207.2 g/mol. It is a silver - bluish white metal that is found in small amounts in the earth‘s crust although it is rarely found naturally as a metal. Usually it is found combined with two or more other elements to form lead compounds. It is highly malleable, ductile and a relatively poor conductor of electricity. Lead is very resistant to corrosion but tarnishes upon exposure to air (Korn *et al*., 2006).

The main oxidation states of lead are +2 and +4, although in the environment +2 is the prevalent form. Inorganic lead compounds, such as lead phosphate and lead carbonate, usually contain lead in its divalent state (+2). The solubility of lead compounds in water is a function of pKa, hardness, salinity and the presence of humic material (ATSDR, 2007). Industrially synthesised organic lead compounds, such as alkyl - lead compounds, have been used mainly as fuel additives as anti - knock agents in combustion engines. Tetraethyl lead (Pb $(C_2H_5)_4$ and tetramethyl lead (Pb $(CH_3)_4$) are the most commonly used alkyl - lead compounds. They are both highly volatile, lipid soluble liquids. Human exposure to these compounds is mainly through inhalation of leaded petrol vapours, dermal exposure to leaded petrol and ingestion of lead-contaminated soil, food or water. Once absorbed into the body, these compounds may be dealkylated to divalent lead ions by cytochrome P450 mono - oxygenase activity and this must be considered in total exposure assessment

(WHO/IPCS, 1995). Alkyl-lead compounds were included on the EPA‘s Persistent Bioaccumulative and Toxic (PBT) chemicals programme, aiming at reducing their use and developing safer alternatives.

Although Lead as a metal is found in small amount in the earth’s crust, but it is the most abundant of the heavy metals in the earth’s crust. It has been used since prehistoric times, and has become widely distributed and mobilized in the environment. Exposure to and uptake of this non-essential element has consequently increased the lead exposure challenges to environmental. Both occupational and environmental exposures to lead remain a serious problem in many developing and industrializing countries, as well as in some developed countries (Smith, 1984).

Lead is a naturally occurring metal, found throughout the environment. High levels of lead have entered the environment through human activities such as mining, industrial processes and burning fuels. Lead is used in hundreds of products, for example, as an additive in gasoline, in the production of batteries, as an additive in some paints, in solder, in making tainted glass and crystal for ammunition, in ceramic glazes, and in some cosmetics and traditional medicines. Drinking water delivered through lead pipes or pipes joined with lead solder may contain lead (Nriagu and Pacny, 1988).

Lead in the environment can easily contaminate food through water or through atmospheric lead deposition on agricultural crops. Control measures have been taken to regulate lead in paint, food cans, water pipes and petrol in Europe since the 1970s. Leaded petrol was banned from use in the European Union in 2000 with exemptions possible until 2005 and continued use only allowed in vintage cars.

The general population is exposed to lead via food, water, air, soil and dust. Food is the major source of exposure to lead, although for children ingestion of soil and dust can also be an important contributor (WHO, 2007a; EFSA, 2010). Absorption of lead from the gastrointestinal tract depends on host characteristics and on the physicochemical properties of the ingested material. Absorption of ingested soluble lead compounds appears to be higher in children than in adults. Absorption is lower in the presence of food (Alexander *et al*., 1974; Ziegler *et al*., 1978; Heard and Chamberlain, 1982; Rabinowitz and Needlman, 1982; James *et al*., 1985).

Absorption of inhaled sub-micron sized particles occurs in the respiratory tissues whereas larger-sized particles are transferred into the pharynx and are then swallowed (Hursh *et al*., 1969; Hursh and Mercer, 1970; Morrow *et al*., 1980). Lead can accumulate in the body, primarily in the skeleton. From the skeleton, it is released gradually back into the blood stream, particularly during physiological or pathological periods of bone demineralisation such as pregnancy, lactation and osteoporosis, even if lead exposure has already ceased. Maternal transfer of lead occurs through the placenta and subsequently during breast feeding. Half-life for inorganic lead in blood is approximately 30 days and for bone it is between 10 and 30 years (Rabinowitz *et al*., 1976).

Although the acute toxicity of lead is low, chronic oral exposures to inorganic lead by experimental animals and observations in occupationally exposed humans have been shown to affect multiple organs. Due to its long half-life in the body, chronic toxicity of lead is of most concern when considering the potential risk to human health. The central nervous system is the main target organ for lead toxicity. In adults, lead-associated neurotoxicity was found to

affect central information processing and short-term verbal memory, to cause psychiatric symptoms and to impair manual dexterity. There is considerable evidence demonstrating that the developing brain is more vulnerable to the neurotoxicity of lead than the matured brain and this is of particular concern even at relatively low lead exposure. In adults, a number of studies have also identified an association between blood lead concentration, elevated systolic blood pressure and chronic kidney disease at relatively low blood lead levels (IARC, 2006; ATSDR, 2007). The International Agency for Research on Cancer (IARC) classified inorganic lead as probably carcinogenic to humans (Group IIA) in 2006 (IARC, 2006).

International and European health-based guidance values for lead exposure have been amended several times as new information has come to light. In 2010, the European Food Safety Authority's (EFSA) Panel on Contaminants in the Food Chain concluded that the provisional tolerable weekly intake (PTWI) of 25 μg/kg body weight set by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1986 and endorsed by the European Commission's (EC) Scientific Committee for Food (SCF) in 1989 was no longer appropriate and that, as there was no evidence for a threshold for a number of critical endpoints including developmental neurotoxicity and adult nephrotoxicity, it would not be appropriate to derive a PTWI (EFSA, 2010; WHO, 1986; EC, 2006).

In light of the particular concern for lead exposure in children, it was considered important to better identify major dietary sources of lead in Europe. The current report provides updated information on the levels of lead found in a range of foods on the European market and estimates dietary exposure using detailed

individual data from the Comprehensive European Food Consumption Database covering seven age groups from infants to the very elderly.

Scientists have suggested that all exposure to Lead should be avoided and that there is no safe exposure concentration (Joint UNEP/OCHA, 2010)

1.2 History of Lead Poisoning in Zamfara State

In March 2010, an unusually high number of deaths (about 163), primarily among children under the age five years in Bukkuyum and Anka Local Government Areas (LGAs) of Zamfara State were reported by Médecins Sans Frontières (MSF-Holland) to state health authorities (Joint UNEP/OCHA, 2010).

Further study of blood samples taken by MSF revealed that the increased mortality was the result of acute lead poisoning, determined to be caused by massive environmental contamination from artisanal mining and processing of gold found in lead-rich ore. The grinding of the ore into fine particles resulted in extensive dispersal of lead dust in the villages concerned, including within family compounds. Ingestion and inhalation of the fine lead particles was determined to be the major reason for high blood lead levels in victims' bodies. Joint mission stated that blood lead levels (BLLs) were 'unprecedented' for human beings, according to the Centres for Disease Control and Prevention (CDC).

The Joint UNEP/OCHA Environment Unit (JEU) in 2010 led a sampling and analysis mission to investigate the lead pollution emergency in Zamfara State, following requests for assistance from the Federal Ministry of Health of Nigeria and the UN Resident Coordinator. Specifically, the mission focused on

determining the quantities of lead in ground and surface water, building on investigations already conducted by the CDC, the World Health Organization (WHO), and the National Water Resources Institute of Nigeria (NWRI), and a team from TerraGraphics Environmental Engineering/The Blacksmith Institute, as it was determined that there was insufficient information in these domains. The mission also took the opportunity to look at lead levels in soil, and mercury levels in air (Joint UNEP/OCHA, 2010).

Field work focused on five villages in Anka Local Government Authority (LGA), two of which had been confirmed as lead-contaminated (Abare and Sunke); two of which were newly suspected of contamination (Kirsa and Bagega); and one of which had been remediated (Dareta).

Blacksmith/TerraGraphics used the US Environmental Protection Agency (US EPA) guidelines of 400 parts per million (ppm) in soil (dry) to indicate a potential health risk. The concentrations of lead in soil in the villages varied significantly within villages, suggesting a human factor in the dispersion of lead. Higher concentrations (up to 8%) were seen close to drinking water wells and to other surface water sources. At other locations, concentrations were often much lower. At ore processing locations, incidental ingestion of soil (via hand-to-mouth behaviour and eating food with dirty hands) by young children could be a substantial exposure route. Since processing is often done within the walls of home compounds, infants and toddlers would be particularly exposed (Joint UNEP/OCHA, 2010).

Measurements of Lead in drinking water showed that 25-30% of wells in the villages assessed did not meet the WHO guideline for lead in drinking water, although in most wells, the limit of 10µg/l was exceeded by no more than

several µg/l. However, in some wells, concentrations of up to 10-15 times the guideline were found. In most cases, exceeding of the guideline was coupled with high concentrations of lead in the soil around the well. Therefore, the mission suspects that the contamination of the wells has been caused by dust deposition and soil run-off from sites where lead-contaminated ore has been/is being processed. This is consistent with the mission's findings that drinking water from boreholes was never contaminated (Joint UNEP/OCHA, 2010).

This would imply that even the relatively low concentrations found in most wells by the mission might be a risk, especially for young children.

Food pathways and crops contamination of the common grains in the mining communities was part of the major contributions of this research to knowledge and literature. Results obtained would further guide on the sources of lead exposure and lead control.

1.2.1 Findings of the joint mission

Blacksmith Institute's World´s Worst Pollution Problems (WWPP) Report 2010 disclosed that in early 2010, doctors from Medicines Sans Frontiers (MSF) were conducting field visits in Zamfara State, in northwest Nigeria, when they noticed an absence of children in several villages. When these doctors inquired with the local population about the low numbers of children, they were told that most children had died unexpectedly. This was reported to the State Health Authorities, who invited international specialists to investigate the cause of death. Investigations led by the US Centres for Disease Control and Prevention (US-CDC), in collaboration with Federal and Zamfara State authorities, MSF, Blacksmith Institute, and the WHO, revealed that the outbreak was caused by

acute lead poisoning. The source was massive environmental contamination from the informal processing of lead-rich ore to extract gold. Men from the villages had brought rocks containing gold ore into the villages from small-scale mining operations; however, the villagers did not know that the ore also contained extremely high levels of lead. The ore was crushed inside village compounds, spreading lead dust throughout the community (Blacksmith Institute, 2010).

1.2.1.1 Blood lead levels (BLL)

Blacksmith Institute joined a CDC field investigation that measured blood-lead concentrations in 113 samples from young children in the villages of Yargalma and Dareta. The results showed that 100% of the children had blood-lead levels exceeding 10µg/ dL (the international standard for the maximum safe levels of lead in blood), 96% exceeded 45µg/dL, and 84% exceeded 70µg/dL. It was also discovered that there were 78 deaths in Yargalma (30% of the population was less than 5 years old in the village) and 40 deaths in Dareta (20% of the population was less than 5 years old), totalling 118 deaths in these two communities since the beginning of the year (2010). 95% of all deaths were in children under the age of five (Blacksmith Institute, 2010).

As of September 2010, it was estimated that a total of 2,500 children had life-threatening levels of lead in their blood. Further investigation identified at least five additional villages where similar ore processing activities are common (Blacksmith Institute, 2010 and WWPP, 2011).

1.2.1.2 Water

The mission found that drinking water from wells did not meet WHO and Nigerian standards (10µg/l) for lead limits – in at least one case, exceeding this by more than tenfold. Water in ponds was often highly contaminated (frequently reaching 200 µg/l). However, no boreholes were found to have been contaminated, indicating that lead pollution most likely remains confined to areas (wells and ponds) where processing has taken place, and has not (yet) spread throughout the groundwater aquifer. The lead found in wells and ponds was likely to have come from external sources (processing) rather than to be naturally occurring (Joint UNEP/OCHA, 2010)

High concentrations of lead in surface waters (up to more than 1,000µg/l – 10 times higher than the exposure limit suggested by FAO for livestock) were often found in ponds, rivers and lakes. This is not surprising since surface water sources are often used for processing ore and livestock drink from same source. However, it is common practice to use most or the entire animal after it has been slaughtered, including using bones for soup. Since people in the villages report illness and death among livestock, it is reasonable to suspect that the consumption of such meat might also be an important exposure route for humans. The geographic extent of this crisis and the number of people potentially affected are still not known, according to CDC and Nigerian Field Epidemiology and Laboratory Training Program (NFELTP, 2013)

Lead concentrations found by the mission are consistent with earlier (pre-rainy season) measurements taken by the National Water Resources Institute (NWRI), suggesting that concentrations were not significantly affected by the particularly abundant rainy season.

1.2.1.3 Soil

The mission believes that contaminated water was less of a concern than contaminated soil, due to the levels and extent of contamination, meaning that priority should be given to soil in remediation efforts. In the four as-yet unremediated villages visited, the soil was often highly polluted with lead: while (for example) the US standard is 400 parts per million (ppm), readings were sometimes as high as 60,000 ppm. Since young children readily ingest soil as part of normal hand-to-mouth behaviour, such high concentrations expose children to potentially harmful amounts of lead (Joint UNEP/OCHA, 2010).

In many areas in all villages sampled, including family homes and compounds, the soil lead concentration exceeded 100,000 ppm, far above the recommended maximum of 400 ppm considered acceptable for residential areas. Ingestion of contaminated soil has been the primary pathway of lead exposure (Blacksmith Institute, 2010).

Joint UNEP/OCHA (2010), suggested that further study of food pathways (livestock, crops) should be undertaken by Federal and State experts, with support from international partners, as livestock was seen to be drinking from contaminated ponds, and crops were found to be growing in contaminated soil near affected wells.

The concentrations of lead in soil in the villages varied significantly within villages, suggesting a human factor in the dispersion of lead. Higher concentrations (up to 8%) were seen close to drinking water wells and to other surface water sources. At other locations, concentrations were often much lower.

As for concentrations of mercury in air, for which 50 nanograms per cubic meter is the maximum exposure for non-industrial workers in the Netherlands, for example, readings of up to 24 micrograms per cubic meter – nearly 500 times the acceptable limit – were measured. Toxic effects cannot be ruled out, especially as the exposure is more or less chronic (Joint UNEP/OCHA, 2010).

Blacksmith Institute's World´s Worst Pollution Problems Report (WWPP-2010-Report) estimated that about 10 to 20 million people worldwide work in artisanal gold mining. Artisanal miners often use toxic materials to separate metals from the surrounding ore and silt. In artisanal gold mining, the most common separation process is known as mercury amalgamation. Due to lack of awareness, as well as lack of environmental, health, and safety regulations in these small mining industries, miners are often exposed to dangerous levels of toxic materials. The mercury used in these mining activities can also be responsible for the contamination of water and soil, posing health risks for communities near and far, but also to the global population. About 1/3 of the global annual release of mercury into the environment is due to artisanal gold mining (Blacksmith Institute, 2010).

The list of top sources as in Table 1.1 varies slightly from the list of the top ten pollution problems. This is because the list of the top ten pollution problems counts only sites where a specific activity releases a specific pollutant. For example, mining and ore processing makes the top pollution problems list twice, once for mining operations that emit mercury, and again for mining operations that emit lead. Petrochemical industries and municipal/industrial waste disposal sites do not make the list of top pollution problems, but they do make the list of top sources. This is because these sources emit a variety of

pollutants and no single pollutant shows up often enough to put the source and a specific pollutant in the top ten pollution problems list (Blacksmith Institute, 2010).

Table 1.1: Top Ten Sources of Toxic Pollution Hotspots

Source Activity	Estimated Global Population at Risk
Mining and Ore Processing	7,023,000
Metal Smelting	4,955,000
Chemical Manufacturing	4,787,900
Artisanal & Small-Scale Mining	4,233,400
Mixed Industrial Estates	3,862,800
Agricultural Production	3,274,400
Industrial/Municipal Waste Disposal	3,210,800
Heavy Industry (metal casting, rolling, stamping)	2,771,900
Petrochemical Industries	1,917,700
Tannery Operations	1,890,600

Populations' estimates are preliminary and based on an ongoing global assessment of known polluted sites.

The WWPP-2010 report revisits the subject of pollution problems, but draws upon the substantial volume of research the organizations have conducted on polluted sites over the last two years to identify the specific pollutants that are causing the most harm.

The second section of the report shown in Table 1.2 focuses on the six toxic pollutants that have the greatest impact on human. The list of the top six toxic health threats was generated using research from site assessments conducted by

Blacksmith Institute field investigators. The number of people currently estimated to be at risk from these sites exceeds 56 million; however, this number will rise as more sites are evaluated.

Table 1.2: The list of the top six toxic health threats

Top Six Toxic threats	Estimated Population at risk at identified sites (millions of people)	Estimated Global impact (millions of people)
1. Lead	10	18-22
2. Mercury	8.6	15-19
3. Chromium	7.3	13-17
4. Arsenic	3.7	5-9
5. Pesticides	3.4	5-8
6. Radio nuclides	3.3	5-8

1.3 Environmental Lead Pollution

1.3.1 Soil pollution

Lead is considered one of the environmentally hazardous elements because, along with cadmium, mercury, copper, zinc and chromium it poses a particularly high risk of disturbing the chemical balance in the ecosystem (Kabata-Pendias *et al*., 1979).

The content of lead in the soil is directly related to its granularity and mineral composition, as well as the origin of its parent rock. The natural content of lead in the soils formed of sands does not normally exceed 16 mg per kg of soil, and in more packed soils it is usually within the range of 13 to 60 mg/kg of soil

(Kabata-Pendias and Pendias, 1993; Piotrowska *et al*., 1994 ; Dudka *et al*., 1995).

Soil contamination by heavy metals is a significant problem, which leads to changes of soil characteristics and limits productive and environmental functions. Polluted soils are no longer appropriate for agricultural production, because they are unable to produce healthy food. Assessment of soil pollution by heavy metals in Slovakia is determined by limit values of risk elements, which were set by law.

It has been repeatedly shown that heavy metals have a negative impact not only on the size, activity and diversity of soil microbial communities (Chander *et al*. 2001), but they also affect soil enzymes (Belyaeva *et al*., 2005). Soil enzyme activity is a reliable indicator reflecting the biological situation in soils and it is possible to very quickly obtain credible results of soil pollution. Reaction of enzymes to soil pollution is faster in comparison with monitoring of the chemical and physical properties (Hinojosa *et al*., 2004).

The factors contributing to the excessive intake of lead and other heavy metals are: acidity of soil, low content of humus and its low sorption capacity. The results reported in Pulawy suggest that in Poland normal content of lead in the soil does not exceed 20 mg per kg of soil. The mean content of lead in cultivated land in Poland is 13.8 mg per kg of soil, the range of variation from 0.1 to 1723 mg per kg of soil. However, the development of industry and motorization and increasing use of communal and industrial waste water for liming and fertilizing soil as well as long-lasting use of some pesticides may lead to excessive concentrations of lead, cadmium, zinc, copper or mercury in the soil (Szrzedzinski *et al*., 1983). It has been discovered that there is a

correlation between content of heavy metals in soils and in vegetables grown on them (Szymczak *et al*., 1983; Czarnowska and Gworek, 1987). There is evidence that the content of lead, cadmium or zinc in the vegetables grown using environmentally safe methods may be up to 60% lower than in those grown in a traditional way (Leszczynska, 1999).

This is also of great importance to human health, as lead belongs to toxic elements which can accumulate in the human organism (the maximum daily dose of lead which does not lead to accumulation is 0.50 mg (Sikorowska, 1966). Excessive amounts of this element cause disorders in the metabolism of other microelements, e.g. iron (which may be manifested as anemia), copper or zinc (which has a negative effect on the function of heart and kidneys) (Oledzka, 1999).

1.3.2 Water pollution

The major portion of our planet is occupied by water. Water is a precious resource of life and forms the base of survival. It occupies maximum portion of the earth, out of which 97% is occupied by the salt water, 2% is trapped under glaciers and 1% is drinking water. Once the disposal of waste material begins to exceed the volume of water, it triggers the issue of water contamination. There are many water pollution causes and effects but it is necessary to analyze the causes and effects to save our planet from destruction. Being an important resource, humans must work towards conserving water (Galkwad, 2011).

Groundwater contamination by heavy metals is often associated with mining activities and the subsequent processing of ores. Heavy metals enter the surface water in dissolved form and in association with substances washed off the ground, where they can migrate over long distances (Frankowski *et al*., 2009).

Polluted water sources may become the source of undesirable substances, which are dangerous for human health causing various cancers, cardiovascular or neurological diseases (Galušková *et al*., 2010).

1.3.3 Causes of water pollution.

There are many factors that lead to water pollution. It is essential to first understand the sources that contribute to this problem. There are point and non point sources that raise the problem of water pollution. Harmful chemicals from the factories, industries or the hazardous smoke released by the vehicles, fall under point source as they directly target water contamination. However, the pollution created by the factories is well regulated and so fall under non point source, and same goes for transport (Galkwad, 2011).

1.3.3.1 Agriculture

Lakes, rivers and streams, are sources of fresh water which are under constant threat of getting contaminated. The farmers or the landowners that are situated near the freshwater bodies contribute to the pollutants which mix up into the water polluting it. The farmers who produce crops on large-scale, need to treat and nurture their crops well to provide good quality of food to the increasing population. Taking care of these crops comes at a great price due to the usage of pesticides and fertilizers. These pesticides get washed away due to rain which eventually flows into the freshwater bodies harming the aquatic life and bringing down the quality of drinking water (Frankowski *et al.*, 2009).

1.3.3.2 Oil Leakage

Oil Leakage poses a great problem to the local life and also the coastal business. However, aquatic life gets severely affected due to the oil spills by tankers and ships. Oil forms a thick layer which blocks the light and suffocates the fish causing them to die (Galušková *et al*., 2010).

1.3.3.3 Industrial Waste

The waste from the industries or factories contributes a lot to the problem of water pollution. They use injection wells to insert all the harmful and insolvent materials into the water bodies. Emission of such materials proves to be fatal due to its ill effects on the health of humans and animals. Marine water is an easy source of water that can easily get contaminated. It is easily available to dump industrial wastes and local wastes. The water bodies that are already contaminated ultimately flow into the ocean bringing in all the harmful and dirty waste with it (Galkwad, 2011).

1.3.3.4 Dumping of Plastics

Plastic is an inevitable material which is used and produced on a large-scale. Many products are manufactured from plastic which are greatly consumed around the world. Plastic takes around 400 years to degrade and kill many fish and sea animals. Though the body of the dead animal decomposes faster, the plastic will remain and pose a threat to other sea animals. The water gets contaminated with plastics due to dumping it straight into the water or if it is thrown on the road it gets washed away by the rain, eventually landing into the freshwater bodies (Galkwad, 2011).

1.3.3.5 Household Products

The products that are used while performing these activities include soaps, detergents, shampoos, which are harmful chemicals and cannot get completely filtered when they get mixed up water. Ultimately the water gets spoiled and makes living difficult for humans and freshwater animals.

1.3.3.6 Sewage

Sewage often contains faeces and the waste from household chores. These wastes are controlled through sewage pipes which are treated in water treatment plants. However, if sewage is left unattended, it causes water contamination and gives rise to many waterborne diseases. Many developing countries that do not have access to good living conditions and water face the problem of sewage and water pollution; even in the developed countries if the sewage is not treated well, it will create many health problems and affect the fresh water by killing the organisms living in it (Galkwad, 2011).

1.3.4 Air pollution

Clean air is considered to be a basic requirement for human health and well being, (WHO, 2000). Individual and population exposure to air pollution is caused by both indoor and outdoor sources. Although the components of indoor and outdoor pollution may be the same, and the exposure–response relationship is not affected by the source of a specific pollutant, outdoor and indoor sources can usefully be treated separately as they are determined by different factors and require different management policies.

Anthropogenic air pollution (*i.e.* that superimposed on the background of natural pollution originating from plants, radiological decomposition, forest fires, volcanic eruptions, *etc.*) has existed since people learned how to use fire, but has increased rapidly with industrialization. The well known and severe air pollution episodes in Europe and North America before 1960 provided indisputable evidence that those high levels of air pollution can have very important adverse health effects, including a significant increase in mortality. Until the mid 1980s, it was generally thought that ambient pollution levels in Europe did not threaten human health (Holland *et al.*, 1979). However, results from epidemiological studies during the last 15 years have consistently shown that moderate and low concentrations of traditional pollutants such as ambient particles can have both short- and long-term effects on health.

The traditional ambient particle indicator in Europe has been Black Smoke (BS), measured by reflectometry, representing black particles of aerodynamic diameter <4μm. Particles derived from combustion sources (vehicles, power plants, *etc.*) are generally smaller whilst those coming from abrasion (road dust, windblown soil) are often larger. The European Union adopted a general framework directive for air pollution in 1996 and a daughter directive (CEC, 1999), including ambient particulate matter regulations in 1999.

The short-term effects (acute effects) of particles have are well established for total non-accidental, respiratory, cardiopulmonary and cardiac daily mortality, as well as respiratory hospital admissions (WHO, 2000). There is also evidence of acute effects on respiratory function, lower respiratory symptoms and increased medication use by asthmatic subjects (WHO, 2000).

It is also apparent that the acute effects of air pollution do not represent only short-term harvesting: analyses using distributed lag models have indicated that the effects persist over a longer period of time (>1.5 month) and the extent of mortality displacement may be considerable, depending on the cause of death (Zanobetti *et al*., 2002).

Long-term effects of chronic exposure to ambient particle concentrations have been studied less. An estimate for three countries (Austria, France and Switzerland) using the effects reported from the US cohort studies concluded that about 6% of the annual total mortality may be attributed to air pollution exposure, whilst in the WHO 'Global Burden of Disease' project about 1,000,000 premature deaths are attributed to high PM concentrations worldwide (Ezzati *et al*., 2002).

1.3.4.1 Ozone (O_3)

Ozone is one of a range of photochemical oxidants which are formed as secondary pollutants by the action of solar radiation in the presence of primary pollutants, mainly nitrogen oxides and volatile organic compounds (WHO, 2000). Tropospheric ozone pollution should be distinguished from the problem of stratospheric ozone depletion, which is linked to global warming and risks of UV radiation. Because of its generation procedure, tropospheric ozone is a more important problem in the summertime and in areas with more prolonged sunshine. In the presence of precursor primary pollutants (especially NO), ozone is 'scavenged'. As a result, low concentrations tend to occur in busy city centres, where NO concentrations are high, whilst higher concentrations are observed downwind in city suburbs to which ozone is transported but where NO

and other precursor concentrations are relatively low. Thus the spatial distribution of ozone and resulting personal exposure patterns differ from those of other pollutants. Ozone measurements are often expressed as ppb or $\mu g/m^3$ (1 ppb = 2 $\mu g/m^3$ at 20°C) (Katsouyanni, 2003).

The WHO guidelines for ozone give a level of 120$\mu g/m^3$ for an 8-h average WHO, (2000). The US EPA regulations US-EPA (1996), comprise an 8-h standard of 157$\mu g/m^3$ and a 1-h level of 235$\mu g/m^3$.

Ozone, as a potent oxidant, may react with a variety of biomolecules, potentially causing both short- and long-term effects. They include an increase in the daily total number of deaths, especially for the warm season, an increase in hospital respiratory admissions, increased respiratory symptoms, pulmonary function changes, increased airway responsiveness and airway inflammation (CEOHA, 1996).

A number of field studies done in children and young individuals indicate that pulmonary function decrements can occur at levels of 120–240 $\mu g/m^3$. There is also evidence for association of short-term peaks in O_3 exposures and lung epithelial damage (Broeckaert *et al*., 2000). There are a few studies indicating that long-term ozone exposure may be a risk factor for asthma incidence (McDonnell *et al*., 1999; McConnell *et al*., 2002), lung function growth (Horak *et al*., 2002), and lung cancer incidence and mortality (Beeson *et al*., 1998; Abbey *et al*., 1999).

1.3.4.2 Nitrogen dioxide (NO_2)

Nitrogen dioxide is mainly produced as a result of emissions from vehicles and is thus considered a good indicator of ambient, traffic-generated air pollution

(Rijnders *et al*., 2001). Power plants and fossil-fuel burning industries also contribute to NO_2 pollution. There are also significant indoor sources of NO_2, such as gas stoves (CEOHA, 1996; WHO 2000), and indoor NO_2 levels may dominate the total personal exposure to NO_2.

During high temperature combustion, nitric oxide (NO), NO_2 and other nitrogen oxides (NO_x) are generated. Part of the NO is converted to NO_2 through oxidation reactions which involve oxygen and ozone. NO_2, in the presence of sunlight, participates with hydrocarbons and oxygen in the formation of ozone and other secondary photochemical oxidants and is therefore an important precursor of O_3 formation. NO_2 also reacts with aerosols to form secondary (often acidic) particles (Spengler *et al*., 1990; CEOHA, 1996).

NO_2 is measured routinely by monitoring networks and is expressed either as $\mu g/m^3$ or ppb (1 ppb = 1.913 $\mu g/m^3$ at 20°C).

WHO guidelines provide a 1-h limit of 200$\mu g/m^3$ and an annual limit of 40 $\mu g/m^3$. The US-EPA only provides for an annual standard of 100$\mu g/m^3$. Healthy subjects experience reductions in pulmonary function and increased airway reactivity only at levels of NO_2 exposure much higher (>1500 mg/m^3) than those measured outdoors (CEOHA, 1996; WHO, 2000a).

Epidemiological studies have mainly focused on indoor exposures (Samet *et al*., 1987). From these, there is evidence of increased respiratory symptoms and illness with increased long-term average indoor NO_2 concentration (Neas *et al*., 1991). The increase in mortality due to particles was higher in cities where the long-term NO_2 concentrations were higher (Katsouyanni *et al*., 2001). This was interpreted as an indication that a greater proportion of particles originated from traffic in places with higher NO_2 levels. Animal studies have suggested that

effects on alveolar macrophage antimicrobial function can occur at NO_2 concentrations of 1000 μg/m3 or higher.

1.3.4.3 Sulphur dioxide (SO_2)

Sulphur dioxide (SO_2) together with particles was derived primarily from coal combustion the effects of SO_2 and particles were often considered together (CEOHA, 1996).

Since the 1970s, SO_2 concentrations in both Europe and the USA have declined as a result of changing fuel quality and fuel use. However, in large cities outside those areas (*e.g.* in China), where coal is still used for domestic cooking and heating, high concentrations of are still observed; SO_2 concentrations are expressed in ppb or μg/m3 (1 ppb = 2.704 $\mu g/m^3$ at 20°C).

WHO provides a guideline of 125 $\mu g/m^3$ for 24-h SO^2 exposure, 500$\mu g/m^3$ for 10 min and an annual average of 50 $\mu g/m^3$, independent of the presence of particles. The US-EPA gives a 3-h average standard of 1300 $\mu g/m^3$, a 24-h average of 365$\mu g/m^3$ and an annual standard of 80$\mu g/m^3$. The EU has limit values for 1 h of 350 $\mu g/m^3$ not to be exceeded by 2005; 125 $\mu g/m^3$ for 24 h and an annual average of 20 $\mu g/m^3$ for the protection of ecosystems with no margin of tolerance (CEC, 1999). Short-term epidemiological studies in the past were unable to distinguish between the effects of SO_2 and particles.

1.3.4.4 Carbon monoxide (CO)

Carbon monoxide is mainly produced by incomplete combustion of carbonaceous fuels such as gasoline and natural gas. Outdoors it is mainly emitted from vehicles. Its concentration is relatively high in traffic canyons and

may be very high in road tunnels, multi-storey car parks and other such microenvironments. Also CO concentrations inside vehicles may be higher than outdoors, while a range of indoor sources exist, such as ETS and gas appliances (WHO, 2000b). It has been shown that individual exposure to CO in non-smokers mainly happens during motor vehicle travel (CEOHA, 1996).

CO is routinely measured by monitoring networks and is usually expressed in mg/m^3 or ppm (1 ppm = 1.165 mg/m^3 at 20°C)

WHO air quality guidelines give a guideline of 100 mg/m^3 for 15-min exposure, 60 mg/m^3 for 30-min, 30 mg/m^3 for 1-h and 10 mg/m^3 for 8-h exposure. There is no long-term average guideline. The US-EPA has adopted a standard of 10 mg/m^3 as an 8-h and 40 mg/m^3 as a 1-h average (US-EPA, 1996). The WHO air quality guidelines are set to prevent levels of COHb (carboxyhaemoglobin) in the blood exceeding 2.5%.

The toxic effects of CO are largely attributed to its high affinity with haemoglobin and myoglobin. Its affinity to haemoglobin is 200–250 times that for oxygen. Approximately 80–90% of absorbed CO binds with haemoglobin to form carboxyhaemoglobin (COHb). High exposures to CO cause acute poisoning, but such exposures are not encountered in outdoor urban settings. Unlike other gaseous pollutants presented above, CO appears to have no toxic effect on the lung but its health effects are manifested through the interference with oxygen transport (CEOHA, 1996; WHO, 2000a). For continuous exposures to CO concentrations up to 200 ppm at sea level, the COHb% at equilibrium can be approximated as COHb % = CO ppm × 0.16.

A limited number of recent epidemiological studies have provided evidence on the association of CO exposure to cardiac arrhythmia (Peters *et al*., 2000),

hospital admissions for heart disease and mortality (Touloumi *et al*., 1996 and Samet *et al*., 2000).

1.3.5 Food and lead poisoning

Recent research findings on lead poisoning in Zamfara State have not found any literature so far, on lead contamination from grains or food grown in mining areas. However, other research from the literature carried out indicated analysis on lead in urban areas rather than in mining communities.

Lead is commonly present in food and is regulated as a contaminant. Over the past decades, lead concentrations have decreased significantly due to the phase-out of leaded petrol, other actions, and the concomitant significant decrease in lead air pollution. A general decrease in lead concentrations had occurred, whereas the contents of cadmium, nickel, mercury and selenium were stable or declined only slightly (Larsen *et al*., 2002).

In total diet surveys carried out in the U.S. between 1991 and 2005, 382 different product types were analysed for lead. The overall mean for the tested products was 0.003 mg/kg, varying between not detected and 0.033 mg/kg.

Absorption of lead from the gastrointestinal tract depends on host characteristics and on the physicochemical properties of the ingested material. Absorption of ingested soluble lead compounds appears to be higher in children than in adults. Absorption is lower in the presence of food (Alexander *et al*., 1974)

Lead is a metal that exists both in inorganic and organic forms. In the environment, inorganic lead predominates over organic lead and the former is also the only type found in food. Lead in the environment can easily

contaminate food through water or through atmospheric lead deposition on agricultural crops.

According to Makokha *et al*. (2008) environmental lead pollution and contamination in food around Lake Victoria, Kisumu, Kenya and the lead levels in the grain samples were below the maximum WHO limits, and are comparable to those observed in other studies. This might have been partly due to the fact that most of these grains are brought from rural areas, where the risk for lead pollution is low. Yang *et al*. (1994) reported that cereals in China had lead levels of 0.06 mg/g. In Denmark, the National Food Agency established lead levels of 0.03 mg/g in cereals (National Food Agency of Denmark, 1992; Andersen *et al*., 1996). Another study by Urieta *et al*. (1996), found mean lead levels of 0.02 mg/g in cereals from Spain. In the United Kingdom, Ysart, (1994) reported mean lead levels of 0.02 mg/g in cereal products. In Poland, lead level was found to be 0.07 mg/g in cereals. In Japan, Muramatsu *et al*. (1994), established lead concentration of 0.05 μg/g in wheat and rice.

1.3.6 Lead dietary intake

Food is the major source of exposure to lead. The primary techniques for analysing lead in food samples are based on atomic absorption spectrometry, atomic emission spectrometry and mass spectrometry after digestion of organic material with concentrated acids.

Lead occurs primarily in the inorganic form in the environment. Human exposure is mainly via food and water, with some via air, dust and soil. In average adult consumers, lead dietary exposure ranged from 0.36 to 1.24, up to 2.43 μg/kg body weight (b.w.) per day in high consumers in Europe. Exposure

of infants ranged from 0.21 to 0.94 μg/kg b.w. per day and in children from 0.80 to 3.10 (average consumers), up to 5.51 (high consumers) μg/kg b.w. per day. Cereal products contribute most to dietary lead exposure, while dust and soil can be important non-dietary sources in children (EFSA, 2010).

