LEVELS OF LEAD, CADMIUM AND CHROMIUM IN BLOOD, URINE, NAIL AND HAIR SAMPLES OF ELECTRONIC REPAIRERS IN KADUNA METROPOLIS, NIGERIA

BY

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Declaration

I declare that the work in this thesis entitled "Levels of Lead, Cadmium and Chromium in Blood, Urine, Nail and Hair Samples of Electronic Repairers in Kaduna Metropolis, Nigeria" was performed by me in the Department of Chemistry, Ahmadu Bello University, Zaria under the supervision of Prof. A. Uzairu and Dr M. S. Sallau. The information derived from the literature has been duly acknowledged in the text and a list of references provided. No part of this work has been presented for another degree or diploma at this or any other institution.

Muhammad Abubakar Lawal _____ _____

Signature Date

Certification

This thesis entitled "LEVELS OF LEAD, CADMIUM AND CHROMIUM IN BLOOD, URINE, NAIL AND HAIR SAMPLES OF ELECTRONIC REPAIRERS IN KADUNA METROPOLIS, NIGERIA" by MUHAMMAD ABUBAKAR LAWAL meets the regulations governing the award of the degree of Master of Science Degree in Analytical Chemistry of the Ahmadu Bello University, and is approved for its contribution to knowledge and literary presentation.

Prof. A. Uzairu Chairman, Supervisory Committee	Signature	Date
Dr M. S. Sallau Member, Supervisory Committee	Signature	Date
Prof. V. O. Ajibola Head of Department	Signature	Date
Prof. A. A. Joshua Dean, School of Postgraduate Studies	Signature	Date

Dedication

This research work is dedicated to my parents; Alhaji Abubakar Lawal and Hajiya Khadija Saidu who put me on this path of knowledge so that I may become knowledgeable and impart others with knowledge and to my lovely wife for her patience and support throughout the work.

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Finally, my profound gratitude goes to the electronic repairers who volunteered to be part of this work, without whose cooperation this work may have been impossible.

Abstract

The Levels of lead (Pb), cadmium (Cd) and chromium (Cr) in blood, urine, nail and hair samples of electronic repairers in Kaduna-Nigeria were assessed using Atomic Absorption Spectrophotometry (AAS). The mean blood concentrations of Pb, Cd and Cr in the subjects were 29.33 ± 4.80, 7.78 ± 10.57 and 24.78 ± 21.77 µg/dL, respectively. The mean urine concentrations of Pb, Cd and Cr were 24.18 ± 2.98, 6.81 ± 10.05 and 14.78 ± 14.20 µg/dL, respectively. Mean nail metal values of 37.13 ± 4.08, 1.00 ± 1.21 and 18.49 ± 12.71 µg/g were obtained for Pb, Cd and Cr, respectively while mean hair metal values of 39.41 ± 5.63, 1.09 ± 1.14 and 19.13 ± 11.61 µg/g for Pb, Cd and Cr, respectively. Positive Pearson correlation coefficients were also observed between Pb/Cd, Pb/Cr and Cd/Cr in all samples and they indicate the metals are likely from same pollution source. The mean concentrations of the metals in all samples were higher than the WHO, ILO and ACGIH standards, implying the repairers are occupationally exposed and are subject to serious health concerns. Smoking was suspected to significantly affect the concentrations of these metals. The level of education, use of safety devices, period of exposure (length of period in the job), the type of electronics and the age of the electronic repairers were also suspected to remarkably affect the concentrations of the metals. However, the sources of food eaten at work were found to have no significant effects on the levels of the metals present.

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GLOSSARY/ ABBREVIATION

ANOVA - Analysis of variance

SD - Standard deviation

WHO - World Health Organization

Ppm - parts per million same as milligram per cubic decimeter

Pb - Lead

Cd - Cadmium

Cr - Chromium

MESS - Multi-element Standard Solution

ILO - International Labour Organization

BDL - Beyond Detection Limit

ACGIH - American Conference of Governmental Industrial Hygienists

CDC - Centre for Disease Control and Prevention

AAS - Atomic Absorption Spectroscopy

ICP-AES - Inductively Coupled Plasma Atomic Emission Spectroscopy

ICP-MS - Inductively Coupled Plasma Mass Spectroscopy

EFSA - European Food Safety Authority

CHAPTER ONE

INTRODUCTION

1.1 Background of the Study

People engage in diverse occupations in order to achieve some set goals; the primary of which usually is to earn a living. These occupations may range from unskilled, semi skilled to skilled occupations. A vast population of people fall under the semi-skilled category especially in developing countries of Asia and Africa. The dominant mode of training is through apprenticeship, a system of training a new generation of practitioners of a structure competency based set of skills. Most of the training here is done while working for an employer who helps the apprentices learn their trade or profession, in exchange for their continuing labour for an agreed period after they have achieved measurable competency. Examples of occupations under this category are electronic repairers, mechanics, carpenters, painters, welders, and so forth. Most people under this category are either illiterate or semi literate usually with little or no formal training or education. In the developing world, employment in the informal sector may reach 70 percent, with the contribution to the gross domestic product (GDP) ranging from 10 to 60 percent (ILO, 2002)

Virtually all occupations have some level of hazards inherent in them. Occupational health has been an issue of cognisance since the 1950 with a joint committee of the International Labour Organization (ILO) and the World Health Organization (WHO) sharing a common definition of occupational health at its first session in 1950.

According to the World Health Organization (WHO) report in 1998, there were about 250 million cases of work related injuries through the year worldwide (WHO, 1998). More recent reports estimates work related diseases at 160 million every year while more than 2.2 million occupational deaths and 250 million industrial accidents occur every year worldwide (Bankole and Ibrahim, 2012; ILO, 2011). Work accidents or occupational accidents constitute an important cause of sickness and absence from work in industries particularly in developing countries (Asogwa, 1978), thereby causing huge losses in revenue for the government.

These hazards could be as a result of materials, equipment and appliances frequently used or some form of physical energy demanding stage or stages or the working environment. These hazards could be electrical in nature, chemical, physical or radioactive.

Electronic repairers like their counterparts in other occupations are exposed to various occupational hazards which could be classified as follows:

- Electrical hazards: electrical shock, electrocution, and so forth
- Chemical hazards: lead fumes and dust, lead ingestion, fumes from heated solder constituents, fumes from decomposition products from heating of oil, paints or coatings present on the surfaces heated during soldering, fumes from fluxes, gases and vapour from cleaning agents.
- Physical hazards: burns and fire.

- Risk of radioactivity: radioactivity from some USA-made common voltage regulator tubes which had radioactive isotopes added, apparently to achieve more stable operation.

Both electronic and hard soldering present potential exposure issues via airborne, skin contact and hand to mouth routes, from both the products used or by- products of the soldering process. There are also physical hazards from heat and in some cases from open flame (Manton and Malloy, 1983). There are virtually no published data on the statistics of electronic repairers and the cases of occupational accidents amongst them in Nigeria.

According to the International Labour Organization, approximately 35 metals are of major concern in regard to occupational exposure. Two thirds of them can cause health hazards if not properly handled and may result in well-defined toxic effects in humans. Some metals are not poisonous in small amounts and may, on the contrary, be necessary for good health. On the other hands, some metals, even in small doses, may cause both immediate and chronic poisoning. Damage may involve disturbances of the blood composition or nervous system, or injury to the liver or kidneys. Long-term exposure to certain metal compounds may cause cancer. Allergic reactions may result from repeated long term contact with some metals and metal compounds.

Mercury, lead, cadmium, nickel, chromium, manganese, arsenic, antimony, zinc, copper, cobalt, vanadium and beryllium are used in industries and are known to cause adverse health effects both as the metal and as metallic compounds. Cadmium and chromium [VI] are listed by the International Agency for Research on Cancer (IARC) as carcinogenic to humans while lead is classified as possibly carcinogenic to humans.

1.2 The Research Problem

Occupations are a major source of exposure to heavy metals to adults; these occupations are an integral part of human lives since survival is heavily dependent on them. Thirty-eight per cent of the male working population in Nigeria are engaged in agriculture (20% of women) and 56% of the female working population are engaged in sales and service occupations (19% of men). Twenty-one per cent of men and 9% of women are engaged in skilled manual trades and 16% of men and 8% of women in professional and technical jobs (NPC and ORC Macro, 2004). No matter how small the fraction of these metals that pose a threat at the work place, it becomes obviously dangerous considering that most people normally engage in such occupations for a substantial part of their lives (usually to old age) and spend more than 8 hours a day at work especially in poor developing countries. Common symptoms of patients frequently reported at hospitals in Nigeria may be as a result of exposure to heavy metals, often scrutiny for heavy metals exposure is not usually considered.

Electronic repairers are exposed to lead (Pb) from solder fumes and dust, cadmium (Cd) and chromium (Cr) from batteries, coatings, pigments etc. These metals could pose serious health problems over a long period and could indirectly affect children at home as 'take home metals' on clothing, bags e.t.c. The level of adherence to safety measures at work due primarily to ignorance and poverty even makes the problem a bigger one. Poor statistics available on the level of these metals in electronic repairers in Nigeria does not make glaring the impending problem in the job, making also awareness about the problem low or nonexistent.

1.3 Justification for the Study

Exposure to lead, cadmium and chromium at work is an issue of concern especially in developing countries like Nigeria where the level of education and awareness about hazardous metal exposure is low and poverty is high (98% of adults and 99% of children affected by exposure to lead live in low- and middle-income countries (WHO, 2009)). Many research reports have shown serious exposure of workers in artisan professions to heavy metals. Welders have been reported to be exposed to heavy metals (Sabitu *et al.*, 2009), auto mechanics (Babalola *et al.*, 2005 and Dioka *et al*., 2004), painters (Orisakwe *et al.*, 2007), drivers (Fatoki and Ayoade, 1996) e.t.c have also been reported to be exposed to heavy metals.

With a lot of researches conducted on different occupations to assess hazards and exposure, there are no reported studies on occupational exposure of electronic artisans, hence this study was conceived. The study is focused at assessing the level of exposure of the repairers to lead (Pb), cadmium (Cd), chromium (Cr) and habits affecting the level of exposure.

Nigeria, just as most other developing countries lacks accurate and up to date statistical figures on occupational exposure to lead, cadmium and chromium. There is the need to provide such statistics to facilitate analyses on occupational health in Nigeria and would come handy in the formulation and/or amendment of policies affecting such occupations.

1.4 The Research Questions

The questions that could arise from this study are;

1. What are the levels of lead, Cadmium and Chromium exposures in electronic repairers in the study area?
2. Are the levels of the metals higher than allowed?
3. What is the relationship between the level of exposure of the repairers with their social and personal habits?
4. What is the correlation between the metals and are the metals likely from same pollution source?
5. What nature of sensitization should be undertaken to educate electronic repairers of the hazards in the profession?

1.5 Aim and Objectives of the Study

This study is aimed at assessing the levels of lead, cadmium and chromium in blood, urine, nail and hair samples of electronic repairers in Kaduna metropolis, Nigeria, with a view to comparing the results obtained with the international allowed limits for lead (Pb), cadmium (Cd) and chromium (Cr) in the body. This shall be achieved through the following objectives:

i) To collect blood, urine, nail and hair samples from electronic repairers and to analyze the samples collected for Pb, Cd and Cr in order to determine levels and therefore exposure.

ii) To correlate the levels of the metals in the samples and ascertain whether the metals are likely from same source.

iii) To compare the concentrations of the metals with WHO and ILO acceptable limits to determine whether the repairers are at risk or not.

iv) Assess the effect of factors like smoking, drinking, age, length of period in the job, the brand of electronics specialized in, adherence to safe practice and level of formal education on the concentrations of the metals in the repairers.

1.6 The Research Hypotheses

The study was guided by the following hypotheses:

a) The null hypothesis (H_0) which states that:

i) The levels of the metals (Pb, Cd and Cr) in the system of the electronic repairers in Kaduna metropolis, Nigeria are within the WHO acceptable levels and the repairers are therefore, not at risk.

ii) There is no correlation between the metals in the system of the electronic repairers and are likely not from same pollution source.

iii) Personal and social habits do not significantly affect the levels of the metals.

b) The alternate hypothesis (H_1), which states that:

i) The levels of the metals (Pb, Cd and Cr) in the system of the electronic repairers in Kaduna metropolis, Nigeria exceed the WHO acceptable levels and the repairers are therefore at risk.

ii) There is a positive correlation between the metals in the system of the electronic repairers; the metals are likely from same pollution source.

iii) Personal and social habits significantly affect the levels of the metals.

1.7 Significance of the Study

The study would provide data on occupational exposure of electronic repairers to heavy metals. The result of the research would further guide the government in policy making with respect to occupational issues. The relationship between the level of occupational hygiene of the repairers and the levels of the metals would also give an indication of the exposure risk of the family members in form of 'take home' metals

1.8 Scope and Limitation of the Study

This study is aimed at assessing the levels of lead, cadmium and chromium exposures in electronic repairers in Kaduna metropolis, Nigeria. Cooperation from

electronic repairers to willingly participate in the research was very low; most of them have this superstitious believe of anything that has to do with blood is evil and hence were not ready to submit themselves for collection of samples. Environmental conditions were not taken into account during the research work, as the conditions would have little or no effect on the microenvironment of the repairers (working area).

