# Blood and Urine Lead Levels in Adults Attending Harare Polyclinics

**By**

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Dissertation Submitted in Partial Fulfillment of

Masters of Clinical Pharmacology Degree

University of Zimbabwe

November 2013

## Declaration

I, Prudence Manyuchi, certify that this dissertation is my original work and has been prepared in accordance with guidelines of the Master of Clinical Pharmacology Program, University of Zimbabwe. I further attest that this work has not been submitted, in part or in full, for any other degree at any university and/or any publication.

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I, have supervised and read this dissertation, I am satisfied that this is the original work of the author in whose name it is being presented. I confirm that the work has been completed satisfactorily for presentation in the examination.

Name of Supervisor_____

Signature_____Date_____

Chairman of the Department of Clinical Pharmacology, CHS University of Zimbabwe

Signature_____Date_____

## Dedication

To my father Simon Usiku Manyuchi, who has taught me life is worth living no matter what circumstance you are in.

To my husband Albert Kulupi, thank you for loving me and having faith in me.

To my children, Maxine and Atinatsa, you are loved dearly.

To my mother, you are my pillar of strength and close friend.

To You God, the love of my life.

## Acknowledgements

I want to acknowledge with thanks, my supervisors, Prof CFB Nhachi and Dr Tagwireyi, who tirelessly offered their guidance and wisdom during the course of the year.

Mrs Madyambudzi, Mrs Mabhugu and Sr Chifamba for their moral support.

Mr Julius and Mr Murambiwa for their laboratory expertise and the rest of the clinical pharmacology.

MSc Clinical Pharmacology Class of 2013

The SAZ staff.

## List of Abbreviations

| | |
|---|---|
| **BLL** | Blood Lead Levels |
| **CDC** | Centre for Disease Control |
| **EPA** | Environmental Protection Agency |
| **HC** | Head Circumference |
| **ICP-AES** | Inductively Coupled Plasma – Atomic Emission Spectroscopy |
| **IQ** | Intelligence Quotient |
| **JREC** | Joint Parirenyatwa Hospital & College of Health Sciences Research Ethic Committee. |
| **MRCZ** | Medical Research Council Of Zimbabwe |
| **UZ-CHS** | University of Zimbabwe College of Health Sciences |
| **WHO** | World Health Organisation |

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## Abstract

Lead is a toxic environmental pollutant and exposure to it can produce serious adverse health effects in adults and children. There is no safe exposure to lead, however the CDC recommends adult blood lead level (BLL) of < 20µg/dl to be safe and <5 µg/dl for children. However evidence suggests subclinical toxicity at lower levels. In Harare, people are exposed to lead from exhaust fumes from leaded petrol, old leaching lead plumbing and flaking leaded paint.

The main objective of the study was to evaluate the level of lead exposure of ordinary residents in Harare. The secondary objective was to determine if lead levels were affected by serum protein levels.

Three urban areas were chosen for their potential of increased risk of environmental exposure. One rural area was chosen to act as a control. Water lead levels were also measured from the different study areas. The lead levels were measured using the inductively coupled plasma-atomic emission spectroscopy (ICP-AES). Protein levels were determined using the Lowry method.

The mean blood lead levels that were obtained for the urban areas were 0.80±0.69µg/dl and 0.96±0.98µg/dl for the rural clinic. The urine lead levels for the urban areas were 0.57±0.67 µg/dl and 0.51±0.13µg/dl for the rural clinic. The water lead levels from the different study populations were 1.1µg/dl for Mbare, 1.6µg/dl for Highfield, 0µg/dl for Goromonzi and 0.4µg/dl, for Mabvuku/Tafara respectively.

The mean serum protein levels were 24.45±3.76g/l for the study population. The Pearson correlation coefficient for the serum protein levels and blood lead levels was 0.225 at a p-value of 0.124.

The ordinary residents in Harare urban have minimal environmental exposure to lead. There was no significant correlation between serum protein levels and blood lead levels.

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# CHAPTER 1

## 1.0 General Introduction

Lead is abundant in the earth's crust. It is a toxic, heavy metal with no physiological benefit to man[1]. Old civilizations used this metal because of its easy accessibility, abundance and malleability[2]. Lead's toxic effects have been known for centuries. The symptoms range from the dramatic lead colic and encephalopathy usually characterized by confusion in acute, severe exposure[2,4]. To asymptomatic or non specific and may be mistaken for other disease processes[3,4] in chronic subclinical exposure .One of the worst symptom is mental retardation and reduction of IQ in children. There is also peripheral neuropathy, cardiovascular disease, recurrent miscarriages, and reduced sperm count and motility amongst other symptoms in adults.[5, 6, 7]

Lead exposure can be occupational or non occupational[7,8]. Countries like the United Kingdom, United States of America have put in measures into place to protect workers from exposure to lead through their trade unions[3,9]. These include protective gear, regular screening of blood and urine lead levels and chelation of lead in workers with the elevated lead levels [3,9,10]. Non occupational exposure to lead include inhalation of exhaust fumes from cars using leaded petrol, emissions from industrial plants which use lead e.g. battery manufacture and recycling, or from ingesting food/water contaminated with lead. [8,9,10] The contamination of water occurs when using leaded water pipes and/or solder joints. The lead leaches out in water with an acidic pH [10]. Another major source of non occupational exposure to lead is living in a lead based paint[1,4,7]. Exposure includes inhaling the dust and ingesting the flaking paint[1]. Harare residents are not spared from any of the above, since leaded petrol is still available on the Zimbabwean market. Most of the houses in Harare's high residential areas were built before the banning of leaded paints internationally. So most of the houses still contain lead based paint which may be acting

as a continuous source of lead exposure. The plumbing system used in this country was also put in place before the international banning of lead, so lead might still be leaking into household water. There are two battery manufacturing and recycling plants in Harare and may act as sources of airborne lead.

Nutritional state of an individual affects the absorption of lead from its entry points and its metabolism into lead storage into tissues[11]. Absorption from the gastrointestinal is increased if the lead is ingested in a fasting period than on a full stomach[4,12]. However there is not much evidence on how protein levels are related to blood lead levels except for a study done by Swaran [12], showed that low protein levels were associated with high toxicity of lead.

The reduction of IQ due lead exposure in childhood has serious economic and health implications[2,4]. Most countries have appreciated this and as result have put in legislation that reduces the risk of exposure to lead for ordinary citizens[2,7]. The legislation include, banning of leaded petrol, banning lead based paint and changing the plumbing system. Public awareness on the toxicity of lead was also put in place [13]. The reduction of lead from the environment is an ongoing process. Follow up studies done in countries which adopted these policies showed a significant decline of blood lead levels[2, 4 , 13].

Lead poisoning is a permanent health hazard which is preventable[4]. Doing a study on evaluation of blood lead levels in adults in Harare would give a picture of how much lead are the people exposed to.

## 1.1 LITERATURE REVIEW

Lead is a heavy metal, malleable and can easily be combined with other metals to form alloys .It has a low melting point of 327.46$^{o}$C. Its use today is widespread in products as diverse as pipes, storage batteries, pigments and paints, glazes, vinyl products, shots and ammunition, cable covers and radiation shielding [2,4,] . Lead ores constitutes 0.002% of the earth's crust and exists mainly as sulphides. Inorganic forms of lead are the form of lead that is found in old paint, soil, dust and various consumer products[14]. The colour varies depending on the chemical form, lead white (lead carbonate), yellow lead (lead chromate, lead monoxide) or red lead (lead tetraoxide). The organic form of lead is tetra-ethyl lead[15,16]. This is the one mainly used in leaded petrol to enhance the engine's performance. Tetra-ethyl lead is the most dangerous as it can be absorbed through the skin and is highly toxic to the brain and the central nervous system[15,16,17,].

## 1.2 Sources of Lead Exposure

Exposure to lead usually is classified into two main categories; occupational exposure and environmental exposure[1,2]. The most common sources and products that account for environmental exposure to lead are:

a) Leaded petrol[1,16,17]

b) Lead from an active industry, such as mining (especially in soils) lead-based paints and pigments[2,3,]

c) Lead solder in food cans[5,6]

d) Ceramic glazes and cooking earthenware.[7,8]

e) Drinking-water systems with lead solder and lead pipes[10,12,13]

f) Lead in products, such as herbal and traditional medicines, folk remedies, cosmetics[18] and toys

g) Lead released by incineration of lead-containing waste lead in electronic waste (e-waste)[16,]

i) Lead in the food chain, via contaminated soil[19,20]

j) Lead contamination as a legacy of historical contamination from former industrial sites[21].

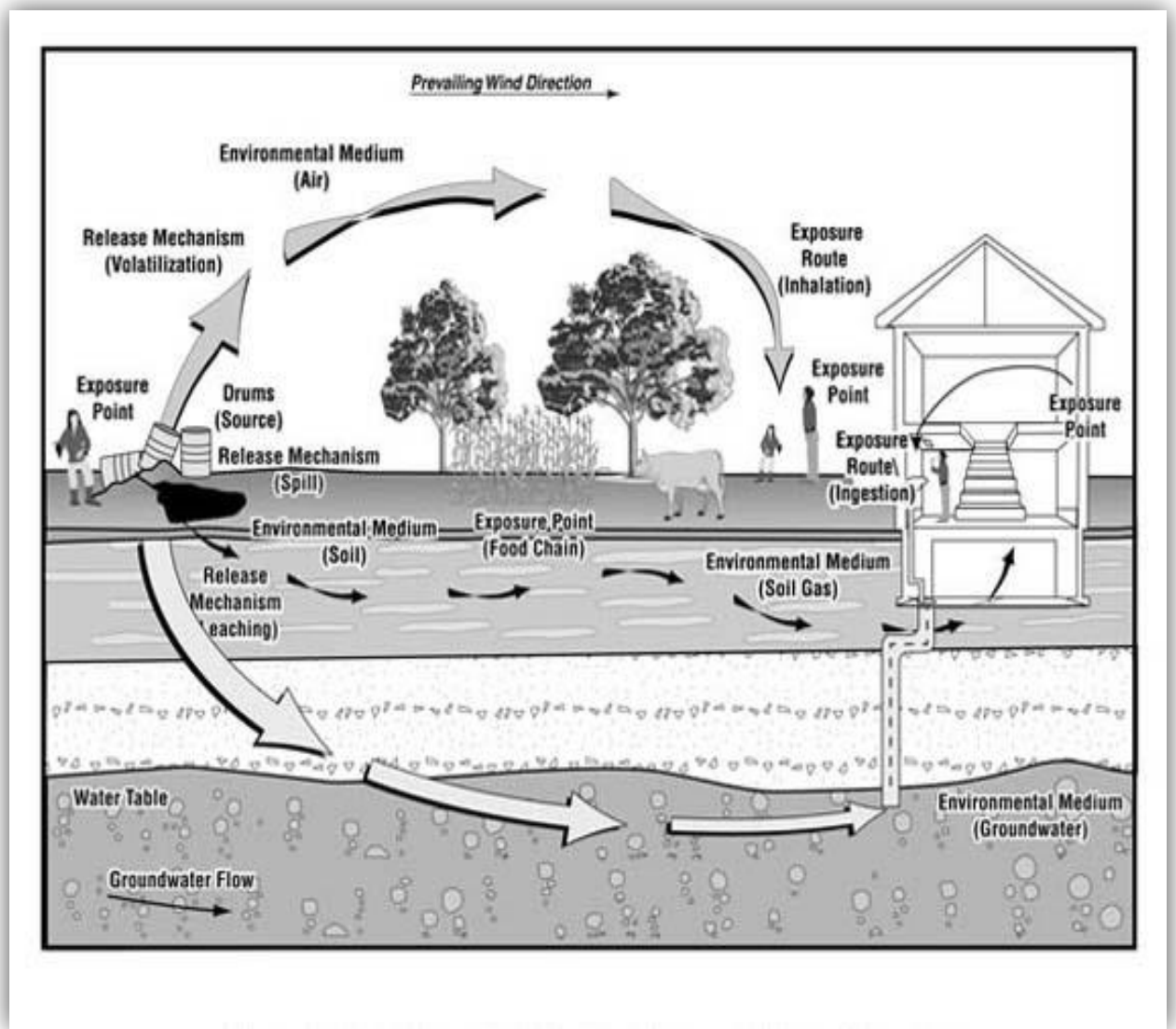



**Figure 1.1: showing the lead exposure pathway**. (Adopted from CDC.http//www//atsr.cdc.gov.hac/PAHManual/Ch6.html)[19]

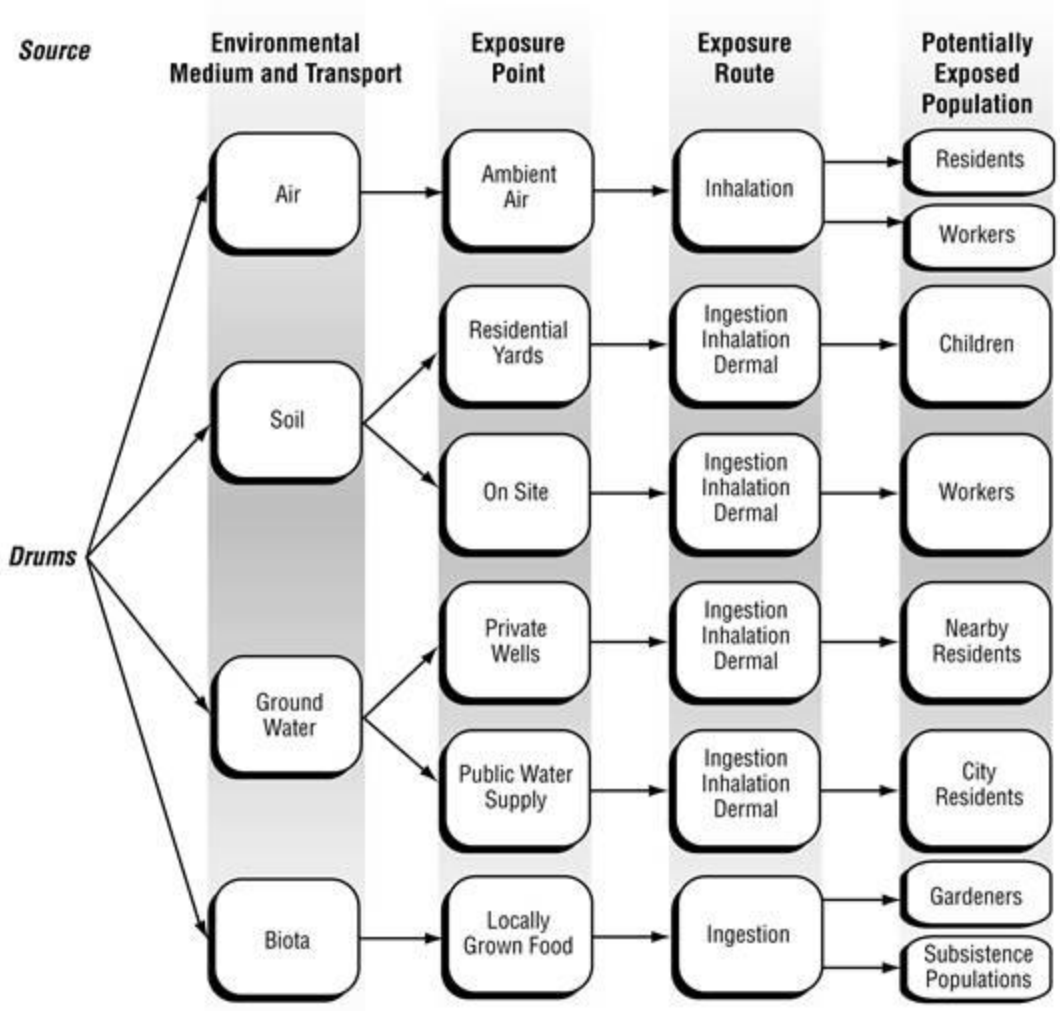



**Figure1. 2: Exposure pathway**.

Images adopted from CDC.