The primary techniques for analyzing lead in food samples are based on atomic absorption spectrometry (AAS), atomic emission spectrometry (AES) and mass spectrometry (MS). When very high concentrations occur, X-ray fluorescence spectroscopy (XRFS) is also applicable.

Flame AAS (FAAS) and graphite furnace AAS (GFAAS) are the two atomic absorption spectrometry techniques used for measuring trace elements in foodstuffs. Empirical limits of detection for lead in analysis solution are in the range of 0.5 and 0.1 μg/L for FAAS and GFAAS, respectively (EFSA, 2010).

Control measures have been taken to regulate lead in paint, petrol, food cans and pipes in Europe since the 1970s. Human exposure to lead can occur via food, water, air, soil and dust.

Lead levels in ambient air range from 7.6×10^5 μg/m^3 in remote areas such as Antarctica, to >10 μg/m^3 near stationary sources such as smelters (ATSDR, 2007).

Long-term measurements of background lead concentrations have demonstrated significant reduction in lead levels in the environment after the phase-out of leaded petrol in many countries (EMEP, 2006). According to EMEP data (which should be considered as background values for Europe), lead concentrations in air, averaged over a number of EMEP monitoring stations during the period 1990-2003, decreased from about 0.020 to about 0.005 μg/m^3 (EMEP, 2001).

It has been estimated that an average child may ingest up to 100 mg of soil/day WHO, (2007). If the soil contains 100 mg/kg of lead (conservative upper bound), an average child may be exposed to as much as 10 μg of lead/day from this source alone, and, at the median value in Europe of 23 mg/kg, the exposure would be up to 2.3 μg of lead per day.

1.4 Statement of Research Problem

In June 2010, the world's attention was drawn to the monumental death toll in some communities in Zamfara State, Nigeria, as a result of lead (Pb) poisoning, with at least 400 victims (mostly children) dead from illegal gold mining using rudimentary tools. In addition to health and environmental challenges, there is the likely tendency that at least 5, 000 children were likely to suffer from brain damage (MSF, 2012).

The processing of the lead-rich ore resulted in direct contamination of soil, underground and surface water; with a consequent toxicity to human and animals, as a result of exposure to lead through dust inhalation and ingestion. The results of health and environmental assessment carried out in selected affected communities in Anka and Bukkuyum Local Government Areas indicated that the lead poisoning to human and contamination to environment was above tolerable limits of the World Health Organization (WHO), Food and Agricultural Organization, (FAO) and US-EPA (Joint UNEP/OCHA Environment unit, 2010).

1.5 Justification of the Study

In 2010, high number of deaths toll, particularly among children below the age of 5 years in the mining communities of Bukkuyum and Anka Local Government Areas of Zamfara State, Nigeria, was reported by Medicines Sans Frontiers' (MSF) to the state health authorities. Further investigation revealed that it was as a result of acute lead poisoning from lead pollution from the illegal gold mining of lead-rich ore. As part of the emergency measures taken to bring the situation under control, was soil remediation of the affected mining communities. Following the soil remediation in addition to other measures that had taken place, there was the need to determine the lead levels in soil, water, grains, human and animal blood samples to establish whether lead levels were still significant.

1.6 Aim and Objectives of the Study

1.6.1 Aim of the study

To determine the lead levels in selected mining communities of Zamfara State, Nigeria as a result of reported lead pollution and poisoning.

1.6.2 Objectives of the study

a. To determine the geographical position of the sampling point within the selected mining communities using geographical positioning system (GPS).

b. To collect samples of residential compound soil, underground water, surface water, human and livestock blood samples, and common grains grown in selected mining communities for analysis.

c. To determine the lead levels in the collected samples.

d. To compare the lead levels of residential compound soil, underground water, surface water, human and livestock blood samples, and common grains grown

in selected mining communities with those of internationally accepted threshold level of concern.

e. To determine whether the demographic distribution of socio-economic opportunities has any influence on the status of the selected mining communities.

1.6.3 Scope of the study

The scope of this research was limited to the ten (10) selected mining communities of Zamfara State, Nigeria, namely: Bagega, Kadauri, Kawaye, Kwali, Magami, Sunke, Tsunami, Tungar-Guru, Tungar-Kudaku and Yarglma. And the samples to work on have direct bearing on the life of the inhabitants, which include: soil, underground and surface waters, common grains grown in the area, human and animals' blood samples.

1.7 Research Hypothesis

Soil remediation had taken place in gold-mining communities of Zamfara State, Nigeria, due to outbreak of lead pollution, in which lead poisoning had been reported, with the remediation, lead levels is supposedly not significant (null hypothesis).

Although remediation had taken place in the gold-mining communities of Zamfara State, Nigeria, lead levels is still significant (alternate hypothesis).

CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Heavy Metals and their Health Effects

Heavy metals are commonly defined as those having a specific density of more than 5 g/cm^3. The main threats to human health from heavy metals are associated with exposure to lead, cadmium, mercury and arsenic (arsenic is a metalloid, but is usually classified as a heavy metal).

Heavy metals have been used in many different areas for thousands of years. Lead has been used for at least 5000 years, early applications including building materials, pigments for glazing ceramics, and pipes for transporting water. In ancient Rome, lead acetate was used to sweeten old wine, and some Romans might have consumed as much as a gram of lead a day. Mercury was allegedly used by the Romans as a salve to alleviate teething pain in infants, and was later (from the 1300s to the late 1800s) employed as a remedy for syphilis (Järup, 2003).

Although adverse health effects of heavy metals have been known for a long time, exposure to heavy metals continues and is even increasing in some areas. For example, mercury is still used in gold mining in many parts of Latin America. Arsenic is still common in wood preservatives, and tetraethyl lead remains a common additive to petrol, although this use has decreased dramatically in the developed countries (Järup, 2003). At the end of the 20th century, however, emissions of heavy metals started to decrease in developed countries: in the UK, emissions of heavy metals fell by over 50% between 1990 and 2000 (DETR, 2001).

Lead emissions are mainly related to road transport and thus most uniformly distributed over space. Cadmium emissions are primarily associated with non-ferrous metallurgy

and fuel combustion, whereas the spatial distribution of anthropogenic mercury emissions reflects mainly the level of coal consumption in different regions. People may be exposed to potentially harmful chemical, physical and biological agents in air, food, water or soil. However, exposure does not result only from the presence of a harmful agent in the environment. The main concern in any exposure is contact with the harmful agent (Berglund *et al*., 2001).

2.2 Lead

During the last century, lead emissions to ambient air have further polluted our environment, over 50% of lead emissions originating from petrol. Over the last few decades, however, lead emissions in developed countries have decreased markedly due to the introduction of unleaded petrol. Subsequently blood lead levels in the general population have decreased (Järup, 2003).

Occupational exposure to inorganic lead occurs in mines and smelters as well as welding of lead painted metal, and in battery plants. Low or moderate exposure may take place in the glass industry. High levels of air emissions may pollute areas near lead mines and smelters. Airborne lead can be deposited on soil and water, thus reaching humans *via* the food chain.

Up to 50% of inhaled inorganic lead may be absorbed in the lungs. Adults take up 10–15% of lead in food, whereas children may absorb up to 50% *via* the gastrointestinal tract. Lead in blood is bound to erythrocytes, and elimination is slow and principally *via* urine. Lead is accumulated in the skeleton, and is only slowly released from this body compartment. Half-life of lead in blood is about 1 month and in the skeleton 20–30 years (WHO, 1995).

In adults, inorganic lead does not penetrate the blood–brain barrier, whereas this barrier is less developed in children. The high gastrointestinal uptake and the permeable blood–brain barrier make children especially susceptible to lead exposure and subsequent brain damage. Organic lead compounds penetrate body and cell membranes. Tetramethyl lead and tetraethyl lead penetrate the skin easily. These compounds may also cross the blood–brain barrier in adults, and thus adults may suffer from lead encephalopathy related to acute poisoning by organic lead compounds (Järup, 2003).

2.2.1 Health effects of lead

The symptoms of acute lead poisoning are headache, irritability, abdominal pain and various symptoms related to the nervous system. Lead encephalopathy is characterized by sleeplessness and restlessness. Children may be affected by behavioural disturbances, learning and concentration difficulties. In severe cases of lead encephalopathy, the affected person may suffer from acute psychosis, confusion and reduced consciousness. People who have been exposed to lead for a long time may suffer from memory deterioration, prolonged reaction time and reduced ability to understand. Individuals with average blood lead levels under 3μmol/l may show signs of peripheral nerve symptoms with reduced nerve conduction velocity and reduced dermal sensibility. If the neuropathy is severe the lesion may be permanent. The classical picture includes a dark blue lead sulphide line at the gingival margin. In less serious cases, the most obvious sign of lead poisoning is disturbance of haemoglobin synthesis, and long-term lead exposure may lead to anaemia (Järup, 2003).

Recent research has shown that long-term low-level lead exposure in children may also lead to diminished intellectual capacity. Efforts to define relationship between body

burden of lead and blood pressure or other effects on the cardiovascular system has not been demonstrated in humans (WHO, 1995).

Acute exposure to lead is known to cause proximal renal tubular damage. Long-term lead exposure may also give rise to kidney damage. Blood lead levels in children below 10μg/dl have so far been considered acceptable, but recent data indicate that there may be toxicological effects of lead at lower levels of exposure than previously anticipated. There is also evidence that certain genetic and environmental factors can increase the detrimental effects of lead on neural development, thereby rendering certain children more vulnerable to lead neurotoxicity (WHO, 1995; Lidsky and Schneider, 2003).

International Agency for Research on Cancer (IARC) classified lead as a 'possible human carcinogen' based on sufficient animal data and insufficient human data in 1987. Since then a few studies have been published, the overall evidence for lead as a carcinogen being only weak, the most likely candidates are lung cancer, stomach cancer and gliomas (Steenland and Boffetta, 2000).

2.2.2 Sources of exposure to lead

Lead is found at low levels in Earth's crust, mainly as lead sulfide (IARC, 2006). However, 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 (UNEP, 2008); 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 (IPCS, 1995). Because of lead's persistence and potential for global atmospheric transport, atmospheric emissions affect even the most remote regions of the world (WHO, 2007b).

Lead is used mainly in the production of lead-acid batteries, plumbing materials and alloys. Other uses are in cable sheathing, paints, glazes and ammunition. Human occupational exposure can also take place during the application and removal of protective lead-containing paints, during the grinding, welding and cutting of materials painted with lead-containing paints, such as in shipbuilding, construction, demolition industries, and fabrication of heavy lead glass and crystal, and in crystal carving. Mining, smelting, and informal processing and recycling of electric and electronic waste can also be significant sources of exposure. Lead has been used widely in the form of tetraethyl and tetramethyl lead as antiknock and lubricating agents in petrol, although the majority of lead is emitted from vehicles in the form of inorganic particles. This use has been phased out in most countries, which has resulted in a significant reduction of human exposure and mean blood lead levels (IPCS, 1995; Fewtrell *et al*., 2003).

2.2.2.1 Leaded gasoline

Leaded gasoline accounted for 80–90% of airborne lead pollution in large cities where it was used worldwide (Lovei, 1999). Countries that have phased lead out of gasoline have reported corresponding decreases in lead concentrations in air and human blood. The decreases in population BLLs have been dramatic. For example, mean BLLs in the United States, Ontario, Canada, and the United Kingdom were reduced by >70% as lead in gasoline was reduced (Meyer and Brown, 2008).

Similarly, in a study on BLL conducted by George Foundation in 1997 (when leaded gasoline was still used in Bombay), using similar blood–collection procedures, the study conducted before leaded gasoline was banned showed that 61.8% of children had BLLs ≥ 10 μg/dL compared with 33.2% after the phase-out (Nichani, 2006).

2.2.2.2 Smelters

Metal smelters are another potential source of contamination for air and soil and can contribute to lead poisoning. Children's BLLs and risk factors was conducted in Torreo´ n, Mexico, in March 2001 (Albalak *et al*., 2003). The mean BLL of children living there was 6.0 mg/dL and 20% had BLLs ≥ 10 mg/dL in 2001 (Meyer and Brown, 2008).

After smelter emissions stop, historic soil contamination can pose an ongoing threat. In June 2005, reports of symptomatic lead poisoning among refugee children in Kosovo reached the US CDC. The children most affected by the lead contamination in Mitrovica, Kosovo, were the Roma whose homes were destroyed during the war. Operations ceased in 1999 when routine testing of United Nations peacekeeping forces identified soldiers with elevated BLLs. This finding motivated testing of children living in the nearby refugee camps. All children had blood-lead levels >10 μg/dL, the level of concern for young children. The mean BLL was 47μg/dL in the camps as compared to 29μg/dL in the non-Roma children in the area (WHO, 2005).

2.2.2.3 Lead-battery recycling

Informal lead-smelting operations often operated at or near the home, can be a source of lead exposure for nearby residents. ‘‘Backyard’’ or ‘‘cottage’’ lead smelting has

been described in the scientific literature for decades (Koplan *et al*., 1977; Kawai *et al*., 1983; Matte *et al*., 1991). Backyard smelters is a cause of lead poisoning worldwide continue. These smelters are usually unregulated and the magnitude of the problem is not documented. A study estimated that about one third of spent lead-acid batteries in Jamaica were recycled by backyard smelting operations or by small battery repair shops scattered over the island (Matte *et al*., 1991). These can be especially dangerous to workers' families and neighbours because the work areas rarely have proper ventilation or control of worker exposure or lead release. These operations create lead air emissions that then settle and contaminate nearby soil, water, and food and can result in high levels of lead exposure for families (Meyer and Brown, 2008)

Melting lead to make fishing sinkers has also been reported as a source of lead exposure. In Cartagena, Colombia, the parents of all children with BLLs ≥ 10 mg/dL worked in and lived near small metal smelting factories with poor indoor air quality, or were fishers who made lead sinkers for their fishing nets. The highest BLLs were in children from a neighbourhood near a shoreline where many family incomes were derived from fishing (Olivero-Verbel *et al*., 2007).

2.2.2.4 Lead-based Paints

Lead was commonly used in residential paint before 1950; in the 1960s the paint industry voluntarily began to limit lead in residential paint. Although regulations limiting lead content in paint for residential use were enacted in the United States in 1978, leaded paint remains in many old homes. A wide distribution of housing built before 1950 exists across the United States and poses a potential health threat (CDC, 2001)

Children aged 1–5 years who were tested for lead poisoning (Mayan *et al.*, 2001). BLLs ranged from 4.7 mg/dL to 42.5 mg/dL with a mean of 13.9 mg/dL; 85.8% of children had BLLs ≥ 10 mg/dL. Qualitative lead-paint tests of the walls of the homes were positive for 91% of children with BLLs ≥ 20 mg/dL and 14% for children with BLLs <10 mg/dL. In India, studies conducted in Mangalore and Karnataka found lead-based paint in the homes of 3 of the 10 children with BLLs of at least 40 mg/dL. These 10 children were part of a blood-lead study of 107 school children who were randomly selected (Kuruvilla *et al*., 2004).

2.2.2.5 Electronics

Most electronic devices contain lead and may represent another source of lead when discarded (Brown, 2004). A study sponsored by the US Environmental Protection Agency (US EPA) reported that electronic items in landfills leached lead at levels exceeding the threshold for hazardous waste. The European Union recognized this potential threat to human health and the environment and acted by banning certain electronic items from landfills. However, the US EPAs most recent estimate is that more than two million tons of electronic waste is dumped in US landfills each year. EPA estimates that only about 10% of all obsolete consumer electronics are recycled. The rest are stored somewhere, passed on to second users, or simply tossed in the trash. US recyclers and watchdog groups estimate that 50% of the used US computers, cell phones, and TVs that are sent to recyclers are shipped overseas for recycling in facilities in Taizhou, China, or Lagos, Nigeria, as permitted by federal law. Much of this obsolete equipment ends up as toxic waste with hazardous components exposed, burned, or allowed to degrade in landfills. Waste by-products produced during informal recycling are heavily contaminated with lead (Meyer and Brown, 2008).

2.2.3 Global dimensions of lead exposure

There is a long history of public exposure to lead in food and drink. Lead poisoning was common in Roman times because of the use of lead in water pipes and earth ware containers, and in wine storage. Lead poisoning associated with occupational exposure was first reported in 370 BC (Kazantzis, 1989). It became common among industrial workers in the 19th and early 20th centuries, when workers were exposed to lead in smelting, painting, plumbing, printing and many other industrial activities.

Exposure to lead occurs mainly through inhalation of dust and air and ingestion of foodstuffs, water and dust. Inhalation is an important route of exposure for people in the vicinity of point sources, including open burning of wastes containing lead products, in countries that still use lead in petrol, and in some occupational settings including secondary lead recovery. Ingestion of lead in dust and soil is a major exposure pathway in children, because of their biological and behavioural characteristics. In general, ingestion of lead through food and water is the major exposure path way for lead in adults.

Lead exposure is estimated to account for 0.6% of the global burden of disease, with the highest burden in developing regions (WHO, 2007c). Recent reductions in the use of lead in petrol (gasoline), paint, plumbing and solder have resulted in substantial reductions in lead levels in the blood (Fewtrell *et al.*; 2003). However, significant sources of exposure to lead still remain, particularly in developing countries.

Exposure to and uptake of this non-essential element have consequently increased, both occupational and environmental exposures to lead remain a serious problem in many developing and industrializing countries, as well as in some developed countries.

In Australia, lead poisoning in children was first reported in 1892, although it was not until 12 years later that the source, peeling lead-based paint, was identified in a series of ten children with lead colic (Gibson, 1904). In 1943 a follow-up study of 20 school children in the USA who had experienced acute lead poisoning in infancy or early childhood found that exposure to environmental lead at levels insufficient to produce clinical encephalopathy was associated with long-term deficits in neuropsychological development (Byers, 1943).

2.2.4 Lead contamination before and after industrial revolution

Exposure of human populations to environmental lead was relatively low before the industrial revolution but has increased with industrialization and large-scale mining. Lead contamination of the environment is high relative to that of other nonessential elements (Flegal and Smith, 1986). Globally, the extensive processing of lead ores is estimated to have released about 300 million tonnes of lead into the environment over the past five millennia, mostly within the past 500 years. (Flegal and Smith, 1986).

Following the advent of motor vehicles at the beginning of the 20th century, there was a substantial increase in environmental lead contamination because of the use of lead in petrol (Smith, 1984; CDC, 1991). This resulted in an increase in community exposure to environmental lead throughout much of the century.

World lead consumption rose steadily between 1965 and 1990, when it reached about 5.6 million tons. Between 1980 and 1990 the consumption of lead in developed countries increased only slightly, whereas between 1979 and 1990 in developing countries it increased from 315, 000 tonnes to 844, 000 tonnes per annum. Global lead contamination, attributable to the greatly increased circulation of lead in soil, water and air as a result of human activities, remains significant (Nriagu and Pacnya, 1988).

Lead levels in human skeletal remains, which indicated that the body lead burden of today's populations is 500–1000 times greater than that of their pre-industrial counterparts (Ericson *et al*., 1979; Patterson *et al*., 1991).

Elevated lead levels continue to be a particular problem among socially and economically deprived children. Poor people are more likely to live in substandard housing and be near industry and heavy traffic, to be exposed to lead dust brought home by lead workers, and to be nutritionally deprived and therefore susceptible (Tong *et al*., 2000).

2.2.5 Occupational lead exposure

Workers are exposed to lead in many occupations, including motor vehicle assembly, panel beating, battery manufacture and recovery, soldering, lead mining and smelting, lead alloy production, and in the glass, plastics, printing, ceramics and paint industries. In most highly industrialized countries, stricter controls and improvements in industrial methods have helped to ensure that occupational lead poisoning is less prevalent than formerly. In developing countries, however, it remains a problem of potentially huge dimensions (Von Schirnding, 1999).

Workers are also at particular risk in battery manufacture, demolition work, welding, pottery and ceramic ware production (often a home-based occupation involving women and children), small businesses repairing automobile radiators, and the production of jewellery and decorative items by artisans. The last-mentioned category is of particular concern since the work is predominantly carried out at home or in unregulated workshops, often by women and children. Although adults are mainly involved, in many countries, especially those with developing industries, and in small home-based industries, there is little distinction between home and the workplace, and children are

consequently exposed to lead. Because of the transfer of lead to the foetus in uterus and the introduction of lead into people's homes on clothing, where young children thus become exposed, problems of occupational exposure become community problems (Von Schirnding, 1999).

2.2.6 Environmental lead exposure

In most developed countries, concerted efforts have led to a reduction in the introduction of lead into the ambient environment in recent years, reflecting a decline in the commercial use of lead, particularly in petrol (CDC, 1991; Edwards-Bert *et al*., 1994). Blood lead levels in the general population in these countries have fallen dramatically over the past 20 years, thanks to the phasing out of lead from petrol and the reduction of environmental exposure to the metal (Robinowitz and Needleman, 1982; Annest, 1983; Edwards-Bert *et al*., 1994).

In the USA between 1976 and 1991 the mean blood lead level of persons aged 1–74 years dropped by 78%, from 12.8 mg/dl to 2.8μg/dl (Pirkle *et al*., 1994). Mean blood lead levels of children aged 1–5 years declined by 77% (from 13.7 μg/dl to 3.2 μg/dl) for non- Hispanic white children and by 72% (from 20.2 mg/dl to 5.6 μg/dl) for non-Hispanic black children. The prevalence of blood lead levels of ≥10 μg/dl for children aged 1–5 years declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children. Similar declines were found in population subgroups defined by age, sex, race/ethnicity, income level and urban status. The major cause of the observed decline in blood lead levels was the removal of lead from petrol.

Between 1978 and 1988, marked decreases in the average blood lead levels of adults were noted in many countries, including Belgium, Federal Republic of Germany, New Zealand, Sweden, and the United Kingdom (Von Schirnding, 1999). Recently, lead

exposure was examined among the population of Barcelona and the changes that had occurred during the previous 10 years were evaluated (Torra *et al*., 1997).

Lead continues to be a significant public health problem in developing countries (Tong *et al*., 1998)., where there are considerable variations in the sources and pathways of exposure. For example, in many Latin American countries, leaded paint is not a significant source of recurrent exposure, whereas lead-glazed ceramics are such a source (Rojas-Lopez *et al*., 1994). Exposure attributable to miscellaneous sources may be even more significant than universal exposure associated with leaded petrol, especially for people living in poverty. Exposure to lead from lead mining, smelting, battery factories and cottage industries is a significant environmental hazard in developing countries.

In Jamaica a survey was conducted to determine the distribution and determinants of environmental and blood lead levels in populations living near conventional and cottage lead smelters (Matte *et al*., 1991). Geometric mean blood lead levels in exposed groups were nearly twice as high as those in unexposed groups; 44% of exposed under-6-year olds had blood lead levels ≥ 25 μg/dl. In Berat and Tirana, Albania, the mean observed blood lead levels in 84 preschool children living less than 2 km from a battery plant was 43.4 μg/dl, significantly higher than the value of 15.0 μg/dl in 45 preschool children living more than 2 km from the plant; 98% of preschool children and 82% of schoolchildren had blood lead levels >10 μg/dl (Tabaku *et al*., 1998).

In China, childhood lead poisoning may be widespread as a result of rapid industrialization and the use of leaded petrol (Shen *et al*., 1996). Children residing in industrial areas and in areas with heavy traffic had average blood lead levels of 21.8–67.9 μg/dl. The proportion of blood lead levels > 10 μg/dl ranged from 64.9% to 99.5%. Even about 50% of children living in non-industrialized areas had blood lead

values >10 μg/dl (Shen *et al*., 1996). There is also evidence of an increase in blood lead levels among non-smoking women between 1983 and 1998, associated with a rapid increase in the number of motor vehicles. The problem of lead exposure in children is particularly significant in small towns with numerous small factories (Zheng, 1999).

The mean blood lead concentration among 93 randomly selected rickshaw pullers in India was 53μg/dl (Viswanathan *et al*., 1991). Also in India, direct testing for blood lead was carried out randomly on 2031 children and adults in five cities with high population densities where leaded petrol had contributed to environmental lead levels. Approximately 51% had levels >10 μg/dl, and 13% had values >20 μg/dl. The proportion of children with levels ≥ 10μg/dl ranged from 40% in Bangalore to 62% in Mumbai (George Foundation, 1999).

A random sample of 200 children under the age 5 years living in an area of Mexico City (Romieu *et al*., 1995), and samples of floor, window and street dust, paint, soil, water and glazed ceramics were obtained from the participants' households, as well as blood samples and dirt from their hands. Blood lead levels ranged from 1 μg/dl to 31μg/dl, the mean being 9.9μg/dl. Among children aged ≥18 months, 44% had blood lead levels exceeding 10μg/dl. Except for glazed ceramic, the lead content of environmental samples was low. The major predictors of blood lead levels were the lead content of the glazed ceramics used in the preparation of children's food, exposure to airborne lead from vehicle emissions, and the lead content of dirt from the children's hands.

African children may be particularly predisposed to environmental lead exposure because of their lifestyle and socio-ecological factors. However, the true picture of childhood lead poisoning in Africa remains unclear. Petrol sold in most African countries contains 0.5–0.8 g/l lead, which may be among the highest levels in the world

(Nriagu *et al*., 1996). In urban and rural areas and near mining centres, average atmospheric lead concentrations reach 0.5–3.0 mg/m^3 and exceed 1000 mg/g in dust and soils. In addition to automotive and industrial sources, the following are hazards in individual households: cottage industries and the burning of paper products, discarded rubber, battery casings, and painted wood for cooking and heating. Lead paint, lead solders and lead cosmetics are unregulated in some countries.

In Cape Province, South Africa, over 90% of the children in some urban and rural communities have blood lead levels ≥ 10μg/dl. The mean blood lead level in inner-city, first-grade schoolchildren in the country was 18μg/dl; 13% of mixed race children, but not white children, had blood lead levels ≥ 25 μg/dl. Over 90% of children had blood lead levels >10 mg/dl (Von Schirnding *et al*., 1991). The levels of lead in blood, air and dust samples taken from schools close to roads carrying heavy traffic were higher than in those from schools further away (Von Schirnding *et al*., 1991; Von Schirnding and Fuggle, 1996). Raised blood lead values were also associated with dusty houses, houses in a poor state of repair, overcrowding, low parental educational and income levels, and other factors related to family structure and socioeconomic status (Von Schirnding *et al*., 1991).

The level of exposure to lead is, however, falling in some developing countries because of the reduced use of lead in petrol and elsewhere. In Thailand, for example, leaded petrol was phased out during the period 1984–96, and was associated with a marked decline in atmospheric lead levels in Bangkok (Boontherawara *et al*., 1994). A recent survey of 1000 children aged 6–72 months in Chiang Mai, Thailand revealed that their average blood lead level was 4.2 μg/dl; only 4.6% of the children had blood lead levels ≥10 μg/dl (Prapamontol *et al*., 1996).

In Bangkok, where air pollution levels were higher than in Chiang Mai, 5.2% and 2.4% of 500 pregnant women and their newborn babies, respectively, had blood lead levels >10 mg/dl. These values compared favourably with those determined in a previous study in Bangkok before the introduction of unleaded petrol (Phupradit *et al*., 1994).

In Nigeria, in addition to developing countries challenges in lead levels from lead exposure sources of leaded fuel, smelting, iron and still industries, lead paints, and water pipes, a greater challenge on lead poisoning and blood lead levels to epidemic level was discovered in Zamfara State, North-western Nigeria in 2010 (Joint UNEP/OCHA, 2010; TerraGraphics, 2011).

The blood lead levels among children and adults in the affected mining communities was at critical level, in some cases, BLL > 100 μg/dL. Mean Lead concentration for soil, drinking water, surface water and common grains exceeded the acceptable limits of US-EPA, WHO and FAO that resulted in so many deaths and neurological challenges (Lowry, 2010). Zamfara State mining communities are at the moment in the list of epidemic lead poisoning in modern history. Since 2011, efforts has been on to get the epidemic under control and re-direct the mining activity towards safer mining practices in order to save the future teaming population from permanent incapacitation, as contained in the report of Kevin Telmer, (2011), on Emergency assessment and recommendations.

Exposure to environmental lead is clearly a major public health hazard of global dimensions. As measures to control the transfer of lead to the environment are implemented in most developed countries through, for example, the phasing out of lead in fuel, paints and other consumer products, and tighter control of industrial emissions, environmental exposure to lead can, in general, be expected to continue to decline. However, because of rapid industrialization and the persistence of lead in the

environment, exposure is likely to remain a significant public health problem in most developing countries for many years. Much work needs to be done to identify and treat children with elevated blood lead levels and reduce lead exposure in the community (Tong *et al*., 2000).

2.2.7 Toxic effects of lead in children and adults

Lead affects at least three major organ systems: (1) the central and peripheral nervous systems; (2) the heme biosynthetic pathway; and (3) the renal system. Clinical manifestations differ somewhat between children and adults. In the child, the most serious symptoms are found in the central nervous system with subtle effects (e.g., decreased IQ and cognitive effects) occurring at lower levels and severe effects (e.g., seizures, encephalopathy) occurring at higher levels. Chelation therapy has reduced the mortality rate and morbidity substantially at higher levels. However, chelation therapy at lower levels (< 45μg/dL), it has not been shown to be as effective as removal of the lead source from the child's environment. Children are much more sensitive than adults to the neurocognitive and behavioural effects of lead, probably primarily for two reasons: (1) children absorb 40 to 50% of dietary lead whereas adults absorb about 10%; and (2) the nervous system develops rapidly in the young child. The blood lead threshold (if there is one) for neurocognitive and behavioural effects is probably lower in children than in adults (Bellinger *et al*., 1992; Lanphear *et al*., 2005)

In the child, lead appears to have an effect on renal function even at levels below10 μg/dL (Longhman-Adham, 1998), this especially true if the lead exposure occurs over a sustained period of time. Subtle abnormalities in renal tubular function, associated with aminoaciduria, glycosuria, and increased excretion of low-molecular weight proteins can occur. Lead has been clearly demonstrated to produce tubular nephrotoxicity and

chronic interstitial nephritis in humans and rodents after chronic exposure. In addition, lead in the kidney interferes with activation of vitamin D 1,2-dihydroxy cholecalciferol, a p450-dependent process (NRC, 1993).

Lead interferes in the formation of active vitamin D, which has an important role in its influence on calcium metabolism. The active form of Vitamin D is produced, primarily, from activation of Vitamin D by sunlight on the skin. The circulating hormone binds to Vitamin D Receptors (VDRs) in the nucleus of cells in the gastrointestinal tract, kidney and bone. Because of their similar biochemical nature, lead can be absorbed by this mechanism especially in children who have decreased calcium intake.

It is known that lead interferes with the utilization of iron for the formation of heme. This probably occurs in every cell, although it is best studied in the blood-forming organs. In chronic, moderately severe lead poisoning, anaemia is commonly found (Anderson *et al*., 1996). A decrease in haemoglobin is reported to occur in iron-sufficient children when blood lead concentration exceeds 60μg/dL.

Anaemia in lead poisoning results from impairment of haemoglobin production and changes in the red blood cell membrane. Lead's interference in heme biosynthesis is characterized by several unique enzyme blockades causing increased urinary delta-aminolevulinic acid (ALA), urinary coproporphyrin, and erythrocyte zinc protoporphyrin. While anemia may not be seen until blood lead concentrations are markedly elevated, the effect on hemoglobin synthesis occurs at lower levels. ALA dehyratase is inhibited at levels of 15 μg/dL children. At levels of 30 μg/dL, elevation in erthyrocyte protoporphyrin may be seen. Finally, at levels of 40, reduced hemoglobin synthesis may be found. The basophilic stippling of red cells is due to the presence of aggregated ribosomes, which may also include mitochondrial fragments.

Conditions, such as lead poisoning, can result in altered ribosomes to have a higher propensity to aggregate (Bull, 2006).

The central nervous system can be affected by lead in children. Over the past several decades, epidemiologic studies have demonstrated that chronic, low-level lead poisoning may lead to CNS injury in young children (Weitzman *et al*., 2005). Earlier studies suggested that altered electrophysiologic responses and adverse effects on IQ occurred at blood lead concentrations of 30μg/dL or higher. However, more recent data suggests environmental lead exposure in children at blood lead concentrations < 7.5 μg/dL is associated with cognitive deficits (Lanphear *et al*., 2005). In fact, studies suggest that a permanent pattern of cognitive dysfunction may result from lead poisoning in the first several years of life (White *et al*., 1993). It should be noted that the variability in blood lead testing allows for statistical significance to be seen at levels, only, above 5μg/dL. Consequently, it seems appropriate that blood lead test reports now inform providers that results in the range 5-9μg/dL are associated with adverse health effects in young children aged 6 years and younger. Acute lead poisoning may produce encephalopathy in children. Ataxia, altered state of consciousness, and seizures has been reported in children with blood lead concentrations over 80μg/dL.

2.2.8 Reproductive and developmental effects

The reproductive toxicity which results from high dose lead exposure was well known in the last century. This has only changed in the last 30 years, with the return of women to the work force. The obvious effects of lead in the 19th century were stillbirth and spontaneous abortion, which was usually recognized in women with occupational exposure to lead and other clinical manifestations of lead poisoning (Paul, 1860).

With the advent of human chorionic gonadotropin measurement procedures, it is now possible to detect the onset of pregnancy and early fatal loss as early as the first one to two weeks of pregnancy (NRC, 1993). Sexual dysfunction in the male has not been as closely studied. More recently, it has been found in workers employed for more than three years that serum testosterone and free-testosterone indices are decreased, at mean blood lead concentrations in excess of 60μg/dL (NRC, 1993).

Prospective studies on the adverse effects of low-level increase in lead absorption have revealed that there is no association between blood lead concentration at birth and neurobehavioral effects beyond 24 months of age. However, these and other studies suggest that the effects on learning behaviour are associated with the degree of lead exposure occurring between 12 and 36 months of age.

The consensus is that lead has an adverse effect on neurodevelopment and cognition. For an increase of 10μg/dL (0.48 μmol/L), during the preschool years, an average IQ loss of 2.6 points is predicted (NRC, 1993).

Furthermore, in the few studies that have had the chance to study children with blood leads below 10μg/dL (0.48μmol/L), some adverse effects on neurodevelopment have been found. Indeed, there may be no blood lead threshold for subtle adverse effects on neurodevelopment.

2.2.9 Treatment of lead poisoning

The clinical signs and symptoms of lead poisoning are nonspecific; therefore, a lead measurement, preferably a venous blood lead measurement, is essential for diagnosis. Ancillary tests such as those involving heme precursors (urinary delta-aminolevulinic acid, coproporphyrin, and erythrocyte protoporphyrin) may be helpful in making a diagnosis, but by themselves are inadequate for definitive diagnosis. In the majority of

cases, children with lead poisoning are asymptomatic resulting in a delay in the appropriate diagnosis. However, during this time effects on a cellular level are occurring resulting in subtle changes in the child. These include impairment of Intelligence Quotient (IQ) and other cognitive effects, decreased heme synthesis, and interference in vitamin D metabolism (Lowry, 2010).

The total body burden of lead may be divided into four compartments. The residence times of lead in these four compartments are estimated at about: 35 days in blood; 40 days in soft tissues; 3 to 4 years in trabecular bone; and 16 to 20 years in cortical bone. The disappearance time is largely dependent upon the degree of overall excess exposure. The greater the body lead burden the slower the rate of disappearance from the tissues, including blood (Rabinowitz *et al*., 1976; US-EPA, 2010). Injury from lead (for kidneys and CNS) may remain long after blood lead levels have decreased due to distribution and elimination. At present, there is no established way to make a retrospective diagnosis of lead toxicity in a child on the basis of current blood lead alone.

Lead is distributed throughout the body with the major fraction being absorbed in the bone (95% in the adult and about 70 to 75% in young growing children) (Ziegler *et al*., 1978). The rate of turnover of lead in bone is higher in children than in adults. The two nonosseous organs with the highest lead contents are the liver and the kidney, the organs of excretion of lead. In general, the concentration of lead in other organs is comparable to that found in blood. Approximately 99% of the lead in blood is bound to red blood cells. The remaining 1%, i.e., plasma lead, serves as an intermediate in transporting lead from the erythrocytes to other body compartments.

