DETERMINATION OF BLOOD LEAD LEVELS AND CHARACTERIZATION OF POTENTIAL ENVIRONMENTAL EXPOSURES AMONG CHILDREN IN KIBERA, NAIROBI

THESIS SUBMITTED IN PARTIAL FULFILMENT FOR THE AWARD OF THE DEGREE OF MASTERS OF PUBLIC HEALTH OF THE UNIVERSITY OF NAIROBI, KENYA

DECLARATION

I, Tom H.A.M. Olewe, do hereby declare that this research is my original work and has not been presented to any other institution for the purpose of obtaining a degree.

Signed: Date: J ? f / f A X

APPROVAL

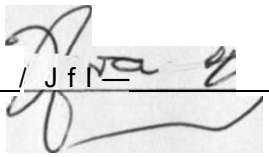
The thesis has been submitted as-part fulfillment for the award of the degree of Masters of Public Health of the University of Nairobi with our approval as supervisors:

Internal Supervisors:

PROF M.A MWANTHI, Ph.D., MSEH, B.Sc.

Signed: J V I I F I V w W A J U ^ r r ^ - r i____ Date:

PROF J.K WANGOMBE, Ph.D. (Health Economics), MA, BA

Signed:  Date

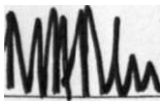
External Supervisor:

PROF J. GRIFFITHS, MD, MPH & TM

Signed: Date: f f J v ^ j 1#08

Approved by Chairman, Department of Community Health, University of Nairobi

PROF M.A MWANTHI, Ph.D., MSEH, B.Sc.

Signed:  ^ Date:

DEDICATION

This work is dedicated to my son, Tom Joseph Olewe who was born on August 10th 2006, about ten (10) hours before my own birthday, for rekindling my passion to promote healthy environments for the children of Kenya.

ACKNOWLEDGEMENTS

This study is a product of awesome support and unequalled supervision. I would like to acknowledge the following individuals and organizations:-

My beautiful wife, Mercy and only begotten son, Jojo for your love, prayers and patience;

My supervisors: Prof. Mwanthi, Prof Wangombe and Dr. Griffiths for their advice, guidance and patience;

Ms. Rose, Mr. Nyabola and entire staff of Department of Community Health for superb coordination of the entire academic program;

Dr. Oduor, Department of Chemistry, University of Nairobi, for partnership in analysis of l e ^ levels in environmental samples;

Anne and Anthony for being invaluable research assistants;

Tufts University, Boston, USA through Dr. Giffiths, for funding the blood lead testing and giving me a profitable experience in the USA;

Yes to Kids Programme of Medical and Sports Evangelism Ministries for permitting the use of their clinic;

Many other individuals who played a part in making this work possible,

Without you all this work would not have been possible.

Thank you, all.

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

AIHA	American Industrial Hygiene Association
AIDS	Acquired Immune Deficiency Syndrome
AMREF	African Medical Research Foundation
ARC	The Arc's Questions and Answers on Lead Poisoning
ASTM	American Society for Testing and Materials
ATPase	Adenosine Triphosphatase
BLL	Blood Lead Levels
CBS	Central Bureau of Statistics
CDC, USA	Centre for Disease Control and Prevention, United States of America
CEHRC	Community Environmental Health Resource Center
CLSI	Clinical and Laboratory Standards Institute
DMOH	District Medical Officer of Health
DPHO	District Public Health Officer
EDTA	Ethylene Diamine Tetra-acetic Acid
EP	Erythrocyte Protoporphyrin
ELPAT	Environmental Lead Proficiency Analytical Testing
ELCI	Environment Liaison Centre International
GFAAS	Graphite Furnace Atomic Absorption Spectrophotometer
GoK	Government of Kenya
HCL	Hydrochloric Acid
HIV	Human Immunodeficiency Virus

$HN0_3$	Nitric Acid
HPBL	Health Protection Branch Laboratories, Bureau of Chemical Safety, Ottawa, Canada
ILO	International Labour Organization
MD	Medical Doctors
MDEQ	Michigan Department of Environmental Quality
NEMA	National Environmental Management Agency
NCBD	Nairobi Central Business District
NYCDHMH	New York City Department of Health and Mental Hygiene
OSHA	Occupational and Safety Health Administration
SoilPB	Soil Lead levels in ug/kg
TB	Mycobacteria Tuberculosis
TCBD	Thika Central Business District
UNICEF	United Nations Children's Fund
UNEP	United Nations Environmental Program
UNDP	United Nations Development Program
VIPS	Vision, Integrity and Passion to Serve, a registered clinic
WHO	World Health Organization
Y2K	Yes to Kids program

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

BACKGROUND: Lead is a heavy metal which is introduced to the environment by human activities. Excessive lead exposure through air, water, soil and food is harmful to the health and intellectual development of millions of children (Markowitz et al, 2000). For instance, lead has ability to cause neurotoxic (nerve poison) effects, particularly in children whose growing bodies are highly susceptible (Markowitz et al, 2000).

There is overwhelming evidence • of higher concentration than allowable environmental lead levels in Nairobi, yet relative dearth of information and action regarding lead poisoning in Kenya. Widespreao and potentially excessive lead exposure has been reported in Nairobi by Mungatana et al. (2004). Lead levels in kales, maize, tap water and soil were 5,053ug/kg, 1,948ug/kg, 5.5ug/l and 44,350ug/kg respectively. These were higher than the acceptable World Health Organization lead levels in kales, maize, tap water and soil, which are 300ug/kg, 200ug/kg, 10ug/l and 100 - 120ug/kg respectively.

OBJECTIVE: A study was carried out in Kibera slums, Nairobi between April and August 2007, with the main objective to determine the blood lead levels among 6 - 59 months old children born in Kibera and their potential environmental exposures and risk factors for elevated blood lead concentrations (BLL >10 Hg/dl). .

METHODS AND MATERIALS: This was a descriptive, cross-sectional study of 387 children, who presented at Yes to Kids (Y2K) program, VIPS Health Services at Woodley, Nairobi between June and August 2007. Upon approval of the study by the Ethic and Research Committee, training of interviewers and Laboratory technicians was held in tandem with pre-testing of the questionnaires. Parental, guardians or care givers gave consent for children's participation in the study. Participating children were carefully screened for inclusion criteria by medical doctors. Trained laboratory technologists at the clinic using the capillary blood collection protocol collected blood for lead analysis using both LeadCare II blood lead analyzer on 387 samples at Y2K program and Graphite furnace, Atomic Absorption Spectrometer on 22 samples, following a standardized analytical methods (Flajnik et al, 1994) at the Massechussetts Public State Laboratory in Boston, USA.

Coded and close ended questionnaires were introduced to parents, guardians or care givers, asking about socio-demographic profiles, and residence characteristics, as well as potential risk factors for lead exposure.

Pooled samples from selected potential sources of exposure (water, soil, kales) were collected from the villages from which the tested children came. Environmental samples were collected following the sampling methodology according to the Community Environmental Health Resource Center (www. Cehrc.org, 2006) and analyzed using flame Atomic Absorption

Spectrophotometry, following a Shimadzu AA6300 standardized analytical method (Shimadzu, 2002).

DATA ANALYSIS: Data management and analysis was done using Statistical Package for Social Sciences (SPSS) software, version 10. The CDC permissible childhood blood lead level of 10pg/dl was used as a cutoff point in the analysis. The socio-demographic characteristics, residence characteristics, and potential risk factors for exposure to lead among children with a blood lead levels > 10 pg/dl were compared to those of children with a blood lead levels <10 pg/dl. Chi-square test for independence, Spearman's correlation, Eta correlation and Analysis of Variance (ANOVA) were used to determine measures of association and statistical significance. Statistical significance was set at p = 0.05.

RESULTS: Three hundred and Eighty Seven (387) children were involved in the study, with 52.8% and 47.2% being boys and girls respectively. The .mean blood lead level (BLL) was 5.997ug/dl (median = 5.400ug/dl, SD =2.42, Range 3.30 - 24.70ug/dl). There were 27 (7%, N = 387) children with BLL > 10 *hg/6*l, which was above the WHO/CDC cut off for lead poisoning. Blood lead level > 10 ug/dl was associated with non-permanent housing (X^2 = 0.0565, df = 1, p = 0.812), playing on potentially lead contaminated grounds (OR = 0.89; 95%CI: 0.25 - 2.34, p = 0.627) and pica behaviour (OR = 0.72; 95%CI: 0.31 - 1.68, p = 0.439). Low risk parental occupation (OR = 14.28; 95% CI: 3.05 - 66.75; p = 0.001) was significantly associated with BLL > 10ug/dl among the children. The

questionnaire was probably not the best surrogate for occupational lead exposure hence those classified as low risk for lead exposure could, infact have been high risk for occupational lead exposure.

Kales sourced from the market/kiosks (OR = 14.24; 95% CI: 3.05 - 66.45; p = 0.001) were significantly associated with BLL £ 10ug/dl yet concentration of lead in analyzed kales were below detectable levels. It is possible that the kales analyzed for lead concentration were from sources different from those ingested by the children in the study.

Soil lead levels (SoilPb) ranged from 3,000 to 90,000ug/kg, which was very high compared to WHO acceptable range of 100 - 200ug/kg. There was weak linear association (r^2 = 0.0160) between SoilPb and mean BLL for a given village. There were no detectable levels of lead in kales and tap water.

CONCLUSIONS: About 7% (N = 387) of the children tested had childhood lead poisoning (BLL > 10ug/dl), which is higher than in economically advantaged countries. Soil was the significant source of exposure to lead (Range in Kibera slums: 3,365 - 89,570 ug/kg; WHO allowable range: 100 - 120ug/kg), among the children in Kibera slums. With such high soil lead levels, the prevalence of childhood lead poisoning in Kibera could be higher than found using convenient, clinic - based sample in this study. Knowledge of the prevalence will determine the choice of lead screening strategy and devices. The knowledge on lead

poisoning (5.4%, N = 387) and potential sources of exposure (3.1%, N = 387) were very low. Intervention strategies at the community will require advocacy and education about childhood lead poisoning. In comparison with other parts of the world, socioeconomic factors seemed to play an important role in childhood lead poisoning. Given the socioeconomic status of most Kenyans, the 7% prevalence in the study and higher figures e.g. 10% in Kariobangi North (UNEP, 2006) have raised a health flag that must be addressed.

CHAPTER 1: INTRODUCTION AND BACKGROUND

1.1. STATEMENT OF THE PROBLEM

Lead poisoning is a serious health hazard with major socio-economic implications. Due to human activities, lead, a heavy metal, pollutes the environment and poisons children. Lead is a potent neurotoxin (nerve poison), particularly in children whose growing bodies are highly susceptible (Markowitz et al, 2000) Exposure to excessive levels of lead in air, water, soil and food is harmful to the health and intellectual development of millions of children (Markowitz et al, 2000)

The public health problem of environmental lead exposure has been widely investigated in developed countries like the United States of America, where actions taken have led to significant reductions in children's blood lead concentrations (Henry, 2003). In contrast, there is a relative dearth of information and action regarding lead poisoning in developing countries, particularly in African countries like Kenya, despite evidence of widespread and excessive childhood lead exposure.

The work of United Nations Environmental Program (UNEP) on the levels of lead in various foodstuffs, water and soil in Nairobi **(Tables 1)** revealed lead levels way above those acceptable by World Health Organization (WHO) (Mungatana, 2004).

Table 1: Lead levels in Selected Environmental samples

Environmental Samples (units of measurement)	WHO allowable Lead levels	Mean Lead Levels in Samples (Nairobi Area)
Kales (ug/Kg)	300	5.053.6
Maize (ug/Kg)	200	1,948.1
Tap Water (ug/l)	10	5.5
Soil (ug/Kg)	100 - 120	44,350

Source: Mungatana, 2004

In spite of these alarming statistics, the extent of lead poisoning among the Nairobi population is not known The extend of childhood lead poisoning is also not known and has not even made the list of national public health priorities in spite of the fact that children are more vulnerable and suffer more serious consequences (Mathee et al, 2004).

Growing evidence suggest that lead in a child's body, even in small amounts, can cause disturbances in early physical and mental growth, and later interferes with intellectual functioning and academic achievements (de Burbure et al, 2006; Papanikolaus et al, 2005). Hence Centre for Disease Control (CDC) in United States of America (USA) has lowered the blood lead concentration to 10jjg/dl, above which public health action should be taken. This, in addition to the alarming statistics on the environmental lead levels, warrants an active approach to understand the magnitude and determinants of lead poisoning among children in Kenya, which is currently lacking.

The aim of the study was to determine the blood lead levels among children aged 6 -59 months, and identify possible risk factors for elevated blood lead levels in children of Kibera, an urban slum population in Kenya exposed to potential sources of lead poisoning. The magnitude of the lead poisoning among children in Kibera was compared with those of children in other parts of the world. The study provided baseline data for evidence-based advocacy and action, as regards lead poisoning and its effects on children of Kenya.

1.2. RATIONALE FOR THE STUDY

1. The study contributed to the understanding of the level and extent of lead pcroning among children in Kibera slums, Kenya.

2. The study provided valuable data that could initiate a policy and enhance evidence-based enforcement of controls on potential sources of lead poisoning as well as for monitoring and evaluation of the impact of unleaded gasoline on blood lead levels among the Kenyan children. With the policy in place, it will be possible to reduce exposure levels.

3. The study provided base-line data essential for future studies and development of intervention strategies.

CHAPTER 2: LITERATURE REVIEW

Lead is classified as a metal. Chemically, its low melting point and ability to form stable compounds has made it useful for hundreds of products. The result is widespread dissemination of lead in the human environment. Clinically, it is purely a toxicant; no organism has an essential function that is lead dependent.

2.1 DEFINITION OF LEAD POISONING

Prior to 1970, blood levels greater than 60 pg/dl defined significant lead poisoning. In 1971 the threshold for blood lead levels was reduced to 40 pg/dl (CDC, 2000). This was subsequently reduced to 30 pg/dl in 1975 and to 25 pg/dl in 1985 (CDC, 2000). In 1991, the Centers for Disease Control and Prevention, USA statement concerning lead poisoning in young children redefined elevated blood lead levels as >10 pg/dl and recommended a new set of guidelines for treatment of blood lead levels >15 pg/dl (CDC, 2002). However, since the early 1990s, accumulating data have provided evidence that toxic effects occur at levels below 10 pg/dl.

2.2 LEAD METABOLISM

Elemental lead and inorganic lead compounds are absorbed through ingestion or inhalation. Organic lead (e.g., tetraethyl lead, the lead additive to gasoline) is absorbed to a significant degree through the skin as well. Pulmonary absorption is efficient, particularly if particle diameters are <1 um e.g. in fumes from burning

lead paint, exhaust from vehicles (Lauralynn et al, 2001). Children absorb up to 50% of the amount of lead ingested, whereas adults absorb only ~10 to 20% (Anderson et al, 1995). Gastrointestinal absorption of lead is enhanced by fasting and by dietary deficiencies in calcium, iron, and zinc; such absorption is minimal, however, for lead in the form of lead sulfide, a common constituent of mining waste. Lead is absorbed into blood plasma, where it equilibrates rapidly with extracellular fluid, crosses membranes (such as the blood-brain barrier and the placenta), and accumulates in soft and hard tissues (LaDou et al, 1990). In the blood, ~95 to 99% of lead is sequestered in red cells, where it is bound to hemoglobin and other components. As a consequence, lead is usually measured in whole blood rather than in serum. The largest proportion of absorbed lead is incorporated into the skeleton, which contains >90% of the body's total lead burden. Lead is excreted mainly in the urine and in the feces. Lead also appears in hair, nails, sweat, saliva, and breast milk. (Markowitz et al, 2000). The half-life of lead in blood is ~25 days; in soft tissue, ~40 days; and in the nonlabile portion of bone >25 years (Richard et al, 2003). Thus, blood lead levels may decline significantly while the body's total burden of lead remains heavy.

The toxicity of lead is probably related to its affinity for cell membranes and mitochondria, as a result of which it interferes with mitochondrial oxidative phosphorylation and sodium, potassium, and calcium ATPases. Lead impairs the activity of calcium-dependent intracellular messengers and of brain protein kinase C. In addition, lead stimulates the formation of inclusion bodies that may

translocate the metal into cell nuclei and alter gene expression (Markowitz et al, 2000).

2.3 LEAD PATHOPHYSIOLOGY

The non-nutritive hand-to-mouth activity of young children provides the pathway for lead to enter the body in the majority of children (Markowitz et al, 2000). In nearly all cases lead is ingested either as a component of dust licked off surfaces or in swallowed paint chips. Less commonly, water contaminated by its flow through lead pipes or brass fixtures is drunk or food is contaminated by contact with lead-glazed ceramic ware (CDC, 1991). Cutaneous contamination with inorganic lead compounds, as are found in pigments, roes not result in a substantial amount of absorption. In contrast, organic lead compounds such as tetraethyl lead may penetrate through skin (ELCI, 2003).

Once lead is in the intestine, the percentage absorbed depends on several factors such as particle size, pH, other material in the intestine, and nutritional status of essential elements. Large paint chips are difficult to digest and mainly are excreted. Fine dust can be dissolved more readily, especially in an acid medium. Lead eaten on an empty stomach is better absorbed than if taken with a meal (www.emedicine.com/EMERG/topic293.htm, 2007). The presence of calcium and iron'may decrease lead absorption by direct competition for binding sites. On the other hand, iron, and probably calcium, deficiency results in enhanced lead absorption, retention, and toxicity (Markowitz et al, 2000).

After absorption, lead is disseminated throughout the body by way of the blood. It circulates bound to erythrocytes; about 97% is bound on or in the red blood cell. The plasma fraction is too small to be measured by conventional techniques such as atomic absorption spectroscopy or anodic stripping voltammetry; it is, however, presumably the plasma portion that may enter cells and induce toxicity (Fernandos et al, 2005). In cells, lead has multiple effects. It binds to enzymes, particularly those with available sulfhydryl groups, changing the contour and diminishing function. The heme pathway, present in all cells, has three enzymes susceptible to lead inhibitory effects. The last enzyme in this pathway, ferrochelatase, enables protoporphyrin to chelate iron, thus forming heme. Protoporphyrin is readily measurable in red blood cells. Levels greater than 35pg/dl are abnormal and are consistent with lead poisoning, iron deficiency, or recent inflammatory disease. Lack of heme affects multiple metabolic pathways. The accumulation of excess amounts of protoporphyrin and other heme precursors is toxic as well, independently of lead. Measurement of the erythrocyte protoporphyrin (EP) level is, therefore, a useful tool for monitoring biochemical lead toxicity (Markowitz et al, 2000).

A second mechanism of lead toxicity is by way of its competition with calcium. Many calcium-binding proteins have a higher affinity for lead. Lead bound to these proteins may alter function, resulting in intracellular and intercellular communications breakdown. For example, neurotransmitter release is in part a

calcium-dependent process that is adversely affected by lead (Markowitz et al, 2000).

Although these two mechanisms of toxicity may be reversible, a third way that lead may cause harm is by preventing the development of the normal tertiary structure in the brain (Canfield et al, 2003). In immature mammals, the normal pruning process that results in elimination of multiple intercellular brain connections is inhibited by lead. This process is part of the timed developmental sequence that humans undergo as well. Failure to construct the appropriate tertiary brain structure during the first few years of life may, therefore, result in a permanent abnormality (Markowitz et al, 2000).

2.4 BIOMARKERS FOR MONITORING HUMAN EXPOSURE TO LEAD

Biomonitoring for human exposure to lead reflects an individual's current body burden, which is a function of recent and/or past exposure. Thus, the appropriate selection and measurement of biomarkers of lead exposure is of critical importance for health care management purposes, public health decision making, and primary prevention activities.

Lead concentration in whole blood (BLL) is the primary biomarker used to monitor exposure to this metallic element with blood lead levels of 10 ng/dl considered the threshold of concern in young children (CDC, 2002). However,

recent studies have reported the possibility of adverse health effects, including intellectual impairment in young children, at blood lead levels < 10 jag/dl, suggesting that there is no safe level of exposure (CDC, 1997).

It appears impossible to differentiate between low-level chronic lead exposure and a high-level short lead exposure based on a single blood lead levels measurement; therefore, serial blood lead levels measurements offer a better estimation of possible health outcomes (Fernando et al. 2005). The difficulty in assessing the exact nature of lead exposure is dependent on the complex toxicokinetics of lead within various body compartments including cycling of lead between bone, blood, and soft tissues. To differentiate more effectively between lead stored in the body for years and lead from recent exposure, information on other biomarkers of exposure may be needed.

Many studies have reported statistically significant associations between blood lead levels and various health effect outcomes. Some, however, have been statistically weak, with the magnitude of the effect relatively small. According to Haward et al. (1998), such weaknesses of association may occur because blood lead levels is not a sufficiently sensitive biomarker of exposure or dose at the target organ(s) or because the relationships involved are biologically irrelevant and are only found because of an uncontrolled confounding factor. Furthermore, in view of the kinetics of lead distribution within the body (cycling among blood, bone, and soft tissues), differentiation of low-level chronic exposure from a short

high-level exposure is not possible on the basis of a single blood lead levels measurement (Haward et a!., 1998). Consequently; there is renewed interest in alternative biomarkers that may aid diagnosis of the extent of lead exposure. Such alternatives include lead determinations in plasma/serum, saliva, bone, teeth, feces, and urine. However, none of these matrices has gained convincing acceptance"as an alternative to blood lead levels.

2.5 MAGNITUDE OF LEAD POISONING IN KENYA

Lead is an environmental and public health hazard of global proportions. Yet, the magnitude of lead poisoning in Kenya remains poorly understood, due to the persisting lack of information and policy regarding this subject matter. Some studies have been carried out on environmental lead levels in Kenya (Mungatana, 2004), but very little on the blood lead levels, especially among the children. Previous studies have established that 5.8%, 10% and 15.2% of children drawn from Waithaka, Kariobangi North and Babadogo, Nairobi respectively have BLL > 10ug/dl (UNEP, 2006). There was however no data on the blood lead levels of children drawn from Kibera slums of Nairobi.

It is known that causes of lead poisoning are local, varying from community to community and urban to rural areas. Urban children in developing countries are most at risk. In the developing countries, over 80% of children between three and five years of age and 100% under two years have average blood lead levels exceeding the threshold of 10 ug/dl (CDC, 1994).