Humans are exposed to lead mainly via inhalation of contaminated fumes, ingestion of lead contaminated foods e.g. vegetables from contaminated soils or drinking lead contaminated water[15]. Dermatological exposure also occurs with the application of lead contaminated cosmetics[18]. The diagrams above summarize the lead exposure pathways.

### 1.3 Metabolism of Lead

When lead enters the body it binds to any ligand with a sulphyhdryl group and the most affected is haemogloublin[3,4,5]. Lead interferes with the dehydration of aminolevulenic acid and the incorporation of iron into aprotoporyphin molecule which results in a decrease in the production of haem[8,9,13]. Lead also has high affinity for the nervous tissue, kidney, liver, bones teeth and ovaries[3,7,15] . Lead crosses the placenta and it is also found in breast milk. Lead has a half-life of approximately 30 days in the intravascular space 90% of which is stored in bone with a half-life of decades[3,7,18,].Blood lead exists in equilibrium with the lead in the bone, such that conditions that increase bone turn over e.g. pregnancy or a fracture would result in an increase in blood lead levels. The toxic effects of lead are from the lead which is in circulation[7,9,14]. Lead is primarily excreted in urine and bile and excretion rate varies depending on the tissue absorbing the lead[3,5].

Lead poisoning is often asymptomatic or the symptoms are non-specific and can be mistaken for other disease processes[2,3]. The severity of the symptoms is dependent upon the amount and frequency of exposure. Essentially all systems are affected by elevated blood lead levels[20,21] In the neurological system, centrally there is impaired cognitive development, associated mental retardation and reduced IQ in children[21,22], irritability, depression and encephalopathy[22,23,25]. Peripherally there is segmental demyelination of mainly motor nerves and often results in peripheral neuropathy, wrist and foot drop[3, 5, 6]. Elevated lead levels also results in defective haem synthesis which results in anaemia and the presence of immature red cells in circulation[6]. In the gastrointestinal system there is nausea, dyspepsia, constipation, colic and lead gingival effects[3, 6, 7, 8]. Renal effects include chronic interstitial fibrosis, chronic nephropathy with tubular damage and hypertension[3,6.] Cardiorespiratory effects include aneurysms, hypertension, ventilation, perfusion mismatch and pulmonary granulomas[6,7]. Elevated blood lead levels also

affect the reproductive system, resulting in recurrent miscarriages/stillbirths in females and abnormal sperms and reduced sperm count/motility[1, 3, 5,]

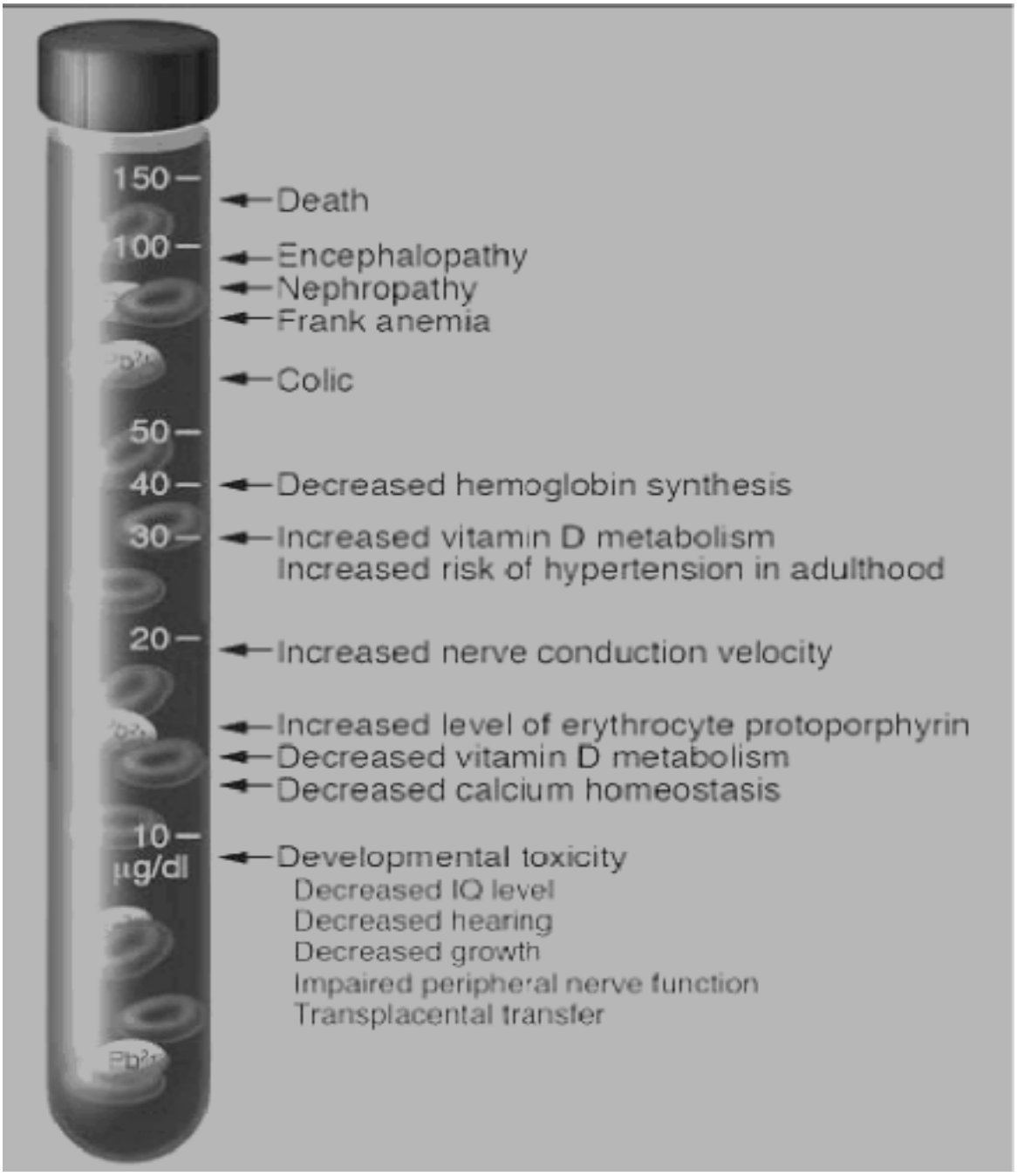



**Figure 1.3. Showing blood lead levels and symptoms** (Adapted from Bellinger and Bellinger ,2006)[20].

Lead is associated with wide range toxicity to the human body. Various extends of exposure have been studied from the lowest known blood concentration (which results in subclinical toxicity) [22,23] to the high levels of exposure which results in obvious clinical symptoms. Essentially all systems are affected by elevated lead levels[15,]. The main organs that are affected are the central and peripheral nervous system, the cardiovascular, gastrointestinal, renal, endocrine, immune and hematological systems are affected as well[15,16]. Acute toxicity can result from, high, intense exposure to lead and usually result in colic, constipation, fatigue and neurologic deficit. The insult to the neurological system can result in acute encephalopathy with ataxia, coma and convulsions. Usually the insult to the neurological system is irreversible. Subclinical lead toxicity also occurs at low lead levels which were thought to be relatively safe but can cause harmful effects which are not evident in a standard clinical examination[, 20, 21]. This has led to the continuous revision of the accepted blood lead levels for children from 70µg/dl in 1960s to 40µg/dl in the 1970s and down to 30µ/dl. In the 1980s the level was revised to 25µg/dl down to 10µg/dl in the 1990s[15]. Currently the recommended paediatric level is <5µg/dl [5]. This continuous revision of the acceptable levels is a clear indication of the knowledge of deleterious subclinical toxicity. The recommended levels for adults are currently at, ≤ 20ug/dl in adults with no occupational exposure to lead and ≤ 40ug/dl with occupational exposure to lead[5].

The most affected system by lead exposure is the neurological system, both centrally and peripherally. Motor neurons are the principal target in the peripheral nervous system[7]. Lead induced pathological changes include segmental demyelination and axonal degeneration, which results in ankle drop and wrist drop. This is as a result of extensor muscle palsy and is usually a sign of chronic lead poisoning[7, 8, 9]. Centrally high doses of lead exposure can result in an encephalopathy, confusion or even coma[14]. Asymptomatic impairment of neurobehavioral

function in children at doses as low as 1-3µg/dl has been reported. This usually results in lower IQ scores and mental retardation[22, 23, 24]. One of the mechanisms that has been postulated to explain the underlying irreversibility of lead's neurotoxicity, lies in the ability of lead to substitute for other polyvalent cations [(particularly divalent cations, such as calcium ($Ca^{2+}$) and zinc ($Zn^{2+}$)] and lead binds with greater affinity than calcium and zinc ions to protein binding sites. This allows lead to affect different significant biological processes. These include metal transport, energy metabolism, apoptosis and ionic conduction. Other processes that are affected are cell adhesion, intercellular and intracellular signaling, diverse enzymatic processes, protein maturation, and genetic regulation. Membrane ionic channels and signaling molecules seem to be one of the most relevant molecular targets that contribute to lead's neurotoxicity.[25, 26]

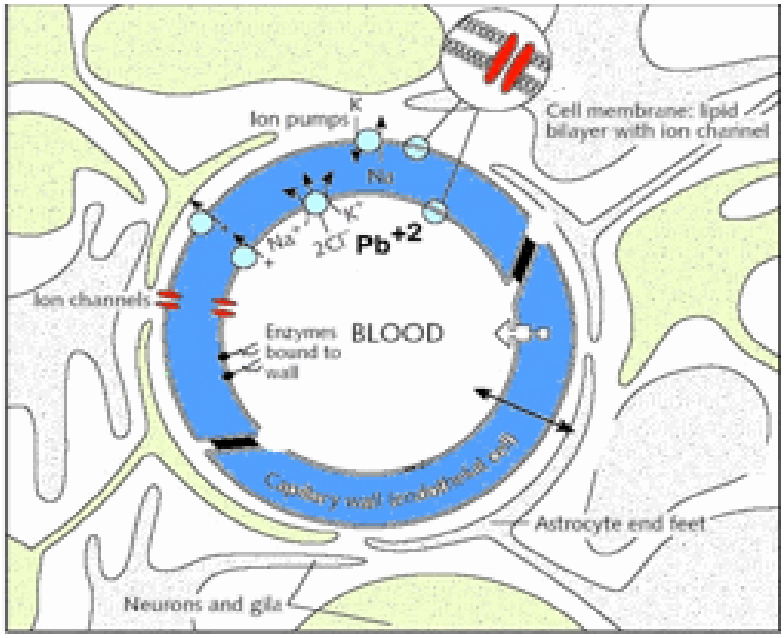



**Figure1.4: Lead movement through the blood brain barrier.**

Lead ions are able to permeate the blood brain barrier via ion channels and interfere in communication between astrocytes and endothelial cells. Adopted from http://content.answers.com/main/content[27](Oxford_Body/19852403X.blood -brain-barrier1.jpg

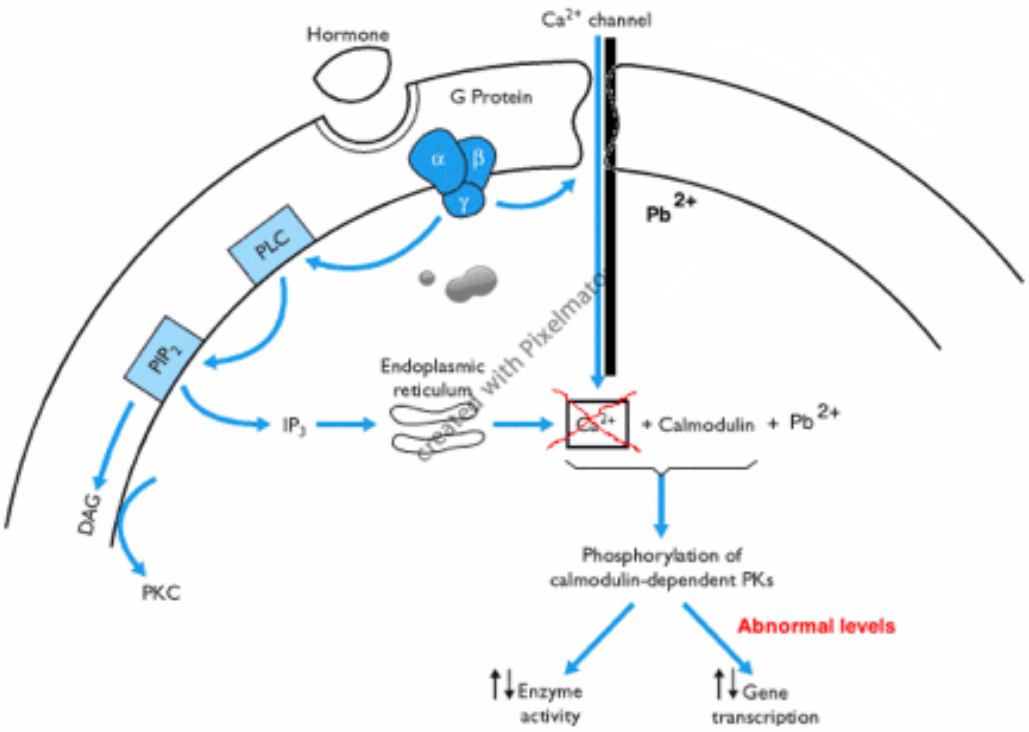



**Figure 1.5: Intracellular mechanisms of lead.**

$Pb^{2+}$ enters the cell through open $Ca^{2+}$ channel (stimulated by a G-protein) and readily binds than Calcium does. This causes phosphorylation of dependant protein kinases leading to abnormal enzyme activity and gene transcription. Alternatively, the G-protein activates Phosholipase C which in turn activates the hydrolysis of phosphotidylinositol 4,5 –biphosphate ($PIP_{2)}$ to inositol triphosphate ($IP_3$) and DAG. $IP_3$ normally releases stored calcium that combines with

calmodulin but it case where lead is stored in a cell organelle $IP_3$ may stimulate the release of $Pb^{2+}$ to combine with calmodulin[28].