2.2.9.1 Chelation therapy

Once lead has entered the body, especially bone, it is very difficult to remove. Accordingly, prevention is the mainstay of treatment. However, chelation therapy may be used to decrease the blood lead concentrations acutely. The final component of treatment is chelation therapy. Chelating agents bind metals at two or more sites. Ideally, the chelated metal would be excreted; however, the lead: chelate complex may persist in tissues where the binding occurred or be redistributed to other tissues. An optimal chelating drug should increase lead excretion, be administered easily, and be affordable and safe. Lead removal should halt further toxicity and reverse previous effects (Makrowitz, 2000).

Several chelating agents are effective in lead excretion, but the chelator of choice depends on the blood lead concentration, the patient's symptoms and the environmental lead burden. Symptomatic patients should be hospitalized and chelation therapy with Edentate Calcium Disodium ($CaNa_2EDTA$). $CaNa_2EDTA$ is an intravenous formulation that has been shown to be effective with British AntiLewisite (BAL, Dimercaprol) for removal of lead in patients with encephalopathy. Edentate calcium disodium, used alone, may aggravate symptoms in patients with very high blood lead levels (Chisolm, 1971).

Oral chelating agents are available for treatment of lead poisoned patients who have elevated blood lead concentrations and asymptomatic. In the Unites States, 2, 3 Dimercaptosuccinic Acid (DMSA, Succimer) is the drug most commonly used. Other oral agents that may be used are DMPS (Unithiol) and penicillamine.

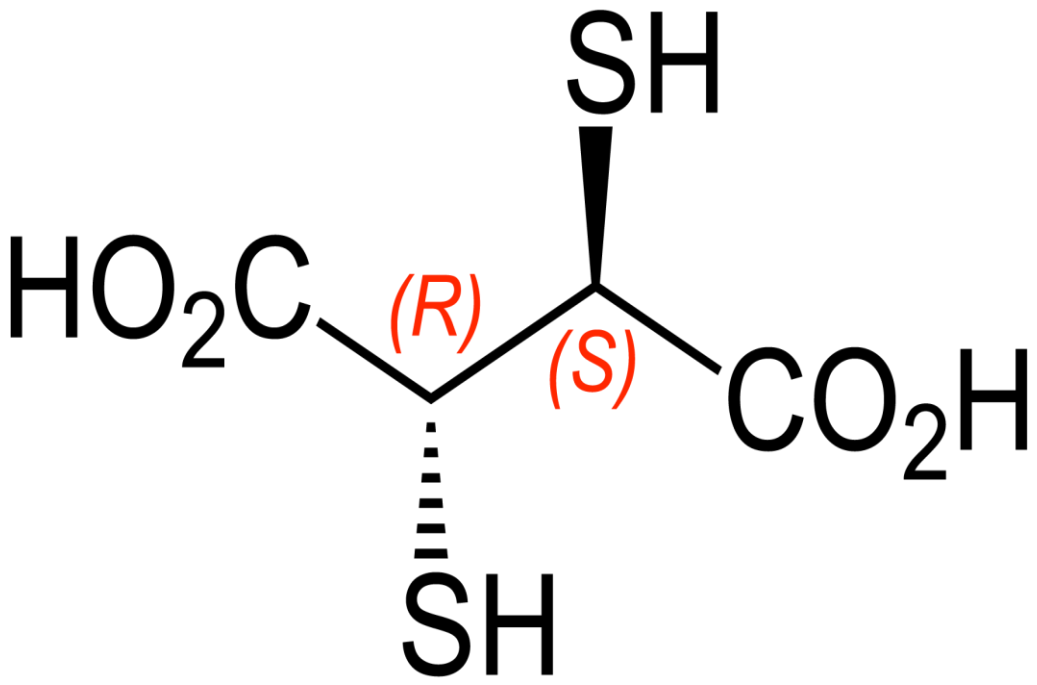


Figure 2.1: 2,3-Dimercaptosuccinic Acid (DMSA, Succimer)

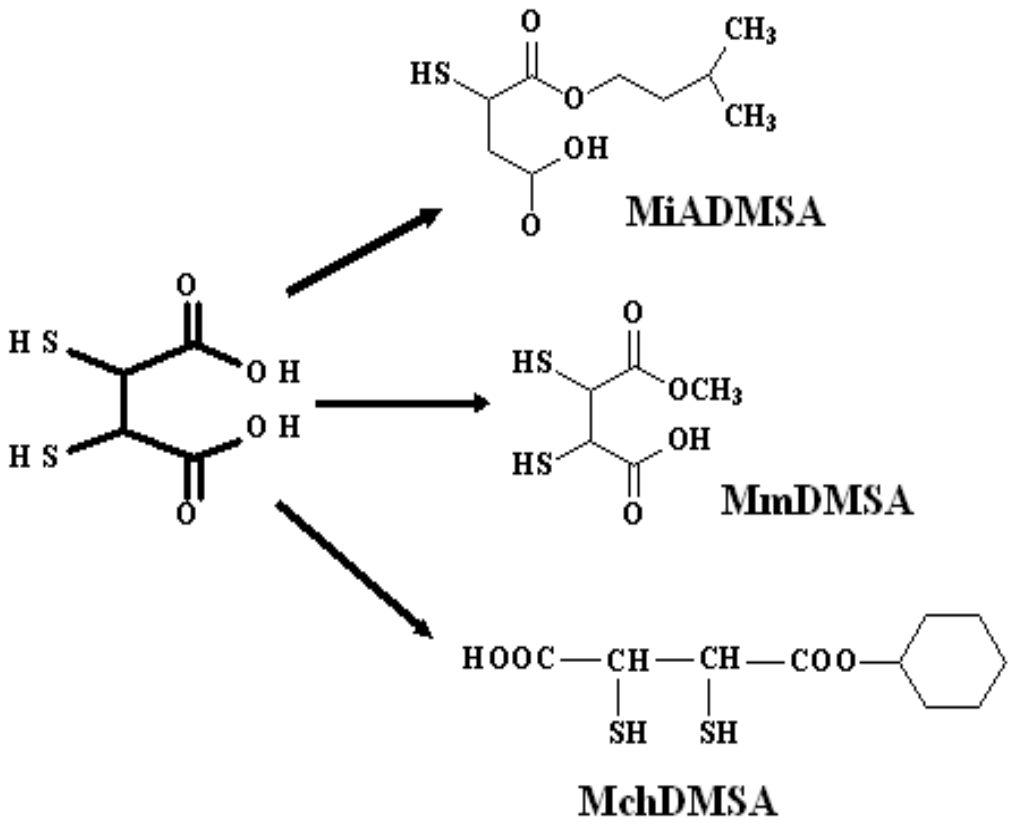


Figure 2.2: Newly synthesized monoesters of DMSA

Succimer is an orally chelating agent that is commonly used for the treatment of blood lead concentrations above 45µg/dL in the United States. It is a water soluble analog of dimercaprol. However, it has a wider therapeutic index and has advantages over dimercaprol and $CaNa_2EDTA$.

The majority of the elimination occurs within 24 hours and as DMSA-cysteine disulfide conjugates.[31] Renal clearance is greater in healthy adults than in children or adults with lead poisoning (Dart *et al*., 1994). The majority of the elimination occurs within 24 hours and as DMSA-cysteine disulfide conjugates (Maiorino *et al*., 1989). Renal clearance is greater in healthy adults than in children or adults with lead poisoning (Dart *et al*., 1994).

While few studies have been performed to determine appropriate dosing in humans, only one paediatric study is available (Graziano *et al*., 1988). Oral DMSA at 30 mg/kg/day (1050 mg/m^2/day) was used and based on previous adult studies (Graziano *et al*., 1988). This dose in children produced significantly ($p<0.0001$) greater lead excretion than 10 mg/kg/day (350 mg/m^2/day) or 20 mg/kg/day (700 mg/m^2/day). The current recommended dose for DMSA in the United States for children is 30 mg/kg/day for 5 days followed by a 14-day course of 20 mg/kg/day to prevent or blunt the rebound of the blood lead concentration. However, the duration of dosing has been controversial.

Use of DMSA for chelation treatment has resulted in few adverse effects. A number of studies have assessed the impact of DMSA on other metals (Graziano *et al*., 1988; Chisholm, 2000). The only essential metal that has consistently been found to be adversely affected by DMSA is zinc, which affects the metabolism of vitamin D. However, differences have been found between children and adults.

DMSA (meso-2,-3-dimercaptosuccinic acid) is a sulfhydryl-containing, water-soluble, non-toxic, orally administered metal chelator[1] that has been in use as an antidote to heavy metal toxicity since the 1950s.

Research confirms this substance's efficacy and safety, and supports its use as the premier oral metal chelator for mercury—and its efficacy for other heavy metals. DMSA's water-solubility, oral dosing, large therapeutic window and low toxicity make it the chelator of choice for many applications.

About 20 percent of orally administered DMSA is absorbed from the gastrointestinal tract. This is two to four times the percentage of EDTA that is absorbed. It is believed that one of the sulfhydryls in DMSA binds to a cysteine molecule on albumin, leaving the other S-H to chelate metals (Maiorino *et al*., 1989; Aposhian *et al*., 1992).

2.2.10 International standards for lead levels

The threshold lead levels have been set for many lead containing matrices, and periodically reviewed to ensure safety and maintain an internationally acceptable standards that would guide level of exposure. International organizations like the WHO, FAO, CDC, ATSDR, EPA, EFSA, etc has set standards of different parameters resulting from research and other scientific investigations. Threshold level of concern, permissible exposure limit and withdrawal from exposure and medical attention limits were summarised for the parameters used in this research shown in Table 2.1.

Table 2.1: International Standards for Lead Levels

Agency	Nature of Sample	Lead Levels	Comment
EPA	Soil (residential)	< 50 ppm	Uncontaminated soil contains lead concentrations less than 50 ppm.
EPA	Soil	> 200 ppm	Soil lead levels in many urban areas exceed 200 ppm. (AAP 1993)
EPA	Soil	400 ppm	The EPA's standard for lead in bare soil in play areas is 400 ppm by weight and 1200 ppm for non-play areas.
EPA	Water (Drinking)	15 µg/L	Levels determined to be safe by toxicological and biomedical considerations, independent of feasibility.
			EPA's final rule establishes an action level is set at 15 µg/L.
WHO	Water (Drinking)	10 µg/L	
FAO	Water (surface)	100 µg/L	From rivers, ponds and lakes. Mostly for livestock consumption and other human activity.
OSHA	Air (Workplace)	50 µg/m^3	OSHA has set a PEL (enforceable) of lead in workplace air at 50 µg/m^3 averaged over an 8-hour workday for workers in general industry.
		30µg/m^3	Above the action level of 30 µg/m^3 for more than 30 days per year, OSHA mandates periodic determination of BLLs.
EPA	Air (Ambient)	0,15 µg/m^3	
CDC/NOISH	Air (Workplace)	100 µg/m^3	REL (Non-enforceable)
WHO/CDC	Blood	10 µg/dl	Ten µg/dL (micrograms /deciliter) was adopted by CDC in 1991.
		5 µg/dl	CDC guidelines are designed to keep children's BLLs below 10 µg/dL (CDC, 2002)
FDA	Food	0.5 µg/Kg	For lead in products intended for use by infants and children and has banned the use of lead-soldered food cans. (FDA 1994 and FDA 1995 as cited in ATSDR 1999)
EFSA	Food	0.3 µg/Kg	Lead dietary exposure in Europe
WHO/FAO	Food	3.0 µg/Kg	Threshold level of concern for grains

2.3 Methods of Lead Determination in Different Samples

Many of the analytical methods used for environmental and Biological samples are the methods approved by federal agencies and organizations such as EPA and the National Institute for Occupational Safety and Health (NIOSH). Other methods presented in this chapter are those that are approved by groups such as the Association of Official Analytical Chemists (AOAC) and the American Public Health Association (APHA). Additionally, analytical methods are included that modify previously used methods to obtain lower detection limits and/or to improve accuracy and precision.

2.3.1 Analysis of biological samples

Several analytical methods are available to analyze the level of lead in biological samples. The most common methods employed are flame atomic absorption spectrometry (AAS), graphite furnace atomic absorption spectrometry (GFAAS), anode stripping voltametry (ASV), inductively coupled plasma-atomic emission spectroscopy (ICP/AES), and inductively coupled plasma mass spectrometry (ICP/MS). According to Flegal and Smith (1995), GFAAS and ASV are the methods of choice for the analysis of lead. In order to produce reliable results, background correction, such as Zeeman background correction that minimizes the impact of the absorbance of molecular species, must be applied. Limits of detection for lead using AAS are on the order of μg/mL (ppm) for flame AAS measurements, while flameless AAS measurements can detect blood lead levels at about 1 ng/mL (Flegal and Smith, 1995). A detection limit of 0.05ng/mL has been achieved for lead in blood samples analyzed by GFAAS (Flegal and Smith, 1995). ICP/MS is also a very powerful tool for trace analysis of lead and other metals. Although ICP/MS instruments are more costly than GFAA instruments, their ability to analyze multiple metals from a single sample, low

detection limits, reliability, and ease of use have increasingly made them popular for trace metal analysis.

Other specialized methods for lead analysis are x-ray fluorescence spectroscopy (XRFS), neutron activation analysis (NAA), differential pulse anode stripping voltametry, and isotope dilution mass spectrometry (IDMS). The most reliable method for the determination of lead at low concentrations is IDMS (EPA, 1986), but due to the technical expertise required and high cost of the equipment, this method is not commonly used. It is primarily used for the development of certified standard reference materials by which other methods can determine their reliability since results of lead analyses from numerous laboratories often do not agree.

2.3.2 Analysis of environmental samples

The primary methods of analyzing for lead in environmental samples are AAS, GFAAS, ASV, ICP/AES, and XRFS (EPA, 1993). Less commonly employed techniques include ICP/MS, gas chromatography/photo ionization detector (GC/PID), IDMS, DPASV, electron probe x-ray microanalysis (EPXMA), and laser microprobe mass analysis (LAMMA). The use of ICP/MS for the analysis of trace metals (including lead) has increased in recent years due to its high sensitivity and ease of sample preparation.

Chromatography (GC, HPLC) in conjunction with ICP/MS can also permit the separation and quantification of organometallic and inorganic forms of lead. In analyzing lead concentrations in the atmosphere, a distinction between the levels of inorganic lead, which exists predominantly in the particulate phase, and alkyl lead, which occurs predominantly in the vapor phase, is necessary. Particulate-phase lead can be separated from the gas phase using a filter technique. The filter collects the

particulate matter and allows the dissolved material to pass through for separate analysis of each form. As with the analysis of biological samples, the definitive method of analysis for lead is IDMS.

2.4 Atomic Absorption Spectrophotometry (AAS)

Atomic Absorption Spectrometry, or AAS, is an analytical technique commonly used for the quantitative and qualitative determination of elements in samples such as aqueous solutions, waters, sea-waters, metals and alloys, glass, drugs, food, environmental samples, industrial wastes, biological samples among others.

This technique is based on measuring the amount of electromagnetic energy of a particular wavelength (ultraviolet or visible region), which is absorbed as it passes through a cloud of atoms of a particular chemical element (the analyte) coming from samples and standards. An appropriate mathematical treatment allows relating the amount of absorbed energy to the number of absorbed atoms by providing a measurement of the element concentration in the sample. This technique is established, relatively quickly, economically affordable and allows to determine more than 60 chemical elements from a huge type of samples. It is used by most of research laboratories and industry quality control around the world (Filho *et al.*, 2012)

The concentration is calculated based on the Beer-Lambert law. Absorbance is directly proportional to the concentration of the analyte absorbed for the existing set of conditions. The concentration is usually determined from a calibration curve, obtained using standards of known concentration in parts per million (ppm).

For the calibration curve, Prepare standard solutions of at least three different concentrations, measure the absorbance of these standard solutions, and prepare a calibration curve from the values obtained. Then measure the absorbance of the test

solution adjusted in concentration to a measurable range, and determines the concentration of the element from the calibration curve. The measurement procedure is as follows. A small quantity of the extracted sample is injected into a flame where the ions are reduced to elements and vaporized. The elements present in the sample absorb light at specific wavelengths in the visible or the ultraviolet spectrum. A light beam with a single specific wavelength for the element being measured is directed through the flame to be detected by a monochrometer (Flegal and Smith, 1995).

The light absorbed by the flame containing the extract is compared with the absorption from known standards to quantify the elemental concentrations. One of the disadvantages of this method is that only one element can be quantified at a time. AAS requires an individual analysis for each element, and sometimes a large filter or several filters are needed to obtain concentrations for a large variety of elements. Samples having high concentrations of elements beyond the linear range of the instrument should be diluted prior to the analysis.

The dilution factor (d.f), if used, as described below:

$$\text{d. f .} = \frac{\text{(Volume of diluted sample solution in ml)}}{\text{(Volume of aliquot taken for dilution in ml)}}$$

Báez *et al*., (2007) and García *et al*., (2009) characterized atmospheric aerosols, metals and ions that play an important role in the content of chemical species and of many elements in atmospheric ecosystem interfaces. Sodium, K^{+}, Ca^{2+} and Mg^{2+} were analyzed with a double beam atomic absorption spectrophotometer. Deuterium and hollow cathode lamps (Photron Super lamp) were used for background correction and analysis.

The advantages of using atomic absorption spectroscopy are:

- Good sensitivity and detection limits.

- Direct analysis of some types of liquid samples.

- Low spectral interference.

- Very small sample size.

The procedure consists of three general steps: atom formation, excitation, and emission. For UV and visible spectroscopy, the input energy must be sufficient to raise an electron from the ground state to the excited state. Once the electron is in the excited state, the atom emits light, which is characteristic of that particular element. Before excitation, an element that is bound in a specific matrix must be separated from that matrix so that its atomic emission spectra are free from interferences.

The elements present in a sample are converted to gas phase atoms in the ground state. The UV-Vis absorption of these gas phase atoms are then measured by irradiation of light at a highly specific wavelength causing transition of some of the gas phase atoms to a higher energy level. The extent to which light is absorbed is related to the original concentration of ground state atoms. This situation is completely analogous to the Beer-Lambert law in conventional liquid UV-Vis absorption Spectrophotometry. Conversion of the sample from its native state to the atomic state can be achieved using a flame (flame-AAS) or an electric furnace (electro-thermal or graphite furnace AAS).

In the furnace, the sample undergoes a number of pre-treatment steps prior to analysis.

1. Sample is dried by evaporating the solvent (in this case the water).

2. The organic matrix is decomposed by heating of the sample $\geq$1000ºC, (taking care not to lose any of the analyte through evaporation processes).

3. Furnace is rapidly heated to temperatures around 2400ºC to produce vaporized neutral atoms.

This method provides both sensitivity and selectivity since other elements in the sample will not generally absorb the chosen wavelength and thus, will not interfere with the

measurement. However, molecular species may also be formed during the atomization step. This can alter the spectral characteristics of the analyte metal or can cause spectral interference at the wavelength being monitored. To reduce background interference, the wavelength of interest is isolated by a mono-chromator placed between the sample and the detector. Additional techniques such as D2 or Zeeman background correction may also be used for complex matrices such as beer.

The GFAA and flame AAS measurement principle is the same. The difference between these two techniques is the way the sample is introduced into the instrument. In GFAA analysis, an electrothermal graphite furnace is used instead. The sample is heated stepwise (up to 3000ºC) to dry. The advantage of the graphite furnace is that the detection limit is about two orders of magnitude better than that of AAS.

The graphite furnace has several advantages over a flame furnance. First it accepts solutions, slurries, or solid samples. Second, it is a much more efficient atomizer than a flame furnace and it can directly accept very small absolute quantities of sample. It also provides a reducing environment for easily oxidized elements. Garcia *et al*., (2009) determined Pb in total suspended particles in southwest Mexico City, using a nitric acid digestion–based AAS method.

Atomic absorption analyses are most commonly and routinely performed on solutions.

Therefore a sample must be converted to liquid form prior to analysis using a microwave to digest the sample, leaving a solution that can then be analyzed. However this method has the following limitations:

Limitations:

1. Elemental range is limited to metals and metalloids.

2. Sample preparation is tedious and time consuming.

3. The sample is destroyed by the analysis.

4. Only one element at a time can be measured.

An atomic absorption Spectrophotometry analysis was used for the determination of lead levels in this work under the selected conditions shown in the table 2.2.

Table 2.2: Standard Atomic Absorption Conditions for Pb

Wavelength (nm)	Slit (nm)	Relative Noise	Characteristic Concentration (mg/L)	Characteristic Concentration check (mg/L)	Linear range (mg/L)
283.3	0.7	0.43	0.45	20.0	20.0
217.0	0.7	1.0	0.19	9.0	20.0
205.3	0.7	1.4	5.4	250.0	----
202.2	0.7	1.8	7.1	350.0	----
261.4	0.7	0.35	11.0	500.0	----
368.3	0.7	0.40	27.0	1200.0	----
364.0	0.7	0.33	67.0	3000.0	----

1. Recommended Flame: air-acetylene, oxidizing (lean, blue)

2. Data obtained with a standard nebulizer and flow spoiler. Operation with a High Sensitivity nebulizer or impact bead will typically provide a 2-3 sensitivity improvement.

3. Characteristic Concentration with a N_2O-C_2H_2 flame at 283.3 nm: 2.7 mg/L.

4. Table contains Hollow Cathode Lamps (HCL) data. Electrodeless Discharge Lamps (EDL) sensitivity values approximately the same.

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Materials

3.1.1 Equipment and glassware

- Shimadzu AAS machine, Model spectra AA-6800 of the National Research Institute for Chemical Technology (NARICT),.
- Varian AAS model 240FS machine, with GTA-120 graphite furnace of the Multi-User Science Research Laboratory/ Ahmadu Bello University, Zaria (MUSRL/ABU).
- Geographical Position System (GPS) mobile trackers (Extrex, Garmin, USA).
- Medical facilities and supplements (Hand gloves, air masks, syringes: 2ml, 5ml and multivitamins, sweets, etc)
- Analytical balance (Mettler-Toledo balance model AE163 No. C4 1075)
- Hot plates: Mode HC500, BIBBY.
- Electrical Oven: Model MIR-162, SANYO.
- Fume cupboard: Model Air one 1000RS, SAFELAB.
- Refrigerator: Model Casart, HAIER THERMOCOOL.
- Laboratory pestle and mortar
- Specimen bottles (sterilized and pre-treated) for Blood and Water samples
- Polythene packs (for solid samples).
- Weighing balance (Mettler-Toledo GmbH, Laboratory and Weighed Technologies, Switzerland)
- Beakers: 10, 50, 250ml (Pyrex)
- Volumetric flasks: 25, 50, 100ml (Pyrex)

- Measuring cylinders: 5, 10, 50, 100ml (Pyrex)
- Filter paper (Whatman 110mm)
- Funnel (Pyrex)
- Micro pipette (with adjustable volume)
- Polythene sample bottles: 5, 100, 120ml

3.1.2 Chemicals and reagents

- All chemicals used for the analysis are of analytical grade obtained from British Drug House (BDH). Standard stock solution used for the calibration curves for the standards are of standard grade. Other reagents used include the following:
- Hydrochloric acid, HCl (Analytical grade)
- Trioxonitrate (V) acid, 0.5% HNO_3 (Analytical grade)
- 10% Trioxonitrate (V) acid (Analytical grade)
- Hydrogen peroxide, 30% H_2O_2 (Analytical grade)
- Deionised distilled water.

3.1.3 Instrumentation

(a) Analysis of Soil, Human and Animal Blood samples, underground and surface water samples were performed by Atomic Absorption Spectrophotometry (AAS) using Varian AAS model 240FS machine, with GTA-120 graphite furnace a Multi-User Science Research Laboratory/ Ahmadu Bello University (MUSRL/ABU), Zaria, with the following conditions:

- Element – Matrix: Pb – Water (Flame)
- Lamp: Lead Hollow Cathode
- Lamp current: 5.0 mA

- Wavelength: 217.0nm
- Slit With: 1.0 nm
- Flame type: Air / Acetylene
- Fuel flow rate: 2.0 L/min
- Concentration Unit: ppm

(b) Analysis of Common Grains Samples: Sorghum (GS1), Millet (GS2), and Maize (GS3) were performed by Atomic Absorption Spectrophotometry (AAS) using Shimadzu AAS machine, (Model spectra AA-6800) at NARICT with the following conditions:

- Element – Matrix: Pb
- Lamp Mode: BGC-SR
- Lamp current: 8.0 mA
- Wavelength: 283.3 nm
- Slit With: 1.0 nm
- Flame type: Air – C_2H_2
- Fuel flow rate: 2.0 L/min
- Concentration Unit: ppm

3.2 Methods

3.2.1 Geographical location of the study areas

Zamfara State located about 240Km from the city of Kaduna, both located in the North-Western Nigeria. The State is located at $12^0 10`$ N $6^0$ 15`E, bordered by Kaduna and Katsina States in the east, Sokoto and Kebbi States in the North and South West respectively. Another portion in the North bordered with Niger Republic. Zamfara State consists of 14 Local Government Areas with a total population of 3,838,160 (2011 updated census) and ranked 23rd of the 36 state of Nigeria, and its capital is Gusau shown in Figure 3.1 and 3.2. It has a land area of 39,762 Km^2 and rank 7th of 36 in terms of land area. The research covered ten villages from four LGAs, namely: Anka, Bukkuyum, Gusau and Maru. The choice of villages was at random guided by the availability of mining communities in those villages and the security challenges in other mining communities. The villages that constitute the study area include: Bagega, Kadauri, Kawaye, Kwali, Magami, Sunke, Tsunami, Tungar-Kudaku, Tungar-Guru and Yargalma. In all these villages, they have historical records of gold mining and still are involved in active gold-mining and processing, though remediation has taken place in most of the villages, reversal effect remained the major set-back in the control of lead poisoning (TerraGraphics, 2011).

The four LGAs where the ten selected villages from the mining communities include:

Anka LGA with an area of 2,746 Km^2 and a population of over 142,280 (2006 Census). It has many mining sites and the selected villages for this research include Bagega, Kawaye, Sunke and Tungar-Kudaku. Bukkuyum LGA with a land area of 3,214 Km^2 and a population of over 211,633 (Census, 2006). The villages selected include Kwali, Yargalma and Tungar-Guru. Gusau being the capital of Zamfara State also has some historical record of mining; Tsunami (known as Unguwan Danbaba) and Magami were

the selected locations for this research. Gusau LGA located at 12^0 09`N $6^0$ 40`E, with land area of 3,364 Km^2 and a population of over 383,162 (Census, 2006). Another local government covered by the research was Maru LGA, located about 70Km from Gusau, and the selected mining community was Kadauri located along Gusau- Anka Road. Maru with a population of 291,900 (Census, 2006) and a land area of 6,654 Km^2. The GPS coordinates of the five selected compounds in each village was recorded as at the time of sample collection using GPS mobile trackers (Extrex, Garmin, USA).



Figure 3.1: Map of Nigeria showing Zamfara State

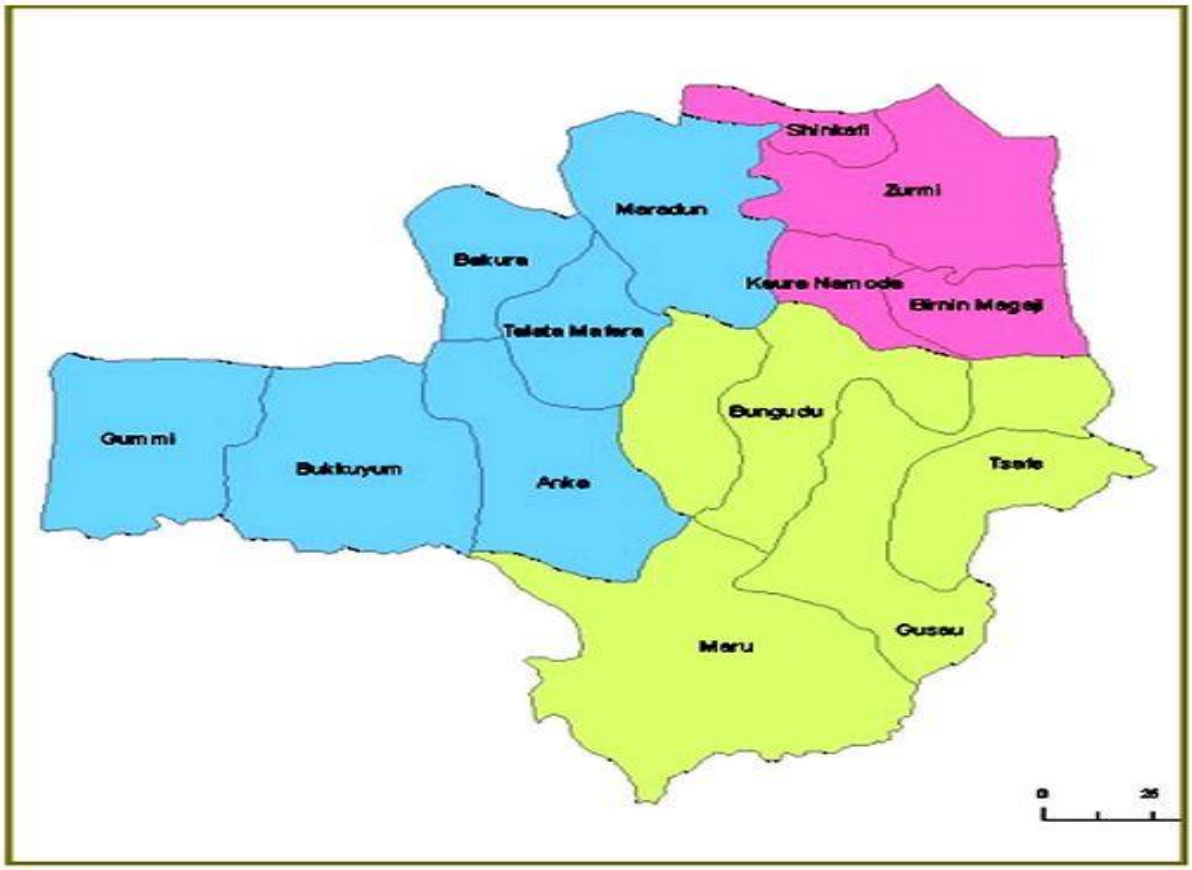


Figure 3.2: Map showing the LGAs of Zamfara State – Nigeria

Ten selected mining communities from four Local Government Areas were identified at random, but the selection of these communities was guided by the security challenges in the state and around those communities. The communities selected were:

- **Anka:** Bagega (BAG), Kawaye(KWY), Sunke (SUK), Tungar-Kudaku (TKD).
- **Bukkuyum:** Kwali (KWL), 'Yargalma (YGM), Tungar-Guru (TGR).
- **Gusau:** Magami (MGM), U/Danbaba/Tsunami (TSM).
- **Maru:** Kadauri (KAD).

3.2.2 Advocacy and community relations

For a start, in September 2013, introductory letters were obtained from the Faculty of Pharmaceutical Sciences, ABU, Zaria, to the Ministries of Health and Environment respectively, introducing me as a research fellow from Ahmadu Bello University in respect of the lead poisoning in the selected mining communities. The Ministry of Environment referred the request to the Zamfara State Environmental Sanitation Agency (ZESA), for contact and advocacy at the selected emirates of Anka, Bukkuyum, Maru and Gusau. A total of ten villages from the listed emirates/LGA were visited a total compliance from almost all the villages was achieved with the research design as shown in the Figure 3.3.

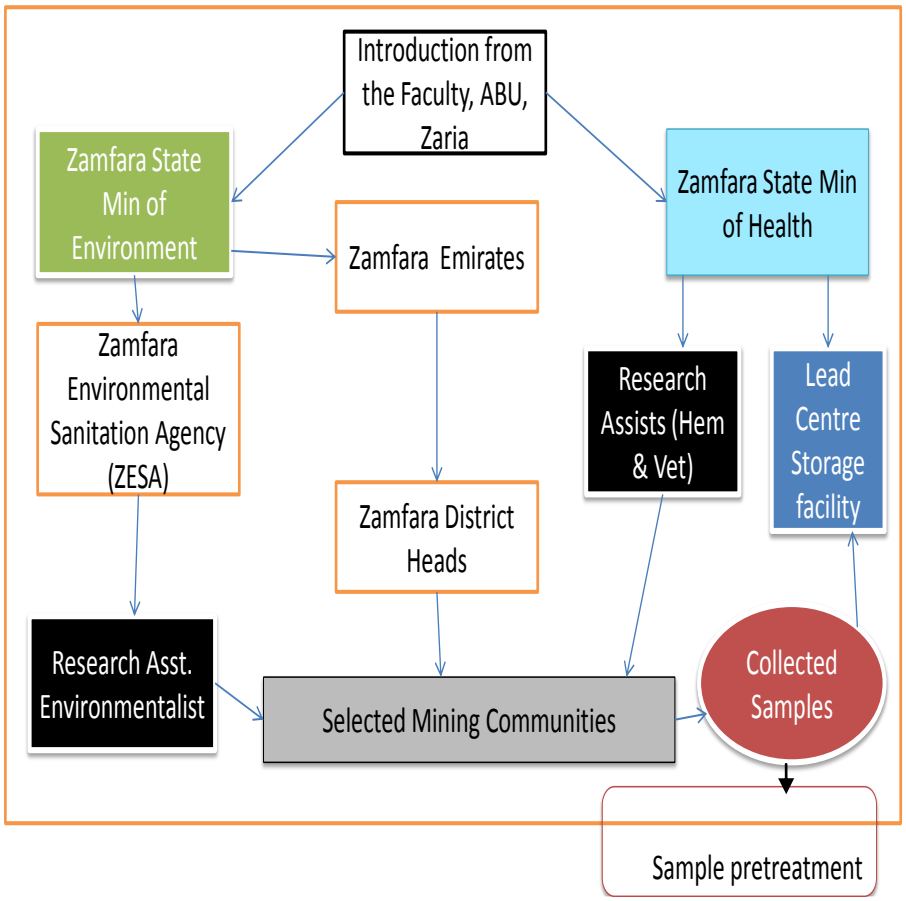


Figure 3.3: Advocacy and Research design

3.2.3 Sample and sampling site selection

Samples were selected by stratified sampling, (Harper 1976; Elwood 1982). Five sampling units were selected from each village, often based on Traditional Heads guide and assistance. Each sampling unit represents a family compound. From each compound of the sampling community, samples of soil, common grains (sorghum, millet and maize), human blood sample (from children and adults) and animal blood samples were collected. However, underground water and surface water, were often shared within the mining communities rather than from each compound of the selected mining community. The listed represent the basic necessities of daily/periodic need or contact in the life of each community:

A well defined pattern of sample selection could not be achieved, as selection of samples was based on what was available in the selected compound, rather than what was desirable, particularly in terms of animal blood samples and surface water samples.

In terms of human blood samples, the selection from each selected compound was based on age. Children below or equal the age ranged of six years was considered, while in the adult category, adults of age ranged 18 years and above were considered. Selection indicated that, a minimum age of two years from children and a maximum age of 60 years were among sampling groups.

3.2.4 Samples collection and preservation

For the success of this research, the service of three other field research assistants was employed. The entire team was involved in the field trips and sample collection. The research assistants include: an environmentalist from the Zamfara State Environmental Sanitation Agency (ZESA), a hematologist from the King Fahd children and women

specialist hospital, Gusau, and a veterinary officer from the veterinary division of the Ministry of Health, all from Zamfara State.

The list of samples was collected from the selected compounds under the usual collection protocol of samples and simple questionnaire administered to the head family of each selected compound from December, 2013 to January, 2014. The coordinates of the Geographical Position System (GPS) of each sampling compound was taken and could be used as reference in case of follow up study or confirmation during or after treatment, for documentation of the selected compounds' parameters.

3.2.4.1 Soil samples

Soil samples were collected in a clean polythene plastic pack; about 10g of soil sample from the surface to approximately 2cm dip were collected and stored in dry place for digestion.

3.2.4.2 Grain samples

Sorghum, millet and maize were collected in three separate clean and dry polythene bags. About 10 g of each grain sample were provided by each compound house hold, labeled and stored in a dry place until ready for digestion.

