EGERTON UNIVERSITY LIBRARY

HUMAN EXPOSURE TO LEAD IN SELECTED AREAS IN OLKALOU AND NAIROBI, KENYA

EGERTON UNIVERSITY LIBRARY
30 AUG 2013
P.O. BOX 536,
EGERTON
ACQUISITION SECTION

NJOROGE G. KIMANI

SEPTEMBER 2007

EULIB
042 654

EGERTON UNIVERSITY LIBRARY
EGERTON UNIVERSITY LIBRARY
27 MAY 2014
P.O. BOX 536,
EGERTON
AFRICANA SECTION

ACKNOWLEDGEMENT

I would not have confidently gone through this research were it not for the dedicated contributions made by my academic mentors and co-investigators Prof. Eliud N. M. Njagi and Dr. George O. Orinda from Kenyatta University-School of Pure and Applied sciences, Prof. Christine S. Kigondu and Dr. Joshua K. Kayima of Nairobi University-College of Health Sciences and whole heartedly appreciate their participation without which the objectives of the study would not have been realized. I unreservedly acknowledge the Kenyatta National Hospital Ethics and Research Committee for the approval of this research without which its implementation would not have been possible. Likewise, I greatly appreciate the entire management of Kenyatta National Teaching and Referral Hospital where I have gained much experience during my service at the hospital that has played a big input into my success for creating the enabling environment that promotes personal development.

The financial requirement for this study was immense and fundamental to the success of this study and I greatly appreciate the financial contribution made by the United Nations Environment Programme (UNEP) - Partnership for Clean Fuels and Vehicles (PCFV) and for facilitating the equipment contribution by the United States Environment Protection Agency (USEPA). I greatly appreciate the Chairman and the entire staff of the Department of Clinical Chemistry at the College of Health Sciences, University of Nairobi for their contribution in allowing me to use the facilities of the department to carry out analytical work. The unrivaled dedication and implementation of research activities by the dedicated technical team of Cromwell Kibiti, Joel Mukaya, Antony Waswa and Alice Gathoni as well as the superb statistical analysis by George N. Michuku was paramount to the timely completion of this work and will always be remembered and appreciated. I acknowledge the inputs done by the following: - Dr. Ahmed E. Ogwell, former Head, Division of Non-Communicable Diseases, Ministry of Health and the staff of the Division for their assistance and collaboration in this exercise; Dr. Daniel Nguku-Medical Officer of Health Nairobi City Council, the Medical Officer of Health- Olkalou Sub-District Hospital and the entire staff for their cooperation and assistance.

During the entire period of the research, I received a lot of support and co-operation from colleagues at the Intensive Care Unit of Kenyatta National Hospital, friends and all those that I interacted with whom I can't be able to thank individually but request that they accept my gratitude. Last but not the least, I make a special note to all the children and adults who participated in the study and whose participation will forever remain a beacon of hope and inspiration towards achieving better health for all. The almighty God gives us the breath, knowledge and the power to interact that makes it possible for mankind to achieve great heights in life and glory goes to him for making us all participate and contribute to the success of this study.

Author,
Njoroge G. Kimani, MSc Medical Biochemistry
Clinical Biochemist/ Principal Investigator.

EXECUTIVE SUMMARY

Lead is a multitargeted toxicant, causing toxic effects to almost all organs and systems in the human body. Children are more susceptible to lead poisoning and suffer irreversible neuropsychological and cognitive impairments. WHO estimates that 12 million children in developing countries suffer from some form of permanent brain damage due to lead poisoning. In addition, hundred of millions of children and pregnant women suffer from various toxic effects of lead exposure. Though substantial progress has been made from the global response to lead poisoning, chronic exposure to low levels of lead in the environment still remains a serious public health problem particularly in developing countries where occupational lead poisoning is equally not adequately addressed. The problem is exacerbated by lack of appropriate diagnostic facilities. Though several biomarkers are used as effective tools in diagnosis of lead poisoning, measurement of blood lead concentration is regarded as the most reliable for general clinical use and public health surveillance. The World Health Organisation and the United States Center for Disease. Control places a blood lead level (BLL) of 10µg/deciliter of blood as toxic although no threshold levels has been determined due to the observed effects of lead exposure in children. This study was done to determine the prevalence of lead exposure in selected areas of Nairobi and in Olkalou. The latter study site served as a covariate. Blood lead levels were determined in a total number of 308 subjects using anodic stripping voltammetry (ASV). Children and adolescents (individuals aged 20 years and below) made 40% of the study subjects. The results obtained indicated 25% of the study population as exposed to high levels of lead. Agewise, 21% of children and adolescents were exposed to unacceptable levels of lead. Within the occupational setup of Ziwani Jua kali works, 89% of the workers had BLLs above 10µg/dl. The results of this study indicated significant environmental and occupational human exposure to unacceptable levels of lead in Nairobi. It is recommended that measures to address all sources of lead exposure and eradication of the problem be instituted. Based on the scope of this study, a comprehensive study to assess and identify childhood lead poisoning and the contribution of the various sources of lead, and an audit of industrial lead exposure is recommended.

ABBREVIATIONS

CBD	Central Business District
UNEP	United Nations Environment Programme
USA	United States of America
USEPA	United States Environment Protection Agency
KNH-ERC	Kenyatta National Hospital Ethics and Research Committee
MO	Ministry of Health
PHD	Public Health Department
HIV	Human Immunodeficiency Virus
ANOVA	Analysis of Variance
BLL	Blood Lead levels
Pb	Lead
WSSD	World Summit on Sustainable Development
CNS	Central Nervous System
PNS	Peripheral Nervous System
ATSDR	Agency for Toxic Products and Disease Registry
ALAS	Aminolaevulinic Acid Synthetase
ALAD	Aminolaevulinic Acid dehydratase
ZPP	Zinc Protoporphyrin
CDC	Centre for Disease Control and Prevention
NCC	Nairobi City Council
EPP	Erythrocyte Protoporphyrin
WHO	World Health Organisation
MDG	Millennium Development Goals
NCCLS	National committee for Clinical Laboratory Standards

ESA	Environment Science Associates
EDTA	Ethylene Diamine Tetra acetic Acid
OSHA	Occupational Safety and Health Administration
ASV	Anodic Stripping Voltammetry
TWA	Time Weighted Average
GFAP	Glial Fibrilliary Acidic Protein
PKC	Protein Kinase C
PbG	Porphobilinogen
NIOSH	National Institute of Occupational Safety and Health
IQ	Intelligent Quotient
Kda	Kilo Daltons
NAG	N-acetyl B D glucosaminidase
RBP	Retino Binding Protein
GFAAS	Graphite Furnace Atomic Absorption Spectrophotometry
IARC	International Agency for Research on Cancer

TABLE OF CONTENTS

ACKNOWLEDGEMENT ..... *II*
EXECUTIVE SUMMARY ..... *III*
ABBREVIATIONS ..... *IV*
TABLE OF CONTENTS ..... *VI*
LIST OF TABLES ..... *IX*
LIST OF FIGURES ..... *X*
LIST OF PHOTOGRAPHS ..... *XI*

CHAPTER ONE *1*
INTRODUCTION AND LITERATURE REVIEW *1*
1.1 Introduction *1*
1.1.1 Physical and chemical properties of lead **1**
1.1.2 Distribution of lead in the environment **2**
1.1.3 Uses of lead **3**
1.2 Literature Review *4*
1.2.1 Public health concern on lead exposure **4**
1.2.2 Sources of lead exposure **6**
1.2.3 Forms of lead intoxication **9**
1.2.4 Toxicokinetics of lead **9**
1.3 Problem statement and justification *37*
1.4 Hypothesis *39*
1.5 Study objectives *39*
1.5.1 Broad objective **39**
1.5.2 Specific objectives **39**

CHAPTER TWO *40*
MATERIALS AND METHODS *40*
2.1 Type of study *40*
2.2 The Study area *40*
2.2.1 Kariobangi North (E1) and Babandogo (E2) **40**
2.2.2 Waithaka (E3) **42**
2.2.3 Pumwani (E4) **42**
2.2.4 Ziwani Jua Kali works (OC) **42**

2.2.5 Olkalou (CA) 42
2.3 Study population *43*
2.3.1 Inclusion and exclusion criteria 44
2.4 Questionnaire administration *44*
2.5 Collection of blood samples *45*
2.6 Blood lead level determination *46*
2.6.1 Principal of analysis 46
2.6.2 Calibrating the analyzer 48
2.6.3 Quality control/assurance 48
2.6.4 Sample analysis and waste disposal 50
2.7 Ethical considerations *51*
2.8 Statistical analysis *51*

CHAPTER THREE *52*
RESULTS *52*
3.1 Blood lead levels in the total sample *52*
3.1.1 Distribution of blood lead levels in the study population 54
3.2 Blood lead levels in specific study areas *55*
3.2.1 Blood lead levels in Kariobangi North 55
3.2.2 Blood lead levels in Babadogo 56
3.2.3 Blood lead levels in Waithaka 56
3.2.4 Blood lead levels in Pumwani 57
3.2.5 Blood lead levels in Olkalou 58
3.2.6 Blood lead levels at Ziwani Jua kali (OC) 59
3.3 Comparison of blood lead levels in Nairobi and Olkalou for the entire study population *61*
3.4 Blood lead levels by age sets *62*
3.4.1 Comparison of blood lead levels for adults (> 20 years of age) 64
3.4.2 Comparison of blood lead levels for individuals below and above 20 years of age within and between study areas 65
3.5 Blood lead levels for children and adolescents (≤ 20 years) *65*
3.5.1 Distribution of blood lead levels for children and adolescents 68
3.5.2 Comparison of blood lead levels between study areas for individuals' ≤ 20 69
3.6 Blood lead levels by age sets (≤ 20 years) *69*
3.7 Blood Lead levels for children aged ≤ 10 years in Kariobangi North, Babadogo, ... *71*

3.8 Blood lead levels by sex *72*

CHAPTER FOUR *75*

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS *75*

4.1 Discussion *75*

4.2 Conclusion *81*

4. 3 Recommendations *82*

REFERENCES *83*

LIST OF TABLES

Table 1: Interpretation of adult worker BLLs 28

Table 2: Interpretation of erythrocyte protoporphyrin levels 30

Table 3: Chelating agents 37

Table 4: Study subjects in respective study areas 43

Table 5: Control target levels and post analysis control levels 50

Table 6: Blood lead levels (µg/dl) in the total sample and prevalence of BLLs ≥10 53

Table 7: Distribution of BLLs (µg/dl) at Olkalou 58

Table 8: Blood lead levels (µg/dl) at Ziwani Jua Kali 60

Table 9: Mean blood lead level (µg/dl) concentrations by age 62

Table 10: Blood lead levels (µg/dl) for individuals' ≤ 20 years in the total sample 67

Table 11: Mean ±1SD (µg/dl) blood lead levels by age sets for subjects' ≤20 years 70

Table 12: Blood lead levels for children 10 years for environment assessment areas in Nairobi and Olkalou 72

Table 13: Mean ±SD BLLS for females and males for the entire study sample 73

LIST OF FIGURES

Figure 1: Lead production in the twentieth century 2

Figure 2: Trend in reduction of BLLs action level. 5

Figure 3: Principle pathways of human lead exposure 8

Figure 4: Effects of lead on heme synthesis 22

Figure 5: Effects of lead poisoning on children and adults, lowest observable adverse health 25

Figure 6: Prevention and control of environmental lead exposure 33

Figure 7: Study areas in Nairobi and Nyandarua Districts 41

Figure 8: Distribution of BLLs (µg/dL) in the study population 54

Figure 9: Distribution of BLLs (µg/dL) in Kariobangi North 55

Figure 10: Distribution of BLLs (µg/dL) in Babadogo 56

Figure 11: Distribution of BLLs (µg/dL) in Waithaka 57

Figure 12: Distribution of BLLs in Pumwani 58

Figure 13: Distribution of BLLs at Ziwani 61

Figure 14: Mean blood lead level (µg/dL) concentrations by age 64

Figure 15: Distribution of blood lead level (µg/dL) for individuals' ≤ 20 years 68

Figure 16: Mean blood lead levels by age sets (≤ 20years) 71

Figure 17: Percentage of males and females (total sample) with BLLs above 10µg/dL ... 74

Figure 18: Percentage of males and females ≤ 20 years with BLLs above 10µg/dL 74

LIST OF PHOTOGRAPHS

Photograph 1: Questionnaire administration 45

Photograph 2: Collection of blood samples 46

Photograph 3: The LeadCare blood lead analyzer 47

Photograph 4: Calibration button and sensor containers 48

Photograph 5: Control materials 49

CHAPTER ONE

INTRODUCTION AND LITERATURE REVIEW

1.1 Introduction

1.1.1 Physical and chemical properties of lead

Lead (Pb) is a naturally occurring element, the most widely distributed and accessible of the heavy metals in the earth's crust that exists as a sulphide in galena (ore). Natural lead is a combination of four stable isotopes: - ^{208}Pb (51-53%), ^{206}Pb (23.5-27%), ^{207}Pb (20.5-23%), and ^{204}Pb (1.35-1.5%), which are decay products of three naturally radioactive elements; ^{208}Pb from thorium, ^{207}Pb from actinium and ^{206}Pb from uranium. Lead exists in three oxidative states: - metallic lead: Pb (O), organolead: Pb (II) and inorganic lead: Pb (IV). The primary form of lead in the environment is organolead while inorganic lead is formed under extremely oxidizing states and its compounds are not found under normal environmental conditions (ATSDR 2005).

Metallic lead is rarely found naturally as a metal but usually combined with two or more other elements to form lead compounds. It's a soft, bluish-white/silvery gray malleable metal that melts at 327.5 degrees centigrade and has no characteristic taste or smell. Metallic lead is resistant to corrosion (not easily attacked by air or water), but is attacked by pure water and weak organic acids in the presence of oxygen. Lead sulphate, lead oxides and lead carbonates are formed as protective films which prevent or slow down the corrosion of the underlying lead metal on its exposure to air or water. Elemental lead reacts to hot or boiling hydrochloric acid, nitric acid and sulphuric acid to form plumbous and plumbic salts; and alkali to form plumbites and plumbates. Solubility of lead salts in water varies from insoluble to soluble depending on the type of salt;

i. Lead sulphate, phosphate, carbonate and oxides are insoluble in water,

ii. Lead chloride, lead azide and lead iodide is slightly soluble in water,

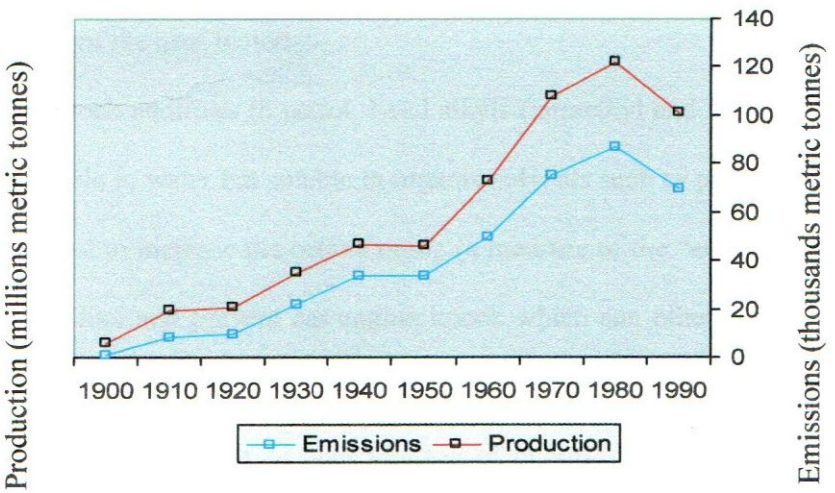
iii. Lead acetate, sub acetate and nitrate are highly soluble in water.

Some of the lead compounds can burn; for example lead chromate, lead nitrate and tetraethyl/tetramethyl lead in some gasoline (Goyer 1988, USEPA 1989, ATSDR 2005).

1.1.2 Distribution of lead in the environment

Due to its resistance to corrosion, density and low melting point, lead has widely been mined and used; its production and emission in the twentieth century has drastically risen. The increased distribution and mobilization of lead in the environment (Figure 1) has resulted to increased human exposure to and uptake of this non-essential element (Smith 1984, Nriangu *et al.* 1996).

Figure 1: Lead production in the twentieth century



Source: Nriangu *et al.*, 1996

Lead is naturally persistent in water and soil. Most of the lead in the environment is of anthropogenic sources and the concentration in various media depends on the proximity to

source. Studies in the US indicated that lead concentration in various media varied depending on the point of source and automobile exhaust emissions. The mean concentration of lead in surface water was more than in seawater, while the content in soil was lowest in the rural areas as compared to urban areas (Goyer 1988, USEPA 1989a /1989b, ATSDR 2005). Worldwide, elevated blood lead levels are noted among people living in substandard and overcrowded houses near industries and heavy traffic, nutritionally and economically deprived (USEPA 1990, Tong *et al.* 2000). Occupationally, people dealing with lead during their manufacturing process, lead smelting and other industrial activities are at high risk of overexposure to lead. Low social economic status and low parental education contributes to high levels of lead exposure (USEPA 1990, Tong *et al.* 2000, Pruss-Ustin 2004).

1.1.3 Uses of lead

Elemental lead and its compounds have numerous uses in industrial processes and domestic appliances. Some of the uses include:-

i. Antiknock additives in petrol. Lead alkyls (tetraethyl and tetra methyl lead) are insoluble in water but soluble in organic solvents such as petrol. They are added in petrol to increase the octane rating (a measure of the "anti knock" resistance) of the fuel and prevent car engine knock which can otherwise be achieved by greater fuel refining. Level of octane rating determines the resistance to engine knocking as a result of auto ignition of unburnt hydrocarbon mixture in the combustion chamber. Addition of the alkyl lead in petrol results into leaded petrol whose use is being phased out all over the world,

ii. Paint additive (lead acetate; lead chromate; lead sulphate) in houses and in the printing (lead iodide; lead nitrate) industry due to its virtues as a stable compound and luster,

iii. Manufacture of lead acid storage batteries (lead oxide; lead sulfate), steel/iron pipes (lead alloys) and ammunition (lead oxide; lead styphnate; lead azide; lead nitrate). Other uses include the production of plastic and rubber (lead phosphate; lead bromide; lead chromate), utensils (kitchen ware), weights and balances, solder (lead bromide; lead sulphide; lead sulfate) and sheet lead,

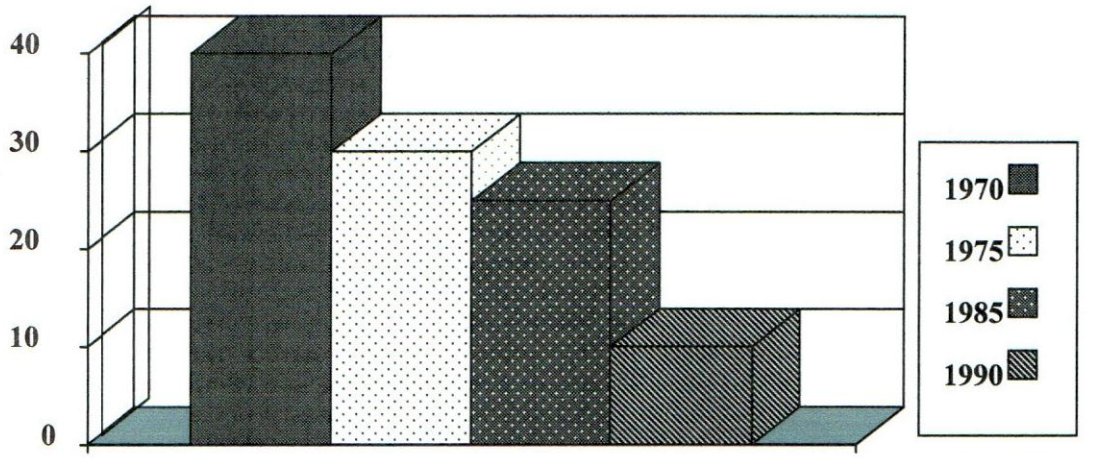
iv. Domestic use as parts in:-radiation protection in computer, radiation protection in x-ray units, television, diagnostic magnetic imaging and in circuit boards (lead sulfate; lead sulphide).

1.2 Literature Review

1.2.1 Public health concern on lead exposure

Lead is a cumulative poison whose toxicity affects multiple systems and organs in the human body. Depending on whether lead exposure is acute or chronic, serious physiologic effects, including death or long-term irreversible damage to brain function and organ systems occur. Lead primarily affects the peripheral and central nervous systems, renal function, heme synthesis and the metabolism of vitamin D and calcium. Lead also causes hypertension, reproductive toxicity, and developmental effects. Blood lead levels at which health effects have been observed are constantly being revised as new data is being generated (USEPA 1990, ATSDR 2005). WHO and the United States Centers for Disease Control (CDC) defines blood lead levels of 10 µg/dl of blood as toxic but there are considerations of lowering this level and the WHO is taking action. A reduction in action levels had been in process since 1970 due to the observed effects in children (Figure 2).

Figure 2: Trend in reduction of BLLs action level.