### 1.4 History of lead exposure.

Before human exploitation, people were not exposed to lead. Human activities, primitive as the mining of lead ores to as recent as recycling of electronic waste (e-waste) are responsible for the increased lead in the environment. Once lead is introduced into the environment it persists[2,14]

Lead toxicity has been known since at least 2000 BC. Lead poisoning was common in Roman civilization, due to their use of leaded water pipes, lead earthenware, and wine storage vessels and a leaded wine sweetener called sapa[29]. The Japanese Samurai era ended due to lead poisoning. The main source of lead was from the women's make up. The women painted their faces with a white powder made up of white lead and mercury chloride. This led to exposure of their children via breastmilk and the environment[18]. Hippocrates is reported to describe a case of lead colic in 370 BC[30]. Sir Benjamin Franklin obtained a list of patients admitted at La Charite Hospital in France with symptoms unknowingly to them were evident of lead poisoning. All the patients' occupations exposed them to lead[30]. At the same hospital in 1830-1838, a French physician, Tanqueral des Planches described the symptoms of acute lead poisoning based on 1213 admissions for the 8years[3]. During the 19$^{th}$ and early 20$^{th}$ centuries many industrial workers (smelters, plumbers, painters and printers) were exposed to lead with various deaths from lead toxicity. In 1883 the Factory and Workshop Act was passed in the United Kingdom. The act required lead factories in the United Kingdom to conform to certain minimum standards aimed at reducing the worker's exposure to lead. This also gave birth to the routine medical surveillance of lead exposed workers[3,32].

Lead toxicity secondary to lead based paint was first observed in Australia in 1892. Children were the most affected as they were eating old peeling paint on their verandas. The source was established to be peeling old paint 12 years later[33]. Various anti-leaded paint campaigns were done, which led to the banning of lead based in Western countries in 1978[34]. Most countries have appreciated the toxicity of lead and have banned the use of leaded petrol[35]. In Zimbabwe there are no measures to control the amount of lead that individuals are exposed to environmentally. A study done by Kasilo and Nhachi did show that children in Harare were indeed exposed to lead [36].

The Zimbabwean government understands the toxicity of lead, such that it is making strides to try and reduce the amount of lead in petrol. A study done by Chipindu in 1999 showed the lead content in petrol to be 4g/l which was higher than the permitted values[37].In 2005 Zimbabwe met Mozambique and Malawi in an effort to reduce the imported leaded petrol. Currently 2 out of 3 depots are dedicated to the procurement of unleaded petrol. However the 2 out of 3 does not account for petrol that is imported in the country by private companies/sector. So the amount of leaded petrol that gets into the country might be higher than what the government of Zimbabwe estimates [38].

Harare, the capital city of Zimbabwe with a population of 2,5million[39] has the highest vehicle population of about 1, 2 million, which is about two thirds of the vehicles in the country. Most of the vehicles are second hand Japanese and European vehicles some of which are sometimes not roadworthy. Harare also has growing industries, in which they have lead battery manufacturing and recycling plants. These (vehicular and industrial smoke) put the Harare residents at risk of inhaling lead contaminated fumes. A study done by Mujuru[40] showed that air lead levels for Harare were highest in Southerton and Highfields. This could be attributed to industrial activity

and increased vehicles. Another study done in Harare by Mupa[41] reported that the air quality was much compromised in terms of lead pollution. This is sad situation because one of the ways to be exposed to by lead is through inhalation. So if Harare air is heavily polluted then her residents are at risk of exposure. Another study done by Madyiwa[42] showed that soil lead levels were elevated beyond recommended levels, as a result the lead levels in *Cynodon nlemfuensis* grass were also elevated which is the main grass that cattle graze on in Zimbabwe[42, 43]. This confirms the fear that there is a lot of lead in the food chain.

Other countries in the SADC region have not been spared against the effects of lead poisoning. Zambia has been struggling with lead exposure mainly from a closed lead mine in Kabwe, a mining city in the Northern part of Zambia's copper belt. The mine was closed in 1994. Lead blood concentrations measured in 2007 had a range of 50-100µg/dl[44]. A recent study done in Kabwe reported lead levels in free range chickens were elevated and unfit for human consumption[45]. The study demonstrated the persistent nature of lead in the environment. Once lead finds its way into the environment it is difficult to get rid of it. Currently Kabwe is in the top ten of the worst polluted places in the world according to the National Geographic Channel[44]. Nigeria is another African state that is suffering the ills of mining lead. Nigeria mines lead and other heavy metals in her Zamfara state[46]. Human rights and Environmental activists are already lobbying against the practice as they appreciate the health consequences that lead exposure has on the miners, the residents in the mining town as well as the environment[47].

South Africa has made major strides in trying to reduce the burden of lead exposure. South Africa banned the use of leaded paint in 2009[48,49]. However a recent study done on the amount of lead pigments on households in South Africa showed elevated of lead than that permitted according to WHO[15] guidelines. This has not been South Africa's problem alone, even Australia

which banned leaded paint a century ago still has "leaks" of leaded paint households or from companies that may have failed to abide to regulations[14]. United States banned leaded paint in 1978 but still has not managed to clean up lead from her streets[48, 59]. South Africa has recently managed to phase out leaded petrol.

Egypt managed to phase out leaded petrol in 1999. A follow up study showed a significant decline in blood lead levels in children who were resident in high traffic areas[1]. United States managed to phase out leaded petrol from 1976-1995. During that phase, studies that were done on children's blood lead levels were encouraging, the blood lead levels were declining as the amount of lead was declining in the petrol[49]. The reduction in blood lead levels in Egypt and the United States children, clearly indicate that lead poisoning is preventable.

### 1.5 Lead, Nutrition and Socioeconomic Status

Lead exposure is a hazard for the people with a low socioeconomic status as compared to people with a higher socioeconomic status[4.] A study done in South Africa by Nisha et al in 2010 showed that blood lead levels were higher in mothers and babies born to mothers with low educational levels and socioeconomic status[49]. Another study also done in South Africa by Yvon Schindling et al in 1991 showed that blood lead levels were elevated in black and mixed race children than in whites [51]. This was during the apartheid era in South Africa and blacks' residential areas were in the Southern Western Suburbs where the industrial gases flow to, as well as high traffic density. In Mexico high lead levels were noted in children who lived in town near trash dumping sites. The children were exposed as a result of scavenging lead contaminated trash e.g. storage batteries, toys and earthenware, which they would recycle for resale or use in their homes[51, 52]

Another thing that put the poor at increased risk of exposure to lead is backdoor industries. People with home industries sometimes use recycled e-waste (cell phones, computers) which

contain heavy metals like lead and mercury[14, 51]. Nutritional state of an individual has been shown to affect the amount of lead absorbed by an individual. Poverty is associated with decrease food intake as well as the quality of food eaten. Lead is absorbed more if ingested on an empty stomach than a full stomach[11]. In a study done by Flora et al, rats on a protein free diet retained more lead as compared to those with adequate protein[12]. This is worrisome since in Zimbabwe protein energy malnutrition is prevalent. Nutrients also affect how lead is deposited in tissues once it has been absorbed from the environment. Diet low in calcium results in greater deposition of lead in osseous and non osseous tissue[16]. Iron deficiency increases susceptibility to lead toxicity. A greater part of the Zimbabwean population has micronutrient deficiencies[53]. Exposure to lead also results in Vitamin D deficiency since lead interferes with enzyme that is responsible for the metabolism 1,25 hydroxycholecalciferol metabolism.

### 1.6 The Economic burden of lead Exposure

The economic cost associated with the environmental lead exposure is substantial[16] and the economic benefits associated with effective and successful interventions is beneficial[54,55]. WHO made an assessment of the global burden of environmental lead exposure in 2000, and discovered that the total burden of disease attributable to approximately 9 million disability-adjusted life years (DALYs), which represents about 0.6% of the global burden of disease[56, 57]. The direct costs of lead exposure include treating a patient who has acute exposure to lead. The indirect costs which are more costly are the economic effects of reduced IQ in adults who were exposed to lead as children. When children have mental retardation and reduced IQ as a result of lead exposure, there is an increased number of children who do poorly at school. Who in turn require special schooling and remedial work and may not contribute fully when they become adults and a loss of economic productivity[23, 58]. The decrease in intelligence attributable to each

1µg/dl increase in blood is 0.25IQ points, and the loss of economic productivity with loss of IQ point is 2.4% [58.]

Strategies to reduce /prevent lead exposure have shown multiple benefits. A study done by Grosse et al in 2002 estimated that the increase in children's intelligence following the removal of lead from petrol, would produce a benefit of US$110 -319 billion in each birth cohort[55]. The cost-benefit analysis done by Gould in 2009 suggested that for every dollar spent on reducing/preventing lead hazards there would be a benefit of US$17-220[56].

World over people have understood the toxicity of lead for many centuries and have taken measures to prevent or reduce the burden of lead poisoning [1, 2, 4]. Some countries do studies at regular intervals checking on how much lead is in the environment and people living in that environment and use the data to define any strategies that may need to be adopted to reduce the lead blood levels of a particular population[1, 4, 5, 9, 10, 13.] Workers who are also at risk of lead poisoning have regular screening of their lead levels and these help the employer to have an idea on how much he needs to step up his protective gear and improve the health safety of his/her employees[3, 4]. So the evaluation of lead levels should be an on-going process in a population with an associated risk of exposure[4] a strategy which Zimbabwe needs to adopt.

### 1.7 Analytical methods

Methods of measuring blood lead levels have changed over time. Atomic absorption spectrometry and anodic stripping voltammetry are the methods most frequently used for determining the levels of lead in blood.

**Table1.1 Overview of analytical methods for blood lead measurement.**

| Method | Advantages | Disadvantages |
|---|---|---|
| Flame atomic absorption spectrometry (FAAS) | -Requires only basic laboratory expertise<br>-Rapid analysis<br>-Small sample size using Delves cup (50–100 µl)<br>-Low purchase and running costs<br>-Relatively few interferences<br>-Robust interface | -Relatively high detection limit (~10 µg/dl)<br>-Time needed for sample digestion/preconcentration if not using Delves cup<br>-Large sample size needed for nebulization methods<br>-Should not be left to run unattended |
| Graphite furnace atomic absorption spectrometry (GFAAS) | -Good detection limit (<1–2 µg/dl)<br>-Small sample size<br>-Moderate purchase and running costs<br>-Some multielement capacity<br>-Relatively few interferences (although more than with FAAS)<br>-Widely used, available from multiple vendors | -Longer analysis time<br>-Requires some laboratory expertise (more than FAAS)<br>-Greater potential spectral interference than with FAAS |
| Laboratory anodic stripping voltammetry (ASV) | -Good detection limit (2-3 µg/dl)<br>-Low purchase and running costs<br>-Rapid<br>-Small sample size (~100 µl)<br>-Relative simplicity of equipment | -Requires some laboratory expertise (similar to GFAAS)<br>-Sample pretreatment needed<br>-Some factors might affect measurement (e.g. presence of copper)<br>-Becoming less available |
| Portable ASV | -Portable; measurement at point of care possible<br>-Simple to use; does not require skilled laboratory personnel<br>-Very low purchase and running costs | -Not as accurate as other methods<br>-Can determine levels only up to 65 µg/dl<br>-Levels above 8 µg/dl should be confirmed by a laboratory method |

| | | |
|---|---|---|
| | -Reasonably good detection limit for a portable device (3.3 μg/dl)<br>-Rapid | |
| Inductively coupled plasma mass spectrometry (ICP-MS) | -Excellent method detection limit (~0.1 μg/dl)<br>-Rapid<br>-Small sample size (50–100 μl) Relatively few, well-understood, spectral interferences<br>-Isotopic measurements possible<br>-Economic if very large number of samples<br>Multi-element capability | -High purchase and running costs<br>-Highly skilled laboratory operator required |

### 1.8 Study Rationale

Evidence has shown that lead poisoning is a preventable health hazard and measures can be taken to prevent it. Countries that have legislation that prevent/reduce the release of lead into environment have had positive feedback. Studies that have been done in the countries that have taken measures to reduce the burden of lead have shown significant decline in the blood lead levels. Results from this study will provide evidence on how people in Harare are exposed to lead. Since there is not much evidence of how much people in Zimbabwe are exposed.