3.2.4.3 Water samples

Water sample from both underground source (well water) and surface water from ponds and lakes were collected in a 50 ml plastic container properly closed, labelled and stored for digestion. Preservation of water samples was done by adding 2 drops of concentration HNO_3 to each water sample to maintain a pH of between 2.2 to 2.8, before storage below 4^0C until analysed.

3.2.4.4 Blood samples

Human and animal blood samples were collected by the hematologist and the veterinary officer respectively, using sterilized equipment and clinical protective wears. 2 ml syringe was used in taking blood samples from both human and animal's samples, and the specimen was transferred emptied into a sterilized 5 mls sample bottle coated with anticoagulant. The blood samples were refrigerated at Lead Centre of Excellence, Gusau, before it was transferred to the Multi-User Science Research Laboratory (MUSRL)/ABU, Zaria and stored appropriately for digestion.

3.2.5 Preparation of lead stock solution for calibration curve

A 1000 μg/L Pb solution was prepared by dissolving 1.598 g lead nitrate in 5.0 cm^3 10% nitric acid and was diluted with water to mark in a 100ml volumetric flask.

3.2.6 Calibration curve for lead in soil samples

1ml of the stock solution of lead was diluted to 9.0 ppm. And this standard was serially diluted to 1.0, 3.0, 6.0 and 9.0 ppm and set for analysis. The serially diluted samples were aspirated using AAS machine and subjected to same treatment as reported in 3.2.7 and shown in Figure 4.1.

3.2.7 Calibration curve for lead in grain samples

1ml of the stock solution of lead was diluted to 4.0 ppm. And this standard was serially diluted to 1.0, 2.0, 3.0 and 4.0 ppm and set for analysis. The serially diluted samples were then aspirated into the AAS machine. Calibration curves were based on four standards. The instrument was programmed to take three readings per sample and average the absorbance. Instrument blanks (0.5 % HNO_3) and check standards were

processed with all samples. Sample concentrations were then corrected for deviations from the standards, and final wet weight was factored into the calculation of final values. The calibration curve was used to obtain the lead content in the grain samples as shown in Figure 4.2.

3.2.8 Calibration curve for lead in blood samples

1ml of the stock solution of lead was diluted to 9.0 ppm. And this standard was serially diluted to 1.0, 3.0, 6.0 and 9.0 ppm and set for analysis. The serially diluted samples were aspirated using AAS machine and subjected to same treatment as reported in 3.2.7 and shown in Figure 4.3

3.2.9 Calibration curve for lead in underground and surface water

1ml of the stock solution of lead was diluted to 9.0 ppm. And this standard was serially diluted to 1.0, 3.0, 6.0 and 9.0 ppm and set for analysis. The serially diluted samples were aspirated using AAS machine and subjected to same treatment as reported in 3.2.7 and shown in Figure 4.4.

3.2.10 Preparation and analysis of samples

3.2.10.1 Sample pretreatment for grains

Grain samples were sieved of sandy particles, and then washed with tap water to remove traces of dust particles. They were then washed with distilled water and allowed to air dry. The air dried samples were placed in an oven for 12 hours. The dried samples were then grinded into a powder using laboratory mortar and pestle. Each grain

in separate polythene bag, making a total of 150 samples labeled and stored for digestion.

3.2.10.2 Analysis of lead in grain samples

The sample wet digestion was carried out by weighing accurately 2 g of the dried powdered grain sample and transferred into 250 ml beaker containing 4ml HNO_3. The mixture was subjected to heat using a hot plate until all brown fumes ceased from the mixture. The mixture was then allowed to cool and 20 ml of deionized water added and filtered with filter paper. More water was added to make up to 100 ml, and the sample analysed for lead in the AAS machine under the conditions stated earlier (Horwitz, 1982; Ellen and Van Loon 1990).

3.2.10.3 Sample pretreatment for soil

Soil samples collected from 50 compounds were grinded and sieved off from stones and other debris, stored in dry oven condition for 24 h for preparation and analysis.

3.2.10.4 Analysis of lead in soil samples

2 g soil sample was placed in a tube and 10 ml of concentrated HNO_3. The tube is connected to a funnel condenser overnight at a temperature from 80 to 90 ◦C. The apparatus was removed on the furnace support after heating to dryness at 125–130 ◦C. Then 1 ml of concentrated HNO_3 and 4 ml of concentrated $HClO_4$ were added and the mixture was heated to dryness at 200–210 ◦C. The tube container was then cooled and 4 ml of HCl and 50 ml of distilled water were added. The solution is passed through an acid-washed filter membrane and was then transferred quantitatively to a 100 ml volumetric flask by adding enough volume of 1% HNO_3 (Burau, 1982).

3.2.10.5 Analysis of lead in human and animals blood samples

Whole blood samples were digested according to NIOSH, (1977c) modified procedures described by (Hogstrand *et al*., 1996; U.S. EPA, 1997; Shaw *et al.*, 1998). A 500µL sub-sample of whole blood was removed from the original sample and digested with 2.0 ml trace metal grade concentration HNO_3. The sub-samples were then heated (100^0 C/2h), until brown fumes were all evolved. The mixture was allowed to cool at room temperature and, once complete digestion was achieved, 300 µL of 30% H_2O_2 was added to each sample followed by heat-instilling until dry. The samples were then reconstituted in 5.0 ml of 0.5 % HNO_3 with deionized water.

These prepared samples were then aspirated into the AAS machine. The instrument was programmed to take three readings per sample and average the absorbance. Instrument blanks (0.5 % HNO_3) and check standards were processed with all samples. Sample concentrations were then corrected for deviations from the standards, and final wet weight was factored into the calculation of final values. The lead contents of the blood samples were determined from the calibration curve.

3.2.10.6 Analysis of underground water samples

1 ml of water sample was digested by mix with 4 ml of HNO_3. The mixture was heated gradually until the brown fumes disappear. The resulting mixture was cooled and made to 10 ml mark and stored for analysis using AAS according to modified procedures (Chau *et al*., 1979).

3.2.10.7 Analysis of surface water samples

Water samples were digested by taking 1 ml of water sample and mixed with 4 ml of mixture of H_2O_2 and HNO_3 (1 : 3). The physical clarity and turbidity of the surface

water was much higher than the underground water. The mixture was heated gradually until the brown fumes disappear. The resulting mixture was cooled and made to 10 ml mark and stored for analysis using AAS (Chau *et al*., 1979).

3.2.11 Validation of the adopted methods

3.2.11.1 Precision

Validation of the adopted methods was done through determination of precision and accuracy. According to Horwitz (1982), as cited from Gonzalez and Herrador (2007), the maximum RSD value acceptable for the analyte level of 10 μg/mL is 16 %. AOAC set the maximum acceptable RSD value at 11% for the same analyte level (Gonzalez and Herrador, 2007). Since the percentage of coefficient of variation (%CV) for the replicate analysis of each measurement was < 15% CV calculated from the formula:

$$\% \text{ CV} = \text{S/X x100}$$

Where X is the mean and S is the standard deviation

Therefore, it can be stated that the proposed method showed good precision based on %CV (RSD %) values obtained as shown in Table 4.1.

3.2.11.2 Accuracy

According to previous published study (Huber, 1998), the acceptable recovery percentage range is 80-110% for the analyte level of 10 μg/mL. Five replicate analysis of the analyte solution was use for the determination. Accuracy is expressed as percentage relative error (% Er) calculated using the formula, and recovery was within acceptable range, with results shown in Table 4.1.

$$\% \text{ Er} = \text{X - } \mu/\mu \text{ x } 100$$

Where X is the mean and μ is the expected value.

3.2.12 Demographic Distribution of Socio-Economic Opportunities of the Selected Mining Communities

A demographic data was collated using a questionnaire (Appendix XXVII), from which, information based on interview/oral responses gave a fair status of the socio-economic opportunities of the selected mining communities.

3.2.13 Statistical analysis

The statistical analysis of data collected was carried out using scientific statistical package of GraphPad Prism 6 Statistical package. Descriptive statistics of each set of data was carried out to test the normality, standard deviation, mean, standard error of mean, difference between means and variance.

The statistical analysis indicated similarities and differences in all the parameters used as samples. Depending on the nature of the data, in addition to descriptive statistics, column statistics, one sample t-Test, 2way ANOVA, and multiple comparisons test were deploy to analyze various tables of values. 2way ANOVA, Multiple comparisons test was used to compare differences within each community and across different communities at confidence interval of 95% and alpha = 0.05. At confidence interval of 95% ($P < 0.05$) was considered statistically significant.

CHAPTER FOUR

4.0 RESULTS

4.1 Geographical Position System (GPS) of the Sampling Compounds

The coordinates of each sampling location in relation to the compounds number (I – V) were recorded for accurate grouping of collected samples and possible follow-up in subsequent research, as shown in Appendix I.

4.2 Calibration Curves and Validation of Adopted Methods

4.2.1 Calibration curves for the adopted methods

The calibration curves for the adopted methods of analysis residential soil, grain samples, human and animals blood samples, and water samples as shown in Figures: 4.1, 4.2, 4.3, and 4.4.

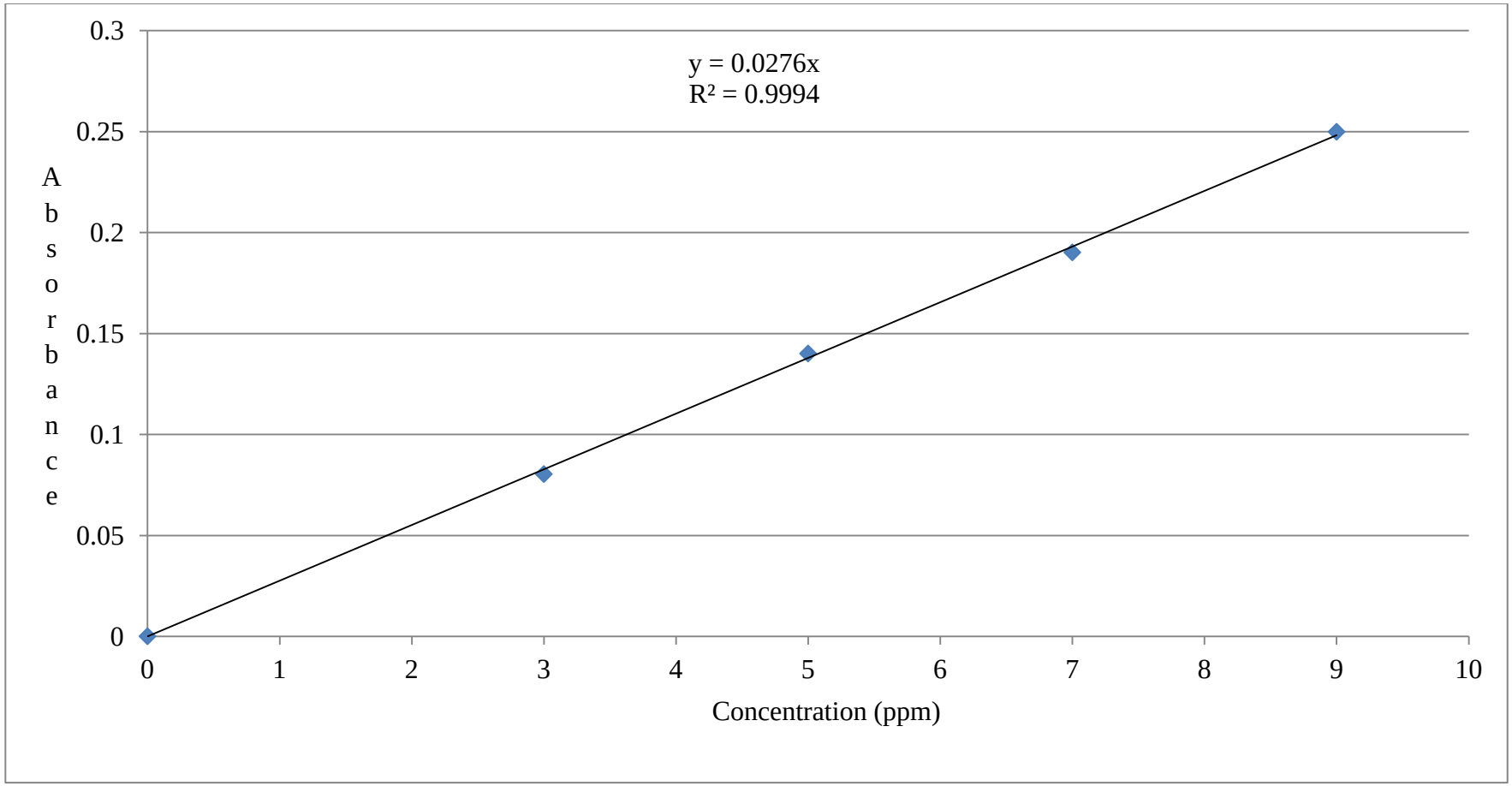


Figure 4.1: Calibration curve for lead in residential soil from selected mining communities

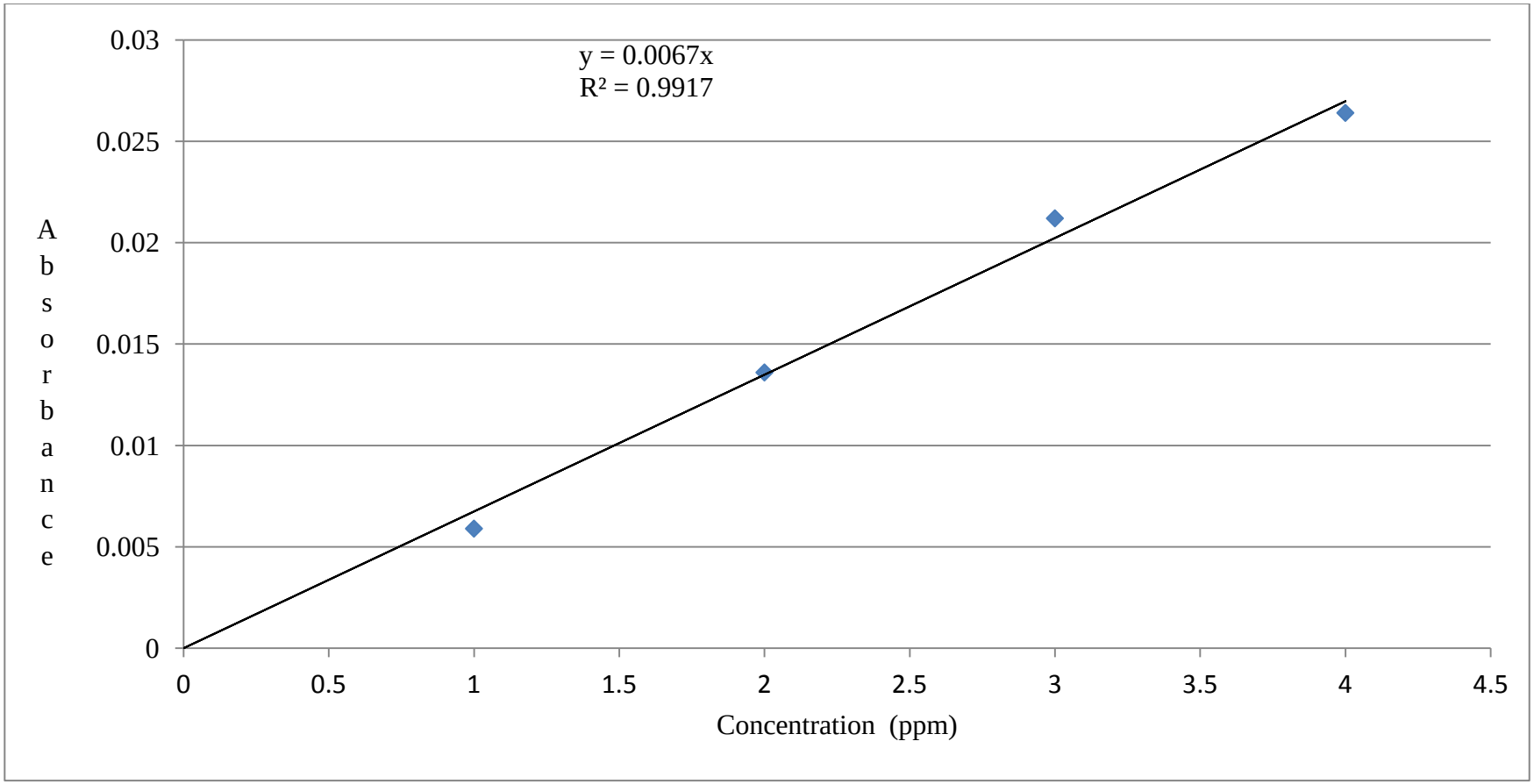


Figure 4.2: Calibration Curve for lead in grain samples from the selected mining communities

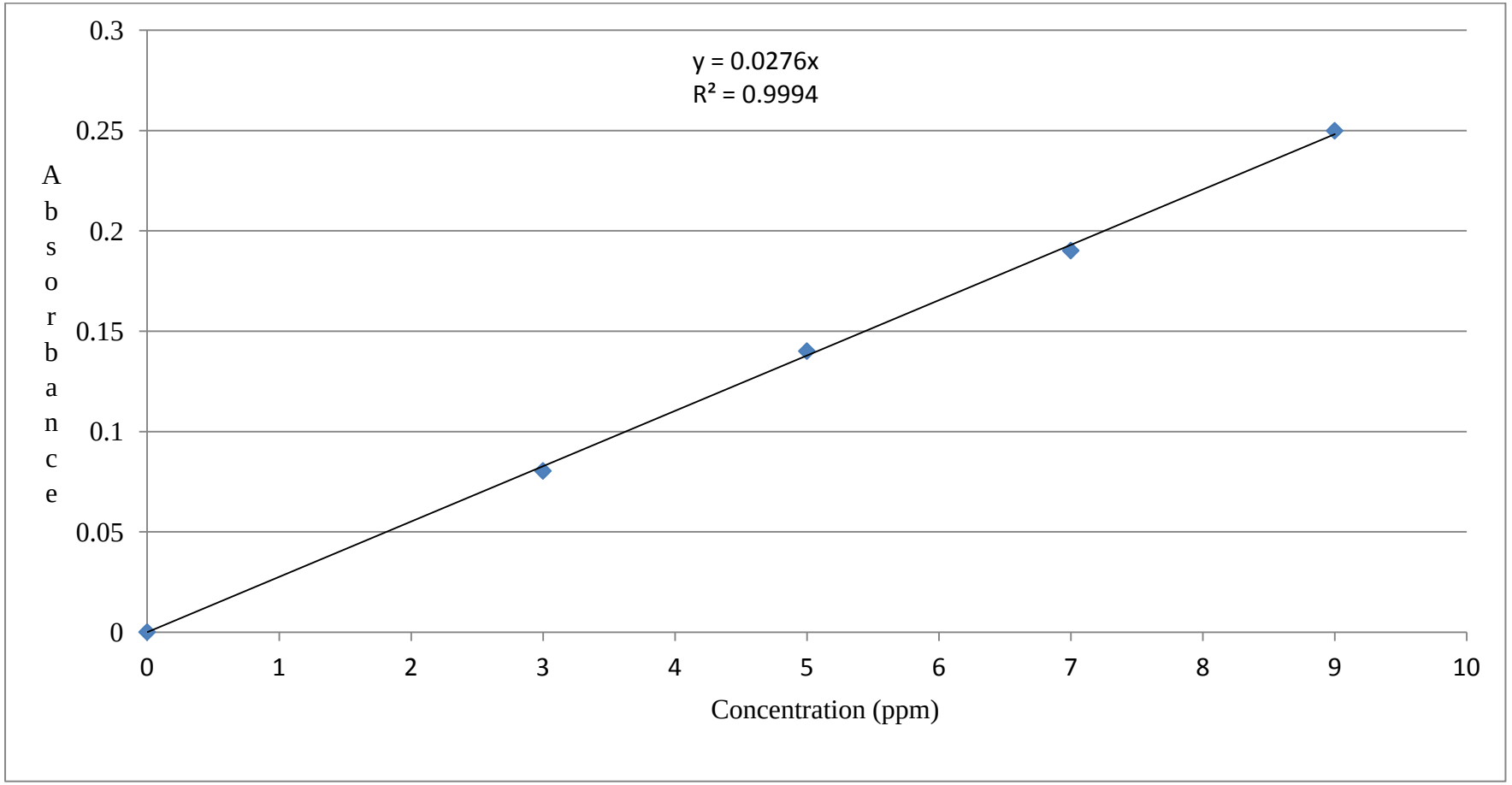


Figure 4.3: Calibration Curve for lead in human and animals blood samples from the selected mining communities

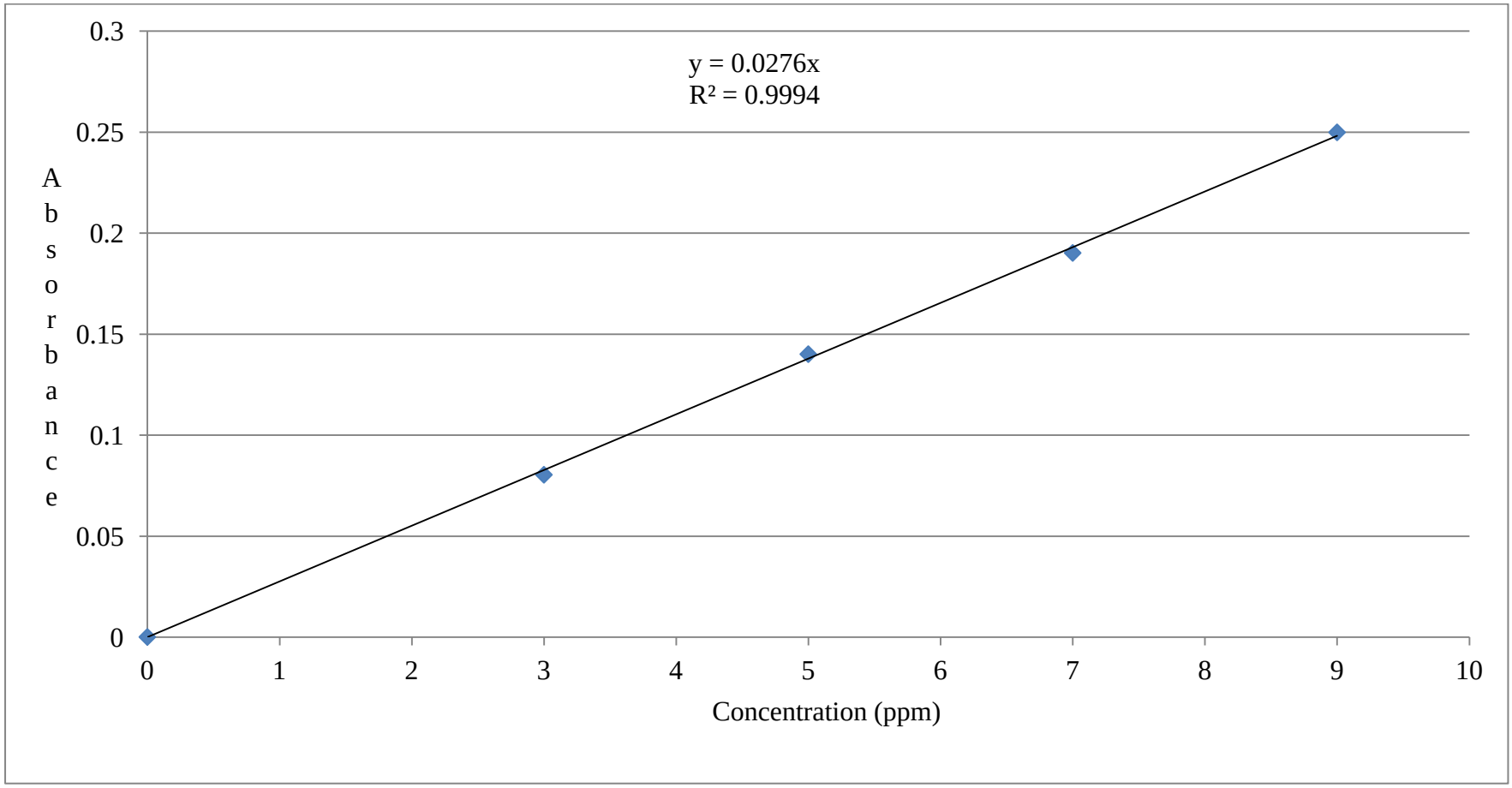


Figure 4.4: Calibration curve for lead underground and surface water from the selected mining communities

4.2.2 Validation results of the adopted methods

Table 4.1: Validation results of the adopted methods

parameter	For soil sample	For grain sample	For blood sample	For water sample
λmax (nm)	217.0	283.3	217.0	217.0
Precision (% CV)	4.9	9.5	6.0	4.7
Accuracy (% Er)	95.7	96.5	97.7	89.0

The percentage recovery was found to be within the accepted limit of 88 – 110%

4.3 Lead Concentration in Soil from Residential Compounds

The Lead concentration in soils from residential compounds (sampling units I – V) were determined for each village of the selected mining communities. Although four out of the ten villages, namely: Bagega, Sunke, Tungar-Guru and Yargalma have undergone soil remediation between September 2010 and December, 2011 (TerraGraphics, 2011).

Results of the residential compound soil analysis indicated that, lead concentration in soil in all the selected villages; except for Tungar - Guru, with 700 mg/Kg, were below the United States – Environmental Protection Agency (US – EPA, 1997) acceptable level for lead in compound soil of (400 mg/Kg) as shown in Figure 4.5 and Appendix II.

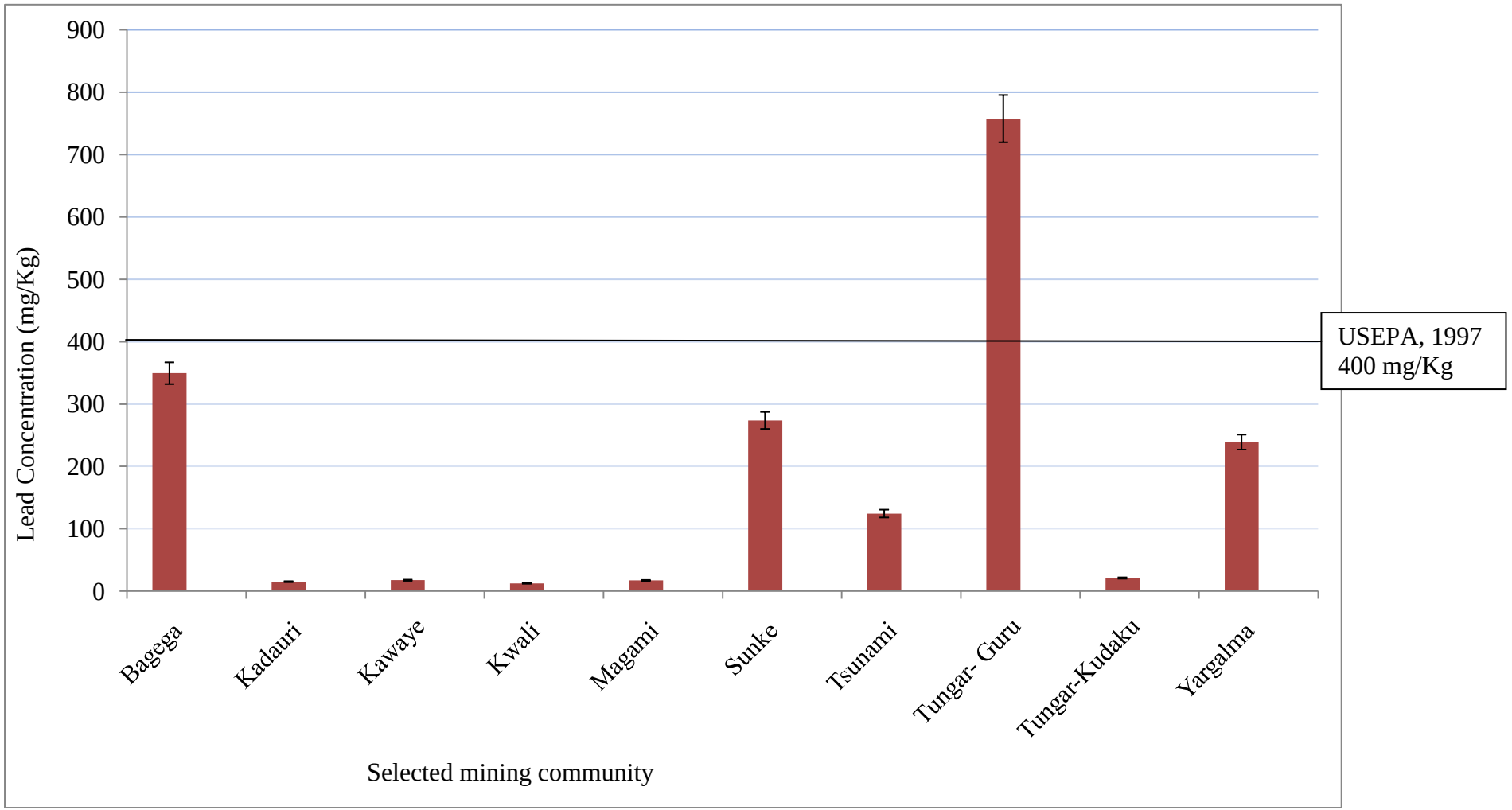


Figure 4.5: Mean concentrations of lead in soils from residential compounds in the selected mining communities

4.4 Lead Concentration in Sorghum, Millet and Maize Samples

The common Grains Samples (GS) were: Sorghum (GS1), Millet (GS2) and Maize (GS3). Except for millet from Kwali which recorded very high mean lead concentration (72.07 ± 57.36mg/Kg), the mean lead concentration among the three grains ranged from 5.56 ± 2.02 to 28.18 ± 5.93 mg/Kg. What was observed in Kwali may be influenced by one of the millet samples from residential compound I, where excessive contamination might not be ruled out. The mean lead concentration in all the grains analysed were above the 3.0 µg/Kg WHO/FAO lead threshold level of concern as shown in Figures 4.6, 4.7 and 4.8 and Appendices III, IV and V.

Statistical analysis indicated no significant difference ($P < 0.05$) within community but there were significant difference ($P < 0.05$) between communities. Test for equality of variance (F- Test) indicated unequal variance between communities while there was no difference community.

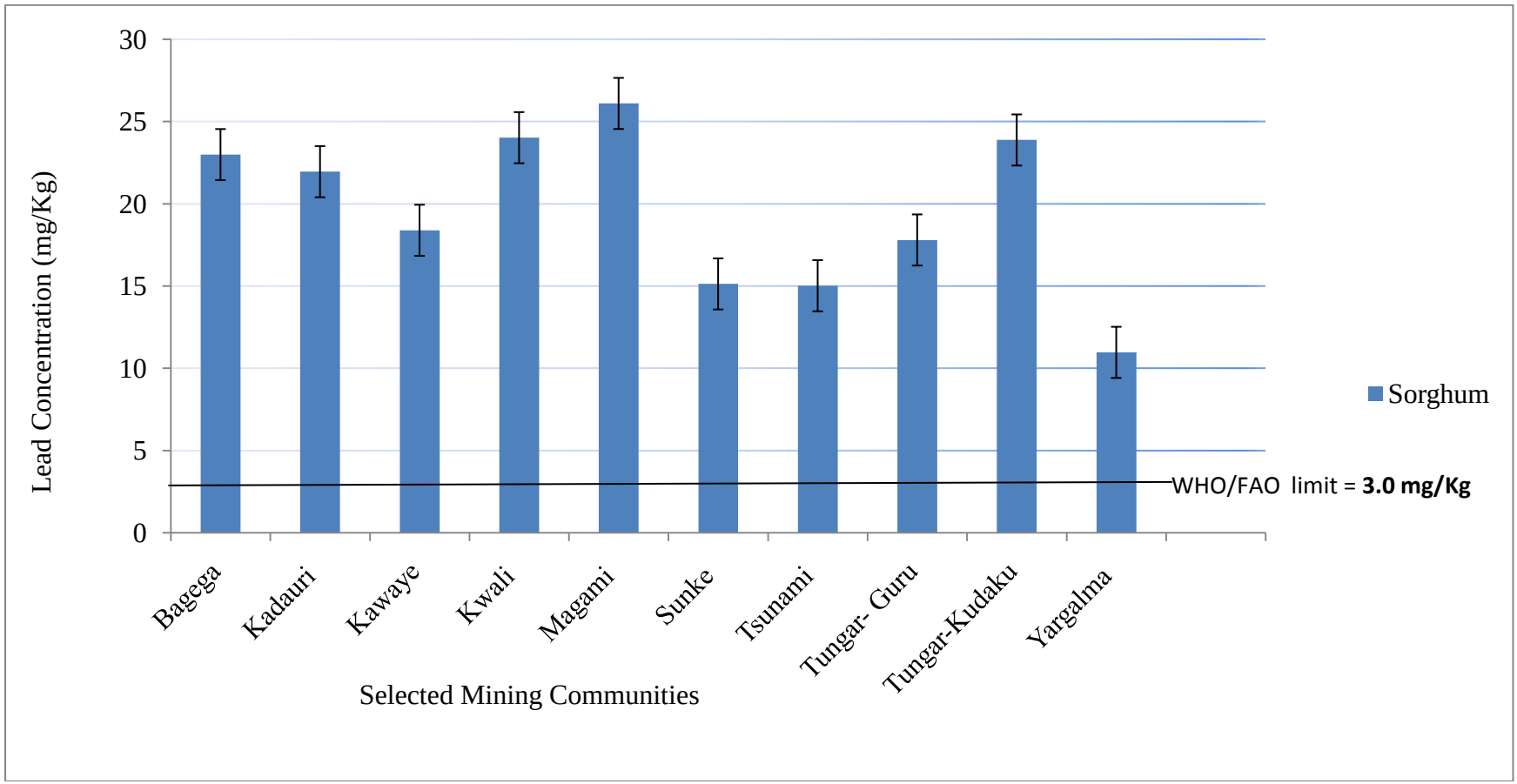


Figure 4.6: Mean lead concentration in Sorghum (GS1) from the selected mining communities

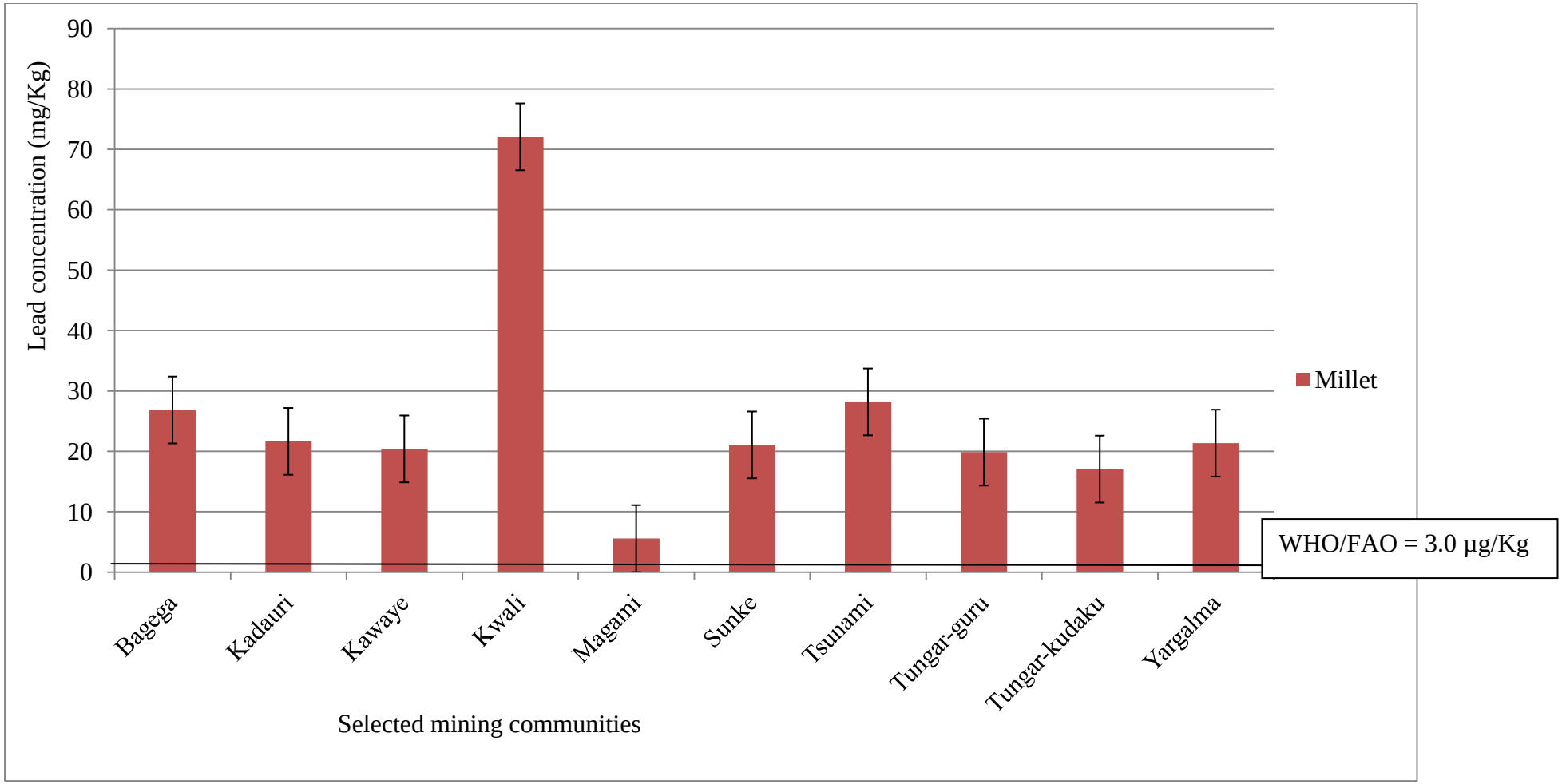


Figure 4.7: Mean lead concentration in Millet (GS2) from the selected mining communities