Source: CDC 1991

Lead intoxication is dangerous to all people in all sectors but children are more vulnerable. The WHO (2005) estimates indicate that 12 million children in developing countries suffer from some form of permanent brain damage due to lead poisoning while other hundred of millions of children and pregnant women are exposed to different sources of lead. All over the world, about 3.5% of minor mental retardations are attributable to lead poisoning. The heightened risk of children to lead poisoning is attributed to increased hand to mouth activity and Pica which results to increased intake of lead per unit body weight in children than adults. Children absorb more of ingested lead (40-70%) than adults (10-20%). The fraction of absorption is higher in children than adults as children's growth rate is rapid and deposition of lead in the developing tissue is increased. Increased susceptibility of children is also attributed to the developing organs and systems, critical stage of brain development and an immature blood-brain barrier (Mushak 1992, WHO 1995, Tong and McMichael 1999). There is currently no lead level that is safe for infants and young children (CDC

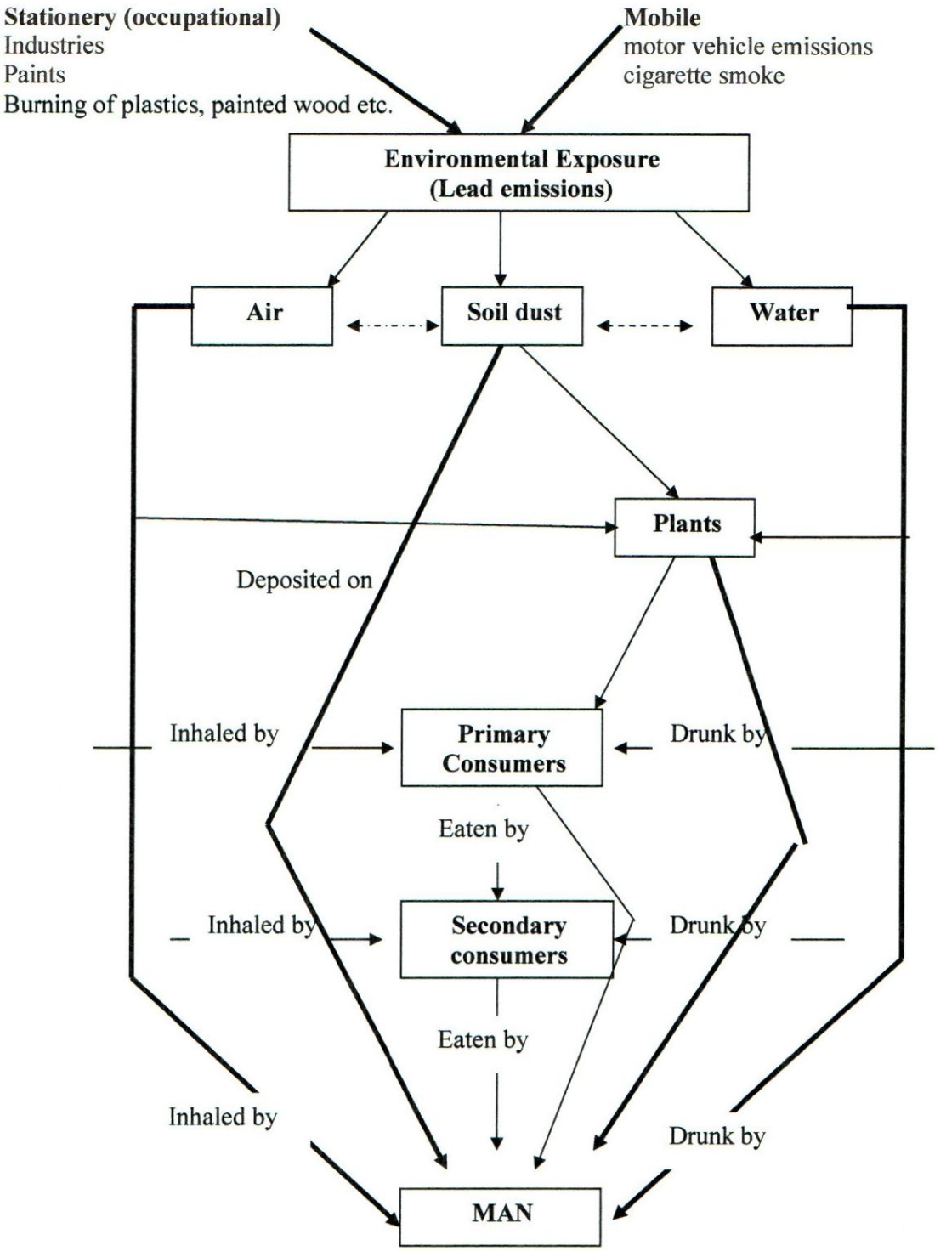
2005). Epidemiological studies of children show that those exposed to lead, at even low levels may have a lower IQ, learning disabilities, behavioral abnormalities and kidney damage. Recent studies have clearly indicated an association between blood lead levels below 10 µg/dl and adverse health effects ranging from cognitive function, neurological function, dental carries, growth retardation and onset of puberty levels (Moss *et al.* 1999, Lanphear *et al.* 2000, Canfield *et al.* 2003, CDC 2005).

1.2.2 Sources of lead exposure

Environmental sources of lead exposure to the public could be stationery and/or mobile as shown in Figure 3 (Goyer 1988). Lead released into the air through combustion of petrol is a major mobile source of environmental lead. Globally, most of the lead found throughout the environment has been attributed to the use of leaded gasoline (Smith 1984, CDC 1991). Cigarettes are also a mobile source of environmental lead contamination. Other sources of lead are release into the air when dust from lead painted surfaces are disturbed or created by abrasion of the paint and from contaminated clothing worn by a worker. Lead becomes airborne when it is burned or melted to a temperature high enough to release a fume into the air. Industrial emissions, burning of solid waste and some cosmetics and burning candle wicks are important sources of lead. Lead particles in the atmosphere have a residence time of about ten days ending up in soil, vegetation and water. Vegetables, grains and fruits exposed to heavy vehicle exhaust or industrial emissions may also contain higher than normal concentrations of lead. Secondary lead contamination to human beings also occurs from consumption of meat from animals fed on contaminated feeds. Lead solder used in cans is the main source of lead in food and many processors have started using nonsoldered cans. This is particularly significant in the case of baby and infant foods (CDC 2001, WHO 2005).

Lead poisoning associated with occupational exposure was first reported in 370 BC. It became common among industrial workers in the 19th and 20th centuries (Tong *et al. 2000*). According to estimates made by the United States National Institute of Occupational Safety and Health (NIOSH 1999), more than 3 million workers in the United States were potentially exposed to lead occupationally. People who work in lead smelting and refining industries, brass/bronze foundries, rubber products and plastic industries, printing, soldering, steel welding and cutting operations, battery manufacturing plants and lead compound manufacturing industries may be exposed to lead. Policemen who stand for long hours in traffic, construction workers and people who work at municipal waste incinerators, pottery and ceramic industries and radiator repair shops are all exposed to lead (Staudinger and Victor 1998, ATSDR 2005).

Figure 3: Principle pathways of human lead exposure



Source: Goyer 1988

1.2.3 Forms of lead intoxication

Lead intoxication may be organic or inorganic. Organic lead is highly volatile, lipid soluble and absorbed through the skin and respiratory tract. Organic lead poisoning is caused by exposure to tetraethyl or tetramethyl lead used as antiknock agents in gasoline and organolead compounds such as lead stearate or lead naphtharate used in certain commercial processes. Severe poisoning results from deliberate or accidental inhalation, sniffing or huffing of leaded gasoline in the course of cleaning gasoline storage tanks. Organic lead may also be ingested in illicitly distilled whiskies (moon shine whiskies) and from water whose delivery system is mainly made up of leaded pipes (Kosnett 2001, ATSDR 2005).

Inorganic lead poisoning may result by inhalation of large quantities of lead fumes or particulate aerosols in ambient air, ingestion of large quantities of lead based paints especially by children and to a lesser extent through dermal absorption (Kosnett 2001, ATSDR 2005).

1.2.4 Toxicokinetics of lead

Several factors determine the degree of toxicity to an individual exposed to lead (Poklis *et al.* 1996, Plaa 2001, Kosnett 2001). These factors include:-

1.2.4.1 Route of entry to the body

The amount of lead in the human body is largely determined by the route of entry. Major routes of lead intoxication are inhalation and ingestion. Airborne lead dusts or fumes can be inhaled into the lungs and upper respiratory tract, where it is readily absorbed and distributed throughout the body tissues. Absorption by the lungs depends on the size of lead particles.

Ingestion is the most common source of lead intake in children. Small children may eat lead paint chips or play in contaminated soil. While adults do not typically eat paint chips, they may handle cigarettes, food, chewing tobacco or apply make up that has been contaminated by lead, or use these products with lead contaminated hands. Lead can also be ingested from water that has been contaminated by lead in water pipes or containers. 40-70 % of ingested lead is absorbed through the gastrointestinal tract in children while adults absorb 10-20% (ATSDR 2005). Skin absorption is not normally a lead hazard because inorganic lead is not absorbed through the skin. However, organic lead such as tetraethyl lead and tetramethyl lead can be absorbed by the skin (Kosnett 2001).

1.2.4.2. Dose of lead exposure and exposure to other chemicals

The higher the dose one is exposed to, the lethal it is. Lead absorption in humans is a capacity limited process and the percentage of absorbed lead increases with the amount ingested and/or inhaled. The amount that one gets exposed to depends on occupational activity, proximity to source and whether rural or urban. People may be exposed to other chemicals through medicines, food, in the environment or working situations. These chemicals may be toxic to many other organs in the body leading to higher deposition of lead or impaired lead elimination from the body. Others may potentiate the action of lead intoxication at a level that may not have exerted effects of exposure (Kosnett 2001, ATSDR 2005).

1.2.4.3 Duration and frequency of exposure

The exposure time is related to dosage of exposure and frequent exposure to even low levels result to an accumulation of lead in the body. High single or multiple exposures occurring over a period of 1-2 days represents acute exposure. Exposures continuing for a

prolonged period of time represent chronic exposure. Chronic exposure to very high levels of lead even for a short time is highly dangerous to ones health.

1.2.4.4 Age, sex and lifestyle of individual

The potential for adverse effects of lead exposure is more heightened in children than adults. Likewise, there is a higher deposition of lead in the elderly than the middle aged. Males are more likely to be exposed to lead than females. This is most related to the fact that males are involved in most occupational activities dealing with lead than females. Body lead burden is increased in cigarette smokers due to the lead and other chemical contents of cigarettes. Alcoholics are also likely to have high levels due to high lead contents in some alcohols that take place during preparation e.g. moonshine whiskeys prepared using old radiators or lead pipes and dehydration due to the excessive use of alcohol (Kosnett 2001).

1.2.4.5 Nutrition and individual's state of health

Fasting, ingestion on an empty stomach, malnourishment, low intake of calcium (hypocalcaemia), iron (iron deficiency), high zinc levels and vitamin D excess enhances absorption of lead in the gastrointestinal tract (Goyer 1996). Some clinical states may enhance the deposition of lead or worsen its toxicity. Examples are diabetes, hypertension, hyperuricemia and or renal insufficiency:-

i. Diabetes

Diabetes is a chronic disease as a result of impaired carbohydrates metabolism due to either lack, inadequacy of insulin or its insensitivity. Diabetic nephropathy is one of the major complications of diabetes, culminating to renal insufficiency or chronic renal failure. Since

elimination of lead is primarily through the kidneys, accumulation of lead in the body is enhanced due to impaired excretion and high lead levels had been associated with diabetes (Schteingart 1992, Dods 1996, Chatterjea and Shinde 2002).

ii. Hypertension

Hypertension is sustained elevation of blood pressure above the accepted normal of 90 mmHg diastolic or 140 mmHg systolic. This condition may result to renal disease or aggravate nephropathy which in turn will impair excretion of lead from the body (Wilson 1992, Pudek 1996, Benowitz 2001).

iii. Hyperuricemia

Hyperuricemia is an elevation of uric acid concentration in the blood due to the body's overproduction or decreased excretion of uric acid. High levels of uric acid can damage the kidneys which will equally affect the excretion of other substances including lead. The impaired excretion results to an accumulation of lead in the body to dangerous levels. A study by Bennett 1985, found that more than 50% of patients suffering from lead nephropathy also suffered from gout. Lead-associated gout may occur in premenopausal women, an uncommon occurrence in nonlead-associated gout (Goyer 1985, Carter 1992, Chatterjea and Shinde 2002).

iv. Renal insufficiency/ renal failure

This refers to a condition in which over 75% of the nephrons are destroyed. The Kidneys lose their ability to maintain normal volume and composition of body fluids and excretion of toxic substances from the body is greatly affected. Renal failure enhances lead

deposition in the body while accumulation of other toxins may aggravate lead toxicity (Wilson 1992, First 1996, Perazella 1996).

1.2.4.6 Absorption and distribution of lead in the human body

Inhaled lead particles are deposited in the respiratory tract depending on their sizes and individual's breathing patterns (e.g., nose breathing vs. mouth breathing) and is related to age, airway geometry and air movement within the respiratory tract. Particles < 1μm are deposited in the alveolar and are absorbed on cellular dissolution or ingestion by phagocytic cells. Large particles >2.5μm are deposited in the nasopharyngeal and tracheobronchial regions from where they are transferred by mucociliary transport into the oesophagus and swallowed.

Absorption of lead from the gastrointestinal tract is primarily in the duodenum. The mechanism of absorption may involve active transport and diffusion through intestinal epithelial or paracellular cells. In relation to other divalent cations, lead is also thought to be absorbed through saturable transport mechanism or other capacity limiting processes. The less calcium or iron there is in the diet, the more of lead that is absorbed. This mechanism has been attributed to competition for a common transport protein. Adult human absorb about 10-20% of ingested lead, whereas children absorb up to 70% of ingested lead.

Lead absorbed from the respiratory or intestinal tract is bound to several intracellular proteins within erythrocytes. Facilitated transfer of lead across the cell membrane is thought to involve three mechanisms and a pathway inhibited by anion exchange inhibitors and dependent on HCO_3^- is the major pathway. A pathway that is not HCO^{3-} dependent and

not inhibited by anion exchangers is also thought to exist. A permeability pathway involving Ca^{2+} channel and shared between lead and calcium is also involved in cellular lead uptake. About 99% of lead in the circulation is in the erythrocytes while 1% is found in plasma (Bergdahl *et al.* 1997). In plasma, lead exists bound to several plasma constituents in four states; 40-75% of lead in the plasma is loosely bound to albumin and other proteins of low affinity to lead, some complexed to amino acids and carboxylic acid while some is tightly bound to metalloproteins and the rest exist as free ionized lead. The concentration of blood lead changes rapidly with exposure and its half-life is 25-28 days (ATSDR 2005).

The absorbed lead is further distributed to other compartments such as soft tissue and bone. The largest compartment is the bone marrow where 94% of the total body lead burden in adults and about 74% in children is deposited. The crystalline matrix of bone is made up of hydroxyapatite, a calcium-phosphate salt and lead displaces the calcium by forming stable complexes with the phosphate. Lead is mostly deposited in bone in normal mineralization processes during bone growth and remodeling. Lead in bone readily exchanges with the plasma lead pool and replenishes blood lead levels. The half-life of bone lead is more than twenty years. Apart from the bone marrow, other soft tissue that take up lead include; kidney, liver, muscle and gonads. Lead also crosses the placenta to the fetus and to the infant through maternal milk. The half-life of lead in the soft tissues is forty days (USEPA 1986a, Kosnett 2001, ATSDR 2005).

1.2.4.7 Systemic biotransformation and elimination of lead

Organic lead such as tetramethyl and tetraethyl lead is biotransformed in the liver through a P-450 mediated oxidative dealkylation to highly neurotoxic metabolites: trimethyl and

triethyl lead or completely oxidized to inorganic lead. Inorganic lead forms reversible complexes with amino acids and nonprotein thiols and binding to proteins. The inorganic lead is conjugated to glutathione and excreted primarily in the urine. Lesser amounts are excreted through sweat, skin hair, nails, bile and breast milk. About 99 % of the amount of lead taken into the adult body will leave in waste in a couple of weeks but only about 32 % of the lead taken into the body of a child will leave in the waste. Under conditions of continued exposure, not all lead that enters the body will be eliminated, and this may result to accumulation of lead in the tissues notably bone. In patients with high bone lead burden, slow release from the skeleton may elevate blood lead concentration for years after exposure ceases. Pathological high bone lead turnover states occurs in conditions such as hyperthyroidism, pregnancy, lactation, menopause, osteoporosis and/or or prolonged immobilization and result in frank lead intoxication (Kosnett 2001, ATSDR 2005).

1.2.4.8 Mechanism of lead toxicity and effects on body organs

The biochemical basis for the multiple toxic effects of lead is its ability to form bonds with a number of commonly occurring negatively charged groups in a number of enzymes, proteins, amino acids and nucleic acids. Lead ions complexes with ligands such as sulphate, carboxylic and the imidazole groups of proteins and interacts with the function of enzymes, signal systems and membranes. On a molecular level, lead toxicity involves fundamental biochemical processes and exerts multisystemic toxic effects through at least three mechanisms:-

i. Inhibiting or mimicking the actions of essential cations particularly calcium, iron and zinc which affect the ions dependent or related processes.

ii. Inhibiting enzyme activity by structural alterations or prevention of binding of substrate to normal active sites thus interfering with the normal synthesis and functions of protein and enzymes.

iii. Altering the structure of cell membranes and receptors. Resultant general depression of energy metabolism and of protein and nucleic acid synthesis causes a reduction in cell division and therefore affects growth (USEPA 1990, Kosnett 2001, ATSDR 2005).

Described below, are the effects of lead toxicity on specific organs and systems in the body.

1.2.4.8.1 Neurological system

The nervous system is the most sensitive target of lead exposure. Neurological effects in children generally occur at lower BLLs than in adults. In children, acute exposure to very high levels of lead may produce encephalopathy and its clinical manifestations (e.g. hyperirritability, ataxia, convulsions, stupor, and coma or death). Intellectual deficits observed in children have been studied in animals enabling determination of the molecular and biochemical basis of lead neurotoxicity. Mimicking of calcium action and/or disruption of calcium homeostasis is the most important of the numerous mechanisms on the effects of lead on the nervous system. This affects cell-signaling systems as calcium acts as an important cofactor in many cellular processes. One of the processes mostly affected is the activation of protein kinase C (a serine/threonine protein kinase) pathway by lead. PKC is involved in various synaptic transmission processes such as synthesis of neurotransmitters, ligand-receptor interactions, conductance of ionic channels and dendrick branching. PKC is made up of several isoenzymes which exhibit different enzymatic cofactor requirements, tissue expression and cellular distributions. The PKC γ-isoform which is neurospecific and

involved in long-term potentiation, spatial learning and memory processes is calcium dependent and a target of lead neurotoxicity.

AP-1 transcriptional regulatory complex formation is induced by PKC. This complex regulates the expression of several target genes through AP-1 promoter elements. One of the genes is the glial fibrillary acidic protein (GFAP) which is induced during reactive astrocytic gliosis. The astrocytes and endothelial cells make up the blood brain barrier. Chronic exposures to low levels of lead alter the normal pattern of GFAP gene expression in the brain and have largely been demonstrated during the developmental period in rats. Most of protein kinase C is found in the cytosol of immature brain microvessels while in mature brain, the enzyme is membrane-bound. Exposure of immature brain microvessels to lead, results to a change in PKC distribution from cytosol to the membrane on activation of PKC. Immature PKC activation due to high lead exposure may thus impair microvascular formation and function which is attributed to defects in the blood brain barrier that contributes to acute lead encephalopathy. A disruption of the blood brain barrier function of excluding plasma proteins and many organic molecules while limiting the passage of ions, results to free entry of molecules such as albumin. To maintain osmolarity, ions and water enters the brain resulting to edema and an increase in intracranial pressure (ATSDR 2005).

The neurologic effects of lead toxicity have been documented at exposure levels once thought to cause no harmful effects (CDC 1997a) and toxicity in children has been reported at levels lower than 10µg/dl (CDC 2005). There is a large body of evidence that associates decrement in intelligence quotient (IQ), performance and other neuropsychological defects with lead exposure. Studies have reported a drop in IQ by four to seven points for every 10 µg/dL increase in children's BLL (Lansdown *et al.* 1986, Fulton *et al.* 1987, Winneke *et al.*

1990) and the WHO (2005) states that there is a reduction of 0.25-0.5 points in IQ for every 1μg/dl increase in BLL. Some of the neurologic effects of lead in children may persist into adulthood. Needleman *et al.* 1990 correlated lead exposure with lower class standing (classroom performance), greater absenteeism, more reading disabilities, and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination in young adults more than 10 years after childhood exposure. Neurologic and behavioral effects have been documented in lead-exposed workers with BLLs ranging from 40 to120 μg/dl. Irreversible central and peripheral nerve damage has been reported to occur at blood lead level between 40-60 μg/dl (ATSDR 2005).