### 1.9 Aim

To quantify the amount of lead in adults in Harare.

### 1.1 0 Objectives

1. To determine the amount of lead (in urine and blood) in adults in Harare urban and people living in a rural area.
2. To determine serum proteins levels and relate it to the amount of lead in blood.
3. To determine the amount of lead in Harare water and compare it to the one in rural areas.

## 1.11 Hypotheses

Objective 1

Null hypothesis ( **$H_0$)-** Adults in Harare have environmental lead exposure.

Alternative hypothesis (**$H_A$) -** Adults in Harare have no environmental exposure to lead.

Objective 2

**$H_0$-** There is a relationship between serum protein levels and blood lead levels.

**$H_A$-** There is no relationship between serum protein levels and blood lead levels.

Objective 3

**$H_0$** – There is a difference between the water lead levels in Harare and the rural area.

**$H_A$ -** There is no difference between the water lead levels in Harare and the rural area.

# CHAPTER 2 METHODOLOGY

## 2.0 Study design

This was a cross sectional and a laboratory study, in which lead levels and protein levels were, measured and analysed.

## 2.1 Study population

Participants were drawn from a population attending Harare's polyclinics .Mbare was chosen because of its proximity to the bus terminus, Highfield was chosen because of her proximity to Workington industry with the presence of a battery recycling plant there. Mabvuku/Tafara was chosen because of the proximity to a cement manufacture plant. All these residential areas were built in the pre-independence with old plumbing which may contain lead and solder. The houses may also have been painted with lead based paint, since the international ban of lead was in 1978. The last clinic was from Goromonzi district which acted as a comparative.

## 2.2 Sampling

The sample size was based on the evidence from similar studies which had similar sample sizes.[1,9,74]

## 2.3 Ethical Considerations

The study was approved by the Harare City Health Department Ethical Review Board, JREC and Medical Research Council of Zimbabwe. Verbal and written informed consent was obtained from each participant before the questionnaire and sample collection was done.

## 2.4 Recruitment

The participants were selected from relatives who had accompanied their sick. Health Education about non-occupational exposure was given to everyone attending the clinic on the particular day. Those willing to join the study and were eligible were offered the informed consent. A

questionnaire was conducted to act as a screening tool, to help to exclude people with occupational exposure, or who were non-resident in the study area.

### 2.5 Informed Consent

All participants were given written informed consent to participate in the study. The informed consent was administered either in English or Shona. The informed consent form contained information about the aims and objectives of the study. It also informed the participant of the procedures, risks and benefits of the study. The participant was given two copies of the informed consent to sign as study staff witnesses. The participant left one copy of the signed informed consent with study staff to be kept in the participant binder for verification. The other copy was taken home by the participant. No study procedure was commenced before signing of the informed consent.

### 2.6 Enrolment

After verification of signing of informed consent, the study staff assigned a unique participant ID (PTID) to the participant. The PTIDs were assigned chronologically from PTID list. The same PTID was also used for labeling the samples. The PTID was also listed with the patient's national identity number to avoid co-enrolment. Addresses were also collected for correspondence of the lead levels after the study is finished or if the lead levels were elevated. Assignment of the PTID indicated enrolment into the study and all study documents for the participants were identified by the PTID.

### 2.8 Procedures and techniques

5mls of venous blood was collected from the cubital vein into a heparinised tubes (BD-Plymouth PL6BP) and plain tubes (SGVac VP4011). Spot urine samples were collected using sterile containers. The samples were transported from the clinics in a vaccine box maintained at 0-4$^{o}$C, to the laboratory at UZ CHS where they were stored at 0-4$^{o}$C.

The samples were prepared for Inductively Coupled Plasma Spectroscopy or Atomic Emission Spectroscopy ICP- AES determination within 2 weeks of sample collection.

**2.9 Blood**

The blood samples were put in a rotatory vibrator for 10mins and then 50µL of whole blood was haemolysed with 270µL of DW, and 1,2ml Nitric Acid. (0,15N).The sample was capped and was mixed on a rotatory vibrator for 2minutes. 30µL of Bismuth nitrate was added to each sample. The processed samples were maintained at $4^0$C until they were analysed

**2.10 Urine**

The urine was filtered before sample preparation.50µl of urine was added to 2.4ml of nitric acid. The sample was capped put on a rotatory vibrator for 2 minutes and 50µL of bismuth solution was added to each sample. The processed samples were maintained at $4^0$C until they were analysed.

**2.11 Inductively Coupled Plasma Spectroscopy –Atomic Emission Spectroscopy (ICP-AES)**

ICP-EAS (Model 6500MK2 Thermo-scientific with ITEVA software, 2011) was used for analysis of lead in the sample solutions at Standards Association of Zimbabwe. The operating conditions were as follows: RF Power 1150W, Auxiliary gas flow 0.5L/min, Nebulizer gas flow 0.5L/min, Coolant gas flow 12L/min and Radial viewing height 12mm respectively. The settings for the sample pump were: Flush pump rate 50 rpm, Analysis Pump Rate 25 rpm and pump stabilization time was 5 seconds respectively. Each sample was determined in triplicate and the total analytical time was 30seconds. . The instrument was calibrated using an analysis solution (Certificate of Analysis) of single element aqueous CRM- Lead at 0.1ppm, 0.5ppm and 1ppm.

### 2.12 Protein

The protein levels were determined using the Lowry protein determination method[60,61,62]. See appendix.

### 2.13 Analysis

The analysis of the results was done using GraphPad Prism Version 6 and SPSS Version 17 statistical outputs. One way Anova was to be used to compare the means of blood lead levels for all the study populations, after being tested for normality. If not normally distributed, the Kruskal-Wallis test was perfomed. The urine and the blood lead levels from each urban area were compared to the rural area using an unpaired student t-test. $\mathbf{H_0}$ for objective one was to be rejected if $p > 0.05$. Logistical correlation was to be carried to ascertain if there is a relationship between serum protein levels and blood lead levels. $\mathbf{H_0}$ for the second objective was to be rejected if $p > 0.05$.

# CHAPTER 3 RESULTS

## 3.0 Descriptive Statistics

They were sixty (60) healthy adults aged 16years and above. Forty-five (45) of the participants were from Harare's high density suburbs, Mbare, Highfield and Mabvuku/Tafara, the rest of the participants Goromonzi district.

**Table 3.1: Descriptive statistics of the studied population in 4 studied areas**

| | Variable | | | | |
|---|---|---|---|---|---|
| **Studied area** | Age /years | BLL/µg/dl | Serum protein levels g/l | Head Circumference /cm | Water lead levels/µg/dl |
| **Mbare n=15** | 56.93±1.98 | 0.8868±0.727 | 24.41±1.71 | 56.93±1.98 | 1.1 |
| **Mabvuku/Tafara n=14** | 57.33±2.32 | 0.700±0.5684 | 22.97±2.95 | 57.33±2.32 | 1.6 |
| **Highfield n=15** | 55.80±2.97 | 0.827±0.769 | 28.17±6.79 | 55.80±2.95 | 0.4 |
| **Goromonzi n=15** | 56.40±1.35 | 0.962±0.9830 | 22.26±3.60 | 56.40±1.35 | 0 |

In Table 3.1, the studied population has been classified into four groups according to their residential areas, representing the 4 activity areas.The mean age of the participants from Mbare was 56.93±1.98 years, 57.33±2.32 years for Mabvuku/Tafara, 55.80±2.97 years for Highfield and 56.40±1.35years for Goromonzi. The mean head circumference for participants from Mbare was 56.93±1.98cm, 57.33±2.32cm for participants from Mabvuku/Tafara, 55.80±2.95cm for participants from Highfield and 56.40±1.35cm for participants from Goromonzi. The mean protein levels were 24.41±1.71g/l for Mbare, 22.97±2.95g/l for Mabvuku/Tafara 28.17±6.79g/l for Highfield and 22.26±3.60g/l for Goromonzi respectively.

### 3.1 Lead levels

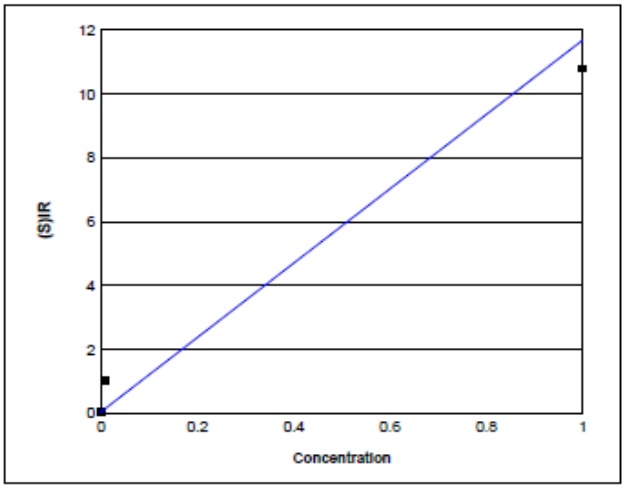
The blood lead levels were obtained using the standard curve below. The blood lead level result for participant number seven from Mabvuku/Tafara was not used in statistical analysis due to contamination

**Flexible Report By Sample**

Author:

Published: 10/25/2013 3:52:29PM

Method Name: lead in blood (6)




| | |
|---|---|
| **Element Name:** | Pb |
| **Element Wavelength:** | Pb 220.353 nm |
| **Concentration Units:** | ppm |
| **Date of Calibration:** | 10/15/2013 12:29:24PM |
| **Date of Fit:** | 10/15/2013 12:29:24PM |
| **Type of Fit:** | Linear |
| **Correlation:** | 0.79763 |
| **A0 (Offset):** | 0.036227 |
| **A1 (Gain):** | 11.636 |
| **A2 (Curvature):** | 0.00000 |
| **n (Exponent):** | 1.0000 |

| **Reslope** | | **QC Normalize** | |
|---|---|---|---|
| **Slope:** | 1.0000 | **Slope factor:** | 1.0000 |
| **Y Int:** | 0.00000 | **Offset:** | 0.00000 |

| Standard Name | Stated | Found | Diff | % Diff | (S)IR | Stddev | Emphasis |
|---|---|---|---|---|---|---|---|
| BLANK | 0.00000 | 0.000000000 | -0.000074823 | 0.00000 | 0.035356 | 0.71969 | 1 |
| CalibStd-1 | 0.010000 | 0.090000 | 0.075579 | 755.79 | 1.0320 | 0.60223 | 1 |
| CalibStd-3 | 1.0000 | 0.92000 | -0.075579 | -7.5579 | 10.792 | 0.57841 | 1 |

| Sample Name | Acquisition Date | Correction Factor |
|---|---|---|
| CRM SAZ | 10/15/2013 12:33:15PM | 1.00 |

**Concentration**

| | |
|---|---|
| **Element/Wavelength** | **Pb2203** |
| **Units:** | ppm |
| **Avg. of Repeats:** | 0.217836 |
| **Std Dev:** | 0.0503551 |
| **%RSD:** | 23.1161 |
| **Repeat: 1** | 0.182328 |
| **Repeat: 2** | 0.275465 |

**Figure 3.1. Standard curve used for obtaining lead levels**

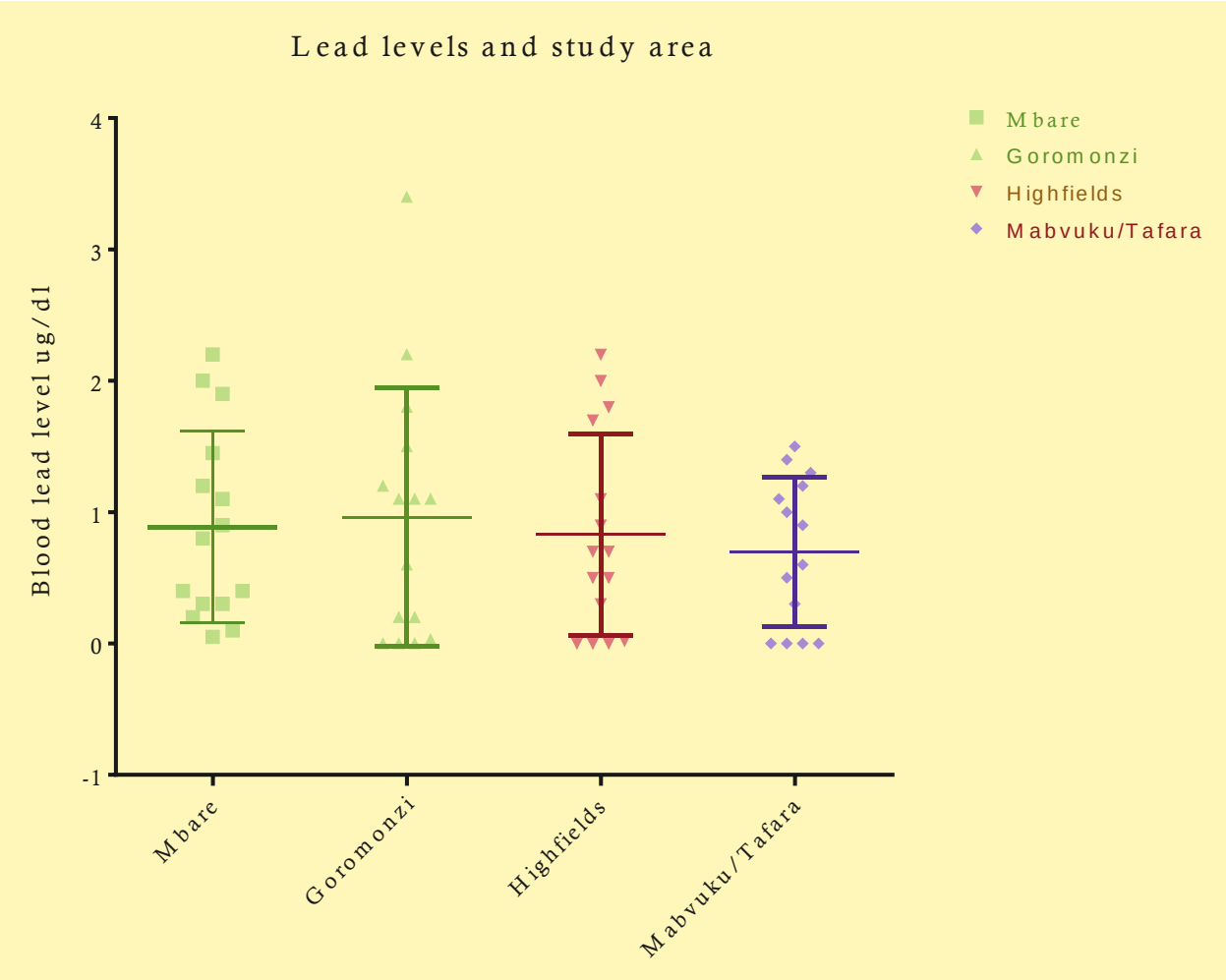



**Figure 3.2 Graph showing blood lead levels in the different study populations.**

The mean blood lead levels were 0.8868±0.727µg/dl for participants from Mbare, 0.700±0.5684µg/dl for Mabvuku/Tafara. The mean blood lead level for Highfield was 0.827±0.769µg/dl and 0.962±0.9830µg/dl for Goromonzi. The maximum measured levels in individual clinics were 2.2 µg/dl, 3.4µg/dl, 2.2µg/dl and 1.5µg/dl for Highfield, Goromonzi, Mbare and Mabvuku/Tafara respectively. The lowest recorded blood lead levels were zero from each study group.