CHAPTER TWO

LITERATURE REVIEW

2.1 Occupational Health

Occupational health have been an issue of cognisance since the 1950s, Hippocrates, in the fourth century B.C. recognized the toxic properties of lead and recorded a series of attack of lead colic in miners. About 100 years later, Galen accurately described the pathology of Lead poisoning (Waldron, 1989). The evolution of modern industrial safety movement had its roots in England, at the dawn of the 18th century industrial revolution era. By 1750, machines had been invented and mining and manufacturing industries became established. Men, women and children were employed to work in factories under very terrible conditions. They worked for many hours under crowded conditions and with little or no food or good water to drink (Entwistle, 1983). The progress and growth of occupational health has not been linear; occupational health has been influenced primarily by events outside the field, namely social movements and changes in the delivery of health care and perception of health as is evident from its history in the US and many industrialized countries (Ashford and Caldart, 1996, Emmett, 1997, Cullen, 1999, and Hunter, 1978).

Workers around the world—despite vast differences in their physical, social, economic, and political environments—face virtually the same kinds of workplace hazards. These hazards are traditionally categorized into four broad types: chemical, biological, physical, and psychosocial (Rosenstock *et al*., 2006). What emerges from our incomplete knowledge of their risk, however, is that more than 80 percent of the world's workforce that

resides in the developing world disproportionately shares in the global burden of occupational disease and injury. Several classic occupational diseases, such as silicosis and lead poisoning, that have been substantially eliminated in industrialized countries remain endemic elsewhere in the world. Whether this high and preventable burden of ill health faced by workers in the developing world is the result of ignorance, inattention, or intent, compelling evidence indicates that work-related health conditions could be substantially reduced, often at modest cost (Rosenstock *et al*., 2006).

2.2 Occupational Health and Exposure to Heavy Metals in Developing Countries

In developing countries, the growth of occupational health has been hindered by lack of governmental interest in occupational health, poor data and data collection systems, and weak enforcement of health and safety regulations as has been reported in Nigeria (Asuzu, 1996), South Africa (Joubert, 2002), Bangladesh (Ahasan *et al.*, 1999) and Thailand (Yingratanasuk *et al.,* 1998). Inability by any country or organization to provide occupational health and safety services to all categories of workers is in contravention to the International Labour Organization (ILO) convention 161 and recommendation 171 of 1985, which requires that occupational health and safety (OHS) services should be developed for workers in all branches of economic activities and all undertakings. The availability of occupational health and services will protect workers from the effects of occupational hazards prevalent in almost all occupations.

Numerous researches have been undertaken to assess the levels of lead, cadmium and chromium exposures in workers in various occupations. The blood lead levels of apprentices working in lead-related industries were analyzed in Bursa, Turkey to evaluate

the influence of such occupational exposure; the apprentices were receiving training in engineering, welding, electrical, and dye curricula at Bursa Vocational Education Centre. The results show that occupational exposure increases the blood lead levels of apprentices (Pala *et al*., 2009). Neuropsychiatry has been linked to chronic occupational lead poisoning among workers of a car battery industry in Iran, blood lead concentration of 398.95 ±177.40 µg/L and urine lead concentration 83.67±49.78 µg/L were reported in the workers (Kianoush *et al.*, 2013). Lead exposure has been linked with increased risk of abnormal sperm morphology in lead-exposed workers from a South African lead acid battery facility (Robins *et al.*, 1997). A related study on lead smelters also show current blood lead concentrations of 40 micrograms/dl or more had an increased risk of below normal sperm concentration (odds ratio (OR) 8.2, 95% confidence interval (95% CI) 1.2-57.9) and total sperm count (OR 2.6, 95% CI 0.4-15.7), based on World Health Organization standards. Sperm concentration, total sperm count, and total motile sperm count have been found to be inversely related to measures (concentrations) of long term lead exposure, independent of current lead exposure. No association was found between lead exposure and measures of sperm motility, sperm morphology, or serum concentrations of reproductive hormones (Alexander *et al.*, 1996).

Cadmium exposure during abrasive blasting has been studied (Conroy *et al.*, 1996). The report shows that airborne levels in the containment exceeded the Occupational Safety and Health Administration's permissible exposure limits by factors of 3.1. Workers exposed to cadmium in an alkaline storage battery manufacturing and polyvinyl chloride (PVC) compounding plants have been examined. The urine cadmium excretion in these workers

exposed to cadmium in the alkaline storage battery manufacturing and PVC compounding plants increased with working time and there was also a good correlation between the blood and urine cadmium levels (Chan *et al.*, 1982, Hassler *et al.*, 1983 and Dewell, 1994). A follow up study by Chan *et al.*, (1988) on the lung function status of workers in the cadmium–nickel battery factory showed a mild restrictive effect and the blood and urine cadmium concentrations were significantly lower than previously reported but consistent with the decreased cadmium-in-air levels observed. The rate of respiratory symptoms also decreased particularly in the workers who were no longer exposed to cadmium. Similar findings were also established by McDiarmid *et al*., (1997) on a follow-up study of biological monitoring results in cadmium workers removed from exposure. Findings by Marek *et al.* (1982) suggest that the cadmium concentration in the blood and urine after the body stores it to saturation is a better informant of current exposure (bioavailability) than of cadmium stored in the body (bioaccumulation). Considering dose–response relationship, an increase of low molecular protein excretion in urine can be seen in one-tenth of the cases of cadmium in urine, amounting to 10–15 μg/g creatinine and cadmium in blood years of about 300–400 (Jakubowski *et al.*, 1987).

The content of chromium was determined in the organs of six chromate workers who had worked in a chromate chemical manufacturing plant, had been exposed to a high amount of chromium for over 10 years and had died of lung carcinoma (Kishi *et al*., 1987 and Tsuneta *et al.*, 1980). They reported that it was apparent that the metal remained in the lungs long after exposure to chromate had ceased. The concentration of chromium in the upper lobes is usually significantly higher than that in the lower lobes, suggesting regional

differences either in clearance from or deposition in the lung. Biological monitoring of hexavalent chromium and serum levels of the senescence biomarker Apolipoprotein J/Clusterin (ApoJ/CLU) in Welders in Greece was carried out, the result from the preliminary study gives evidence that the human ApoJ/CLU gene is responsive to the acute oxidative stress induced by heavy metals such as hexavalent chromium (Alexopoulos *et al.*, 2008). Clinical findings of irritation among chromium chemical production workers, have been established, skin irritation and ulcerated skin were reported in 15% and 32%, respectively, of workers first employed between 1950 and 1974 at a chromate production plant (Gibb *et al.*, 2000).

2.3 Occupational Health and Exposure to Heavy Metals in Nigeria

In Nigeria, the factories decree of 1987 was a landmark in legislation on occupation health. A substantial revision of the colonial legislation, factories act 1958, the 1987 decree, changed the definition in providing oversight for the numerous small-scale enterprise that engaged majority of the workforce. Enforcement of legislation is carried out by the factory inspectorate of the Ministry of Labour. This ministry produced a national policy on safety and health which details the responsibilities of the employers, workers, manufacturers and government agencies in the maintenance of the health and safety of workers (Peter, 2013). The Factory Act generally covers the hygiene and safety requirement of work environment. It however focuses purely on factory workplace, and only by its extension could it be applied to non- factory workplace. Also, the health issues addressed in the Act are merely factory hygiene issues that do not take cognizance of serious health issues that may arise in the workplace. In the Workman Compensation Act, where various degree of accidents could occur in the workplace and the compensation for them were identified, no mention or

description of such was made concerning occupational diseases except that compensation would be made for them as if they were accidents arising from the course of work. However, as stated earlier, the Workman Compensation Act has been replaced by the Employee Compensation Act. The new Act deals with every aspect of the contract of employment entered between employees and employers. Issues therein include: wages, working hours, holiday, compensation during illness or infirmity, and suspensions among others (Peter, 2013).

The cases of work accidents reported in Nigeria were 82 in 2001, 62 in 2002, the figures progressively increased until it peaked in 2005 with 120 cases reported and dropped by half in 2006 with 57 cases (ILO, 2006). These figures are much smaller compared to the reality on ground as many cases of work accidents are not ever documented or reported (Omokhodion, 2009). Work accidents or occupational accidents constitute an important cause of sickness and absence from work in industries particularly in developing countries (Asogwa, 1978). The Factories Act Cap 126, Laws of the Federation of Nigeria, 1990 is the legislation for the enforcement of safety and health standards in Nigerian workplaces, however not much has been achieved due to poor monitoring and implementation. Experts in the field of occupational health have carried researches and assessments over the years to collate data that would be helpful in policy formulation, implementation and assessment.

Nigerian artisanal fisheries have been reported to have one of the highest occupational fatality rate; estimated to be between 999 and 3,329 per 100,000 fishermen (Udolisa *et al*., 2013). The research identified piracy, boundary disputes between fishing communities, unemployment and societal low value for human life as the reasons for the high fatality rate. Patchy awareness and poor to average knowledge of preventive measures

against work related zoonotic diseases have been attributed to be responsible for occupational work related diseases in workers of animal related occupations in Abeokuta southwestern Nigeria (Awosile *et al.*, 2013; Ajetomobi *et al.*, 2010; Omokhodion and Adebayo, 2012). The design and layout of offices/workstations and access to equipment has also been discovered as a source of occupational hazard for office workers. This is said to reduce efficiency and productivity of the workers (Ijadunola *et al.*, 2003). The lack of use of protective devices among welders in Benin City in Edo-Nigeria have been linked to these most prevalent health complaints; arc eye injuries, foreign bodies in the eyes, back/waist pain, metal fume fever and cuts/injuries from sharp metals (Isah and Okojie, 2006). Low level of education, awareness, and the low utilization of safety devices and measures have been reported in nurses and welders in Abeokuta-Ogun state and Kaduna state respectively in Nigeria (Amosu, 2011 and Sabitu, *et al*., 2009). Similarly, musculoskeletal disorders have been reported among nurses in Ibadan (Tinubu *et al.*, 2010). Low awareness, poor safety measures and the perceived influence of health education among factory workers and sawmill workers have also been reported (Bankole and Ibrahim, 2012 and Osagbemi *et al.*, 2010). The need for further studies relating the toxicity of these metals and their health impacts on cement factory workers have been reported by Ogunbileje *et al.*, (2013). Results from assessment of injuries in small scale sawmill industry of southwestern Nigeria indicated that mill workers suffer highest injury rate of 83% while moving logs to mill from log yard or stack while timber stacking accident is 36%. Other accident cases recorded include; log transport to the mill is 22%, milling operation 41% and maintenance accident 38%. Injuries occurring to body area include upper limb injuries (Neck and head, arm, wrist, hand and shoulder) 68%, back and lower

back injuries 58%, and less prominent lower limb (Legs, knees and ankle) injuries at 13% (Segun and Yahaya, 2010).

Recent lead poisoning outbreaks in Senegal and Nigeria illustrate the potential severity of hotspot exposures. According to the WHO, 18 children died in Thiaroye sur Mer, Senegal, during November 2007-March 2008 from encephalopathy caused by severe lead intoxication as a result of environmental contamination from informal used lead acid battery (ULAB) recycling. Blood lead levels (BLLs) of 50 children (three months-19 years old) included in the WHO investigation ranged from 39.8-613.9 μg/dL, with a mean (SD) of 129.5 (92.4) μg/dL (Haefliger *et al.*, 2009). In Zamfara State, Nigeria, 200 children have reportedly died from lead intoxication since March 2010 and an estimated 18,000 children and adults have been affected by widespread lead contamination resulting from the informal extraction of gold from lead-bearing ore (WHO, 2010; Adeniyi and Anetor 1999).

Lead assay in blood of occupationally and non-occupationally exposed donors have been undertaken by Fatoki and Ayoade in Nigeria. The blood lead levels of non-occupationally exposed group were found to be low, and generally lower than the quoted threshold values. However, the lead levels were higher in blood of exposed group and most of the values exceed the quoted threshold values although lower than critical lead value that could cause poisoning. The mean blood levels in painters, drivers and motor mechanics are 30.4 μg/100 ml, 37–39 μg/100 ml and 36.7 μg/100 ml, respectively (Fatoki and Ayoade, 1996). Another research on auto mechanics in Abeokuta-Nigeria carried out to assess the levels of lead in blood, scalp hair and fingernail samples reported the mean blood lead, hair lead and fingernail lead to be 48.50 ± 9.08 μg/dL, 17.75 ± 5.16 μg/g and 5.92 ±

3.30 μg/g respectively (Babalola *et al.,* 2005). Results by Orisakwe *et al*. (2007) on 25 occupationally exposed artisans in Anambra State- Nigeria showed that occupational exposure of humans to paints increased the blood lead, serum cadmium and nickel, when compared with non-paint factory workers. Similar research on mechanics in Nnewi-Nigeria was carried out by Dioka *et al.* (2004) to assess some toxicological effects of occupational exposure to petroleum products (especially petrol which contains tetraethyl lead) amongst twenty five occupationally exposed artisan mechanics. The results show a significantly increased blood lead (59.6 ± 15.9 microg/dL). Blood lead levels range of 18.5 ± 3.5 - 69.7 ± 13.2 mg/L (18.5 ± 3.5 - 69.7 ± 13.2 μg/dL) have been reported in occupationally exposed workers in Osogbo city- Nigeria. Significant increases in mean plasma concentration of creatinine (84.9 ± 13.6-97.4 ± 28.7 μmol/L), sodium (131.9 ± 3.9-134.8 ± 3.7 mmol/L) and chloride (98.2 ± 4.0-100.4 ± 3.9 mmol/L) were also observed. These renal indices indicate increased susceptibility to renal failure (Onuegbu *et al.*, 2011). Occupationally lead exposed subjects in Port Harcourt- Nigeria were reported by Alasia *et al.,* (2010) to have higher mean blood lead 50.37 ± 24.58 μg/dI, than controls 41.40 ± 26.85 μg/dL ($p = 0.008$). The mean values of serum urea, creatinine and uric acid were significantly higher in study subjects compared to controls. Njoku and Ibe (2009) reported high accumulation of heavy metals in electronic technicians, in a study carried out in Imo State-Nigeria.