1.2.4.8.2 Renal system

Accumulation of lead in the kidney produces characteristic and pathological changes in structure and function. Cells lining proximal convoluted tubules are the most sensitive to lead. The lowest level at which lead has an adverse effect on the kidney remains unknown but damage to an adult kidney can occur at exposures of about 40 micrograms/dl (NSC 2004). Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (40-60 μg/dl), and some population-based studies show accelerated (*i.e.*, greater than that for normal aging) increases in serum creatinine or decrements in creatinine clearance at BLLs below 60 μg/dl (Staessen *et al.* 1992, Payton *et al.* 1994, Kim *et al.* 1996, El-Safty *et al.* 2004). The vulnerability of the kidney nephrons to lead toxicity is attributed to: -

i. renal blood flow which accounts for 25% of the cardiac output exposing the kidneys to large amounts of the chemicals,

ii. the hyperosmotic interstitium which allows chemicals to be concentrated in relatively hypovascular region and the kidney being an obligatory excretory route for most drugs,

iii. such that renal insufficiency results in drug accumulation and increased concentration in the tubular fluid (Wilson 1992).

Lead reacts with sulphur in most of enzymes to form metallothions and eventually accumulate in the kidney which can lead to irreversible damage to the kidney (Smith *et al.* 1994). Acute, high dose lead-induced impairment of proximal tubular function manifests in aminoaciduria, glycosuria, and hyperphosphaturia (a Fanconi-like syndrome); these effects appear to be reversible (Wedeen *et al.* 1986, ATSDR 2005). Low-level environmental lead exposure may accelerate progressive renal insufficiency in patients without diabetes who have chronic renal disease (Lin *et al.* 2003, Brewster and Perazella 2004).

Lead nephropathy in adults is a silent process characterized by absence of proteinuria in its early phase (Galagher-legak 1998). Diagnosis of chronic lead nephropathy is more difficult since the laboratory abnormalities seen with acute lead intoxication are not present with chronic lead exposure. Much of the evidence has been derived from renal histology in experimentally lead exposed animals and biopsies materials from occupationally exposed workers (Goyer 1985). Lead nephropathies have also been associated with an increased urinary excretion of various renal biomarkers that can be used to study human nephrotoxicity at an early stage (WHO 1991, Mueller *et al.* 1997, Awad *et al.* 2002, Ferah *et al.* 2002, Weaver *et al.* 2003, El-Safty *et al.* 2004). Higher NAG and RBP were associated with high lead levels and hence worse renal function (Weaver *et al.* 2003). Lead exposure may be a causal agent in kidney disease associated with essential hypertension and assessment of persons with hypertension should follow closely the renal functions and

a past history of lead exposure. In addition, other known causes of renal disease or damage, such as diabetes mellitus and gout, should be well controlled in patients with excess past or current lead exposure (WHO 1991, ATSDR 2005).

1.2.4.8.3 Haematological system

Lead inhibits the body's ability to make hemoglobin (heme biosynthesis) by interfering with several enzymatic steps in the heme pathway. The alteration of ALAS (aminolaevulinic acid synthase), ALAD (δ-aminolaevulinic acid dehydratase) and ferrochelatase activities diminishes or inhibits heme synthesis. The mitochondrial enzyme ALAS catalyzes the condensation of Glycine and Succinyl-coenzyme A to form ALA (aminolaevulinic acid). ALAS activity is regulated by heme through feedback mechanism. Lead indirectly stimulates ALAS resulting to more ALA formation. ALAD is the second enzyme in the biochemical pathway for heme synthesis. ALAD a zinc containing cytosolic enzyme catalyzes the condensation of two ALA molecules to one porphobilinogen (PbG) molecule to form Uroporphyrinogen. Lead inhibits the activity of ALAD (Mwt 280 kDa) by noncompetitive binding to vicinal sulfhydryls at the active sites of ALAD. Zinc binds to only one of the vicinal sulfhydryls while lead bridges the sulfhydryls found at this site. Inhibition of ALAD like the feedback derepression of ALAS results to the accumulation of ALA. It has been suggested that a genetic polymorphism in the human ALAD gene may cause certain individuals to be more sensitive to lead toxicity than others (Wetmur *et al.* 1991, Bergdahl *et al.* 1997).

Ferrochelatase catalyzes the insertion of iron into protoporphyrin IX to form heme. Lead decreases the activity of ferrochelatase by binding to the vicinal sulfhydryl groups of the active site. The disruption of the mitochondrial structure by lead resulting to impaired

transport to the mitochondrion is an indirect mechanism of ferrochelatase inhibition. A decrease in the activity of this enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP) in the red blood cells. Other enzymes in the pathway contain only one sulfhydryl groups at their active sites and not very sensitive to lead. The threshold BLL for a decrease in hemoglobin is 50μg/dL for occupationally exposed adults and approximately 40μg/dL for children, although other studies have indicated a lower threshold (e.g. 25μg/dl) for children.

The heme synthesis pathway (including cytochromes) on which lead has an effect is involved in many other processes in the body including neural, renal, endocrine, and hepatic pathways. Lead's impairment of heme synthesis can affect other heme-dependent processes in the body outside of the haematopoietic system and there is a concern about the significance and possible sequelae of these biochemical and enzyme changes at lower levels of lead (USEPA 1986b, ATSDR 2005). Figure 4 shows a scheme of the heme synthesis pathway and effects of lead.

Figure 4: Effects of lead on heme synthesis

Succinyl CoA + Glycine
↓← Aminolevulinate synthetase ▲
δ-Aminolevulinate (ALA) ▲
↓←δ-Aminolevulinate dehydratase (ALAD) ▼
Porphobilinogen
↓← Porphobilinogen deaminase
Uroporphyrinogen III
↓← Uroporphyrinogen decarboxylase
Coproporphyrinogen III
↓← Coproporphyrinogen oxidase
Protoporphyrinogen IX
↓← Protoporphyrinogen oxidase
Protoporphyrin IX (erythrocyte protoporphyrin-EPP) ▲
↓← Ferrochelatase (heme synthetase) ▼
Heme

▲increase in substrate
▼decreased activity

Source: Chatterjea and Shinde 2002

1.2.4.8.4 Endocrine system

Lead impedes vitamin D conversion into its hormonal form, 1-25-dihydroxyvitamin D, which is largely responsible for the maintenance of extracellular and intracellular calcium homeostasis. Diminished 1-25-dihydroxyvitamin D, in turn may impair cell growth, maturation, and tooth and bone development. These adverse effects seem to be restricted to children with chronically high BLLs and chronic nutritional deficiency, especially with regard to calcium, phosphorus, and vitamin D (Koo *et al.* 1991). Rosen *et al.* (1980) noted that 1-25-dihydroxyvitamin D levels in lead-exposed children, with blood lead levels of 33-55 µg/dl, were reduced to levels comparable to those observed in children with severe renal insufficiency (ATSDR 2005). Increase in duration of exposure in relation to decreasing thyroxin and free thyroxin levels have been reported suggesting that chronic lead exposure

could adversely affect the thyroid over time. No effects of lead on thyroid function have been found in children (ATSDR 2005).

1.2.4.8.5 Cardiovascular system

Various mechanisms been attributed to the observed increase in blood pressure due to chronic lead poisoning. Hormonal and neural system involved in the regulation of peripheral vascular resistance, heart rate and cardiac output are affected by lead. Several studies support an association between lead exposure and elevations in blood pressure and greater exposures (primarily occupational) increase the risk for hypertensive heart disease and cerebrovascular disease as latent effects (Victery *et al.* 1988, Hu 1991, Schwartz 1995, Korrick *et al.* 1999). It is estimated that, on a population mean basis, systolic blood pressure may rise 1-2 mm with each doubling of blood lead, and that blood lead can account for a 1 to 2% variance in blood pressure.

1.2.4.8.6 Reproductive system and pregnancy

Decreased total sperm counts and increased abnormal sperm frequencies have been reported in humans in long-term and ongoing lead exposure levels and effects may begin at BLLs of 40 µg/dl (Gennart *et al.* 1992, Lerda 1992, Alexander *et al.* 1996, Telisman *et al.* 2000, ATSDR 2005). There appears to be an association between higher (occupational) lead exposure levels and adverse pregnancy outcomes as opposed to those women exposed to lower environmental levels of lead. An increased frequency of miscarriages and stillbirths among women working in the lead trades has been reported and women living near smelters had high frequency of spontaneous abortions, miscarriages and stillbirths than those living some distance away (McMichael *et al.* 1986, Baghurst *et al.* 1987).

Increasing evidence indicates that lead readily crosses the placenta and adversely affects fetus viability as well as fetal and early childhood development. Prenatal exposure to low maternal BLLs of 14μg/dl or less may increase the risk of reduced birth weight, premature birth, congenital abnormalities, and postbirth effects on growth or neurologic development (Needleman *et al.* 1984, Ernhart *et al.* 1986, McMichael *et al.* 1986, ATSDR 2005).

1.2.4.8.7 Carcinogenic effects of lead

The carcinogenicity of lead in humans has not been demonstrated sufficiently but elemental lead and inorganic lead compounds has been classified as probable human carcinogens (Group 2B) by EPA. Lead acetate and lead phosphate are classified as potential carcinogens by the National Toxicology Program in the US (ATSDR 2005). Occupational exposure studies which primarily examined lead smelters, involved confounding exposures to other chemicals such as arsenic, cadmium, antimony, and toxicants from worker smoking habits and hence could not conclusively associate exposure to lead with cancer cases (IARC 1987). Figure 5 gives a summary of observed adverse effects in relation to lead levels.

Figure 5: Effects of lead poisoning on children and adults, lowest observable adverse health Effects

Children	**Lead concentration in blood (µg /dl)**	**Adults**
	-150-	
Death ►		
Encephalopathy→	**-100-**	◄Encephalopathy
Nephropathy→		
Frank anaemia→		◄Frank anaemia
Colic→		←Decreased longevity
	-50-	←Hemoglobin synthesis▼
		←Peripheral neuropathies
	-40-	Infertility(men)
Hemoglobin synthesis ▼→		Nephropathy
Toxicity at lower exposures		
		←Systolic blood pressure(men)▲
Vitamin D metabolism▼→	**-30-**	Hearing acuity
		←Erythrocyte protoporphyrin(men)▲
Nerve conduction velocity ▼→	**-20-**	
		←Erythrocyte protoporphyrin (women)▲
▲Erythrocyte protoporphyrin		
▼Vitamin D metabolism (?)		
Developmental toxicity→	**-10-**	Hypertension▲
IQ ▼		
Hearing▼		
Growth ▼		
Transplacental transfer		
Increased function▲		**▼Decreased function**

Source: ATSDR 1992

1.2.4.9 Clinical manifestations and diagnosis of lead intoxication

Lower levels of lead exposure are usually characterized by subtle, nonspecific symptoms. The infrequency of classic diagnostic signs and the nonspecific nature of the symptoms frequently contribute to misdiagnosis of lead poisoning (Keogh 1992). Since many of the neurologic and gastrointestinal symptoms are nonspecific, the diagnosis of lead toxicity may be easily overlooked unless the examining physician is aware of the history of lead exposure. As such, arriving at a diagnosis of lead poisoning requires a high index of suspicion and a careful history taking. Children and adults may also exhibit varying symptoms at different BLLs (Fischbein 1992). Clinical presentations may vary depending on whether the lead poisoning is acute or chronic and if mild, moderate or severe.

In acute lead poisoning, there is an abrupt onset of severe symptoms, which results after several days or weeks of the recurrent exposure and presents with symptoms of encephalopathy and colic. Excessive occupational exposure to lead over a brief period of time can cause a syndrome of acute lead poisoning. Classic clinical findings in this syndrome include abdominal colic, constipation, fatigue and central nervous system dysfunction. With even greater doses, acute encephalopathy with coma and convulsions may occur. In milder exposures, headaches and personality changes may be the only signs of neurologic toxicity. The diagnosis of acute lead poisoning is difficult and depending on the prominent symptoms the presentations may be mistaken for appendicitis, peptic ulcer, pancreatitis or infectious meningitis. Sub acute presentations featuring headache, fatigue, intermittent abdominal cramps, myalgias, and arthralgias has often been mistaken for flu like viral illness and may not come to medical attention (ATSDR 1992, Landrigan 1994).

Chronic toxicity is a latent illness with unspecific manifestations. Chronic inorganic lead poisoning is manifested by constitutional complaints of anorexia, fatigue and malaise and lead poisoning should strongly be suspected in any patient presenting with headache, abdominal and vague gastrointestinal difficulties, anaemia, weakness and depression, and less commonly with motor neuropathy, gout, renal insufficiency, loss of libido and impotence. Late effects may include chronic renal failure, hypertension, gout and chronic encephalopathy (ATSDR 1992, Landrigan 1994). Slowed nerve conduction and forearm extensor weakness (wrist drop), as late signs of lead intoxication, are more classic signs in workers chronically exposed to high lead levels (ATSDR 2005).

Due to the asymptomatic or unspecific manifestation of lead intoxication, an effective diagnosis is arrived at by carrying out diagnostic investigations which may demonstrate the body lead burden or effects of exposure. These diagnostic investigations include:-

i. Determination of blood lead level

The blood level concentration is the single best indicator of recent, acutely elevated lead absorption and provides information on lead absorption in persons with relatively brief exposure. It is considered to be the most reliable biomarker for general clinical use and public health surveillance. Various methods are used to determine blood lead levels. Most commonly used methods are graphite furnace atomic absorption spectrophotometry (GFAAS) and anodic stripping voltametry (ASV). Environment Science Associates (ESA) manufactures a simple to use and portable device for blood lead level determination which employs ASV. This electrochemical analysis for blood lead level determination allows the measurement of low levels using very low sample volumes. This is the method that was used to carry out blood lead levels determinations in this study. The higher the test result,

the more lead in the system and the more potential danger there is to health (CDC 1991, ATSDR 2005).

Blood lead level rises rapidly within hours after an acute exposure and remains elevated for several weeks. However, a single blood lead level measurement cannot distinguish between low level intermediate exposure or chronic exposure and high level chronic exposure. Likewise, the single BLL measurement may fail to establish higher exposure that may have occurred several months earlier. Several measurements done over a period of time are a good indicator of long-term exposure. The interpretation of BLLs for adults occupationally exposed to lead is as shown in Table 1.

Table 1: Interpretation of adult worker BLLs

Test value	Interpretation
≤ 9 µg/dl	Unexposed or "normal"
10 to 40 µg/dl	Acceptable levels for long-term exposure; retest in 6 months
40 to 50 µg/dl	Close observation and follow-up indicated; retest in 2 months
≥ 50 µg /dl or average of last three ≥ 60 µg/dl	Removal from exposure; retest within 1 month

Source: Staudinger and Victor, 1998

ii. Determination of aminolaevulinic acid dehydratase

This demonstrates the effect of lead intoxication to the haematological system and involves the measurement of the activity of the enzyme δ-aminolaevulinic acid dehydratase (ALAD) in red blood cells. A decrease in the activity of red cell δ -aminolaevulinic acid dehydratase

EGERTON UNIVERSITY LIBRARY

(ALAD) is seen even at low levels of lead exposure. Inhibition of ALAD is 50 percent at a blood lead level of 20μg per dl and almost complete when blood lead concentrations reach 50μg per dl. However, ALAD activity may be decreased in other disease conditions such as porphyria, liver cirrhosis and alcoholism, hence not specific (ATSDR 2005).

iii. Determination of zinc protoporphyrin

A zinc protoporphyrin or free erythrocyte protoporphyrin level reflects the toxic effect of lead on the erythrocytic enzyme ferrochelatase and a better indicator of impairment of heme synthesis than determination of ALAD activity. Inhibition of ferrochelatase is detected by a build-up on the red cell of protoporphyrin, and increases in plasma and tissue iron. Red cell protoporphyrin is in the form of a highly fluorescent Zn-protoporphyrin (ZPP) which can be measured readily with a filter fluorimeter. Zinc protoporphyrin levels begin to rise in adults when the blood lead level exceeds 30 to 40 μg per dl. Once elevated, zinc protoporphyrin levels tend to remain above background level for several months post exposure and even when the blood lead level has fallen, and hence does not discriminate between recent and past exposure but provides average lead levels at the site of erythropoiesis over the previous for months (Landrigan 1994, Staudinger and Victor 1998). The determination of protoporphyrin is limited by the fact that an increase of ZPP/EPP can also reflect IDA, sickle cell anaemia and hyperbilirubinaemia. Porphyrin is also photo labile and false low levels can be obtained if sample was exposed for long in light. The increased urinary excretion of porphyrin metabolites, coproporphyrin II and δ-aminolaevulinic acid are also useful on a group basis but less sensitive and convenient than blood porphyrin measurements (ATSDR 2005). Protoporphyrin levels can be interpreted alongside BLLs as shown in Table 2.

Table 2: Interpretation of erythrocyte protoporphyrin levels

Test EPP Value	Interpretation
Up to 80 µg/dl	Normal range for adults
80 to 250 µg/dl	Typical range for occupationally lead exposed adults whose BLLs range from 20-40µg/dl.
250 to 500 µg/dl	High range for lead-exposed adults (BLLs range from 40-55 µg/dl).
> 500 µg/dl	Excessive, indicating heavy exposure or extreme sensitivity to lead; symptoms likely

Source: Staudinger and Victor, 1998

iv. Assessment of peripheral blood films

Continued inhibition of heme biosynthesis by lead and a reduction in protein synthesis causes anaemia as a late effect of lead poisoning and is evident only when the BLL is significantly elevated for prolonged periods. Lead induces two types of anemia, often accompanied by basophilic stippling of the erythrocytes which can be seen in a peripheral blood film of lead exposed individuals. Acute, high-level lead exposure has been associated with hemolytic anemia. Chronic lead intoxication induces anemia by interfering with heme biosynthesis and diminishing red blood cell survival resulting to hypochromic, and normocytic or microcytic anaemia with associated reticulocytosis. Basophilic stippling; inclusions of aggregated ribosome in the red blood cells has been used for a long time as an indicator of lead poisoning. However, it's unspecific and inconstant as it can be seen in benzene and arsenic poisoning and is not universally present in all cases of lead poisoning (ATSDR 2005, WHO 2005).

v. Determination of urinary lead levels

Urinary levels of lead are determined to measure the excretion of lead in urine after provocation with edetate calcium disodium (calcium EDTA; Calcium Disodium Versenate) or another chelating agent. This serves to determine an individual's bone lead burden and establish or exclude past lead exposure as the etiology of a current disease. This is particularly useful especially when all functional tests are normal after several years since the last known lead exposure. An abnormally high body lead burden shows a relationship between past lead exposure and present illness. The investigation is affected by kidney dysfunction and variability in urine output (Staudinger and Victor 1998, ATSDR 2005).

vi. Assessments of body lead burden by x-ray fluorescence

The x-ray fluorescence method has been used as a noninvasive, relatively rapid approach to the assessment of body lead burden and an indicator of cumulative lead absorption. It does not indicate present exposure. Lead lines are a sign of significant chronic exposure to lead in a child, heavy lead lines forms at the distal part of the femur and the proximal side of the tibia and fibula. The threshold for the occurrence of lead lines is a BLL of 45 µg/dL. Due to its safety, specificity and simplicity, it's an alternative to chelation testing in monitoring of body lead burden for workers with documented chronic exposure (Landrigan 1994, WHO 2005).

1.2.4.10 Prevention of lead poisoning

The most effective way to ensure both the general public and workers are free of lead intoxication is by prevention of exposure. The approach in prevention of environmental and occupational lead exposures is different and should target all sources of lead.

1.2.4.10.1 Prevention and control of exposure to lead in the environmental

World wide, the reduction of lead in petrol or total eradication of leaded gasoline in several states has been instituted as the principal measure to guard against lead intoxication (CDC 1991, von Schirnding *et al.* 2001, Luo *et al.* 2003). The international action plan for preventing lead poisoning provides guidelines toward achieving an environment free of lead. Recommended initial step of action include:-

i. Completion of the worldwide phase out of leaded gasoline. A complete phase out of leaded gasoline was declared in Kenya on Dec 31st 2005. Atmospheric lead is also greatly reduced by regulation of emissions from vehicles, mining, smelting and refining operations.

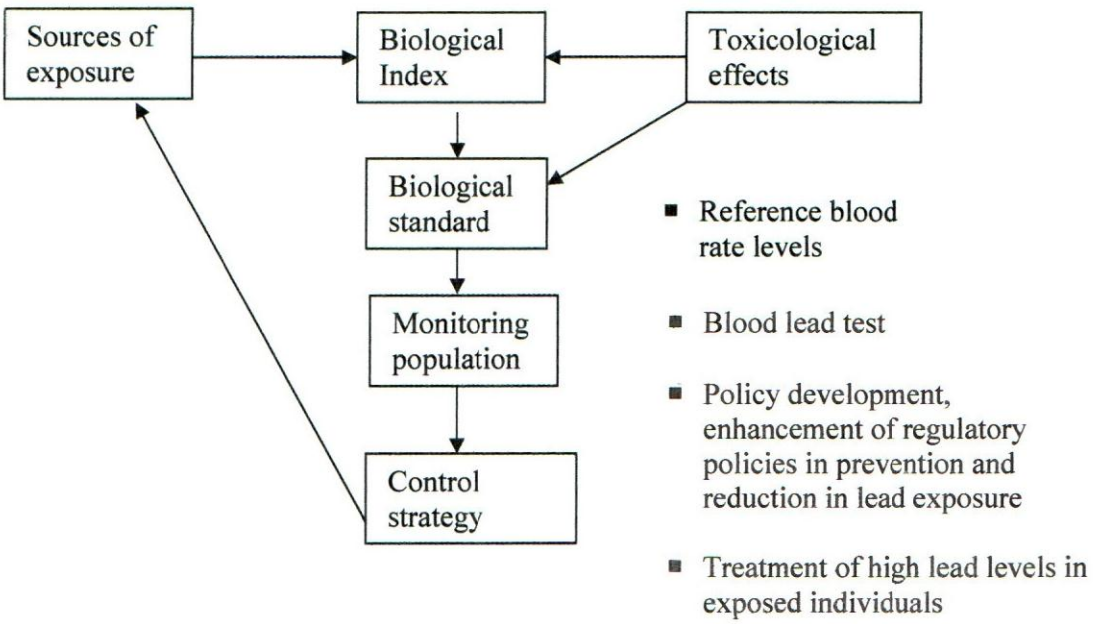
ii. Assessment of the problem which entails collection of data on population's blood lead levels, occupational health and safety, environmental contamination and industrial regulation.

iii. Setting priorities so as to coordinate the activities of controlling hazards from current uses. Replacing water delivery pipes with non- lead plumbing and establishment of good manufacturing practices for lead such as use of acid resistant ceramic ware reduces lead entering food through cooking utensils and canned food.

iv. Raising public awareness on dangers of lead, available substitute of lead containing substances, personal and family's protective measures and engagement in the preventive programs.