Lead poisoning is the most common, chemical related, environmental health problem ' in children. This is especially pronounced in economically disadvantaged subgroups of the population. Poverty can cause malnourishment or physical stress, which intensifies disabilities caused by lead absorption (Harvard, 1996).

In developed countries where most gasoline sold is still unleaded, typical annual average concentrations of lead in the air are between 0.1 and 0.2 j*xg / m³* in urban areas and between 0.01 and 0.05 l*ig / m³* in rural parts. Concentrations in excess of 1 l*ig I rrP* are recorded in developing country cities that still heavily rely on leaded gasoline (UNDP/World Bank, 2003). The World Health Organization (WHO) standard for a maximum lead concentration in air is 0.5 l\l*g I m³.* However, due to the significant negative impacts of lead on human health, in 1995, the WHO recommended that the use of lead additives in motor fuels be phased out and that exposure to other sources of lead should be reduced (UNDP/World BanK, 2003). Evidence suggests that population living in urban areas are exposed to inordinately high concentrations of lead. For example, a study conducted by UNEP in Nairobi shows that the average lead levels of soils in the Central Business District are higher than those of a nearby rural area by a multiple of 95 **(Tables 2,3,4,5,6).** The results for lead concentrations in water and other consumables (vegetables and maize) show a similar pattern.

Table 2: Lead levels in Kale [Mg/kg]

	Range	Mean
WHO standard		300.00
.Nairobi Central Business DistrictJNCBD)	4,114-6,092	5,053.6
Nairobi residential	783-.1,601	1,408.2
[Juja	2,603-3,200	2,840.5
Thika business district (TCBD)	846 - 3,302	2,243.0
Ithanga	245 - 680	448.9

Source: ***Mungatana, 2004***

Table 3: Lead levels in Maize [pg/kg]

	Range	Mean
WHO standard		200.00
NCBD	1,610-2,200	1,948.1
Nairobi residential	671 - 1,651	1,000.6
Juja	1,569-3,125	2,454.4
TCBD	622- 1989	1,352.0
Ithanga	167-767	548.8

Source: ***Mungatana, 2004***

Table 4: Lead levels in Milk [pg/1^{4}]

i	Range	Mean
[WHO standard		20.00
Nairobi Raw	30.0-80.0	46.0
Juja Raw	10.0-70.0	34.0
Thika Raw	24.7-83.3	44.4
Brand 1	10.0-50.0	30.0
Brand 2	10.0-30.0	18.0

Source: ***Mungatana, 2004***

Table 5: Lead levels in Water [jig/1]

	Range	Mean
[WHO Standard		10.00
[River	3.2 - 35.0	19.1
1 Bore Hole	3.1-4.0	13.4
[Tap	2.0-8.0	5.5
[Rain	1.0-10.0	5.8

Source: ***Mungatana, 2004***

Table 6: Lead levels in Soils [pg/kg]

¡	Range	Mean
I WHO standard	100 - 120	
[NCBD	96,160-663,470	265,918
Nairobi Residential	36,760 - 63,940	44,350
Nairobi - Thika Road	10,380-60,540	26,732
TCBD	47,060 - 374,420	133,790
Thika Residential	10.670-90,840	39,966
Ithanga	470 - 5,730	2,784

Source: ***Mungatana, 2004***

Kibera slums is situated in the peri-urban Nairobi, so it's reasonable to expect its residents to be exposed to environmental lead levels similar to those of Nairobi residential in the UNEP study **(Tables 2,3,4,5,6).**

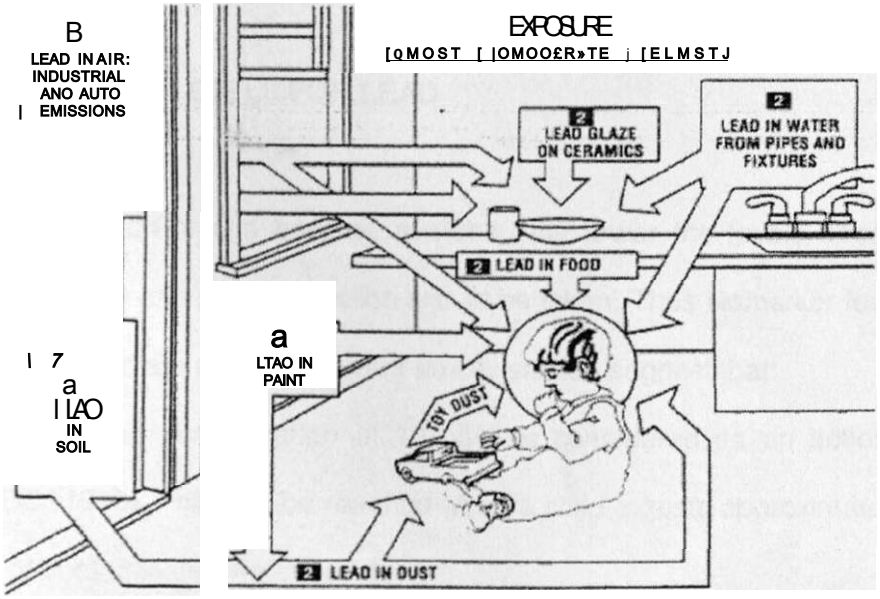
2.6 SOURCES OF LEAD EXPOSURES FOR CHILDREN

Lead in air occurs primarily due to gasoline additives (Bangkok Post, 1996). The combustion of leaded gasoline causes fallout of lead oxides in dust, which can be inhaled. It can also contaminate soil used to grow crops and feed livestock as well as soil found in playgrounds and gardens (Momeshora et al, 1981). Cars that rely on leaded gasoline account for up to 95% of airborne lead pollution in developing countries (NYDHMH, 2004).

Lead in dust derives from soil and airborne pollutants or from parents who bring it into the home after having been exposed at the workplace. Dust settles on 'friction' surfaces like doors or windows, which are, used daily. In addition to inhaled dust, children ingest it through regular hand-to-mouth behavior (NYDHMH, 2004). Lead in water occurs through contamination at various points in the drinking water delivery system, e.g. lead pipes and solder, water faucets (CDC, 1991). Lead can also leach into groundwater from soil pollu- - with fallout from leaded gasoline, crop irrigation and food processing in factories and homes (ARC, 1996). About 15 mg/l of lead in drinking water is currently considered as an acceptable level (Chivian et al, 1994). Lead in food results from food and drink cans, manufactured in many countries, which continue to contain lead solder that can leach into the food and drink. Concentrates of lead can be found in vegetables, particularly green leafy ones. Lead in paints is the major source of lead in homes. Children are at risk from peeling paint chips, which they tend to, take in their mouths (Markowitz et al, 2000). Home gardens can be contaminated

by paint peels or rain-washed run-offs from lead-based paints. Lead in ceramic glazes can leach out of ceramic ware, china and crystal, especially if the food is acidic. Lead in cosmetics and folk remedies is found in substantial quantities, especially in the developing world (Chivian et al, 1994). Sources of childhood exposures within the household are summarized in **Figure 1.**

Other sources of lead include: lead smelters, incinerators, battery recycling plants and disposal of products containing lead (e.g. pencils). Some activities such as battery recycling, can take the form of heavily polluting 'home backyard' industries that are difficult to identify (Matte et al, 1998). In a study done in Oporto, Portugal, important sources of childhood lead poisoning were father's occupation, mother's smoking habits, and poor hygiene and pica associated with contaminated soils and lead paint (Mayan et al, 2001).



Source: *USA Consumers reports, 1993; Exposure levels 1 = Most;2 = Moderate; 3 = Least*

Figure 1: Various sources of Lead

2.7 CHILDREN VULNERABILITY TO LEAD

Children's nervous and digestive systems are developing. They are thus more susceptible to lead uptake. Children absorb up to 50% of lead taken into their bodies, compared to 10-15% in adults. (Haward et al, 2001). Children may receive 3 times the dose of adults, because they have a larger surface to volume ratio (CDC, 1991). Children have a propensity to explore the world through their mouths (Markowitz et al, 2000). Lead in the air attaches to dust, which is widespread in household environments where young children are crawling, playing and touching. Lead in dust and dirt can be ingested via children's hands and toys, for example by thumb sucking or by putting objects in their mouths. Young children might ingest as much as 200 mg per day, particularly if they live in cities or near busy streets (UNEP/UNICEF, 1997).

2.8 TOLERANCE LEVELS FOR LEAD

As more research results become available, the lower the levels become at which preventive and remedial action should be taken. Thus biomarker levels for tolerance for lead are unclear. Current toxicity studies suggest that:

I. A blood-lead concentration of 10 ug/dl is considered as an action level (CDC, 1996). This can be reached when a child ingests approximately 225 ml of contaminated water per day.

II. An amount of 45 ug/dl demands that treatment begin within 48 hours (CDC, 1997).

III. More than 70 ug/dl in blood presents a medical emergency (CDC, 1997).

IV. Over 120 ug/dl in blood is highly toxic and potentially lethal (CDC, 1997).

Studies have shown that even at 10ug/dl blood-lead levels, detrimental effects on child development and behavior can-be observed. Further studies suggest that at any level the detrimental effects of lead on child health can be detected **(Figure** 7).

Table 7: Effects of lead poisoning on children

Health effects among children with BLL < 5pg/dl	**Health effects among children with BLL 55-70ug/dl**	**Health effects among children with BLL > 70ug/dl**
• Reduction in intelligence quotient (IQ) and attention span • Reading and learning disables • Hyperactivity and behavioral problems • Impaired growth • Impaired visual and motor functioning • Hearing loss	• Anemia • Brain, liver/kidney, nerve damage • Coma • Convulsions • Death	• Long-term and potentially irreversible • Intensified with repeated exposure and accumulation of lead in body

Source: ***UNEP/UNICEF, 1997***

CHAPTER 3: OBJECTIVES AND HYPOTHESES OF THE STUDY

3.1. GENERAL AND SPECIFIC OBJECTIVES

3.1.1 GENERAL OBJECTIVE

The aim of the study was to determine the blood lead levels among children aged 6 - 5 9 months living in Kibera slums, Nairobi and characterizing the potential sources of exposures.

3.1.2 SPECIFIC OBJECTIVES

1. To determine the study subjects demographic profiles;
2. To determine the blood lead levels (BLL) of children aged 6 - 5 9 months, residing in Kibera slums;
3. To determine the lead levels in various types of exposure, specifically foodstuffs, drinking water and soil sampled from Kibera slums;
4. To determine the level awareness of environmental sources of childhood lead poisoning among the parents, guardians or care givers of the children living in Kibera slums;
5. To compare the blood lead levels of Kibera children with data and guidelines from WHO/CDC and other countries.

3.2. RESEARCH HYPOTHESES

The following hypotheses were tested for the study;

1) The BLL of the children in Kibera were higher than the CDC/WHO allowable levels of urban resident population
2) Lead in water, soil and vegetables (Kale) in Kibera slums were higher than CDC/WHO allowable levels hence potential sources of childhood exposures
3) Parents, guardians or care givers of children living in Kibera were neither aware of childhood lead poisoning nor the potential sources of environmental exposures.

To facilitate the testing of these hypotheses, data were generated as follows;

1. Blood was collected from children who met the inclusion criteria and analysed for lead levels, as a biomarker for childhood lead exposure.
2. Questionnaires were administered to collect data on sociodemographic profiles of these children and their potential sources of exposure to lead.
3. Drinking water and kales from sources identified during the interviews, as well as peri-residential soil were collected and lead levels measured as surrogates for environmental lead levels.

Data from questionnaires generated independent variables used to explain the dependent variables, that is, lead levels in blood and environmental samples.

CHAPTER 4: METHODS AND MATERIALS

t

4.1. STUDY DESIGN

This was a descriptive, cross-sectional study among the Kibera children aged 6 - 59 months. To determine the blood lead levels, blood samples were drawn from children who met the inclusion criteria for the study. Structured, coded questionnaires were administered to the parents, guardians or care-givers accompanying the children. Information generated from the interviews guided the collection of environmental samples of soil, kales and water; from the children's residential areas within Kibera slums.

4.2. DESCRIPTION OF THE STUDY AREA

The study was carried out in Kibera slums of the city of Nairobi, where majority of the children visiting Yes to Kids (Y2K) program, at VIPS clinic, lived.

The name "Kibera" is derivtu from *kibra,* a Nubian word for "forest" or "jungle". The slum originated in 1920 as a Nubian soldiers' settlement. The Nubians were stable residence of Kibera, because in addition to residing there, they were also landowners. Kibera is situated in Nairobi's South-western, peri-urban zone approximately 7km from the Nairobi City Centre. Kibera division borders Dagoreti, Makadara, Pumwani, Central, Westlands and Kasarani to the North and Embakasi to the Northeast **(Appendix I).** It borders Machakos District to the East and Kajiado District to the West and South (GoK / UNHABITAT, 2004).

Administratively, the Kibera division comprised of 7 locations and 16 sub-locations. The l'ocations include Kibera, Lang'ata, Karen, Mugumoini, Nairobi West, Laini-Saba and Sera Ngo'mbe **(Appendix II).** There were a number of villages, including Kianda, Soweto, Gatwekera, Kisumu Ndogo, Lindi, Laini Saba, Siranga/Undugu, Makina and Mashimoni (Central Bureau of Statistics, 1999), as shown on the appended map of the villages in Kibera **(Appendix III).**

It was the largest and most populous of the eight divisions comprising Nairobi Province. Kibera covered an area of 223.4 square kilometres and had a total population of 286,739 people distributed among 89,086 household units (Central Bureau of statistics, 1999). More than half (60%) were Protestants from different denominations. The Catholics constituted about a third of the population. The recent emergence of many evangelical churches (constituting various denominations of the Protestant Church) targeting the poor in Kenya probably explained why there were more Protestants than Catholics in Kibera. The Moslems only constituted 6% and these were likely to be Nubians wno were predominantly Moslems.

Overcrowding and congestion was widespread and experienced at room occupancy level with an average overcrowding index of 5.0. This meant that a single room in Kibera of average size 9.4 square metres was occupied by an average of 5 persons. The mean number of years lived in a village ranged from 8.5 years in Mashimoni to 12.8 years in Makina (GoK / UNHABITAT, 2004).

Internal overcrowding compromised the health of the occupants, especially through susceptibility to contracting infectious such as tuberculosis, skin infections and other diseases. Similarly food contamination, domestic accidents, and psychological stress due to the limited space per person were common. The indoor environment was worsened by use of crude energy (wood, charcoal, kerosene, waste material) for cooking-and lighting, which produced smoke and other hazardous gases. Internal overcrowding also resulted in rapid wear and tear of the structures and denied privacy and family life to the occupants (Syagga et al, 2001).

Most of the residents worked as semi-skilled or unskilled labourers, often on casual basis in the city's industrial area. A few of the residents, particularly women, engaged in small-scale enterprises such as selling of vegetables, food and water (African Population and Health Research Centre, 2002).

Data on nutritic ?l status measured by sufficiency of food supplies showed that quite a large percentage (41%) of the population did not access adequate (quantity and quality) food on a daily basis. As a coping mechanism, many of these households (47%) skipped meal. This phenomenon was explained by the limited income potential (GoK / UNHABITAT, 2004).

Health-related data from Kibera showed that malaria was the leading health problem (cited by 69%) followed by HIV/AIDS (cited by 31%). Poor sanitation in Kibera offered conducive breeding environment for the mosquitoes and rodents.

The other common diseases included: typhoid, TB, respiratory track infection and diarrhoea. The frequency of illness in Kibera appeared to vary with age and gender. Children below 12 years fell sick more frequently than adults (GoK / UNHABITAT, 2004).

Quality of shelter in Kibera was poor. Indeed, most houses were built of rudimentary materials such as mud, timber, polythene paper, corrugated iron sheets among other poor quality building materials. Fifty Seven percent (57%) had cemented floors; the remaining 43% were made of natural earth. Nearly all units (88%) were built of mud; a small proportion of 5% were built of corrugated iron sheet and another 4%, timber. Most of the houses, as a result of being built of very low quality materials, had poor internal conditions and were permeated by elements of weather (rain, wind, cold, etc). Leaking roofs, poor insulation and ventilation, pollution by noise and dust, and lack of privacy were all problems resulting from these poor shelter conditions. Physical infrastructures were nonexistent or minimal at Kibera. For example, roads and pathways were made of natural earth and vehicles could not access most households (GoK / UNHABITAT, 2004).

Even though slums in general were characterized by lack of social amenities, the Kenyan Government and other development agencies had made appreciable efforts to provide slum communities with basic facilities. For example, African Medical Research Foundation (AMREF) had provided financial support to improve the situation of toilets in Kibera. There were many other development

agencies in Kibera attempting to provide more social amenities like schools, health clinics and training institutions. Surprisingly, even though there was a long list of different amenities available in Kibera, virtually all were inadequate. The local authority did not provide water as it considered those settlements illegal and therefore providing water would be misconstrued to be legitimizing them. Therefore, various other sources of water were available to the residents irrespective of the quality, namely private (3%), piped (19%), communal piped or wells or tank (1%), vendors (19%), river and dam (19%), and others (40%) (GoK/ UNHABITAT, 2004).

In Kibera, pit latrines were the main mode of excreta disposal. However, they were inadequate and often overflowing. In this settlement, up to 75 people shared one pit latrine. The location of most latrines did not conform to public health requirements. The mode of excreta disposal were private pit latrine (35%), communal pit latrine (49%), pay-for-use latrine (11%), flying toilets (4%) and open spaces (2%)(GoK / UNHABITAT, 2004). Paying latrines were well maintained and residents are charged a nominal fee for use and maintenance. Flying toilets involved defecation in plastic bags which were then thrown promiscuously, thus posing serious health hazards.

Electricity was scantily provided owing to the poor housing structures. Most of the electrical fittings were located within the government administrative units and social institutions as schools and health care facilities.

Due to the fact that most of the roads and pathways were not paved, they generated dust during the dry spells. Some open and school playgrounds, which are were paved or grass covered, also generated dust especially because of heavy usage.

Smoke emanated from outdoor burning waste and indoor use of crude fuels such as wood, charcoal and kerosene. Atmospheric gaseous pollution is linked to respiratory diseases and poor general health, especially among children. Exposure to dust may precipitate skin irritation, enhances eye infection especially among children and increase particulate matter in the atmosphere.

At the fringes of the Kibera slums, ran Motoine - Ngong River. It was heavily polluted by silt, solid wastes, overflowing pit latrines, waste waters and all forms of dumping including medical wastes from upstream and the surrounding areas. Nairobi dam was also heavily clogged by pollution to a point where the colour of water had turned sewer- like and smells foul, rendering the waters unpotable. Unfortunately, some residents of Kibera and downstream still used the heavily polluted water source to meet their domestic and animal water needs.

4.3. REASONS FOR SELECTION OF THE STUDY AREA

1. Kibera is in Nairobi, a heavily motorized urban city. The environmental lead levels in Nairobi were alarmingly high (Mungatana et al, 2004). The children of Kibera were thought to be predisposed to lead poisoning.

2. Documented blood lead levels of some children living is settings similar to Kibera slums e.g. 10% in Kariobangi North, Nairobi (UNEP, 2006), were above WHO/CDC cut off for lead poisoning (BLL > 10 ug/dl).
3. Economic activities within Kibera slums, that could be potential sources of environmental lead, were carried out, in or close to homes.
4. Farming activities, a source vegetables, were carried out near or irrigated with, the heavily pollute waters from Motoine - Ngong river.
5. Kibera children were economically disadvantaged thus likely to suffer malnutrition which is a known risk factor for lead body burden.
6. The collection of blood sample required pollution free environment. The choice for children most likely to visit the clinic and hence access the laboratory, was aimed at reducing the chances of extraneous contamination in the course of sample collection
7. Kibera slums was a catchment of the Y2K program of VIPS clinic, an established community clinic

4.4. TARGET POPULATION

Universal screening is recommended for children aged less than 6 years except in communities where the prevalence of elevated BLLs is known to be very low and therefore is not a practical or cost-beneficial investment of limited resources (CDC, 1991). The BLL of Kibera children were not known. This coupled with the fact that younger children face greater harm from elevated blood lead concentrations (Haward et al, 2001) led to the selection of children aged 6 to 59

months who had lived in the Kibera area since birth as the study population. Respondents to the structured questionnaires were parents, guardians or care givers of the sampled children and gave informed consent to participate in the study.

4.5. STUDY POPULATION

The study population consisted of people who had information about a child or children. Information on exposure to lead in the study area was attributed to an individual's position within the family unit or within the community. Participants in the study therefore included parents and family care givers/guardians as those with information from the family unit. Health care workers, social workers, local leaders and staff from institutions dealing with the welfare of children, water and environmental issues in the community and wider Nairobi area were respondents from the community point of view.

Parents

Parents who brought children to the Y2K program were interviewed to determine the eligibility of their children in the study, according to the inclusion criteria. Parents who consented to participate in the study and whose children qualified were guided through the pre-coded, structured and closed ended questionnaire and the children's blood sample collected. Where parents were not alive or too sick to communicate, the family care giver or guardian were interviewed.

Family care givers or guardians

These consisted of extended kinship to the children including elderly siblings, uncles and aunts. Social workers were also included.

Community key informants

Key informants provided information concerning knowledge on lead poisoning, morbidity trends and how the community solved its health problems. The key informants included health care workers, local leaders, social workers, staff of government and non-governmental institutions dealing with environment and health e.g. District Medical Officer of Health (DMOH), District Public Health Officer (DPHO), Selected Medical Doctors (MD) running clinics, National Environmental Management Agency (NEMA) staff.

4.6. SAMPLE SIZE DETERMINATION

To determine how many children should be observed to obtain a reasonable picture of the prevalence of BLL > 10pg/dl in the descriptive study, the sample size formula according to Dobson (Dobson, 1984) was used.

$$n = \frac{Z^2 p(1 - p)}{d^*}$$

Where:

n = Sample size

p = 0.5 (assumed that prevalence of children with BLL > 10ng/dl. It was estimated in 1994 that over 80% of those between three and five years of age and 100% under two had average blood lead levels exceeding the threshold of 10 ug/d£ set by the US Centers for Disease Control and Prevention).

z = 1.96 (reliability coefficient corresponding to 5% significance level),

d = 0.05 (degree of precision).

Using the formula given above, the sample size (n) was calculated as below,

$n = (1.96)^2 0.5(1 - 0.50)/ (0.05)^2 = 384.16$

387 children were observed to assure that the 95% confidence interval for the estimated proportion was within ± 5% of the true prevalence.