2.4 Heavy Metals in the Work Place

2.4.1 Lead (Pb) in the workplace

Lead is a naturally occurring element that people have used almost since the beginning of civilization. Lead is bluish-gray in colour and has no characteristic taste or smell. Lead has many different uses. The major uses of lead and its compounds are storage batteries, pigments, ammunition, solders, plumbing, cable covering, bearings, and caulking. It is also used to attenuate sound waves, atomic radiation, and mechanical vibration. A variety of human activities have spread widely throughout the environment, such as leaded gasoline which in the mid 20^{th} century became a significant cause of lead poisoning, especially in children (Kersten *et al*., 2003). Efforts are still being made to limit the use of lead containing products to minimize harmful effects on people and animals.

Most adult exposures to lead are occupational and occur in lead related industries such as lead soldering in the electronic industry, painters and remodelers, battery manufacturing and recycling, automotive radiator manufacturing and repair, casting and machining lead, brass, bronze, pewter, white metal, metal plating operations, gun firing ranges, salvaging and recycling scrap metals, manufacturing leaded glass or crystal, auto-body repair (Clausen and Rastogi, 1977).

Lead exposure is arguably the oldest known occupational health hazard with the potential for causing irreversible health effects, including hypotension, central nervous system problems, anaemia, and diminished hearing acuity before it is clinically recognised (Staudinger and Roth, 1998). The evaluation of occupational lead exposure has been topical especially in developing countries (Khan *et al*., 2009). Exposure routes for lead have been

discovered, predominant among these are; occupational contamination of the environment as a result of industrial use of lead such as in lead-acid batteries, lead wire or pipes, metal recycling or contact with lead in air, house hold dust, soil, water and commercial products (Rossi, 2008).

The probability of lead poisoning increases when workers inhale lead dust and lead fumes or when they eat, drink or smoke in or near contaminated areas. Between 0.5 and 1.5 million workers are exposed to lead in the work place. Workers can accidentally expose family members by bringing lead dust on their skin, shoes and clothing. Lead brought home in this way is referred to as 'take home lead' (Gerson, *et al.,* 1996).

According to estimates made by the National Institute of Occupational Safety and Health (NIOSH), more than 3 million workers in the United States alone are potentially exposed to lead in the work place (Staudinger and Roth, 1998). Human exposure to lead is estimated to account for 143 000 deaths every year and 0.6% of the global burden of disease (WHO, 2009).

When lead gets into the body' the main body compartments that store lead are blood, soft tissues (brain, spleen, kidneys, liver, lungs, and so forth) and bone. The half life of lead in these tissues is measured in weeks for blood, months for soft tissues and years for bone (Karri *et al*., 2008). Lead in bones, teeth, hair and nails is bound tightly and not available to other tissues and is generally thought not to be harmful (Rubin and Strayer, 2008). About 94% of absorbed lead in adults is deposited in the bones and teeth. As a result lead exposure takes place over years; clearance is much slower, partly due to the re-release of lead from bone. A portion of lead in the body is stored in the brain, spleen, kidneys,

liver, lungs, and so forth and can be removed from the body very slowly through mainly urine. Smaller amounts of lead are also eliminated through the faeces, and very small amounts in hairs, nails and sweat (Kosnett, 2006). This explains why some of them are used as bio indicators of lead exposure. Adults with certain risk factors might be more susceptible to toxicity; these include calcium and iron deficiencies, old age, disease of organs targeted by lead (for example the brain, the kidneys), and possibly genetic susceptibility (Kosnett *et al.*, 2007) . Differences in vulnerability to lead-induced neurological damage between males and females have also been found, but some studies have found males to be at greater risk, while others have found females to be (Bellinger, 2004).

The estimated half life of lead in bone is 20 to 30 years and the bone can re-introduce lead into the blood stream long after the initial exposure is gone. The half life of lead in the blood in men is about 40 days, but it may be longer in children and pregnant women whose bones are undergoing remodelling which allows the lead to be continuously re-introduced into the blood stream (Pearson and Schonfeld, 2003).

Lead taken internally in any of its forms is highly toxic. Lead has been shown to be absorbed by the skin even from a short contact (Filon *et al*., 2006). Decontamination of skin exposed to lead powder or dust done after 30 minutes of exposure did not decrease skin absorption occurring over 24 hours (Filon *et al.*, 2006). The absorption of metal powders generally through intact (undamaged) and damaged skin is possible (Filon *et al*., 2009). Generally speaking invitro percutaneous absorption of metal compounds has also been reported to occur (Larese *et al*., 2007).

The effects are usually felt after it has accumulated in the body over a period of time. It is rapidly absorbed into the blood stream and is believed to have adverse effects on the central nervous system, the cardiovascular system, kidneys, and the immune system (Bergeson and Lynn, 2008; Patrick, 2006). Epidemiology studies have suggested that lead is immunosuppressive (Goyer and Clarkson, 2001). A study in Finland provided support for the existence of a connection between occupational exposure of the father to lead and birth defects in children (Sallmen *et al.*, 1992), decreased sperm count in worker exposed to lead, as well as decreased motility and lifespan of sperm has also been reported to have direct relation to the level of lead in the blood (Xuezhi *et al.*, 1992).

Lead interferes with a variety of body processes and is toxic to many organs and tissues including the heart, bones, intestines, kidneys and reproductive nervous systems. It interferes with the development of the nervous system and is therefore particularly toxic to children causing potentially permanent learning and behavioural disorders. Lead pollution has been stated as a risk factor for cardiovascular disease in Asian developing countries (Pak, 2012), brain damage has also been reported to occur in adults after massive exposure (Sahu *et al*., 2008). Signs and symptoms of lead poisoning vary usually depending on the individual and the duration of exposure (Karri *et al.*, 2008 and Kosnett, 2005). Early symptoms of lead poisoning in adults are commonly non specific and include depression, loss of appetite, forgetfulness, fatigue, irritability, intermittent abdominal pain, headache, nausea, diarrhoea, constipation and muscle pain. These symptoms may be due damage to the central nervous system (Goyer and Clarkson, 2001). Early signs could include malaise, fatigue, decreased libido and problems with sleep. However, these symptoms and signs vary based on the period of exposure from the chronic exposure to acute symptoms from

brief, intense exposure. Principally, the amount of lead in the blood and tissues, as well as the duration of exposure determines the toxicity. Blood lead levels are usually measured in micro grams per decilitre (µg/dL) and micrograms per 100 g of whole blood (µg/100 g whole blood) which is about equal to the first measurement µg/dL.

The US centres for disease control and prevention (CDC) and the World Health Organization state that a blood lead level of 10 µg/dL or above is a cause for concern, however, lead may impair development and have harmful health effects even at lower levels and there is no known safe exposure level (Rossi, 2008 and Barbosa *et al*., 2005).

2.4.2 Cadmium (Cd) in the workplace

The principal uses of cadmium are in batteries, as a coating and plating agent for inhibiting corrosion, in pigments, as a heat stabilizer in plastics and synthetic products, and as a component in brazing of low melting alloys. Cadmium is a component of some solder metals and baths used for electroplating. Many pigments contain cadmium compounds, often to make yellow or red colours.

In the occupational setting, absorption of cadmium occurs chiefly through inhalation. However, gastrointestinal absorption may significantly contribute to the internal dose of cadmium. 5%–35% of inhaled cadmium is absorbed into the blood depending on its form, site of deposition, and particle size. If the cadmium penetrates to the alveoli, it is estimated that there is 100% absorption into the blood (Satarug and Moore, 2004). One important characteristic of cadmium is its long biological half-life in the body, exceeding 10 years. In tissues, cadmium is mainly bound to metallothionein. In blood, it is mainly

bound to red blood cells (Hoet *et al.*, 2011). The human body lacks the ability to expel this metal from its system. Practically all the cadmium taken in during the lifetime is stored in the kidneys, which gradually results in reduced kidney function. Cadmium compounds can cause skin lesions, decrease in heart contractility, blood vessel damage and injuries of the nervous system, kidney and liver. Cadmium can be particularly very harmful for those who are occupationally exposed to it and who are also cigarette smokers since one cigarette contains 1 to 2 µg of cadmium, 10% of which is inhaled when the cigarette is smoked (Goyer and Clarkson, 2001). Cadmium compounds can also produce skin and lung tumors in humans (Jenne *et al*., 2011). Co-exposure to cadmium, cobalt and lead can lead to multiplicative effects of the metals (Hengstler *et al.*, 2003). The normal levels of cadmium in blood and in urine are usually below 0.5 µg/dL and 2 µg/g creatinine respectively. They are higher in smokers than in nonsmokers. In workers chronically exposed to cadmium, the risk of renal impairment is negligible when urinary cadmium levels never exceed 10 µg/g creatinine. An accumulation of cadmium in the body which would lead to a urinary excretion exceeding this level should be prevented. However, some data suggest that certain renal markers (whose health significance is still unknown) may become abnormal for urinary cadmium values between 3 and 5 µg/g creatinine, so it seems reasonable to propose a lower biological limit value of 5 µg/g creatinine. For blood, a biological limit of 0.5 µg/100 ml has been proposed for long-term exposure. It is possible however; that in the case of the general population exposed to cadmium via food or tobacco or in the elderly, who normally suffer a decline of renal function, the critical level in the renal cortex may be lower (Hoet *et al.*, 2011). The American conference of Governmental and Industrial Hygienists (ACGIH) recommends a blood cadmium level of 5 µg/L and 5 µg/g in urine.

A balanced diet can reduce the amount of cadmium taken into the body from food and drink. Animal studies suggest that more cadmium is absorbed into the body if the diet is low in calcium, protein, or iron, or is high in fat (Min, *et al*., 2008; Washko and Cousins, 1977).

2.4.3 Chromium (Cr) in the workplace

Chromium, especially in salt and as chromic acid, is widely used in different types of industries. Chromium is part of stainless steel and several other alloys; it is used in electroplating various metal parts, from electrical equipment to car parts. Chromium compounds are used in dyeing of textiles and they can be present in small amounts in concrete. Chromium compounds may cause cancer (ATSDR, 2000). Apart from the carcinogenic potential, prolonged exposure can result in bronchitis, rhinitis, or sinusitis and the kidney is often a target organ for chromate toxicity (Rom, 2007).

In persons non-occupationally exposed to chromium, the concentration of chromium in serum and in urine usually does not exceed 0.05 μg/100 ml and 2 μg/g creatinine, respectively. Recent exposure to soluble hexavalent chromium salts (e.g., in electroplaters and stainless steel welders) can be assessed by monitoring chromium level in urine at the end of the work shift since the major route of excreting chromium from the body is through urine (Hoet *et al.*, 2011). Chromium [VI] is corrosive and can cause chronic ulcerations of skin surfaces. Allergic skin reactions to chromium [VI] can also occur with chronic exposure (Goyer and Clarkson, 2001).

2.5 Occupational Hazards of Electronic Repairers

Electronic repair technicians have been associated with a high risk of brain tumor as a result of exposure to microwave and radio frequency, the risk of astrocytic tumors among the electronics manufacture and repair workers increases with duration of exposure to about tenfold among those employed for 20 or more years (Thomas *et al.*, 1987). Electrical tradesmen have also been reported to be exposed to extremely low frequency electromagnetic radiation, while men in some jobs associated with electronics manufacture and repair are said to be exposed to electromagnetic radiation in the very high frequency and ultra-high frequency ranges and also may be exposed to soldering fumes, solvents, and a variety of other chemicals (Thomas *et al.,* 1987 and Floderus *et al.,* 1996). Investigators have hypothesized that occupations involving electric and magnetic field exposure are associated with a variety of health problems, including neurological disease (Savitz *et al*., 1998). Exposure to irritants and allergens during common operations by electronic repairers and electronic workers such as soldering, cleaning operations pose dermatological hazards for such workers (Koh *et al*., 1990), furthermore, soldering flux has been identified as one of the commonest irritants in the electronics industry (Goh, 1994). Skin problems usually also arise from contact with rosin flux (Courtney, 1985). A study conducted in Taiwan showed a high risk of hand dermatitis among electronic workers; skin symptoms like itching, redness and scaling (Shiao *et al.*, 2004). Female electrical technicians have been linked to high risk of breast cancer (Loomis and Savitz, 1990; Loomis *et al.*, 1994). Work in electronics assembly by women has been reported to be significantly associated with delivering low birth weight infants (Lipscomb *et al.*, 1991). Electronics workers exposed to soldering have been found to have significantly higher risk of developing occupational

asthma symptoms in comparison to those not exposed (Burge *et al.*, 1979), the rosin type of organic flux used to provide flow for the solder joint and non-tarnished surfaces for bonding has been reported to be responsible for 5% cases of occupational asthma in the UK (Ross *et al*., 1995 and Burge, 1982). Occupational asthma is important both as a potentially curable and preventable cause of asthma and as a model of adult onset asthma. It is induced by sensitization to a specific agent inhaled at work (Newman-Taylor, 1988). Association between exposure to soldering in electronics workers and an increased risk of wheezing has been reported (Mendy *et al.*, 2012). Exposure to heavy metals was identified as the second most common occupational hazard among female electronic workers in China (Yu *et al.*, 2013), this occupational exposure to heavy metals is an important risk factor for cancer (t Mannetje *et al.*, 2011). Amyotrophic lateral sclerosis (ALS) (a neurodegenerative disease affecting the motor neurons of the spinal cord and brain) has been reported in workers of electronic manufacturing industry and has been strongly linked to high exposure to lead metal (Oh *et al.*, 2007). However, there are virtually no published works on assessment of the occupational hazards of electronic repairers in Nigeria.