Figure 6 shows a protocol of preventive strategies of environmental lead exposure.

Figure 6: Prevention and control of environmental lead exposure



Source: Goyer 1988

1.2.4.10.2 Prevention and control of exposure to lead occupationally

According to the occupational safety and health administration of the United States (OSHA 1995, Staudinger and Victor 1998), primary activities that are used to prevent and control occupational lead poisoning include:-

i. Enhancement of safe working practices which entails educating workers on personal hygiene techniques and preventive strategies, housekeeping activities to remove lead dust accumulation and periodic inspection of maintenance of machinery and industrial setup,

ii. Establishment of engineering controls which involves substitution with less hazardous material, isolation through containment structure and ventilation via local exhaust system,

iii. Providing workers with personal protective equipment, protective clothing, face masks, and gloves,

iv. Establishment and monitoring of lead air standards.

Secondary measures that prevent individual overexposure include periodical medical examinations, workers medical removal and chelation therapy (OSHA 1995). A single case of occupational lead poisoning presents a sentinel health event and serves as a warning of potential overexposure to the workers. As such, the patient's workplace should be investigated for possible additional cases and causes. Appropriate health departments should be notified and referral to an occupational medicine specialist should be done (OSHA 1995, Staudinger and Victor 1998).

All individuals exposed to exceedingly high levels of lead should have a medical examination done yearly if any blood level exceeds 40 μg per dl. A medical exam should also be done prior to assignment in an area where air lead levels are at or above the action level and if a worker exhibits signs or symptoms of lead intoxication. The diagnosis of lead intoxication in adults requires the demonstration of excess lead absorption, documentation of impairment in an organ system consistent with the effects of lead and exclusion of other causes of disease. Thus, a medical examination of the highly exposed individual should focus on occupational and nonoccupational history, personal and workplace hygiene, history of gastrointestinal, hematological, renal, reproductive and neurological disorders (OSHA 1995, WHO 2005) and entail:-

i. Physical examination

Particular attention should be directed to neurologic and hematological abnormalities. Pulmonary status should be evaluated in workers required to wear respiratory protective devices.

ii. Blood pressure measurement

Several measurements of blood pressure should be taken over a period of time and should be correlated with other findings. Other causes of elevated blood pressure should also be ruled out.

iii. Blood testing

An elaborate blood evaluation in lead poisoning should include:

- Blood lead level
- Zinc protoporphyrin or free erythrocyte protoporphyrin level
- Hemoglobin, hematocrit and peripheral smear
- Serum creatinine, urea and electrolyte levels.

iv. Urinalysis and urinoscopic examination

A full urine analysis should be done which should include measurements of urinary porphyrin and aminoaciduria.

A worker, whose blood lead level has been determined to be dangerously elevated, should be removed from the source or work place to prevent further exposure. General industry workers found to have a single blood lead level of 60μg/dl or greater, or an average blood lead level of 50μg/dl or greater must be removed from the high-exposure job. The workers should subsequently receive more frequent medical evaluation and blood testing. A worker should not be allowed to return to a job with the potential for high lead exposure until his

or her blood lead level has fallen below 40μg/dl on two successive tests. Level must be confirmed with second follow-up level within 2 weeks of first report (OSHA 1995, Landrigan 1994). Permissible airborne exposure limit in absence of respirator use for workers removed from work due to an elevated blood lead level is $30\mu g/m^3$ per 8 hour time weighted average (TWA). Employees exposed to action level of workplace air lead of $30\mu g/m^3$ TWA must have blood lead level checked on a regular basis. Zinc protoporphyrin test is also strongly recommended at each occasion that a blood lead level is obtained. Categories and frequencies of monitoring are recommended every 6 months if last blood lead level was below 40μg/dl, every 2 months if last blood level is between 40μg/dl and level requiring medical removal and monthly if an employee has been removed from work because of an elevated blood lead level.

Chelation therapy is taken as the last step on complete physical and clinical assessment, removal from source of exposure and correction of nutritional/iron deficiencies. Chelation therapy should be administered depending on the blood lead concentration, the severity of clinical symptoms, the biochemical and hematological abnormalities, and the nature of the exposure. Chelation can be effective at reducing the total lead body burden and is critical in managing acute toxicity, but is generally not indicated for individuals with BLLs below 45μg/dl (Fischbein 1992, Landrigan 1994). Chelating agents themselves may have significant adverse effects, which present a risk apart from the lead toxicity. Chronic administration of oral agents to workers who continue to be exposed to unacceptably high levels in their workplace has been used by employers in the past but is inappropriate (Landrigan 1994, Staudinger and Victor 1998). Common chelating include are indicated in Table 3 (WHO 2005).

Table 3: Chelating agents

Generic	Chemical	Abbreviation
Edetate disodium calcium	Calcium disodium ethylenediamine-tetra-acetate	CaNa2EDTA
Dimercaprol	2,3-Dimercaptopropanol	BAL
D-Penicillamine	3-Mercapto-D-valine	D-Penicillamine
Succimer	Meso-2,3-dimercaptosuccinic acid	DMSA

Source: WHO 2005

Edetate calcium disodium has widely been used as the drug of choice but is now being replaced with succimer. Dimercaprol is used rarely in cases of lead encephalopathy in children and in combination with EDTA (WHO 2005).

1.3 Problem statement and justification

Over the last decade, global concern on the possible adverse effects attributed to chronic human exposure to lead in the environment has grown. Governments must therefore respond in a timely and effective manner to mitigate the problem. Focus must not only be on policy change but also on education and formulations of regulations geared towards eradication of the problem. As part of the interventions made to reduce and prevent human lead exposure, a resolution to implement a global phase out of lead from petrol (realized to be the biggest source of environmental lead), was adopted at the 2002 United Nations World Summit on Sustainable Development (WSSD) in Johannesburg, South Africa. As

more countries move towards implementation of the resolution and other measures to combat the problem, monitoring and evaluation of long-term effects of lead exposure to public health is crucial.

Lead exposure remains a serious public health problem particularly in several developing nations where measures to eradicate the problem are yet to be instituted. The high risk of exposure to environmental lead within our set up has been demonstrated by the high levels of lead that were established in soils, kale (sukumawiki), maize, water and milk sampled in Nairobi, Thika town and along the Thika – Nairobi highway. Most of the levels obtained were over a hundred times above the WHO standard mean levels (Mutuku *et al.* 2003). In addition, occupational exposure to high levels of lead may be on the increase as industries relocates to the developing countries due to cheap labour and less stringent regulations. The problem is further aggravated by the fact that numerous people work in the informal industrial setup (Jua Kali) where numerous activities are unregulated and if any, little monitoring is done. Likewise, with the growing urban population, numerous people who settle around industries in pursuit of jobs and shelter may be exposed to emission from industries dealing with lead unaware of the hazards attributed to lead exposure. The problem is further compounded by lack of appropriate diagnostic equipments and facilities that can be used to address lead exposure within our setup. Despite the potential risk, the status of human lead exposure has never been established in Kenya. Consequently, there is need to carry out an evaluation to establish the magnitude of the problem and the most affected populations. This will serve as a guide towards formulation of guidelines, direct focus of interventions and allocation of resources. The information obtained will also be handy in future monitoring and evaluation of interventions to mitigate lead exposure in Kenya.

1.4 Hypothesis

Environmental and occupational exposure to lead is a significant public health problem in Kenya.

1.5 Study objectives

1.5.1 Broad objective

To determine the status of public lead exposure in selected areas of Nairobi and Olkalou in order to provide baseline information that would assist in creating public awareness and formulation of policy guidelines towards mitigating the problem in Kenya.

1.5.2 Specific objectives

i. Determine blood lead levels (BLL) in the general population in Nairobi and the rural inhabitants at Olkalou.

ii. Determine blood lead levels of workers occupationally exposed to lead at the Ziwani Jua kali works in Nairobi.

iii. Compare blood lead levels (BLL) between the rural, urban and occupational study populations.

CHAPTER TWO

MATERIALS AND METHODS

2.1 Type of study

This was a cross sectional analytical study of blood lead levels in selected areas in Nairobi and the rural town of Olkalou in Nyandarua district. Random sampling was done for those present and willing to participate in the study on the day selected for fieldwork in the respective areas.

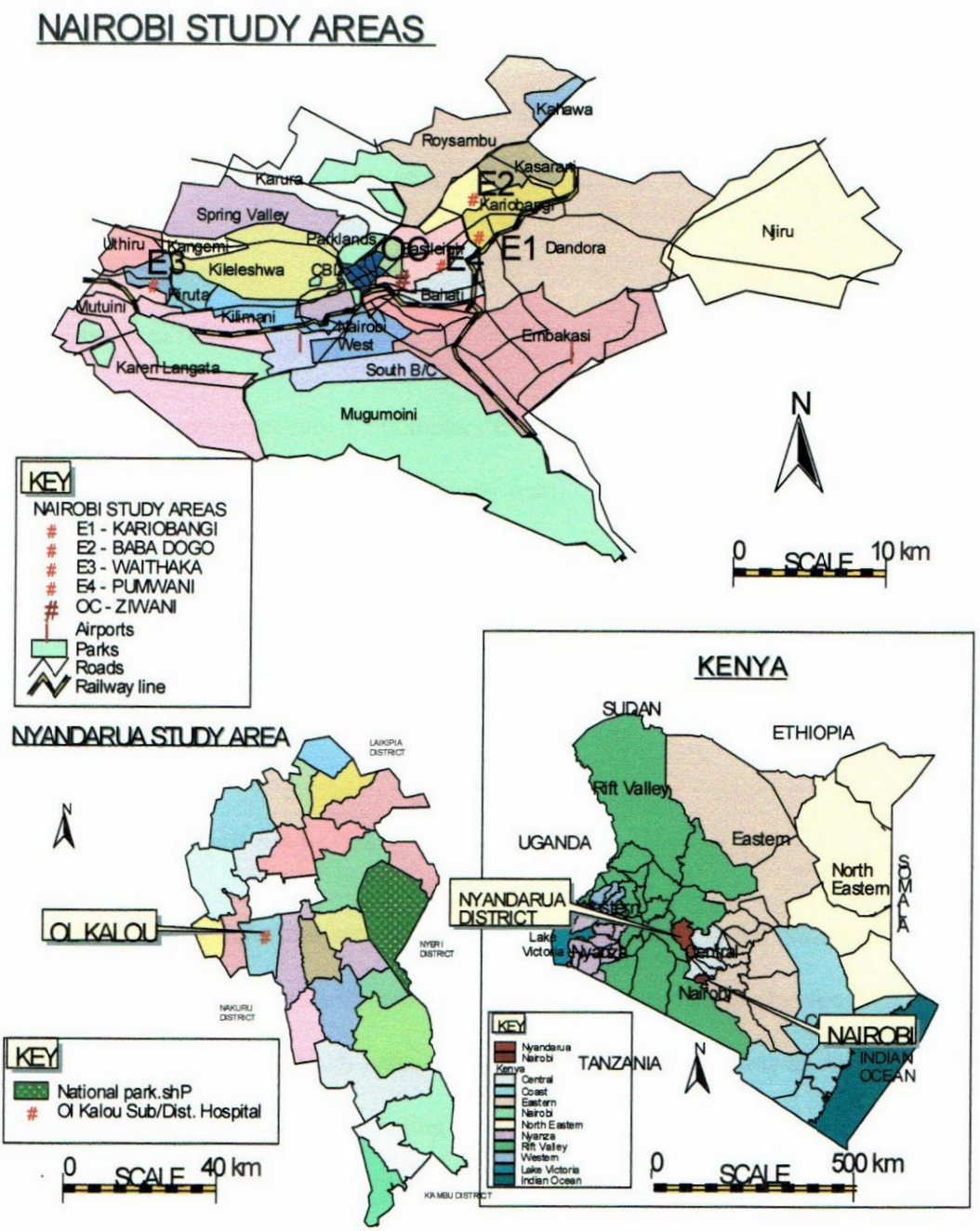
2.2 The Study area

The study on blood lead levels was conducted in five areas within Nairobi and one in Nyandarua District (Figure 7). Four study sites in Nairobi were coded as E1 to E4 and were meant to establish environmental exposure, while one was coded as OC for determination of occupational exposure. Olkalou was the covariate study area and was coded CA.

2.2.1 Kariobangi North (E1) and Babandogo (E2)

These are estates at the East of Nairobi, approximately 20 kilometres from the city centre and located next to each other with no clear boundary between them. Kariobangi North runs along Outer ring road, a major highway connecting the Mombassa and Thika highways and serves as link between other estates within the Eastland of Nairobi. Nearby are the Kariobangi North light industries dealing with paint, leather and timber works, ceramics and weighing balance manufacture. Most of the activities are carried out within residential flats and could be potential sources of environmental lead contamination. Babadogo is located near the Thika highway and off outer ring. Major industries dealing with steel, plastic and polythene in their production processes are located nearby and may be sources of lead exposure to the public. It was anticipated that environmental lead exposure would be high in this study areas.

Figure 7: Study areas in Nairobi and Nyandarua Districts



2.2.2 Waithaka (E3)

Waithaka is a periurban residential estate, North West of Nairobi and approximately 15 kilometres away from the City centre. Apart from simple mechanics and welding, no major industrial activity is evident in the locality. Vehicular traffic is greatly reduced in comparison to other areas within Nairobi. This area was considered as of low environmental lead exposure to the public.

2.2.3 Pumwani (E4)

This is an area approximately 5 kilometers from the city center. Within and around the estate are major roads which are of high vehicular traffic. No industrial activities are carried out in this area. It's an area of low income earners and most people live in overcrowded and deplorable housing. This area was considered to be of moderate public lead exposure from environmental lead. Recruitment was done at an informal school at the St John community center.

2.2.4 Ziwani Jua Kali works (OC)

This is an informal industrial setup located at close proximity to the Nairobi CBD. Numerous activities involving lead and its products are carried out within the Jua kali sheds. These activities include spray painting, panel beating, metal cutting, welding as well as motor vehicle mechanics. Most of the spray painting is done within a building where training of young artisans is also carried.

2.2.5 Olkalou (CA)

Olkalou in Nyandarua district is an urban centre at the slopes of the Aberdares Mountains and located about 200 kilometers from Nairobi. Agriculture is the major livelihood for the

inhabitants. The levels of exposure in this area were presumed to be negligibly low due to the fact that no industries and large traffic is evident within the area.

2.3 Study population

Children, adolescents and adults were randomly recruited for the study. A total of 308 subjects participated. Table 4 shows a breakdown of study subjects who participated in individual study area. Children and adolescents (ages ≤20 years) made up 40.9% of the study population. Recruitment of study subjects in Nairobi were done at City Council of Nairobi Health Centers at Kariobangi North, Babadogo and Waithaka and an informal school at Pumwani. The Olkalou district hospital served as the recruitment site for study subjects in the region. Most of the recruitment was done within health facilities since they served as suitable catchments site. Public awareness programmes were conducted in all areas of study prior to recruitment of study subjects.

Table 4: Study subjects in respective study areas

Code	**Study area**	**Total number of subjects**	**No of subjects age ≤20**
E1	Kariobangi north estate	90	36
E2	Babadogo estate	46	26
E3	Waithaka estate	52	19
E4	Pumwani	30	30
OC	Ziwani Jua Kali works	55	5
CA	Olkalou	35	10
	Total	308	126

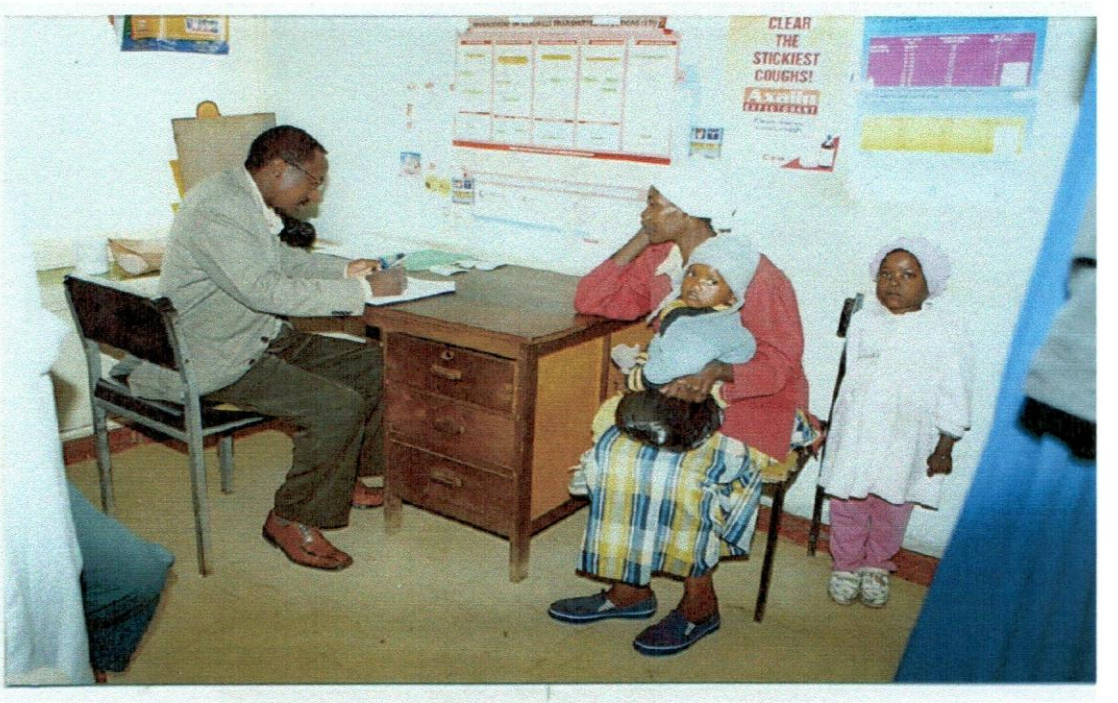
2.3.1 Inclusion and exclusion criteria

All individuals whose individual, parent's or guardian's consent of participation in the study was obtained were included in the study. Study subjects who were found to be critically ill, those who withdrew their consent of participation at a point during the study and those whose blood samples could not easily be obtained by venepuncture were excluded from the study.

2.4 Questionnaire administration

All adults and parents or guardians accompanying children at the recruitment venue and having presented themselves to the interviewer, were briefed on the purpose of the study through a standard explanation and individual consent for inclusion to the study was requested. Consent was registered from each participant in the presence of a member of the research team upon which, structured questionnaire seeking information on age, sex, occupation and brief medical history of the study subjects was administered in either Kiswahili or English. Client identification slips were issued to all consenting individuals to facilitate results notification to the subjects.

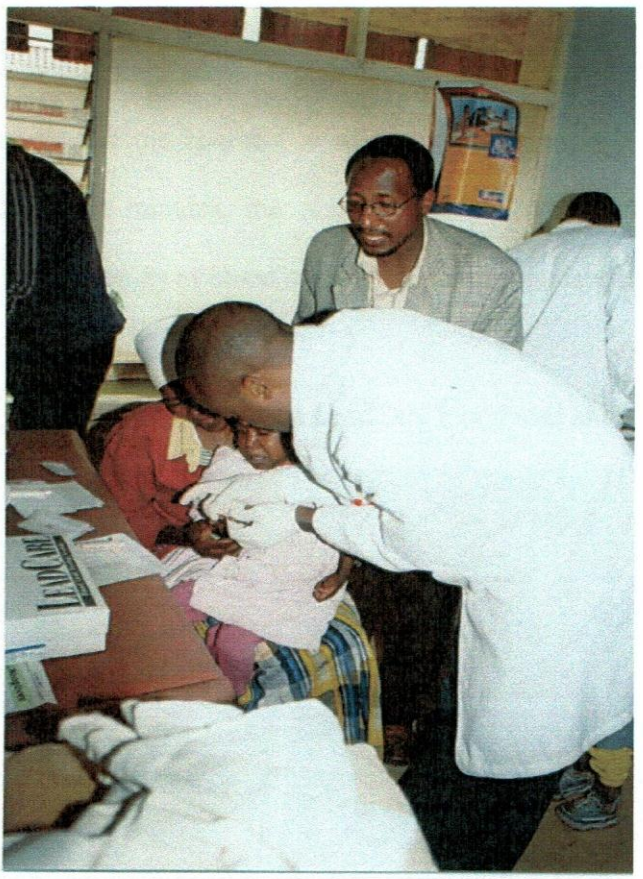
Photograph 1: Questionnaire administration



2.5 Collection of blood samples

Capillary blood samples were obtained from all participating individuals and collected in heparinized capillary tubes from Manienfeld, Germany. Sample collection was done by qualified Medical Laboratory Scientific Officers registered with the Kenya Medical Laboratory Technicians and Technologists Board. The standard procedure (NCCLS 1991) for obtaining capillary blood samples was followed. Fifty microlitres of the sample in the capillary tube were collected using a calibrated micropipette and put into a treatment reagent. Appropriate labeling of the sample/treatment reagent preparations was done and samples were dispatched for analysis.