The blood lead levels failed the Shapiro-Wilk normality test. The Kruskal -Wallis test was done. There was no statistical difference among the means. The Kruskal-Wallis test statistic was 0.659 and a p value of 0.8842. The sign test was used to compare each urban study population to the rural study population. There was no significant statistical difference between the means of Mbare and Goromonzi with a sign statistic of 0.2386 and a p value of 0.81. There was no difference between Highfield and Goromonzi, the sign statistic was 0.416 and a p value of 0.681. There was no statistical difference between the mean blood lead level of, Mabvuku/Tafara and Goromonzi with sign statistic of 0.8703 and a p value of 0.391.

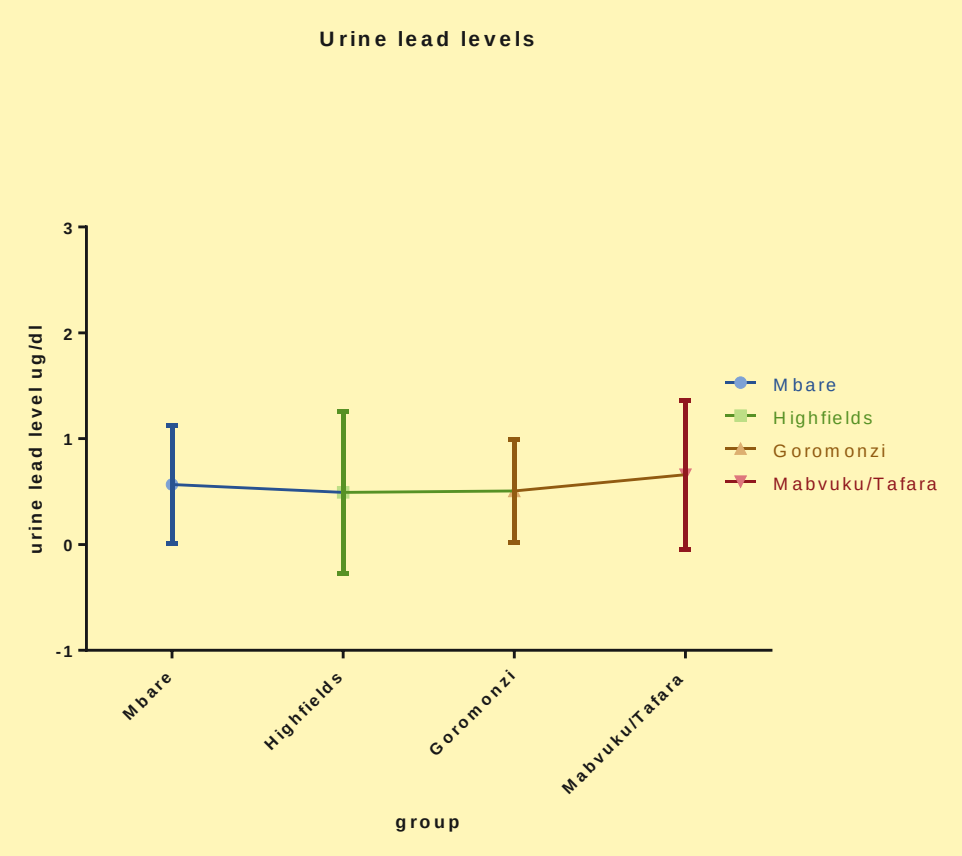



**Figure3.3: Showing the urine lead levels and study population.**

The mean for the urine lead levels were 0.56±0.55µg/dl for Mbare, 0.4933±0.7630µg/dl for Highfield, 0.5067±0.1267µg/dl for Mabvuku/Tafara and 0.660±0.7059µg/dl for Goromonzi respectively. The highest measured urine levels were 1.9µg/dl, 2.4µg/dl, 1.5µg/dl and 2.5µg/dl for Mbare, Highfield, Goromonzi and Mabvuku/Tafara respectively. The urine lead levels were subjected to a Kruskal-Wallis test. There was no statistical difference among the mean of the four groups. The Kruskal-Wallis test statistic was 1.468 and a p-value of 0.6898. The urine lead levels from the urban areas were subjected to a sign test each being compared to the rural urine lead levels. There was no statistical difference among all the means in the urban areas and the rural area levels.

The water lead levels from the different study populations were 1.1µg/dl, 1.6µg/dl, 0µg/dl and 0.4µg/dl, for Mbare, Highfield, Goromonzi and Mabvuku/Tafara respectively.

### 3.2 Protein Levels

**Table 3.2 Absorbance values for Standard Curve at 600nm**

| BSA Concentration/ mg/ml | 0.00 | 0.1 | 0.3 | 0.4 | 0.6 | 0.8 | 0.9 | 1.0 | 1.5 | 2.0 |
|---|---|---|---|---|---|---|---|---|---|---|
| Absorbance/ Λ | 0.00 | 0.037 | 0.107 | 0.132 | 0.186 | 0.247 | 0.286 | 0.324 | 0.460 | 0.555 |

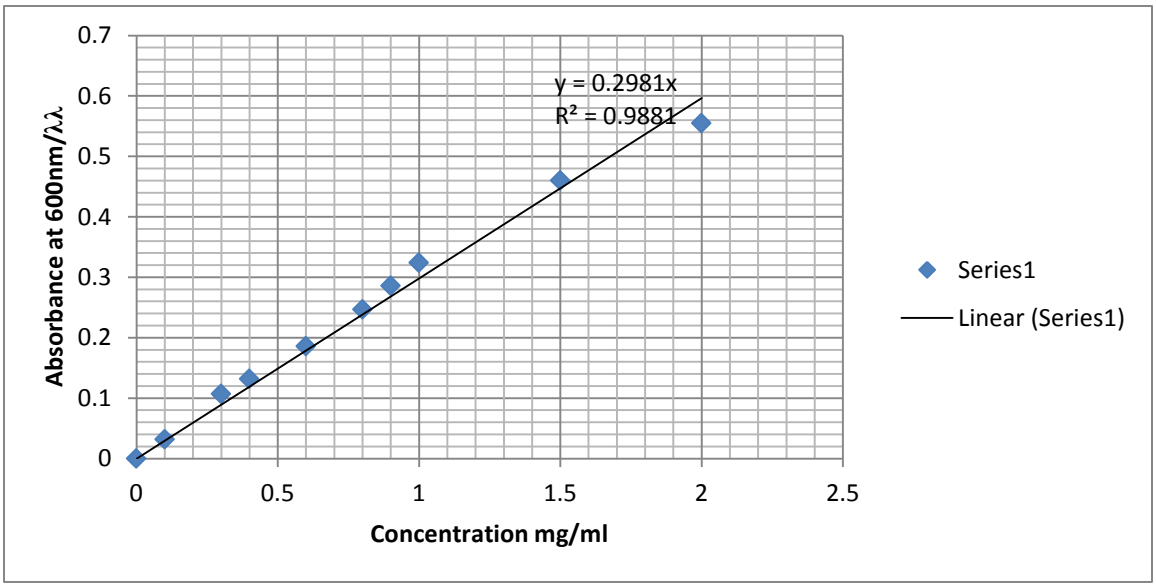



**Figure 3.4. Standard Curve from the Lowry protein determination method.**

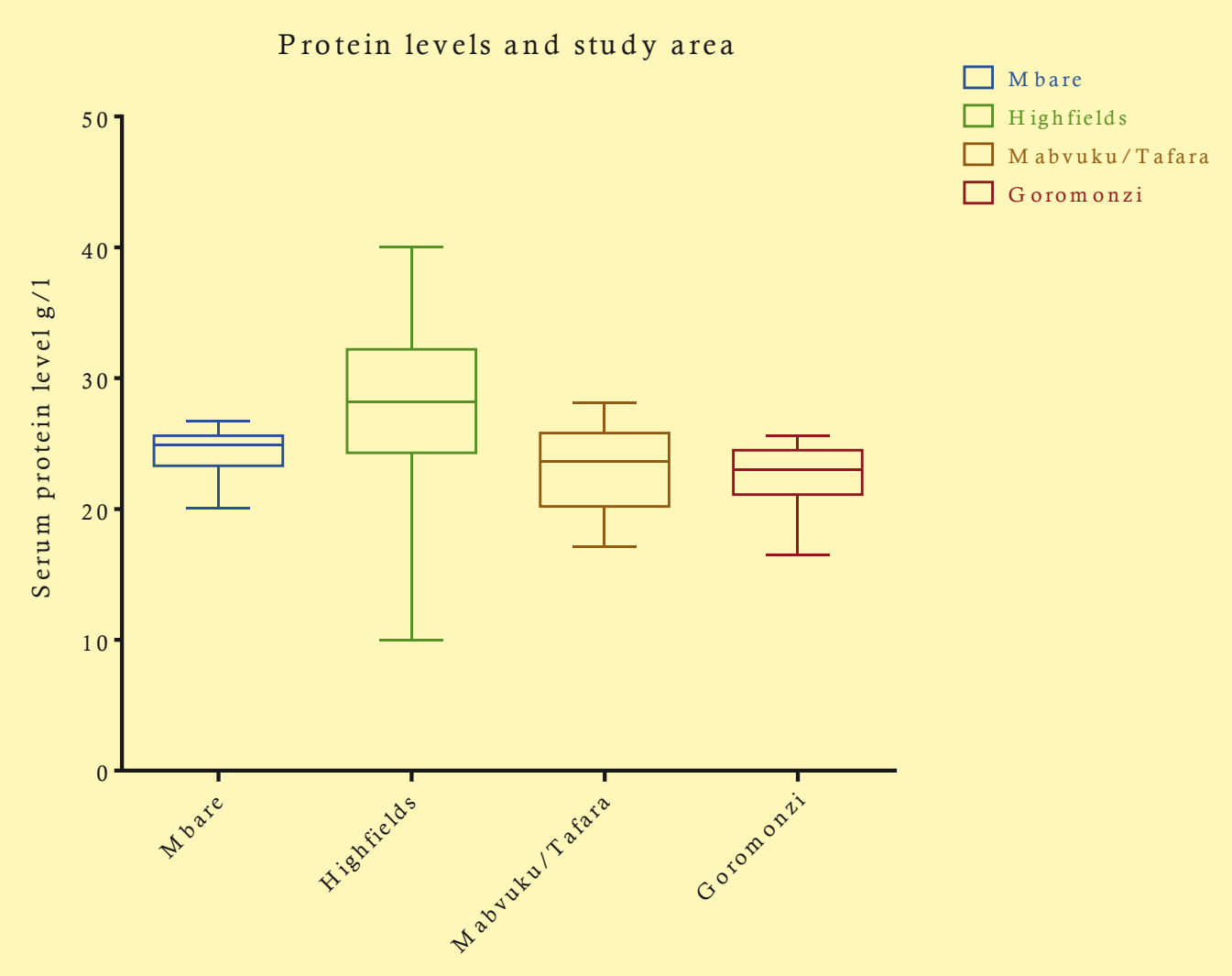



**Figure 3.5: Serum protein levels and study population.**

The mean serum protein levels for the participant from Mbare, Highfield, Mabvuku/Tafara and Goromonzi were 24.41±1.71g/l, 28.17±6.79g/l, 22.97±2.95g/l and 22.26±3.60g/l respectively. Anova revealed a significant difference in the serum protein levels from the four different areas. The F test statistic was 5.888, p-value 0.0014 and $R^2$ 0.234. The post hoc analysis using a Tukey showed a significant difference between Mabvuku/Tafara and Highfield with a p value of $p < 0.05$ as well Highfield and Goromonzi with a p value $<0.05$.