2.6 The Use of Biomarkers to Detect and Quantify Heavy Metal Exposure

Biological markers or biomarkers have been used to provide important information about the presence of metals in biological systems. Biomarkers are observable end points in a continuum of events leading from exposure to these metals to effects (diseases or death). Biomarkers help to extrapolate the data on accessible sites to processes in inaccessible sites (Kakkar *et al.*, 1996)

Biomarkers example blood (whole blood), saliva, urine, nail, tissue, bone and so forth have been used to monitor lead exposure (Manton and Malloy, 1983 and Foo *et al*., 1993). This is because lead distributes itself in body fluids after ingestion (Manton and Malloy, 1983), a fraction of toxins absorbed by the human body is accumulated in various tissues and organs. The rest is excreted unchanged or as polar metabolites. The body has a tremendous ability to eliminate a wide variety of chemicals absorbed with the diet or as a result of exposure to the environment. The majority of poisons, particularly polar substances, leave the body with exhaled air and urine. Smaller amounts of chemicals are removed with saliva, bile, sweat, milk, and faeces (Kozłowska, 2003). The International Atomic Energy Agency (IAEA) started a Coordinated Research Programme in 1984 on the significance of hair mineral analysis as a means for assessing internal body burdens of environmental mineral pollutants.

Lead concentration in whole blood (BPb) is the primary biomarker used to monitor exposure to this metallic element (Barbosa *et al*., 2005). Human nails are extensively employed for monitoring exposure to excessive levels of elements. Nails can be studied easily and economically in subjects of residential areas and industrial workers and patients, and sometimes may indicate that high concentrations of elements are related to various illnesses. Deficiency of certain elements can also be determined with use of nails of subjects with specific or unknown deficiencies. Although nails are different from other biological samples in bioaccumulation, physiological activities may be partially due to chemical properties of the elements. Comparison of multi-elements between nails and other samples is relevant and found significant, because of the status of chronic exposure to high levels of elements (Sukumar, 2006). Results from researches suggest that hair is a reliable

biomarker to determine different levels of exposure of populations to lead and other heavy metal pollution (Muramatsu and Parr 1988), and they indicate that females tend to accumulate lead in the hair more than males of the same age (Sanna, *et al*., 2002). Of the biomarkers of lead exposure, blood lead (Pb-B), mainly red cell lead, is a representative of soft tissue lead and most widely used as measures of body burden and absorbed (internal) doses of lead. Urine lead (Pb-U) as well as plasma lead (Pb-P) increases exponentially with increasing Pb-B under a steady-state situation and is a reflection of recent exposure (Sakai, 2000).

Cadmium in blood has been used as a biomarker for exposure to cadmium, blood cadmium mainly reflects recent exposure, but it has also been shown to be a good indicator of cadmium body burden (Jarup *et al.*, 1988). In addition to blood cadmium, urine Cd has also been used as a biomarker for cadmium exposure (Jin *et al.*, 2002). Cadmium accumulates in the kidney with age and urinary cadmium is considered a valid biomarker of lifetime kidney accumulation (Orlowski *et al*., 1998) and consequently used in the assessment of cadmium-induced health effects (EFSA, 2009 and WHO, 2011b). Cadmium concentration in hair was used to assess the interaction between zinc, cadmium, and lead in scalp hair samples of Pakistani and Irish smokers who have rheumatoid arthritis in relation to controls (Afridi *et al.*, 2012). Cadmium in hair and nails has been used as biomarkers of individual cadmium exposure. Because of the affinity for SH-groups, cadmium is likely to accumulate in keratin-rich tissues like nails and hair.

Chromium in blood and urine has been used as a means of monitoring the metal ion in biological fluids of stainless steel welders using the flux-cored-wire (FCW) welding method (Stridsklev *et al.*, 2004)**.** Chromium levels in body fluids, such as urine and serum

are reliable markers of exposure to chromium in oxidation states (VI) and (III) and provide a measure of the internalized dose of chromium (Kortenkamp, 1997; Bonde and Christensen, 1999). Biomonitoring of Cr, Mn, Co and Fe in hair of 116 subjects of 40-50 years of age living near heavy traffic area and less traffic area in the state of Rajasthan in India was carried out by Mehra *et al*., (2010). Hair, nail, blood, urine and saliva were used to biologically monitor the occupational exposure of stainless steel welders to chromium, iron, manganese, molybdenum, nickel and vanadium in the workplace in manufacturing of stainless steel constructions (Kucera *et al.*, 2001). The report indicated that biological fluids and tissues like blood, urine, saliva, hair and nail are good biological indices for heavy metals exposure.

The advantages of hair and nail tissues analysis over other biological samples are that trace metal concentrations in them are not subjected to rapid fluctuation due to diet, air and water hence a long term stable nutritional status. More advantages include the non-invasive sampling, the reflection of exposure over several months, and the possibility to store at room temperature for long period of time (Kucera *et al.*, 2001). The Global Environmental Monitoring System (GEMS) has chosen human hair as a biopsy material to estimate metal body burden (Vahter, 1982). In a carefully conducted study, a major research laboratory at the Centers for Disease Control and Prevention (CDC) reported the determination of 28 elements in hair from non-occupationally exposed U.S. populations. These investigators used ICP-AES for all elements except mercury, which was measured with an LDC mercury monitor. The required minimum hair sample weight was 0.5 gram. More recently reported results for ICP-MS analysis of a suite of elements from hair in a

Brazilian population showed improved sensitivity compared to the ICP-AES values. The amount of hair required was approximately 0.3 gram (Ayodele and Bayero, 2009).

2.7 Toxic Metal Detection in Biological Samples

Toxic effects from heavy metals (Pb, As, Se, Cd, Cr, Bi, Tl, and Hg) have been characterised in medical literature for decades. Originally, complex extractions were performed to isolate the poisonous components from the complex biological matrices: blood serum, urine, tissues. Recently, optical emissions and atomic absorption spectroscopy techniques have been developed to determine trace elements. Lead (Pb) and other metals have been determined using Electrothermal Atomization Laser- Excited Atomic Fluorescence Spectrometry (Eugene *et al*., 1996), square wave anodic stripping analysis with electrode arrays (Timothy *et al*., 1997), size exclusion chromatography and inductively coupled plasma/ mass spectrometry (Gercken and Barnes, 1991), direct flameless atomic absorption (Hwang *et al*., 1973 and Nise *et al*., 1978) and a host of other techniques.

However, Atomic Absorption spectrometry (AAS), Graphite Furnace Atomic Absorption spectrometry (GF-AAS), Anodic Stripping Voltammetry (ASV) and Differential Pulse Anodic Stripping Voltammetry (DPASV) are the most frequently used because of their sensitivity, relative low cost, speed, simplicity of operation and minimum maintenance requirements (Yu *et al*., 1999; Brouwer, 2005; Szkoda and Zmudzki, 2005). As analytical instrumentation performance improved, real limits of detection were also improved. The advent of inductively coupled plasma (ICP) and graphite furnace AAS (GF-

AAS) brought the limits of analysis into the parts per billion range for many matrices. This capability has been invaluable in screening blood and urine samples from paediatric and chronically ill patient to track the exposure and elimination of lead.

Basically, the method involves transferring samples to a nichrome wire by placing a small sample on a wire loop or touching the heated wire in the AAS. By choosing the appropriate radiation source and wavelength, the presence of different metals may be determined qualitatively. An atom absorbs light at discrete wavelengths, in order to measure this narrow light absorption with maximum sensitivity, a line source is used. The line source emits the specific wavelengths, which can be absorbed by the atom because they possess just the right energy to result in the discrete electronic transitions. The common source of light in AAS is a hollow cathode lamp which contains a tungsten anode and a cylindrical hollow cathode made of the element to be determined. These are sealed in a glass tube filled with an inert gas like neon or argon. Each element has its own unique lamp, which must be used for that analysis. Applying a potential difference of about 300-400 V between the anode and the cathode leads to the ionization of some gas atoms. These gaseous ions bombard the cathode in a process called sputtering. Some sputtered atoms are in the excited states and emit radiation characteristic of the metal as they fall back to the ground state sputtering (PerkinElmer Corporation, 1996).

CHAPTERTHREE

MATERIALS AND METHODS

The apparatus, equipment and reagents used for the study are listed in Appendix I. All reagents used were of analar grade.

3.1 Preparation of Stock Solutions

3.1.1 Stock solutions

1000 mgdm^{-3} lead solution: Prepared by dissolving 1.5990 g $Pb(NO_3)_2$ in a 1000cm^3 volumetric flask with distilled deionized water and then making it up to mark. This gives a 1000 ppm solution of Pb.

1000 mgdm^{-3} Cadmium solution: Prepared by dissolving 2.7500 g $Cd(NO_3)_2$. $4H_2O$ in a 1000 cm^3 volumetric flask with distilled deionized water and then making it up to mark to give the 1000 ppm Cd solution.

1000 mgdm^{-3} Chromium solution: Prepared by dissolving 2.5192 g $Na_2Cr_2O_7$ in a 1000 cm^3 volumetric flask with distilled deionized water and then making it up to mark to give the 1000 ppm Cr solution.

3.1.2 Standard working solutions

Working standards were prepared from the stock solutions using the dilution formula:

$C_{\text{stock solution}} \times V_{\text{stock solution required}} = C_{\text{standard working solution}} \times V_{\text{standard working solution}}$

$V_{stock\ solution\ required} = (C_{standard\ working\ solution} \times V_{standard\ working\ solution}) / C_{stock\ solution}$

Working standard concentrations were prepared from the 1000 $mgdm^{-3}$ solution of each metal stock solution by serial dilution as follows; 1.0, 2.0, 4.0, 5.0 and 10.0 $mgdm^{-3}$ Pb, 1.0, 2.0, 3.0, 6.0, 8.0 and 10.0 $mgdm^{-3}$ Cd and 5.0, 10.0, 20.0, 40.0 and 80.0 $mgdm^{-3}$ Cr.

3.2 Description of the Study Area

Kaduna occupies a central position in the northern part of Nigeria. It is situated between a latitude of 10° 31' 23N and longitude of 7° 26' 25E with an area of approximately 48,473.2 square kilometers. It has a population of 6,113,503 according to the 2006 census, with an estimated current population of 7,474,369 in 2013 (using 3.18% growth rate as allowed by the National Population Commission). It shares common borders with Niger, Kano, Bauchi, Plateau, Zamfara and Katsina States and to the South-West, the State shares a border with the Federal Capital Territory, Abuja (the capital of Nigeria) It has a long historical significance as it was the capital of the old northern Nigeria (between 1917 and 1967). The Map of Nigeria showing Kaduna state is shown in figures 3.2a.

Nine electronic repair shops within Kaduna metropolis were the points of collection of samples from ten (10) volunteer electronic repairers, these locations are Hayin banki (1), Unguwar Dosa (2), Badarawa (3 & 4), Tudun wada (5), Sheikh Abubakar Gumi Market (6), Unguwar kanawa (7), Kawo (8), Malali (9) and Unguwar Rimi (10), the numbers in paranthesis represent the volunteers who were serialized in sequence of sample collection. However, subjects 2 and 6 did not avail themselves for nail and urine collection

respectively while subject 9 is bald as such hair samples from the subject could not be collected.

3.2.1 Hayin Banki (1)

Blood, urine, nail and hair samples of subject one were collected at his shop along Baso road, Hayin Banki, Kaduna. The street is located off Ali Akilu Road, Kawo Kaduna.

3.2.2 Unguwar Dosa (2)

This was the point of collection of samples from subject 2. The repair shop is located on layin mai unguwa close, off College road. It is a residential area. There is a very high traffic on College road as it is the main link to Legislative Quarters (U/Dosa new extension), Rafin Guza. It is also the main route to Kawo market from Badarawa.

3.2.3 Badarawa (3 & 4)

This was the point of collection of samples of subjects 3 and 4, working in the same shop; the shop is located along Majalisa road off Shagari close, Badarawa. The shop is situated just by the road side and there is relatively high traffic along the Majalisa axis as the road connects Badarawa, U/Yero and Malali from the north.

3.2.4 Ungwar Mu'azu (5)

Subject 5 is located along Na'ibi Street off Ungwar mu'azu by U-turn on the Nnamdi Azikiwe bye pass.

3.2.5 Doka (6)

This was the point of collection of samples from subject 6 along Ahmadu Bello Way, close to Katsina round about in the heart of Kaduna town. The vehicular traffic is enormous as it is situated just after the Kaduna Central Market; the Sheikh Abubakar Gumi Market, the heart of business activities in Kaduna.

3.2.6 Unguwar Kanawa (7)

Samples were collected from subject 7 at layin mai unguwa, Unguwar Kanawa, Kaduna.

3.2.7 Kawo (8)

Samples from subject 8 were collected at Dalma road off Nasarawa road, Kawo Kaduna.

3.2.8 Malali (9)

Samples from subject 9 were collected at Malali, Chad cresent by liberia road.

3.2.9 Unguwar Rimi (10)

Samples from subject 10 were collected from Fulani road, off Kinshasa road Ungwar Rimi Kaduna. The vehicular traffic is relatively high and the road serves as a link to many other areas of Ungwar Rimi.

The map showing the sampled points in Kaduna metropolis is shown in figure 3.2b, photographs of the shops are also presented in figures 3.2.1 – 3.2.9.