Photograph 2: Collection of blood samples



2.6 Blood lead level determination

Blood lead level analysis was done using a LeadCare ® Blood Lead Testing System and LeadCare ® Blood Lead Testing Kits manufactured by ESA of Chelmsford, USA (Photograph 3).

2.6.1 Principal of analysis

The LeadCare ® Blood Lead Testing System and LeadCare ® Blood Lead Testing Kits uses the electrochemistry principal based on Anodic Stripping Voltammetry in determination of blood lead levels. Briefly, when a blood sample is added to the treatment reagent (metexchange), the bound lead in the Red blood cells is released and is made available for detection. When an

aliquot of the sample /treatment reagent is added onto the active part of the sensor, the lead is preconcentrated by application of a potential (electric current) on the working electrode. Application of an increasingly positive potential strips off (remove) the analyte from the electrode by oxidation and the resultant anodic current signal is a measure of the analyte concentration. After three minutes, the result indicating the blood lead concentration in micrograms per deciliter (μg/dl) of blood of an individual is displayed on the LED.

Photograph 3: The LeadCare blood lead analyzer



2.6.2 Calibrating the analyzer

The analyzer is electrically (AC) or battery (DC) powered. The system carries out an automatic self test to check its electronic functions every time it's turned on. Calibration of the analyzer was done for every testing kit that was used. Calibration was performed using a coded calibration button supplied with the kits. On successful calibration, the sensor number is displayed on the LED and sensors corresponding to the calibration code were used (Photograph 4). Calibration was performed as per the protocol of the operator's manual supplied.

Photograph 4: Calibration button and sensor containers



2.6.3 Quality control/assurance

For every day that sample analysis was done, high and low controls were run to ensure quality results and optimal performance of the analyzing system. The control materials were lyophilized bovine blood and leadcare water (Photograph 5).

Photograph 5: Control materials



The lyophilized whole bovine blood containing metabolized lead was reconstituted using two milliliters of purified water containing isothizolones (< 0.002%) as preservative. Fifty microlitres of the reconstituted controls were added into the treatment reagent and mixed on the day of analysis. The running of the controls was performed as a patients blood sample through the procedure explained below. The results obtained for the controls were confirmed to be within the expected control ranges before sample analysis commenced. The mean target control levels and the post analysis control levels for the high and low controls were as shown in Table 5.

Table 5: Control target levels and post analysis control levels

	Level 1 control lot number CCL 030405	**Level 1 control lot number CCL 032405**
Mean target value	8.8 ± 3	27.3 ± 4
Day 1	8.8	27.3
Day 2	8.7	26.5
Day 3	8.8	23.2
Day 4	8.4	27.7
Day 5	9.1	27.0
Day 6	8.6	27.7
Day 7	8.0	28.0
Postrun Mean±SD levels	8.7±0.2	26.8±1.7

2.6.4 Sample analysis and waste disposal

A sensor was put into the sensors holder and enough sample placed to cover the sensors active area. The sensor was pushed the rest of the way into the sensor holder until the area carrying the sample was covered. Testing commences on pressing the start button and results were displayed in micrograms per deciliter of blood after a count of 180 seconds. Results were double recorded by two analysts to rule out errors in registration. All medical wastes such as used swabs, gloves and sharps objects (needles and lancets) were put into appropriately labeled biological waste containers and sealed as per the manufacturers' instructions. The bags and containers were then dispatched to appropriate service providers for incineration.

2.7 Ethical considerations

Research activities commenced upon approval by the Kenyatta National Hospital Ethics and Research Committee vide Ref: KNH-ERC 01/2572. Further approval was obtained from the MOH Nairobi City Council, Ref: PHD/MOH/E.3 VOL 1(30). These approvals were communicated to all head of stations or departments that were concerned. The Ministry of Health, Kenya, Division of Non-Communicable Diseases (Ref: research/NCD/05) collaborated logistically in this research.

There was a concern from the study subjects over testing for the human immunodeficiency virus (HIV) as well as fear to infection from contaminated pricking needles or lancets. These concerns were addressed by satisfactory explanations and assurance that no testing for HIV will be done and sterile non-used sharps and consumables will be used. No participant was coerced to take part in the study. All individuals whose BLLs were determined were informed of the tests outcome and any required advice given. Those whose levels required medical attention were referred to a physician for followup and management.

2.8 Statistical analysis

Principal goals of the statistical analysis were to compare the blood lead levels obtained in the different study areas in Nairobi and Olkalou. Data collected by questionnaire and all blood lead levels obtained was coded using epi-info.v.6.0 software. Statistical analysis was done using Graphpad instant^{tm} version 2.04. ANOVA, post ANOVA Tukey Kramer and Dunn's multiple comparison tests were used to compare associations between and within study areas. $P < 0.05$ was considered to be significant variation in BLLs between comparisons.

CHAPTER THREE

RESULTS

3.1 Blood lead levels in the total sample

Table 6 presents Blood lead levels (BLLs) obtained for the entire sample (n = 308) ranged from 0.4 to 65µg/dL of blood. BLLs for individuals in the study areas meant for environmental assessment ranged from 1.4 to 22.0µg/dL and the lowest mean BLL recorded was 4.1 while the highest was 8.4µg/dL. The lowest and highest BLLs in µg/dL obtained for respective areas were: Kariobangi North (1.4, 22.0), Babadogo (1.5, 21.8), Waithaka (1.4, 13.0) and Pumwani (3.5, 20.3). The means±1SD recorded were as follows for individual study area:- Kariobangi North: 5.0±3.6; Babadogo: 5.8±4.1; Waithaka: 4.1±2.7 and Pumwani: 8.4±3.8µg/dL. In order of increasing prevalence, those with BLLs ≥10µg/dL were 5.8% for Waithaka, 10% for Kariobangi North, 15.2% for Babandogo and 30% for Pumwani.

The highest BLLs were recorded in Ziwani and the concentrations significantly varied to those obtained in Kariobangi North ($p < 0.001$), Babadogo ($p < 0.001$) Waithaka ($p < 0.001$) and Pumwani ($p < 0.05$). The mean BLL for the occupationally exposed (Ziwani Jua kali) was 22.6±13.4µg/dL while the lowest and highest BLLs obtained for this group were 5.8 and 65µg/dL respectively. 89% of the subjects had BLLs above 10µg/dL.

The lowest blood lead concentrations were obtained in Olkalou and the levels differed significantly ($p < 0.001$) from those recorded in other study areas. The covariate group at Olkalou had a mean BLL of 1.3±0.9µg/dL. The lowest BLL recorded in Olkalou was 0.4µg/dL and the highest 4.1µg/dL.

Table 6: Blood lead levels (µg/dL) in the total sample and prevalence of BLLs ≥ 10 µg/dL

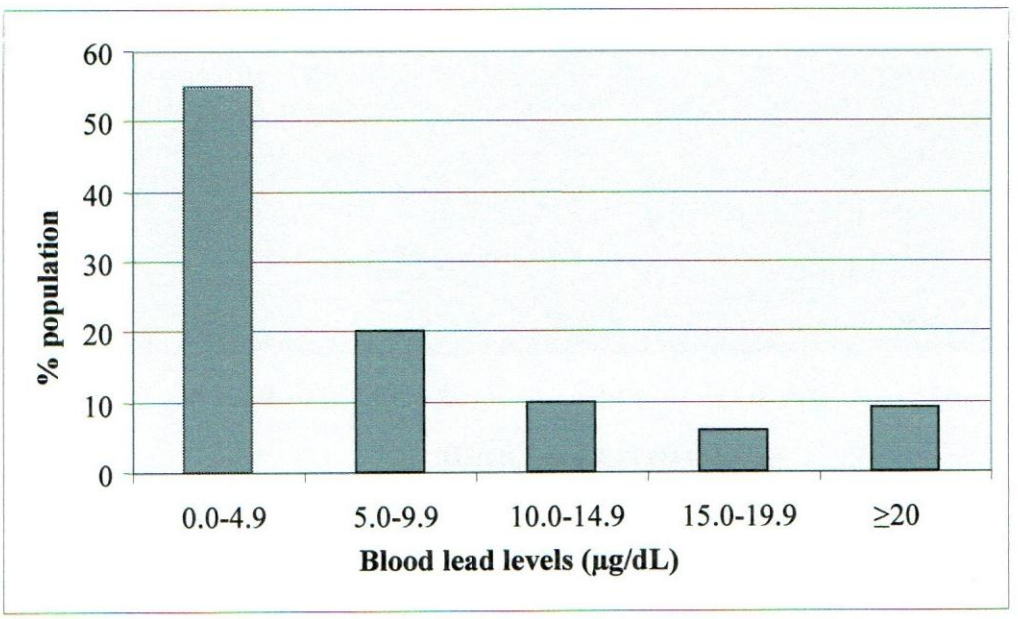
Region	n	Min	Max	Mean ±1SD	Prevalence (%) ≥10µg/dL
Kariobangi North	90	1.4	22.0	5.0±3.6	10.0
Babadogo	46	1.5	21.8	5.8±4.1	15.2
Waithaka	52	1.4	13.0	4.1±2.7	5.8
Pumwani	30	3.5	20.3	8.4±3.8[af]	30.0
Ziwani Jua Kali	55	5.8	65.0	22.6±13.4[bdgi]	89.1
Olkalou	35	0.4	4.1	1.3±0.9[cehjk]	0.0
Total sample	308	0.4	65.0	8.0±9.4	25.0

The values are expressed as BLL means ± standard deviation in µg/dL. [a] $p < 0.01$ between Kariobangi North and Pumwani; [b] $p < 0.001$ between Kariobangi North and Ziwani; [c] $p < 0.001$ between Kariobangi North and Olkalou; [d] $p < 0.001$ between Babandogo and Ziwani; [e] $p < 0.001$ between Babandogo and Ziwani; [f] $p < 0.001$ between Waithaka and Pumwani; [g] $p < 0.001$ between Waithaka and Ziwani; [h] $p < 0.001$ between Waithaka and Olkalou; [I] $p < 0.05$ between Pumwani and Ziwani; [j] $p < 0.01$ between Pumwani and Olkalou; [k] $p < 0.001$ between Ziwani and Olkalou.

3.1.1 Distribution of blood lead levels in the study population

Figure 8 presents the distribution of BLLs in the whole sample. 55 % (n = 169) of the total sample had levels equal to or below 4.9µg/dL, 20% (n = 62) of the population had levels ranging from 5.0 to 9.9µg/dL while 25% (n = 77) of the total sample had levels above the action level of 10µg/dL. Within Nairobi, 15.3% (n = 32) of the study subjects in areas meant for assessment of environmental lead exposure (i.e. Kariobangi North, Babandogo, Waithaka and Pumwani) had levels above the CDC/WHO action levels. 15% of the entire sample in Nairobi had BLLs above 15µg/dL and a big proportion of this percentage was made up of Babandogo (2%), Pumwani (4%) and the occupationally exposed at Ziwani (73%).

Figure 8: Distribution of BLLs (µg/dL) in the study population



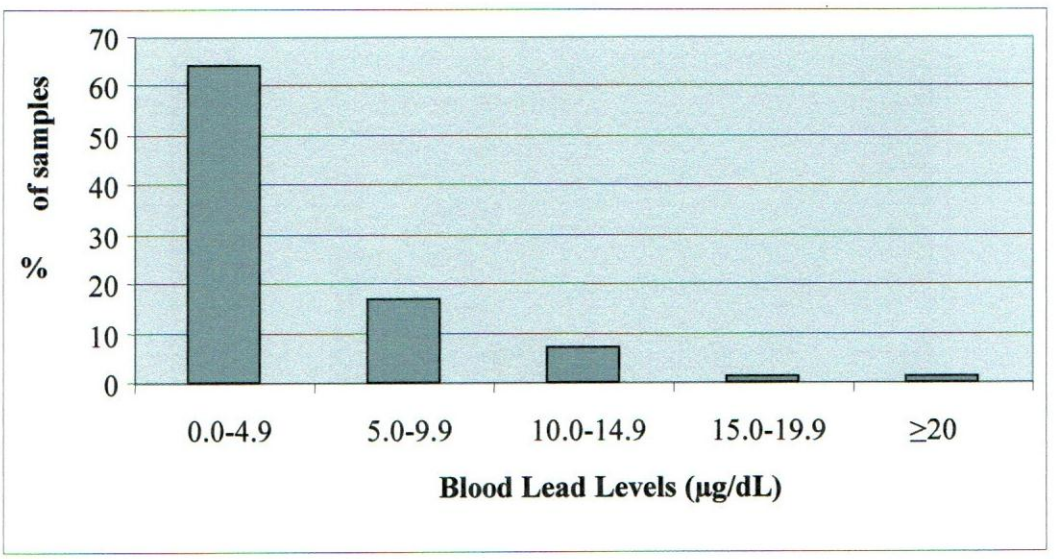
n=308
Mean= 8.0µg/dL
SD=±9.4µg/dL
Prevalence of elevated BLL (>10 µg/dL) = 25% (n = 77)

3.2 Blood lead levels in specific study areas

3.2.1 Blood lead levels in Kariobangi North

A total number of ninety subjects were recruited in this area and their blood lead levels determined. The youngest was 2.5 years and the oldest 55 years. 71.1 % of the subjects were females while males made 28.8 % of the group. Children and adolescents (≤ 20 years of age) made 40 % (n = 36) of the sample. Figure 9 shows percentage distribution of BLLs in Kariobangi North. 10 % of the sample had levels above the action level of 10µg/dL. 71 % of the sample or 64 subjects had levels equal to or below 4.9µg/dL. 29 % of the sample had levels above or equal to 5.0µg/dL. The mean BLL obtained in this study area was 5.0±3.6µg/dL.

Figure 9: Distribution of BLLs (µg/dL) in Kariobangi North

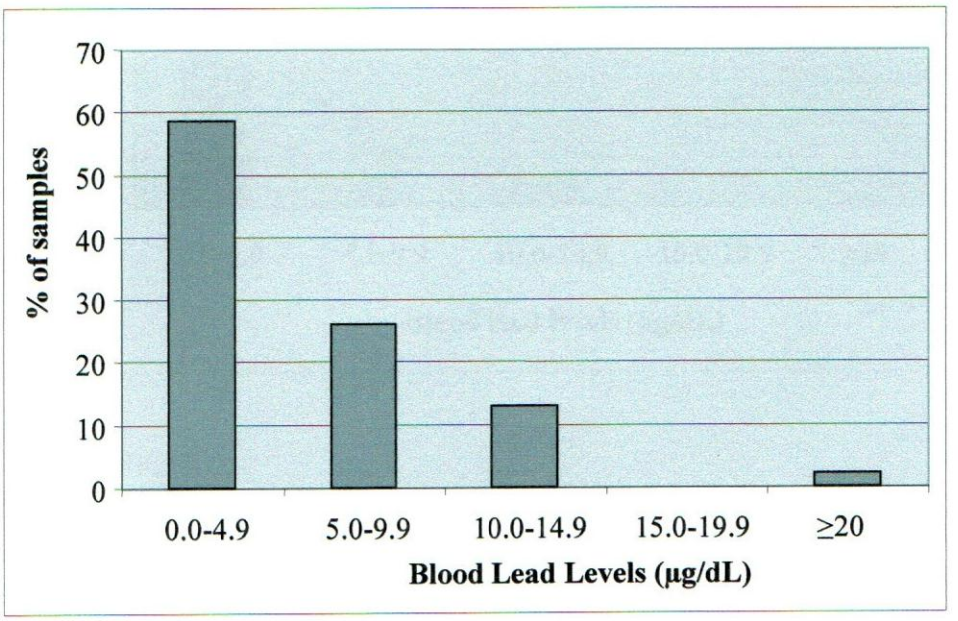


n = 90
Mean = 5.0µg/dL
SD = ±3.6µg/dL
Prevalence of elevated BLL (>10 µg/dL) = 10% (n = 9)

3.2.2 Blood lead levels in Babadogo

A total number of forty six subjects were recruited in this area and their blood lead levels determined. The youngest was 5 years and the oldest 54 years. Males made 71.7 % of the sample while females made 28.2 % of the group. Individuals below or equal to 20 years of age made 56.5 %(n = 26) of the sample. 15 % of the sample had levels above the action level of 10µg/dL. 59 % (n = 27) of the sample had levels below or equal to 4.9µg/dL (Figure 10). The mean BLL obtained for this area was 5.8±4.1µg/dL.

Figure 10: Distribution of BLLs (µg/dL) in Babadogo



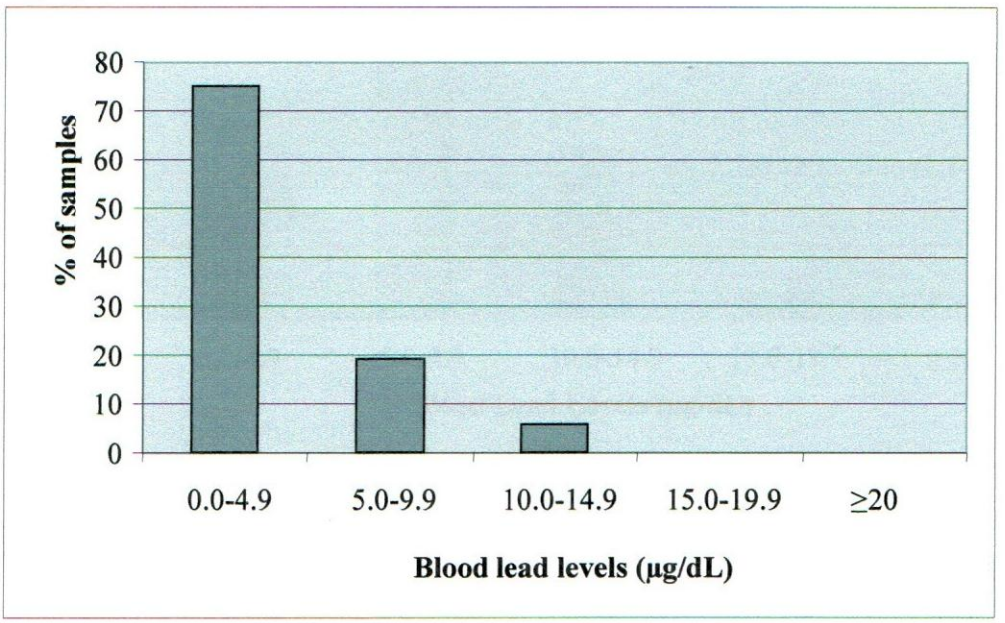
n = 46
Mean = 5.8µg/dL
SD = ±4.1µg/dL
Prevalence of elevated BLL (>10 µg/dL) = 15% (n = 7)

3.2.3 Blood lead levels in Waithaka

A total number of fifty two subjects were recruited in this area and their blood lead levels determined. The youngest was 5 years and the oldest 76 years. Females made 73.1 % of the

sample while males made 26.9 % of the group. Individuals below or equal to 20 years of age made 36.5 % (n= 20) of the sample. Figure 11 presents the distribution of BLLs in Waithaka. 6 % of the sample had levels above the action level of 10μg/dL. 75 %(n = 39) of the sample had levels below or equal to 4.9μg/dL. The mean BLL obtained in this area was 4.1±2.7μg/dL.

Figure 11: Distribution of BLLs (μg/dL) in Waithaka



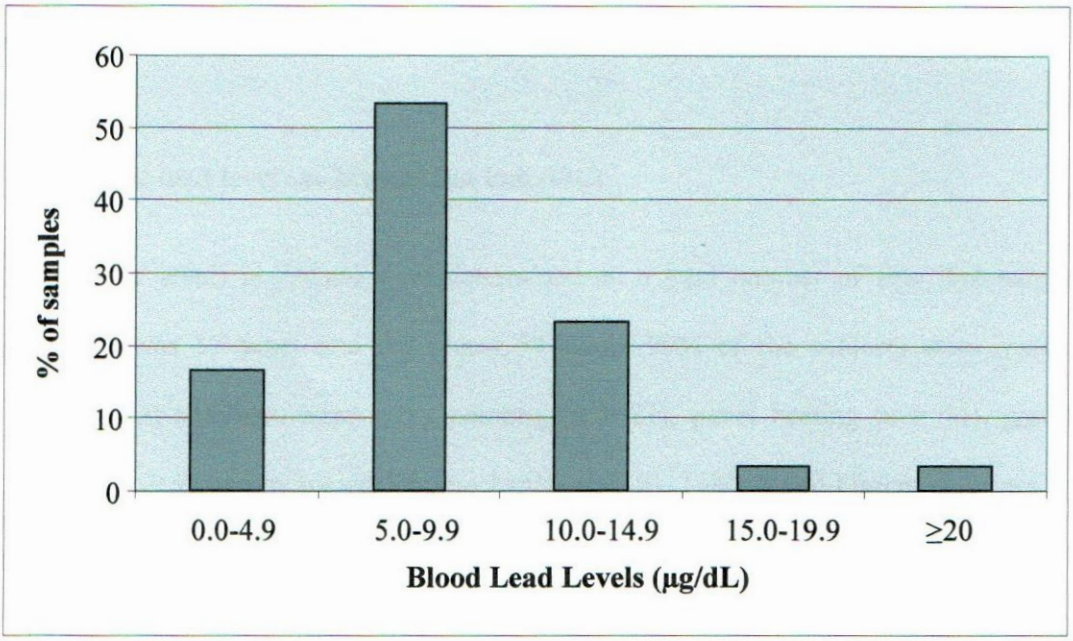
n = 52
Mean = 4.1μg/dL
SD = ±2.7μg/dL
Prevalence of elevated BLL (>10 μg/dL) = 6% (n = 3)

3.2.4 Blood lead levels in Pumwani

A total number of thirty subjects were recruited in this area and their blood lead levels determined. The youngest was 8 years and the oldest 15 years. Females made 50 % of the sample while males made the other half of the group. 30 % of the sample had levels above the action level of 10μg/dL. 82% of the subjects had levels above or equal to 5.0μg/dL. 53% of the

subjects had blood lead levels between 5.0-9.9µg/dL and 6% had blood lead levels above 15µg/dL (Figure 12). Mean BLL for Pumwani was 8.4±3.8µg/dL.