4.7. SAMPLING AND TESTING TECHNIQUES

a) Sampling of Children

Convenient, non-probability sampling was done on eligible 6-59 month old children presenting for routine check-up and treatment at the Yes to Kids (Y2K) program, VIPS clinic at Woodley Estate, Nairobi situated less than 500m from Kibera slums. The ratio of male to female among children aged between 0 - 5 9 months old of approximately 1:1 in Kibera, was used given that the population of male and female in Kibera was 159,083 and 127,656 (CBS/GoK, 1999) respectively, according to census 1999.

During the study, children presenting at the cllinc were registered by the records clerk at the reception area. Those presenting with non-emergency cases according to the medical doctors, regular check -up or vaccination were targeted for the study Two medical officers provided the names of children eligible for the study. Trained interviewers then approached parents, guardians or care givers in the waiting area and informed them about the study. Those consenting signed an informed consent form to allow participation of their children in the study. Trained interviewers then used a pre-tested questionnaire to collect information on demographic and socioeconomic characteristics, household and child behavior in the context of potential lead exposures. All interviewed parents, guardians or care givers were given coded cards to reduce the chances of re-interviews as well as for ease of follow up later in the study and thereafter. No information was

collected from non-participating parents, guardians or care givers or their children.

b) Sampling and determination of Blood Lead levels

The LeadCare II blood lead analyzer developed by ESA Biosciences Incorporated, USA with partial funding from CDC was used for BLL determination. The principal investigator and the laboratory technologists were trained in its use.

Principles of LeadCare II Blood Lead Analyzer Model 70 - 6529

According to the instructions manual (2004 - 06), LeadCare II used the principle of anodic stripping voltammetry (ASV), an electrolytic method in which a mercury electrode held at a negative potential to reduce metal ions in solution formed an amalgam with the electrode. The solution was stirred to carry as much of the analyte metals to the electrode as possible for concentration into the amalgam. After reducing and accumulating the analyte for some period of time, the potential on the electrode was increased to reoxidize the analyte and generate a current signal. LeadCare system relied on a unique sensor that contained a small amout of gold particles in an inert matrix, to detect lead in whole blood. Samples of whole blood were mixed with treatment reagent containing 250ug of a diluted hydrochloric acid solution in water (0.1mol/l). This caused lysis of red

blood cells, which carry most of the lead and making lead available for detection. When a test was run, the analyzer applied a potential that caused lead to collect on the LeadCare II sensor. After three minutes the analyzer measured the amount of lead collected on the sensor and displayed the result in ug/dl.

Blood Collection

Trained laboratory technologists wearing protective gear, followed the capillary blood collection protocol (Schonfeld et al, 1995). The children's hands were thoroughly washed with soap and water, and air dried. The ball or pad of the finger to be punctured was cleaned with the alcohol swab. The fingertip was then dried using the sterile gauze or cotton ball. The grasped finger was quickly punctured with a sterile lancet in a position slightly lateral to the center of the fingertip.

Pre-EDTA treated capillary tube provided with LeadCare II analyzer held almost horizontally with the green band on top was filled to the black line with 50ul of whole blood. Excess blood was removed from the outside of the tube with clean wipe. Capillary tubes were inspected for proper filling, ensuring there were no gaps, bubbles or excess blood on the outside of the capillary. Properly filled capillary tubes were then placed in uncapped tube containing treatment reagent labeled with client unique code. The entire volume of the blood in the capillary tube was dispensed into the bottom of the tube with aid of a plunger inserted into

the top of the capillary tube. The cap of the tube was replaced and the latter inverted 8 to 10 times to mix the sample completely, until the mixture turned brown. The mixture was left at room temperature and tested within 48 hours.

Blood Lead Analysis

LeadCare II analyzer was powered and calibrated using the calibration button provided for the lot of test kits being used. Calibration was considered successful when the message "CALIBRATION SUCCESSFUL" flashed on the screen for two seconds, then "PREPARE SAMPLE" appeared. Each time new lots of test kits were started or when the analyzer displayed a recalibration message, calibration was done. A sensor was then inserted and one drop of controls added to the sensor at the designated spot, when prompted to do so by the message on the screen were tested. Caution was taken to ensure that the sensor lot number matched the lot number on the screen of the analyzer. After three minutes, the analyzer beeped and displayed tne results in ug/dl. The results found during the study were within the acceptable range for level one (6.5 ± 3.0 ug/dl) and two (26.5 ± 4.0 ug/dl) controls respectively. The subjects blood were then tested using same procedure. Results below 3.3 ug/dl were displayed as "LOW" and those above 65ug/dl as "HIGH". The results were recorded on the LeadCare II worksheet provided. Used sensors, lancet and other consumables were discarded in appropriate biohazard containers and later incinerated.

Validation of LeadCare II Analyzer as Blood Lead Screening Device

To validate the BLL results obtained using LeadCare II, 22 randomly selected blood samples were shipped to Massachessetts Childhood Lead Screening Laboratory, USA. Universal precautions for collection and handling of blood lead samples (CLSI, 2004) were followed. First, the child's hand was thoroughly washed with soap and water. It was then cleaned with alcohol and contact with the cleaned area avoided to decrease the chance of contamination. The finger was punctured with a sterile lancet and the first drop of blood wiped off with gauze. The finger was gently milked to establish blood flow. The Sarstedt microvette CB300 tubes with EDTA anticoagulant were used to collect the capillary blood samples. The tubes were tapped to release the anticoagulant, and then held upright. As the tube filled automatically by capillary action, it was slowly turned to facilitate the mixing of the anticoagulant and the blood until it was filled to at least *3A* full, ensuring proper blood to EDTA ratio. The capillary tip of a full tube was sealed with the small cap and the large opening sealed with the l ^ e cap attached to the tube. The collection tube was then inserted into the amber protective tube and the latter inverted to further mix anticoagulant with the blood specimen. The primary specimen containers were labeled with the participants' name and date of birth. The Childhood lead screening sample submission forms, CLSL1 (09/03) were completed for each participant. All samples were refrigerated at 2 - 8°c and transported in conformity with the United States Postal Service and Department of Transportation Regulations for "Exempt Human Specimens" (http://pe.usps.gov/text/dmm300/601.htm. 2007).

c) **Sampling and Analysis of Lead levels in Environmental Samples**

A total 19 villages in which participating children were born were purposively selected for collection of environmental samples (water, soil, kales).

1. WATER

Sampling and Sample Treatment

Half litre plastic containers were thoroughly cleaned with deionized water. 500 millilitres of water sample was collected from central water collection points within each village.

Sample Digestion and Extraction

The method for determining total Lead in water was used. A known volume of well mixed water was transferred to a beaker and 3ml $HN0_3$ added. The solution was heated and evaporated to dryness then cooled. 3ml $HN0_3$ was added and heated until digestion was complete as indicated by light colored residue. 2ml HCL (1+1) was added and heated gently to dissolve residue. The watch glass and beaker were washed with water and the mixture filtered. The resultant filtrate was diluted with water to concentration within range of instrument.

2. SOIL

Sampling and Sample Treatment

Sampling sites within the villages included the grounds and areas around the homes, used by children as playing grounds. Half a kilogram (500gm) of soils was sampled by auger to a 15-cm depth at random positions in the sampling sites for each selected village. Each sample was packed into Ziploc bag, previously flushed with deionized water. The sampled soil was air dried in a dust free atmosphere. The dry soil was then disaggregated with pestle and mortar and screened through 80 mesh using a nylon sieve. All processed materials were categorized according to sampling location.

Sample Digestion and Extraction

Soil samples were extracted according to the procedure of Forayee et al (1994) with slight modifications. Duplicate sample aliquots (5g) were dried at 105°c and accurately weighed. Each aliquot was heated on a magnetic stirrer with 4M HCl (20ml) under reflux conditions for 1 hr. The digest was cooled and filtered through glass wool previously washed with 4M HCl. The filtrate was slowly evaporated to a volume of about 5ml before adding 15ml 1M ammonium acetate. The contents were boiled and quantitatively filtered (Whatman No. 42 double circle) into a 25ml

volumetric flask. The contents were allowed to cool and diluted to volume with deionized water. The solutions are then stored for lead analysis.

3. KALE

Sampling and Sample Treatment

Pooled sample of ten leaves of kale were bought from the markets and Kiosks identified by villagers as sources of vegetables during the interviews. Each pooled sample was packed into Ziploc bag, previously flushed with deionized water and labeled accordingly. At the chemistry laboratory, the leaves were flushed with deionized water to remove soil particles prior to drying in a forced draught, dust free oven for 24 hours at 105°C. Samples were then ground in a Wiley Laboratory Mill using a 0.5-mm stainless steel screen and stored in minigroup polythene bags.

Sample Digestion and Extraction

Samples were ashed according to the procedures of Jumba et al (1996a, 1996b). Each duplicate sample (2g) was placed into a preweighed 100-ml tall form beaker and dried in an oven at 105°C for 16 hours. The samples were removed from the oven and the actual dry matter was determined after cooling in a desiccator for 2 hours. The sample aliquots were wet-ashed under reflux conditions with 20ml $HN0_3$ for 1 hour at 150°C and then heated with 2ml $HCI0_4$

at 200 - 250°C until fumes of $HClO_4$ appear consistently for 20 minutes. The digests were allowed to cool, boiled with 15ml 2M HCl, cooled, filtered (whatman No. 40) to remove silica, and diluted to 25ml with deionized water. The filtrates were reserved for determination of lead concentrations.

Chemical Analysis using Flame Atomic Absorption Spectrometer

Lead levels in water, soil and kales were analysed by Flame Atomic Absorption Spectrometry [AAS] (Shimadzu AA 6300) using prescribed settings (Shimadzu Corporation, 2002). After choosing the proper hollow cathode lamp for the analysis, the lamp was allowed to warm up for a minimum of 15 minutes. During this period, the instrument was aligned; the monochromator positioned at the correct wavelength (283.3nm), the proper monochromator slit width selected (0.7nm), and the hollow cathode current adjusted according to the manufacturer's recommendation. Subsequently, the flame was lit and the flow of fuel (acetylene) and oxidant (air) regulated at 2litre/minute. The burner and nebulizer flow rate were adjusted for maximum percent absorption and stability, and the photometer balanced. A series of standards of the lead solution with concentration of lead 0, 2, 5, 10, 15, 20 ppm respectively, were run and a calibration curve constructed by plotting the concentrations of lead in the standard solutions against the absorbance **(Figure 2).** Shimadzu AA 6300 read the concentrations directly as the curve corrector was set to read out the concentrations.

Table 8: Concentration of Lead in Standard Lead solutions by Flame AAS

Standards	Test Concentration (ppm)	Absorbance
0	0	0.0004
2	2.1711	0.0272
5	4.8529	0.0548
10	10.6537	0.1145
15	15.2497	0.1618
20	19.5056	0.2056

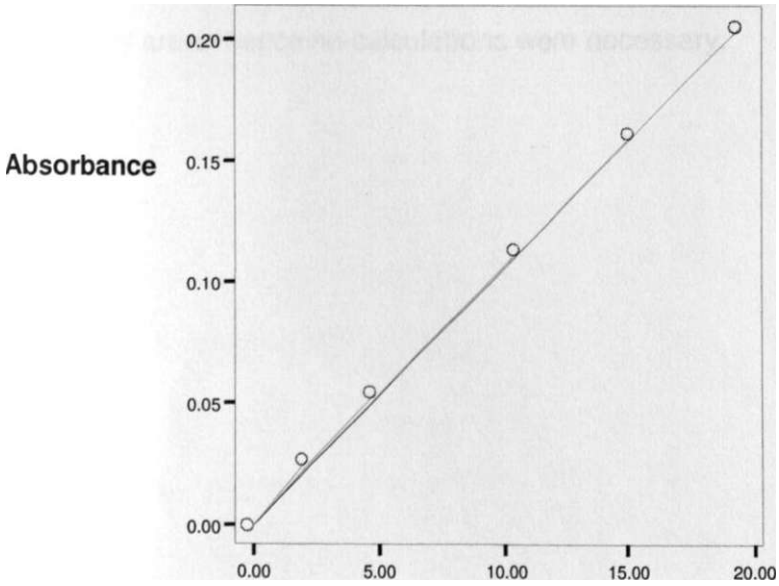


Figure 2: Calibration Curve for Flame AAS

The concentrations of lead in soil were reported as mg/kg (then converted to ug/kg by multiplying by 1000) dry weight. Since dried sample of soil were used, the formula (US/EPA, 1983) was used to calculate the lead concentration as follows:

$$\text{mg metal/kg sample} = \frac{A \times V}{D}$$

A = mg/l of metal in processed sample from calibration curve

V = final volume of the processed sample in ml, i.e 50ml

D = weight of dry sample in grams

There were no detectable levels of lead in Kales and drinking water drawn from the study areas hence no calculations were necessary.

4.7.1. INCLUSION AND EXCLUSION CRITERIA

a) **INCLUSION CRITERIA**

A child aged 6 - 5 9 month, who visited the Y2K program, had lived in Kibera since birth with consent to participate from the parents, guardians or care givers and had no exclusion criteria participated in the study.

b) • **EXCLUSION CRITERIA**

Children who did not meet the inclusion criteria according to medical assessment by the doctors were not included. These included but were not limited to all children with neurological e.g. celebral palsy, or digestive symptoms, with emergency conditions and those with a diagnosis of a chronic disease e.g. seizures disorders, diabetes, cancers or hematolof :al disorder or congenital disease or lead poisoning.

4.7.2 VARIABLES

1. **INDEPENDENT VARIABLES**

The following were the independent variables in the study:

a) Age of the child.

b) Sex of the child.

c) Source of water.

d) Source of Kales (a common vegetable foodstuff).

e) Parents, guardians or care givers awareness of childhood lead poisoning and sources of exposure.

2. DEPENDENT VARIABLES

Lead levels in blood, drinking water, Kales (vegetables) and soil were the dependent variables.

3. CONFOUNDERS

The following were the confounding variables in the study:

a) Respondents' educational level could have influenced responses to the questionnaire.

b) Children with previously undiagnosed nodical conditions that were not part of exclusion criteria.

c) Parents occupation may have introduced para-occupational hazards, which this study was, not be able to capture.

d) Breast-feeding history of the children.

e) History of paint pica.

f) Other unknown sources of exposure.

4.8. DATA COLLECTION

1. Quantitative Data

Sampling and analysis methods described in **section 4.7** were used to generate quantitative data.

2. Qualitative Data

Trained interviewer, using a standardized and coded questionnaire **(Appendix IV** and V) with mostly closed-ended questions, interviewed the parents, guardians or care givers of children who met the inclusion criteria, according to the medical officers at the clinic. After informed consent **(Apperdix VI and VII)** had been given, household data were collected pertaining to drinking water source, type of eating and drinking utensils, education and occupations of the parents, housing material, flaking interior paint, and the proximity of the house to a car battery smelter, gasoline seller, welder, and a major road, individual data was collected pertaining to age, gender, breast-feeding status, pica and use of lead containing traditional cosmetics or herbal preparations. Parental occupational exposure to lead were classified as low or high depending on whether or not they worked in industries known to process lead as well as by the nature of their job. Clearly, responses on risks for occupational lead exposure were not surrogates for biological monitoring hence blood lead testing on respondents would have been the best approach. Additional qualitative data were gathered through informal interviews of key informants for example, District Medical Officer of Health, District Public Health Officer, selected medical clinics in Kibera, Officer from

NEMA. Informal individual and group discussions with community and women groups were held. Observations were also conducted on water and food source, environmental sanitation and housing.

4.9. QUALITY CONTROL AND PROCESSING OF DATA

1. Quantitative Data

a) Blood Lead Analysis

I. Training of the principal investigator on use of LeadCare II at the Massechussett Public Health Laboratory, USA.

II. Training of involved Laboratory technologist in use of LeadCare II

III. Lead free environment for sample collection, using CDC guidelines for capillary blood sample collection.

IV. Use of certified controls for the LeadCare II prior commencement of testing daily.

V. Re-testing of randomly selected blood samples using the GFAAS at Massechussett Public Laboratory, USA.

b) Environmental Samples Analysis

i. Strict adherence to samples collection guidelines by Community Environmental Health resource centre (CEHRC, 2006)

II. Lead and dust free environment for storage, trea[1] lent and analysis

III. Experienced Environmental Chemists from the Department of Chemisty, University of Nairobi, Kenya

IV. Use of certified standards to calibrate the Flame AAS.

V. Adherence to the internal chemical laboratory quality controls

2. Qualitative Data

Prior to pre-testing the questionnaire, several key informants including parents, guardian, care givers, community based health workers, medical personnel and environmental experts were interviewed regarding general aspects of the community life with a focus on lead poisoning, maternal and child health, sources of water and local food consumption patterns. Observations were conducted on water and food sources, environmental situation and housing. Informal individual and group discussions were held with community and women groups' leaders. Information obtained was useful in improving the study instruments and results interpretation. The investigator personally conducted the informal key informants' interviews..

The personnel involved in the study were trained on the use of the various instruments of data collection. A training session was held prior to the onset of the survey, which included an orientation outlining the purpose of the survey, general lead information, and instructions on how to complete the questionnaires. Proper verbal etiquette, potentially difficult situations, and administrative details were discussed. Targets were assigned to each trained interviewer at conclusion

of the training session. The questionnaires were pretested during the training of the interviewers. Parents, guardians or care givers visiting the clinic who agreed to be interviewed were used to pretest the questionnaires. These parents, guardians or care givers then did not participate in the study proper. The findings of the pretest were used to improve the questionnaire and increase the reliability of the data collected.

4.10. DATA MANAGEMENT AND ANALYSIS

1. Data Management

Data were entered into Statistical Package for Social Sciences (SPSS) software, version 10.0.

2. Data Analysis

The CDC permissible childhood blood lead levels of 10 pg/dl was used as a cutoff point. The socio-demographic characteristics, residence characteristics, and potential risk factors for exposure to lead among children with blood lead levels £ 10 pg/dl were compared to those of children with blood lead levels <10 pg/dl. Where the numbers of children with blood lead levels £ 10|jg/dl was small and whenever possible, categorical variables were collapsed into a smaller number of categories to avoid cells with less than 5 individuals. Chi-square analysis was used to test statistical significance for categorical variables. Regression analysis was used to determine the association between

interval/ratio data and the strength of association, as well as to draw the estimation curves. Pearson's, Spearman's and Eta correlations were used to determine the measure of association. Statistical significance was set at p value of 0.05. Validity and predictive values of LeadCare II analyzer as a blood lead screening and confirmatory device for BLL were calci tted.

4.11 ETHICAL CONSIDERATIONS

1. The study protocol was presented to the Ethical and Research Committee which constituted the Kenyatta National Hospital and the College of Health Services, University of Nairobi, and approved.
2. Approval for shipment of blood samples for quality control purposes, to the USA was received from the Ethic committee, Kenya and CDC, USA.
3. Approval to conduct the study was granted by the administrator of Y2K program.
4. Parents, guardians or care givers gave informed consent.
5. Information on potential sources of lead and advisable activities that would reduce children's exposure was given at the end of the interview to all contacted parents, guardians or care givers, regardless of partic'nation.
6. When results were obtained, subjects were informed of their blood lead level.
7. Those with levels < 10 ug/dl were advised to avoid potential sources of lead exposure.

8. Parents, guardians or care givers of children with elevated blood lead levels were referred to Kenyatta National Hospital in accordance with CDC's guidelines **(Table 9).**

Table 9 - Medical management of children based on blood lead levels

BLOOD LEAD ! LEVELS (ug/dl)	RECOMMENDED ACTION
<10 **ug/dl,** Risk **Level** I	• Obtain careful environmental history • Provide risk reduction and nutrition education • If risk assessment indicates exposure to lead is likely, consider
10-14 **ug/dl,** Risk **Level II: Moderate**	• Report BLL to Ministry of Health • Obtain careful environmental history • Provide risk reduction and nutrition education • Repeat all capillary samples, confirming with a venous sample within 1 month for new cases and 1 to 3 months for known cases
15-19 **ug/dl,** Risk **Level II: Moderate**	• Follow steps above • If BLL remains 15 to 19 ug/dl for 3 months, proceed with actions for BLL 20 to 44 ug/dl. • Collaborate with NEMA, which can provide home inspection and other services • If initial sample was capillary, repeat with venous sample in 1 week to 1 month. The higher the BLL, the more urgent
20-44 **ug/dl,** Risk **Level III:** High	• Follow steps for child who has BLL 10 to 14 ug/dl. • Provide complete medical evaluation, including detailed environmental history, developmental assessment, physical examination and evaluation for iron deficiency. If particulate ingestion is suspected, obtain abdominal radiograph and order bowel decontamination if indicated. • Consider chelation therapy in consultation with a clinician experienced in lead toxicity treatment • Collaborate with NEMA, which can provide home inspection and other services
45- 69 ug/dl, Risk Level IV: Urgent	• Consider BLL with venous sample within 24 to 48 hours before initiating chelation • Provide or refer for chelation therapy within 48 hours. Child must be in a lead-safe environment during chelation • Follow all steps for BLL of 20 to 44 ug/dl • Perform complete neurological examination and consider free erythrocyte (FEP) or Zinc protoporphyrin (ZPP) testing to assist in evaluation child's response to management
>70 ug/dl, Risk Level V: Emergency	• Arrange immediate hospitalization and chelation at a facility that has expertise in treating lead-poisoned children. • Confirm BLL immediately with venous sample processed as an emergency laboratory test • Follow all steps for BLL 20 to 44 ug/dl • Perform complete neurological examination and consider FEP or ZPP testing to assist in evaluating child's response o management

Sources: 1. CDC, 2002

2. American Academy for Pediatrics, 1998

CHAPTER 5: RESULTS AND DI6 ;USSION

5.1: ORGANIZATION OF RESULTS

The study determined the blood lead levels (BLL) of children drawn from Kibera slums using LeadCare II analyzer. The sociodemographic characteristics of the children categorized by their blood lead levels as BLL > 10ug/dl and BLL < 10ug/dl respectively, consistent with WHO/CDC cut off level for lead poisoning, were compared. Selected blood samples were analyzed using both LeadCare II kits and GFAAS to facilitate comparison of values as well as determination of the validity measures and predictive values of LeadCare II analyzer as a blood lead screening device.