### 3.3 Correlation between serum protein levels and blood lead levels

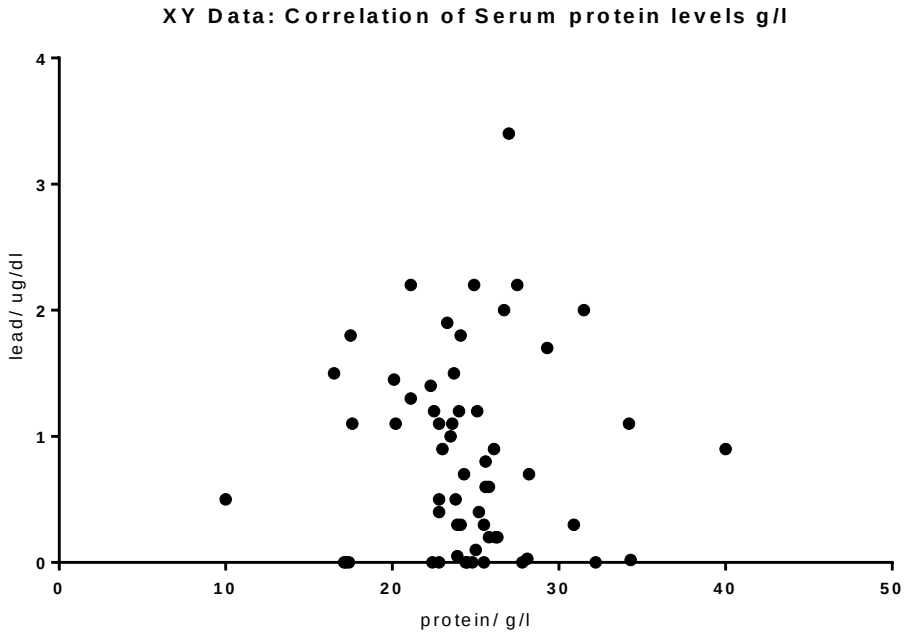



**Figure 3.6: Correlation plot between serum protein levels and blood lead levels.**

The graph above is the correlation plot of serum protein levels and the blood lead levels. There was a negative non significant correlation between serum protein levels and the blood lead levels. Pearson Correlation coefficient $r^2$= 0.225, p=0.1244.

# CHAPTER 4 DISCUSSION

This study had three objectives. The first objective was to determine the amount of lead in adults in Harare urban and compare it to people living in a rural area. The second objective was to determine serum protein levels and relate it to the lead levels in blood. The third was to determine the amount of lead in Harare water and compare it to the one from a rural area.

The mean lead level obtained for the population under study in urban area was 0.8046±0.6881µg/dl, which is below the recommended value of 20µg/dl of CDC[5]. The means in others for different study groups were <1µg/dl. These results show a significant decline in blood lead levels, compared to a study done by Kasilo and Nhachi in 1999 in children in Harare which the highest recorded mean was 31.07±7.33µg/dl[36]. There seems to be a great reduction in blood lead levels in the past fourteen years. However the lead levels might have been different if this current study was carried in a pediatric population. As children are more prone to exposure than adults due to their higher metabolic need per kilogram of weight, playing habits and no control of their environment[21]. Children also absorb lead from the gastrointestinal tract five times more than an adult[4].

The lead in drinking water for Mbare, Mabvuku/Tafara, and Highfield were 1.1µg/dl, 1.5µg/dl and 0.4µg/dl respectively. These values are above the recommended 0µg/dl by the EPA and CDC[63]. One value is at par with the action level for treatment set at 1.5µg/dl[64]. According to WHO guidelines if a concentration of 5µg/dl in drinking water is assumed, the total intake of lead from this source can be calculated to range from 3.8µg per day for an infant to 10 µg per day for an adult[65].

The lead in water could be as a result of its dissolution from pipes, solder pipes in the plumbing system. Polyvinyl chloride (PVC) pipes also contain lead compounds that can be leached from them and result in high lead concentrations in drinking-water[65]. The amount of lead dissolved from the plumbing system depends on several factors, including the presence of chloride and dissolved oxygen, pH, temperature, water softness and standing time of the water, soft, acidic water being the most plumbosolvent[4,10,65]. The level of lead in drinking-water may be reduced by corrosion control measures such as the addition of lime and the adjustment of the pH in the distribution system from <7 to 8–9[10, 65]. Lead can also be released from flaking lead carbonate deposits on lead pipe and from iron sediment from old galvanized plumbing that has accumulated lead from lead sources such as plumbing and service connections, even when the water is no longer plumbosolvent[65].

The ranges of the urine and blood lead levels obtained in this study were low. This maybe because of the public health interventions that are being put in place to reduce the burden of environmental exposure to lead. The United States banned the use of leaded pipes in 1986[63,64], Australia had leaded pipes phased out in the mid 20$^{th}$ century[66]. The United States has managed to ban leaded paint by 1978[4]. Closer home, South Africa has recently managed to phase out leaded petrol[49] and is the process of putting control measures to control the amount of lead in paint. At the moment Zimbabwe is in the process of phasing out leaded petrol[4]. In Zimbabwe there is no active lead mining, as a result the obtained levels as high as 3.4µg/dl are consistent with the exposure of contaminated water and air from the exhaust fumes.

In this study values of BLL as high as 3.4µg/dl was obtained from an adult participant. The same value would have serious detrimental effects in a pediatric participant. This level, though

below the recommended CDC values, would result in subclinical toxicity[20,67]. The most affected system is the neurological system. These low levels would cause subclinical damage to the growing brain and interfere with many delicate processes. This may later result in anti-social behaviors, truancy and reduced IQ[23].

The observed lead levels were low enough not to be associated with the detrimental effects of lead exposure in adults such as renal disease, hypertension and cardiovascular disease. These usually occur with levels above 40µg/dl. Appearance of lead- protein complexes in the renal proximal tubular epithelial cells occurs at blood lead levels between 40-80 µg/dl[65].

The mean serum protein levels obtained in this study were 24.41±1.71g/l for Mbare, 28.17±6.79g/l for Highfield, 22.97±2.95g/l and 22.26±3.60g/l for Goromonzi. The measure of serum protein is more specific to the serum albumin concentration[68]. The values obtained in this study are below the reference range of 35-50g/l. One of the reasons for reduced serum protein level is chronic protein energy malnutrition[68]. Malnutrition is rampart in Zimbabwe. According to UNICEF, one in every three children under five suffers from malnutrition[69]. The effects of malnutrition carry on into adulthood.

Lead levels are affected by the nutritional state of an individual. Micronutrient deficiency affect lead metabolism[4,11]. Blood lead levels in the range of 12-120µg/dl interfere with calcium metabolism by interfering with haeme pathway of generating vitamin D precursor 1,25-

Dihydroxycholecalciferol[4,65]. This results in a significant decrease in the level of circulating 1,25-dihydroxycholecalciferol, when the lead level s reach the mentioned concentration[65]. Other micronutrients affected by lead exposure are, iron, zinc, and vitamin C[70].

Several studies have been done to try and find the effect of blood lead levels on protein levels and vice versa. A study done by Patel and Ventakratishna-Bhatt[71] in 1992, in albino rats showed a 72% increase in the serum total and PCA sialic acid in lead treatment group[71]. A study done by Hristev et[72] al in 2009, in rabbits with chronic administration of lead and cadmium showed a significantly raised content of the cholesterol, ASAT and ALAT; hypo-albuminemia and hyperbetaglobulinemia of the background of one hypoproteinemia and low A/G coefficient are established. The obtained result also showed some degree of liver parenchyma damage[72]. In the study done by Jaremin in 1983[73] in adults who had occupational exposure to lead, showed increased lead levels resulted in a decrease in serum immunogloublins G and M and the total lymphocyte count. However the was no change in serum proteins[73].

In a study done by Pachathundikandi and Varghese in 2006[74], in adults who had chronic lead exposure and elevated blood lead levels showed that there was mean decrease in the serum protein and the total cholesterol levels. In comparison to their counterparts with lower lead levels[74]. The exact mechanism in which lead interferes with protein metabolism is unknown[74]. In this current study, correlation was done between the serum protein levels and the blood lead levels. The Pearson correlation factor was $r^2$ 0.225 at a p-value of 0.124. This shows that there was no statistical significance in the lead levels and protein levels in the studied populations.

This could have been as a result of the low lead levels observed in the study. Comparisons that were made in the past studies were done with elevated lead levels[71,72,73,74].

Head circumferences of the participants were also measured to assess for effects of chronic malnutrition and chronic lead exposure. The mean head circumferences for the study populations were 56.93±1.93cm for Mbare, 57.33±2.32cm for Mabvuku/Tafara, 55.80±2.95cm for Highfield and 56.40±1.35 for Goromonzi. There was no statistical difference among the groups. Head circumference is usually used in the paediatric population to measure insult to the brain, assess nutritional status and growth. Childhood effect on the head circumference may spill into adulthood.

A study done by Hernandez –Avilla et al in 2002, on the effect maternal bone lead concentration on the newborn showed that a higher concentration of maternal bone lead was associated with a small head circumference, decreased bone length and reduced birth weight[75]. A study done in older children (preschoolers) showed a marginal inverse relationship between blood lead levels and head circumference[76]. Not much evidence is available for the effect of BLL and head circumference in adults. The head circumferences obtained in this study were within the 97$^{th}$ centile of the Tanner staging charts for head circumference for 16 year olds. Currently there are no charts for head circumference for adults[78,79].

## Conclusion

The present study suggests that residents in Harare have low blood and urine lead, indicating minimal environmental exposure to lead. There is subchronic malnutrition in the adult population and there is no relationship between blood lead levels and serum protein levels.

## Recommendations

Blood and urine lead levels should be measured in the pediatric population of Harare since children absorb more lead than adults and the effects are more catastrophic, even at lower doses in children. Regular screening of lead levels in drinking water and air quality is recommended. The ongoing process of the phasing out of leaded petrol from Zimbabwe is also recommended.

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# APPENDICES

## APPENDIX 1

### SUBJECT INFORMED CONSENT

**PROTOCOL TITLE**: Blood and urine lead levels in adults in Harare polyclinics

**PRINCIPAL INVESTIGATOR:** Dr Prudence Manyuchi (MB, ChB) 0772 957 984

**SUPERVISORS:** Prof CFB Nhachi

Dr D Tagwireyi

### PROJECT DESCRIPTION

You are being asked to volunteer for a research survey conducted by an MSc student in Clinical Pharmacology at the University of Zimbabwe. The study involves the measurement of blood and urine lead levels in adults attending Harare polyclinics. Lead is a heavy metal with no biological benefit to man. Exposure to lead can occur after breathing of air contaminated with lead (which is usually from car exhaust fumes using leaded petrol, which is most of the petrol found in Zimbabwe) or eating food or drinking water contaminated with lead. High lead levels can cause mental slowness or reduced mental capacity, numbness and tingling sensation of hands and legs, hypertension, heart and renal disease. High lead levels can also caused depletion of normally formed blood cells, reduced sperm motility, abnormal sperms, stillbirths and miscarriages. The lead levels will be compared to the internationally recognized standards. Blood protein levels will also be measured to see if the amount of lead is comparable to the nutritional state in an individual.

### YOUR RIGHTS

Before you decide whether or not to volunteer for this study, you must understand its purpose, how it may help you, and what is expected of you. This process is called informed consent.

### PURPOSE OF RESEARCH STUDY

The study is to determine the amount of lead in urine and blood in adults in Harare. As well as trying to establish if there is a relationship between blood lead levels and protein levels.

### PROCEDURES INVOLVED IN THE STUDY

You will be asked questions through a questionnaire and a physical examination. Then 5mls (approximately a teaspoon) of blood will be drawn. You will also be given a urine collecting bottle, where you will collect your urine in a private secluded toilet. No other tests will be done except for the tests agreed above. The procedures (urine and blood collection) will be done once.

**DISCOMFORTS AND RISKS**

Apart from slight of discomfort giving blood, no other discomfort is anticipated. Going through the interview and physical examination may take you about 20 minutes.

**POTENTIAL BENEFITS**

You are going to contribute to the body of knowledge. The results in this study will help us to know if people in Harare are exposed to unacceptable levels of lead. You shall be informed about your results after the samples have been processed in the laboratory. If the lead levels are high you shall be referred to the nearest hospital to be managed by specialist physicians. You shall be given a token of lunch and transport money, a sum of US$5 for the time spent participating in the study.

**STUDY WITHDRAWAL**

You may choose not to enter the study or withdraw from the study at any time(from the beginning, during or towards the end).You will not be asked questions or try to be held accountable for your decision.

**CONFIDENTIALITY OF RECORDS**

Every effort will be made to protect participant privacy and confidentiality to the extent possible. No information identifying you will be published without your permission.

**PROBLEMS OR QUESTIONS**

Please ask questions about this research or consent now. If you have any question in future please ask.

**AUTHORIZATION**

I have read this paper about the study or it was read to me. I understand the possible risks and benefits of this study. I know being in this study is voluntary. I choose to be in this study. I know I can stop being in the study and I will not lose any benefits entitled to me. I will get a copy this consent form. (Please initial all the previous pages of the consent form.)

_____

Client Signature Date

_____

Client Name (printed)

_____

Researcher Signature Date

_____

Witness Signature Date

Initials_____

## GWARO RETENDERANO

**PROTOCOL TITLE**: Blood and urine lead levels in adults in Harare polyclinics

**PRINCIPAL INVESTIGATOR:** Prudence Manyuchi (MB, ChB) 0772 957 984

**SUPERVISORS:** Prof CFB Nhachi

Dr D Tagwireyi

### ZVINOENDERANA NECHIRONGWA

Pane chirongwa ye Zimbabwe ari kuita Masters yeClinical Pharmacology. Chirongwa ichi ndechetsvagiridzo yemunyu unonzi lead. Munyu uyu unenge uchitsvagwa muropa nemuweti. Munyu uyu unopinda mumunhu mushure mekunge afema mhepo ine munyu uyu, kunyanya mhepo inobva mudzimotokari dzinoshandisa mafuta epeturo. Kunwa mvura, kana kudya chikafu chakasanganiswa nemunyu uyu Munyu uyu hauna zvaunoshanda mumunhu kunze kwekukonzera zvirwere zvakaita sekushomeka kweropa mumuviri, BP, zvirwere zvemwoyo, zvirwere zveitsvo, nekusanyatsoshanda zvakanaka kwepfungwa. Munyu uyu ukawandisa unoita kuti mbeu yababa isakwanisa kusamhanya zvakanaka nekubva pamuviri mumadzimai. Kana munyu uyu wawanikwa uchaongororwa zvichitariswa kuti unoendereana zvakadini nemumyu unotenderwa kunge uchiwanikwa muvanhu pasi rese. Uyezve muropa menyu muchatariswa kuti zvinowaka muviri zvine ukama hwakadii nemunyu uyu.