Figure 12: Distribution of BLLs in Pumwani



n = 37
Mean = 8.4µg/dL
SD = ±3.8µg/dL
Prevalence of elevated BLL (>10 µg/dL) = 30% (n = 11)

3.2.5 Blood lead levels in Olkalou

A total number of thirty five subjects were recruited in this area and their blood lead levels determined. The youngest was 4 years and the oldest 73 years. Females made 60 % of the sample while males made 40 % of the group. Individuals below or equal to 20 years of age made 28 % of the sample. All the study subjects (100%) had levels below 4.9µg/dL (Table 7) and the mean BLL was 1.3±0.9µg/dL.

Table 7: Distributions of BLLs (µg/dL) at Olkalou

BLLs(µg/dL)	0.0-4.9	5.0-9.9	10.0-4.9	15.0-9.9	≥20
% of Samples	100	0	0	0	0
n	35	0	0	0	0

3.2.6 Blood lead levels at Ziwani Jua kali (OC)

Blood lead levels at Ziwani were determined on a total number of fifty five subjects. The youngest was 17 years and the oldest 54 years. 99% of the subjects were males. Major occupational activities were spray painting (n = 21), panel beating (n = 11), gas/electrical welding (n = 7) and motor vehicle mechanics (n = 8). Table 8 and Figure 13 shows the BLLs obtained and their distribution at the Jua kali sheds in Ziwani. The only female subject in this area had a BLL of 21.7µg/dL. The mean blood lead levels were recorded as 18.1±4.2µg/dL for others (general businessmen, hawkers and milk vendors) and 25.3±17.8µg/dL for welders. Painters, panel beaters and those dealing with both spray painting and panel beating had mean BLLs of 21.8±11.6, 22.4±14.2 and 24.1±12.7 respectively.

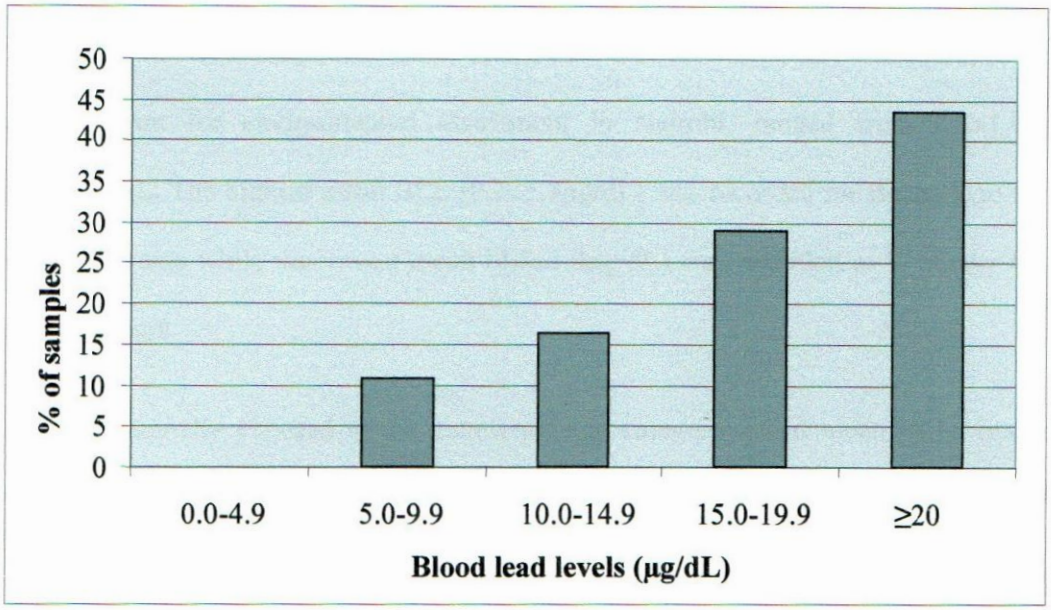
Table 8: Blood lead levels (µg/dL) at Ziwani Jua kali

Occupational activity	n	Mean ± 1SD	median	min	max	%>10µg/dL
Motor vehicle Painters	21	21.8±11.6	21.2	10	65	100
General Welders	7	25.3±17.8	17.0	11.2	58.2	100
Painters/P.beaters	3	24.1±12.7	22.3	12.4	37.6	100
General Panel beaters	11	22.4±14.2	18.3	7.9	50.8	72.7
Mechanics	8	23.0±19.0	13.9	5.8	52.9	62.5
Others	5	18.1±4.2	18.6	11.3	21.7	100
Total	55	22.5±13.4	18.6	5.8	65	89.2

The values are expressed as BLL means ± standard deviation in µg/dL. $P > 0.05$

11 % of individuals whose blood lead levels were determined in this area had BLLs below 10µg/dL. 44 % of the subjects had levels above 20µg/dL. There was no significance difference ($p > 0.05$) between the BLLs obtained for the various occupations. The mean BLL for the whole group was 22.5±13.4µg/dL.

Figure 13: Distribution of BLLs at Ziwani



n = 55
Mean = 22.5µg/dL
SD = ±13.4 µg/dL
Prevalence of elevated BLL (>10 µg/dL) = 89% (n = 49)

3.3 Comparison of blood lead levels in Nairobi and Olkalou for the entire study population

The variation in BLL at Olkalou was highly significant ($p < 0.001$) to the BLLs obtained in all the study areas in Nairobi. While the BLL obtained at Kariobangi North was statistically not different ($P > 0.05$) from those at Babandogo and Waithaka, the variation in BLLs at Kariobangi North and Pumwani was significant ($p < 0.01$). There was no significant difference in BLLs obtained at Babandogo to those obtained at Pumwani ($p > 0.05$). Waithaka had the highest concentration of low blood lead levels in Nairobi and varied significantly ($p < 0.001$) to BLLs obtained at Pumwani.

3.4 Blood lead levels by age sets

Mean BLLs for various age sets (≤ 10, 11-20, 21-30, 31-40, 40-50 and >50 years) for the entire sample meant for environmental assessment in Nairobi, ranged from 2.8±1.0µg/dL to 9.7±5.7µg/dL. The highest mean BLL (9.7±5.7µg/dL) was recorded for Babandogo for the age group ≤10 years while the lowest mean (2.8±1.0µg/dL) was recorded at Waithaka for the age set 31-40 years.

The occupationally exposed at the mentioned age categories, had mean BLLs ranging from 14.0±5.9µg/dL to 24.4±7.4µg/dL. The lowest mean was recorded for the age group 41-50 years while the highest was recorded for the age group 11-20 years. No significant variation ($p > 0.05$) in BLLs obtained in Olkalou between the various age sets was recorded. The lowest mean recorded for Olkalou was 0.9±0.6µg/dL and the highest 1.7±1.1µg/dL.

While excluding the occupationally exposed, highest mean BLLs were noted for the age group ≤ 10 years who's mean BLLs (µg/dL) in ascending order were recorded as 1.7±1.1, 6.5±3.0, 7.4±3.9, 7.9±3.7 and 9.7±5.7 for Olkalou, Waithaka, Kariobangi North, Babandogo and Pumwani respectively. There was no significant variation ($p > 0.05$) in BLLs obtained between the age sets ≤ 10 years and 11-20 years at Pumwani where the recorded means were 7.9±3.7, 8.5±3.9 µg/dL for the age sets ≤ 10 years and 11-20 years respectively. A decrease in BLLs was noted for Waithaka, Kariobangi North and Babandogo with an increase in age up to 50 years (Table 9 and Figure 14).

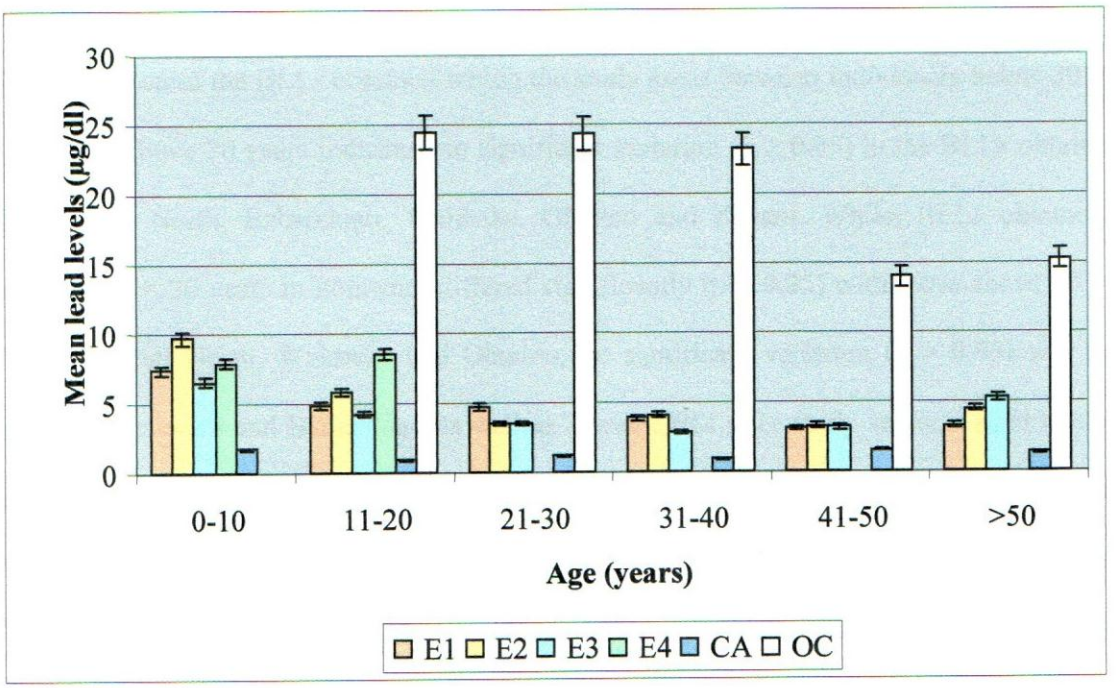
EGERTON UNIVERSITY LIBRARY

Table 9: Mean blood lead level (µg/dL) concentrations by age

Age(years)	0-10 (n = 50)	11-20 (n = 76)	21-30 (n = 89)	31-40 (n = 44)	41-50 (n = 30)	>50 (n = 19)
Kariobangi North	7.4±3.9 (n =21)	4.9±2.8 (n =15)	4.7±3.8 (n =27)	3.9±3.4[a] (n =17)	3.1±0.9 (n =5)	3.3±0.4 (n = 5)
Babandogo	9.7±5.7 (n=10)	5.8±3.6 (n=16)	3.5±0.7[a] (n =6)	4.1±0.8[b] (n = 6)	3.3±1.5[c] (n = 5)	4.5±0.8 (n =3)
Waithaka	6.5±3.0 (n = 7)	4.2±3.4 (n = 12)	3.5±1.8 (n =18)	2.8±1.0 (n =4)	3.2±0.9 (n = 7)	5.3±3.8 (n= 4)
Pumwani	7.9±3.7 (n =5)	8.5±3.9[c] (n =25)				
Olkalou	1.7±1.1[ab] (n = 7)	0.9±0.6[e] (n = 3)	1.2±0.7 (n = 7)	0.9±0.8 (n = 8)	1.5±1.3 (n = 6)	1.3±0.3 (n = 4)
Ziwani		24.4±7.4[abdfg] (n = 5)	24.3±15.0[abcd] (n =31)	23.2±14.4[abcd] (n =9)	14.0±5.9[abcd] (n =7)	15.3±2.3[abcd] (n = 3)

The values are expressed as BLL means ± standard deviation in µg/dL. [a] $p < 0.05$ between age sets ≤ 10 years and 31-40 years in Kariobangi North: $p < 0.05$ between age sets ≤ 10 years and 21-30 in Babandogo: $p < 0.05$ on comparison of BLLs obtained in Kariobangi North versus Olkalou for the age set ≤ 10 years: $p < 0.001$ Kariobangi North vs. Ziwani for age category 11-20 years: $p < 0.001$ Kariobangi North vs. Ziwani for age set 21-30 years: $p < 0.001$ Kariobangi North and Ziwani for age set 31-40 years: $p < 0.001$ Kariobangi North and Ziwani for age set 41-50 years: $p < 0.001$ Kariobangi North and Ziwani for age category above 50 years; [b] $p < 0.05$ between age sets ≤ 10 years and 31-40 years in Babandogo: $p < 0.01$ between Babandogo and Olkalou for age sets ≤ 10 years: $p < 0.001$ between Babandogo and Ziwani for age category 11-20 years: $p < 0.001$ between Babandogo and Ziwani for age set 21-30 years: $p < 0.001$ between Babandogo and Ziwani for age category 31-40 years: $p < 0.001$ between Babandogo and Ziwani for age set 41-50 years: $p < 0.001$ between Babandogo and Ziwani for age set above 50 years; [c] $p < 0.05$ between Waithaka and Pumwani for the age sets 11-20 years: $p < 0.001$ between Waithaka and Ziwani for age set 21-30 years: $p < 0.001$ between Waithaka and Ziwani for age category 31-40 years: $p < 0.001$ between Waithaka and Ziwani for age set 41-50 years: $p < 0.001$between Waithaka and Ziwani for age set above 50years; [d] $p < 0.001$ between Waithaka and Ziwani for age sets 11-20 years: $p < 0.001$ between Olkalou and Ziwani for age set 21-30 years: $p < 0.001$ between Olkalou and Ziwani for age set 31-40 years: $p < 0.001$ between Olkalou and Ziwani for age set 41-50 years: $p < 0.001$ between Olkalou and Ziwani for age set above 50 years; [e] $p < 0.05$ between Pumwani and Olkalou for age category 11-20 years; [f] $p < 0.001$ between Pumwani and Ziwani for age set 11-20 years; [g] $p < 0.001$ between Olkalou and Ziwani for age set 11-20 years.

Figure 14: Mean blood lead level (µg/dL) concentrations by age



Key to Figure 14
E1= Kariobangi north, E2= Babadogo, E3= Waithaka, E4= Pumwani, CA=Olkalou, OC= Ziwani

3.4.1 Comparison of blood lead levels for adults (> 20 years of age)

Comparison of blood lead levels for all adults (> 20 years) indicated that the blood lead concentrations obtained at Olkalou differed significantly ($p < 0.05$) with BLLs obtained for all adults in Nairobi. Likewise, blood lead concentrations in Ziwani differed significantly ($p < 0.001$) with blood lead levels obtained in all the other study areas. There was no significant difference ($P > 0.05$) in BLLs obtained in Waithaka, Kariobangi North and Babandogo for adults. A comparison of BLLs obtained in Pumwani to the rest of the study areas for adults could not be done since there were no study subjects' ≥ 20 years in Pumwani.

3.4.2 Comparison of blood lead levels for individuals below and above 20 years of age within and between study areas

A comparison of the BLLs obtained within the study areas between individuals below 20 years and those above 20 years indicated no significant variation ($p > 0.05$) in the BLLs obtained at Kariobangi North, Babandogo, Waithaka, Olkalou and Ziwani. While, BLLs obtained for individuals < 20 years in Pumwani differed significantly ($p < 0.05$) with those above 20 years in Kariobangi North, Waithaka and Olkalou, no significant variation ($p > 0.05$) was noted between Pumwani and Babandogo as well as Ziwani. BLLs for study subjects aged below 20 years in Ziwani varied significantly ($p < 0.05$) to the blood lead values obtained for individuals above 20 years in Kariobangi North, Babandogo, Waithaka and Olkalou. Blood lead levels for individuals under 20 years in Olkalou did not have any significant variation ($p > 0.05$) with those obtained in Kariobangi North, Babandogo and Waithaka for individuals aged 20 years and above but differed significantly ($p < 0.05$) with those at Ziwani aged ≥ 20 years.

3.5 Blood lead levels for children and adolescents (≤ 20 years)

Subjects in the entire study sample (n = 126) aged 20 years and below had blood lead levels ranging from 0.4 to 35.6µg/dL. Children and adolescents in Kariobangi North (n = 36), Babadogo (n = 26), Waithaka (n = 20) and Pumwani (n = 30) had BLLs ranging from 1.4 to 21.8µg/dL. The lowest and highest BLLs (µg/dL) obtained for individual study areas in Nairobi for children and adolescents were recorded as: Kariobangi North (2.1; 16.5), Babandogo (1.5; 21.8), Waithaka (1.4; 13.0) and Pumwani (3.5; 20.3). Mean±1SD BLLs recorded for children and adolescents were 6.3±3.7, 7.3± 4.8, 5.1 ±3.4 and 8.4 ±3.8µg/dL for Kariobangi North, Babandogo, Waithaka and Pumwani respectively. The covariate group at Olkalou (n = 10) had a mean BLL of 1.5±1.0µg/dL and the BLLs ranged from 0.4µg/dL to

3.8µg/dL. Only 9% (n = 5) of the occupationally exposed study subjects were equal to or below 20 years of age. Actual BLLs obtained for the five were 35.6, 22.1, 15.7, 27.0 and 21.7µg/dL and the mean was 24.4±7.4µg/dL.

In order of increasing prevalence, individuals in the areas meant for assessment of environmental exposure in Nairobi, whose blood lead levels exceeded 10µg/dL and 5µg/dL in specific study areas were; Waithaka (10%, 40%), Kariobangi North (17%, 44%), Babandogo (27%, 62%) and Pumwani (30%, 83%). In general and excluding individuals occupationally exposed, 21% and 57.3% of study subjects up to 20 years in Nairobi had BLLs exceeding 10µg/dL and 5µg/dL respectively. All (100%) of the occupationally exposed had levels above 10µg/dL. None (0%) of the subjects at Olkalou had levels above 5µg/dL (Table 10).

Table 10: Blood lead levels (µg/dL) for individuals' ≤ 20 years in the total sample

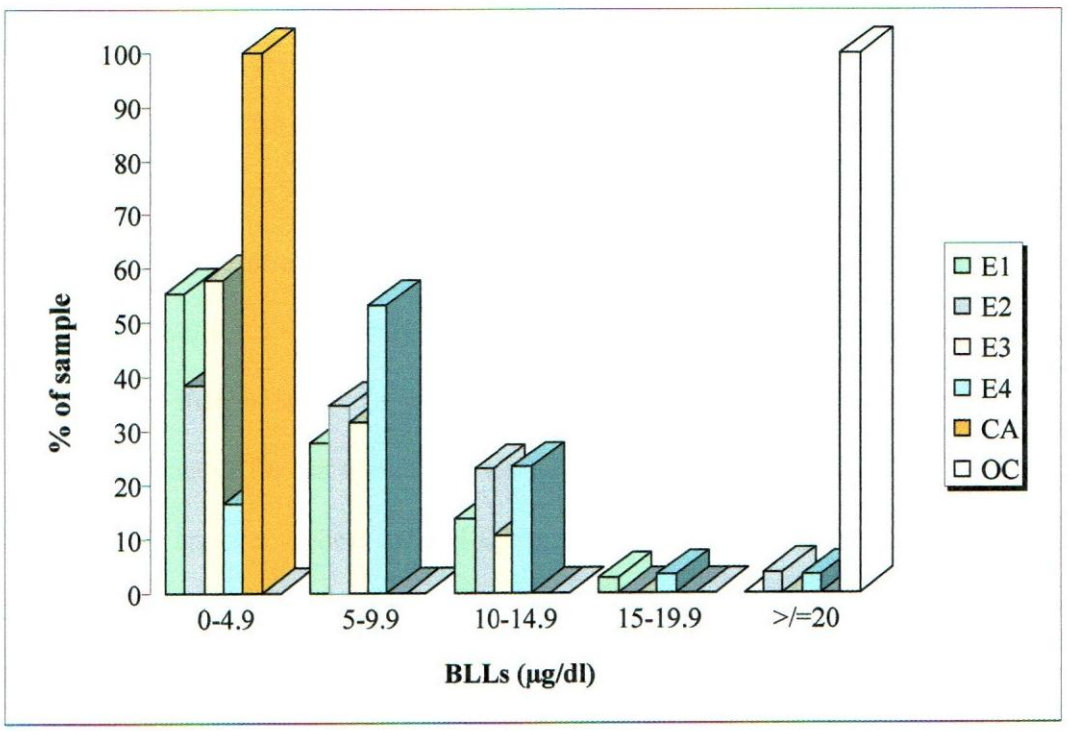
Area of Study	n	Mean±1SD	Mode	% >10µg/dL	% >5µg/dL	min Bll	max Bll
Kariobangi North	36	6.3±3.7	4.5	17.0	44.0	2.1	16.5
Babadogo	26	7.3±4.8	3.6	27.0	62.0	1.5	21.8
Waithaka	19	5.1±3.4	3.2	10.0	40.0	1.4	13.0
Pumwani	30	8.4±3.8	8.3	30.0	83.0	3.5	20.3
Ziwani Jua Kali	5	24.4±7. 4[c]	N/A	100.0	100.0	15.7	35.6
Olkalou	10	1.5±1.0[abde]	0.8	0.0	0.0	0.4	3.8

The values are expressed as BLL means ± standard deviation in µg/dL. [a] $p < 0.01$ between Kariobangi North and Olkalou; [b] $p < 0.01$ between Babandogo and Olkalou; [c] $p < 0.05$ between Waithaka and Ziwani; [d] $p < 0.001$ between Pumwani and Olkalou; [e] $p < 0.001$ Olkalou and Ziwani.