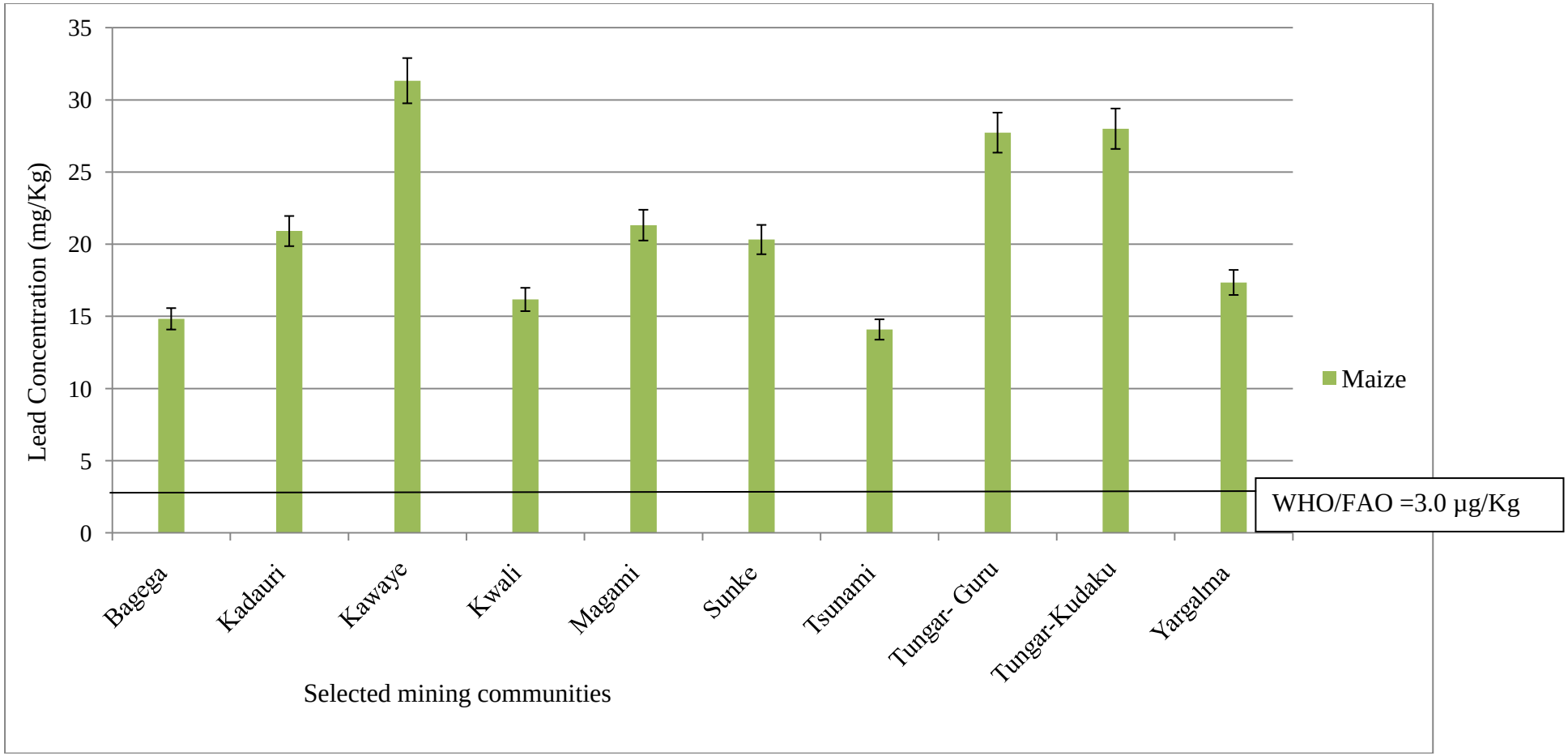


Figure 4.8: Mean lead concentration in Maize (GS3) from the selected mining communities

4.5 Comparison of Mean Lead Concentration in the Three Common Grains

Comparison of the mean lead concentration of the three common grains (Sorghum, Millet and Maize) show some similarities and one distinct difference in the concentration of millet from Kwali. From the lead concentration in the three grains, Sorghum appears to have the highest concentration from 7 out of the 10 villages, followed by millet and then maize as shown in Figure 4.9 and Appendix VI.

Statistical analysis indicated that there was no significant difference ($P < 0.05$) within communities except in one case involving the millet sample from Kwali. Also there was no significant difference ($P < 0.05$) across communities except in the comparison involving millet from Kwali and the rest of the communities.

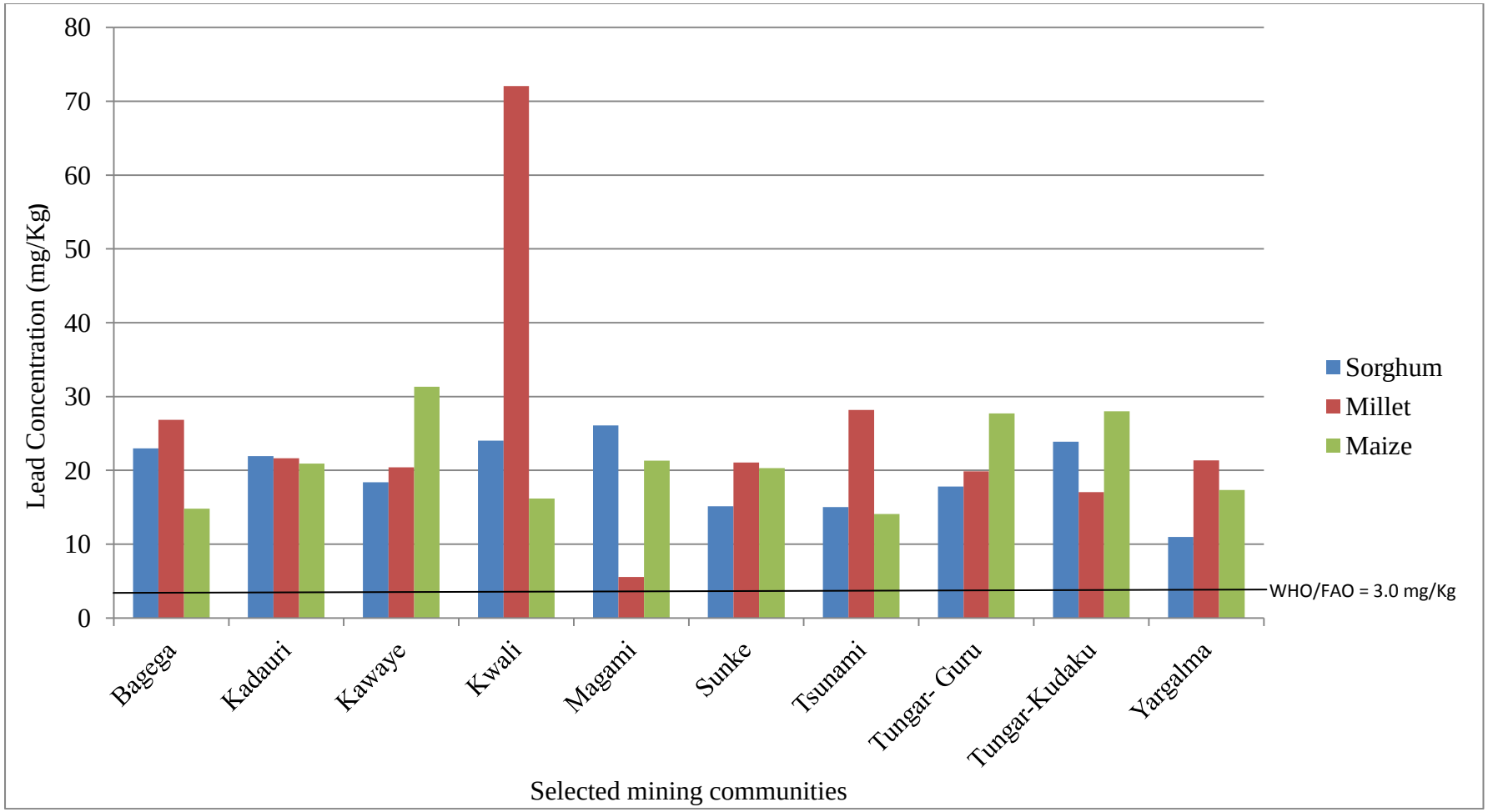


Figure 4.9: Comparison of lead concentrations in common grains from the selected mining communities

4.6 Lead Concentration in Human (Children and Adult) Blood Samples

There are similarities generally in the blood lead level trends of both children and adults from the villages. The mean blood lead concentration in children was lowest in Kwali and highest in Bagega, while that of adults was highest in Magami and lowest in Tungar-Kudaku as shown in Figures 4.10 and 4.11 and Appendices VII and VIII.

Statistical analysis indicated that there is no significant difference ($P < 0.05$) within a community, but significant difference ($P < 0.05$) was observed in children's blood lead level between some communities (Bagega Versus Kwali, . A significant difference ($P < 0.05$) was also observed in adults' blood lead level across some communities (Bagega, Kwali, Kawaye, Sunke,Tsunami, Magami, Tungar-Kudaku, Tungar-Guru, and Yargalma) in multiple comparisons between each community with the rest of the communities.

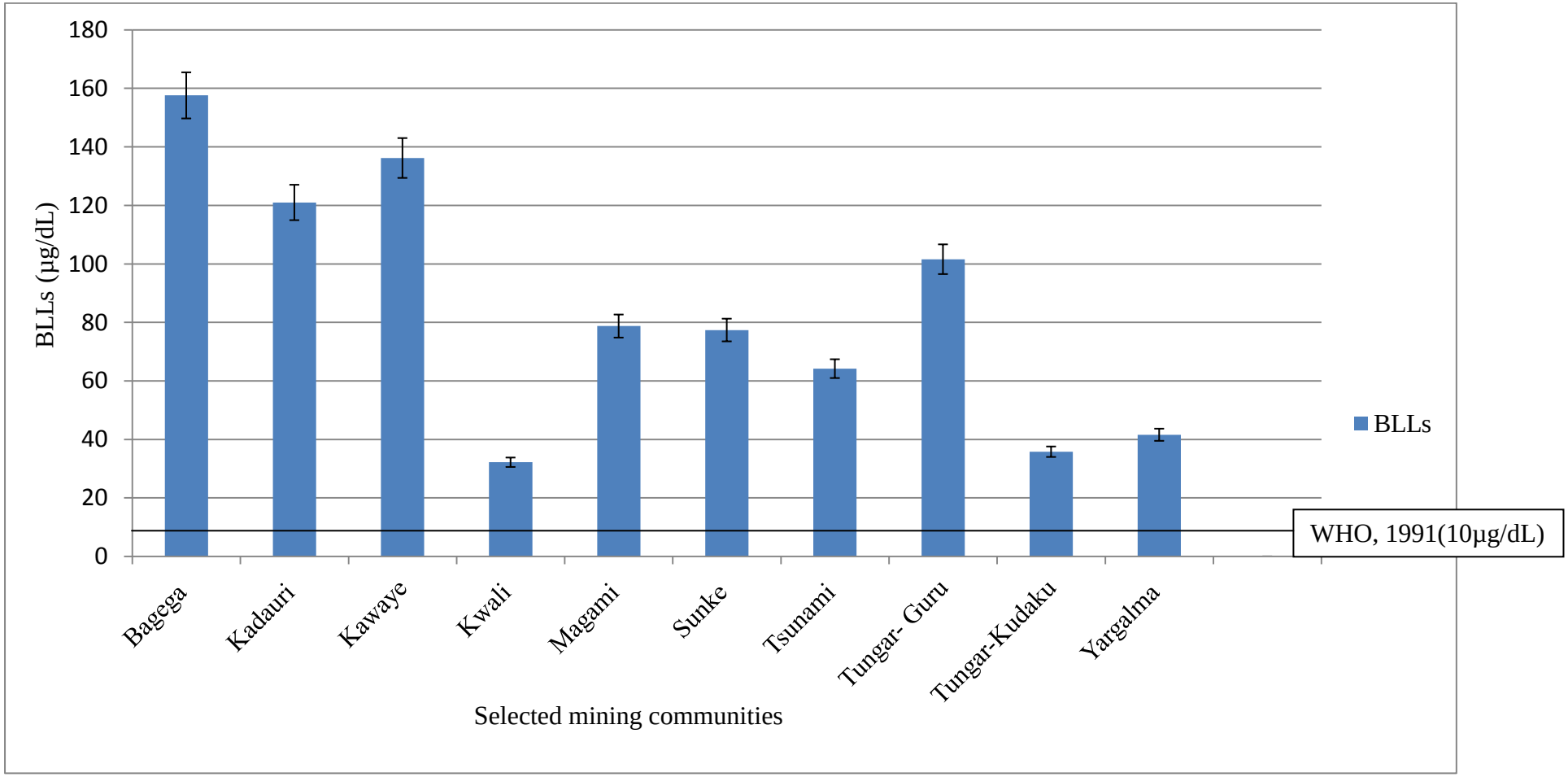


Figure 4.10: Blood lead levels (BLLs) in children from the selected mining communities

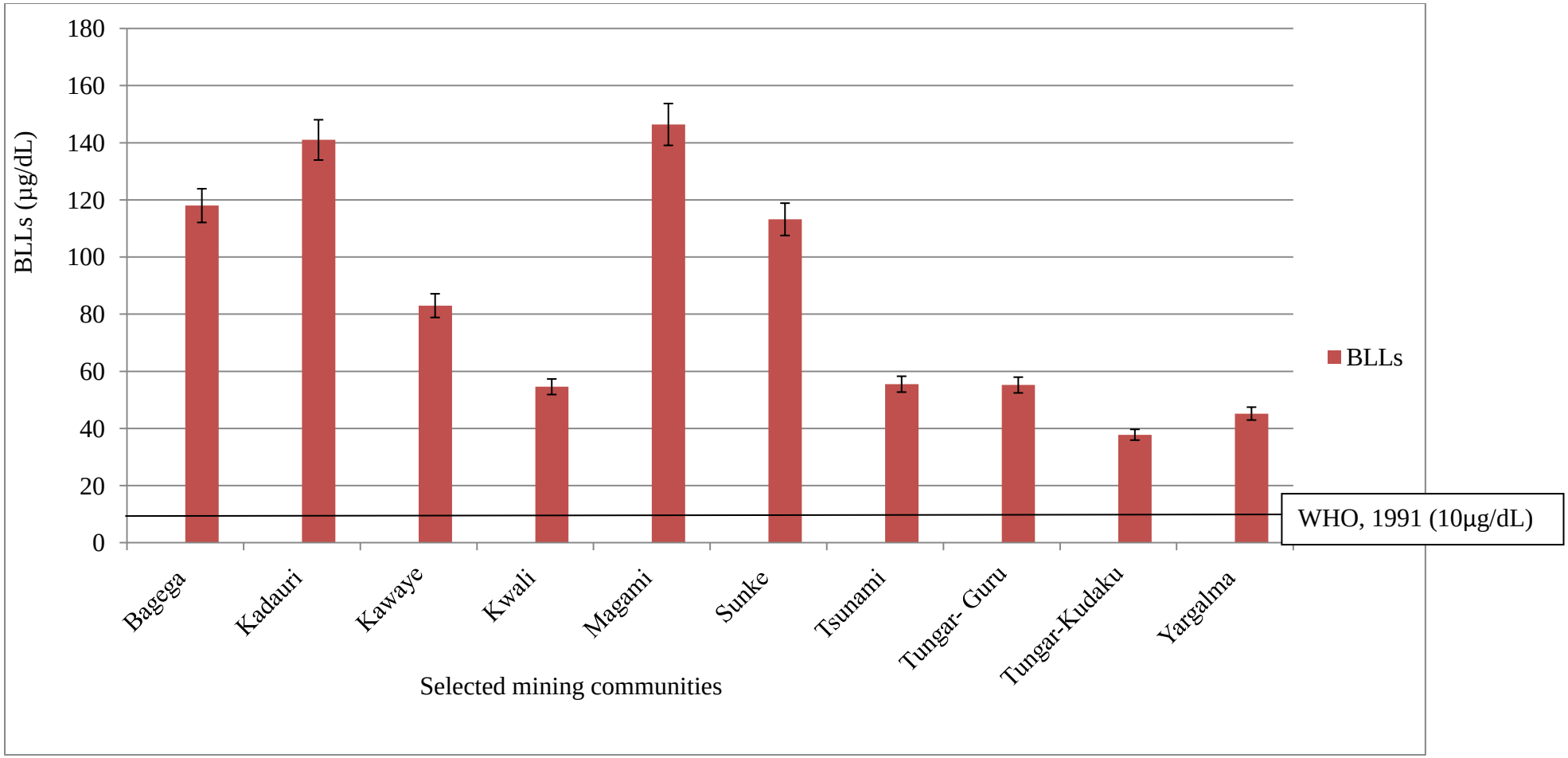


Figure 4.11: Blood lead levels (BLLs) in adults from the selected mining communities

4.7 Comparison of Lead Concentration between Children and Adults

Comparison of the mean blood lead concentration of children and adults indicated a similar trend with alternating peaks of lead levels between the children and adults, as the maximum concentration were found to be 157.60 ± 9.11 and 146.40 ± 27.40 μg/dL for children and adults respectively, as shown in Figure 4.12 and Appendix IX.

Statistical analysis indicated that there is no significant difference ($P < 0.05$) in comparison between the mean blood lead concentration of children and that of adults.

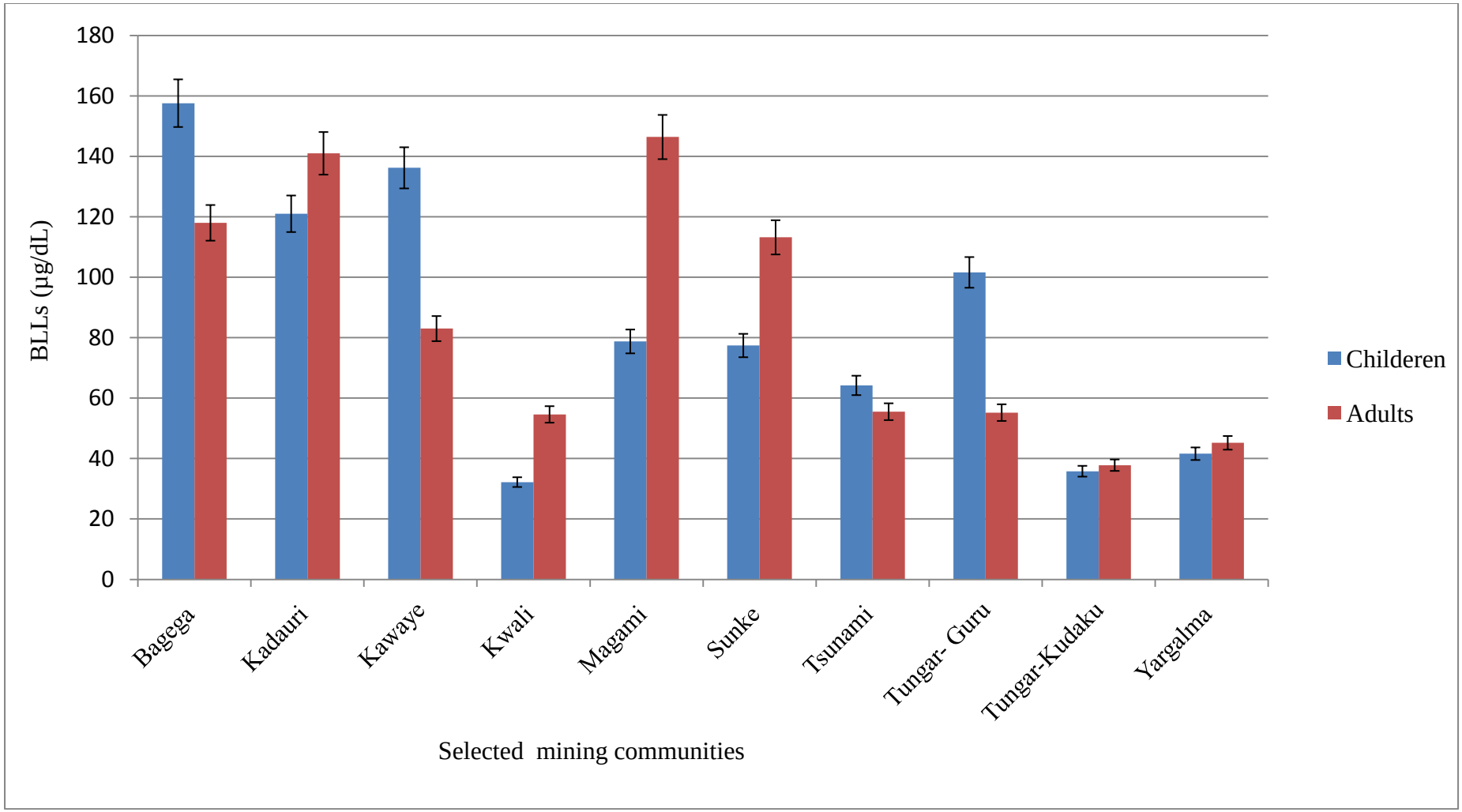


Figure 4.12: Comparison of blood lead levels (BLLs) in children and adults from the selected mining communities

4.8 Lead Concentration in Animals Blood Samples

The Mean blood lead concentration of the selected animals is shown in Figures 4.13 and Appendices X, XI and XII.

Statistical analysis indicated that significant difference ($P < 0.05$) exist in animal blood lead concentration both within community and across different communities.

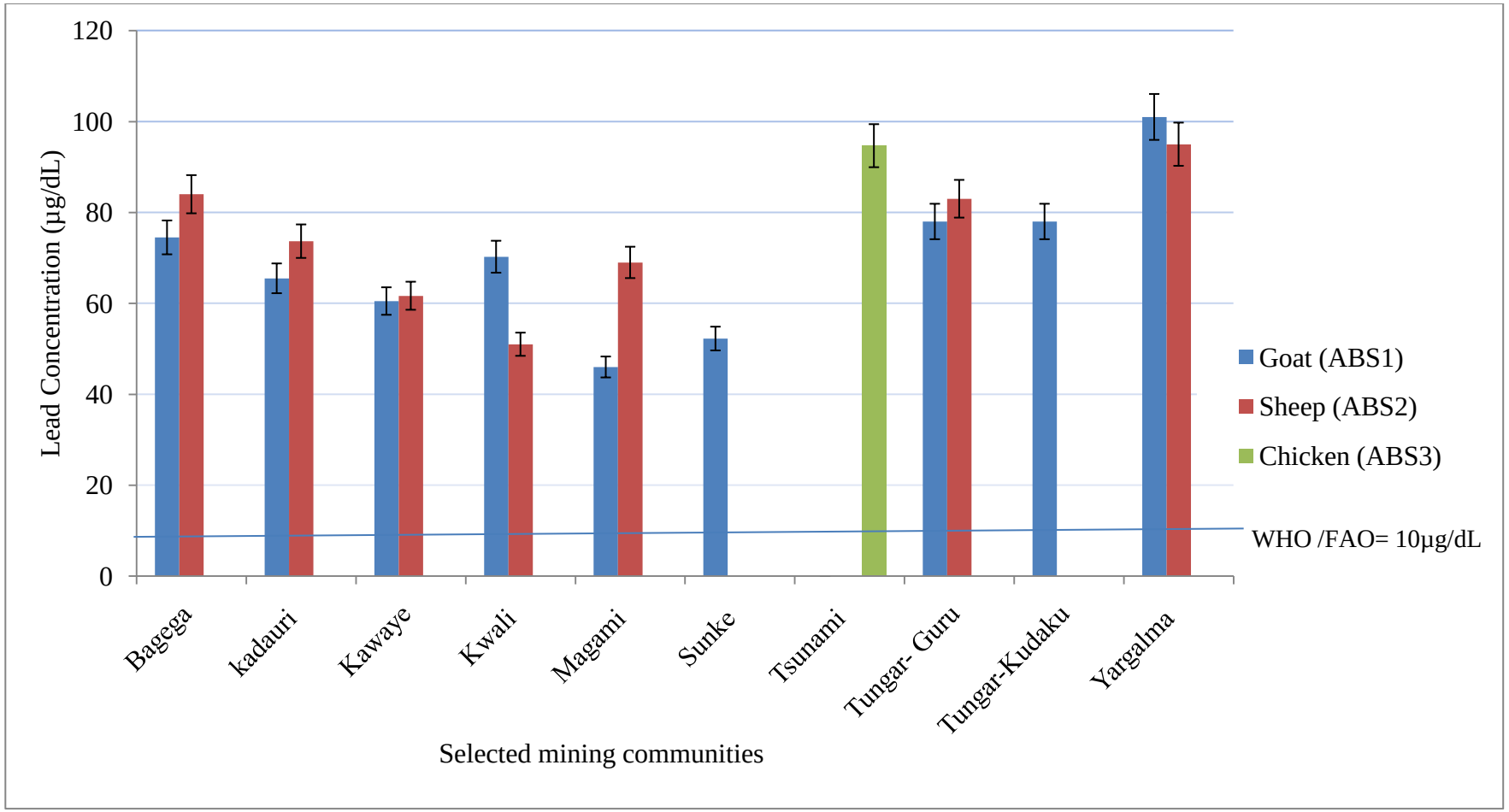


Figure 4.13: Comparison of mean blood lead levels in animals from the selected mining communities (µg/dL)

4.9 Lead Concentration in Underground and Surface water

The mean lead concentration for the underground water and surface water indicated serious contamination of water sources. The highest mean lead concentration for underground water was from Tsunami concentration and the lowest from Yargalma concentration; while the most contaminated surface water with high mean lead concentration was from Bagega concentration and the lowest was from Kadauri concentration. Comparism between the two sources of water indicated higher concentration of lead in surface water compared to underground water, as shown in Figures 4.14, 4.15 and 4.16 and Appendices XIII, XIV and XV respectively.

Statistical analysis indicated that there is significant difference ($P < 0.05$) in lead concentration in underground water within the communities, while in surface water there is no significant difference. This is because most of the communities share the same stream, lake or pond and such waters are involved in gold ore processing.

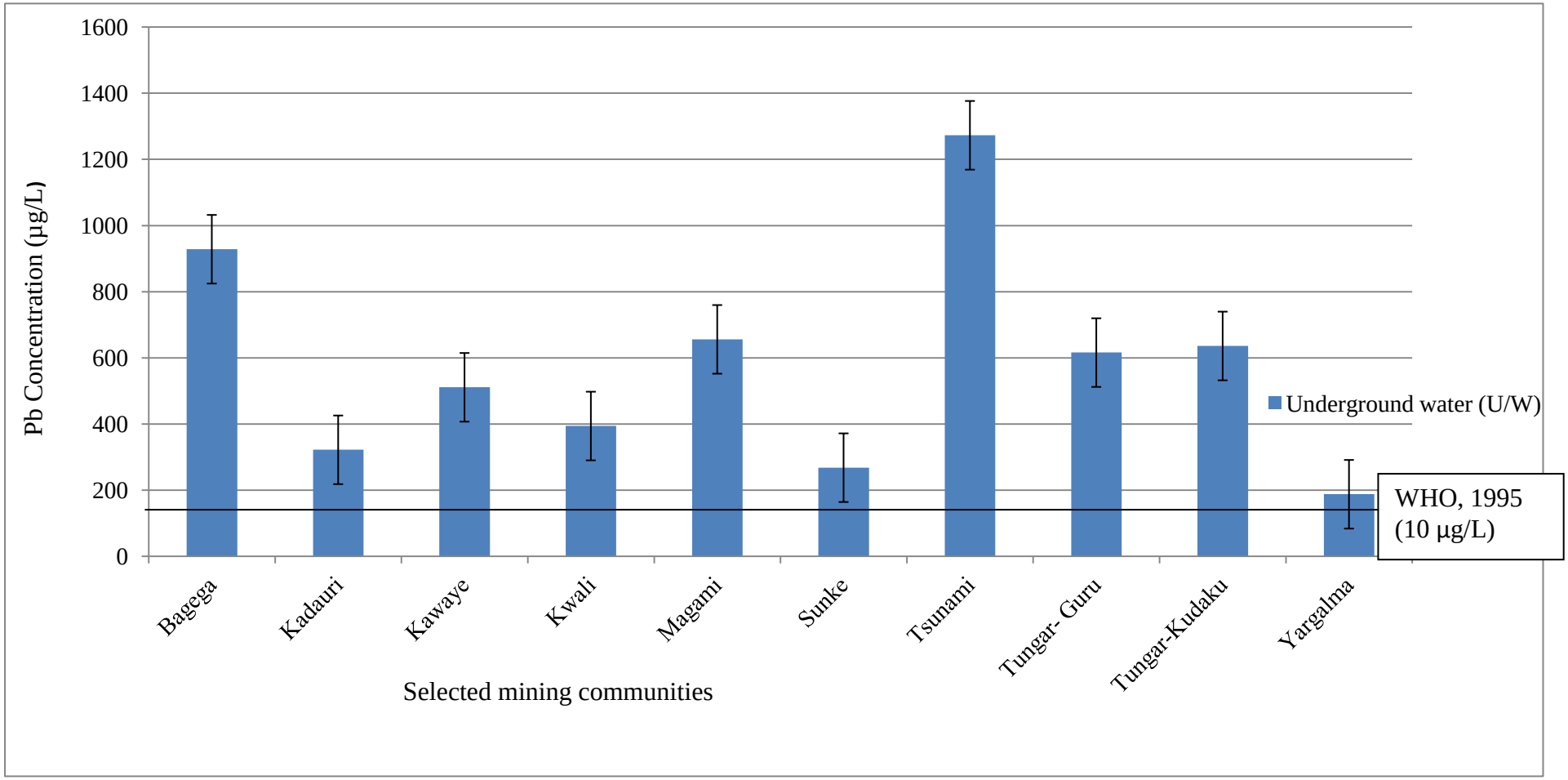


Figure 4.14: Mean lead levels (µg/L) in underground water (UW) from the selected mining communities

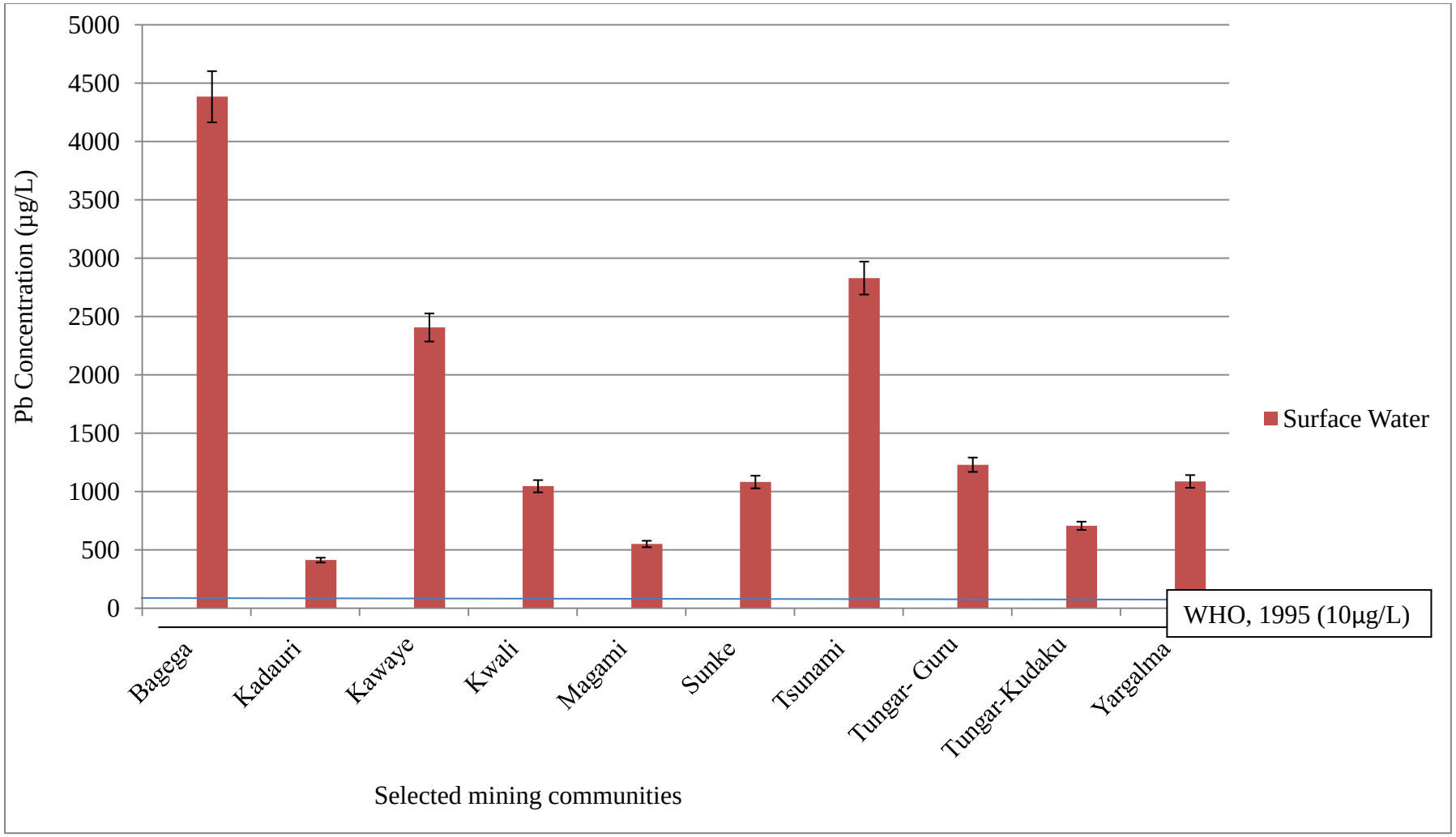


Figure 4.15: Mean lead levels (µg/L) in surface water (SW) from the selected mining communities

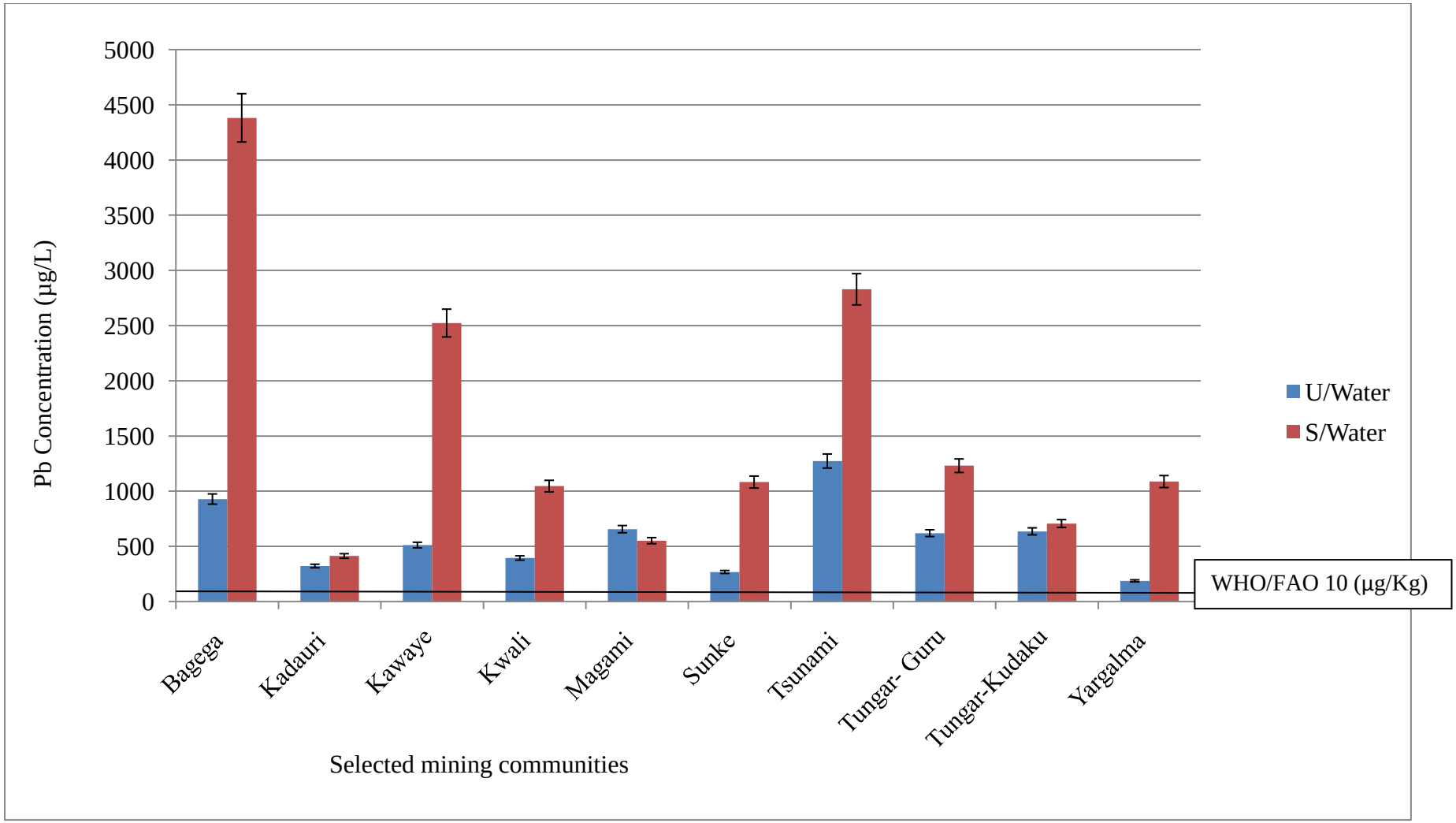


Figure 4.16: Comparison of lead levels in underground water & surface water

4.10 Demographic data of the Socio-Economic Opportunities of the Selected Mining Communities

The data collated and analysed indicated how the selected mining communities were not faring well in terms of education, occupational opportunities, and basic social amenities as presented in Table 4.2.