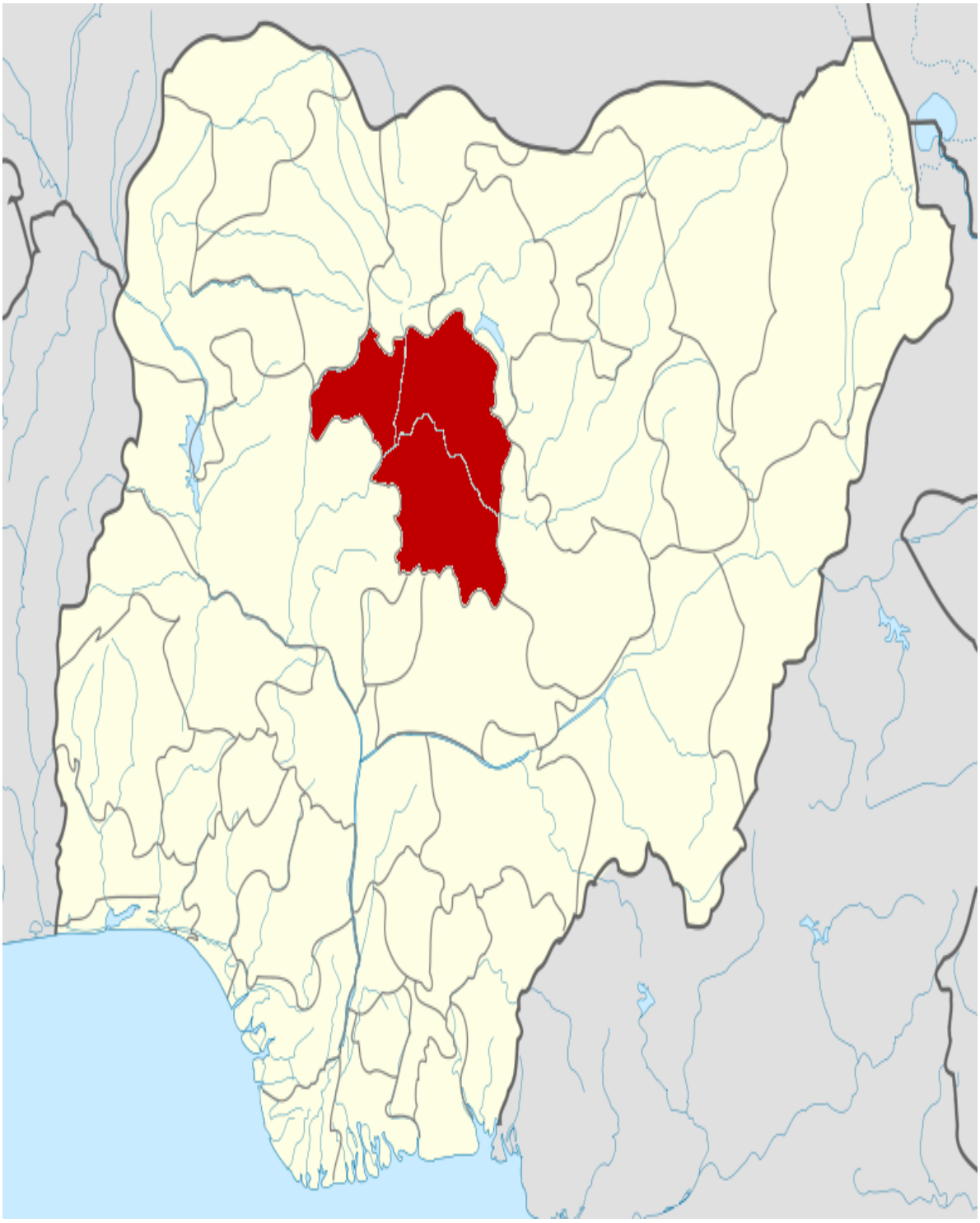


Figure 3.2a: Map of Nigeria Showing Kaduna State (in red)

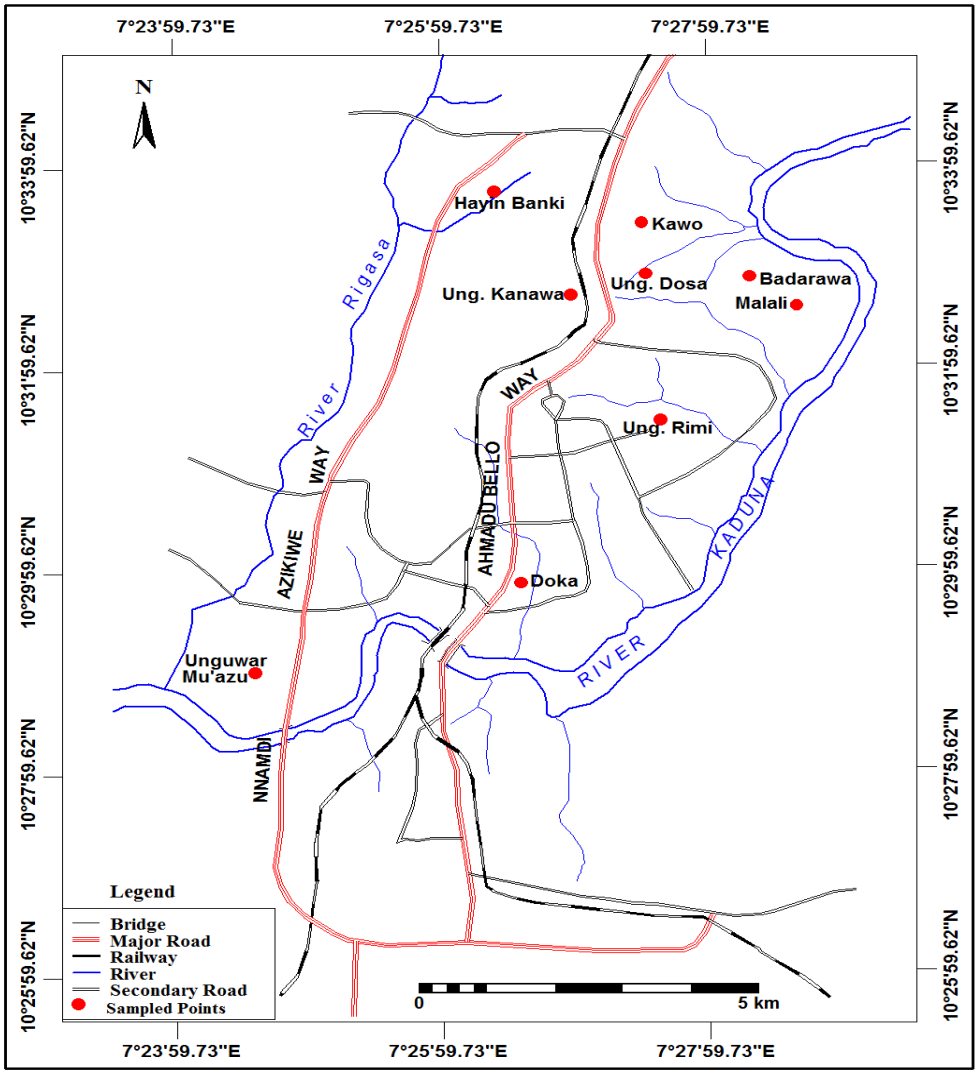


Figure 3.2b: Map of Kaduna Metropolis Showing Sample Collection Points



Figure 3.2.1: Sample Collection Point 1, Hayin Banki



Figure 3.2.2 Sample Collection Point 2, Ungwar Dosa



Figure 3.2.3: Sample Collection Point 3 and 4, Badarawa



Figure 3.2.4: Sample Collection Point 5, Ungwar Mu'azu



Figure 3.2.5 Sample Collection Point 6, Doka

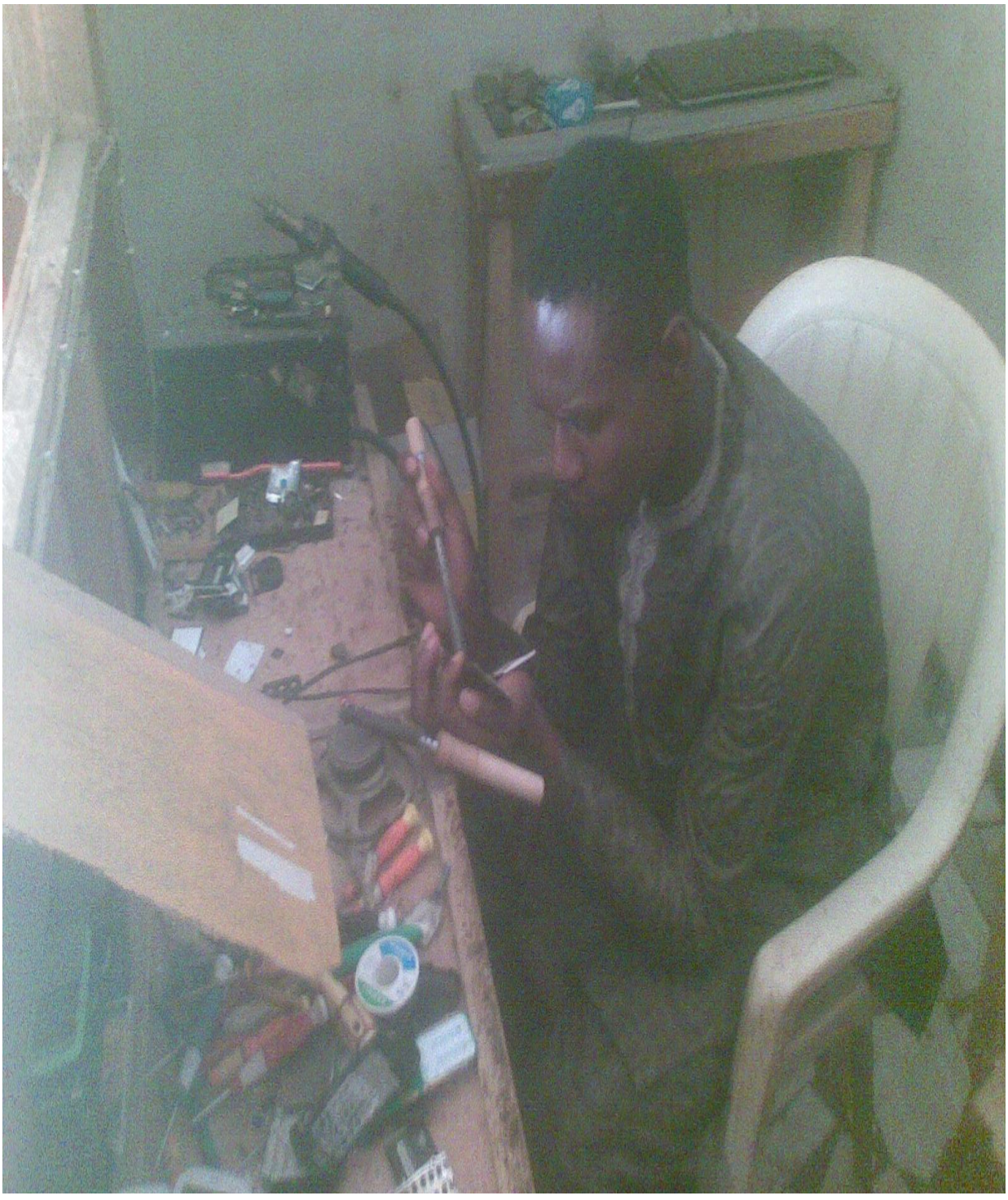


Figure 3.2.6 Sample Collection Point 7, Ungwar Kanawa



Figure 3.2.7 Sample Collection Point 8, Kawo



Figure 3.2.8 Sample Collection 9, Malali



Figure 3.2.9 Sample Collection 10, Ungwar Rimi

3.3 Sample Collection

Sampling was carried out according to standard procedure as reported by Memon *et al.*, (2007) and Olmedo *et al.*, (2010).

3.3.1 Blood samples

1 ml of whole blood was collected in 5 ml EDTA tubes using a stainless needle and syringe. The blood samples were then shaken and frozen for 3 days till further treatment.

3.3.2 Urine samples

Urine samples were collected in acid-washed, decontaminated 20 ml polyethylene tubes. Urine samples were then acidified with reagent grade concentrated HNO_3 (1% v/v) and frozen for 3 days till further treatment.

3.3.3 Hair samples

Hair samples, as close as possible to the scalp were collected using a stainless scissors and stored in clean plastic bags with identity numbers for further treatment.

3.3.4 Nail samples

Nail samples were cut using a sterilised stainless scissors and sealed in plastic bags with identity numbers prior to analysis

The personal, medical history and general information of the subjects were obtained through a questionnaire filled by the subjects. The questionnaire is a modified version of

the WHO health assessment questionnaire (WHO and UNEP, 2008); modified to suite the problem at hand. A sample of the questionnaire is shown in appendix II.

3.4 Sample Preparation and Analysis

3.4.1 Sample pre-treatment and digestion

3.4.1.1 *Blood and urine samples*

Blood and urine samples were digested using conventional wet acid digestion as reported by Memon et *al* (2007); 0.5 ml of blood and urine was directly taken into a Pyrex flask, 3 ml of freshly prepared mixture of concentrated HNO_3-H_2O_2 (2:1, v/v) was added and allowed to stand for 10 minutes. The flask was then covered with a watch glass and digested at 60-70^oC for 1-2 hours, the digest was treated with 2ml nitric acid and few drops of H_2O_2, heated on a hot plate to about 80^oC until a clear digested solution was obtained. The excess acid mixture was evaporated to a semi-dried mass, cooled and diluted with 0.1 mol/dm^3 nitric acid.

3.4.1.2 *Hair samples*

200 mg of the hair samples was washed as described by Afridi *et al.* (2006).The hair specimens were cut into 0.2 to 0.3 cm pieces and washed four times with a 1:200 v/v dilution of triton X-100, then rinsed three times with distilled water and two times with acetone. The samples were then dried in an oven at 75-85^oC. The washed hair samples were digested as described by Memon *et al.*, (2007) above.