The study also determined the associations between the lead levels in various environmental samples (water, kales and soil) and blood lead levels found in the

<

children drawn from Kibera slums.

5.2: BLOOD LEAD LEVELS

The total number of children in the study was 387 children, with 52.8% and 47.2% being boys and girls respectively. The mean blood lead level (BLL) was 6.0ug/dl (median = 5.40ug/dl, SD =2.42, range 3.30 - 24.70ug/dl) **[Figure 3].** There were 27 (7%, N = 387) children with BLL > 10 ug/dl, which was above the WHO/CDC cut off level for lead poisoning. However, using the LeadCare II, it is recommended that a child with level £ 8 ug/dl should be re-tested with Atomic

absorption spectrophotometer (AAS) using venous blood samples. About 16.3% (N = 387) of the children tested had BLL £ 8ug/dl and recommendation for further testing was communicated to the parents, guardians or care givers.

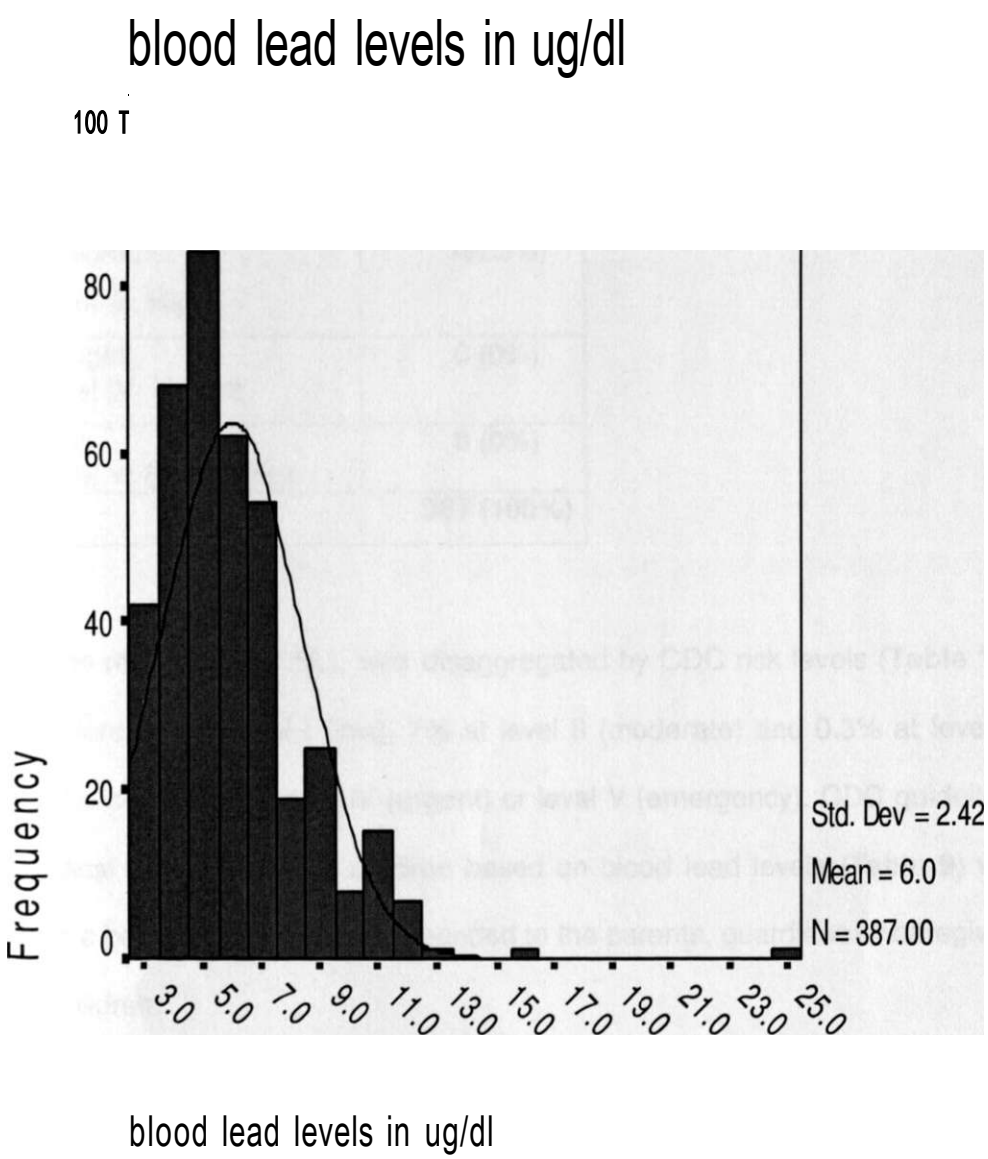


Figure 3: Frequency of Blood Lead Levels among 6 - 5 9 months Old in Kibera

Table **10: BLL Disaggregated by CDC Risk Levels (N = 387)**

Blood Lead Levels (ug/dl) categorized by risk levels	**n (%)N = 387**
<10 ug/dl, Risk Level I	359 (92.8%)
10-14 ug/dl, Risk Level II: Moderate	26 (6.7%)
15-19 ug/dl, Risk Level II: Moderate	1(0.3%)
20 - 44 ug/dl, Risk Level III: High	1(0.3%)
45 - 69 ug/dl, Risk Level IV: Urgent	0 (0%)
>70 ug/dl, Risk Level V: Emergency	0 (0%)
Total	**387 (100%)**

When the distribution of BLL was disaggregated by CDC risk levels **(Table 10),** 92.8% were at risk level I (low), 7% at level II (moderate) and 0.3% at level III (high). No child was at level IV (urgent) or level V (emergency). CDC guidelines for medical management of children based on blood lead levels **(Table 9)** was used as a basis for actions recommended to the parents, guardians or caregivers of the children.

5.3: BLL AND SOCIO-DEMOGRAPHIC PROFILES OF CHILDREN

Table 11 is a summary of the findings of sociodemographic characteristics of the children disaggregated by the two categories of blood lead levels.

Table 11: Socio-Demographic Characteristics of Children by Blood

	Blood Lead Concentration (ug/dl)	
	< 10 (N = 359) n(%)	**> 10 (N = 28) n (%)**
Mean BLL (Range)	5.54 (3.3 - 9.8)	11.85 (10.0-24.7)
Age (months)		
6 to 9	68 (18.9)	3(11.1)
10 to 19	102 (28.4)	7 (25.9)
20 to 29	53(14.5)	4 (14.8)
30 to 39	41 (11.4)	7 (25.9)
40 to 49	45 (12.5)	4(14.8)
50 to 59	50 (13.9)	2 (7.4)
Missing	1 (0.3)	0
Child's Sex		
Male	169 (47.1)	13(48.1)
Female	190 (52.9)	14 (51.9)
Breastfeeding History		
Never breast-fed	7(1.9)	o
Stopped > 1yr	112 (31.2)	9 (33.3)
Stopped < 1yr	75 (20.9)	6 (22.2)
Currently breastfeeding	165 (46.0)	12 (44.4)
Respondent's Sex		
Male	34 (9.5) .	1 (3.7)
Female	325 (90.5)	26 (96.3)
Respondent's Education		
Primary	167 (46.5)	13 (48.1)
Secondary	136 (37.9)	10(37.0)
Tertiary	45 (12.5)	4(14.8)
Others	11 (3.1)	0

Table 11: Socio-Demographic Characteristics *of* Children by Blood Lead Concentration (Continued)

Respondent's Occupation				
High Risk of lead exposure at work	2 (0.6)	2 (7.4)	4(1.0)	***OR = 14.28 (95% CI: 3.05 - 66.75) p = 0.001***
Low Risk of lead exposure at work	357 (99.4)	25 (92.6)	382 (99.0)	
House wall type				
Stone	87 (24.2)	6 (22.2)	93 (24.1)	
Mud	228 (63.5)	17(63.0)	245 (63.5)	
Iron Sheet	40 (11.1)	3(11.1)	43 (11.1)	
Wood	4(1.1)	0	4(1.0)	
Others	0	1 (3.7)	1 (0.3)	'"0.812
Source of Drinking Water				
Tap	355 (98.9)	27(100)	382 (99.0)	
Borehole	1 (0.3)	0	1 (0.3)	
Others	3 (0.8)	0	3 (0.8)	" " N
Source of Kales				
Kitchen garden	2 (0.6)	2 (7.4)	4(1.0)	***OR = 14.24 (95% CI: 3.05, 66.45) p = 0.001***
Market	238 (66.5)	18 (66.7)	256 (66.4)	
Others	118 (32.9)	7 (25.9)	125 (32.4)	
Awareness of Pb-Poisoning				
i es	21 (5.8)	0	21 (5.4)	
No	338 (93.9)	27(100)	366 (94.3)	"0.230
Number of Known sources of Lead				
Nil	346 (96.4)	27 (100)	373 (96.6)	
1 to 2	7(1.4)	0	5(1.3)	
3 to 4	5(1.9)	0	7(1.8)	"0.395

"Chi Square test for independence, A for nominal data with all cells having expected count more than 5*
***Spearman's correlation, /> for ordinal by ordinal variables*
****Collapsed to determine Chi square test of independence*
*****N- 100% of children with BLL > 10ug/dl*

There was an almost equal distribution by gender (48.1% male and 51.9% female) among the children found with blood lead levels £10 ug/dl **(Table 11).** Comparison between the children with BLL > 10ug/dl and those with higher BLL, did not yield a statistically significant difference in distribution of blood lead levels by sex (p=0.314).

Table 12: Mean Blood lead levels in ug/dl by Children's sex (N = 386)

Child's Sex	Mean BLL	N	Standard Deviation	Range	Minimum BLL in ug/ר,ו	Maximum BLL in ug/dl
Male	6.1088	182	2.6314	21.40	3.3'J	24.70
Female	5.8735	204	2.1892	9.10	3.30	12.40
Total	5.9845	386**	2.4075	21.40	3.30	24.70

*ANOVA: F = 0.918, p = 0.339; Eta = 0.049, Eta Squared = 0.002; **1 Missing value*

The mean BLL by sex was 6.11 ug/dl (SD 2.63) among males and 5.87ug/dl (SD 2.19) among females (p = 0.339) **(Table 12).** The proportion of the total variability in the BLL that was accounted for by variation in the sex was 0.002 (Eta-squared), hence a weak and statistically insignificant association (F= 0.918, p = 0.339).

Table 13: Blood Lead Levels by Age of the children In months (N = 386)

Age Vs BLL	0-19	20-39	40-59	Total
BLL< 10ug/dl	170	94	95	359
BLL > 10ug/dl	10	11	6	27
Total	180	105	101	**386

*Y = 2.908, df = 2,p = 0.234; **1 Missing value*

Modal age interval for blood lead levels £10 pg/dl wa< 20-39 months (41 %, n = 27). It was observed that proportionally fewer children had BLL > 10ug/dl after the 20 - 39 age interval **(Table** 13). There was no statistically significant association between the age of the children and BLL (p = 0.234).

Table 14: BLL by Respondents' highest educational level (N = 386)

BLL vs EDU	Primary	Secondary	Others	Total
BLL< 10ug/dl	167	136	56	359
BLL > 10ug/dl	13	10	4	27
Total	180	146	60	**386

*X^2 = 0.0134, df = 2, p = 0.993; **1 Missing value*

According to **Table 14,** 48.2% (n =27) of the respondents with children having BLL > 10ug/dl had primary level of education. Higher education level was not significantly associated with lower BLL (p = 0.993).

Table 15: Blood Lead Levels by children's Housing Type (N = 386)

BLL vs House Type	Stone (Permanent)	Others (Non-permanent)	Total
BLL< 10ug/dl	87	272	359
BLL > 10ug/dl	6	21	27
Total	93	293	**386

*X^2 = 0.0565, df = 1, p = 0.812; **1 Missing value*

Among the children with BLL > 10ug/dl, higher proportion (77.8%, n = 27) resided in semi - permanent and non-permanent housing **(Table 15).** There was however no significant association (p = 0.812) between type of housing and BLL.

Table 16: Blood Lead Levels by parental Lead Exposure risk (N = 386)

Occupational risk of lead exposure	BLL > 10ug/dl	BLL < 10ug/dl	Total
High Risk	2	2	4
Low Risk	25	357	382
Total	27	359	**386

*OR = 14.28 (95% CI: 3.05 - 66.75) p = 0.001; **1 Missing value*

Higher proportion **(92.6%,** n = **27)** of children with Bl ' £ 10 yg/dl **(Table 16),** had parents, guardians or care-givers who were occupational^ low-risk for lead exposure, and primarily used kales from market or kiosks **(92.6%,** n = **27)[Table 17].**

A child whose parent had low risk occupation and ate market/Kiosk vegetables was 14.28 (OR = 14.28; 95% CI: 3.05 - 66.75; p = 0.001) and 14.24 (OR:14.24; 95% CI: 3.05 - 66.45; p = 0.001) times respectively, more likely to have BLL > 10 (jg/dl than one whose parents had high risk occupation and ate kitchen garden vegetables **(Tables 16 and 17).**

Table 17: Distribution of Blood Lead Levels by Vegetables Source (N = 385)

Source of Vegetables	BLL > 10 pg/dl	BLL < 10ug/dl	Total
Kitchen Garden	2	2	**4**
Market/Kiosks	25	356	381
Total	27	358	385***

*OR = 14.24 (95% CI: 3.05 - 66.45) p = 0.001; ***2 Missing values omitted*

All the children with BLL >10 ug/dl (100%, n = 27) used tap water provided by the City council of Nairobi, as primary source of drinking water.

None of the parents, guardians or care givers of children with BLL £ 10ug/dl were either aware of lead poisoning (p = 0.230) or potential sources of lead (p = 0.395) **[Table 11].**

Table 18: Respondents' Education by the knowledge of Lead Poisoning (N = 385)

Knowledge Vs Education level	Yes	No	Total
Primary	4	176	180
Secondary	9	136	'45
Tertiary/others	8	52	60
Total	21	364	385***

*W- 11.043, df = 2, p = 0.004; ***2 Missing values omitted*

According to **Table 11,** about 6% (n = 359) of respondents whose children had BLL > 10ug/dl were aware of lead poisoning. Among this'group **(Table 18),** higher education level was significantly associated with more awareness of lead poisoning (X^2 = 11.043, df = 2, p = 0.004).

5.4: POTENTIAL RISK FACTORS FOR CHILDREN'S EXPOSURE TO LEAD

According to **Table 19,** Children with BLL > 10ug/dl, generally played on suspected contaminated grounds (77.8%, n = 28), and had soil or dust pica behavior (70.4%, n = 28). Compared with children who had blood lead levels below 10ug/dl, there was no statistically significant association between BLL £ 10ug/dl, and playing in contaminated grounds (OR = 0.89; 95%CI: 0.25 - 2.34, p = 0.627) or pica behavior (OR = 0.72; 95%CI: 0.31 - 1.68, p = 0.439). Similarly, there was no statistically significant association (p > 0.05), between BLL and the potential risk factors shown on **Table 19.**

Potential Risk Factors	Blood Lead Concentration (ug/dl) f< 10 (N = 359) n (%)	' > 10 (N = 28) n (%)	I Total (N = 387) n (%)	p value
Paint EXPosure				
Yes	108 (30.1)	7 (25.9)	115(29.8) •	OR - 1.23 (95%CI: 0.51 - 2.98). p= 0.649
No	251 (69.9)	20 (74.1)	271 (70.2)	
Contaminated Plavarounds				
Yes	272 (75.8)	21 (77.8)	293 (75.9)	OR = 0.89 (95%CI: 0.25 - 2.34), p = 0.627
No	87 (24.2)	6 (22.2)	93 (24.2)	
Knows Poisoned playmate				
Yes	2 (0.6)	1 (3.7)	3 (0.8)	OR = 0.073 (95%CI:0.004 - 0.75), p = 0.069
No	352 (99.4)	26 (96.3)	379 (99.2)	
OccuDationallv exposed parent				
Yes	105 (29.2)	10 (37.0)	115 (29.8	OR - 0.70 (95%CI:0.302 - 1.58), p = 0.356
No	254 (70.8)	17(63.0)	271 (70.2)	
Living near Lead industry				
Yes	89 (24.8)	4(14.8)	93 (24.1)	OR = 1.90 (95%OI:1.54 - 5.54). p = 0.279
No	270 (75.2)	23 (85.2)	293 (75.9)	
Pica Behaviour				
Yes	226 (63.0)	19 (70.4)	245 (63.5)	OR = 0.72 (95%CI: 0.31 - 1.68), p = 0.439
No	133 (37.1)	8 (29.6)	141 (36.6)	
Signs of Lead Poisoning				
Yes	34 (9.5) .	2 (7.4)	36 (9.3)	OR = 1 li (95%CI:0.30 - 5.76), p = 0.722
No	325 (90.5)	25 (92.6)	350 (90.7)	
Use glazed pottery				
Yes	64 (17.8)	5(18.5)	69(17.9)	OR = 0.10 (95%CI:0.35 - 2.62). p = 0.928
No	295 (82.2)	22 (81.5)	319 (82.1)	
Cosmetic Exposure				
Yes	52 (14.5)	5 (18.5)	57(14.8)	OR = 0.75 (95%CI:0.27 - 2.06), p =0.569
No	307 (85.5)	22 (81.5)	329 (85.2)	
Mental Development Concerns				
Yes	8 (2.2)	0	8(2.1)	
Mo	351 (97.8)	27 (100)	380 (97.9)	" N

It was noted on **Table 19 (**N)** that 100% of children ' rth BLL £ 10ug/dl had no mental development concerns.

5.5: CORRELATION OF BLL MEASURED BY LEADCARE II AND GFAAS

Graphite furnace atomic absorption spectrophotometer is considered the gold standard for BLL assay. To facilitate comparison of values of BLL obtained by using LeadCare II and GFAAS, lead analyses were done on duplicate blood samples drawn from 22 children **(Table 20).**

Table **20: BLL by LeadCare and GFAAS on duplicate blood samples (n = 22)**

Duplicate Sample code	Blood Lead Concentration (ug/dl) readings	
	LeadCare II	GFAAS
1	10.6	9.0
2	11.1	15.0
3	12.1	9.0
4	9.5	9.0
5	13.2	8.0
6	4.7	3.0
7	8.5	6.0
8	3.4	3.0
9	3.3	3.0
10	8.1	11.0
11	9.7	7.0
12	10.5	11.0
13	11.3	13.0
14	8.9	8.0
15	8.9	11.0
16	7.8	12.0
17	6.0	8.0
18	12.0	18.0
19	9.1	9.0
20	4.8	5.0
21	9.0	5.0
22	6.9	7.0

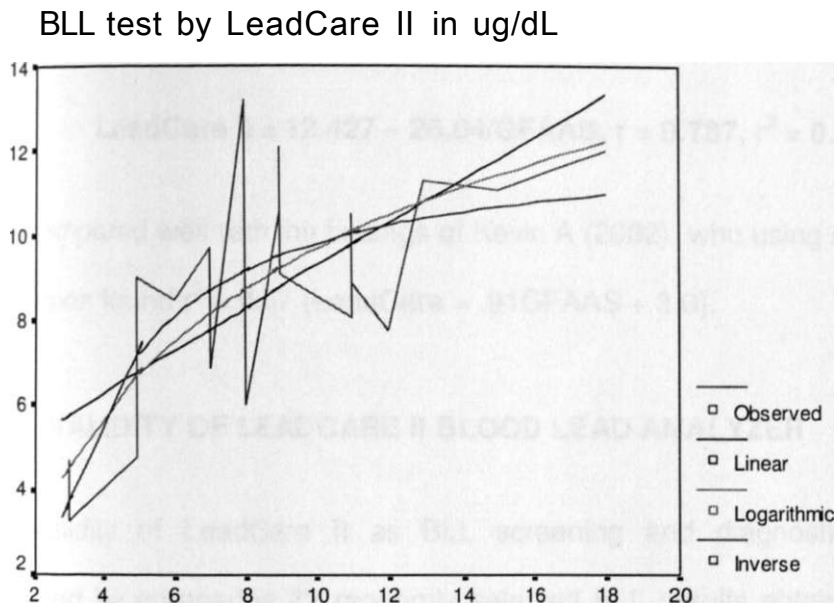


Figure 4: Regression Curves for BLL measured by GFAAS Vs LeadCare II

Figure 4 demonstrates the correlation between the 22 values of BLL obtained by using LeadCare II and GFAAS on duplicate samples as shown in **Table 20.** Various models were applied to determine which provided a strong correlation between the variables of interest. The Inverse model of regression generated a strongly positive (Pearson's correlation, $r = 0.787$, $r^2 = 0.62$), statistically significant correlation ($F = 32.64$, $p<0.05$) between the BLL determined using LeadCare II and GFAAS. Inverse regression estimated 62% of the reading of BLL by LeadCare II for a given value by GFAAS.

Regression equation using the inverse model generated is shown below:

LeadCare II = 12.427 - 26.04/GFAAS, r = 0.787, r^2 = 0.620

This compared well with the findings of Kevin A (2002), who using simple linear regression found r^2 = 0.67 (LeadCare = .91GFAAS + 3.0).

5.6: VALIDITY OF LEADCARE II BLOOD LEAD ANALYZER

The validity of LeadCare II as BLL screening and diagnostic device was assessed by comparing 22 randomly selected BLL results obtained using both LeadCare II and GFAAS **(Table 20),** the later being considered the gold standard for BLL assay. Validity is a measure of extent to which the LeadCare was capable of correctly diagnosing the presence (BLL > 10ug/dl) or absence (BLL > 10ug/dl) of Lead poisoning. Validity was expressed in terms of sensitivity and specificity. In order for these calculations to be made, parameters shown in Table 21 were computed.

Table 21: Relationship between Lead poisoning and screening test results

LeadCare Vs GFAAS	BLL > 10ug/dl	BLL < 10ug/dl
BLL > 10ug/dl	4 (TP - true positive)	3 (FP - false positive)
BLL < 10ug/dl	3 (FN - false negative)	12 (TN - true negative)

Table 22: Validity and Predicitive values using LeadCare II analyzer

	Measures	Formula	Value
Validity Testing	Sensitivity	TP/(TP + FN) X 100	57.14%
	Specificity	TN/(TN + FP) X 100	80%
Predicitive Values (PV)	Positive PV	TP/(TP + FP)	0.57
	Negative PV	TN/(FN + TN)	0.80

LeadCare II correctly identified 57% **(Table 22)** of children with BLL > 10ug/dl given the 57% sensitivity.