3.5.1 Distribution of blood lead levels for children and adolescents

Figure 15 shows the distribution of blood lead levels for individual ≤ 20 years in the entire sample. The distribution of blood lead levels for assessment of environmental exposure in Nairobi was positively skewed. 17%, 38%, 56% and 58%in Pumwani, Babandogo, Kariobangi North and Waithaka respectively had levels ≤ 4.9µg/dL. The highest percentage (53%) of study subjects having BLLs between 5.0 to 9.95µg/dL was recorded at Pumwani while the lowest (28%) was recorded in Kariobangi North. For individuals who's BLLs ranged from 10 to 14.9µg/dL, Babandogo and Pumwani each had 23% while Kariobangi North and Waithaka had a prevalence of 14% and 11% respectively. 1.6% of the total sample in Babandogo, Pumwani, Kariobangi North and Waithaka had BLLs above 15µg/dL.

Figure 15: Distribution of blood lead level (µg/dL) for individuals' ≤ 20 years



Key to Figure 15
E1= Kariobangi north, E2= Babadogo, E3= Waithaka, E4= Pumwani, CA=Olkalou, OC= Ziwani

3.5.2 Comparison of blood lead levels between study areas for individuals' ≤ 20 years of age

There was no significant variation ($p > 0.05$) of blood lead levels obtained for children and adolescents (≤ 20 years) in Kariobangi North to those recorded in Babandogo, Pumwani and Ziwani. Likewise, BLLs in Babandogo for adolescents and children did not vary significantly ($p > 0.05$) with those obtained in Waithaka, Pumwani and Ziwani. However, BLLs obtained at Ziwani varied significantly ($p < 0.05$) with BLLs obtained at Waithaka. There was no significant variation ($p > 0.05$) in BLLs obtained in Waithaka to those obtained at Olkalou for this age group. BLLs obtained for the children in Pumwani were not significantly different ($p > 0.05$) to those obtained in Ziwani.

3.6 Blood lead levels by age sets (≤ 20 years)

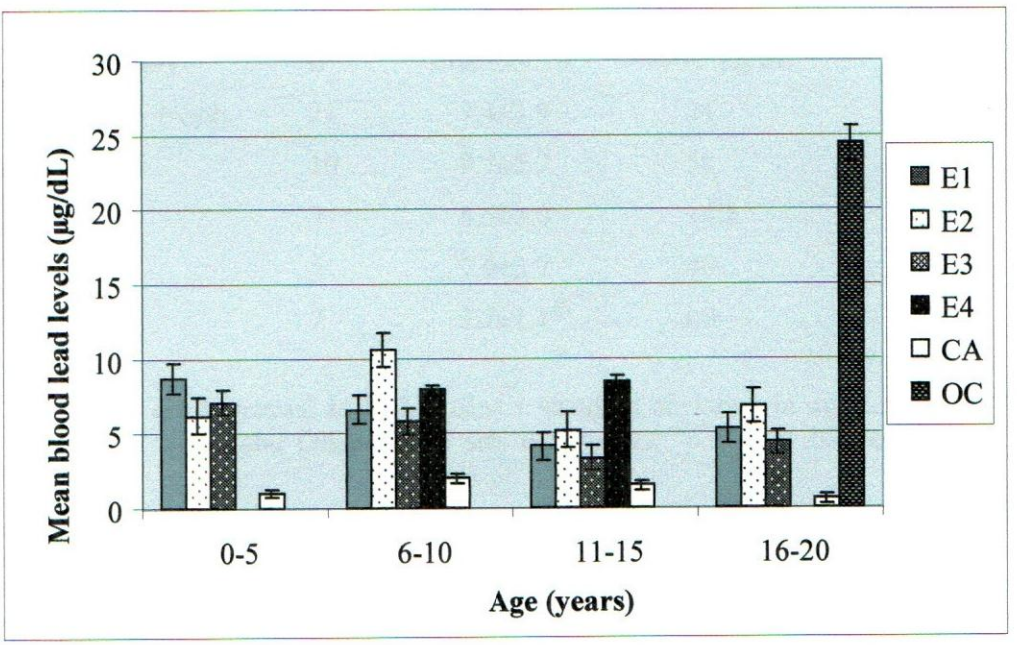
Table 11 and Figure 16 shows the mean BLLs grouped in age categories (≤ 5, 6-10, 11-15 and 16-20 years) within the study subjects of 20 years and below. Mean BLLs ranged from 4.1 to 10.6μg/dL. The lowest and highest means (μg/dL) in age categories for individual study areas in Nairobi were: Kariobangi North (4.1±0.2, 8.7±4.5), Babadogo (5.2±3.1, 10.6±5.9) and Waithaka (3.3±0.1, 7.1±4.0). Only two age categories: 6-10 and 11-15 years were identified for Pumwani and their mean BLLs were recorded as 7.9±3.7 and 8.5±3.9μg/dL respectively. In Olkalou, the means for the age categories were almost the same with the lowest mean being 0.9±0.6μg/dL and the highest 1.7±1.1μg/dL. The only age category which encompassed the occupationally exposed group was 16 to 20 years and the mean was recorded as 24.4±7.4μg/dL.

Table 11: Mean ±1SD (µg/dL) blood lead levels by age sets for subjects ≤ 20 years

Age (years)	**0-5**	**6-10**	**11-15**	**16-20**
	(n =16)	**(n =34)**	**(n = 42)**	**(n = 34)**
Kariobangi North	8.7±4.5	6.6±3.1	4.1±0.2	5.3±3.3
	(n = 8)	(n= 13)	(n=5)	(n = 10)
Babandogo	6.2±3.6	10.6±5.9	5.2±3.1	6.8±4.3
	(n = 2)	(n = 8)	(n = 10)	(n = 6)
Waithaka	7.1±4.0	5.8±3.0	3.3±0.1	4.4±3.7
	(n = 4)	(n = 3)	(n = 2)	(n =11)
Pumwani		7.9±3.7	8.5±3.9	
		(n= 5)	(n =25)	
Olkalou	1.0±0.6 9	1.7±1.1[a]		0.9±0.6
	(n = 2)	(n = 5)		(n = 2)
Ziwani				24.4±7.4[abcd]
				(n = 5)

The values are expressed as BLL means ± standard deviation in µg/dL. [a] $p < 0.01$ between Babandogo and Olkalou age sets 6-10 years: $p < 0.001$ between Kariobangi North and Ziwani for the age category 16-20 years; [b] $p < 0,001$ between Babandogo and Ziwani for individuals of 16-20 years; [c] $p < 0.001$ between Waithaka and Ziwani for individuals of 16-20 years; $p < 0.001$ between Olkalou and Ziwani for individuals of 16-20 years.

Figure 16: Mean blood lead levels by age sets (≤ 20years)



3.7 Blood Lead levels for children aged ≤ 10 years in Kariobangi North, Babadogo, Pumwani, Waithaka and Olkalou

Table 12 presents the mean blood lead levels for study subjects of ten years and below. A total number of 50 individuals' ≤ 10 years were recruited and their BLLs determined. There were no children in this age set that were studied in Ziwani. 32% of the children ≤10 years had BLLs exceeding 10µg/dL. In individual study areas within Nairobi, the mean BLLs and percentage of children in this age category with BLLs > 10µg/dL in ascending order, were reported as: Waithaka (6.5±3.0; 14.2%), Kariobangi North (7.4±3.9; 24%), Pumwani (7.9±3.7; 40%) and Babandogo (9.7±5.7; 50%). 65.7% of the children in Kariobangi North, Babandogo, Pumwani and Waithaka ≤10 years had levels above 5µg/dL while non in Olkalou exceeded this level.

Table 12: Blood lead levels for children ≤ 10 years for environment assessment areas in Nairobi and Olkalou

Area of study	n	Mean±1SD	%>10 µg/dL	% > 5 µg/dL
Kariobangi North	21	7.4±3.9	24	61.9
Babandogo	10	9.7±5.7	50	70
Waithaka	7	6.5±3.0	14.2	71
Pumwani	5	7.9±3.7	40	60
Olkalou	7	1.7 ± 1.1^{ab}	nil	nil

The values are expressed as BLL means ± standard deviation in µg/dL. $^{a}p < 0.05$ between Kariobangi North and Olkalou age sets 6-10 years; $^{b}p < 0.01$ between Babandogo and Olkalou.

3.8 Blood lead levels by sex

Table 13 and Figures 17 and 18 presents BLLs by sex in the entire sample, those ≤ 20 years and individuals above 20 years. There was no significant difference ($p > 0.05$) in the BLLs obtained between males and females for the entire sample as well as between adults and children (≤ 20 years). However, significant variation ($p < 0.05$) was recorded between the sexes in the proportion of females and males who had levels above 10µg/dL. Compared to females, 41% of males had BLLs above 10µg/dL while only 9% of the females had elevated BLLs within the entire sample.

Table 13: Mean±SD (µg/dL) BLLs for females and males for the entire study sample

Study area	All ages		≤ 20 years (n=126)		> 20 years (n = 182)		total
sex	female	male	female	male	female	male	
n	152	156	73	53	79	103	308
Mean±1SD	7.8±9.4	7.9±9.4	8.1±9.4	6.7±6.7	4.8±3.6	7.9±9.4	8.0 ±9.4
%>10 µg/dL	8.6(n=13)	41(n=64)	16.4(n= 12)	32(n =17)	1.3(n= 1)	45.2(n =47)	25(n =77)
P value	P > 0.05		P > 0.05		P > 0.05		
P value (≤ 20 years vs. > 20 years)			P > 0.05				

Figure 17: Percentage of males and females (total sample) with BLLs above 10µg/dL

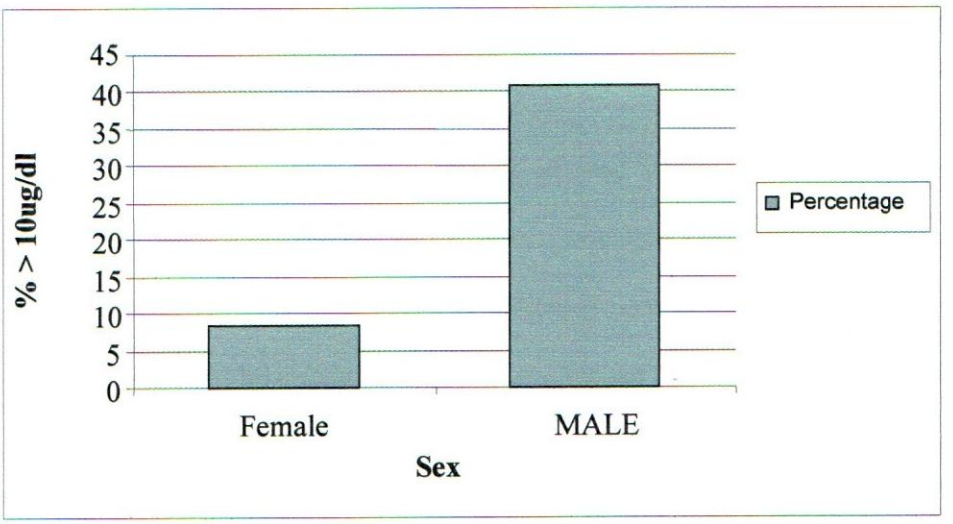
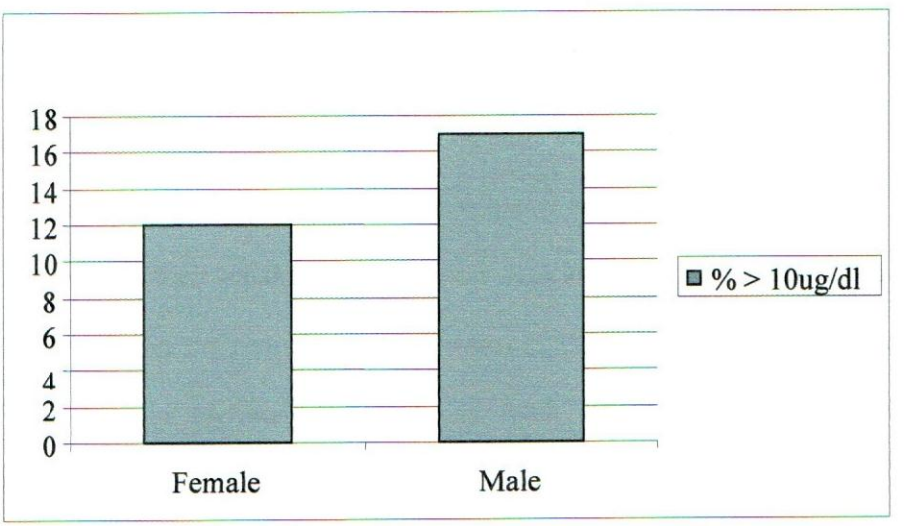


Figure 18: Percentage of males and females ≤ 20 years with BLLs above 10µg/dL



CHAPTER FOUR

DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

4.1 Discussion

Human exposure to unacceptable levels of lead within environmental and occupational setups has been established from the results obtained in this study. However, geographical distribution of BLLs obtained, largely indicated nonexposure in Olkalou where none of the study subjects had levels above 10μg/dL as opposed to Nairobi where significant exposure is indicated with 15% of the population having elevated blood lead levels. The results equally indicate a clear distinction of exposure levels between the environmental and occupational setups.

Wide spread human exposure to environmental lead in Nairobi has been demonstrated with the prevalence of lead poisoning in Nairobi shifting from low to high, on a rural-urban to urban trend. While the prevalence of elevated levels was lowest in Waithaka-the rural-urban setup in Nairobi, it was highest in Pumwani which is at close proximity to Nairobi's central business district. It was expected that blood lead levels in Babandogo and Kariobangi as attributed to their proximity to industries such as plastic, paint and steel industries, burning of solid waste in the nearby dumping waste at Dandora as well as vehicular traffic along the Thika-Nairobi highway and Outering road, would be higher than those in Pumwani. However, blood lead levels obtained at Pumwani were significantly elevated than those found in other study areas within Nairobi. A major predictor of elevated BLLs in Pumwani could be attributed to exposure to airborne lead emissions from vehicles as there are no industries in Pumwani. Although the trend in BLLs shifts according to a change in the density of vehicular traffic, other sources of exposure are likely and should be investigated.

Comparatively, the trend in lead exposure levels was similar to what was reported in china (Zheng 1999) where it was noted that although childhood lead poisoning was widespread in china due to industrialization and use of leaded petrol, there was evidence of an increase in blood lead levels relative to a rapid increase of vehicular traffic. The study which was conducted on children indicated that children residing in industrial areas and close to high vehicular traffic had blood lead levels ranging from 21.8 to 67.9µg/dL. 64% to 99% of children had levels above 10µg/dL which was also observed in areas that were not industrialized. Although the prevalence of high exposure is low compared to what was reported in china, the same observations can be accorded to the study in Nairobi where the levels in Pumwani could be associated with high vehicular traffic as compared to Waithaka where traffic is relatively low.

The results obtained for the Nairobi study indicates a lower prevalence of elevated blood lead levels than was reported in South Africa (Mathee *et al.* 2002). The South African study reported blood lead levels in children ranging from 1.0 to 44.4µg/dL (mean = 6.9±3.27µg/dL) while in Nairobi the levels of children (≤ 20 years) ranged from 1.4 to 21.8µg/dL (mean = 7.9±9.4µg/dL) which indicates high levels in South Africa than Nairobi. However, the prevalence of children above 10µg/dL and 5µg/dL in South Africa was reported as 17% and 78% respectively while in Nairobi, 21% and 57.3% of children had levels above 10µg/dL and 5µg/dL respectively. Blood lead levels for younger children (≤ 10 years) in Nairobi ranged from 2.6 to 21.6µg/dL and the proportion above 10µg/dL was 32%, an indication of less poisoning in Nairobi than in South Africa. By individual study sites, mean BLL, peak BLL and the percentage of the population above 10µg/dL in individual study sites obtained in Kariobangi North, Babandogo, Waithaka and Pumwani were lower than those reported by Mathee *et al.* 2002 for Johannesburg (9.1±3.1µg/dL; 44.4µg/dL;

35%), Capetown (6.4±2.9µg/dL; 24.5µg/dL; 10%), Kimberley (7.1±2.7µg/dL; 22.6µg/dL; 10%), Pella and Onseepkans (6.1±2.5µg/dL; 17.1µg/dL; 7.6%). The determining factors in blood lead levels observed in South Africa were established as vehicular density, proximity to source and socioeconomic status and these factors should be evaluated to determine their contribution to the observed blood lead levels in Nairobi.

When the BLLs were compared with age, a decrease in blood lead levels relative to an increase in age was observed which indicates age as a risk factor in lead exposure. The levels obtained for children are a clear indicator that a big proportion of children are exposed to high levels of lead in the environment. Of major concern is the indication that 15.6% of children below five years of age and 32% of children below 10 years had levels above 10µg/dL. This are the ages when lead absorption into the body and development of body organs and systems are at a peak thus most of the children are exposed to the risk of developing impairment in neuropsychological development and deficiency in cognitive functions. A bigger sample would give a better representation of the status on children.

In comparison, the findings in Nairobi indicated high level of exposure in Nairobi for children $\leq$ 10 years than a multinational study carried out in the Middle East in the period from 1996-2000. The study by Safi *et al.* (2006) described a distribution of blood lead levels of children aged 2-6 years depending on factors such as proximity to local small industries, distance from major roads, vehicular density and social economic status. Apart from vehicular emission, the study which was carried out in Israel, Westbank, Gaza and Jordan to assess childhood lead poisoning, identified other sources of exposure to be releases from industries such as smelters, battery factories and radiator shops, burning of solid wastes and flour from traditional stone mills. While the study in Nairobi reported a prevalence of 32% of

children ≤ ten years as having blood lead levels above 10μg/dL, highest levels reported in the Middle East were noted in Gaza (n= 435) where a mean level of 8.6μg/dL was obtained and 17.2% of children had levels above 10μg/dL. The mean, peak and percentage above 10μg/dL were 4.6μg/dL, 25.7μg/dL and 5.2% respectively. Jordan (n=382) had the lowest prevalence (<1%) of children above 10μg/dL in the region. The sampling in Jordan was done in a Maternal Child Health Care (MCH) facility that was 200 meters from the road. The highest level reported was 39.3μg/dL and the mean 3.2μg/dL. The sampling setup in westbank was similar to that of Jordan but the MCH facility was nearer to the heavy traffic at half a distance that of Jordan. The mean, peak and prevalence of elevated levels in west bank were reported as 4.2μg/dL, 25.7μg/dL and 5.2% respectively. The target population in Israel (n = 317) were those living near heavy traffic density and industries dealing with lead and low socioeconomic standards. Although 99% of the population was urban based, lower levels were reported compared to other regions. 2.2% of the children were reported as having blood lead levels above 10μg/dL and a general mean of 3.2 and a maximum of 18.2 was recorded.

Although all the sexes in the study were largely subjected to the same environmental conditions and there was no overall variation in blood lead levels between males and females, the percentage of females having elevated blood lead levels was quite low compared to men for both adults and children. This observation could be attributed to the blood lead concentrations obtained at Ziwani where almost all of the study subjects were males and 89% had levels above 10μg/dl. However, the study indicates males likely to be exposed to high levels of lead than females and mostly through occupations. The 9 % of adult females whose blood lead levels exceeded 10μg/dl is an indicator of an extension of exposure risks to fetal development and pregnancy outcomes.

Despite the low number of children below 5 years that was included in the Nairobi study, the results obtained indicated less exposure than a similar study carried out in Mexico City on 200 children aged below 5 years (ATSDR 2005). In Mexico, blood lead levels ranged from 1-31μg/dL and the mean was 9.9μg/dL. 44% of children above one and a half years had levels above 10μg/dL while in Nairobi, blood lead levels ranged from 3.3 to 16.5μg/dL and 15% had above 10μg/dL. Major predictors of blood lead levels in Mexico were identified as lead content in ceramic glazes used in the preparation of children's food, exposure to ambient air lead pollution from vehicles and dirt on children's hands.