3.4.1.3 *Nail samples*

Collected nail samples were washed using Gammelgaard's method (Gammelgaard *et al.*, 1991); the nail samples were washed thoroughly stirring with different solvents in sequence: acetone, deionized water and 0.5% Triton X-100 solution. The nail samples were then dried in an oven at 110^{o}C for about 30 minutes. The dried nail samples were digested with 10 mL of 6:1 mixture of concentrated nitric and perchloric acid and kept overnight at room temperature to prevent excessive foaming and subsequently heated at 160-180°C until the mixture was water clear and less than 1 mL of the solution was remain. Each sample solution was then diluted with 0.1 N nitric acid (Mehra and Juneja, 2005).

3.4.2 Determination of the metals

The concentrations of the metals were determined using Varian AA240FS Fast Sequential Atomic Absorption Spectrometer.

3.5 Quality Assurance Procedure

Validation of the digestion method of analysis used and certification of the instrument as good enough for the analysis was done by carrying out a recovery experiment. This was done by spiking the predigested blood, urine, nail and hair samples with 10 cm^3 of multi-element metal standard solution (MESS) (20 ppm of Pb, Cd and Cr).

3.5.1 Preparation of multi-element standard solution (MESS)

0.0320 g of $Pb(NO_3)_2$ was weighed into a beaker, about 2 cm^3 of distilled deionized water was then added to dissolve it. The solution is then transferred into a 1000 cm^3 volumetric flask. The beaker was rinsed thoroughly with distilled deionized water into the volumetric flask. 0.0550 g of $Cd(NO_3)_2.4H_2O$ was then weighed into another beaker, about 2 cm^3 of distilled deionized water was added to dissolve it. The solution was transferred into the same volumetric flask. The beaker was rinsed thoroughly with distilled deionized water into the flask. 0.0504 g of $Na_2Cr_2O_7$ salt was also weighed into another beaker, about 2 cm^3 of distilled deionized water was added to dissolve it. The solution was transferred into the same volumetric flask. The beaker was rinsed thoroughly with distilled deionized water into the flask. The flask was made up to mark with distilled deionized water to give the 20 ppm multi element standard solution (MESS) of Pb, Cd and Cr. Aliquot of this solution was used in the recovery experiment.

3.5.2 Recovery experiment

A portion of predigested blood, urine, nail, and hair samples was spiked with 10ml of mixed element standard solution (MESS) of 20 ppm lead, cadmium and chromium (VI) each. These were then digested as described in sections 3.4.1.1 – 3.4.1.3 above. The concentrations of the metals were then determined using the atomic absorption spectrophotometer. The percentage recoveries were then calculated as follows:

Amount of metal used to spike the sample

$$= \frac{\text{Vol. of MESS used x Conc. of metal in MESS}}{1000}$$

= **X** mg of metal used to spike a sample (calculated)

Amount of metal in unspiked sample

$$= \frac{\text{Vol. of digest of unspiked sample x Conc. of metal in digest of unspiked sample}}{1000}$$

= $\mathbf{Y_0}$ mg of metal in Zg of unspiked sample

Amount of metal in spiked sample

$$= \frac{\text{Vol. of digest of spiked sample x Conc. of metal in digest of spiked sample}}{1000}$$

= $\mathbf{Y_1}$ mg of metal in Zg of sample spiked

Estimate of amount of metal used to spike sample

= Amount of metal in spiked sample ($\mathbf{Y_1}$) – Amount of metal in unspiked sample ($\mathbf{Y_0}$)

= **X**□ mg of metal used to spike a sample (determined)

$$\% \text{ Recovery of metal} = \frac{\mathbf{X}\square \text{ mg of metal used to spike a sample (determined)}}{\mathbf{X} \text{ mg of metal used to spike a sample (calculated)}} \times 100$$

= **(X ÷ X□)** x 100

3.6 Statistical Analysis

The values of the mean metal concentrations in the blood, urine, nail and hair samples are expressed as the arithmetic mean value with standard deviation and tabulation to illustrate concentration profile over each group. Some of the results are presented in bar charts to give a clearer and simpler picture of the relationships over each group.

The statistical significance of the difference of the mean values between different groups was determined by applying the student‘t’ test with the level of significance at $P<0.05$. The correlation between the metals was observed using Pearson correlation analysis. The correlation coefficient ‘r’ is a measure of linear association between two variables. Values of the correlation coefficient are always between −1 and +1. A correlation coefficient of +1 indicates that two variables are perfectly related in a positive linear sense; a correlation coefficient of −1 indicates that two variables are perfectly related in a negative linear sense, and a correlation coefficient of 0 indicates that there is no linear relationship between the two variables. Analyses of variance ANOVA was employed to test the hypotheses made at the onset of the work.

All statistical analyses were performed using Microsoft Excel 2007 and SPSS 16.0 version.

CHAPTER FOUR

RESULTS

4.1 Quality Assurance (Recovery Experiment)

Figure 4.1 shows the pattern of the recoveries of the metals in all samples.

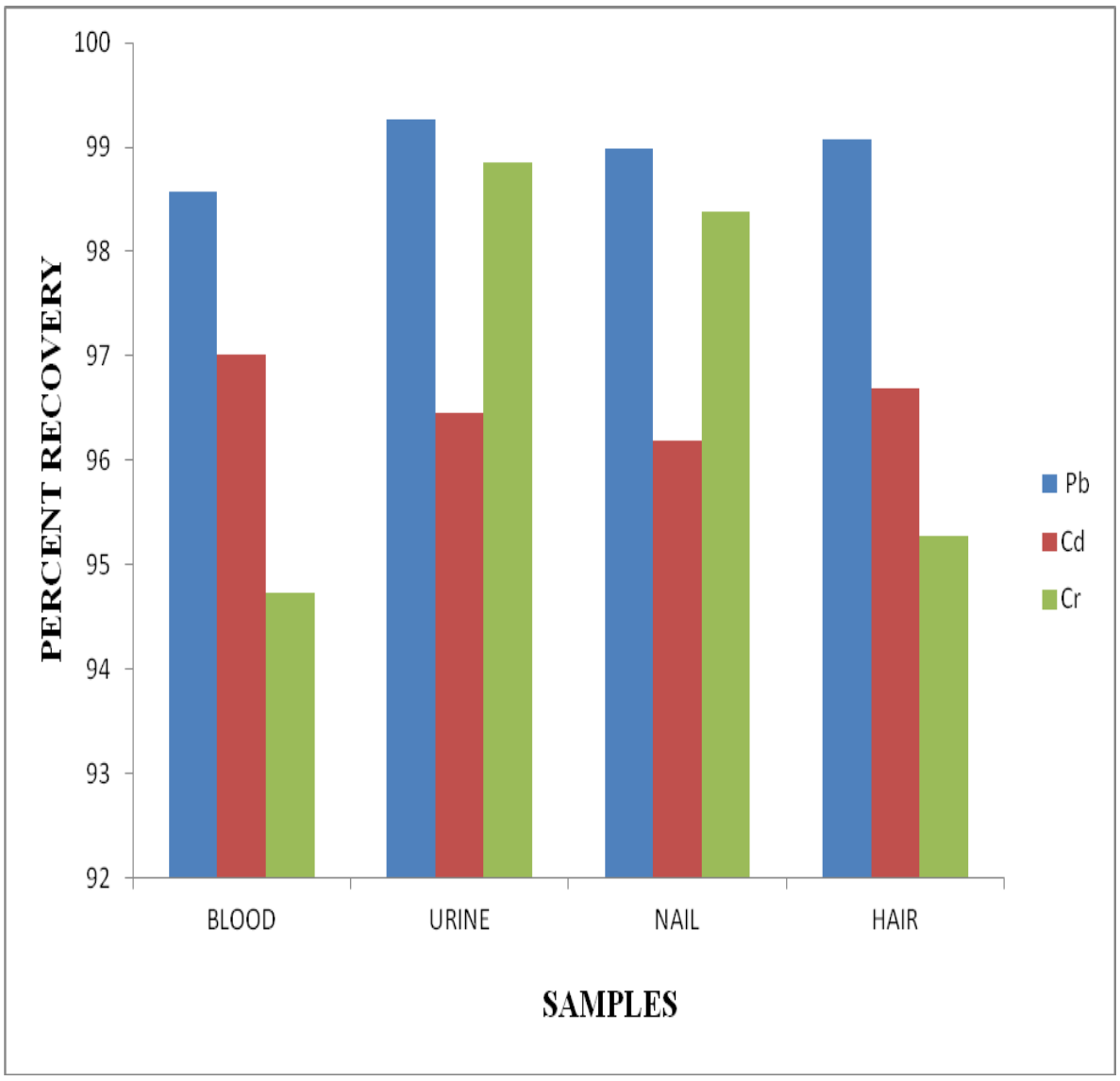


Figure 4.1: Percentage Recoveries of Pb, Cd and Cr in Blood, Urine, Nail and Hair samples

4.2 Result of Pb, Cd and Cr Analyses in Blood Samples of the Subjects

The concentrations of Pb, Cd and Cr metals in the blood samples of the electronic repairers are shown in figure 4.2.

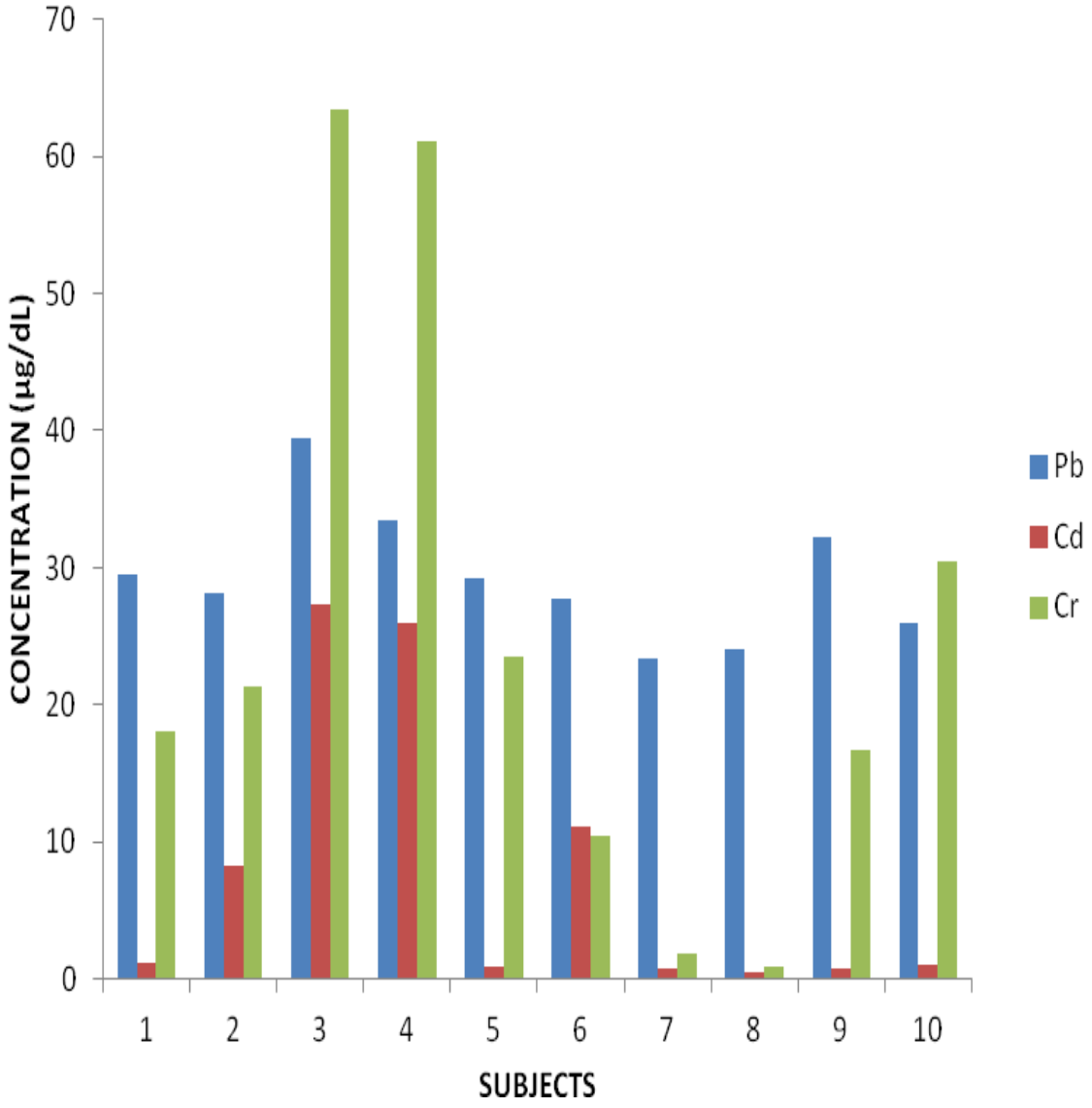


Figure 4.2: The Distribution of Pb, Cd and Cr (µg/dL) in Blood Samples of Electronic Repairers

4.3 Results of Pb, Cd and Cr Analyses in Urine Samples of the Subjects

The concentrations of Pb, Cd and Cr in the urine samples of the subjects are shown in figure 4.3.