LeadCare II correctly identified 80% **(Table 22)** of c< ildren with BLL < 10ug/dl, given the 80% specificity. LeadCare II has a potential for use as a confirmatory testing device for lead poisoning.

5.6.1: PREDICTIVE VALUES

Given the test results obtained using LeadCare II analyzer, the likehood of lead poisoning actually being present or absent was tested by computing the predictive values.

Positive predictive value (PPV) of 0.57 **(Table 22)** showed about 60% chance that children who tested BLL £ 10ug/dl were actually lead poisoned. It is important to note that PPV increases with higher prevalence. Hence, the use of

LeadCare II as a screening device in the high prevalence settings would potentially be more cost effective.

Negative predictive value (NPV) of 0.8 **(Table 22),** showed about 80% chance that children who tested BLL < 10ug/dl were actually below the action level (BLL < 10ug/dl) for lead poisoning. NPV decreases as prevalence increases.

It is important to note that predictive value depends on the sensitivity and specificity of the test as well as on the prevalence of the disease in the population being tested. Even with a high sensitivity and high specificity, if the prevalence is low, the positive predictive value of a test may be low. Therefore, to improve on positive predictive value it would be beneficial to screen populations with high prevalence of the disease, using the targeted as opposed to universal screening strategy.

5.6.2: VARIANCE IN CLINICAL CLASSIFICATION BASED ON BLL

Using GFAAS as the gold standard, LeadCare II anaiyzer misclassified14% (N = 22) of samples as having BLL 2 10ug/dl when infact the converse was true. A similar percentage was misclassified as having BLL < 10ug/dL LeadCare II analyzer misclassified risk levels children based on the BLL **(Table** 9) of 36% (N = 22). Caution should therefore be exercised in the use of LeadCare II to direct medical management of the children based on BLL.

5.7: LEAD LEVELS IN ENVIRONMENTAL SAMPLES

The environmental samples analyzed in the study were soil, water and kales.

The results are detailed in the subsequent sections.

5.7.1: SOIL LEAD LEVELS

Duplicate soil samples per village were analyzed for lead levels and mean of the two readings reported as mean soil lead level for the given village. The findings are shown on **Figure 5** and **Table 23.**

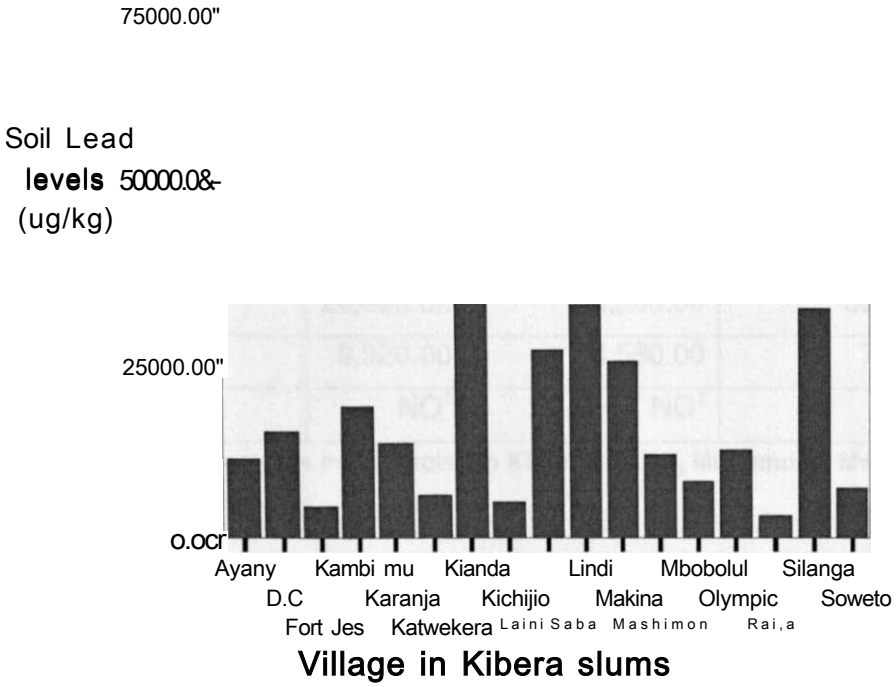


Figure 5: Soil Lead Levels in ug/kg by Village in Kibera slums

Table 23: Soil Lead Levels in ug/kg by Village in Kibera slums

	Soil Lead levels (ug/kg)		Mean Soil Pb (ug/kg)
	Sample 1	Sample 2	
Ayany	9,910.00	12,820.00	11,365.00
D.C	17,710.00	12,830.00	15,270.00
Darajani	3,416.00	29,890.00	16,653.00
Fort Jesus	4,580.00	4,590.00	4,585.00
Kambi muru	20,120.00	17,690.00	18,905.00
Karanja	16,700.00	10,420.00	13,560.00
Katwekera	6,040.00	0.00	6,040.00
Kianda	90,580.00	88,560.00	89,570.00
Kichijio	1,478.00	8,940.00	5,209.00
Laini Saba	28,380.00	25,470.00	26,925.00
Lindi	35,110.00	31,790.00	33,450.00
Makina	20,120.00	30,320.00	25,220.00
Mashimoni	15,270.00	8,470.00	11,870.00
Mbobolulu	6,030.00	10,410.00	8,220.00
Olympic	9,780.00	15,260.00	12,520.00
Raila	5,560.00	1,170.00	3,365.00
Silanga	24,990.00	40,500.00	32,745.00
Soweto	9,920.00	4,580.00	7,250.00
Others	NO[1]	NO[1]	NO[1]

1. NO = Soil samples not collected in Kisumu ndogo, Mugumoini, Mwemwbeni

The mean soil lead concentration in Kibera slums was 19,180 ug/kg (Range 3,365 - 89,570 ug/dl, SD = 20,521 ug/kg) which was above the WHO acceptable range of 100 - 200 ug/kg. Kianda village had the highest concentration of lead in the soil of 89,570 ug/kg way above the second highest at Lindi village at 33,450

ug/kg **(Figure 5** and **Table 23).** Further research should establish whether the high soil levels in Kianda were artefactual or indeed represented the true level of soil contamination. Raila village had the lowest concentration of 3,365 ug/kg. Physical visit to the villages however revealed that Kianda was situated at the junction of the railway line and the major road through the Kibera slums, whereas Raila village was along the railway line. Informal key informants' discussions with the local officers of health, environmental expert at NEMA and UNEP did not provide additional information to explain the differences in the soil lead levels in the various villages.

Table 24: Mean Blood Lead Levels by Village in Kibera slums

Name of Village in Kibera	Mean BLL (ug/dl)	N	Std. Deviation	Minimum BLL in ug/dl	Maximum BLL in ug/dl
1 Ayany	5.2852	27	1.7523	3.30	9.60
2 DC[1]	6.1800	35	2.4154	3.30	11.80
3 Darajani	6.4000	1	.ON*	6.40	6.40
4 Kianda	5.3532	77	1.7034	3.30	11.10
5 Kichijio	6.3538	13	3.4246	3.30	16.30
6 Katwekera	6.8037	27	2.4599	3.60	12.20
7 Karanja	5.9909	22	1.7509	3.30	9.30
8 Kisumu Ndogo	5.9833	6	1.1286	4.50	7.80
9 Kambi Muru	7.2750	4	4.1145	3.80	13.20
10 Fort Jesus	5.9187	16	2.6972	3.30	11.00
11 Laini Saba	5.8083	12	1.7207	3.30	9.30
12 Mashimoni	5.9474	19	2.2134	3.30	12.40
13 Mwembeni	5.1000	3	.5292	4.70	5.70
14 Mugumoini	3.3000	1	.ON[2].	3.30	3.30
15 Raila	4.1000	2	.9899	3.40	4.80
16 Soweto	6.9444	9	2.8623	4.30	11.30
17 Silanga	6.400C	£	2.8931	3.30	10.80
'18 Mbombolulu	5.650C	2	.6364	5.20	6.10
19 Olympic	6.2412	3^	4.040C	3.3C	24.70
20 Makongeni	6.050C)	2>	7.07E-02	6.0C	6.10
21 Kwa Nganga	6.700C)	1	.ON[2]	6.7C	6.70
22 Others	6.300C)	(3	2.6351	3.3C	- 10.70
23 Lindi	7.011	3	2.68163	3.3C	12.10
24 Makina	6.261	5'4	2.207C3	3.3C	11.50
jSample Mean	5.996	5 38"7	2.4163	3.3C	24.70

1. DC = District Officer's office area

2. ON = Villages which had only one participant in the study

Kianda village with SoilPb of 89,570ug/kg had mean BLL of 5.353 ug/dl SD=1.703, Range = 3.30 - 11.10 ug/dl) whereas Raila village with SoilPb of 3,365ug/kg had mean BLL of 4.10 ug/dl (SD = 0.99, Range = 3.40 - 4.80 ug/dl). 7.1% (n = 77) of children from Kianda tested had BLL £ 10 ug/dl compared to none from Raila village.

Table 25: **Soil Lead Concentration and Mean Blood Lead Level by Village**

Name of the village in Kibera	Soil Lead concentration (ug/kg)	Mean Blood Lead levels (ug/dl)
Ayany	11,365.00	5.285
D.C	15,270.00	6.180
Darajani	16,653.00	6.400
Fort Jesus	4,585.00	5.919
Kambi muru	18,905.00	7.275
Karanja	13,560.00	5.991
Katwekera	6,040.00	6.804
Kianda	89,570.00	5.353
Kichijio	5,209.00	6.353
Laini Saba	26,925.00	5.808
Lindi	33,450.00	7.011
Makina	25,220.00	6.261
Mashimoni	11,870.00	5.947
Mbobolulu	8,220.00	5.650
Olympic	12,520.00	> 6.241
Raila	3,365.00	4.100
Silanga	32,745.00	6.400
Soweto	7,250.00	6.944

Note: Villages with no figures for SoilPb have been omitted.

Measure of linear association yielded Pearson's correlation coefficient, r = 0.127 hence a weakly positive, statistically insignificant (p = 0.628) linear relationship between SoilPb and Mean BLL by village. Other regression models were used to determine the relationship between the two variables, MeanBLL and SoilPb, and best model that would explain the relationship. Tb-s cubic regression model provided the best correlation coefficient, r = 0.467. The coefficient of determination, r^2 was 0.218 as compared to linear regression with r^2 = 0.0160 (r = 0. 127). Using the cubic regression model, the proportion of the variance of MeanBLL explained by SoilPb was 21.8% compared to 1.6% by linear regression model. Hence, 21.8% of predictions on the value of dependent variable (MeanBLL) for a given value of independent variable (SoilPb) would be made using the formula below.

$$\text{MeanBLL} = 5.435 + 0.000129(\text{SoilPb}) - 5.509(\text{SoilPb})^2 + 4.278(\text{SoilPb})^3,$$

$$r = 0.467,\ r^2 = 0.218$$

There was low confidence (21.8%) in the use of this model for prediction of MeanBLL for a known SoilPb by village. The association between the variables of interest using the cubic model was statistically i: significant (F = 1.210, p = 0.345).

5.7.2: KALES AND DRINKING WATER LEAD LEVELS

Using sample preparation procedures described above **(Section 4.7)** and analyzing with Flame Atomic Absorption Spectrometry [AAS] (Shimadzu AA 6300), there were no detectable levels of lead in the samples of Kales and tap water collected from the villages that participated in the study.

5.8: BLOOD LEAD LEVELS AMONG CHILDREN IN SELECTED PARTS OF THE WORLD

Previous studies showed BLL in some developing countries ranging from a concentration mean of 1.96 jjg/dl in Jordan (Dabbas et al, 2000), to as high as 50-87% of children having BLL > 10 pg/dl in Cape Peninsula, South Africa (von Schirnding et al, 2001), and Dhaka, Bangladesh (Kaiser et al, 2001). Other reported BLL from various parts of the world are summarized in **Table 26**

Table 26: BLL of Children in selected parts of the world

Name of state/city .	Source of data	N	Ages of children (months)	(%)Children with BLL > 10ug/dl
Kibera, Nairobi	Tom Olewe, 2008	387"	0-59	7.0
Boston, USA	Mary Jean, 2001	24,332	0-59	6.2
Detroit, USA	Mary Jean, 2001	24,691	0-59	17.5
Milwaukee, USA	Mary Jean, 2001	19,829	0-59	26.5
[Philadelphia, USA	Mary Jean, 2001	31,146	0-59	28.0
Silesia, Poland	Jarosia, 2004	11, 877	24-84	13.0
Callao/Lima, Peru	Meneses, 2003	2510	*NO*	38.4
Morelos, Mexico	Espinoza, 2003	232	12- 144	29.7
! Mexico City, Mexico	Jimenez, 1993	113	36-84	76.0

NO = data missing

UNEP (2006) reports blood lead levels > 10ug/dl among children in Waithaka (5.8%), Kariobangi North (10%) and Babadogo (15.8%) in Nairobi. The prevalence of childhood lead poisoning in Kenya, however, remains unknown.

5.9: DISCUSSION

The prevalence of blood lead concentrations > 10 pg/dl among children aged 6-59 months old, drawn from Kibera was 7%. UNEP (2006) reported prevalences in Waithaka, kariobangi and Babadogo, Nairobi as 5.8%, 10% and 15.2% respectively. These prevalences are much higher than those reported among children in economically advantaged countries e.g. Boston city, Massechussetts, USA has a prevalence of 2% (Lead Screening Data, 2006) and 4.4% USA national prevalence (CDC, 1991 - 94). In these countries, decrease in blood lead concentrations was mainly attributed to the elimination of leaded petrol and lead soldered food cans, and de-leading of houses with lead paints. In spite of this, the prevalence of lead poisoning is still high among urban low-income populations, due to other sources of exposure (Pirkie et al, 1998; Karr et al, 1997). The same could also be true for Nairobi, where children could still be exposed to lead from leaded gasoline but most likely from other sources such as traditional cosmetics, lead water pipes, contaminated soil, occupational^ exposed parent and leaded cookery or pottery. The reported BLL in some developing countries ranged from a concentration mean of 1.96 pg/dl in Jordan (Dabbas et al, 2000), to as high as 50-87% of children having BLL £ 10 pg/dl in

Cape Peninsula, South Africa (von Schirnding et al, 2001) and Dhaka, Bangladesh (Kaiser et al, 2001).

LeadCare II offers a window of opportunity to cost effectively test for childhood lead poisoning in Kenya due to the strongly, positive correlation (Pearson's correlation, r = 0.787, r^2 = 0.62), between the BLL determined by LeadCare II and GFAAS. Whilst GFAAS is technical, expensive and inaccessible, LeadCare II is portable; battery operated and costs less per test. In this study, LeadCare II scored 57% sensitity, 80% specificity and PPV of 0.8. There is however need for more studies to determine the actual prevalence of childhood lead poisoning in Kenya because higher prevalence increases the PPV. LeadCare II therefore offers the potential for medical office-based measurement of BLL in children as well as, for screening and routine use in high prevalence, resource limited settings.

5.9.1: RISK FACTORS

In this study, living at the intersection of traffic-jammed road and railway line (Kianda village, 89,570ug/kg) more than quadrupled the exposure to high levels of lead in the soil. This finding is consistent with previous reports (Kaiser, 2001; Pirkle, 1994; Hashim, 2000), and could be attributed to the fact that Lead was previously deposited in the soil by vehicles and +-ains operating on leaded gasoline, in the absence of any emission control program in Kenya, until mid

2006. Young children, who spent most of their time at home, may have been exposed to lead through direct inhalation by playing in suspected lead contaminated grounds, or by ingesting deposited lead dust through their pica behaviour.

Another factor found to be strongly associated with elevated BLL is the low-risk occupation of the parents, guardians or care-givers. This could be attributed to fact questionnaires were probably not good surrogates for occupational exposure to lead. The study questionnaire did not probe to know whether workers knew what they were exposed to, how work clothes were handled or washed at home. Analysis of blood lead levels of the parents, guardians or care-givers would have been a better surrogate for exposure. A link has previously been made between elevated BLL and the exposure of children to the contaminated clothes at home (Morales, 1998; Whelan, 1997).

The lack of association between drinking of tap water and elevated BLL was noted. Analysis of the water samples revealed below detection levels of lead. Previously, Mungatana et al (2004) found lead levels in the tap water within the WHO acceptable levels. In Kibera slums, however, this was an interesting finding given the water pipes were proned to bursting hence contamination by the lead polluted soil.

The association between semi-permanent housing and elevated BLL could be explained by the fact that such houses also have dirt floors. Given the exploratory behaviour of the children and high concentration of SoilPb in the study area, the children were likely to get lead poisoned.

Lack of knowledge about lead poisoning and possible sources was not surprising given that the issues of lead have not been accorded publicity like other public health problems. Low level of education among Kibera dwellers (46.6%) could have also bee'n a contributing factor given that higher level of education among the respondents was significantly ($p = 0.004$) associated with more awareness among those whose children had BLL > 10ug/dl.

A few potential risk factors for elevated BLL deserve mentioning. These include the presence of small industries especially battery recycling in residential buildings, which was very common in Kibera. Proximity to industrial activity, a known risk factor (Morales et al, 1998), could also explain the high soil lead levels in Kibera. Other risk factors also include parental, guardians or care givers' low education and low income (Y2K serves the poor). These are socioeconomic indicators whose effects might have been mediated through other variables, such as parental, guardians or care givers' occupation. Other potential risk factors for elevated BLL among children, such as male gender e use of glazed pottery, or eating canned food (WHO, 1995; CDC, 1991) were not found to be significant in this study. No differences were noted in the mean BLL or proportion with elevated BLL by gender. An elevated BLL among boys is usually attributed to

more exploratory activities, which was not assessed in this study. Only 18.5% of the mothers whose children had BLL > 10ug/dl reported using glazed pottery to cook or serve food, and it was more common among the families of children with BLL within the permissible limits. This suggests that better quality glazed pottery were being used (Acra et al, 1981), or that children were not eating food served in pottery.

5.9.2: LIMITATIONS OF THE STUDY

There were certain limitations to the study that merit discussion. The study was cross-sectional in design. Habits and exposures may differ by age, especially among young children, thus affecting the blood lead concentration. The study sample, which was based in a medical center that also served as a primary care provider, may not be representative of the population as a whole. Accounting for refusal to participate and ineligibility, the participation rate was approximately 95%, however no information was collected on the children whose mothers refused to participate. The study sample size did not provide enough statistical power to compare the distribution of different variables between the two groups of children. In addition, exposures and health outcomes were self-reported. The standardized questionnaire, which was introduced by well-trained interviewers, might have reduced the bias but did not resolve the innate limitations of recall information, and lack of knowledge about lead poisoning and its sources.

The village sites selected for collection of environmental samples were not necessarily representative of the village hence the study was not able to identify the area-specific factors (intra-village factors) associated with elevated blood lead concentrations.

As a result of limited funds, pooled environmental samples of Kales, drinking water and soil were analyzed, further masking potential inter and intra-village variations. Lead levels in the air were not analyzed due to methodological and technological limitations

The analysis of the environmental samples for lead was a huge challenge given the limited number of institutions that could competently perform the requisite analysis. The need to establish a quality controlled environmental health laboratory cannot be overemphasized. Strengthening the capacity of the University of Nairobi to provide high quality laboratory services, would substantially contribute to better understanding of environment - mediated health problems in Kenya.

CHAPTER 6: CONCLUSIONS AND RECOMMENDATIONS

6.1: CONCLUSIONS

1. Sociodemograhic characteristic of the children

The study participants were aged 6 - 5 9 months. Out of the 387 children participants, the proportion of boys and girls were 52.8% and 47.2% respectively. Over 98% (N = 387) of children were with either breastfeeding at the time of study or had stopped breastfeeding. About 99% (N = 387) used tap water and market/kiosks as primary sources of water and kales respectively. About 80% (N = 387) of children lived in semi-permanent houses with dirt floors, characteristic of housing in the Kibera slums. About 90% (N = 387) of the respondents were women with 53% (N = 387) having at least secondary level of education. Over 99% of the parents, guardians or care-givers reported working in environments with low risk for lead exposure.

2. Blood lead levels of the participants

The study concluded that there was childhood lead poisoning among the children tested. Twenty seven, 27 (7%, N = 387) children had BLL £ 10 ug/dl, which is above the WHO/CDC cut off level for lead poisoning. The mean blood lead level (BLL) was 6.00ug/dl (median = 5.40ug/dl, SD =2.42, range 3.30 - 24.70ug/dl).

About 16.3% (N = 387) of the children in the study had BLL £ 8ug/dl using the LeadCare II analyzer. Kibera slum is presumed to be potentially a high

prevalence area hence confirmatory testing of venous blood samples using GFAAS for children with BLL > 8ug/dl is recommended. GFAAS is costly and unavailable in most medical centres. LeadCare II blood lead analyzer was found to have sensitivity of 60%, specificity of 80% and PPV of 0.60. It could be a viable alternative for resource limited settings like Kenya. Since low prevalence of BLL > 10ug/dl would lower the PPV, it would be beneficial to screen populations with potentially high prevalence of BLL > 10ug/dl, using the targeted as opposed to universal screening strategy.

Blood lead levels > 10 ug/dl among the children was associated with non-permanent housing (X^2 = 0.0565, df = 1, p = 0.812), playing on potentially lead contaminated grounds (OR = 0.89; 95%CI: 0.25 - 2.34, p = 0.627) and pica behaviour (OR = 0.72; 95%CI: 0.31 - 1.68, p = 0.439).

Low risk parental occupation (OR = 14.28; 95% CI: 3.05 - 66.75; p = 0.001) was significantly associated with BLL > 10ug/dl among the children. The questionnaire was probably not the best surrogate for occupational lead exposure hence those classified as low risk for lead exposure could, infact be high risk.

Kales sourced from the market/kiosks (OR = 14.24; 95% CI: 3.05 - 66.72; p = 0.001) were significantly associated with BLL > 10ug/dl yet concentration of lead in analyzed kales were below detectable levels. It is possible that the kales

analyzed for lead concentration were from sources different from those ingested by the children in the study.