Accessibility to major industries dealing with lead production or use during manufacture of various industrial products was not possible as consent to have access to the workers could not be obtained. Reasons given for consent denial were incitement of workers which would result to mitigations and a fear that the industries were to be investigated on workplace practices and conditions. Nevertheless, the various activities carried out at the Jua kali and the BLLs obtained have demonstrated the contribution of various activities to lead poisoning occupationally. Some of the workers had levels $\geq$ 40μg/dL which would warrant removal from work. However, workers removal from their jobs at the Jua Kali maybe difficult since most of the workers engaged in these activities are self employed and this could be their only source of daily livelihood. Ten of the workers were referred to a physician for further evaluation and management. Five of the workers had levels of 65, 58.2, 52.9,50,8 and 42.8μg/dL which place the workers at high risks of developing renal failure. Although there was no significance difference between the various activities at the Jua kali, major source of exposure can be attributed to paint as evidenced by the levels obtained for spray painters who had a mean of 21.8μg/dL.

The variations in blood lead levels observed between the occupationally and non-occupationally exposed groups in Nairobi, although at higher levels are similar to what Ferah *et al.* (2002) reported in a study carried out in Turkey. The study on adolescent workers in an auto repair workshop (n=39) who included auto electricians, repairmen, painters and bonnet straighters, 13 adults in a battery production unit and a rural covariate group (n=29), reported the levels for the auto repair workers (mean: 8.13±7.41μg/dL) to be significantly high than the rural population (mean: 3.49±1.39μg/dL). However, the levels obtained for workers in the battery manufacturing unit (mean: 25.7±9.82μg/dL) were higher than those obtained for the auto repair workers. The research findings of the Nairobi study is in agreement with other findings that numerous workers are at a major risk of occupational lead exposure (ATSDR 2005).

The workers at the Jua kali sheds are industrial end product users and the situation in the people involved in manufacture of paints, welding rods, metal and steel bars as well as battery manufacture and lead sheet production could be worse. Lead dust taken home by lead exposed workers is a source of exposure to children and should be given a lot of concern. The high exposure to the workers could also be attributed to lack of knowledge concerning lead and the danger to their health, poor working conditions, and lack of protective gear. Most of the motor vehicle spray painting is either done within a building where ventilation is inadequate and air purification equipment is lacking or in the open. While the environment within the spraying chamber exposes the workers to high concentration of lead in paint, spraying in the area exposes all within the vicinity due to dissemination of aerosols into a wide area. Those not directly involved in lead associated activities but spend most of their time in this areas exhibit high levels of blood lead indicating high environmental exposure within the vicinities.

4.2 Conclusion

This study has demonstrated that there is significant exposure to lead both from the environment and occupational setups and hence a risk to public health which should be addressed. While the population in Nairobi is exposed to high levels of lead in the environment, there is no exposure that was indicated in Olkalou. The prevalence of blood lead levels above the WHO value has been demonstrated to be higher in Nairobi while the prevalence in Olkalou was low than was anticipated. For instance, while the anticipated level of lead poisoning was placed at 20% for Kariobangi North and Babandogo, the results indicate a prevalence of 25% for the two areas. Likewise, the proportion of the occupationally exposed was placed at 5% but has come out as 89%. Overall, 21 % of children and adolescents studied in Nairobi had blood lead levels >10µg/dL that puts them at risk of impairment in neuropsychological development, hyperactivity, violent behavior, poor school performance and other social and health inadequacies as a result of being exposed to unacceptable levels of lead. Due to limitations in facilities, time and funds, it was not possible to evaluate a classical representative sample for the Nairobi region and target other industrial hot spots of lead exposure. However, the results obtained serves as an indicator on lead exposure to the general urban population in Nairobi but should not be construed to be the highest or lowest within Nairobi.

Occupational lead exposure has been identified to be at worrying levels and majority of industrial workers could be suffering from undetected complications due to elevated BLLs. Human exposure to lead within the informal industrial setup is high than the environmental setup.

4. 3 Recommendations

In line with the findings of this study, it is recommended that there is a need to put in place measures geared towards prevention and eradication of exposure to unacceptable levels of lead. It is recommended that;

i. Based on the scope of this study, further research should be conducted to establish other sources of lead and their contribution to human lead exposure in Kenya.

ii. An assessment of the extent of childhood lead poisoning in all major urban centers in Kenya should be carried out and appropriate measures taken.

iii. Evaluation and establishment of air quality standards in and around industries dealing with lead or lead containing substances in their processes be instituted.

iv. Institutionalization of a general public awareness programme on lead and related hazards should be done. This entails an interdisciplinary approach, bringing together the Ministries of Health, Environment and Housing, National Environment Management Authority (NEMA), children welfare organizations and all other concerned authorities in combating lead exposure.

v. A followup of the Jua kali workers with elevated blood lead levels; as per the recommended protocol on lead poisoning, should be done and the workers advised on possible removal from work as indicated medically. Blood lead level determination for all the workers in the Jua kali should be done to detect all cases of elevated blood lead levels that may require medical intervention.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR) (1992). Case studies in Environmental Medicine: lead toxicity. Atlanta, GA: US Department of Health and Human Services, Public Health Service. www.atsdr.cdc.gov

Agency for Toxic Substances and Disease Registry (ATSDR) (1992). Effects of inorganic lead poisoning on children and adults-lowest observable adverse health effects. Atlanta, GA: US Department of Health and Human Services, Public Health Service. www.atsdr.cdc.gov

Agency for Toxic Substances and Disease Registry (ATSDR) (2005). Toxicological profile for lead. (Draft for Public Comment) Atlanta, GA: US Department of Health and Human Services, Public Health Service. www.atsdr.cdc.gov

Alexander H, Checkoway H, van Netten C (1996). Semen quality of men employed at a lead smelter. *Occupation and Environmental Medicine* **53**:411-416.

Awad H, EL Safty lAM, EL-barbary M, Imam S (2002). Evaluation of renal Glomerular and Tubular function and structural integrity in neonates. *American Journal of Medical Sciences* **324(5)**:261-266.

Baghurst PA, Robertson EF, McMichael AJ (1987). The Port Pirie cohort study: lead effects on pregnancy outcome and early childhood development. *Neurotoxicology* **8**: 395-401.

Bennett WM (1985). Lead nephropathy. *Kidney International* **28**:212-220.

Benowitz NL (2001). Antihypertensive agents. ***In***: Katzung BG (Ed); Basic and Clinical Pharmacology; 8th edition. pp 155-157.

Bergdahl IA, Gerhardsson L, Schütz A, Desnick RJ, Wetmur JG, Skerfving S (1997). Delta-aminolaevulinic acid dehydratase polymorphism: Influence on lead levels and kidney function in humans. *Archives of Environmental Health* **52(2)**:91-96.

Brewster DC, Perazella MA (2004). A review of chronic lead intoxication: an unrecognized cause of chronic kidney disease. *American Journal of Medical Science* **327(6)**:341-347.

Canfield RL, Henderson CR, Cory-Sletchta DA, Cox C, Jusko TA, Lanphear BP (2003). Intellectual impairment in children with blood lead levels below 10μg per deciliter. *New England Journal of Medicine* **348**:1517-1526.

Carter MA (1992). Gout. ***In***: Price SA, Wilson LM (Eds); Pathophysiology: Clinical concepts of disease processes; 4th edition. pp 986-989.

Centers for Disease Control and Prevention (CDC) (1991). Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta: US Department of Health and Human Services. http://www.cdc.gov/

Centers for Disease Control and Prevention (CDC) (1997a). Screening young children for lead poisoning: guidance for state and local public health officials. Atlanta: US Department of Health and Human Services. http://www.cdc.gov/

Centers for Disease Control and Prevention (CDC) (2005). Preventing lead poisoning in young children: a statement by the Centers for Disease Control and Prevention. Atlanta, USA. http://www.cdc.gov/

Chatterjea MN, Shinde R (2002). Digestion and absorption of carbohydrates. Text book of Medical Biochemistry. pp 315-323.

Chatterjea MN, Shinde R (2002). Metabolism of purines and pyrimidines. Text book of Medical Biochemistry. pp 211-213.

Dods RF (1996). Diabetes Mellitus. ***In***: Kaplan LA, Pesce AJ (Eds); Clinical Chemistry: Theory, Analysis and correlation; 3rd edition. pp 613-641.

EL-Safty lAM, Afiti AMH, Shouman AE, EL-Sady AKR (2004). Effects of smoking and lead exposure on proximal tubular integrity among Egyptian industrial workers. *Archives of Medical Research* **35**:59 - 65.

Ernhart CB, Wolf AW, Kennard MJ (1986). Intrauterine exposure to low levels of lead: the status of the neonate. *Archives of Environmental Health* **41**:287-291.

Ferah S, Osman D, Hulk MO, Ahmet KN, Caner K, Serg M (2002). Lead exposure and urinary N-acetyl-β~D-glucosaminidase activity in adolescent workers in auto repair workshop. *Journal of Adolescent Health* **30**: 213 - 216.

First MR (1996). Renal function. ***In***: Kaplan LA, Pesce AJ (Eds); Clinical Chemistry: Theory, Analysis and correlation; 3rd edition. pp 485-493.

Fischbein A (1992). Occupational and Environmental lead exposure. ***In***: Rom WN(Ed); Environmental and Occupational medicine 2nd edition. Boston: Little Brown: pp 735-758.

Fulton M, Raab G, Thomson G (1987). Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet* **1**:1221-1226.

Galagher - Legak S (1998). Renal failure. ***In***: Perth CM (Ed). Pathophysiology: Clinical Concepts of altered Health States 5th edition pp 670-672.

Gennart PH, Buchet JP, Roels H (1992). Fertility of male workers exposed to cadmium, lead or manganese. *American Journal of Epidemiology* **135**:1208-1219.

Goyer RA (1985). Renal changes associated with lead exposure. ***In***: Mahaffey KR(Ed); Dietary and Environmental lead: Human Health Effects. Elsevier. New York. pp 315-338.

Goyer RA (1988). Lead. ***In***: Sigel S (Eds); Handbook on Toxicity of Inorganic Compounds. Seiler HG, Marcel Decker Inc: Newyork, pp 359-382.

Goyer RA (1996). Results of lead research: Prenatal exposure, neurological Consequences. *Environmental Health Perspective* **104**:1050-1054.

Hu H (1991). Knowledge of diagnosis and reproductive history among survivors of childhood plumbism. *American Journal of Public Health* **81**:1070-1072.

International Agency for Research on Cancer (IARC) (1987). Monographs on the evaluation of the carcinogenic risk of chemicals to humans: overall evaluations of carcinogenicity. Supplement 7: an updating of the IARC monographs, volumes **1-42**: 230-232

Keogh JP (1992). Lead. ***In***: Sullivan JB Jr, Krieger GR (Eds); Hazardous materials toxicology: clinical principles of environmental health. Baltimore: Williams & Wilkins. pp 834-844.

Kim R, Rotnitzky A, Sparrow D (1996). A longitudinal study of low-level lead exposure and impairment of renal function. The normative aging study. *Journal of the American Medical Association* **275**:1177-1181.

Koo WWR, Succop PA, Bornschein RL (1991). Serum vitamin D metabolites and bone mineralization in young children with chronic low to moderate lead exposure. *Pediatrics* **87**:680-687.

Korrick SA, Hunter DJ, Rotnitzky A (1999). Lead and hypertension in a sample of middle-aged women. *American Journal of Public Health* **89(3)**:330-335.

Kosnett MJ (2001). Heavy Metal Intoxication and Chelators. ***In***: Katzung BG (Ed); Basic and Clinical Pharmacology; 8th edition. pp 999-1002.

Landrigan PJ (1994). Lead. ***In***: Rosenstock L, Cullen MR, (Eds); Textbook of Clinical Occupational and Environmental Medicine. Philadelphia: Saunders. pp 745-754.

Lanphear BP, Dietrich KN, Auinger P, Cox C (2000). Cognitive deficits associated with blood lead levels concentrations <10μg/dl in US Children and Adolescents. *Public Health Report* **115**: pp 521-529.

Lansdown R, Yule W, Urbanowicz MA (1986). The relationship between blood-lead concentrations, intelligence, attainment and behavior in a school population: the second London study. *International Archives of Occupation and Environmental Health* **57**: 225-235.

Lerda D (1992). Study of sperm characteristics in persons occupationally exposed to lead. *American Journal of Industrial Medicine* **22**:567-571.

Lin JL, Lin-Tan DT,Hsu KH, Yu CC (2003). Environmental lead exposure and progression of chronic renal disease in patients without diabetes, *New England Journal of Medicine* **348(4)**:277-286.

Luo W, Zhang Y, Li H (2003). Children blood lead levels after the phasing out of leaded gasoline in Shantou, China. *Archives Environmental Health* **58(3)**:184-187.

Mathee A, von Schirnding YER, Levine J, Ismail A, Huntley R, Cantrell A (2002). A survey of blood lead levels amongst young Johannesburg school children, *Environmental Research* **90(3)**:181-184.

McMichael AJ, Vimpani GV, Robertson EF (1986). The Port Pirie cohort study: maternal blood lead and pregnancy outcome. *Journal of Epidemiology, Community Health* **40**:18-25.

Moss ME, Lanphear BP, Auinger P (1999). Association of dental carries and blood lead levels. *Journal of the American Medical Association* **281(24)**:2294-2298.

Mueller PW, Lash LH, Price RG, Stolte H, Gelpi E, Maack T, Berndt W (1997). Urinary biomarkers to detect significant effects of environmental and occupational exposure to nephrotoxins 1. Categories of tests for detecting effects of nephrotoxins. *Renal Failure* **19**:505 - 521.

Mushak P (1992). Defining lead as the premier environmental health issue for children in America. Criteria and their quantitative application. *Environmental Research* **59**: 281-309.

Mutuku J, UNEP, JKUAT (2003). Lead levels in Nairobi, Kenya. www unep.org/pcfv

National Committee for Clinical Laboratory Standards (NCCLS) (1991). Procedures for the Collection of Diagnostic Blood Specimens by Skin puncture 3rd edition. Villanova, PA. NCCLS Document H4-A3.

National Committee for Clinical Laboratory Standards (NCCLS) (1991). Procedures for the Collection of Diagnostic Blood Specimens by Venipuncture 3rd edition. Villanova, PA. NCCLS Document H3-A3.

National Institute for Occupational Safety and Health (NIOSH) (1999). Pocket guide to chemical hazards. Cincinnati (OH): National Institute for Occupational Safety and Health. http://www.cdc.gov/niosh/homepage.html.

National Safety Council (NSC) USA. (2004). Adult lead poisoning. file://c:/windows/Temp/trilomel.htm.

Needleman HL, Rabinowitz M, Leviton A (1984). The relationship between prenatal exposure to lead and congenital anomalies. *Journal of the American Medical Association* **251**:2956-2959.

Needleman HL, Schell A, Bellinger D (1990). The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *New England Journal of Medicine* **322**:83-88.

Nriangu JO, Blankson MI, Ocran K (1996). Childhood poisoning in Africa: a growing public health problem. *Science of the Total Environment* **18(2)**:93-100.

OSHA (Occupational Safety and Health Administration) (1995). Standards. Code of Federal Regulations (CFR). Appendix C: medical surveillance guidelines. 29 CFR 1910.1025. http://www.access.gpo.gov/nara/cfr/cfr-table-search.html.

Payton M, Hu H, Sparrow D (1994). Low-level lead exposure and renal function in the normative aging study. *American Journal of Epidemiology* **140(9)**:821-829.

Plaa GL (2001). Introduction to Toxicology: Occupational and Environmental. ***In***: Katzung BG (Ed). Basic and Clinical Pharmacology; 8th edition. pp 987-997.

Poklis A, Wong SHY, Pesce AJ (1996). Toxicology. ***In***: Kaplan LA, Pesce AJ (Eds); Clinical Chemistry: Theory, Analysis and correlation; 3rd edition. pp 1017-1030.

Pruss-Ustin (2004). Lead exposure. ***In***: Ezzati (Ed); Comparative quantification of health risks, World Health Organization, Geneva.

Pudek MR (1996). Adrenal hormones and hypertension. ***In***: Kaplan LA, Pesce AJ (Eds); Clinical Chemistry: Theory, Analysis and correlation 3rd edition. pp 912-930.

Rosen JF, Chesney RW, Hamstra AJ (1980). Reduction in 1-25 dihydroxyvitamin D in children with increased lead absorption. *New England Journal of Medicine* **302**:1128-1131.

Safi J, El-haji S, Sansour R, Hashish MA, Suleiman H, Safi N, Jaghabir M, Fischbein A, Abu-Hamda A, Berman T, Ritcher ED, Choudhri Y, Witt JK, Reingold S, Alayyan A (2006). Childhood lead exposure in Palestinian Authority, Israel and Jordan: Results from the Middle Eastern Regional Cooperation project 1996-2000. *Environmental Health Perspective*. http://dx.doi.org/

Schteingart DE (1992). Pancreas: Glucose metabolism and diabetes mellitus. ***In***: Price SA, Wilson LM (Eds); Pathophysiology: Clinical concepts of disease processes; 4th edition. pp 863-889.

Schwartz J (1995). Lead, blood pressure, and cardiovascular disease in men. *Archives of Environmental Health* **50**:31-37.

Smith MA (1984). Lead in history. ***In***: Lansdown R, Yule W (Eds); The lead debate: The Environmental Toxicology and Child Health. London Croom Helm. pp 7-24.

Staessen JA, Lauwerys RR, Buchet JP (1992). Impairment of renal function with increasing blood lead concentrations in the general population. *New England Journal of Medicine* **327(3)**:151-156.

Staudinger KC, Roth VS (1998). Occupational lead poisoning. *American Family Physician* **57(4)**:377-385.

Telisman S, Cvitkovic P, Jurasovic J (2000). Semen quality and reproductive endocrine function in relation to biomarkers of lead, cadmium, zinc, and copper in men. *Environmental Health Perspective* **108**: 45-53.

Tong S (1998). Lead exposure and cognitive development: persistence and a dynamic pattern. *Journal of Paediatrics and Child Health* **34**:114-118.

Tong S, Mcmichael AJ (1999). The magnitude, persistence and health significance of cognitive effects of environmental lead exposure in childhood. *Journal of Environmental Medicine* **1**:103-110.

Tong S, Yasmin E, von Schirnding YER, Prapamontol T (2000). Environmental lead exposure: A public health problem of global dimensions. *The International Journal of Public Health* **78(9):** 1068 – 1074.

United States Environmental Protection Agency (USEPA) (1986a). Air quality criteria for lead. Research Triangle Park, NC: US Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. Report no. EPA 600/8-83-028F. http://www.epa.gov/

United States Environmental Protection Agency (USEPA) (1986b). Determination of reportable quantities for hazardous substances. Washington: US Environmental Protection Agency. Code of Federal Regulations 40 CFR 117. http://www.epa.gov/

United States Environmental Protection Agency (USEPA) (1989a). Evaluation of the potential carcinogenicity of lead and lead compounds. Office of Health and Environment Assessment. EPA/600/8-89/045A http://www.epa.gov/

United States Environmental Protection Agency (USEPA) (1989b). Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation. Office of Air Quality Planning and Standards, Research Triangle Park, NC.PBBLL89207914/AS. http://www.epa.gov/

United States Environmental Protection Agency (USEPA) (1990). Air Quality Control for lead: Supplement to the 1986 Addendum. Environmental Criteria and Assessment Office, Research Triangle Park.NC.EPA-600/8-89/049F. http://www.epa.gov/

Victery W, Throler HA, Volpe R (1988). Summary of discussion sessions: symposium on lead blood pressure relationships. *Environmental Health Perspective* **78**:139-155.

von Schirnding YER (1999). The impact of lead poisoning on the workforce and society. In: Proceedings of the international conference on lead poisoning. Bangalore. The George Foundation. pp 41 -47.

von Schirnding YER, Mathee A, Robertson P, Strauss N, Kibel M (2001). Distribution of blood lead levels in school children in selected Cape Peninsula suburbs subsequent to reductions in petrol lead. *South African Medical Journal* **91(10)**:870-872.

Weaver VM, Lee BK, Ahmed KD, Lee GS, Toidd AC, Stewart WF, Wen I, Simon DJ, Parsons PJ, Schwartz BS (2003). Association of lead biomarkers with renal function in Korean lead workers. *Occupational and Environmental Medicine* **60**:551-562.

WHO (1991). Principles and methods for assessment of nephrotoxicity associated with exposure to chemicals. Environmental Health Criteria No. 119.

WHO (1995). Inorganic lead. Environmental Health Criteria No. 165.

WHO (2005). Lead. ***In***: Children's Health and the Environment. WHO training package for the health sector. www.who.int/ceh

Wilson LM (1992). Chronic renal failure. ***In***: Price SA, Wilson LM (Eds); Pathophysiology: Clinical concepts of disease processes 4th edition. pp 647-665.

Winneke G, Brockhaus A, Ewers U (1990). Results from the European multicenter study on lead neurotoxicity in children: implications for risk assessment. *Neurotoxicological Teratology* 12:553-559.

Zheng W (1999). Blood lead screening in china: Organisation, quality assurance and results. ***In***: proceedings of the international conference on lead poisoning, Bangalore, India, 8-10 February. The George Foundation, pp119-122.

EGERTON UNIVERSITY LIBRARY