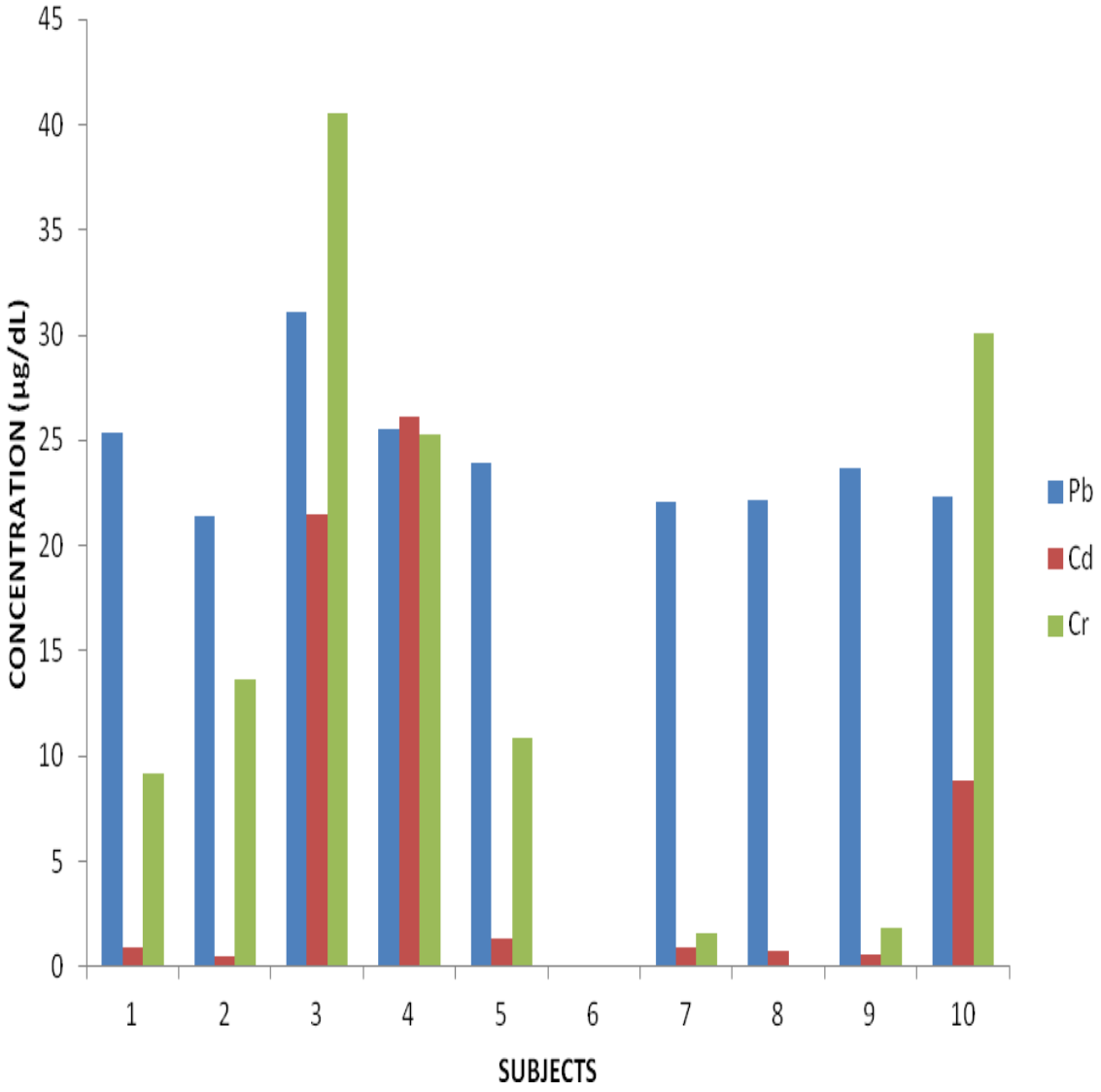


Figure 4.3: The Distribution of Pb, Cd and Cr in Urine Samples of Electronic Repairers

4.4 Results of Pb, Cd and Cr Analyses in Nail Samples of Subjects

Figure 4.4 shows the concentrations of Pb, Cd and Cr in the hair samples of the subjects.

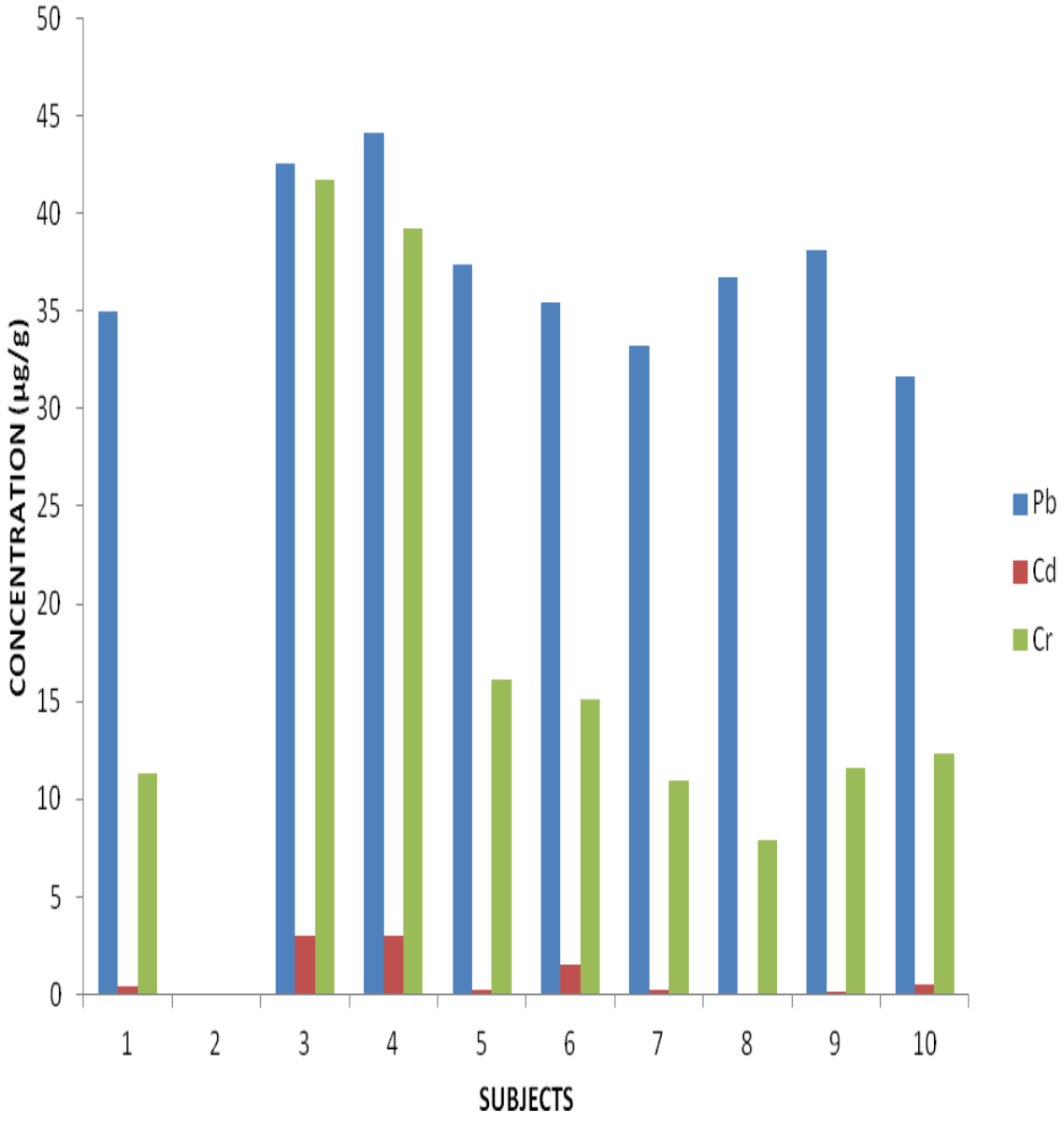


Figure 4.4: The Distribution of Pb, Cd and Cr (µg/g) in Nail Samples of Electronic Repairers

4.5 Results of Pb, Cd and Cr Analyses in Hair Samples of Subjects

The concentrations of Pb, Cd and Cr in the nail samples of subjects are shown in Figure 4.5.

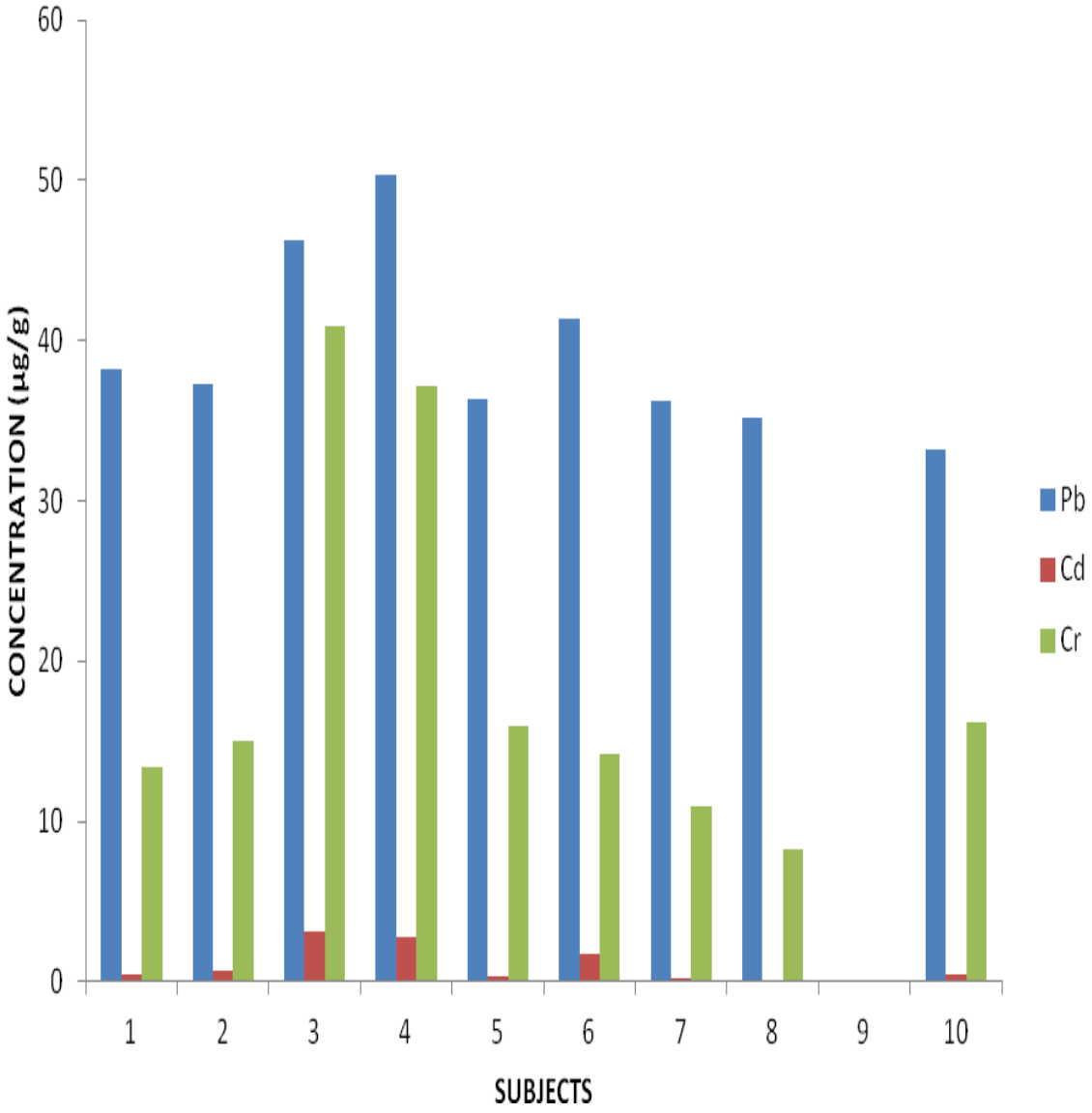


Figure 4.5: The Distribution of Pb, Cd and Cr (µg/g) in Hair Samples of Electronic Repairers

4.6 Correlation of Each Metal Across the Blood, Urine, Nail and Hair Samples

The correlation values between Pb/Cd, Pb/Cr and Cd/Cr pairs in the blood samples is shown in table 4.1

Table 4.1: Parametric Pearson Correlation Coefficients for Pb, Cd and Cr in the Blood Samples

	Pb	**Cd**	**Cr**
Pb	1		
Cd	0.766**	1	
Cr	0.839**	0.854**	1

** Correlation is significant at 0.01 level (1 tailed)

Correlation coefficients between Pb/Cd, Pb/Cr and Cd/Cr pairs in the urine samples is shown in table 4.2

Table 4.2: Parametric Pearson Correlation Coefficients for Pb, Cd and Cr in the Urine Samples

	Pb	**Cd**	**Cr**
Pb	1		
Cd	0.679*	1	
Cr	0.661*	0.807**	1

** Correlation is significant at the 0.001 level (1 tailed)

* Correlation is significant at the 0.005 level (1 tailed)

Correlation coefficients between Pb/Cd, Pb/Cr and Cd/Cr pairs in the nail samples is shown in table 4.3

Table 4.3: Parametric Pearson Correlation Coefficients for Pb, Cd and Cr in the Nail Samples

	Pb	**Cd**	**Cr**
Pb	1		
Cd	0.776**	1	
Cr	0.851**	0.954**	1

**. Correlation is significant at the 0.01 level (1-tailed).

Correlation coefficients between Pb/Cd, Pb/Cr and Cd/Cr pairs in the nail samples is shown in table 4.4

Table 4.4: Parametric Pearson Correlation Coefficients for Pb, Cd and Cr in the Hair Samples

	Pb	**Cd**	**Cr**
Pb	1		
Cd	0.958**	1	
Cr	0.897**	0.924**	1

**. Correlation is significant at the 0.01 level (1-tailed)

4.7 Concentration of Pb, Cd and Cr in the Subjects by Different Categories

4.7.1 Concentration of Pb, Cd and Cr in the test subjects by social habits (smoking)

The mean concentrations of Pb, Cd and Cr in all the samples for smokers and non smokers are shown in table 4.5.