3.- Environmental lead levels

Soil was a significant potential source of lead exposure among the children in Kibera slums. The mean soil lead concentration was 19,180 ug/kg (Range 3,365 -89,570 ug/kg, SD = 20,521 ug/kg) which was above the WHO acceptable range of 100 - 200 ug/kg. Proximity to the road and railway line was associated with higher soil lead levels e.g Kianda which is at the junction of a main road and railway line had levels as high as 89,570 ug/kg i.e over 400 times above the WHO allowable levels. Subsequent to banning of leaded fuel, the deposition of lead compound from combusted fuel in the soil is expected to reduce. However, other potential sources of environmental lead exposu i are still unknown.

4. Awareness of the lead poisoning and potential sources of lead exposure

The knowledge of lead poisoning and potential sources of exposure was very low. Only 5.4 % the respondents interviewed were aware of lead poisoning while about 3% knew more than one potential source of lead poisoning. Tertiary education and above, was associated with awareness of lead poisoning and knowledge of at least one potential source of exposure, among the respondents whose children had BLL £ 10ug/dl. Advocacy would be critical to bridging the knowledge and awareness gap.

5. Comparison of BLL in Kibera, Kenya with other countries

A prevalence of 7% of blood lead concentrations > 10 pg/dl among the study participants was both higher than those reported among children in economically advantaged countries. Whilst USA national prevalence (CDC, 1991 - 94) was 4.4%, poor neighbourhood in some states had higher rate e.g Boston city, Massachussetts 6.2%; Detroit, Michigan 17.5% (Mary et al, 2001). Prevalence in developing countries were generally higher than the study findings, for instance, as high as 50-87% of children had BLL > 10 pg/dl in Cape Peninsula, South Africa (von Schirnding et al, 2001), and Dhaka, Bangladesh (Kaiser et al, 2001). Socioeconomic factors seem to play some roles in childhood lead poisoning. Given the socioeconomic status of most Kenyans, the 7% prevalence among study participants raised a health flag that must oe further investigated and addressed.

6.2: RECOMMENDATIONS

This study established the presence of childhood lead poisoning in Kibera slums. Previously, UNEP (2006) reported prevalence of BLL £ 10ug/dl among studied children in Waithaka, Kariobangi North and Babadogo, Nairobi as 5.8%, 10% and 15.2% respectively. There is sufficient evidence of high levels of environmental lead in Nairobi as documented by Mungatana et al (2004) and UNEP (2006), hence the need to address childhood lead poisoning in Nairobi and probably, Kenya can not be overemphasized.

Strategies to prevent childhood lead poisoning are diverse but must be locally specific and should be supported at national and international levels (Chisolm, 2001; Landrigan, 2000; Needleman, 1998; UNEP - UNICEF information series, 1997). They should be sensitive to differing cultural, political, economical, technological and developmental circumstances. Three levels of interventions are recommended (UNEP/UNICEF, 1997), namely:-

A. HOUSEHOLD LEVEL

The local leaderships e.g. chiefs, community based rivironmental organizations and medical practitioners should be involved in educating the parents. Educating parents has proved to be effective approach for children with mild and moderate lead poisoning (MMWR, 2005) and therefore should be encouraged to:

1. Test child blood for lead levels at 12 or 24 months of age (or even at 6 months, depending on the presence of sources of lead in the child's environment), and if possible, routinely until age six (6).
2. Detect possible symptoms, as a child may no't feel ill. Symptoms which are commonly associated with other childhood ailments and thus can be difficult to distinguish include: stomach or headaches, poor appetite, disturbed sleeping pattern or irritableness.
3. Feed the children on a nutritious diet (regular meals, foods rich in calcium and iron).

B. COMMUNITY LEVEL

The environmental protection division of the local authorities in collaboration with community agencies and leaders should engage the communities. Everyone who cares for children should be aware of. the problems caused by lead.

1. Community-wide lead poisoning prevention and educational activities should be promoted, including the discouragement of the use of local cosmetics and fold remedies containing lead. Environmental intervention programs are more effective when the affe< ad communities carry out health education/intervention programs geared towards the development of individualized strategies for handling the risk implied by the presence of lead in the atmosphere (Saiud Publica Mex. 45 Suppl 2, 2003).
2. Local authorities should be trained to identify and monitor for the presence of point sources of community lead pollution e.g. battery recycling plants.
3. The ability of medical facilities and practitioners to monitor and treat lead poisoning should be strengthened by the Ministry of health and the local authorities through their medical officers of health.
4. Improvement of housing among the urban poor and children's playing grounds under slum upgrading program of Ministry of housing e.g. by planting grass, as a long term strategy would go along way to protecting the children from the highly lead contaminated soil.

c. **NATIONAL LEVEL**

Environmental, public health, economic and legal aspects of the issue should be examined in an integrated way to enable comprehensive and feasible strategies, to be implemented at the local level.

1. Strengthen the legal framework of control through parliament to enable NEMA provide adequate enforcement ana oversight measures to eliminate lead, otherwise both the existing standards and the new ones will be useless to improve the situation.
2. Parliament should enact legislation to strengthen the legal framework to enable NEMA to encourage and/or reinforce the regulation, reduction or abolition of lead in products such as; gasoline, paint, water (and water piping and fixtures), ceramic glazed and solder on food cans etc.
3. Government through NEMA should develop community (environment community-based organizations), national (universities, research institutions) or international collaboration (UNEP, UNICEF, ILO), in order to ensure high quality laboratory analysis of blood and environmental samples for lead. This capability is absolutely essential for the generation of reliable and accurate information on lead-related public health problems. Monitoring of populations and env:"C ment should be backed.

4. Technological changes should be supported.

- The long-term benefits and costs of reducing and substituting lead in products and industrial processes should be determined. For example, technical solutions for phasing out lead in gasoline were relatively easy and the costs were modest, making it particularly cost-effective policy.
- Use of more cost effective lead exposure m .nitoring devices e.g. blood lead testing using LeadCare II blood lead analyzer could potentially result in prompt interventions.

5. Exposures to lead in the workplace should be strictly regulated and limited to reduce para-occupational exposures at home. This should be done by the Ministry of labour through the inspectorate of occupational health and safety in collaboration with Central organization of trade unions (COTU) and Federation of Kenya employers (FKE). •
6. Advances in clinical care should be supported and fostered. Currently in Kenya, screening for lead poisoning is an exception in clinical settings. Ministry of public health and sanitation should facilitate the mainstreaming of issues around childhood lead poisoning, its causes, prevention and clinical management.
7. Government through NEMA should develop policies through the participation of all stakeholders with a direc interest in reducing lead exposure such as; government agencies, large and small industries, organized labor, public health care providers, and environmental and community groups.

Future research should among other areas:

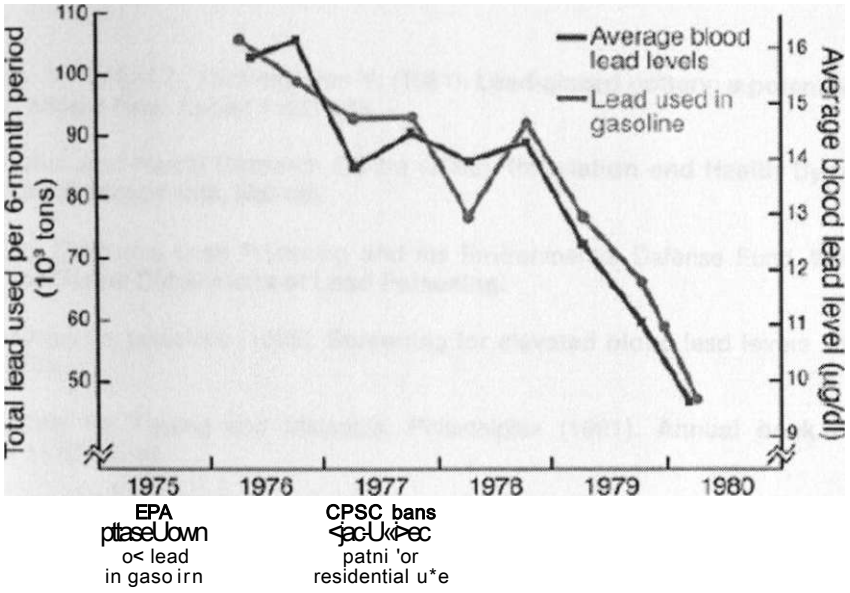
a) Endevour to document the actual prevalence of childhood lead poisoning in the general population of children. This is an important consideration because the results of this study were based on clinic population and sampling not random. Given that the predictive values are affected by prevalence, knowledge of the prevalence of childhood lead poisoning would go a long way in improving the PPV of LeadCare II analyzer as a potential blood lead screening device.

b) Validate LeadCare II analyzer and other affordable devices available in the market, for use as blood lead screening devices in resource limited settings.

c) Determine the socio-demographic determinants of high BLL among children in Kenya

d) Identify and examine the contribution of potential environmental sources, such as drinking water (not tap), vegetables irrigated with water contaminated with heavy metals and paint, to elevated BLL.

e) Develop strategies to monitor the trends in childhood lead poisoning following the ban of.leadri fuel in Kenya in 2006. Following the rulings of the Environmental Protection Agency, USA to phasedown the use of lead in gasoline in 1975 and U.S. Consumer Product Safety Commission (CPSC). that paint intended for residential use could not contain more than 0.06% lead by dry weight (David et al, 2006), there was reduction in BLL **(Figure 6).**



Source: National Health and Nutrition Examination Survey II (David et al, 2006)

Figure 6: Parallel decreases in blood lead levels and the amount of lead used in gasoline, 1976-1980, USA.

f) Determine the role of transplacental transfer of lead from mother to child as well as breastfeeding, in childhood lead poisoning.

g) Compare the magnitude of the childhood lead poisoning in rural and urban settings of Kenya.

h) Re-test the children tested in this study, one year after the first blood lead testing to determine the current blood lead burden.

i) Identify environmental hotspots for childhood lead exposures and map them using geographical information system (GIS) to inform the policy makers and guide lead screening strategies.

REFERENCES

Acra A., Dajani R., Raffoul Z., Karahagopian Y. (1981). **Lead-glazed pottery: a potential health** hazard **in the Middle East.** ***Lancet*** 1:433-434.

i

African Population and Health Research Centre (2002). **Population and Health Dynamics in** Nairobi**'s informal settlements, Nairobi.**

Alliance to End Childhood Lead Poisoning and the Environmental Defense Fund, Washington D C (1994). **The Global Dimensions of Lead Poisoning.**

American academy **for** pediatrics (1998). **Screening for elevated blood lead levels,** ***Pediatrics;*** 101:1072- 1078.

American Society for Testing and Materials, Philadelphia (1991). **Annual book of ASTM** standards. V 11.01: 45-47.

Anderson AC, Pueschel SM, Linkakis JG (1995). **Pathophysiology of lead poisoning.** In: Pueschel SM, editor. Lead poisoning in childhood. Baltimore: Brookes Publishing, p 75 - 95.

Bangkok Post (1996). In the USA, the banning of tetraethyl lead from gasoline resulted, on average, in a fall of the level of lead in blood by 75% (New York Times, 13 March 1996). Studies have also shown that lead poisoning is decreasing in Bangkok, Thailand, with increased use of unleaded petrol.

Centers for Disease Control and Prevention (2005). **Blood lead levels - United States, 1999-2002;** ***MMWR Morb Mortal Wkly Rep.*** 27; 54(20):513-6.

Boston Public Health commission office of Environmental Health (2006). **Lead Screening Data.**

Canfield RL, Henderson CR Jr, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP (2003). **Intellectual impairment in children with blood lead concentrations below 10 micrograms** per **deciliter.** ***N Engl J Med.*** l348:1517-1526.

Centers for Disease Control and Prevention (2002). **Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood** Lead **Poisoning Prevention.**

Centers for Disease Control and Prevention (2001). **Trends in blood lead levels among children-Boston, Massachusetts, 1994-1999.** ***MMWR Morb. Mortal. Wkly. Rep.;*** **4;** 50(17):337-9.

Centers for Disease Control and Prevention (1991). **Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control, Atlanta, GA: US Dept of Health and Human Services.**

Centers ior Disease Control and Prevention (1997). **Screening Young Children for Lead poisoning. Guidance for State and Local Public Health Officials.**

Centers for Disease Control and Prevention (1991). **Preventing lead poisoning in young children: DHHS report 2230.**

Centers for Disease Control and Prevention (1997). **Blood Lead Levels - United States 1991 -** 1994; ***Morbidity and Martality Weekly Report***, 46:141 - 146.

Centers for Disease Control and Prevention (2002). **Managing elevated blood lead levels among young children: recommendations from the Adv'sory Committee on Childhood Lead Poisoning Prevention.** 128 pp.

Central Bureau of statistics. Ministry of Finance and Planning - Kenya (2001). **1999 Population and housing census.** Volume I.

Chisolm JJ Jr (2001). **The road to primary prevention of lead toxicity in children** *Pediatrics* 107:581-583.

Clinical and Laboratory Standards Institute (2004). **Procedure for the handling and processing of blood specimen; Approved guideline - 3rd Ed.**

de Burbure C., Buchet J.P., Leroyer A., Nisse C., Haguenoer J.M., Mutti A., Smerhovsky Z., Cikrt M., Trzcinka-Ochocka M., Razniewska G., Jakubowski M., Bernard A. (2006). **Renal and neurologic effects of cadmium, lead, mercury, and arsenic in children: Evidence of early effects and multiple interactions at environmental exposure levels.** *Environ Health Perspect* 114(4): 584-90.

Dabbas M.A., Al-Zoubi M.A. (2000) **Blood lead level in the Jordanian population.** *Saudi Med J* 21:964-967.

David C.B., Andrew M.B. (2006). Childhood lead poisoning: The torturous path from science to policy; ***J. Clin. Invest.*** 116:853-857.

Dobson, A.J. (1984), **Calculating sample size.** *The Menzies Foundation,* Vol. 1, Pg. 75 - 79.

Chivian E. (1994). **Toxic exposures have been reported among residents in China, India, the Middle-East and South America: Critical Condition.**

Environmental intervention in sites contaminated by lead: the United States of America experience (2003). *Salud Publica Mex.* 45 Suppl 2:S232-6.

Environment Liaison Centre International Office for Africa (2003). **Get the lead out!** *Ecoforum,* Volume-26 Number 1.

Espinoza R., Hernendez-Avila M., Narciso J., Castaaga C., Moscoso S., Ortiz G.,Carbajal L., Wegner S., Noonan G. (2003). **Determinants of blood-lead levels in children in Callao and Lima metropolitan area.** *Salud Publica Mex.* 45 Suppl 2:S209-19.

Fernando B., Jose E.T., Raquel F.G., Patrick J.P. (2005). **A Critical Review of Biomarkers Used for Monitoring Human Exposure to Lead: Advantages, Limitations, and Future Needs.** *Environ Health Perspect.* **113:1669-1674.**

Flajnik C., Shrader D. (1994). **Determination of lead in blood by GFAAS-Deuterium and Zeeman Background Correction.** *American Clinical Laboratory* 13:45-7.

Flores J., Albert L.A. (2004). **Environmental lead in Mexico, 1990-2002.** *Rev Environ Contam Toxicol* 181:37-109.

GoK/ UNHABITAT (2004). **Kibera Social and Economic Mapping; Household Survey report.**

Hashim J.H., Hashim Z., Omar A., Shamsudin S.B. (2000). **Blood lead levels of urban and rural Malaysian primary school children** *Asia Pac J Public Health* 12:65-70.

Haward H. (2001). **Harrison's Principles of Internal Medicine 15th Edition © *by Graw-Hill Companies, Inc;***

Harvard Medical School (1996). A recent American national survey estimated that one in eleven children under age 5 had high lead concentrations in their blood. Among some minority sub-groups a much higher rate was found. For example, one in five non-Hispanic black children under age 5 had high blood-lead concentrations.

Health Protection Branch Laboratories, Bureau of Chemical Safety, Ottawa (1980). **Laboratory Procedure LPFC-110.**

Henry F. (2003). **Case Study of Lead Poisoning; *Pediatrics*** 112: 259-264.

n:tp://pe.usDs.Qov **(2007). Packaging exempt human or animal specimen**

Jarosia D., Peddada S., Rogan W.J. (2004). **Assessment of lead exposure and associated risk factors in urban children in Silesia, Poland. *Environ. Res.*** 95(2):133-42.

Jimenez C., Romieu I., Palazuelos E„ Murtoz I., Cortis M„ Rivero A., Catala J. (1993). **Environmental exposure factors and the concentrations of blood lead in Mexico City children; *Salud Publica Mex.*** 35(6):599-606.

Jumba I., Suttle N.F., Hunter E.A., Wandiga S.O. (1996). **Mineral composition of tropical forages in the Mount Elgon region of Kenya, 1. *Macromineral. Trop Agric (Trinidad)*** 7:108 - **112.**

Jumba I., Suttle N.F., Hunter E.A., Wandiga S.O. (1996). **Mineral composition of tropical forages in the Mount Elgon region of Kenya,** 2. ***Trace elements. Trop Agric (Trinidad)*** 73: 114 -419.

Karr M., Mira M., **Causer** J., Burn M. (1997). **Blood lead concentrations and iron status of preschool children from low income families. *Med J Aust,*** 1

Kaiser R., Henderson A.K., Daley W.R., Naughton M., Khan M.H., Rahman M., Kieszak S., Rubin C.H. (2001). **Blood lead levels of primary school children in Dhaka, Bangladesh. *Environ Health Perspect*** 109:563-566.

Kevin A. (2002). **Analytical Instruement Performance Criteria, On-site Measurement of Blood-Lead Concentrations using Field Portable Electro-analysis. *App Occ Environ Hygiene,*** 17(12):818 - 821.

LaDou J. (1990). **Occupational medicine.** Norwalk: Appleton and Lange 595 p.

Landrigan P.J. (2000). **Pediatric lead poisoning: is there a threshold? *Public Health Hep,*** 115:530-531.

Lauralynn T., Robert L.J., Lorna K., James A.D., Kevin A., Wayne T.S. (2001). **Evaluation of a Portable blood lead analyzer with occupational^ exposed populations. *Am. J. Ind. Med,*** 40:354-362.

Markowitz M. (2000): **Lead Poisoning. *Pediatr Rev;*** 21: 327-35.

Mathee A., von Schirnding Y., Levin J., Ismail A., Huntley R., Cantrell A. (2002). **A survey of blood lead levels among young Johannesburg school children. *Environ. Res***; 90(3):181-4.

Mathee A., von Schirnding Y., Montgomery M., Rollin H. (2004). **Lead poisoning in South African children: the hazard is at home.** *Rev Environ Health;* 19(3-4): 347-61.

Matte T.D., Figueroa J.**P.,** Ostrowski S. **(1989). Lead poisoning among household members exposed to lead-acid battery repair shops in Kingston, Jamaica,** *int J Epidemiol.* **18:874-881.**

Mayan O.N., **Henriques A.T.,** Calheiros J.M. (2001) **Childhood lead exposure in Oporto, Portugal.** *IntJOccup Environ Health.* 7(3):209-16.

Meneses-Gonzalez F., Richardson V., Lino-Gonzalez M., Vidal M.T. (2003). **Blood lead levels** and **exposure factors in children of Morelos state, Mexico.** *Salud Publica Mex.* 45 Suppl 2:S203-8.

Meyer P.A., Ugeehin M.A., Falk H. (2003). **A global approach to childhood lead poisoning prevention.** *Int J Hyg Environ Health* 206(4-5): 363-9.

Michigan Department of Environmental Quality (2005). **Soil fraction preparation for lead analysis.** *MDEQ SOP #213, REVISION #2.*

Momeshora C., Osibanjo O., Ajayi S.O. (1981). **Pollution studies on Nigerian rivers. Toxic heavy metals status on surface waters in Ibadan city.** *Environmental International 5:49-53.*

Morales B.C., Mauss E.A. **(1998). A community-initiated study of blood lead levels of Nicaraguan children living near a battery factory** *Am J Public Health* **88:1843-1845.**

Mungatana A. (2004). **Policy Instruments for the lead phase out in Kenya.**

Needleman H.L. (1998). **Childhood lead poisoning: the pron se and abandonment of primary prevention** *Am J Public Health*, 88:1871 -1877:

New York City, **Department** of Health and Mental Hygiene (2004). **Childhood Lead Poisoning Prevention and Management,** Vol. 23(5):23-28.

New York Times (2/2/1996); Studies in the USA show that aggressiveness and delinquency in boys is associated to lead in bones. It is suggested that this could lead to more serious antisocial behaviour in later life.

Papanikolaou N.C., Hatzidaki E.G., Belivanis S„ Tzanakakis G.N., Tsatsakis A.M. (2005). Lead toxicity **update. A brief review.** *Med Sci Monit'* 11(10): RA329 36.

Pirkle J.L., Kaufmann R.B., Brody D.J.,' Hickman T., Gunter E.W., Paschal D.C. (1998). **Exposure of the U.S. population to lead, 1991-1994** *EnvironHealth Perspect* 106:745-750.

Pirkle J.L., Brody **D.J.,** Gunter E.W., Kramer R.A., Paschal D.C., Flegal K.M., Matte T.D. (1994). **The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES)** *JAMA* 272:284-291.

Sanborn M.D., Abelsohn A., Campbell M., Weir E. (2002). **Identifying and managing adverse environmental health effects: 3. Lead exposure.** *CMAJ.* 14; 166(10):1287-92.

Schlenker T.L., Fritz C.J., Mark D. (1994)1. **Screening for pediatric lead poisoning: comparability of simultaneously drawn capillary and venous blood samples.** *JAMA* 271:1346-8.

Schonfeld D.J., Rainey P.M., Cullen M.R., Showalter D.R., Cicchetti D.V. (1995). **Screening for** lead **poisoning by fingerstick in suburban pediatric practices.** *Arch Pediatr Adolesc Med;* 149(4): 447-50.

Schoen E.J. (1992). **Lead toxicity in the 21st century: will we still be treating it? [letter]** *Pediatrics* 90:481-482.

Shimadzu Corporation Analytical and Measuring Instruments Division (2002). Shimadzu **AA6300: Atomic Absorption Spectrometer instruction manual.**

Syagga, P.M. (2001). **Integrated Multi Sectoral and Sectoral Urban Development in Kenya: Working Paper 3.** *The Schumacher Centre for Technology and Development, Rugby, United Kingdom*, pp. 30 33; 2001.