### KODZERO DZENYU

Musati mapa sarudzo kuti mopinda muchirongwa here kana kutikwete. Tinoda kukutsananguriraichinangwa chechirongwa. Kubatsirikana kwamungaita kubvapa chiri, njodzi nezvinotarisirwa kubva kwamuri. Izvi zvinoita kuti muite sarudzo yenyu muchiziva.

### CHINANGWA CHECHIRONGWA

Donzo rechirongwa ndere kuona kuti vagari venzvimbo ino vane munyu unonzi lead wakawanda zvakadini. Uchitsvagwa muropa nemuweti. Uye zvinowaka muviri zvakawanda zvakadini.

### ZVICHAITWA MUCHIRONGWA

Kana mabvuma kupinda muchirongwa muchabvunzwa mibvunzo nekutariswa nachiremba. Zvapera muchatorwa ropa rinokwana kuita 5mls kana kuti tii spunu imwechete. Muchapihwa

pamwe pekuitira weti yamunonoitira zvenyu kuchimbuzi. Zvinhu zvese izvi zvichaitwa kamwechete pamunhu.

## ZVIKANGAIDZO KANA NJODZI YAMUNGASANGANA NAYO

Hatitarisire kuti pangave nenjodzi yamungasangane nayo kunze kwekuswinyiwa zvishomashoma pamunenge muchitorwa ropa. Kubvunzwa mibvunzo nekuongororwa nachiremba zvinogona kukutorerai maminetsi anokwana kuita makumi maviri.

## ZVAMUNGAWANA KUBVA MUCHIRONGWA

Hapana mibairo yamuchawana kupinda muchirongwa. Zvichabuda muchirongwa ichizvinogona kuzokubatsirai imi nevamwe mune ramangwana paruzivo rwekuwanda kwemunyu welead muHarare. Asi munyu wenyu ukaonekwa wakawandisa munonyorerwa tsamba yeknonoona chiremba wepamusoro kuti akubatsirei. Zvakare muchapihwa mari inokwana kuita US$5 kudzorera panguva yamapambadza muchipinda nuchipinda muchirongwa.

## SARUDZO YOKUBUDA MUCHIRONGWA

Sarudzo ndeyenyu kuva muchirongwa chino kana kusapinda. Kana mapinda muchirongwa munokwanisa kubuda machiri chero nguva pasina kubvunzurudzwa kuti ngenyi mafunga kubuda.

## KUCHENGETEDZEKA KWEZVOSE ZVATAURWA NEMI

Zvose zvichaitwa muchirongwa kana weti neropa zvichachengetedzwa pakahwanda. Hakuna munhu anozoziva kut imhindiro, weti kana ropa ndezvenyu.Kana nzvimbo yamunoshandira ndeipi sezvo hakuna mazita achanyorwa pasi pabepa remibvunzo kana magaba eweti neropa.Hapana zvinwe zvichaongororwa muropa kana muweti kunze kwezvanyorwa pamusoro.

## MIBVUNZO KANA ZVICHEMO

Kana mukaita mibvunzo yechirongwa kana yebepa rino, sunungukai kubvunza.Chero mukazofunga imwe mibvunzo mune remangwana sunungukai kubvunza.

## KUBVUMA KUPINDA MUCHIRONGWA

Ndaverenga bepa rino rechirongwa chirikuitwa. Ndanzwisisa njodzi nezvandingawana muchirongwa chino.Ndanzwisisa kuti isarudzo yangu kupinda kana kusapinda muchirongwa.Ndasarudza kupinda muchirongwa. Ndanzwisisa kuti ndinogona kubuda muchirongwa chero nguva pasina bvunzurudzo kana kutongeswa. Ndichapiwa bepa resarudzo rino. (Isayi mavara okutanga emazita enyu papeji yoga-yoga)

Runyoro rwenyu Zuva ranhasi

_____

Zita renyu (rakanyorwa)

_____

Zita reari kuitisa chirongwa Zuva ranhasi

_____

Runyoro rwemufakazi Zuva ranhasi

Initials_____

**APPENDIX 2**

**Questionnaire**: Assessment for risks factors for exposure to lead

This questionnaire seeks to act as a separation tool between people with occupational and non occupational exposure to lead. To assess the diet history of the participant and symptoms suggestive of exposure. Please assist by completing this questionnaire as accurately as possible. The information given will be treated with confidentiality and will be used for academic purposes only.

Participant ID

SECTION A: DEMOGRAPHIC QUESTIONAIRE

1. Age ( as of last birthday)
2. Sex: Male Female
3. If female- number of pregnancies in her lifetime
4. Marital status

   Single Divorced

   Married Widowed

5. Residential address
6. Length of stay at the physical address

   1 year 2 years >5 years 5-10 years

   >10years

7. Occupational history, have you ever been employed in any of the following occupations.

| | | | |
|---|---|---|---|
| Mining | ☐ | Welding | ☐ |
| Cement manufacture | ☐ | Plumbing | ☐ |
| Battery manufacture and recycling plants | ☐ | Soldering | ☐ |

SECTION B: DIET HISTORY

1. Number of meals per day ☐
2. Number of snacks per day ☐
3. Sources of water

   Tap water ☐ borehole ☐ mixed ☐
4. What do you usually eat at

| | |
|---|---|
| At around 8:00 am | |
| 10:00am | |
| 1-2pm | |
| Supper | |

5. What did you eat yesterday at these times.

| | |
|---|---|
| 08:00 | |
| 10:00 | |
| 1-2pm | |
| Supper | |

SECTION C: PHYSICAL EXAMINATION

This physical examination will act as an assessment tool, to assess signs of chronic lead exposure e.g. anemia, hypertension, neuropathies and other signs of chronic lead exposure. It will also give the general health and nutritional status of a participant. All the findings on physical examination will be treated with confidentiality and will be used for academic purposes only.

Anthropometric measurements

- Weight, height, MUAC, Head circumference, waist circumference.

General examination

JACCOL

Skin examination

CNS examination

CVS examination

Respiratory system examination

Gastro-intestinal system examination

**Mibvunzo:**

Mibvunzo iyi ichabatsira kutsvagiridza munhu anenge aine zvikangaidzo zvinogona kuti ainge ane munyu welead wakawanda.

Mibvunzo iyi ichabatsira kuonesa kuti munhu ari kuwana munyu welead kubva mubasa raanoshanda here kana kuti kubva munharaunda yakamukomberedza. Ichabatsira zvakare kunzwisisa zvakare kunzwisisa madyiro amunoita uyezve nekuona kuti hamuna zvimwe here zvimwe zvinotatidzira kuti munyu uyu wakawanda mumuviri menyu. Tinokumbira kuti mutibatsire nekupindura mibvunzo iyi makasununguka nechokwadi chenyu chose. Hapana munhu achaudzwa mhinduro dzenyu .Zvese zvaitwa nhasi zvichashandiswa mukudzidzwa kwemunyu welead.

chitupa chenyu chemuongororo

CHIKAMU CHEKUTANGA: MIBVUNZO PAMUSORO PENYU)

8. Makore enyu ekuberekwa.( achienderana nebhavhadhi renyu rekpedzisira
9. Munhui: Murume Mudzimai/mukadzi
10. Kana muri mudzimai, makaita pamuviri kangani muupenyu hwenyu.
11. Makaroora kana kuroorwa here

    Hongu

    Kwete

12. Nhamba dzepamba pamunogara.
13. Mava nemakore mangani muchigara pamba ipapo.

Rimwe ☐ Maviri ☐ pazasi pemashanu ☐ mashanu kusvika gumi ☐

kupfuura gumi ☐

14. Makamboshanda mumabasa anotevera here?

Mumughodhi ☐

Kuweredha(, kunama mapoto, kugadzira mahwindo furemu) ☐

Mufekitari inogadzirwa simende ☐

Basa rekuisa pombi dzemvura nemadhireni) ☐

mufekitari munogadzirwa kana kupiswa mabhatiri emotokari ☐

Kunobikwa nekugadzira simbi ☐

CHIKAMU CHEPIRI: MIBVUNZO YECHIKAFU

5. Munodya chikafu chakabikwa kangani pazuva? ☐

6. Munodya chikafu chisina kubikwa kangani pazuva? ☐
7. Mvura yenyu yekunwa munoiwana pai?

mupombi ☐ muchibhorani ☐ chero (chibhorani/pombi) ☐

8. Munodyei mazuva ese panguva idzi?

| | |
|---|---|
| Mangwanani | |

| | |
|---|---|
| Zuva richangoti kwirei | |
| Masikati | |
| Madeko/manheru | |

9. Nezuro makadyei panguva idzi?

| | |
|---|---|
| Mangwanani | |
| Zuva richangoti kwirei | |
| Masikati | |
| Madeko/manheru | |

CHIKAMU CHECHITATU: KUONGORORWA.

Kuongororwa kwamuchaita kuchabatsira kuonesa kuti mune here zvinoratidzira kuti makakanganiswa utano hwenyu zvakadini nemunyu unonzi lead.Munyu uyu unogona kukwidza BP, kuti ropa rishomeke, kuitisa chiveve nezvimwewo. Kuongorowa kwenyu kuchatibatsira zvakare kuziva utano hwenyu huri sei zvichienderana nemadyiro enyu. Zvese zvatichawana mukukuongororai zvichashandiswa mukuziva pamusoro pelead chete. Hakuna kumwe kwazvichashandiswa kana kupa vamwe vanhu.

**Zvichatariswa pakukuongororai.**

Kureba, huremu, MUAC, kukura kwemusoro wenyu, saizi yechiuno.

Muchatariswa kuti BP haina kukwira here, kuti ropa harisi shoma here, nechiveve.

Munogona zvakre kuzoteererwa muchipfuwa chenyu nemudumbu nekuti munyu uyu unogona kukanganisa moyo,mafemero nemudumbu

## APPENDIX 3 RESULTS FROM EACH STUDY AREA

### Table A: Goromonzi participants' characteristics.

| Participant ID number | 01 | 02 | 03 | 04 | 05 | 06 | 07 | 08 | 09 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood lead levels µg/dl | 1.1 | 0 | 0.6 | 1.8 | 2.2 | 0.03 | 1.5 | 0 | 1.1 | 1.2 | 3.4 | 1.1 | 0 | 0.2 | 0.2 |
| Urine lead levels µg/dl | 0 | 1.2 | 0.4 | 0.3 | 0.6 | 0.9 | 0 | 0.6 | 1.5 | 1.1 | 0 | 0 | 0.2 | 0.1 | 0.7 |
| Duration of stay in the area/yr | >5 | 5-10 | >5 | >5 | >5 | 5-10 | >10 | 5-10 | 1 | 5-10 | 5-10 | >10 | >10 | >10 | >10 |
| Age/yr | 29 | 27 | 30 | 35 | 19 | 39 | 63 | 62 | 26 | 39 | 28 | 40 | 18 | 40 | 52 |
| HC/cm | 56 | 57 | 55 | 55 | 56 | 56 | 56 | 58 | 57 | 59 | 57 | 57 | 58 | 54 | 55 |
| Blood Pressure/ mmHg | 120/80 | 120/80 | 110/60 | 100/60 | 120/80 | 110/80 | 120/70 | 110/80 | 120/70 | 100/60 | 100/60 | 110/70 | 120/70 | 100/70 | 120/70 |
| Protein g/l | 23 | 24.1 | 22.3 | 16.5 | 23.5 | 17.2 | 23.6 | 25.1 | 22.8 | 24.5 | 22.4 | 21.1 | 24.8 | 25.6 | 17.4 |
| Weight | 80 | 71 | 68 | 51 | 66 | 64 | 59 | 46 | 73 | 50 | 50 | 54 | 56 | 52 | 62 |
| Height | 175 | 168 | 157 | 177 | 175 | 179 | 158 | 156 | 190 | 160 | 173 | 156 | 169 | 182 | 178 |
| BMI | 26 | 25 | 28 | 16 | 22 | 20 | 24 | 19 | 20 | 20 | 17 | 22 | 20 | 16 | 20 |
| WC | 85 | 86 | 96 | 73 | 76 | 80 | 83 | 76 | 87 | 89 | 70 | 84 | 76 | 74 | 79 |

**Table B. Mabvuku/Tafara participants' characteristics.**

| Participant ID | 01 | 02 | 03 | 04 | 05 | 06 | 07 | 08 | 09 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood lead levels µg/dl | 0.9 | 0.3 | 1.4 | 1.5 | 1.0 | 0 | 92.7 | 1.1 | 1.2 | 0.5 | 0 | 0 | 1.3 | 0 | 0.6 |
| Urine lead levels µg/dl | 0 | 0.9 | 0.5 | 1.5 | 0 | 1.0 | 0 | 0.3 | 2.5 | 0.4 | 0.1 | 1.4 | 0.6 | 0.1 | 0.6 |
| Length of stay in the study area/yrs | 5-10 | >5 | >10 | 1 | 10 | 1 | 5 | >10 | >5 | 5-10 | 2 | >10 | >10 | 2 | 5-10 |
| Age/years | 27 | 45 | 45 | 30 | 48 | 28 | 50 | 71 | 21 | 22 | 26 | 55 | 37 | 18 | 26 |
| H.C/cm | 57 | 56 | 58 | 55 | 57 | 59 | 56 | 55 | 61 | 60 | 58 | 60 | 60 | 54 | 54 |
| BP mmHg | 120/70 | 120/80 | 130/80 | 120/80 | 110/70 | 130/90 | 120/80 | 160/100 | 120/80 | 100/60 | 130/90 | 120/90 | 120/70 | 120/80 | 100/80 |
| Protein g/l | 23.6 | 25.5 | 25.8 | 17.5 | 21.1 | 28.1 | 23.7 | 22.8 | 17.6 | 22.5 | 27.0 | 20.2 | 17.1 | 26.2 | 25.8 |
| Weight | 85 | 65 | 73 | 71 | 62 | 67 | 75 | 52 | 63 | 69 | 56 | 68 | 79 | 56 | 53 |
| Height | 177 | 166 | 181 | 163 | 177 | 175 | 166 | 169 | 168 | 171 | 170 | 174 | 163 | 158 | 165 |
| BMI | 27 | 24 | 22 | 28 | 20 | 22 | 27 | 18 | 22 | 24 | 19 | 22 | 30 | 22 | 19 |
| WC | 104 | 89 | 98 | 87 | 77 | 86 | 96 | 70 | 74 | 86 | 73 | 92 | 96 | 82 | 77 |