Table 4.2: Demographic Distribution of Socio-Economic Opportunities of the Selected Mining Communities

S/N.	Activity	Age Bracket (Years)	Gender (Male)	%	Gender (Female)	%
1.	EDUCATION		N=250		N=234	Total = 484
	a. Primary Pupils	6 – 12	109	44%	77	33%
	b. Secondary Students	12 – 18	63	25%	04	2%
	c. Tertiary Students	≥ 18	11	0.4%	0	0%
2.	OCCUPATION (Adults)	16 – 60	175	-	196	Total = 371
	a. Farming		175	100%	127	65%
	b. Gold mining/Processing		131	75%	-	-
	c. Public Servants		16	9%	-	-
	d. Other Businesses		59	16%	53	23%
3.	BASIC SOCIAL AMENITIES	Category	No. Available	%	NA.	Remark
	a. Source of drinking water	Well water/Bore hole	In all villages	70/30%		
	b. Health clinic	Primary Health care	Inadequate			MSF therapy clinics
	c. Schools	Primary Schools	Inadequate			

NA = Not Applicable. MSF = Medicines Sans Frontiers

CHAPTER FIVE

5.0 DISCUSSION

5.1 Lead Concentration in Residential Compound Soil Samples

There are similarities and significant differences in the Lead levels of the residential compound soils of the ten selected mining communities. An effort at remediation of the contaminated soils by government took place from September 2010 to December 2011 in eight villages, namely: Abare, Dareta, Sunke, Duza, Tungar-Daji, Tungar-Guru, Yargalma and Bagega. Four out of the eight villages involved in the remediation were part of the scope in this research, and these villages (Bagega, Sunke, Tungar-Guru and Yargalma) were observed to have higher lead levels among the ten selected mining communities, which perhaps justified their remediation (TerraGraphics, 2011c).

The results in Figure 4.5 and Appendix II indicated that, the villages remediated have the highest Lead concentration. Apart from Tungar-Guru with 757.69 ± 645.35 mg/Kg that requires another remediation, the lead levels in the other three villages below the tolerable limit of residential compound soil for the US-EPA of 400 mg/Kg.

Five out of the ten villages (Kadauri, Kawaye, Kwali, Magami and Tungar-Kudaku) (50%) have mean soil lead concentration of less than 20 mg/Kg; perhaps that was why they were not part of the soil remediation exercise. Statistical analysis indicated that the unremediated villages have mean soil lead levels that are significantly lower ($P < 0.05$) than the US-EPA threshold level (400 mg/Kg) of concern.

The significantly higher ($P < 0.05$) lead concentration in the remediated Tungar-Guru and the rest of the villages could be due to human factor in the dispersion of lead (Joint UNEP/OCHA, 2010).

Statistical analysis indicated that lead concentrations of the residential compound soil within the unremediated communities were significantly lower ($P < 0.05$) but significantly high deferent ($P < 0.05$) when compared across the rest of the remediated communities. Also equality of variance (F-test) indicated unequal variance between communities. The no significant difference within community was essentially because similar activity of ore processing (grinding, washing, smelting, etc) was taking place within same community. However, such activities vary with different intensity and exposure from one community to the other, which brought about the significant difference of lead concentrations across communities.

In spite of the remediation and the fact that the mean lead levels of Bagega, Sunke and Yargalma were below the US-EPA guideline, heavier lead levels were high and indicated potential danger for the inhabitants due to recontamination. Incidental ingestion of soil, particularly by children due to the oral phase of life, eating of lead pencils and eating food with dirty hands at tender age, could be a potential exposure route (Joint UNEP/OCHA, 2010).

The mean lead concentration indicated in Figure 4.5 and Appendix II has further justified the selection of the villages remediated in 2010 and 2011 remediation plan. The villages of Bagega, Sunke, Tungar-Guru and Yargalma were significantly ($P < 0.05$) higher in the mean soil lead concentration when compared to remaining six other villages (TerraGraphics, 2011).

It's important to note that the mining communities of Kadauri, Kawaye, Kwali, Magami, Tunami and Tungar-Kudaku not remediated had their mean lead concentration of residential soil ranged from 12.44 ± 3.23 - 124.36 ± 62.94 mg/Kg, which are significantly ($P < 0.05$) less than 400 mg/Kg, the US – EPA acceptable limit for lead in soil (TerraGraphics, 2011).

5.2 Lead Concentration in Sorghum, Millet and Maize Samples

The mean lead concentrations of the three common grains (Sorghum (GS1), Millet (GS2) and Maize (GS3) grown in the selected mining communities have indicated lead contamination, even though no significant difference ($P < 0.05$) in lead concentration among each grain was observed, except for a single significantly high ($P < 0.05$) concentration for the Millet from Kwali that was noted as shown in Figures 4.9 and Appendix VI. It was observed that the lowest mean lead concentration among the three grains was the millet from Magami (5.56 ± 2.02 mg/Kg). In all, the results indicated that the mean lead concentrations of all the grain samples have exceeded the 3.0 µg/Kg WHO/FAO threshold level of concern as shown in Figures 4.6, 4.7 and 4.8 and Appendices III, IV and V. Therefore, consumption of such grains from the selected mining communities constitutes a potential source of lead poisoning and health hazards. And these grains could find its way to various markets within and outside the state, which could further spread the health challenges from lead poisoning to the rest of other urban communities.

The role of human factor in the dispersal of lead from place to place has been further confirmed by the significantly high ($P < 0.05$) mean lead concentration in millet samples from Kwali (72.07 ± 57.36 mg/Kg).

A significant difference ($P < 0.05$) in lead concentrations determined in each type of grain within each mining community was obscured. However, the mean concentrations of lead in each type of grain were not significantly different ($P < 0.05$) across the ten mining communities.

In addition to other sources of lead exposure, the staple food of inhabitants of these mining communities now constitutes a potential source of lead poisoning due to

contamination either from the agricultural soil and water or in the processing and storage of the agricultural produce.

The lead levels in the grain samples were above the maximum WHO limits, and comparable to those observed in other studies. Yang *et al*. (1994) reported that cereals in China had lead levels of 0.06 mg/g. In Denmark, the National Food Agency established lead levels of 0.03 mg/g in cereals (National Food Agency of Denmark, 1992; Andersen *et al*., 1996). Another study by Urieta *et al*. (1996) found mean lead levels of 0.02 mg/g in cereals from Spain. In the United Kingdom, Ysart (1994) reported mean lead levels of 0.02 mg/ g in cereal products. In Poland, Krelowska-Kula (1991) found lead levels of 0.07 mg/g in cereals. In Japan Muramatsu *et al*. (1994) established lead concentrations of 0.05µg/g in wheat and rice.

5.3 Lead Concentration in Blood (BLLs) in Children and Adults

The mean blood lead concentrations for children and adults from all the selected mining communities ranged from 32.20 ± 7.33 to 157.60 ± 9.11 µg/dL were above the WHO (10µg/dL) tolerance limit as shown in Figures 4.10 and 4.11and Appendices VII and VIII. Only three (Kwali, Tungar-Kudaku and Yargalma) out of the ten villages have children with mean blood lead concentration less than 45µg/dL, which is the threshold level for chelating therapy treatment in children. At acute level children suffer from impairment of IQ and other cognitive effects, decreased heme synthesis, and interference in vitamin D metabolism (Lowry, 2010).

Statistical analysis for blood lead concentrations in both children and adult indicated significant difference ($P < 0.05$) within each community. However, there was no significant difference ($P < 0.05$) in the blood lead mean concentration across most of the communities, but comparisons between Bagega versus the rest of the communities

indicated significantly higher ($P < 0.05$) blood lead concentration compared to Kwali, Sunke, Tsunami, Tungar-Kudaku, and Yargalma. Comparisons between Kadauri versus Kwali, Tungar-Kudaku, and Yargalma also indicated significantly higher ($P < 0.05$) blood lead concentration. Equally, comparison between Kawaye versus Kwali, Tungar-Kudaku, and Yargalma indicated significantly higher ($P < 0.05$) blood lead concentration for children.

Comparisons in adults' blood lead mean concentration (37.80 ± 5.94 - 146.40 ± 27.40 µg/dL) between Kadauri versus some mining communities indicated significantly higher ($P < 0.05$) blood lead concentration compared to Kwali, Tungar-Guru, Tungar-Kudaku, and Yargalma. Also Comparisons between Kwali versus Magami, Tungar-Guru, Tungar-Kudaku, and Yargalma was not significantly higher ($P < 0.05$) in blood lead concentration. Overall comparison indicated that there was no significant difference ($P < 0.05$) between children and adult mean blood lead concentration.

It was also observed that only Yargalma out of the four remediated villages had children with mean BLL slightly less than 45µg/dL. The lead levels in the residential soil of the remediated villages have justified the reason why children from those villages have high lead concentration in their blood, and lead levels were still significant ($P < 0.05$) even after remediation. The existence of both adults and children in the same environment has subjected both to almost equal exposure to lead pollution and eventually lead poisoning (TerraGraphics, 2011).

Adults' involvement in either gold mining or processing made them agents of human transmission of lead from the mining and processing fields to their homes. Often, these adults' activities in mining were the potential routes of recontamination even after remediation. Comparison of the mean blood lead concentration have indicated that the

adults had higher mean blood lead level concentration in six out of the ten villages, while in the children mean blood lead concentration were higher in four out of the ten villages, as shown in Figure 4.12 and Appendix IX.

The NFELTP, (2013) preliminary report on Tsunami outbreak on lead poisoning in 2013 flood was accompanied with health challenges affecting both humans and animals with the children blood lead level (BLL) that require chelation ranged from 44.9 to 410.0 µg/dL. Animals BLL ranged from < 3.3 to169.4 µg/dL for Guinea fowl and chicken respectively. Residential soil contamination indicated that six out of eight compounds tested require remediation in children's sleep areas, which ranged from 464.4 to 4,145.7 ppm; while eight out of ten compounds require remediation in children's play areas that ranged from 497.7 to 3,932.3 ppm. These uncomfortable limits of concern would have resulted in serious health and environmental consequences in the Tsunami mining communities.

The deaths toll particularly among children as a result of lead poisoning in 2010, was the main cause of the global alert that attracted concern on health and environmental consequences, which the MSF described as' Unprecedented' Epidemic (MSF, 2012).

5.4 Blood Lead Concentration in Animals of the Selected Mining Communities

The mean blood lead concentrations of all the animals from the selected mining communities were above WHO (10µg/dL) levels of concern for animal blood samples. Goats and sheep from Yargalma have the highest mean blood lead concentration of 101 ± 9.07 and 95.00 ± 5.00 respectively. Livestock meat as a major source of protein in African society was equally a potential source of lead poisoning in view of the lead levels in the animals' blood samples as shown in Figure 4.13 and Appendix XII.

Statistical analysis indicated that there was significant difference ($P < 0.05$) in blood lead concentrations for goats, sheep and chicken both within community and across communities.

The lead poisoning transmission from the livestock to human host through consumption of meat of the affected animals is like direct ingestion of lead into the body system whether the host was involved in lead mining and/or processing or not. The major point of contamination of these animals is through both underground and surface water. All the villages involved in this research had their surface water contaminated through lead ore processing, and the livestock drink and bath from these contaminated ponds, lakes or rivers, and eventually gets ingested into their system. Results of the analysed samples from surface waters indicated high concentration of lead far above the FAO (100µg/L) acceptable limit for surface water. The result of lead level in chickens from Tsunami, who had an outbreak of lead poisoning in September – October 2013, also signified high concentration of lead contamination in their blood (NFELTP, 2013).

5.5 Lead Concentration in Underground Water (UW) and Surface Water (SW)

The overall analysis indicated very high lead concentration in both underground water and surface water. The mean lead concentration for underground water ranged from 187.67 ± 99.05 to 1273.00 ± 444.00 µg/L. For the surface water, the mean lead concentration ranged from 413.00 ± 202.43 to 4235.00 ± 121.75µg/L. These results indicated that, the mean lead concentration for both underground and surface water samples have exceeded the WHO (10µg/L) and FAO/WHO (100µg/L) acceptable guideline for underground and surface water as shown in Figures 4.14 and 4.15 and Appendices XIII and XIV respectively.

Statistical analysis indicated that there was significant difference ($P < 0.05$) in mean lead concentration in underground water. However, there was no significant difference ($P < 0.05$) in the mean lead concentration for the surface water. The insignificant difference for the surface water was because; the water was mostly engaged in similar activities of ore processing and domestic usage and stretched along many villages of the mining communities.

The underground water (UW) and surface water (SW) were some of the major sources of lead contamination in the mining communities, largely due to lead processing. Compounds with historical record of lead processing often had their underground well water contaminated with lead as no precautionary measure was observed during utilization, which led to the closure of many wells. Underground water source from bore holes were hardly contaminated except from external source (Joint UNEP/OCHA Environment Unit, 2010).

It should be noted that most of the water sources were not compound based like grains, but community based just like the surface water being a shared resource for the entire community. Comparison of lead concentration between the underground water and surface water indicated that mean lead concentration in surface water was generally higher due to ore processing activity of the mining communities as shown in Figure 4.16 and XV.

A distinct feature worth noticing was the high lead concentration of underground water in Tsunami, a location in Gusau Local Govt. Area. This high lead concentration might be due to the recent outbreak in lead poisoning in Unguwan Danbaba (Tsunami) due to flood in September – October 2013 (NFELTP, 2013).

It was reported by the Joint UNEP/OCHA Environment Unit, in September/October, 2010 on lead concentration in well waters in some of the remediated villages (Abare,

Kirsa, Sunke, Bagega and Dareta) indicated that, out of 76 well water tested, 28 (36.8%) were above the WHO guideline of 10 µg/L lead in drinking water. For surface water, out of 31 ponds, lakes and rivers tested, 21 (67.7%) were above the exposure limit (100 µg/L) suggested by FAO for Livestock (Joint UNEP/OCHA Environment Unit, 2010).

High concentration of lead (up to more than 1,000 µg/L – 10 times higher than the exposure limit suggested by FAO for livestock) were often found in ponds, rivers and lakes sampled by the mission (Joint UNEP/OCHA Environment Unit, 2010). Once lead falls onto the soil, it usually attaches to the soil, from where small amounts may leach into rivers and lakes and streams as the soil particles are moved by rainwater (Mahaffey *et al*., 1982).

5.6 Demographic Distribution of Socio-Economic Opportunities of the Selected Mining Communities

Wholesome analysis of the data obtained from the questionnaire on the demographic distribution of the socio-economic opportunities of the selected mining communities, has revealed the underdevelopment indices of the communities. Opportunities to education were in adequate and children enrolment into primary education was not up to 50%, while secondary education has only 25% of the children in school. Tertiary education was the lowest with less than 1% and in all cases female gender were at disadvantage. The gender difference particularly in terms of tertiary education and job opportunities in the public service was well pronounced.

Subsistence farming has been the main occupation of the mining communities with almost 100% participation, particularly in men; while about 65% of the women were involved in assisting their house hold in the farming activity and processing or produce. More than 75% of members of the holds were involved in mining activity, which

appeared a major source of income, but with devastating and incalculable consequences that spells doom for the future of the mining communities.

In terms of social amenities, portable drinking water, primary health care centres and schools were totally inadequate; where there are few, sometimes were the ones provided to the communities by the medical missions like the MSF. Most of the water sources (both underground and surface water) have already been contaminated with lead, except the bore halls. The absence of these facilities would continue to pose serious challenges to the public education and safety, safe mining practices and may deny the communities some of the driveable benefits of modern technology in exploration and mining.

CHAPTER SIX

6.0 SUMMARY, CONCLUSION AND RECOMMENDATIONS

6.1 Summary

The discovery of 'unprecedented' death toll as a result of lead poisoning in the mining communities of Zamfara State, Nigeria in 2010 during a medical mission on malaria by the MSF alerted the world's attention. The MSF team observed the absence of children in such a polygamous African society during this medical mission. The inhabitants reported the mysterious deaths of their children, which the mission later confirmed to be due high level of lead in the blood, due to gold mining using rudimentary tools.

Part of the measures taken was soil remediation of the mining communities in addition to chelation therapy, safe mining practices and public education. This research intended to determine whether the lead levels in the mining communities were still significant or not. The lead levels was to be determine from residential soil, common grains (sorghum, millet and maize), human (children and adults) and animal blood samples, underground and surface water. Samples were collected from ten selected mining communities, namely: Bagega, Kadauri, Kawaye, Kwali, Magami, Sunke, Tsunami/Ung. Danbaba, Tungar-Guru, Tungar-Kudaku and Yargalma.

Sampling sites and units were identified and their coordinates recorded using the Geographic Position System (GPS) instrument. The samples collected were pretreated stored and digested using appropriate digestion methods. Atomic Absorption Spectrophotometry (AAS) was used for the analysis using appropriate AAS protocol for the analysis of lead as the element of interest.

Results for residential soil samples analysed from the ten selected mining communities indicated mean lead concentration ranged from 12.44 ± 3.23 – 757.69 ± 645.35 mg/Kg. Out of the four communities involved in this research, the residential soil lead level has reduced, particularly from the soil remediated villages of Bagega, Sunke, and Yargalma, only Tungar-Guru (757.69 ± 645.35 mg/Kg) has lead level above the threshold level (400 mg/Kg) of concern that would require another remediation.

Statistical analysis indicated no significant difference ($P < 0.05$) in lead concentrations of residential soil within community, but there was significant difference ($P < 0.05$) in mean lead concentrations across communities.

Lead concentration in the common grains of the selected mining communities: Sorghum, Millet and Maize have indicated a threshold level of concern. The mean lead concentration of all the grains from other locations ranged from (5.56 ± 2.02 to 28.18 ± 5.93 mg/Kg), except for the millet from Kwali with the highest mean lead concentration (72.07 ± 57.36mg/Kg). Mean lead concentration in common grains indicated that the mean lead concentration of all the grain samples have exceeded the WHO threshold level (3.0 µg/Kg) of concern.

Similarly, the concentrations of lead in human (children and adults) and animal (Goat, Sheep and Chicken) blood samples analysed were obscured. BLLs exceeded the threshold level of concern of WHO and WHO/FAO (10 µg/dL) for human and animals blood sample. The blood lead level concentration for human ranged from 32.20 ± 7.33 to 157.60 ± 9.11 µg/dL, while in animals, BLL ranged from 52.25 ± 5.19 to 101 ± 9.07 µg/dL. It was observed that the animals BLLs were likely to be high as that of the surface water, where the gold ore was being processed, and domestic animals and livestock drink from the same source.

Statistical analysis for lead concentrations for each grain samples within each mining community was obscured, but mean lead concentrations across communities was not significantly different ($P < 0.05$) except for the millet from Kwali.

Lead concentrations in human (both children and adults) blood samples were significantly higher ($P < 0.05$) within community, however, significant difference ($P < 0.05$) was also observed in the mean blood lead concentrations across some communities (listed earlier). Overall comparison between children and adults indicated no significant difference ($P < 0.05$) in their blood lead levels.

The underground water analysed ranged from 187.67 ± 99.05 to 1273.00 ± 444.00µg/L and statistical analysis indicated significant differences ($P < 0.05$) in the mean lead concentrations in underground water within and across the communities. Surface water from ponds, lakes and rivers with had lead concentration higher than the FAO acceptable limit of 100 µg/L. Results further indicated that there was no significant difference ($P < 0.05$) in the mean lead concentration within communities, but significantly different ($P < 0.05$) across mining communities. Well water from Tsunami (in Gusau LGA) suffered lead poisoning outbreak as a result of flood in 2013; the results from this research has further confirmed the NFELTP, (2013) preliminary report on contamination of both underground and surface water. Tsunami had the highest mean lead concentration of underground water and second to only Bagega in mean lead concentration in surface water. This was unusually strange, as Tsunami (Unguwan Danbaba) in Gusau, being an urban settlement was not likely to have suffered from high level lead contamination if not for the outbreak caused by the flood (NFELTP, 2013).

The overall results indicated that, apart from few of the remediated villages whose residential compound soil lead concentration became lower than 400 mg/Kg, the rest of

the samples have lead concentration exceeding various thresholds levels of concern. This has indicated a serious health and environmental challenges to the selected mining communities and even the neighbouring communities. Often, meat and other agricultural products may find its way to the neighbouring communities, and beyond the State.

6.2 Conclusion

The research showed that in spite of the soil remediation and chelation therapy in children that had taken place in some of identified villages; the blood lead levels are generally significantly ($P < 0.05$) high, particularly in two of the villages (Bagega and Tungar-Guru). Other samples analysed had also high lead concentrations that were above internationally accepted threshold level of concern. Water contamination (both underground and surface) as one of the major sources of exposure through ore processing requires a lot of preventive measures. Lead contamination of grains and other staple foods due to handling and soil contamination should be a major source for concern as a result of daily dietary intake that is above FAO (3.0 µg/Kg) and EFSA threshold level of concern, due to these sources of exposure.

Overall results indicated there were no significant differences ($P < 0.05$) in lead concentrations of residential soils within community, but significantly high difference ($P < 0.05$) in mean lead concentration across the communities. Lead concentration for the grains was obscured. However, there was no significant difference in lead concentration in grain samples within community, and in mean lead concentration across each community, except with the millet sample from Kwali. There was also significantly high difference ($P < 0.05$) in the blood lead concentration of both children and adults within community, however, no significant difference ($P < 0.05$) across

communities for children and adults. Comparison between blood lead levels of children and those of adults indicated no significant ($P < 0.05$) difference. Equally, analysis of underground and surface water indicated significant differences ($P < 0.05$) within community and not significant across communities in the lead levels of underground water. In surface water, no significant difference within community, but significant difference exists across the communities. The overall results indicated that lead concentrations in all the tested samples were above various threshold levels of concern of the International standards. A lot still needs to be done in the area of public education, enforcement of relevant laws, safer mining practices, and general improvement of the socio-economic status of the of mining communities in order to save them from health and environmental degradation.

6.3 Recommendations

This research was essentially concentrated in selected mining communities with parameters having direct bearing on the life of communities, like underground water, soil, surface water, grains, human and animal blood samples. In view of the various exposure routes for lead contamination in the mining communities and likely variation in the lead concentrations of the parameters, the following are hereby recommended;

- Periodic analyses of samples in order to determine variation in the lead levels and ensure concentration are always within acceptable tolerance limit of FAO/WHO.
- An in-depth monitoring of blood lead Levels (BLLs) in children and assessment of chelation therapy in all the mining communities.

- Lead level assessment of grains and domestic animals for both local and commercial scale consumption
- Renewed effort in remediation of contaminated soil in unremediated mining communities.
- Total commitment to training and retraining of miners on safer mining practices.
- Special Health Insurance for field and laboratory workers involved in lead poisoning treatment and remediation.

.REFERENCES

Abbey, D.E. Nishino, N. McDonnell, W.F. Burchette, R.J. Knutsen, S.F. Lawrence Beeson, W. Yang, J.X. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *American Journal of Respiratory Criteria Care Medicine*; 159 (4): 373–82.

Albalak, R.R. Hart McElroy, G. Noonan, S. Buchanan, R.L. Jones, W.D. Flanders, C. Gotway-Crawford, D. Kim, T. Dignam, W.R. Daley, J. Jarrett, E. Eduardo, M..A. McGeehin, (2003). Blood lead levels and risk factors for lead poisoning among children in a Mexican smelting community. *Archive of Environmental Health,* 58 (3): 172–183.

Alexander, F.W. Clayton, B.E. and Delves, H.T. (1974). Mineral and trace-metal balances in children receiving normal and synthetic diets: *Quarterly Journal of Medicine*, 43 (5): 89-111.

Alfven, T. Elinder, C.G. Carlsson, M.D. Grubb, A. Hellstrom, L. Persson, B. (2000). Low-level cadmium exposure and osteoporosis. *Journal of Bone Miner Resources*; 15 (2): 1579–86.

Andersen, N.L. Fragt, S. Groth, M.V. Hatkopp, H.B. Moller, A. Ovesen, L. Warming, D.L. (1996). Dietary Habits in Denmark Main Results. Publication No. 235. *National Food Agency*, Denmark. 165 -172.

Annest, J.L. (1983). Trends in the blood level leads of the US population: In:The Second *National Health and Nutrition Examination Survey* (NHANES II) 1976–1980. Rutter M, Jones RR (eds). Lead Versus Health, John Wiley & Sons, New York, 33–58.

Aposhian, H. Maiorinoj, R. Rivera, M. (1992). Human studies with the chelating agents, DMPS and DMSA. *Journal for Toxicology and Clinical Toxicology*, 30:505-528.

ATSDR, (2007). (United States Agency for Toxic Substances and Disease Registry), Toxicological Profile for lead. *U.S. Department of Health and Human Services*, 1-582.

Beeson, W.L. Abbey, D.E. Knutsen, S.F. (1998). Long-term concentrations of ambient air pollutants and incidence lung cancer in California adults: results from the AHSMOG study. *Environmental Health Perspective*; 106: 813–23.

Bellinger, D. Stiles, K.M, Needleman, H.L. (1992). Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study. *Pediatrics*, 90: 855–861.

Belyaeva, O.N. Haynes, R.J. Birukova, O.A. (2005). Barley yield and soil microbial and enzyme activities as affected by contamination of two soils with lead, zinc or copper. *Biology and Fertility of Soils*, 41: 85–94.

Berglund, M. Elinder, C.G. Järup, L. (2001). Humans Exposure Assessment. *An Introduction.* WHO/SDE/OEH/01.3 (Accessed 20/.6/2015).

Birge, W. J. and Price , D. J. (1998): Analysis of Metals in Red-tailed Hawk Blood From PGDP. School of Biological Sciences, University of Kentucky. F*inal Report*, May 28, 1998. 1- 4.

Blacksmith Institute (2010). World´s Worst Pollution Problems Report (WWPR) 2010: 31- 76.

Boontherawara, N. Paisarnutpong, O., Panich, S., Phiu-Nual, K. and Wangwongwatana, S. (1994). Traffic crisis and air pollution in Bangkok. *Thailand Environment Institute Quarterly Environment Journal*, 2: 4–36.

Broeckaert, F. Arsalane, K. Hermans, C. Bergamaschi, E. Brustolin, A. Mutti, A. Bernard, A. (2000). Serum claracell protein: a sensitive biomarker of increased lung epithelium permeability caused by ambient ozone. *Environmental Health Perspective*, 108: 1533–7.

Bull, B.S. (2006). Morphology of the Erythron in Williams Hematology, edited by Marshall A. Lichtman *et al*. 7th ed. *New York Mc Graw-Hill Medical*. Pp 375.

Burau, R. E. (1982), 'Lead', in A. L. Page *et al*. (eds), *Methods of Soil Analysis, Part 2, Chemical and Microbiological Properties*, 2nd ed., Agron. Monogr. Vol. 9, ASA and SSSA,Madison, WI, U.S.A., pp. 347–366.

Byers, R.K. Lord, E. (1943). Late effects of lead poisoning on mental development. *American Journal of Diseases of Children*, 66: 471–494.

Caldas, E. D. and Machado, L. L. (2004). Cadmium, mercury and lead in medicinal herbs in Brazil. *Food and Chemical Toxicology*, 42: 599-603.

CDC (1991) (Centres for Disease Control and Prevention) Preventing lead poisoning in young children: a statement by the Atlanta, GA, Centres for Disease Control and Prevention.12: 115-127.

CDC (2001) (Centres for Disease Control and Prevention) Fatal paediatrics lead poisoning–New Hampshire, MMWR Morb. Mortal 6: 15-29.

CDC (2002) (Centres for Disease Control and Prevention) Preventing Lead Poisoning in Young Children, Atlanta: U.S. Department of Health and Human Services 2002. Available at http://www.cdc.gov/nceh/lead/publications/PrevLeadPoisoning.pdf. Accessed August 10, 2009.

CEOHA (1996). (Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society). Health effects of outdoor air pollution. *American Journal of Respiratory Critical Care Medicine*; 153: 3–50 and 477–98

Chander, K., Dyckmans J., Joergensen R.G.J., Meyer B.G., Raubuch M. (2001): Different sources of heavy metals and their long-term effects on soil microbial properties. *Biology and Fertility of Soils*, **34**: 241–247.

Chau, JY, Grunseit AC, Chey T, Stamatakis E, Brown WJ, Mathew CE (1979). Analytical Methods for Determining Lead in Biological Materials: *Analytical methods* (7), 390 – 398.

Chisholm, J.J Jr. (2000). Safety and efficacy of meso-2, 3-dimercaptosuccinic acid (DMSA) in children with elevated blood lead concentrations. *Journal of Toxicology and Clinical Toxicology*; 38: 365 – 75

Czarnowska, K., Gworek B., (1987) .Rocz. Nauk Roln., 107 (2), 23,

Dart, RC, Hurlbut KM, Maiorino RM, *et al*. (1994). Pharmacokinetics of meso-2,3-dimercaptosuccinic acid in patients with lead poisoning and in healthy adults. *Journal of Paediatrics*; 125: 309-316.

DETR, (2001) (Department of the Environment, Transport and the Regions) *Statistics Release 184 1999 UK Air Emissions Estimates*, (28 March 2001)

Dudka, S., Piotrowska M, Matysiak Z, Witek T., P. (1995) *Journal of Environmental Studies*, 4, (3), 9.

Edwards-Bert P, Calder IC, Maynard EJ. (1994). National review of public exposure to lead in Australia. Adelaide, *South Australian Health Commission*.

EFSA, (2010). European Food Safety Agency (2010) Panel on Contaminants in the Food Chain (CONTAM) *Journal for Scientific Opinion on Lead in Food*: 8(4):1570

Ellen, G. and Van, Loon J. W. (1990). Analytical Methods for Determining Lead in Biological Materials: *Analytical method* (7): 190 (1). 34 – 9.

Elwood, P C. (1982) Randomised controlled trials, sampling: *British Journal of Clinical Pharmacology*, 3: p631-636.

EMEP (2001). (European Monitoring and Evaluation Programme),. Cyromazine summary report (2).Committee for veterinary medicinal products. European Agency for the Evaluation of medicinal products, veterinary medicines and inspections (EMEA). EMEA/MRL/770/00-FINAL.12: 22-45.

EMEP (2006). (European Monitoring and Evaluation Programme),. Cyromazine summary report (9).Committee for veterinary medicinal products. European Agency for the Evaluation of medicinal products, veterinary medicines and inspections (EMEA). EMEA/MRL/770/00-FINAL.18: 110-140.

Ericson, JE, Shirahata H, Patterson CC. (1979). Skeletal concentrations of lead in ancient Peruvians. *New England Journal of Medicine*, 300: 946–951.

Ertas, O.S and Tezel, H. (2004). A validated cold vapour-AAS method for determining mercury in human red blood cells. *Journal of Pharmaceutical and Biomedical Analysis* 36: 893–897.

Eurachem guide. (1998). The fitness for purpose of analytical method: A laboratory guide to method validation and related topics. Downloaded from: *http://www.eurachem.org/guides/pdf/valid.pdf. 18/04/2011*.

EC, (2006) European Commission Regulation , No 1881/2006 of 19thof Dec. 2006. Setting maximum levels for certain contaminants in foodstuffs, *Official Journal of European Union*, L364: 5-24.

Ezzati, M, Lopez AD, Rodgers A, Hoorn SV, Murray CJ. (2002). Selected major risk factors and global and regional burden of disease. *Lancet* 2002; 360: 1347–60.

Fewtrell, L, Kaufmann R, Prüss-Üstün A (2003). Lead*: Assessing the environmental burden of disease at national and local levels*. Geneva, World Health Organization (Environmental Burden of Disease Series, No. 2; http://www.who.int/quantifying_ehimpacts/publications/en/leadebd2.pdf).

Filho, Hélcio José Izário, Rodrigo Fernando dos Santos Salazar, Maria da Rosa Capri, Ângelo Capri Neto, Marco Aurélio Kondracki de Alcântara and André Luís de Castro Peixoto (2012). State-of-the-Art and Trends in Atomic Absorption Spectrometry, Atomic Absorption Spectroscopy, Dr. Muhammad Akhyar Farrukh (Ed.), ISBN: 978-953-307-817-5, In Tech, Available from: http://www.intechopen.com/books/atomic-absorptionspectroscopy/ state-of-the-art-and-trends-in-atomic-absorption-spectrometry.

Flegal, AR, Smith DR. (1986). Current needs for increased accuracy and precision in measurements of low levels of lead in blood. *Environmental Research*, 58: 125–133.

Flegal, AR, Smith DR. (1995). Lead levels in preindustrial humans. *New England Journal of Medicine*, 326: 1293–1294.

Frankowski, M., Sojka M., Frankowska A.Z., Siepak M., Blazejewska S.M. (2009): Distribution of heavy metals in Mala Welna River system (western Poland). *Oceanological and Hydrobiological Studies*, 38: 51–61.

Friberg, L. (1950). Health hazards in the manufacture of alkaline accumulators with special reference to chronic cadmium poisoning. *Acta Med Scand*; Suppl 240: 1–124

Galkwad Amurta, (2011). Water pollution, causes and effects. *Published article*.