UNDP/World Bank ESMAP (2003). **Phase-Out of Leaded Gasoline in Oil Importing Countries** of Sub**-Saharan Africa: The Case of Ethiopia Action Plan.**

UNEP - UNICEF information series (1997). **Childhood lead poisoning; information on** advocacy **and action.**

UNEP (2006). **Environmental pollution and impacts on public health.**

United Nations Children's Fund and United Nations Environmental Programme (1997). Childhood **Lead Poisoning: Information for advocacy and Action.**

US/EPA (1996). **The Arc's Questions and Answers on** Lead **Poisoning: Childhood Lead Poisoning Prevention.**

U. S. Environmental Protection Agency (1983). **Metals (atomic absorption methods) - General procedure for analysis by atomic absorption - Methods for Chemical Analysis of Water and** Wastes, pp. 67-70.

von Schirnding Y., Mathee **A.,** Robertson P., Strauss N„ Kibel M. (2001): **Distribution of blood** lead **levels in schoolchildren in selected Cape Peninsula suburbs subsequent to reductions in petrol lead.** *S Afr Med J* 91:870-872.

W B Saunders (2003).**Nelson Textbook of Pediatrics 17th edition.**

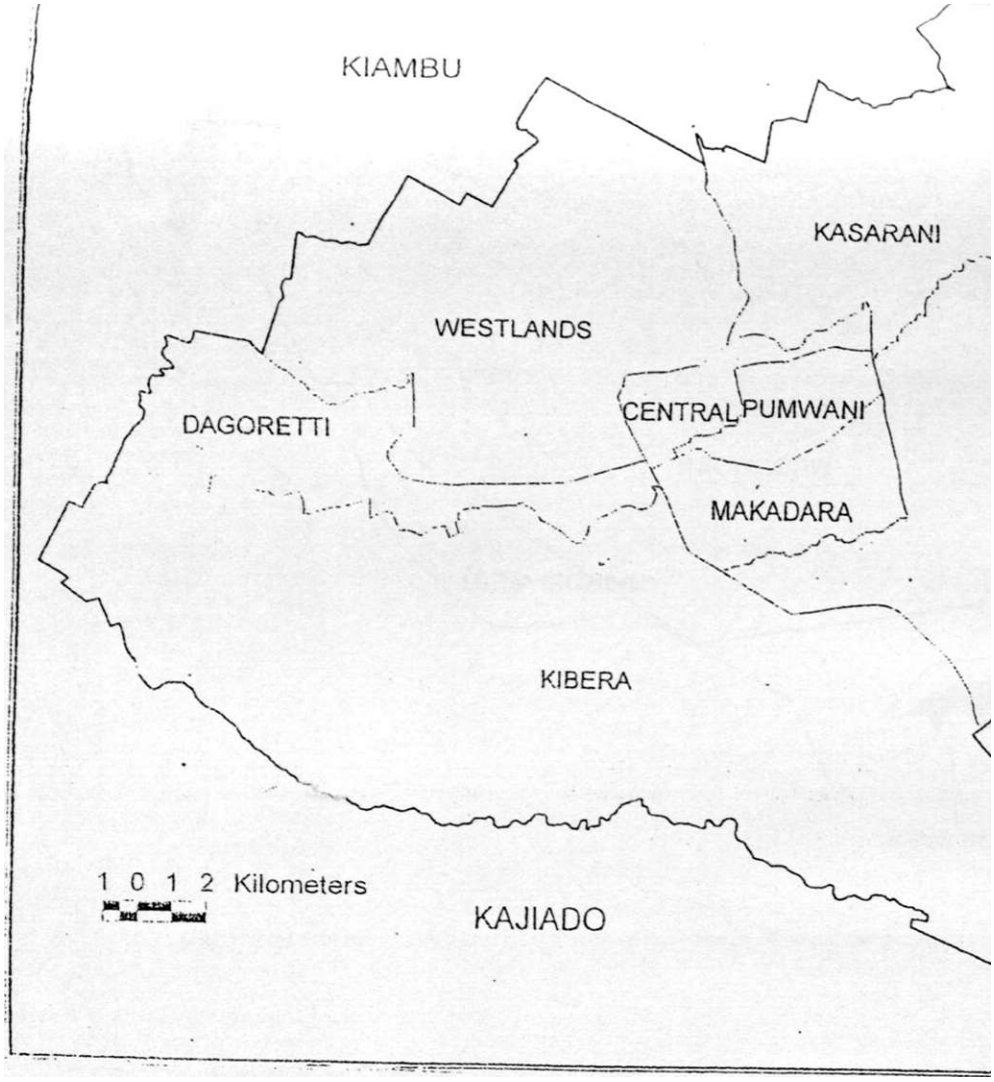
Whelan E.A., Piacitelli G.M., Gerwel B., Schnorr T.M., Mueller C.A., Gittleman J., Matte T.D. (1997). **Elevated blood lead levels in children of construction workers.** *Am J Public Health* 87:1352-1355.

World Health Organization, International Programme for Chemical Safety (1995). **Inorganic Lead: Environmental Health Criteria 165.**

World Resources (1996-1997). **The Urban Environment: A 1990 study in Bangkok.**

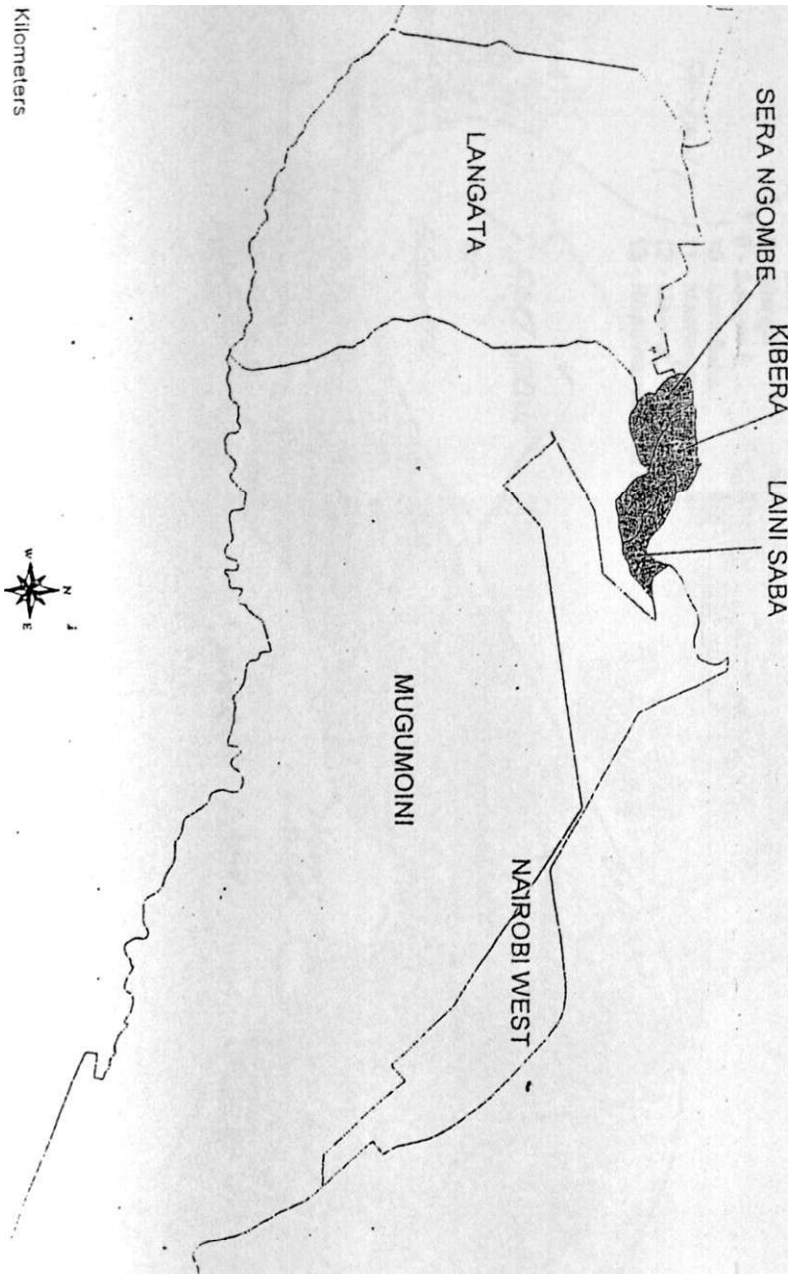
www.cehrc.org **(2006). Tools for detecting hazards**

^ww.emedicine.coni/EMERG/lopic293.htm (2007). Lead Toxicity

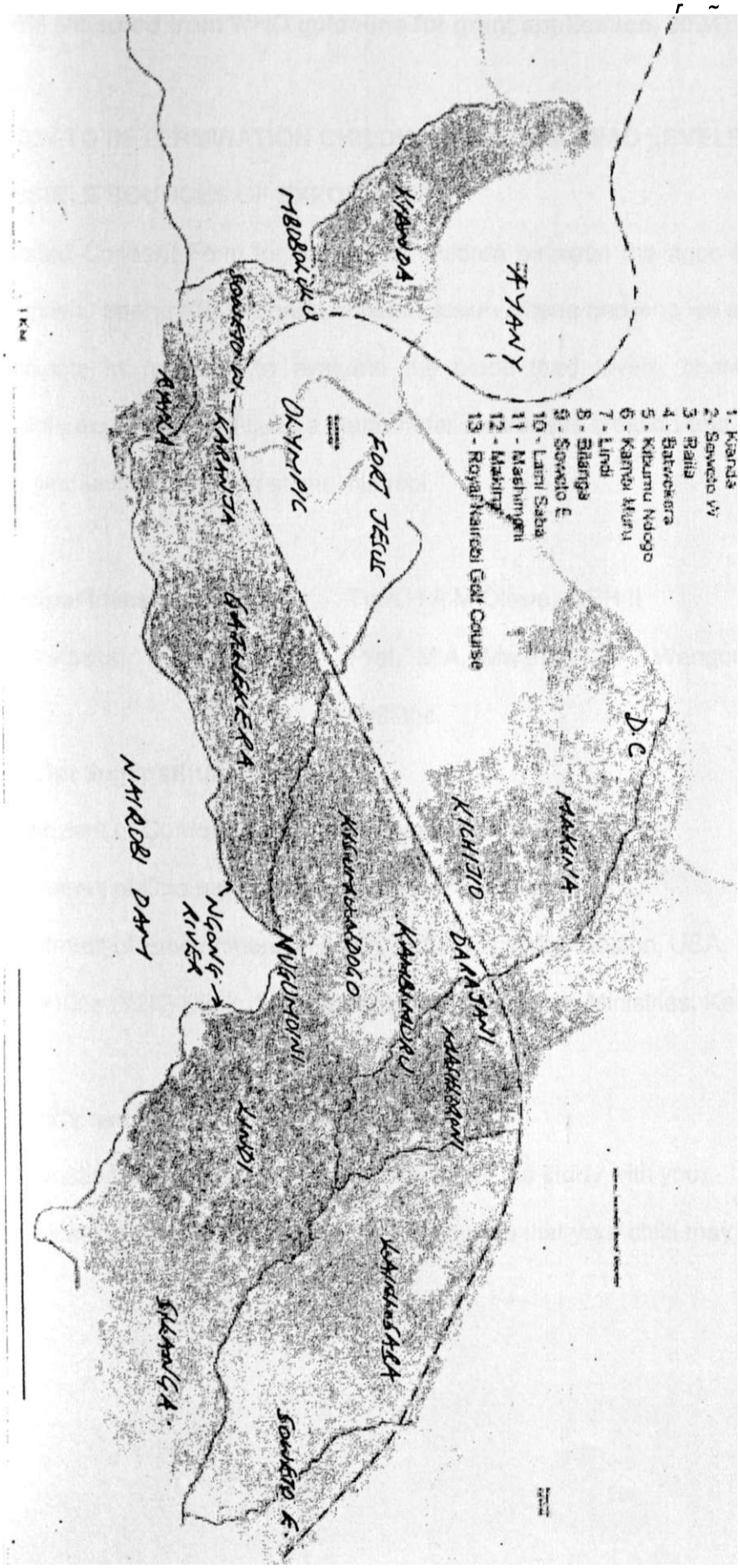

KIAMBU
KASARANI
WESTLANDS
DAGORETTI
CENTRAL PUMWANI
MAKADARA
KIBERA
1 0 1 2 Kilometers
KAJIADO

APPENDIX I: MAP OF NAIROBI DISTRICT INDICATING DIVISIONS

APPENDIX II: MAP OF KIBERA DIVISION INDICATING STUDY LOCATIONS



APPENDIX III: MAP OF KIBERA SLUMS INDICATING STUDY VILLAGES



APPENDIX IV: PARENTAL, GUARDIANS OR CARE GIVERS CONSENT FORM (Adapted from WHO guideline for grant application, 2004)

STUDY TO DETERMINATION CHILDHOOD BLOOD LEAD LEVELS AND POSSIBLE SOURCES OF EXPOSURES

Informed Consent Form for parents of children bet ' een the ages of 6 and 59 month who attend Y2K program, meet inclusion criteria and who we are asking to participate in research to evaluate the blood lead levels, characterize the possible exposures and build a platform for awareness creation and advocacy in the communities of Kibera slums, Nairobi.

Principal Investigator: Tom H.A.M Olewe, MPH II

Supervisors: Prof. M.A. Mwanthi; J.K. Wangombe and J. Griffiths.

Participating institutions:

Department of Community Health, University of Nairobi, Kenya.

Department of Chemistry, University of Nairobi, Kenya.

Department of Environmental Health, Tufts University, Boston, USA.

Yes to Kids (Y2K) Clinic, Medical & Sports Evangelism Ministries, Kenya.

This Informed Consent Form has two parts:

• Information Sheet (to share information about the study with you).

•Certificate of Consent (for signature if you agree that your child may participate).

You will be given a copy of the full Informed Consent Form

PART I: Information Sheet

Introduction

I am Dr Tom Olewe, a master student at the department of Community health, University of Nairobi. I am doing research on ghildhc od lead poisoning, which is not clearly understood in our country but might be very common.

I am going to give you information and invite you to have your child participate in this research. You do not have to decide today whether or not you agree that your child may participate in the research. Before you decide, you can talk to anyone you feel comfortable with. .

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask them of the study doctor, the staff or me.

Purpose

Lead poisoning is a serious health hazard with major socio-economic implications. Lead is a potent neurotoxin (nerve poison) particularly in children whose growing bodies are highly susceptible. Exposure to excessive levels of lead in air, water, soil and food is harmful to the health and intellectual development of children. All children have been exposed to some lead and the parents don't know it. The purpose of this study is to test the blood lead levels of children to help us understand the extent of the problem and intervene according to medical protocols where lead poisoning is found.

Type of Research Intervention

Referral to Kenyatta National Hospital for further treatment. The research will give multivitamins and free consultation services.

Participant selection

t

We are inviting you to take part in this research because it is important that we test the blood lead levels on children who are most at risk of long term adverse effects of lead poisoning if not intervention is undertaken. Because you and your child live in Kibera slums and your child might be at risk, we are asking if you would allow your child to participate.

Voluntary Participation

Your decision to have your child participate in this study is entirely voluntary. It is your choice whether to have your child participate or not. If you choose not to consent, all the services you and your child receive at this clinic will continue and nothing will change. You may also choose to change your mind later and stop participating, even if you agreed earlier, and the services you and/or your child receives at the clinic will continue.

Procedures and Protocol

A. Unfamiliar Procedures

Because we do not know if your child has been exposed to lead, we will ask you some questions to enable us determine whether your child might have been exposed.

B. Description of the Process

You will stay with your child during each of the visit • and during the procedures. In the first visit, after an interview with you and upon you giving your consent for your child to participate, a small amount of blood, equal to about a teaspoon will be taken from your child's arm. This will be tested for the presence of lead. Your child will feel some discomfort when the needle stick goes into her/his arm but this will go away very quickly. There may be slight bruising but this will disappear in a few days. We will give you and your child juice and something small to eat.

In the next visit, you will be given the results of your child's blood analysis and advised accordingly. We will ask your child's physician to give us the details of your child's health and illness related information. If you do not wish us to do that, please let us know. However, because your child's health records are very important for the study, if we cannot look at the health records, we will not be able to include your child in the study.

Your child will receive the treatment for his/her condition according to medical guidelines'on management of lead poisoning in children.

Duration

Include a statement about the time commitments of the research for the participant and for the parent including both the duration of the research and follow-up, if relevant.

The research takes place over one year in total. During that time, it will be necessary for you to come to the clinic/hospital/health facility three days, for one

hour each day. We would like to meet with you six months after your last visit for a final .check-up. Altogether, we will see you and yoit child 4 times over a year.

Side Effects

This study does not currently have known unwanted effects but might have some effects that we are not currently aware of. However, we will follow your child closely and keep track of these unwanted effects or any problems. We will give you a telephone number to call if you notice anything out of the ordinary, or if you have concerns or questions. You can also bring your child to this health facility at anytime and ask to see us. We may use some medicines to decrease the symptoms of the side effects or reactions. If this is necessary we will discuss it together with you and you will always be consulted before we move to the next step.

Risks

By participating in this study, your child will not be at greater risk than he/she would otherwise be. There is a possibility that local infection may happen as a result of taking the blood from your child. While the possibility of this happening is very low, you should still be aware of the possibility. If this happens and your child will receive free medical care of that condition at the clinic.

Discomforts

By participating in this research it is possible that your child may experience some discomfort such as the discomfort of the injections. There may be a slight hardening and/or swelling where the needle stick goes into the skin. This should disappear in one day. Your child may also be fussier than usual. These

behaviors usually stop within one day but ifjou a,e concerned, please call or come to Y2K program at VIPS clinic.

Benefits

If your child participates in this research, he/she will have the following benefits:

1. Any interim illnesses will be treated at no charge to you.

2. If your child falls sick during this, period he/she will be treated free of charge. There may not be any other benefit for your child but his/her participation is likely to help us find the answer to the research question. There may not be any benefit to the society at this stage of the research, but future generations are likely to benefit.

Incentives

You will not be provided any incentive to take part in this research. However, you will be reimbursed with Kshs 100 for your lost time and travel expense.

Confidentiality

The information that we collect from this research project will be kept confidential. Information about your child that will be collected from the research will be put away and no one but the researchers will be able to see it. Any information about your child will have a number on it instead of his/her name. Only the researchers will know what his/her number is and we will lock that information up with a lock and key. It will not be shared with or given to anyone except authorized team of researchers, sponsors and University of Nairobi, Department of Community Health.

Sharing of the results

The knowledge that we get from this study will be shared with you before it is made widely available to the public. Confidential information will not be shared. There will be small meetings in the community and these will be announced. Afterwards, we will publish the results in order that other interested people may learn from our research.

Right to Refuse or Withdraw

You do not have to agree to your child taking part in this research if you do not wish to do so and refusing to allow your child to participate will not affect your treatment or your child's treatment at this clinic in any way. You and your child will still have all the benefits that you would otherwise have at this clinic. You may stop your child from participating in the research at any time that you wish without either you or your child losing any of your rights as a patient here. Neither your treatment nor your child's treatment at this clinic will be affected in any way.

Alternatives to participating

If you do not wish your child to take part in the research, your child will be provided with continue accessing services available at the centre/institute/hospital.

Who to Contact

If you have any questions you may ask them now or later, even after the study has started. If you wish to ask questions later, you may contact the research team at the Y2K program. This proposal has been reviewed and approved by Kenyatta National Hospital - College of Health Sciences, University of Nairobi,

Ethical and Research Committee, which is a committee whose task it is to make sure that research participants are protected from harm. If you wish to find about more about the Ethical and Research committee, please contact the Director, Kenyatta National Hospital and Principal, College of Health Sciences, University of Nairobi.

PART II: Certificate of Consent

Certificate of Consent

I have been invited to have my child participate in research of lead poisoning. I understand that it will involve my child giving some blood sample and three follow-up visits. I have been informed that the risks are minimal. I am aware that there may be no benefit to either my child or myself personally and that I will not be compensated beyond travel expenses. I have been provided with the name of a researcher who can be easily reached at Y2K program.

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily for my child to participate as a participant in this study and understand that I have the right to withdraw my child from the study at any time without in any way affecting either my child's or my own medical care.

Print Name of Participant

Print Name of Parent or Guardian

Signature of Parent or Guardian _

Date_____Day/month/year

If illiterate, a literate witness must sign (if possible, this person should be selected by the participant and should have no connection to the research team). Participants who are illiterate should include their thimbprint as well.

I have witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Print name of witness_

AND

Thumb print of parent

Signature of witness

Date_____Day/month/year

I have accurately read or witnessed the accurate reading of the consent form to the parent or guardian of the potential participant and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Print Name of Researcher_

Signature of Researcher_

Date_____Day/month/year

A copy of this Informed Consent Form has been provided to the parent or guardian of the participant_____(initialed by researcher/assistant)

APPENDIX V - KIAMBATISHO V - FOMU YA MZAZI/MWANGALIZI KUIDHINISHA MTOTO KUCHUNGUZWA

UTAFITI WA KUTAMBUA KIWANGO CHA RISASI (LEAD) KWENYE DAMU YA MTOTO NA VYANZO VINAVYOWEZA KUHATARISHA WATOTO

Fomu ya kukubali (mtu akielewa) kwa wazazi wa watoto wenye kati ya miaka 6 na miezi 59 ambao huhudhuria Kliniki ya Y2K, na wanaofikia vigezo vya kuhusishwa, na wanaoomba kuhusika katika utafiti wa kutathmini kiwango cha risasi kwenye damu, unaainisha (kuonyesha) hatari ambazo zinaweza kuleta maambukizo. Utafiti huu pia utatuwezesha kuwa na maazimio kuweza kuleta kutambua na utetezi katika jamii za mitaa ya mitaa ovyo ya Kibera, Nairobi.

Mchunguzi Mkuu: Tom HAM Olewe, MPH II

Wasimamizi: Maprofesa Mwanthi, Wangombe na Griffiths

Mashirika (Taasisi) vanavohusika:

1. Idara ya Afya ya Jamii, Chuo Kikuu cha Nairobi, Kenya
2. Idara ya Kemia, Chuo Kikuu cha Nairobi, Kenya
3. Idara ya Afya ya Kimazingira,.Chuo Kikuu cha Tufts, Boston, USA
4. Kliniki ya "Yes to Kids", Huduma za Uingilist m Kitibabu na Kimichezo, Kenya

Fomu ya kukubali (mtu akielewa) inazo sehemu mbili:

- Karatasi yenye Taarifa (inakueleza habari kunusu utafiti huu)
- Hati ya kukubali (kwa ajili ya sahihi ikiwa utakubali mwanao ahusishwe)

Utapewa nakili ya Fomu ya kukubali yote.