**Table C. Mbare participant characteristics**

| Participant ID | 01 | 02 | 03 | 04 | 05 | 06 | 07 | 08 | 09 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood lead levels µg/dl | 0.8 | 0.4 | 0.3 | 0.2 | 1.45 | 1.9 | 0.1 | 2 | 0.9 | 2.2 | 0.3 | 1.2 | 1.1 | 0.05 | 0.4 |
| Urine lead levels µg/dl | 1.25 | 1.25 | 0 | 0.3 | 0.6 | 0.3 | 0.6 | 0.3 | 0 | 0 | 0.4 | 1.9 | 0.8 | 0.8 | 0 |
| Length of stay in the study area/yr | >10 | >10 | >10 | >5 | >5 | 5-10 | >10 | >10 | >10 | >10 | 5-10 | >10 | 2 | 1 | >5 |
| Age | 38 | 39 | 26 | 31 | 30 | 40 | 60 | 20 | 33 | 27 | 35 | 23 | 25 | 32 | 30 |
| H.C/cm | 58 | 56 | 60 | 58 | 54 | 54 | 60 | 57 | 56 | 54 | 59 | 58 | 56 | 57 | 57 |
| BP/mmHg | 120/80 | 110/70 | 140/90 | 110/70 | 100/60 | 100/65 | 130/90 | 130/90 | 110/70 | 100/60 | 100/65 | 120/85 | 130/80 | 100/60 | 140/90 |
| Protein g/l | 25.6 | 25.2 | 25.5 | 26.3 | 20.1 | 23.3 | 25.0 | 26.7 | 26.1 | 24.9 | 23.9 | 24.0 | 22.8 | 23.9 | 22.8 |
| Weight | 70 | 53 | 95 | 61 | 57 | 71 | 100 | 86 | 79 | 46 | 74 | 59 | 74 | 59 | 81 |
| Height | 178 | 155 | 162 | 163 | 165 | 147 | 155 | 168 | 157 | 148 | 162 | 164 | 158 | 150 | 168 |
| BMI | 22 | 22 | 36 | 23 | 21 | 33 | 42 | 30 | 32 | 21 | 28 | 22 | 30 | 26 | 29 |
| WC | 80 | 83 | 106 | 88 | 76 | 103 | 105 | 108 | 106 | 75 | 86 | 76 | 86 | 83 | 87 |

**Table D. Highfield participants' characteristics**

| Participant ID | 01 | 02 | 03 | 04 | 05 | 06 | 07 | 08 | 09 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Blood lead levels/ µg/dl | 2.2 | 0.02 | 0 | 1.7 | 1.8 | 0.7 | 0 | 0.9 | 2 | 1.1 | 0.7 | 0.5 | 0.5 | 0 | 0.3 |
| Urine lead levels/ µg/dl | 0.7 | 0 | 2.4 | 0 | 0.2 | 0 | 1.9 | 0.3 | 1.3 | 0.1 | 0 | 0.1 | 0.3 | 0 | 0.1 |
| Length of stay /yrs | 2-5 | 2 | 5-10 | >10 | >10 | >10 | >10 | 5-10 | >10 | >10 | >10 | >10 | >10 | >10 | >10 |
| Age/yrs | 24 | 28 | 38 | 62 | 40 | 31 | 34 | 23 | 36 | 28 | 43 | 47 | 28 | 45 | 54 |
| H.C/cm | 63 | 55 | 55 | 56 | 59 | 57 | 58 | 55 | 56 | 56 | 56 | 55 | 54 | 52 | 50 |
| BP/mmHg | 110/70 | 130/90 | 120/80 | 130/90 | 120/70 | 140/90 | 130/90 | 120/70 | 120/80 | 100/70 | 105/70 | 120/80 | 100/60 | 120/80 | 120/80 |
| Protein g/l | 27.5 | 34.3 | 32.2 | 29.3 | 24.1 | 28.2 | 24.4 | 40.0 | 31.5 | 34.2 | 24.3 | 23.8 | 10.0 | 27.8 | 30.9 |
| Weight | 63 | 54 | 58 | 64 | 63 | 97 | 91 | 94 | 67 | 68 | 57 | 81 | 58 | 71 | 74 |
| Height | 165 | 164 | 182 | 167 | 162 | 169 | 168 | 164 | 174 | 166 | 174 | 173 | 143 | 170 | 153 |
| BMI | 23 | 20 | 18 | 23 | 24 | 34 | 32 | 35 | 22 | 25 | 19 | 27 | 28 | 40 | 32 |
| WC | 82 | 66 | 70 | 78 | 93 | 112 | 93 | 91 | 79 | 79 | 71 | 91 | 82 | 88 | 91 |

**APPENDIX 4**

## Protein Determination- Lowry Procedure

### Reagents

A. 2% Na2CO3 in 0.1 N NaOH
B. 1% NaK Tartrate in $H_2O$
C. 0.5% CuS04.5 $H_2O$ in $H_2O$
D. 48mL of A, mL of B, 1 mL of C
E. Phenol Reagent -1 part Folin-Phenol [2 N] : 1 part water

[Reagents A, B and C may be stored indefinitely]

BSA Standard – 1mg/mL

Bovine Serum Albumin: 5 mg in 5mL of water [1 µg/ µ]

Freeze 1 mL aliquots.

### Procedure

[Run triplicate determination for all samples]

1. Set up eleven sets of three 16x150 mm test tubes in rack.

2. Add BSA [0, 10, 20, 30, 40, 50, 60,70,80, 90, 100 µl] to these tubes.

3. Add 2 mL of solution D to each test tube.

4. Incubate for 10 minutes at room temperature.

5. Add 0.2 mL of dilute Folin-phenol solution to each test tube.

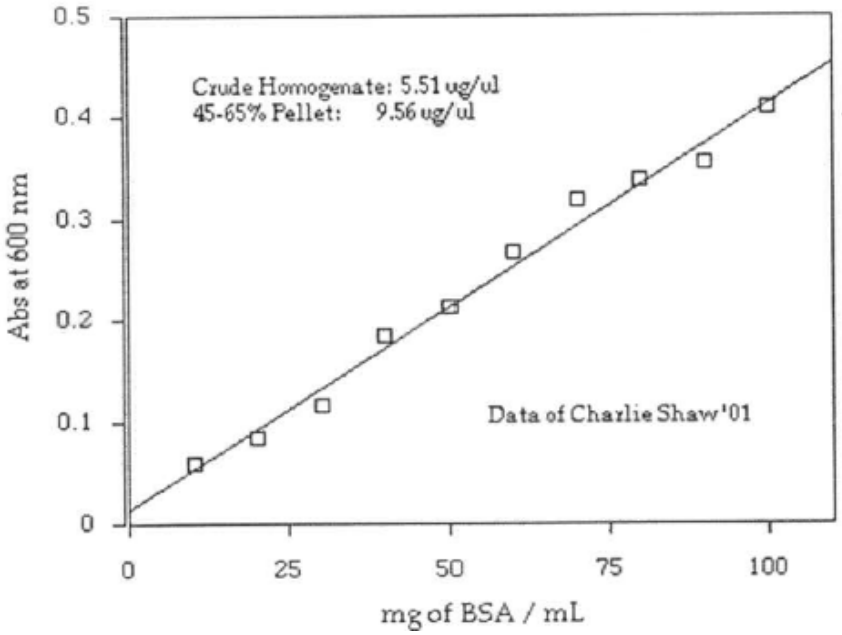
6. Vortex each tube immediately.

7. Incubate at room temperature for 30 minutes.

8. Determine absorbance of each sample at 600 nm.

9. Plot absorbance absorbance *vs* mg protein to obtain standard curve

10. Setup triplicate assays for all "unknowns"




Protein concentrations of crude homogenate and a 45-65% pellet.

**APPENDIX 5**

# CITY OF HARARE

Director of Health Services

DR STANLEY MUNGOFA
MD (Cuba) MPH (Zim)

All correspondence to be addressed to the
DIRECTOR OF HEALTH SERVICES

Ref: ----------

Your Ref: ----------

DIRECTOR OF HEALTH SERVICES
Rowan Martin Building,
Civic Centre,
Pennefather Avenue,
off Rotten Row,
Harare, Zimbabwe.
P.O. Box 596
Telephone: 753326
753330/1/2
Fax: (263-4) 752093

6 June 2013

Prof C F Nhachi
University of Zimbabwe
P O Box A 178
Avondale
**HARARE**

Dear Prof

**RE: PERMISSION TO CARRY OUT A STUDY ON LEAD EXPOSURE IN HARARE**

I acknowledge receipt of your letter dated 21 May 2013 in connection with the above.

Permission is granted for you to carry out the above study at our clinics.

You will be required to pay USD50.00 administration fee prior to commencement of the study. The fee is payable to City Health Department, 6th floor, Rowan Martin Building.

Please be also advised that it is -our institutional policy that written permission should be sought from the department prior to any presentation or publication of research findings.

For further assistance please liaise with the Sisters In Charge of the Clinics.

Yours faithfully

**DIRECTOR OF HEALTH SERVICES**
PNM/rm

c.c. Ethics Committee
S.I.C - Mbare Polyclinic
S.I.C - Highfield Polyclinic
S.I.C - Mabvuku Polyclinic

## APPENDIX 6

Telephone: 791792/791193
Telefax: (263) - 4 - 790715
E-mail: mrcz@mrcz.org.zw
Website: http://www.mrcz.org.zw

Medical Research Council of Zimbabwe
Josiah Tongogara / Mazoe Street
P. O. Box CY 573
Causeway
Harare

**APPROVAL**

**Ref: MRCZ/B/569** **25 September, 2013**

**Prudence Manyuchi**
UZ - Department of Clinical Pharmacology
College of Health Sciences
P.O. Box A 178
Avondale
**Harare**

**RE:- Blood and urine lead levels in adults attending Harare Polyclinics**

Thank you for the above titled proposal that you submitted to the Medical Research Council of Zimbabwe (MRCZ) for review. Please be advised that the Medical Research Council of Zimbabwe has **reviewed** and **approved** your application to conduct the above titled study. This is based on the following documents that were submitted to the MRCZ for review:

a) Research Protocol
b) Informed Consent Form (English and Shona)
c) Questionnaire (English and Shona)

- **APPROVAL NUMBER : MRCZ/B/569**

This number should be used on all correspondence, consent forms and documents as appropriate.

- **TYPE OF REVIEW : Expedited**
- **EFFECTIVE APPROVAL DATE : 25 September 2013**
- **EXPIRATION DATE : 24 September 2014**

After this date, this project may only continue upon renewal. For purposes of renewal, a progress report on a standard form obtainable from the MRCZ Website should be submitted three months before the expiration date for continuing review.

- **SERIOUS ADVERSE EVENT REPORTING**: All serious problems having to do with subject safety must be reported to the Institutional Ethical Review Committee (IERC) as well as the MRCZ within 3 working days using standard forms obtainable from the MRCZ Website.
- **MODIFICATIONS**: Prior MRCZ and IERC approval using standard forms obtainable from the MRCZ Website is required before implementing any changes in the Protocol (including changes in the consent documents).
- **TERMINATION OF STUDY**: On termination of a study, a report has to be submitted to the MRCZ using standard forms obtainable from the MRCZ Website.
- **QUESTIONS**: Please contact the MRCZ on Telephone No. (04) 791792, 791193 or by e-mail on mrcz@mrcz.org.zw
- **Other**
- Please be reminded to send in copies of your research results for our records as well as for Health Research Database.
- You're also encouraged to submit electronic copies of your publications in peer-reviewed journals that may emanate from this study.

Yours Faithfully

.....
**MRCZ SECRETARIAT**
**FOR CHAIRPERSON**
**MEDICAL RESEARCH COUNCIL OF ZIMBABWE**

MEDICAL RESEARCH COUNCIL OF ZIMBABWE
2013 -09- 25
APPROVED
P.O. BOX CY 573 CAUSEWAY, HARARE

**PROMOTING THE ETHICAL CONDUCT OF HEALTH RESEARCH**

**APPENDIX 7**

Parirenyatwa Group of Hospitals

# Joint Parirenyatwa Hospital And College of Health Sciences Research Ethics Committee

5th Floor College of Health Sciences Building
Telephone: +263 4 708140 Email: medirural@medsch.uz.ac.zw

University of Zimbabwe College of Health Sciences

## APPROVAL LETTER

**Date**: 21st August 2013 **JREC Ref: 177/13**

**Name of Researcher:** Dr Prudence Manyuchi
**Address:** University of Zimbabwe, Department of Clinical Pharmacology

**Re: BLOOD AND URINE LEAD LEVELS IN ADULTS IN HARARE POLYCLINICS.**

Thank you for your application for ethical review of the above mentioned research to the Joint Research Ethics Committee. Please be advised that the Joint Research Ethics Committee has reviewed and approved your application to conduct the above named study.

- **APPROVAL NUMBER:** JREC/177/13
- **APPROVAL DATE:** 21st August 2013
- **EXPIRATION DATE:** 20th August 2014
- **TYPE OF MEETING:** **Expedited Review**

This approval is based on the review and approval of the following documents that were submitted to the Joint Ethics Committee:

a) Completed application form
b) Full Study Protocol
c) Informed Consent in English and/or appropriate local language
d) Data collection tool version:

After this date the study may only continue upon renewal. For purposes of renewal please submit a completed renewal form (obtainable from the JREC office) and the following documents before the expiry date:

a. A Progress report
b. A Summary of adverse events.
c. A DSMB report

- **MODIFICATIONS:**
  Prior approval is required before implementing any changes in the protocol including changes in the informed consent.

- **TERMINATION OF STUDY:**

  On termination of the study you are required to submit a completed request for termination form and a summary of the research findings/ results.

Yours Faithfully,

**Professor MM Chidzonga**
**JREC Chairman**