Galušková, I., Borůvka L., Drábek, O. (2010): Urban soil contamination by potentially risk elements. *Soil and Water Research*, 6: 55–60.

Garcia-Rico, G., Leyva-Perez, J. and Jara-Marini, M.E. (2007). Content and daily intake of copper, zinc, lead, cadmium, and mercury from dietary supplements in Mexico. *Food and Chemical Toxicology* 45: 1599–1605.

Garcia, R., R. Belmont, H. Padilla, M. C. Torres and A. Báez. (2009). "Trace metals and
inorganic ions measurements in rain from Mexico City and a nearby rural area". *Reviewed Chemistry and Ecology*. 25(2), pp. 71-86.

George Foundation.(1999). Project lead-free: a study of lead poisoning in major Indian cities. In: Proceedings of the International Conference on Lead Poisoning, Bangalore, India, 8–10 February. Bangalore, *The George Foundation*, 1999: 79–86.

Gibson, J. L. (1904). A plea for painted railings and painted walls of rooms as the source of lead poisoning among Queensland children. *Australian Medical Gaz*ette, , 23: 149–153.

Gonzalez, A.G. and Herrador, M.A., (2007) A Practical Guide to Analytical Method Validation, Including Measurement Uncertainty and Accuracy Profiles, Trends in Analytical Chemistry 26 (3): 227-238.

Graziano, J.H, Lolacono N.J, Meyer P.(1988). Dose-response of oral 2,3-imercaptosuccinic acid in children with elevated blood lead concentrations. *Journal Paediatrics*; 113: 751-757.

Harper, W M. (1976). Statistics 2nd edition M & E Handbooks, Macdonald and Evans, Plymouth.

Heard, M.J and Chamberlain AC, (1982). Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. *Human Toxicology*, 1, 411-415.

Hogstrand, C., F. Galvez, and C.W. Wood. (1996). Toxicity, silver accumulation and metallothionein induction in freshwater trout during exposure to different silver salts. *Environmental Toxicology Chemistry*. 15:1102-1108.

Holland, W. W, Bennett AE, Cameron IR, Florey C duV, Leeder SR, Schilling RSE, Swan AV, Waller RE. (1979). Health effects of particulate air pollution: reappraising the evidence. *American Journal of Epidemiology*; 110: 527–659.

Horak, F, Studnicka M, Gartner C, Spengler JD, Tauber E, Urbanek R, Veiter A, Frischer T. (2002). Particulate matter and lung function growth in children: a 3-yr follow-up study in Austrian schoolchildren. *European Respiratory Journal*; 19: 838–45.

Horwitz, W, (1982). Evaluation of analytical methods used for regulations of foods and drugs. *Analytical Chemist*, 54: 67A – 76A.

Hossn, E, Mokhtar G, El-Awady M, Ali I, Morsy M, Dawood A. (2001). Environmental exposure of the pediatric age groups in Cairo City and its suburbs to cadmium pollution. *Science Total Environment*; 273: 135–46.

Huber, L. (1998). Validation and Qualification in Analytical Laboratories. Interpharm Press. East Englewood CO, USA.

Hursh, JB and Mercer TT. (1970). Measurement of Pb-212 loss rate from human lungs. *Journal of Applied Physiology*, 28, 268-274.

Hursh, JB, Schraub A, Sattler EL and Hofmann HP. (1969). Fate of 212Pb inhaled by human subjects. *Health Physics*, 16, 257-267.

IARC (1993) International Agency for Research on Cancer. Cadmium and cadmium compounds.. In: Beryllium, Cadmium, Mercury and Exposure in the Glass Manufacturing Industry. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. **58**. Lyon: *International Agency for Research on Cancer*; 119–237.

IARC (2006) International Agency for Research on Cancer. Inorganic and Organic Lead Compounds. IARC Monographs on the Evaluation of Carcinogenic Risks to Human. 87, 519.

ICH (1994) (International Conference on Harmonisation).. Validation of Analytical Procedures: Text and Methodology. Downloaded from: *http://www.ich.org/fileadmin/Public/Web_Site/ICH_Products/Guidelines/Quality/Q2_R1/Step4/Q2_R1_Guideline.pdf., 19/04/2011*.

IPCS (1995). *Inorganic lead*. Geneva, World Health Organization, International Programme on Chemical Safety (Environmental Health Criteria 165; http://www.inchem.org/documents/ehc/ehc/ehc165.htm).

James, H.M, Hilburn ME and Blair JA. (1985). Effects of meals and meal times on uptake of lead from the gastrointestinal tract in humans. *Human Toxicology*, 4, 401-407.

Järup, L. Berglund M, Elinder CG, Nordberg G, Vahter M. (1998). Health effects of cadmium exposure— a review of the literature and a risk estimate. *Scand J Work Environ Health*; 24 (Supplementary 1): 1–51.

Järup L. Hellstrom L, Alfven T, Carlsson MD, Grubb A, Persson B *et al.* (2003) Low level exposure to cadmium and early kidney damage: the OSCAR study. *Occupational Environment Medicine*; 57: 668–72.

Järup, L. (2003). Hazards of heavy metal contamination. *British Medical Bulletin*; 68: 167–182.

Joint UNEP/OCHA Environment Unit (2010): Lead Pollution and Poisoning Crisis, *Environmental Emergency Response Mission, Zamfara State, Nigeria.* P 21-23.

Kabata-Pendias A, Pierwiastki Śladowe W Środowisku Biologicznym", Wyd. Geologiczne, Warszawa, (1979) *Polish Journal of Environmental Studies*. Vol. 10, No. 2 (2001), 123-126.

Kabata-Pendias A, Pendias H, Biogeochemia Pierwiastków Śladowych", PWN, Warszawa, (1993) *Polish Journal of Environmental Studies*. Vol. 10, No. 2 (2001), 123-126.

Katsouyanni K, Touloumi G, Samoli E. (2001). Confounding and effect modification in the shortterm effects of ambient particles on total mortality: Results from 29 European cities within the APHEA2 Project. *Epidemiology*; **12**: 521–31.

Kawai M., H. Toriumi, Y. Katagiri, Y. Maruyama, (1983). Home lead work as a potential source of lead exposure for children. *International Archive for Occupational and Environmental Health* 53: 37–46.

Kazantzis G. (1989). Lead: ancient metal—modern menace? In: Smith MA, Grant LD, Sors AI, eds. Lead exposure and child development: an international assessment. *Lancaster,* England, MTP Press: 119–128.

Kevin Telmer (2011). How to bring safer mining practices and a model of formalization to Artisanal and Small Scale Gold Mining to eliminate lead and mercury poisoning in Anka and Bukkuyum LGAs, Zamfara State, Nigeria. In: *Emergency assessment and recommendations*, Pp 1 – 20.

Kolonel, L.N. (1976). Association of cadmium with renal cancer. *Cancer*; 37: 1782–7.

Koplan, J.P., A.V. Wells, H.J. Diggory, E.L. Baker, J. Liddle, (1977). Lead absorption in a community of potters in Barbados, *International Journal Epidemiology*. 6: 225–229.

Korn, MG, de Andrade JB, de Jesus DS, Lemos VA, Bandeira ML, Dos Santos WN, Bezerra MA, Amorim FA, Souza AS and Ferreira SL, (2006). Separation and pre-concentration procedures for the determination of lead using spectrometric techniques: *Journal European Food Safety Authority*: *A review. Talanta,* 69: 16-

Krelowska –Kula, M (1991). Metal contents in certain food products. Die Nehrung; 35:363-367.

Kuruvilla, A., V.V. Pillay, T. Venkatesh, (2004). Portable lead analyzer to locate sources of lead, *Indian Journal of Paediatricians* 41: 495–499.

Lanphear, BP, Hornung R, Khoury J, Yolton K, Baghurst P, *et al*. (2005). Low-level Environmental LeadExposure and Children's Intellectual Function: An International Pooled Analysis. *Environmental Health Perspectives.*113: 894-899.

Larsen, EH, Andersen NL, Moller A, Petersen A, Mortensen GK and Petersen J, (2002). Monitoring the content and intake of trace elements from food in Denmark. *Food Additives & Contaminants*, 19, 33-46.

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

Lovei, M., (1999). Eliminating a silent threat: World Bank support for the global phase out of lead from gasoline, in: A.M. George (Ed.), Lead Poisoning Prevention and Treatment: Implementing a National Program in Developing Countries, The George Foundation, Bangalore, India, pp. 169–180.

Lowry, Jennifer A. MD (2010). Oral Chelation Therapy for Patients with Lead Poisoning: Division of Clinical Pharmacology and Medical Toxicology. *The Children's Mercy Hospitals and Clinics Kansas City, MO 64108*.

Mahaffey, K.R, Annest JL, Roberts J, Murphy RS (1982). National estimates of blood lead levels: United States, 1976-1980; association with selected demographic and socioeconomic factors. New English. *Journal of Medicine.* 307: 537-579.

Maiorino, R, Bruce D, Aposhian H. (1989). Determination and metabolism of dithiol chelating agents. VI. Isolation and identification of the mixed disulfides of meso-2,3-dimercdatosuccinic acid with L-cysteine in human urine. *Toxicology and Applied Pharmacology*, 97:338-349.

Makokha, A O., Leonard R. Mghweno, Happy S. Magoha, Amina Nakajugo and John M. Wekesa. (2008). Environmental lead pollution and contamination in food around Lake Victoria, Kisumu, Kenya. *African Journal of Environmental Science and Technology* Vol. 2 (10). pp. 349-353.

Matte, T.D., J.P. Figureueroa, S. Ostrowski, G. Burr, L. Jackson-Hunt, E.L. Baker, (1991). Lead exposure from conventional and cottage lead smelting in Jamaica, *Archive of Environmental Contamination Toxicology*. 21: 65–71.

Mayan, O.N., A.T. Henriques, J.M. Calheiros, (2001). Childhood lead exposure in Oporto, Portugal, *International Journal Occupational and Environmental Health* 7: 209–216.

McConnell, R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, Avol E, Margolis HG, Peters JM. (2002). Asthma in exercising children exposed to ozone: a cohort study. *Lancet*; 359: 386–91.

McDonnell, WF, Abbey DE, Nishino N, Lebowitz MD. (1999). Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG Study. *Environmental Resources*; 80: 110–21

MSF (2012) (Medicines San Frontiers') Lead Poisoning Crisis in Zamfara State Northern Nigeria: *MSF Briefing Paper*, May 2012. P 1-4(Accessed June, 2012)

Meyer, Pamela A., Mary Jean Brown, (2008). Henry Falk Global approach to reducing lead exposure and poisoning. Mutation Research; 659: 166–175

Mielke, H. (1999). Atomic Absorption Determination of Lead in Soil Samples: *American Scientist*, *87*, 62-73.

Morrow, PE, Beiter H, Amato F and Gibb FR, (1980). Pulmonary retention of lead: an experimental study in man. *Environmental Research*, 21, 373-384.

Muramatsu, Y. Sumiya, M. Yanasigawa, K. Ohmomo, Y. (1994). Trace and Toxic elements in foodstuffs. In: Nuclear Techniques for Toxic Elements in Foodstuffs. *Report on an IAEA Co-ordinated Research Programme*. Vienna, pp.156-172.

Mwegoha, W. J. S. and Kihampa C. (2010): Heavy metal contamination in agricultural, soil and water in Dar es Salaam city, Tanzania. *African Journal of Environmental Science and Technology* Vol. 4(11), pp. 763-769.

National Food Agency of Denmark (1992). Food Monitoring 1988 - 1992, Denmark.

Neas, LM, Dockery DW, Ware JH, Spengler JD, Speizer FE, Ferris BG. (1991). Association of indoor nitrogen dioxide with respiratory symptoms and pulmonary function in children. *American Journal of Epidemiology*; 134: 204–9

Nichani, V., L. Wan-I, M.A. Smith, G. Noonan, M. Kulkarni, M. Kodavor, L.P. Naeher, (2006) Blood lead levels in children after phase-out of leaded gasoline in Bombay, India, *Science. Total Environment* 363: 95–106.

NFELTP (2013),(Nigeria Field Epidemiology and Laboratory Training Program) Response to outbreak of Lead Poisoning in Unguwar Danbaba (Tsunami) Community, Gusau, Zamfara State – Nigeria: *Preliminary Report.* Pp 1 – 33.

NIOSH (1979c), (National Institute of Occupational Safety and Health).Analytical Methods for Determining Lead in Biological Materials: *Encyclopaedia of Analytical methods (7):* 1998 – 4230.

Nishijo, M, Nakagawa H, Morikawa Y, Tabata M, Senma M, Miura K. (1995). Mortality of inhabitants in an area polluted by cadmium: 15 year follow up. *Occupational Environment Medicine*; 52: 181–4.

Nordberg, G, Jin T, Bernard A, Fierens S, Buchet JP, Ye T, Kong Q, Wang H. (2002). Low bone density and renal dysfunction following environmental cadmium exposure in China. *Ambio*; **6**: 478–81.

Nriagu, JO, Pacnya JM. (1988).Quantitative assessment of worldwide contamination of air, water and soils by trace metals. *Nature,* 333: 134–139.

Nriagu, JO, Blankson ML, Ocran K. (1996). Childhood lead poisoning in Africa: a growing public health problem. *Science of the Total Environment,* 181 (2): 93–100.

Okeniyi, S O. (2000). Analysis of levels of lead in water, food and human fluids in Kaduna metropolis. PhD Dissertation for Department of Pharmaceutical and Medicinal Chemistry, Faculty of Pharmaceutical Sciences, Ahmadu Bello University, Zaria – Nigeria.

Oledzka, R., Bromat (1999). Chem. Toksykol., *Polish Journal of Environmental Studies Vol. 10, No. 2 (2001), 123-126* 32, 3, 207.

Olivero-Verbel, J., D. Duarte, M. Echenique, J. Guette, B. Johnson-Restrepo, P.J. Parsons, (2007). Blood lead levels in children aged 5–9 years living in Cartagena, Colombia, *Science Total Environment*. 372: 707–716.

Oskarsson, A Jorhem L, Sundberg J, (1992). Lead poisoning in cattle – transfer of lead to milk. *Science Total Environment,* 111: 83-94.

Patterson, CC, Ericson J, Manea-Krichten M, Shirahata H. (1991).Natural skeletal levels of lead in *Homo sapiens sapiens* uncontaminated by technological lead. *Science Total Environment.* 107: 205–236.

Paul, C. (1860). Study of the effect of slow lead intoxication on the product of conception (in French). *Archive of General Medicine*; 15:513-533.

Peters, A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, Baliff J, Oh JA, Allen G, Monahan K, Dockery DW. (2000). Air pollution and incidence of cardiac arrhythmia. *Epidemiology*; 11: 11–7.

Phupradit, W, Chaturachinda K, Anutlamai S. (1994). Maternal and umbilical cord blood lead levels in Ramathibodi Hospital, 1993. *Journal of Medical Association of Thailand*, 77: 367–372.

Piotrowska, M., Dudka S., Ponce-Hermondes R., Witek T., (1994) *Science Total Environment*. 158, 147.

Pirkle, JL Schwartz, J., Landis, J.R. and Harlan, W.R (1994). The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys. *Journal of the American Medical Association*, 272(4): 284–291.

Prapamontol, T. Tong S, von Schirnding YE. (1996). Health risk assessment of blood lead contamination in Chiang Mai mothers and their foetuses. 20(2): 221 – 228.

Price, D.J. and W.J. Birge. (1998). Analysis of Polychlorinated Biphenyls (PCB) in Redtailed Hawk Blood, Mink and Deer Liver and Kidney. Report submitted to FFOU March 5, 1998. 8 pp.

Rabinowitz, MB, Needleman HL.(1982). Temporal trends in the lead concentrations of umbilical cord blood. *Journal of Clinical and Scientific Investigation*, 216: 1429–1431.

Rabinowitz, MB, Wetherill GW, Kopple JD. (1976). Kinetic analysis of lead metabolism in healthy humans. *Journal of Clinical Investigation.*;58: 260-270.

Rijnders, E. Janssen NAH, Van Vliet PHN, Brunekreef B. (2001). Personal and outdoor nitrogen dioxide concentrations in relation to degree of urbanization and traffic density. *Environmental Health Perspective*; 109: 411–7.

Rojas-Lopez M, Santos-Burgoa C, Rios C,. (1994). Use of lead-glazed ceramics is the main factor associated to high lead in blood levels in two Mexican rural communities. *Journal of Toxicology and Environmental Health*, 42 (1): 45–52.

Romieu, I E. Palazuelos, M. Hernandez-Avila. (1995). Environmental urban lead exposure and blood lead levels in children of Mexico City. *Environmental Health Perspectives*, 103 (11): 1036–1040.

Samet, JM, Marbury MC, Spengler JD.(1987). Health effects and sources of indoor air pollution. Part I. *American Rev Respiratory Dis* ; 136: 1486–508.

Samet, JM, Dominici F, Curriero C, Coursac I, Zeger SL. (2000). Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *New England Journal of Medicine*; 343: 1742–9.

Shaw, J.R., C.M. Wood, W.J. Birge, and C. Hogstrand. (1998). Toxicity of silver to the marine teleost (*Oligocottus maculosus*): effects of salinity and ammonia. *Environmental Toxicology Chemistry,* 17(4):594-600.

Shen, X Rosen JF, Guo D, Wu SM (1996). Childhood lead poisoning in China. *Science of the Total Environment*, 181 (2): 101–109.

Smith, MA. (1984) Lead in history. In: Lansdowne R, Yule W, Eds. The lead debate: *the environmental toxicology and child health. London, Croom Hel*m: 7–24.

Spengler, J, Brauer M, Koutrakis P. (1990). Acid air and health. *Environmental Science and Technology*; 24: 946–56.

Staessen, JA, Roels HA, Emelianov D, Kuznetsova T, Thijs L, Vangronsveld. (2000) Environmental exposure to cadmium, forearm bone density, and risk of fractures: prospective population study. Public Health and Environmental. *Journal of Human Hypertension*, 16(2):123–131.

Steenland, K, Boffetta P. (2000). Lead and cancer in humans: where are we now? *American Journal of Industrial Medicine*; 38: 295–9.

Swant, A. D, (1995). Sample dissolution for elemental analysis/wet digestion: Laboratory Techniques; pp 4503 – 4510.

Szrzedzinski, J., Gozlinski H, Sikora J, Kleczkowski M., Bromat. (1983). Chem. Toksykol., *Polish Journal of Environmental Studies Vol. 10, No. 2, 123-126* 16, 3-4, 225.

Szymczak, I., Ilow R., Regulska-Ilow B., (1983) Rocz. Pzh, *Polish Journal of Environmental Studies Vol. 10, No. 2, 123-126* 44, 331.

Tabaku, A, Bizgha V, Rahlenbeck SI (1998). Biological monitoring of lead exposure in high risk groups in Berat, Albania. *Journal of Epidemiology and Community Health*, 52(4): 234–236.

TerraGraphics (2011). Zamfara, Nigeria Lead poisoning Epidemic Emergency Environmental Response: *Final UNICEF Report.* March, P. 1-42.

Tong, S, Baghurst PA, Sawyer MG, Burns J, McMichael AJ (1998). Declining blood lead levels and changes in cognitive function during childhood: the Port Pirie Cohort Study. *Journal of the American Medical Association*, 280: 1915–1919.

Tong, S, Yasmin E, Schrinding, Prapanontol T, (2000): Environmental lead exposure: a public health problem of global dimensions. *Bulletin of the World Health Organization (WHO),* 78 (9).

Torra, M, Rodamilans M, Montero F, Corbella J. (1997). Exposure to lead among the population of Barcelona: chronologic trends from 1984 to 1995. *Medical Clinic* (Barcelona), 108 (16): 601–603.

Touloumi ,G, Samoli E, Katsouyanni K. (1996). Daily mortality and 'winter type' air pollution in Athens, Greece—a time-series analysis within the APHEA project. *Journal of Epidemiology on Common Health*; 50 (Supply 1): 47–51.

U.S. EPA. (1997). Test methods for evaluating solid wastes, SW-846, Final Update 3. Office of Solid Waste and Emergency Response, Washington, DC.

UNEP (2008). Draft final review of scientific information on lead. Geneva, United Nations Environment Programme, Chemicals Branch (http://www.chem.unep.ch/Pb_and_Cd/SR/Draft_final_reviews/Pb_Review/Final_UNEP_Lead_review_Nov_2008.pdf).

Urieta, I, Jalon M, Eguileor I (1996). Food Surveillance and the Basque country Spain). Estimation of the dietary intake of organochlorine pesticides, heavy metals, arsenic, aflatoxin M1, iron and zinc through the total diet study. *Food Additives Contamination*; 13(1): 29-52.

US-EPA (U.S. Environmental Protection Agency) (2000). *Review of the National Ambient Air Quality Standards for Particulate Matter. OAQPS Staff Paper*. Office for Air Quality Planning and Standards. EPA-452/R-96-013.

Viswanathan, P. Biddle R, Knowlton S, Padmanaban P, Mohanraj D, Ganesh Kumar V. (1991). Biological monitoring and lead and cadmium. In: Krishna Murti CR, Viswanathan P, eds. *Toxic metals in the Indian environment*. New Delhi, Tata McGraw-Hill Publishing Company: (7), 212–235.

Von Schirnding YE Shilu Tong,1 Yasmin E. (1991a). Blood lead levels in South African inner-city children. *Environmental Health Perspectives*, 94: 125–130.

Von Schirnding YE Shilu Tong,1 Yasmin E.. (1991b). Factors associated with elevated blood lead levels in inner-city Cape Town children. *South African Medical Journal*, 79 (8): 454–456.

Von Schirnding YE, Fuggle RF.(1996). A study of the distribution of urban environmental lead levels in Cape Town, South Africa. Science of the Total Environment, , 188 (1): 1–8. 1076 *Bulletin of the World Health Organization*, 2000, 78 (9) Special Theme – Environment and Health.

Von Schirnding YE. (1999). The impact of lead poisoning on the workforce and society. In: Proceedings of the International Conference on Lead Poisoning, Bangalore, India, 8–10 February 1999. Bangalore, the George Foundation, 1999: 41–47.

Weitzman, ML, Matte T., Homa D. (2005). A Review of Evidence of Adverse Health Effects Associated with Blood Lead Levels <10 µg/dL in Children. *Reported by Work Group of the Advisory Committee on Childhood Lead Poisoning Prevention* to: Centers for Disease Control and Prevention, in Preventing Lead Poisoning in Young Children, A statement by the Centers for Disease Control and Prevention, Atlanta, GA August, 2005. http://www.cdc.gov/nceh/lead/ACCLPP/meetingMinutes/lessThan10MtgMAR04.pdf accessed 20/6/15.

White, RF, Diamond R, Proctor S. (1993). Residual cognitive defects 50 years after lead poisoning during childhood. *British Medical Journal*; 50: 613-622.

WHO (1986) (World Health Organization),. Lead (evaluation of health risk to infants and children).Available from http://www.inchem.org/documents/jecfa/jecmono/v21je16.htm.

WHO (1990) Environmental Health Criteria. *Methyl Mercury* vol. 101. Geneva:

WHO (1992) World Health Organization Cadmium. *Environmental Health Criteria*, vol. 134. Geneva: World Health Organization.

WHO (1995) Lead. *Environmental Health Criteria*, vol. 165. Geneva: World Health Organization.

WHO/IPCS (1995) (World Health Organization/ International Programme on Chemical Safety). Environmental Health Criteria 165. Inorganic lead. Geneva.

WHO (2000a) (World Health Organization). Air Quality Guidelines for Europe. Second Edition. WHO Regional Publications, European Series, No 91. Copenhagen.

WHO, (2000b) (World Health Organization). Safety evaluation of certain food additives and contaminants. WHO Food Additives Series 44: Lead. Available from: http://www.inchem.org/documents/jecfa/jecmono/v44jec12.htm.

WHO (2005) World Health Organization, Mission Report: Lead Crisis in the United Nations Administered Province of Kosovo, WHO Bonn, Germany.

WHO (2007a) World Health Organization. Blood lead levels in children. Copenhagen, World Health Organization Regional Office for Europe, *European Environment and Health Information System* (Fact Sheet No. 4.5; http://www.enhis.org/object_document/o4738n27387.html).

WHO (2007b) World Health Organization. Lead exposure in children. Geneva, World Health Organization (http://www.who.int/phe/news/Lead_in_Toys_note_060807.pdf).

WHO (2007c) (World Health Organization). Health risks of heavy metals from long-range transboundary air pollution. *Joint WHO/Convention Task Force on the Health Aspects of Air Pollution*, Copenhagen.

WWPR, (2010) World's Worst Pollution Problems: Report (2011). www.buzzle.com/articles/water-pollution-causes-and effects.html (Accessed 18 May 2015).

Yang, HF, Luo XY, Shen W, Zhou ZF, Jin CY, Yu F, Liang CS, (1994). National Food Contamination Monitoring Programs- Levels of Mercury, lead and cadmium in Chinese foods. *Biomedical and Environmental Sciences*. 7: 362-368.

Ysart, G (1994). Dietary exposure estimates of 300 elements from UK total diet study. *Food Additive Contamination*. 9: 391-403.

Zanobetti, A, Schwartz J, Samoli E. (2002). The temporal pattern of mortality responses to air pollution: a multicity assessment of mortality displacement. *Epidemiology* 2002; **13**: 87–93.

Zheng, W. (1999). Blood lead screening in China: organization, quality assurance and results. In: Proceedings of the International Conference on Lead Poisoning, Bangalore, India, 8–10 February 1999. Bangalore, *The George Foundation*,: 119–122.

Ziegler, E.E, Edwards BB, Jensen RL, Mahaffey KR and Fomon SJ. (1978). Absorption and retention of lead by infants. *Paediatric Research*, 12: 29-34.

APPENDICES

Appendix I: Table 4.3: Geographical Position System (GPS) Coordinates of the village sampling compounds in selected mining communities

S/N	Villages/Comps	I	II	III	IV	V
1.	Bagega	N11^{0}51.864’	N11^{0}51.854’	N11^{0}51.856’	N11^{0}51.850’	N11^{0}51.883’
		E006^{0}00.222’	E006^{0}00.203’	E006^{0}00.247’	E006^{0}00.177’	E006^{0}00.197’
2.	Kadauri	N12˚20.787'	N12˚20.830'	N12˚20.808'	N12˚20.833'	N12˚20.726'
		E006˚19.601'	E006˚19.604'	E006˚19.572'	E006˚19.540'	E006˚19.618'
3.	Kawaye	N11^0 49.014’	N11^0 49.152’	N11^0 49.189’	N11^{0}49.323’	N11^{0}49.322’
		E006^{0}01.906’	E006^{0}01.945’	E006^{0}01.895’	E006^{0}01.897’	E006^{0}01.922’
4.	Kwali	N12^0 06.745’	N12^0 06.749’	N12^0 06.720’	N12^0 06.766’	N12^0 06.798’
		E005^0 41.245’	E005^0 41.266’	E005^0 41.284’	E005^0 41.258’	E005^0 41.304’
5.	Magami	N11^{0}43.299’	N11^{0}43.251’	N11^{0}43.297’	N11^{0}43.281’	N11^{0}43.258’
		E006^{0}35.254’	E006^{0}35.222’	E006^{0}35.274’	E006^{0}35.321’	E006^{0}35.287’
6.	Sunke	N11^0 53.771’	N11^0 53.769’	N11^0 53.791’	N11^0 53.680’	N11^0 53.772’
		E005^{0}54.712’	E005^{0}54.697’	E005^{0}54.674’	E005^{0}54.771’	E005^{0}54.689’
7.	Tsunami	N12^{0}08.652’	N12^{0}08.641’	N12^{0}08.668’	N12^{0}08.635’	N12^{0}08.677’
		E006^{0}40.627’	E006^{0}40.639’	E006^{0}40.628’	E006^{0}40.622’	E006^{0}40.624’
8.	Tungar- Guru	N11^0 55.996’	N11^0 55.957’	N11^0 55.009’	N11^0 55.950’	N11^0 55.017’
		E005^0 29.689’	E005^0 29.731’	E005^0 29.738’	E005^0 29.701’	E005^0 29.725’
9.	Tungar-Kudaku	N11^{0}56.754’	N11^{0}56.727’	N11^{0}56.707’	N11^{0}56.723’	N11^{0}56.618’
		E005^{0}58.126’	E005^{0}58.123’	E005^{0}58.138’	E005^{0}58.123’	E005^{0}58.206’
10.	Yargalma	N11^0 58.408’	N11^0 58.400’	N11^0 58.334’	N11^0 58.473’	N11^0 58.403’
		E005^0 31.016’	E005^0 30.957’	E005^0 31.023’	E005^0 30.940’	E005^0 30.932’

Appendix II: Table 4.4: Lead (Pb) Levels in Residential Compound Soil from the Selected Mining Communities (N=50)

S/N	Villages/Comps	Lead Concentration in Soil Sample (mg/Kg)					Mean ± SEM (mg/Kg)	P Value
	Compound	I	II	III	IV	V		
1.	Bagega	70.10	1335.30	12.80	268.50	60.50	349.44 ± 250.33	.850
2.	Kadauri	0.50	9.00	15.80	29.80	20.60	15.14 ± 4.99	.000
3.	Kawaye	40.60	39.30	1.00	3.60	2.90	17.48±9.19	.000
4.	Kwali	20.00	18.40	13.80	6.30	3.70	12.44±3.23	.000
5.	Magami	13.20	32.20	27.80	2.70	9.30	17.04±5.59	.000
6.	Sunke	286.80	380.40	329.80	155.80	215.80	273.72±39.97	.034
7.	Tsunami	350.90	36.60	169.90	8.30	56.10	124.36 ± 62.94	.000
8.	Tungar- Guru	18.00	3333.00	109.20	49.60	280.00	757.96 ± 645.35	.012
9.	Tungar-Kudaku	29.80	28.50	15.20	10.50	-	21.00 ± 4.81	.609
10.	Yargalma	436.60	4.40	342.00	147.80	264.00	238.96 ± 75.38	.099

P Values < 0.05 Are Statistically Significant *US-EPA Lead Acceptable Limit for Residential Soil = 400 mg/Kg*

Appendix III: Table 4.5: Lead (Pb) Levels in Sorghum (GS1) Sample in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in Sorghum (mg/Kg)					Mean ± SEM (mg/Kg)	P Value
	Compound		I	II	III	IV	V		
1.	Bagega	BAG/GS1	47.46	19.28	-	21.51	3.71	22.99 ± 9.07	.141
2.	Kadauri	KAD/GS1	16.31	28.92	31.89	18.54	14.09	21.95 ± 7.94	.006
3.	Kawaye	KWY/GS1	5.19	31.89	2.97	14.83	37.08	18.39 ± 6.91	.090
4.	Kwali	KWL/GS1	42.27	24.47	23.73	21.51	8.16	24.02 ± 5.44	.018
5.	Magami	MGM/GS1	17.80	41.52	17.06	34.11	20.02	26.10 ± 4.95	.010
6.	Sunke	SUK/GS1	2.97	17.06	6.68	40.04	8.90	15.13 ± 6.64	.142
7.	Tsunami	TSM/GS1	26.70	-	9.64	5.93	17.80	15.02 ± 4.62	.126
8.	Tungar- Guru	TGR/GS1	28.92	30.40	2.97	21.51	5.19	17.80± 5.81	.064
9.	Tungar-Kudaku	TKD/GS1	37.08	13.35	9.64	34.11	25.21	23.88 ± 5.45	.019
10.	Yargalma	YGM/GS1	11.12	3.71	15.57	9.64	14.83	10.97 ± 2.13	.020

P Values < 0.05 are statistically significant *WHO/FAO Threshold limit of concern is 3.0 µg/Kg*

P Values < 0.05 are statistically significant *WHO/FAO Threshold limit of concern is 3.0 µg/Kg*

Appendix IV: Table 4.6: Lead (Pb) Levels in Millet (GS2) Sample in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in Millet (mg/Kg)					Mean ± SEM (mg/Kg)	P Value
	Compound		I	II	III	IV	V		
1.	Bagega	BAG/GS2	18.54	42.27	36.33	17.06	20.02	26.84 ± 5.19	.010
2.	Kadauri	KAD/GS2	32.63	13.35	40.04	2.97	19.28	21.65 ± 6.64	.048
3.	Kawaye	KWY/GS2	27.44	11.87	-	37.82	4.45	20.40 ± 7.53	.135
4.	Kwali	KWL/GS2	300.30	17.06	6.68	35.59	0.74	72.07 ± 57.36	.295
5.	Magami	MGM/GS2	5.93	2.23	11.12	-	2.97	5.56 ± 2.02	.492
6.	Sunke	SUK/GS2	14.83	34.11	20.02	9.64	26.70	21.06 ± 4.31	.014
7.	Tsunami	TSM/GS2	34.11	-	-	-	22.25	28.18 ± 5.93	.312
8.	Tungar- Guru	TGR/GS2	10.38	4.45	39.30	24.47	20.76	19.87 ± 6.03	.049
9.	Tungar-Kudaku	TKD/GS2	22.99	11.12	31.14	2.97	-	17.06 ± 6.24	.146
10.	Yargalma	YGM/GS2	11.87	37.08	5.93	30.40	21.51	21.36 ± 5.73	.033

P Values < 0.05 are statistically significant *WHO/FAO Threshold limit of concern is 3.0 µg/Kg*

Appendix V: Table 4.7: Lead (Pb) Levels in Maize (GS3) Sample in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in Maize (mg/Kg)					Mean ± SEM (mg/Kg)	P Value
	Compound		I	II	III	IV	V		
1.	Bagega	BAG/GS3	10.38	11.87	20.76	6.68	24.47	14.83 ± 3.34	.024
2.	Kadauri	KAD/GS3	22.99	29.66	17.06	21.51	13.35	20.91 ± 2.77	.003
3.	Kawaye	KWY/GS3	49.68	20.76	-	36.33	18.54	31.33 ± 7.29	.059
4.	Kwali	KWL/GS3	11.87	14.83	22.25	18.54	13.35	16.16 ± 1.88	.002
5.	Magami	MGM/GS3	19.28	29.66	26.70	-	9.64	21.32 ± 4.46	.063
6.	Sunke	SUK/GS3	20.76	8.16	34.85	8.16	29.66	20.32 ± 5.45	.034
7.	Tsunami	TSM/GS3	11.87	38.56	1.47	-	4.45	14.09 ± 8.45	.310
8.	Tungar- Guru	TGR/GS3	45.23	34.11	27.44	0.74	31.14	27.73 ± 7.37	.028
9.	Tungar-Kudaku	TKD/GS3	26.70	38.56	34.11	12.61	-	28.00± 5.68	.053
10.	Yargalma	YGM/GS3	28.92	1.49	4.45	34.11	17.80	17.35 ± 6.45	.090

P Values < 0.05 are statistically significant *WHO/FAO Threshold limit of concern is 3.0 µg/Kg*

Appendix VI: Table 4.8: Comparison of Lead Concentration (mg/Kg) in Common Grains in Mining Communities

S/N.	Village/Sample	Sorghum (GS1)	Millet (GS2)	Maize (GS3)
1.	Bagega	22.99 ± 9.07	26.84 ± 5.19	14.83 ± 3.34
2.	Kadauri	21.95 ± 7.94	21.65 ± 6.64	20.91 ± 2.77
3.	Kawaye	18.39 ± 6.91	20.40 ± 7.53	31.33 ± 7.29
4.	Kwali	24.02 ± 5.44	72.07 ± 57.36	16.16 ± 1.88
5.	Magami	26.10 ± 4.95	5.56 ± 2.02	21.32 ± 4.46
6.	Sunke	15.13 ± 6.64	21.06 ± 4.31	20.32 ± 5.45
7.	Tsunami	15.02 ± 4.62	28.18 ± 5.93	14.09 ± 8.45
8.	Tungar- Guru	17.80± 5.81	19.87 ± 6.03	27.73 ± 7.37
9.	Tungar-Kudaku	23.88 ± 5.45	17.06 ± 6.24	28.00± 5.68
10.	Yargalma	10.97 ± 2.13	21.36 ± 5.73	17.35 ± 6.45

WHO/FAO Threshold limit of concern is 3.0 µg/Kg Differences within groups (p = 0.788) not significant
Differences within columns (p = 0.591) not significant