SEHEMU I: Karatasi yenye Taarifa

Utanqulizi:

Jina langu ni Dkt. Tom Olewe, mwanafunzi wa shahada ya pili katika idara ya afya ya jamii katika Chuo Kikuu Cha Nairobi. Ninafanya utafiti kuhusu ugonjwa unaosababishwa na sumu ya risasi kwa watoto, ambao haujaeleweka vizuri Katika taifa hili ingawa umeenea sana.

Ninaenda kukupa maelezo kisha nikualike kukubali mtoto wako ahusishwe katika utafiti huu. Si lazima ufanye uamuzi leo kumhusu mwanao. Unaweza kuongea na yeyote kati yetu utakayejisikia huru naye kabla ya kufanya uamuzi huu.

Yawezekana patakuwa na maneno ambayo hutayaelewa. Naomba unisimamishe ikiwa wataka nieleze maneno fulani. Ukiwa na maswali baadayo, waweza ukamuuliza daktari anayekuuliza maswali, wafanya kazi wengine au uniulize mimi mwenyewe.

Kusudi:

- ugonjwa unaosababishwa na sumu ya risasi ni hatari kuu kwa afya, ambayo inaweza kusababisha madhara ya kijamii na kiuchumi. Risasi ni sumu ya neva (potent neurotoxin/nerve poison) hasa kwa watoto kwa sababu miili yao inaendelea kukua, inawaathiri kwa urahisi.

Ni hatari kwa mtoto anayekutana na risasi kwa wingi kupitia hewa, maji, udongo na chakula. Hii inaweza kudhuru afya na kumfanya mtoto asikue vizuri kimafikira. Watoto wote hukabiliwa na risasi na wazazi hawana habari. Kusudi la utafiti huu ni kuchunguza idadi ya risasi katika damu ya watoto ili tuweze keulewa kiasi cha shinda hii kisha tuingilie kati, koko huu ugonjwa upatikanapo, kulingana na kanuni za matibabu.

Jinsi Utafiti Utakavvo inqilia Kati:

Wagonjwa watapelekwa Hospitali ya Taifa ya Kenyatta kwa matibabu zaidi. Tutapeana vidonge vyenye vitamini nyingi na tutatoa huduma za ushauri bila malipo.

Jinsi Wahusika watakavyo chaquliwa:

Tunakwalika uhusike katika utafiti huu kwa sababu ni muhimu tuchunguze kiw'ango cha risasi kwa watoto walio hatarini ku'u kutokana na madhara ya muda mrefu kutokana na ugonjwa unaosababishwa na sumu ya risasi, hatua za dharura sizipochukuliwa. Kwa sababu wewe na mwanao mnaishi katika mtaa wa ovyo wa Kibera, na mwanao aweza akawa hataririi, tuanakuomba umruhusu huyo mwanao kuhusika.

Kuhusika Kwa Kujitolea:

Uamuzi wako kumhusisha mwanao ni kwa hiari yako pekee. Hakuna kulasimishwa. Wewe ndiye utakayeamua kama mwanao atahusishwa au la. Ukiamua kutoshiriki, huduma zote ambazo Kliniki hii hutoa kwako na mwanao zitaendelea, wala hakuna jambo litakalo badilika. Utaendelea kupokea huduma

zetu hata kama baadaye utaamua kuacha kuhusika, licha ya kwamba ulikubali hapo mwanzoni.

Taratibu na protokali (itifaki)

A. **Taratibu Zisizoiulikana Sana:**

Kwa sababu huna habari kama mwanao amo hatarini kutokana na risasi, tutakuuliza maswali kutuwezesha kuamua kama mwanao yumo hatarini.

B. **Maelezo Kuhusu Niia Itakayotumiwa:**

Utakaa na mwanao unapo tembelea Kliniki yetu, hata uchunguzi unapoendelea. Utakapokuja mara ya kwanza, baada ya mahojiano yetu na wewe, na ukikubali kuwa mwanao atahusika, kiasi kidogo cha damu kitatolewa kutoka kwa mkono wa mwanao (damu inaweza kujaa kijiko kidogo kinachotumiwa kumpa mtoto dawa). Damu hii itapimwa kuthibitisha kama risasi ipo. Mwanao atasikia uchungu mdogo sidano itakapoingizwa kwenye mkono, lakini uchungu hautaendelea kwa muda mrefu. Kuna uwezekano utapata alama (bruise) ndogo, ambayo itatoweka baada ya siku chache sana. Tutakupa wewe na mwanao juisi na kitu kidogo cha kula.

Katika ziara yako itakayofuata, utapewa matokeo ya uchambuzi wa damu ya mwanao, kisha utapewa ushauri kulingana na matokeo hayo. Tutamuuliza daktari anayemtibu mwanao atupe taarifa kuhusu afya ya mwanao, ugonjwa wake, pamoja na habari yoyote kumhusu mwanao.

Tafadhali tujulishe ikiwa hungependa kute/a hivyo. Ingawaje, kwa sababu rekodi za afya ya mwanao ni muhimu sana katika utafiti huu, ikiwa hutaweza kuangalia rekodi hizi, hatutaweza kumweka mwanao katika huu utafiti.

Mwanao atapokea matibabu ya hali alionayo kulingana na taratibu za kitaifa kuhusu matibabu ya ugonjwa unaosababishwa na sumu ya risasi katika watoto.

Muda Wa Utafiti:

Tia maelezo kuhusu wataki ambao mhusika na mgonjwa watapeana kwa ajili ya utafiti - wakati wa utafiti mwenyewe na wakati wa ufuatiliaji- ikiwa inahitajika. Utafiti huu huchukua mwaka mmoja kwa jumla. Katika muda huu, utahitajika kufika katika Kliniki/Hospitali/Kituo Cha Afya siku tatu, saa moja kila siku. Tungependa kututana nawe miezi sita baada ya ziara yako ya mwisho kwa ajili ya kupimwa mara ya mwisho. Kwa jumla, basi, tutakuona wewe na mwanao mara nne kwa kipindi cha mwar.a huu mmoja.

Athari:

Utafiti huu hauna mathara yoyote yanoyojulikana. Ingawaje, upo uwezekano wa athari ambazo hazijajulikana kwa wakati huu. Hata hivyo, tutafuatilia hali ya mtoto wako kwa karibu kuona kama mathara au shida zozote zitakuepo. Tutakupa nambari ya simu ambayo utaweza kupiga ukiwa na jambo lolote la kawaida au ukiwa na shauku au maswali yeyote. Pia unaweza kumleta mwanao kwenye kituo cha afya wakati wowote na kuuliza kutuona. Kuna uwezekano tutatumia madawa kupunguza dalili za athari zozote. Kama hili

itahitajika, tutashauriana nawe na wakati wote utashauriwa kabla hatujachukua hatua jingine.

Hatari/Mashaka:

Kwa kuhusika katika utafiti huu, mtoto wako hatakuwa katika hatari kuliko kama hangehusika. Kunao uwezekano kuwa mwanao aweza akapata maambukizo kwenye sehemu mtoto alipotolewa damu. Hata kama uwezekano huu ni mdogo mno, yakupasa uelewe juu ya uwezekano huu. Ikiwa hayo yatatokea, mtoto wako atapokea matibabu ya bure kukabilia maambukizo hayo katika Kliniki hii.

Usumbufu (Kero):

Kwa kuhusika katika huu utafiti, upo uwezekano mwanao atapata usumbufu kama ule unaoletwa na sidano. Panaweza pakawa na ugumu kiasi na/au kuvimba pahali ambapo sidano illingia ndani ya ngozi ya mtoto. Hali hii huondoka baada ya siku moja. Mwanao aweza akawa msumbufu zaidi ya kawaida. Tabia hizi hukoma baada ya siku moja, lakini ukiwa na shauku, tafadhali tupigie simu au uje katika Kliniki ya Y2K.

Manufaa (Faida):

Mwanao akishiriki katika utafiti huu atapata manufaa yafuatayo:

1. Ugonjwa wowote katika muda huu wa utafiti utatibiwa bila malipo yoyote.
2. Inawezekana mwanao asipate manufaa mengine lakini kuhusika kwake kwaweza kutusaidia kupata jawabu la swali ambalo utafiti unataka kujibu.
3. Yawezekana pasiwe na manufaa kwa jamii katika hatua hii ya utafiti, lakini vizazi vijavyo vyaweza kufaidika.

Marupurupu (Kichocheo):

Hautapewa kichocheo chochote kuweza kushiriki katika utafiti huu. Ingawa hivyo, utarudishiwa Ksh. 100 kusimamia muda wako na matumizi ya usafiri.

Siri (Confidentiality):

Habari zote tutakazokusanya kutoka kwa utafiti huu zitawekwa kama siri. Habari kuhusu mwanao ambazo utafiti huu utakusanya zitafichwa na hakuna atakayeweza kuziona isipokuwa watafiti pekee. Habari zozote kumhusu mwanao zitatambuliwa kwa nambari ya siri wala sio kwa jina lake. Ni watafiti tu ambao wataijua nambari ya mwanao na watafungia habari hii kwa kutumia kufuli na ufunguo. Habari hii haitatolewa kwa mtu yeyote isipokuwa ile timu ya watafiti wenye mamlaka, wadhamini, na Chuo Kikuu cha Nairobi, na idara ya Afya ya Jamii.

Kutoa Matokeo ya Utafiti:

Maarifa tutakayoyapata kutokana na utafiti huu yatatolewa kwako kabla ya kuwekwa wazi kwa umma. Habari zozote za siri hazitatolewa kwa umma. Kutakuwa na mikutano midogo midogo katika jamii. Mikutano hii itatangazwa. Baadaye, matokeo haya yatachapishwa ili watu wote wanaopenda wajifunze na utafiti huu.

Haki ya Kukataa Au Kujiondoa:

Haulazimishwi kukubali mwanao kuhusika kwenye utafiti huu, ikiwa hutaki kufanya hivyo. Pia kutomhusisha mwanao takutaathiri matibabu yako au ya mwanao katika Kliniki hii kwa jia yoyote ile. Wewe na mwanao mtapata manufaa yote ambayo mngepata kutoka Kliniki hiki. kama kawaida. Unao uhuru

kumsimamisha mwanao asiendelee kuhusika kwenye huu utafiti wakati wowote unapotaka, bila wewe au mwanao kupoteza haki zozote kama mgonjwa wetu. Matibabu kwako au kwa mwanao katika Kliniki hii hayataadhiriwa kwa njia yoyote ile.

Njia Tofauti za Kuhusika:

Ikiwa hutaki mwanao ahusike katika utafiti huu, mwanao ataendelea kupokea huduma zinazotolewa katika Kituo/Taasisi/Hospitali

Utawasiliana na Nani?

Ukiwa na maswali yoyote, unaweza kuuliza sasa au baadaye, hata kama utafiti umeanza. Ukipenda kuuliza maswali baadaye, waweza kuwasiliana na kundi la watafiti katika Kliniki ya Y2K.

Mapendekezo haya (mradi huu) yakemakaguliwa rasmi na kupitishwa na Hospitali ya Kitaifa ya Kenyatta - Chuo cha Sayanzi ya Kiafya, Chuo kikuu cha Nairobi, Kamati ya Maadili na Utafiti (ambayo jukumu lake ni kuhakikisha kwamba wahusika wa utafiti wamelindwa kutokana na mathara). Ukitaka kupata habari zaidi kuhusu kamati ya Maadili na Utafiti, tafadhali wasiliana na mkurugenzi wa Hospitali kuu ya Kenyatta na Mkuu wa kituo cha sayansi ya Afya, Chuo kikuu cha Nairobi.

SEHEMU II: Hati ya Kukubali

Hati Ya Kukubali

Nimealikwa kumhusisha mwanangu katika utafiti kuhusu ugonjwa unaosababishwa na sumu ya risasi. Ninaelewa ya kwamba utafiti huu utahusisha mwanangu kutolewa damu na ziara tatu za kufuatilia. Nina habari kwamba yawezekana mimi na mwanangu tusifaidike kibinafsi kwa njia yoyote, na sitapata marupurupu yoyote pasipokuwa kurusishiwa matumizi ya usafiri. Nimepewa jina la mtafiti ambaye anaweza kufikiwa kwa urahisi katika kliniki ya Y2K.

Nimesoma maelezo yaliyotangulia au nimesomewa. Nimekuwa na fursa kuuliza maswali juu yake na maswali yote nilioiuliza nimejibiwa mpaka nikaridhika. Nakubali kwa hiari yangu mwenyewe mwanangu ahusike katika utafiti huu na naelewa kwamba ninayo haki kumuondoa mwanangu kutoka kwa utafiti huu wakati wowote, na hii haitaathiri matibabu kwangu au kwa mwanangu.

Andika Jina la mhusika

Andika Jina la mzazi au Mwangalizi

Sahihi ya mzazi au mwangalizi

Tarehe_____Siku/Mwezi/Mwaka

Kama mhusika hajui kusoma na kuandika, shahidi anayejua kusoma na kuandika lazima atiye sahihi (ikiwezekana, mtu huyu awe amechaguliwa na mhusika na asiwe na uhusiano wowote na kundi la utafiti). Wahusika wasiojua kusoma na kuandika wapaswa kuweka alama ya kidole gumba pia.

Nimeshuhudia usomaji sahihi wa fomu ya kukubali kwake atakaye husika, naye alipewa nafasi kuuliza maswali. Nahakikisha kuwa mtu huyu ametoa idhini kwa hiari yake mwenyewe.

Andika jina la Shahidi

NA

Alama ya kidole gumba ya mzazi

Sahihi ya Shahidi

Tarehe_____Siku/Mwezi/Mwaka

Nimesoma kwa usahihi au nimeshuhudia usomaji sahihi wa fomu ya kukubali kwake mzazi au mwangalizi wa atakaye husika, naye alipewa nafasi ya kuuliza maswali. Nahakikisha mtu huyu amekubali kwa hiari yake mwenyewe.

Andika jina la mtafiti

Sahihi'ya mtafiti

Tarehe_____Siku/Mwezi/Mwaka

Nakila ya fomu ya kukubali yenye maelezo kamili imetolewa kwa mzazi au mwangalizi wa mhusika (imeanzishwa na mtafiti/naibu).

APPENDIX VI: CHILDHOOD LEAD EXPOSURE RISK FACTOR QUESTIONNAIRE [MODIFICATION OF CDC RISK ASSESSMENT QUESTIONNAIRE - 2001]

In some areas of Nairobi, up to 80% of children less than 5 years of age have blood lead levels above what is recommended for good health (CDC, 1994). If your child has too much lead in their blood something **CAN** be done about it. If you know your child has too much lead, you can find out where lead is and how to stop it getting into your child. This will help lessen ihe potential harmful effects of lead, such as learning and behavior problems or lowered IQ. Most children with too much lead in their blood are never tested. These children often have problems at school and families will never know why.

We will take a moment to ask some questions to help us see if your child could be having this problem.

Participant's Code:..... Date:

1. Child's Age: (months) [1] 6 - 9 [2] 10 - 19 [3] 20 - 29 [4] 30 - 39 [5] 40 - 49 [6] 50 - 59
2. Child's Sex [1] M [2] F
3. Breast-feeding history: [1] Current [2] <1yr [3] >1 yi [4] Never
4. Respondent's Sex: [1] M [2] F
5. Respondent's Education Level: [1] Primary [2] Secondary [3] Tertiary [4] Others
6. Type of House Walls: [1] Stone [2] Mud [3] Iron sheet [4] wood [5] Others
7. Parents/Guardians Occupation:
8. Source of drinking Water: [1] Tap [2] Borehole [3] Others
9. Source of Vegetables: [1] Kitchen Garden [2] Market [3] Others
10. Are you aware of Childhood Lead poisoning? [1] Yes [2] No
11. If Yes in question 10, list possible environmental sources of childhood lead poisoning? [1] <2 sources [2] 2 - 4 sources [3] > 5 sources '

For Questions below, answer Yes [1], No [2] Don't Know [3]

1) Live in or regularly visit a house with peeling or chipping paint? Including a day care center, preschool, the home of a babysitter or a relative, etc. [ ]

2) Does the child play outside in dirt that could be co.v aminated with lead from a nearby road, bridge, river, railway line, or a building with peeling paint on the outside? [ ]

3) Have a child or your child's playmate being followed or treated for lead poisoning? [ ]

4) Live with an adult whose job or hobby involves exposure to lead? Such as painting, remodeling, auto radiators, batteries, auto repair, soldering, bullets, stained glass, pottery, going to shooting ranges, hunting or fishing? [ ]

5) Live near an active smelter, battery recycling plant or other industry likely to release lead? [ ]

6) Does your child sometimes eat non-food items such as soil or paint? [ ]

7) Does your child have anemia (low blood), behavior Droblem, and learning problem or did they sit up, walk or talk late? [ ]

8) Do you have pottery or ceramics made in other countries or lead crystal or pewter that are used for cooking, storing or serving fend or drink? [ ]

9) Has your child ever used any traditional, imported or home remedies or cosmetics containing lead e.g Kohl? [ ]

10) Do you have concerns about your child's mental development? [ ]

A positive answer to any of these questions indicates a potentially high risk of lead exposure.

Thank you for participating in our study!

APPENDIX VII - KIAMBATISHO VII - KIPENGELE CHA ADHARI ZA KUHATARISHWA NA RISASI UTOTONI (Marekebisho ya orodha ya maswali kuhusu upimaji wa athari wa CDC - 2001)

Katika sehemu fulani za Nairobi, hadi 80% ya watoto chini ya miaka mitano wana kiwango cha risasi kwenye damu zaidi ya kiwango kinachopendekezwa kwa afya bora (CDC, 1994). Ikiwa mwanao ana kiwango cha juu zaidi cha risasi kwenye damu, kuna jambo LINALOWEZA kuafanywa juu yake. Ikiwa unajua mwanao ana kiwango cha juu sana cha risasi, waweza kugundua ilipo risasi na jinsi ya kuizuia kumuingia mwanao. Hii itasaidia kupunguza mathara mabaya ya risasi, kama vile matatizo ya kusoma na tabia au IQ iliyo chini. Watoto wengi walio na kiwango cha risasi cha juu katika damu hawapati nafasi ya kupimwa. Watoto hawa mara nyingi huwa na matatizo shuleni na fimilia zao hazitawahi kujua ni kwa nini.

Tutachukua muda mfupi kuuliza maswali yatakayotusaidia kujua kana kwamba mwanao anaweza kuwa na tatizo hili.

Alama ya Siri ya Mteja (Code)_____.Tame

1. Umri wa mtoto (miezi) [1] 6 - 9 [2] 10 - 19 [3] 20 - 29 [4] 30 - 39 [5] 40 - 49 [6] 50 - 59
2. Jinsia ya mtoto [1] Mwanamme[2] Mwanamke
3. Historia ya unyonyeshaji: [1] Wakati huu [2] <1mwaka [3] >1 miaka [4] Hajawahi

4. Jinsia ya anayejibu: [1] Mwanamme [2] Mwanamke

5. Kiwango cha Elimu ya anayejibu: [1] Msingi [2] Sekondari [3] Masomo ya juu [4] Zingine

6. Aina ya kuta za nyumba: [1] Mawe [2] Matope [3] Mabati [4] Mbao [5] Zingine

7. Kazi ya mzazi/mwangalizi:

8. Chanzo cha maji ya kunywa: [1] Mfereji [2] Kisima [3] Zingine

9. Chanzo cha mboga: [1] Shamba dogo la jikoni [2] Sokoni [3] Zingine

10. Je, una habari kuhusu ugonjwa unaosababishwa na sumu ya risasi kwa watoto? [1] Ndio [2] La

11. Kama umejibu ndio katika 10, taja vyanzo vya ugonjwa unaosababishwa na sumu ya risasi kwa watoto? [1] vyanzo <2 [2] vyanzo 2 -4 [3] vyanzo > 5

Kwa maswali yafuatayo, jibu Ndio (1), La(2), au Sijui (3)

1. Je unaishi au kutembelea mara kwa mara nyumba yenye rangi inayoambuka au inayomeguka? (Hata iwe mahali pa kutunza watoto wadogo mchana, kituo cha chekechea, au nyumba ya jamaa etc) [ ]
2. Je, mtoto huyu hucheza nje kwenye udongo uliochafuliwa na risasi kutoka kwa barabara iliyo karibu, daraja, mto.njia ya reli, au nyumba iliyo na rangi inayobambuka upande wa nje? [ ]
3. Je, mwanao au mtoto anayecheza na mwanao amekuwa akiangaliwa au kutibiwa kutokana na ugonjwa unaosababishwa na sumu ya risasi? [ ]

4. Je, mtoto anaishi na mtu mzima ambaye kazi yake au jambo la kupitishia muda inahusisha hatari ya sumu ya risasi kama vile kupaka rangi, kutengeneza magari, kuchomelea chuma, risasi, kutia vioo rangi, ufinyanzi, ulengaji shabaha na uvuaji samaki ? [ ]
5. Je unaishi karibu na pahali panapoyeyushiwa madini, mtambo wa kutengeneza betri upya, au viwanda vinginevyo vinavyoweza kutoa risasi ? [ ]
6. Je mwanao wakati mwingine hula vitu kama vile mchanga au rangi ? [ ]
7. Je, mwanao hua na upungufu wa damu, matatizo ya tabia au masomoni na je, alijifunza kutembea, au kuongea akiwa amechelewa ? [ ]
8. Je, unavyo vyombo vya udongo au kauri vilivyotengenezewa nchi za kigeni au chembechembe cha risasi au vyombo vya pyuta vinavyotumika kupikia, kuhifadhi, au kugawia chakula au kinywaji ? [ ]
9. Je, mwanao ashawahi kutumia dawa zozote za kienyeji, au dawa za kinyumbani, au vipodozi vinavyo risasi, kama vile kohl ? [ ]
10. Je, unao shauku kuhusu kukua kwa kimaarifa ya mwanao ? [ ]

Jibu lolote linalokubali swali lolote kati ya yalioulizwa hapa liriaonyesha uwezekano mkubwa wa hadhara ya juu ya hatari ya risasi.

Asante kwa kuchukuwa muda kujibu maswali.