Appendix VII: Table 4.9: Human Blood Lead Levels (BLLs) in Children in the Selected Mining Communities (µg/dL) (N=49)

S/N	Villages/Comps	Age range (Years)	Lead Concentration in Children Blood Samples (µg/dL)					Mean ± SEM (µg/dL)	P Value
	Compound		**I**	**II**	**III**	**IV**	**V**		
1.	Bagega	4 – 6	148.00	164.00	189.00	152.00	135.00	157.60 ± 9.11	.000
2.	Kadauri	5 – 6	70.00	138.00	117.00	140.00	140.00	121.00 ± 13.47	.001
3.	Kawaye	2 – 6	113.00	198.00	130.00	117.00	123.00	136.20 ± 15.71	.001
4.	Kwali	3 – 5	27.00	6.00	44.00	46.00	38.00	32.20 ± 7.34	.107
5.	Magami	3 – 4	52.00	88.00	-	89.00	86.00	78.75 ± 8.94	.005
6.	Sunke	4 – 6	78.00	91.00	107.00	77.00	34.00	77.40 ± 12.14	.005
7.	Tsunami	5 – 6	73.00	72.00	65.00	43.00	68.00	64.20 ± 5,49	.001
8.	Tungar- Guru	4 – 6	169.00	69.00	173.00	68.00	29.00	101.60 ± 29.24	.035
9.	Tungar-Kudaku	3 – 5	56.00	38.00	8.00	46.00	31.00	35.80 ± 8.10	.033
10.	Yargalma	4 – 6	32.00	32.00	55.00	39.00	50.00	41.60 ± 4.70	.003

P Values < 0.05 are statistically significant *WHO BLL of concern is 10µg/dL,* *U.S – CDC ≤ 5µg/dL*

Appendix VIII: Table 4.10: Human Blood Lead Levels (BLLs) in Adults in the Selected Mining Communities (µg/dL) (N=43)

S/N	Villages/Comps	Age range (years)	Lead Concentration in Adults Blood Samples (µg/dL)					Mean ± SEM (µg/dL)	P Value
	Compound		**I**	**II**	**III**	**IV**	**V**		
1.	Bagega	18 – 55	-	108.00	-	135.00	112.00	118.33 ± 8.41	.006
2.	Kadauri	18 – 25	175.00	144.00	117.00	128.00	-	141.00 ± 12.62	.002
3.	Kawaye	30 – 50	218.00	74.00	80.00	45.00	2.00	83.00 ± 36.73	.093
4.	Kwali	30 – 55	60.00	49.00	90.00	33.00	41.00	54.60 ± 9.91	.011
5.	Magami	35 – 45	115.00	84.00	117.00	179.00	237.00	146.40 ± 27.40	.008
6.	Sunke	50 – 60	54.00	55.00	170.00	181.00	106.00	113.20 ± 27.17	.019
7.	Tsunami	38- 50	68.00	43.00	-	-	-	55.50 ± 12.50	.012
8.	Tungar- Guru	40 – 60	32.00	78.00	74.00	63.00	29.00	55.20 ± 10.39	.009
9.	Tungar-Kudaku	40 – 50	22.00	58.00	32.00	36.00	41.00	37.80 ± 5.94	.001
10.	Yargalma	30 – 50	54.00	57.00	39.00	36.00	40.00	45.20 ± 4.28	.006

P Values < 0.05 are statistically significant *WHO BLL of concern is 10µg/dL,* *U.S – CDC ≤ 5µg/dL*

Appendix IX: Table 4.11: Comparison of Blood Lead Levels (Mean ± SEM) in Children & Adult in Selected Mining Communities (µg/dL)

S/N.	Village/Sample	Children (µg/dL)	Adults (µg/dL)
1.	Bagega	157.60 ± 9.11	118 ± 8.41
2.	Kadauri	121.00 ± 13.47	141.00 ± 12.62
3.	Kawaye	136.20 ± 15.71	83.00 ± 36.73
4.	Kwali	32.20 ± 7.34	54.60 ± 9.91
5.	Magami	78.75 ± 8.94	146.40 ± 27.40
6.	Sunke	77.40 ± 12.14	113.20 ± 27.17
7.	Tsunami	64.20 ± 5,49	55.50 ± 12.50
8.	Tungar- Guru	101.60 ± 29.24	55.20 ± 10.39
9.	Tungar-Kudaku	35.80 ± 8.10	37.80 ± 5.94
10.	Yargalma	41.60 ± 4.70	45.20 ± 4.28

Differences within groups ($p = 0.403$) not statistically significant
Differences within columns ($p = 0.0013$) statistically significant

Appendix X: Table 4.12: Mean Blood Lead Levels (BLLs) for Goat (ABS1) from Selected Mining Communities (µg/dL) (N = 19)

S/N	Villages/Comps	Sample ID	Lead Concentration in Goat Blood Samples (µg/dL)					Mean ± SEM (µg/dL)	P Value
	Compound		I	II	III	IV	V		
1.	Bagega	ABS1	89.00	-	60.00	-	-	74.50 ± 14.50	.002
2.	Kadauri	ABS1	53.00	-	78.00	-	-	65.50 ± 12.50	.093
3.	Kawaye	ABS1	58.00	-	-	63.00	-	60.50 ± 2.50	.011
4.	Kwali	ABS1	74.00	72.00	57.00	-	78.00	70.25 ± 4.59	.008
5.	Magami	ABS1					46.00	46.00 ± 0.00	.019
6.	Sunke	ABS1	43.00	67.00	48.00	51.00	-	52.25 ± 5.19	.012
7.	Tsunami	ABS1							
8.	Tungar- Guru	ABS1	-	-	-	-	78.00	78.0 ± 0.00	.009
9.	Tungar-Kudaku	ABS1	74.00	69.00	91.00	-	-	78.00 ±6.66	.001
10.	Yargalma	ABS1	-	-	118.00	98.00	87.00	101.00 ± 9.07	006

Appendix XI: Table 4.13: Mean Blood Lead Levels (BLLs) for Sheep (ABS2) and Chicken (ABS3) the Selected Mining Communities (µg/dL)

S/N	Villages/Comps	Sample ID	Lead Concentration in Sheep Blood Samples (µg/dL)					Mean ± SEM (µg/dL)	P Value
	Compound		I	II	III	IV	V		
1.	Bagega	ABS2	-	112.00	-	60.00	80.00	84.00 ± 15.14	.003
2.	Kadauri	ABS2	-	73.00		83.00	65.00	73.66 ± 6.21	.083
3.	Kawaye	ABS2	-	64.00	54.00	-	67.00	61.67 ± 3.93	.015
4.	Kwali	ABS2	-	-	-	51.00	-	51.00 ± 0.00	.007
5.	Magami	ABS2	63.00	79.00	74.00	60.00	-	69.00 ± 4.49	.020
6.	Sunke	ABS2	-	-	-	-	-	86.50 ± 12.89	.015
7.	Tsunami	ABS3 (Chicken)	87.00	75.00	122.00	62.00	87.00		
8.	Tungar- Guru	ABS2	84.00	72.00	88.00	88.00	-	83.00 ± 3.79	.008
9.	Tungar-Kudaku	ABS2	-	-	-	-	-		.003
10.	Yargalma	ABS2	100.00	90.00	-	-	-	95.00 ± 5.00	.008

Appendix XII: Table 4.14: Comparison of Blood Lead Levels (Mean ± SEM) in Goat, Sheep and Chicken in Selected Mining Communities (µg/dL)

S/N.	Village/Sample	Goat (ABS1)	Sheep (ABS2)	Chicken (ABS3)
1.	Bagega	74.50 ± 14.50	84.00 ± 15.14	-
2.	Kadauri	65.50 ± 12.50	73.66 ± 6.21	-
3.	Kawaye	60.50 ± 2.50	61.67 ± 3.93	-
4.	Kwali	70.25 ± 4.59	51.00 ± 0.00	-
5.	Magami	46.00 ± 0.00	69.00 ± 4.49	-
6.	Sunke	52.25 ± 5.19	86.50 ± 12.89 -	-
7.	Tsunami		-	83.00 ± 3.79
8.	Tungar- Guru	78.0 ± 0.00	-	-
9.	Tungar-Kudaku	78.00 ±6.66	-	-
10.	Yargalma	101.00 ± 9.07	95.00 ± 5.00	-

Differences within columns ($p < 0.05$) were statistically significant.

Appendix XIII: Table 4.15: Lead Levels (µg/L) in Underground Water (UW) in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in Underground Water (µg/L)			Mean ± SEM (µg/L)	P Value
	Compound		I	II	III		
1.	Bagega	BAG/UW	879.00	978.00		928.50 ± 49.50	.156
2.	Kadauri	KAD/UW	54.00	590.00		322.00 ± 268.00	.076
3.	Kawaye	KWY/UW	650.00	666.00	219.00	511.67 ± 146.41	.060
4.	Kwali	KWL/UW	358.00	431.00		394.50 ± 36.50	.047
5.	Magami	MGM/UW	638.00	674.00		656.00 ± 18.00	.215
6.	Sunke	SUK/UW	349.00	300.00	155.00	268.00 ± 58.24	.312
7.	Tsunami	TSM/UW	829.00	1717.00		1273.00 ± 444.0	.215
8.	Tungar- Guru	TGR/UW	945.00	294.00		619.50 ± 325.50	.156
9.	Tungar-Kudaku	TKD/UW	685.00	587.00		636.00 ± 49.00	.076
10.	Yargalma	YGM/UW	385.00	74.00	104.00	187.67 ± 99.05	.060

P value < 0.05 considered statistically significant *WHO Safety level ≤ 10 µg/L for Underground water*

Appendix XIV: Table 4.16: Lead Levels (µg/L) in Surface Water (SW) in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in Surface Water (µg/L)			Mean ± SEM (µg/L)	P Value
	Compound		**I**	**II**	**III**		
1.	Bagega	BAG/UW	4235.00	4530.00		4235.00 ± 121.75	.024
2.	Kadauri	KAD/UW	450.00	415.00		413.00 ± 202.43	.003
3.	Kawaye	KWY/UW	2406.00	2640.00		2523.00 ± 117.00	.059
4.	Kwali	KWL/UW	1231.00	52.00		1046.50 ± 5.50	.002
5.	Magami	MGM/UW	548.00	554.00		551.50 ± 24.50	.063
6.	Sunke	SUK/UW	1083.00	1081.00		1082.50 ± 30.50	.034
7.	Tsunami	TSM/UW	2829.00	293.00		2829.00 ± 1268.00	.310
8.	Tungar- Guru	TGR/UW	1230.00	1319.00		1230.50 ± 44.50	.028
9.	Tungar-Kudaku	TKD/UW	206.00	210.00		207.50 ± 0.500	.053
10.	Yargalma	YGM/UW	1087.00	1089.00		1087.00 ± 28.00	.090

P values < 0.05 are statistically significant *WHO/FAO guideline surface water = 100µg/L*

APPENDIX XV: Table 4.17: Comparison of mean lead levels (µg/L) in underground (UW) and surface water (SW) in the Selected Mining Communities

S/N	Villages/Comps	Village/Sample ID	Lead Concentration in underground water (µg/L)	Lead Concentration in Surface water (µg/L)
	Compound			
1.	Bagega	BAG/UW	928.50 ± 49.50	4235.00 ± 121.75
2.	Kadauri	KAD/UW	322.00 ± 268.00	413.00 ± 202.43
3.	Kawaye	KWY/UW	511.67 ± 146.41	2523.00 ± 117.00
4.	Kwali	KWL/UW	394.50 ± 36.50	1046.50 ± 5.50
5.	Magami	MGM/UW	656.00 ± 18.00	551.50 ± 24.50
6.	Sunke	SUK/UW	268.00 ± 58.24	1082.50 ± 30.50
7.	Tsunami	TSM/UW	1273.00 ± 444.0	2829.00 ± 1268.00
8.	Tungar- Guru	TGR/UW	619.50 ± 325.50	1230.50 ± 44.50
9.	Tungar-Kudaku	TKD/UW	636.00 ± 49.00	207.50 ± 0.500
10.	Yargalma	YGM/UW	187.67 ± 99.05	1087.00 ± 28.00

P values < 0.05 are statistically significant *WHO/FAO guideline surface water = 100µg/L*

Appendix XVI: Advocacy, Field Trips & Collection of Samples

S/N	Village Community	Host	Date of Visit	Time of Visit
1.	Bagega	District Head	26/12/2013	11:40am
2.	Kadauri	Village Head	12/12/2013	3:00pm
3.	Kawaye	District Head	25/12/2013	12:30pm
4.	Kwali	Village Head	14/12/2013	1:00pm
5.	Magami	District Head	27/12/2013	11:00am
6.	Sunke	Village Head	15/12/2013	12:15pm
7.	Tsunami	District Head	27/12/2013	9:30am
8.	Tungar- Guru	Village Head	13/12/2013	12:45pm
9.	Tungar-Kudaku	Village Head	26/12/2013	2:30pm
10.	Yargalma	Village Head	13/12/2013	11:30am
11.	MSF office, Anka	Country Coordinator	09/01/2014	2:00pm

Note, Letter of introduction and familiarization tour took place in September, 2013.

Appendix XVII: AAS Absorbance of Lead in Compound Soil Samples (N=50) Of Selected Mining Communities In Zamfara State Nigeria.

Village/Compound	I	II	III	IV	V
Bagega	0.0215	0.0741	0.0057	0.0762	0.0189
Kadauri	0.0024	0.0047	0.0066	0.0104	0.0079
Kawaye	0.0134	0.0130	0.0025	0.0012	0.0014
Kwali	0.0077	0.0073	0.0060	0.0040	0.0032
Magami	0.0059	0.0111	0.0099	0.0029	0.0048
Sunke	0.0813	0.1071	0.0932	0.0452	0.0617
Tsunami	0.0990	0.0123	0.0491	0.0045	0.0177
Tungar-Guru	0.0072	0.1843	0.0323	0.0159	0.0794
Tungar-Kudaku	0.0104	0.0101	0.0064	0.0051	0.0022
Yargalma	0.1226	0.0034	0.0965	0.0430	0.0750

Appendix XVIII: AAS Absorbance of Lead in Sorghum (GS1) Samples of Selected Mining Communities in Zamfara State Nigeria.

Village/Compound	I	II	III	IV	V
Bagega	0.0064	0.0026	-	0.0029	0.0005
Kadauri	0.0022	0.0039	0.0043	0.0025	0.0019
Kawaye	0.0007	0.0043	0.0025	0.0004	0.0020
Kwali	0.0057	0.0033	0.0032	0.0029	0.0011
Magami	0.0024	0.0056	0.0023	0.0046	0.0027
Sunke	0.0004	0.0023	0.0009	0.0054	0.0012
Tsunami	0.0036	-	0.0013	0.0008	0.0024
Tungar-Guru	0.0039	0.0041	0.0004	0.0029	0.0007
Tungar-Kudaku	0.0050	0.0018	0.0013	0.0046	0.0034
Yargalma	0.0015	0.0005	0.0021	0.0013	0.0020

Appendix XIX: AAS Absorbance of Lead in Millet (GS2) Samples of Selected Mining Communities in Zamfara State Nigeria

Village/Compound	I	II	III	IV	V
Bagega	0.0025	0.0057	0.0049	0.0023	0.0027
Kadauri	0.0044	0.0018	0.0054	0.0004	0.0026
Kawaye	0.0037	0.0016	-	0.0051	0.0006
Kwali	0.0405	0.0023	0.0009	0.0048	0.0001
Magami	0.0008	0.0003	0.0015	-	0.0004
Sunke	0.0020	0.0046	0.0027	0.0013	0.0036
Tsunami	0.0046	-	-	-	0.0030
Tungar-Guru	0.0014	0.0006	0.0053	0.0028	0.0033
Tungar-Kudaku	0.0031	0.0015	0.0042	0.0004	-
Yargalma	0.0016	0.0050	0.0008	0.0041	0.0029

Appendix XX: AAS Absorbance of Lead in Maize (GS3) Samples of Selected Mining Communities in Zamfara State Nigeria

Village/Compound	I	II	III	IV	V
Bagega	0.0014	0.0016	0.0028	0.0009	0.0033
Kadauri	0.0031	0.0040	0.0023	0.0029	0.0018
Kawaye	0.0067	0.0028	-	0.0049	0.0025
Kwali	0.0016	0.0020	0.0030	0.0025	0.0018
Magami	0.0026	0.0040	0.0036	-	0.0013
Sunke	0.0028	0.0011	0.0047	0.011	0.0040
Tsunami	0.0016	0.0052	-	-	0.0004
Tungar-Guru	0.0061	0.0046	0.0037	0.0042	0.0001
Tungar-Kudaku	0.0036	0.0052	0.0046	0.0017	-
Yargalma	0.0039	0.0002	0.0006	0.0046	0.0024

Appendix XXI: AAS Absorbance of Lead in Children Blood Samples (N=49) of Selected Mining Communities in Zamfara State Nigeria

Village/Compound	I	II	III	IV	V
Bagega	0.0044	0.0049	0.0056	0.046	0.0041
Kadauri	0.0023	0.0042	0.0036	0.0042	0.0042
Kawaye	0.0035	0.0058	0.0040	0.0036	0.0038
Kwali	0.0011	0.0002	0.0016	0.0016	0.0014
Magami	0.0018	0.0028	0.0028	0.0027	-
Sunke	0.0025	0.0029	0.0033	0.0025	0.0013
Tsunami	0.0024	0.0023	0.0022	0.0015	0.0022
Tungar-Guru	0.0050	0.0023	0.0051	0.0022	0.0012
Tungar-Kudaku	0.0019	0.0014	0.0006	0.0016	0.0012
Yargalma	0.0012	0.0012	0.0019	0.0014	0.0017

Appendix XXII: AAS Absorbance of Lead in Adults Blood Samples (N=43) of Selected Mining Communities in Zamfara State Nigeria

Village/Compound	I	II	III	IV	V
Bagega	-	0.0033	-	0.0041	0.0034
Kadauri	0.0052	0.0043	0.0036	0.0039	-
Kawaye	0.0064	0.0024	0.0026	0.0016	0.0003
Kwali	0.0020	0.0017	0.0028	0.0013	0.0015
Magami	0.0035	0.0027	0.0036	0.0053	0.0069
Sunke	0.0018	0.0019	0.0050	0.0054	0.0033
Tsunami	0.0024	0.0015	-	-	-
Tungar-Guru	0.0012	0.0025	0.0024	0.0021	0.0012
Tungar-Kudaku	0.0010	0.0020	0.0012	0.0014	0.0015
Yargalma	0.0018	0.0019	0.0014	0.0013	0.0015

Appendix XXIII: AAS Absorbance of Lead in Animals Blood Samples (N=46) of Selected Mining Communities in Zamfara State Nigeria

Village/Compound	I	II	III	IV	V
Bagega – Goat	-	0.0034	0.0020	-	0.0026
-Sheep	0.0028	-	-	0.0020	-
Kadauri – Goat	0.0018	-	0.0025	-	-
-Sheep	-	0.0024	-	0.0027	0.0022
Kawaye – Goat	0.0020	-	-	0.0021	-
-Sheep	-	0.0021	0.0018	-	0.0022
Kwali – Goat	0.0024	0.0024	0.0019	-	0.0025
-Sheep	-	-	-	0.0018	-
Magami – Goat	-	-	-	-	0.0016
-Sheep	0.0021	0.0025	0.0024	0.0020	-
Sunke - Goat	0.0015	-	0.0022	0.0017	0.0018
-Sheep	-	-	-	-	
Tsunami – Goat	-	-	-	-	-
-Sheep	-	-	-	-	0.0021
-Chicken	0.0028	0.0024	0.0037	-	-
Tungar-Guru – Goat	-	-	-	0.0028	0.0025
- Sheep	0.0027	0.0023	0.0028	-	
Tungar-Kudaku - Goat	0.0024	0.0023	0.0029	-	-
-Sheep	-	-	-	-	-
Yargalma – Goat	-	-	0.0036	0.0031	0.0028
-Sheep	0.0031	0.0029	-	-	-

Appendix XXIV: AAS Absorbance of Lead in Underground and Surface Water Samples (N=31) Of Selected Mining Communities in Zamfara State, Nigeria

Village/Compound	I	II	III	IV	V
Bagega – UW	0.0023	-	-	-	-
-SW	0.0014	-	-	-	-
Kadauri – UW	0.0003	0.0016	0.0013	-	-
-SW	0.0104	-	-	-	-
Kawaye – UW	0.0017	0.0018	-	0.0007	-
-SW	0.0060	-	-	-	-
Kwali – UW	0.0010	0.0012	-	-	-
-SW	0.0001	-	-	-	-
Magami – UW	0.0017	-	-	-	-
-SW	0.0011	-	-	-	-
Sunke - UW	0.0010	0.0009	0.0005	-	-
-SW	0.0009	-	-	-	-
Tsunami – UW	0.0022	0.0043	-	-	-
-SW	0.0070	-	-	-	-
Tungar-Guru – UW	0.0024	0.0009	-	-	-
-SW	0.0031	-	-	-	-
Tungar-Kudaku – UW	0.0018	-	-	-	-
-SW	0.0003	-	-	-	-
Yargalma – UW	0.0011	0.0007	0.0004	-	-
-SW	0.0009	-	-	-	-

KEY: UW – Underground Water
SW - Surface Water

Appendix XXV: Peer review Journal publications from the research.

1) Danbaba A [1], Yakasai IA [2], Usman MA [2], Garba M [2], Abdullahi MG [3] (2012).Health and Environmental Effects of Lead in Selected Mining Communities in Zamfara State – Nigeria. *International Journal of Scientific Innovations and Sustainable Development*, Vol. 2, No 1, 2012: 47-55.

2) Danbaba A [1], Usman MA [2] ,Yakasai IA [2], Garba M [2], (2014). Comparison of Blood Lead Levels (BLLs) In Children and Adults of Selected Mining Communities of Zamfara State – Nigeria. *International Journal of Medical, Biological and Pharmaceutical Sciences* (IJMBPS) Vol. 2, No.3: 89-97.

3) Danbaba A [1], Usman MA [2] ,Yakasai IA [2] (2015). Assessment of Lead Levels Contamination of Common Grains (Sorghum, Millet and Maize) Grown In Selected Mining Communities of Zamfara State – Nigeria. *International Journal of Pure and Applied Sciences* Vol.2 No.2: 12-21.

4) Danbaba A[1], Liman M. L [2], Garba M.A [3] (2015). Solid Mineral Exploration: The Effects of Lead and other Heavy Metals Challenges to Future manpower in Zamfara State – Nigeria. *1st Multidisciplinary Journal (with Press).*

Appendix: XXVI: Conference papers from the research.

International Conference on Culture, Science and Sustainable Development, at Songhai Centre, Porto Novo, Republic of Benin. 8 – 12, October, 2012. Presented a paper on: Health and Environmental Effects of Lead in Selected Mining Communities in Zamfara State – Nigeria.

Cambridge Research and Publications International, International Academic Conference on Exploring Sub- Saharan African Resources for Sustainable Development in the Millennium, at Bayero University Kano, Kano State – Nigeria. 13 – 14 November, 2014. Presented a paper on: Comparison of Blood Lead Levels (BLLs) in Children and Adults of Selected Mining Communities of Zamfara State – Nigeria.

Oxford Research and Publications International, International Academic Conference on Exploring Sub- Saharan African Resources for Sustainable Development at Usman Danfodio University, Sokoto – Nigeria. 27-29 January, 2015. Presented a paper on: Assessment of Lead Levels Contamination of Common Grains (Sorghum, Millet and Maize) Grown in Selected Mining Communities of Zamfara State – Nigeria.

1st Multidisciplinary National Conference with Theme, Nigeria today: Security issues and Challenges to National Development at Nuhu Bamalli Polytechnic, Zaria, 14 – 16 September, 2015. Presented a paper titled; Solid minerals exploration: The effects of Lead and other Heavy Metals challenges to future manpower in Zamfara State – Nigeria.

Dept of Science Lab Technology
Nuhu Bamalli Polytechnic
P.M.B 1061, Zaria
Phone: 08036880121
jadanbaba@yahoo.co.uk

APPENDIX XXVII

12/12/13

Questionnaire on Zamfara Lead Poisoning

Dept of Pharmaceutical and Medicinal Chemistry,
Faculty of Pharmaceutical Sciences,
Ahmadu Bello University, Zaria – Nigeria

Dear Respondent,

REQUEST TO FILL QUESTIONNAIRE

In this research on Zamfara State Lead poisoning that has taken a global dimension, I wish to solicit your support and cooperation in answering the questionnaire. The questionnaire is intended to investigate and address the gaps in Lead poisoning research at mining community level as a result of gold ore mining and processing and recommend lasting solutions towards this growing epidemic. The research is aimed at determining the Lead levels in selected mining communities on expanded parameters in order to save the future generation and potential manpower in them.

Please be informed that responses on the questionnaire will be treated as confidential and only for the purpose meant for.

Thank you for volunteering,

Yours Sincerely,

Abduljalal Danbaba

Section A: Location/Compound Details

1. Local Govt. Area:……
2. Village:……..
3. Village/Compound I. D:……
4. Month/Year of Interview:…….

S/N	**Section B: Demographic details**	Within the Compound	Outside the Compound	Remark
1.	Date of Interview			
2.	GPS details: Longitude Latitude			
3.	No. of Children (M) ≤ 6 years			
4.	No. of Children (F) ≤ 6 years			
5.	No. of Adults (M) ≥ 18 years			
6.	No. of Adults (F) ≥ 18 years			
7.	Marital Status – Adult			
	a. Single b. Married c. Divorced d. Widowed			
8.	No. of pregnant women			
9.	No of women that had miscarriage in 2013			

S/N	**Section C: Educational Level/Qualification**	Within the Compound	Outside the Compound	Remark
1.	Quranic Education only Male Female			
2.	Adult Education: Male Female			
3.	Primary Education: Male Female			
4.	Secondary Education: Male Female			
5.	Tertiary Education: Male Female			
6.	No. with Primary Sch. Cert Male Female			
7.	No. with Sec Sch. Cert Male Female			
8.	No. with NCE, Diploma, ND, etc Male Female			
9.	No. Graduates (B. Sc, HND, B.A) Male Female			
10.	No. with Higher degrees			

	Male female			
S/N	**Section D: Employment Profile (≥18)**	Within the Compound	Outside the Compound	Remark
a.	**Self Employment/Trade**			
1	Mining of ore: Male Female			
2.	Processing of ore: Male Female			
3.	Farming: Male Female			
4.	Poultry: Male Female			
5.	Animal Rearing Male Female			
6.	Others (Specify): Male Female			
b.	**Government Employee**			
1.	Local Govt. Male Female			
2.	State Male Female			

3.	Federal Male female			
4.	Others (Specify) Male Female			

Section E: Compound/Family details in relation to Lead (for children ≤ 6 years)

Village/Compound I. D:……

Date:……..……(dd/mm/yyyy)Time:……..

Compound GPS: Longitude…….. Latitude…….

Head of Compound/Father:……..., Age (years):……

Mother/Wife:……..., Age (years)…….

Is the mother currently pregnant? Yes ☐ No ☐ Don't know ☐ ☐ Refused

If yes, how many months pregnant?.....months

Has she had any miscarriage in the past 12 months? Yes ☐ No ☐ Don't kno ☐ Refused

If yes, how many?.....

Child S/N	Sex	Age	Child been tested for Lead?	Child alive or dead?	Died how long ago? (months)
1.	Male…….. … Female …….		Yes……. No…… Result……	Alive…… Dead……..	
2.	Male…….. … Female		Yes……. No…… Result……	Alive…… Dead……..	

	…….				
3.	Male……. … Female …….		Yes……. No…… Result……	Alive…… Dead……..	
4.	Male……. … Female …….		Yes……. No…… Result……	Alive…… Dead……..	
5.	Male……. … Female …….		Yes……. No…… Result……	Alive…… Dead……..	

Does this compound have children over 6 years old? Yes ☐ No ☐ Don't know ☐ ☐
Refused
If yes, how many children over 6 years old?.....
Does the mother participate in ore processing activities? Yes ☐ No ☐ Don't know ☐
Refused
If ore processing, does this mother have their child with them during their ore processing activities?

Yes ☐ No ☐ Don't know ☐ Refused ☐

***THIS PARTICULAR FORM SHOULD BE COMPLETED FOR MOTHERS IN THE COMPOUND

Section F: Compound/Family details in relation to Lead (for Adults ≥ 18 years)

Village/Compound I. D:……Date:……..(dd/mm/yyyy)

Time:……..

Compound GPS: Longitude…….. Latitude…….

Head of Compound/Father:……., Age (years):……

Mother/Wife:……..., Age (years)…….

S/N	Sex	Age	Have been tested for Lead?	Alive or dead?	Died how long ago? (months)
1.	Male…….. … Female …….		Yes……. No…… Result……	Alive……. Dead……..	
2.	Male…….. … Female …….		Yes……. No…… Result……	Alive……. Dead……..	
3.	Male…….. … Female …….		Yes……. No…… Result……	Alive……. Dead……..	
4.	Male…….. … Female …….		Yes……. No…… Result……	Alive……. Dead……..	
5.	Male…….. … Female …….		Yes……. No…… Result……	Alive……. Dead……..	

HUMAN BLOOD SAMPLE ASSESSMENT OF LEAD LEVEL FORM (≤ 6 years)

LGA:…….

Village/Compound I. D:…… Date:……(dd/mm/yyyy)

Time:……

Compound GPS: Longitude…….. Latitude…….

To be taken in area where child with blood sample eats/sleeps

Child S/N	Sex	Age	Child been tested for Lead?	Present BLL (µg/dL)
1.	Male…….. … Female …….		Yes……. No…… Result……	
2.	Male…….. … Female …….		Yes……. No…… Result……	
3.	Male…….. … Female …….		Yes……. No…… Result……	
4.	Male…….. … Female …….		Yes……. No…… Result……	
5.	Male…….. … Female …….		Yes……. No…… Result……	

HUMAN BLOOD SAMPLE ASSESSMENT OF LEAD LEVEL FORM (Adult/pregnant women)

S/N	Sex	Age	Been tested for Lead?	Present BLL (µg/dL)
1.	Male…….. … Female …….		Yes……. No…… Result……	
2.	Male…….. … Female …….		Yes……. No…… Result……	
3.	Male…….. … Female …….		Yes……. No…… Result……	

4.	Male…….. … Female …….		Yes……. No…… Result……	
5.	Male…….. … Female …….		Yes……. No…… Result……	

Participating in Mining operations/ Exposures

In the last 12 months, did any people from this compound do the following gold extraction activities? If yes, indicate whether it was done inside and/or outside your compound.

S/N	Activities	Answer	Inside Compound?	Outside Compound?
1.	Breaking down ore (rocks)	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused…… ……
2.	Grinding of ore gravel	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused…… ……
3.	Washing of grinded ore gravel	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused…… ……
4.	Drying of grinded materials	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused……	Yes…… No,…… Don't know……. Refused…… ……
5.	Bagging of grinded materials	Yes…… No,…… Don't know…….	Yes…… No,…… Don't know…….	Yes…… No,…… Don't

		Refused……	Refused……	know……. Refused…… ……
6.	Separating mercury from Gold	Yes……. No,…… Don't know……. Refused……	Yes……. No,…… Don't know……. Refused……	Yes……. No,…… Don't know……. Refused…… ……
7.	Melting/burning mercury	Yes……. No,…… Don't know……. Refused……	Yes……. No,…… Don't know……. Refused……	Yes……. No,…… Don't know……. Refused…… ……

Where do you get your ore/rock from:……?

What do you do with your ore processing waste……?

In the past 12 months, what has been your main source of drinking water?
Well within the compound……
Well outside the compound……
Bore hole……
Stream ……
Pond……
Others (Specify)……..
In the past 12 months, have you gotten any drinking water anywhere else?
Yes……. No…….. don't know……..refused……
If yes, what are they?
Well within the compound……
Well outside the compound……
Bore hole……
Stream ……
Pond……
Others (Specify)……..
How often is your home cleaned or swept?
Daily …….
At least weekly……..
At least monthly…….
Less than once a moth…….
Never……
Don't know……
Refused…….

ENVIRONMENTAL ASSESSMENT FORM

LGA:……

Village/Compound I. D:…… Date:……(dd/mm/yyyy)

Time:……

Compound GPS: Longitude…….. Latitude…….

XRF Operator/Environmentalist

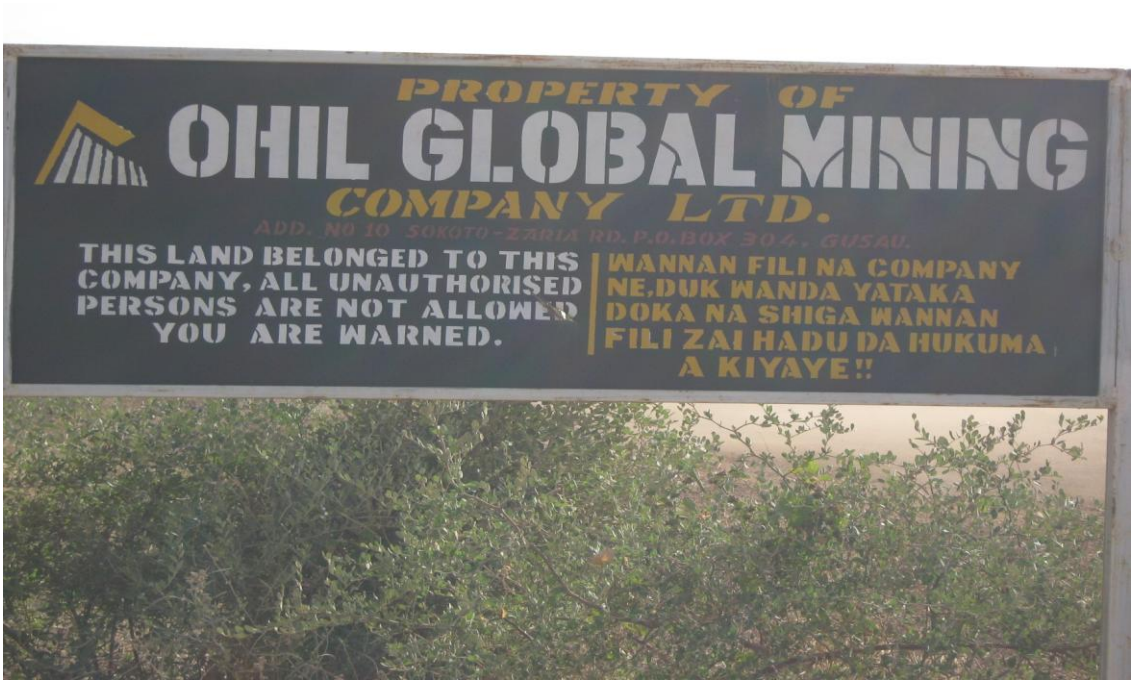
Visual Assessment

Processing	Yes Current	Yes Historic	No.	Comments/location
Ore (rocks) in bags				
Piles of broken ore (rocks)				
Grinding machines				
Grinding wheels				
Tailing piles/sacks of ground ore				
Drying piles				
Sluicing/washing				
Wells suspected of impact from ore processing				

APPENDIX XXVIII: RESEARCH ACTIVITY IN PICTURES



A mining site at Anka LGA using rudimentary tools



One of the Mining sites (Ohil) along Bagega – Anka Road



Copper containing ore at OHIL mining site, Anka



A local Miner demonstrating to the Research team traces of gold from an ore at Ohil Mining site, along Anka Road.



An ore grinding machine outside residential quarters



An abandoned gold ore grinding machine site within residential quarters at Tungar-Guru



Local processing of gold ore by the youths



Surface water used for gold ore processing along Yargalma road



Multipurpose surface water used for gold processing as well for livestock drinking



Multipurpose surface water used by children for domestic activity



A Haematologist taking blood sample from child volunteer Tungar-Kudaku



A Haematologist taking blood sample from an adult volunteer at Kadauri village



The Research team with District Head of Kawaye



The Research team with District Head of Bagega



Country Coordinator of Medicines Sans Frontier (MSF) with research team during a visit in Anka



An overhead bridge along Yargalma road used during rainy season