Table 4.5: the mean concentrations of Pb, Cd and Cr based on the smoking habit of subjects

CATEGORY	SAMPLES	**Pb**	**Cd**	**Cr**
SMOKERS N=2	**BLOOD**	36.50 ± 4.24 µg/dL	26.60 ± 0.01 µg/dL	62.25 ± 1.63 µg/dL
	URINE	28.30 ± 3.96 µg/dL	23.80 ± 3.25 µg/dL	32.90 ±10.75 µg/dL
	NAIL	43.34 ± 1.10 µg/g	2.92 ± 0.27 µg/g	40.48 ± 1.74 µg/g
	HAIR	48.35 ± 2.88 µg/g	3.00 ± 0.02 µg/g	39.05 ± 2.64 µg/g
NON SMOKERS N=8	**BLOOD**	27.54 ± 2.95 µg/dL	3.00 ± 4.00 µg/dL	15.41±10.36 µg/dL
	URINE	23.00 ± 1.39 µg/dL	1.96 ± 3.03 µg/dL	11.99±10.28 µg/dL
	NAIL	31.07 ± 9.12 µg/g	0.52 ± 0.52 µg/g	12.20 ± 2.74 µg/g
	HAIR	36.83 ± 2.50 µg/g	0.50 ± 0.49 µg/g	13.44 ± 2.90 µg/g

4.7.2 Concentration of Pb, Cd and Cr in the test subjects by education

The mean values for the concentration of the metals in the subjects based on the level of formal education are shown in table 4.6.

Table 4.6: The mean concentrations of Pb, Cd and Cr in test subjects with respect to level of education

LEVEL OF EDUCATION	SAMPLE	**Pb**	**Cd**	**Cr**
DIPLOMA (HIGHER EDUCATION) N=2	**BLOOD**	23.75 ± 0.50 µg/dL	0.65 ± 0.21 µg/dL	1.40 ± 0.71 µg/dL
	URINE	22.15 ± 0.07 µg/dL	0.80 ± 0.14 µg/dL	0.80 ± 1.13 µg/dL
	NAIL	34.95 ± 2.50 µg/g	0.15 ± 0.08 µg/g	9.43 ± 2.09 µg/g
	HAIR	35.70 ± 0.77 µg/g	0.17 ± 0.09 µg/g	9.61 ± 1.93 µg/g
SECONDARY N=6	**BLOOD**	29.50 ± 2.87 µg/dL	8.05 ± 9.75 µg/dL	26.35 ±18.23 µg/dL
	URINE	23.66 ± 1.83 µg/dL	7.38 ± 11.04 µg/dL	19.34 ± 8.48 µg/dL
	NAIL	36.86 ± 4.67 µg/g	1.04 ± 0.99 µg/g	17.93 ± 12.01 µg/g
	HAIR	39.26 ± 4.86 µg/g	1.07 ± 1.05 µg/g	19.20 ± 10.10 µg/g
PRIMARY SCHOOL OR LESS N=2	**BLOOD**	34.40 ± 7.21 µg/dL	14.10 ± 18.67 µg/dL	43.45 ± 28.21 µg/dL
	URINE	27.50 ± 5.09 µg/dL	11.40 ± 14.28 µg/dL	25.70 ± 20.93 µg/dL
	NAIL	39.98 ± 3.65 µg/g	1.71 ± 1.98 µg/g	28.93 ± 18.08 µg/g
	HAIR	43.39 ± 9.91 µg/g	1.62 ± 1.97 µg/g	28.46 ± 17.61 µg/g

4.7.3 Concentration of Pb, Cd and Cr in the test subjects by type of electronics

Table 4.7 shows the concentration of the metals in the subjects with respect to the type of electronics the subjects specialize in repairing.

Table 4.7: the mean concentrations of Pb, Cd and Cr in test subjects with respect to type of electronics specialized in

SPECIALITY	SAMPLE	**Pb**	**Cd**	**Cr**
INVERTER	**BLOOD**	24.10 ± 0 µg/dL	0.50 ± 0 µg/dL	0.90 ± 0 µg/dL
	URINE	22.20 ± 0 µg/dL	0.70 ± 0 µg/dL	0.00 ± 0 µg/dL
	NAIL	36.73 ± 0 µg/g	0.09 ± 0 µg/g	7.95 ± 0 µg/g
	HAIR	35.16 ± 0 µg/g	0.10 0 µg/g	8.25 ± 0 µg/g
GENERAL ELECTRONICS	**BLOOD**	29.91 ± 4.70 µg/dL	8.59 ± 10.88 µg/dL	28.60 ± 22.46 µg/dL
	URINE	24.43 ± 3.08 µg/dL	7.58 ± 10.46 µg/dL	16.63 ± 13.98 µg/dL
	NAIL	37.18 ± 4.35 µg/g	1.22 ± 1.15 µg/g	19.80 ± 12.91 µg/g
	HAIR	39.94 ± 5.76 µg/g	1.12 ± 1.24 µg/g	20.49 ± 11.62 µg/g

4.7.4 Concentration of Pb, Cd and Cr by use of protective devices

Table 4.8 shows the variation in concentrations levels of repairers who use safety devices (though not regularly) and those who don't.

Table 4.8: the mean concentrations of Pb, Cd and Cr in test subjects with respect to using safety devices

SAFETY DEVICES	SAMPLE	**Pb**	**Cd**	**Cr**
USERS	**BLOOD**	26.80 ± 3.82 µg/dL	0.85 ± 0.95 µg/dL	9.50 ± 12.16 µg/dL
	URINE	21.80 ± 0.57 µg/dL	0.80 ± 0.14 µg/dL	4.60 ± 6.51 µg/dL
	NAIL	36.73 ± 0.10 µg/g	0.26 ± 0.22 µg/g	9.65 ± 2.41 µg/g
	HAIR	36.22 ± 1.49 µg/g	0.24 ± 0.22 µg/g	10.83 ± 3.65 µg/g
NON USERS	**BLOOD**	29.96 ± 5.02 µg/dL	9.51 ± 11.25 µg/dL	28.60 ± 22.46 µg/dL
	URINE	24.29 ±3.30 µg/dL	8.53 ± 10.92 µg/dL	17.69 ± 14.75 µg/dL
	NAIL	37.49 ± 4.67 µg/g	1.33 ± 1.19 µg/g	21.01 ± 13.45 µg/g
	HAIR	40.18 ± 6.19 µg/g	1.22 ± 1.30 µg/g	21.50 ± 12.16 µg/g

4.7.5 Concentration by lenght of period in the job

The distribution of Pb, Cd and Cr based on the length of period in the job is shown in table 4.9.

Table 4.9: the mean concentrations of Pb, Cd and Cr in test subjects with respect to length of period in the job

Category	Sample	**Pb**	**Cd**	**Cr**
10-15 yrs	**BLOOD**	27.82 ± 3.01 µg/dL	3.87 ± 4.58 µg/dL	16.48 ± 9.68 µg/dL
N=6	**URINE**	22.98 ± 1.59 µg/dL	2.34 ± 3.62 µg/dL	11.27 ± 11.69 µg/dL
	NAIL	34.67 ± 2.46 µg/g	0.54 ± 0.55 µg/g	12.27 ± 1.69 µg/g
	HAIR	37.29 ± 2.97 µg/g	0.72 ± 0.58 µg/g	13.96 ± 1.96 µg/g
>15yrs	**BLOOD**	31.60 ± 2.87 µg/dL	13.65 ± 7.48 µg/dL	37.23 ± 15.17 µg/dL
N=4	**URINE**	25.68 ± 1.93 µg/dL	12.40 ± 6.65 µg/dL	19.17 ± 8.80 µg/dL
	NAIL	40.20 ± 1.85 µg/g	1.58 ± 0.82 µg/g	26.26 ± 8.39 µg/g
	HAIR	42.06 ± 3.73 µg/g	1.56 ± 0.79 µg/g	25.59 ± 7.97 µg/g

4.7.6 Concentration by source of food eaten at work and age of the repairers

The distribution of the metals with respect to the source of food eaten at work and with respect to the age of the repairers are shown in tables 4.10 and 4.11 respectively

Table 4.10: the mean concentrations of Pb, Cd and Cr in test subjects with respect to the source of food eaten at work

SOURCE OF FOOD	SAMPLE	**Pb**	**Cd**	**Cr**
HOME	**BLOOD**	36.50 ± 3.00 µg/dL	26.60 ± 0.70 µg/dL	62.25 ± 1.15 µg/dL
	URINE	28.30 ± 2.80 µg/dL	23.80 ± 2.30 µg/dL	32.90 ± 7.60 µg/dL
	NAIL	43.34 ± 0.78 µg/g	3.00 ± 0.02 µg/g	40.48 ± 1.23 µg/g
	HAIR	48.35 ± 2.04 µg/g	2.92 ± 0.19 µg/g	39.05 ± 1.86 µg/g
RESTAURANT	**BLOOD**	26.50 ± 1.59 µg/dL	0.93 ± 0.22 µg/dL	16.47 ± 8.56 µg/dL
	URINE	23.30 ± 1.05 µg/dL	3.47 ± 2.67 µg/dL	13.10 ± 8.91 µg/dL
	NAIL	34.45 ± 1.50 µg/g	0.32 ± 0.12 µg/g	10.55 ± 1.33 µg/g
	HAIR	35.56 ± 1.45 µg/g	0.34 ± 0.12 µg/g	12.62 ± 2.33 µg/g
VENDOR	**BLOOD**	28.16 ± 1.42 µg/dL	4.36 ± 2.21 µg/dL	14.78 ± 3.91 µg/dL
	URINE	22.77 ± 0.61µg/dL	0.83 ± 0.18 µg/dL	7.00 ± 3.09 µg/dL
	NAIL	36.03 ± 1.11 µg/g	0.52 ± 0.33 µg/g	13.44 ± 1.29 µg/g
	HAIR	37.83 ± 1.22 µg/g	0.74 ± 0.34 µg/g	14.05 ± 1.09 µg/g

Table 4.11: the mean concentrations of Pb, Cd and Cr in test subjects with respect to age of the subjects

AGE	SAMPLE	**Pb**	**Cd**	**Cr**
25-30 YRS	**BLOOD**	27.84 ± 1.51 µg/dL	2.42 ± 1.45 µg/dL	17.68 ± 4.61 µg/dL
	URINE	22.98 ± 0.71 µg/dL	2.34 ± 1.62 µg/dL	11.27 ± 5.23 µg/dL
	NAIL	34.26 ± 1.40 µg/g	0.30 ± 0.08 µg/g	11.55 ± 0.30 µg/g
	HAIR	36.26 ± 1.08 µg/g	0.47 ± 0.10 µg/g	13.90 1.13 µg/g
31-35 YRS	**BLOOD**	27.03 ± 1.54 µg/dL	4.17 ± 3.47 µg/dL	11.63 ± 6.55 µg/dL
	URINE	23.05 ± 0.85 µg/dL	1.00 ± 0.30 µg/dL	5.45 ± 5.45 µg/dL
	NAIL	36.52 ± 0.58 µg/g	0.61 ± 0.45 µg/g	13.08 ± 2.58 µg/g
	HAIR	37.66 ± 1.92 µg/g	0.71 ± 0.51 µg/g	12.82 ± 2.34 µg/g
36-40 YRS	**BLOOD**	39.50 ± 0 µg/dL	27.30 ± 0 µg/dL	63.40 ± 0 µg/dL
	URINE	31.10 ± 0 µg/dL	21.50 ± 0 µg/dL	40.50 ± 0 µg/dL
	NAIL	42.56 ± 0 µg/g	3.01 ± 0 µg/g	41.71 ± 0 µg/g
	HAIR	50.39 ± 0 µg/g	3.11 ± 0 µg/g	40.92 ± 0 µg/g
41-45 YRS	**BLOOD**	33.50 ± 0 µg/dL	25.90 ± 0 µg/dL	61.10 ± 0 µg/dL
	URINE	25.50 ± 0 µg/dL	26.10 ± 0 µg/dL	25.30 ± 0 µg/dL
	NAIL	44.12 ± 0 µg/g	2.98 ± 0 µg/g	39.25 ± 0 µg/g
	HAIR	46.31 ± 0 µg/g	2.73 ± 0 µg/g	37.19 ± 0 µg/g

CHAPTER FIVE

DISCUSSION

Figure 4.1 shows the pattern of the recoveries of the metals in all samples. The percentage recoveries observed for the metals in the blood samples ranged from 94.73 – 98.57, the range in urine samples was 96.45 – 99.26 while the ranges of the percentage recoveries in the nail and hair samples were 96.19 – 98.99 and 95.27 – 99.07 respectively. Pb metal had the highest percent recovery in all samples, the trend of the percent recoveries of the metals in the samples was generally Pb > Cr > Cd except in the blood samples where Cd had a greater percent recovery than Cr (Cd=97.01% anc Cr=94.73%). Similar percent recoveries have been reported in different studies by other researchers who used MESS in spiking blood, urine, nail and hair samples to ascertain recoveries; a recovery profile of Pb>Cd in blood samples was reported by Yahaya *et al.* (2013). A range of 94.6–104.9% has been reported for Al, B, Ba, Cd, Cr, Cu, Mn, Pb and Zn in human blood samples (Momen *et al.,* 2013). The recovery of Pb, Cd and Zn in hair samples has been found to be in the range of 97.5-99.7% of CRMs (Afridi *et al.*, 2012). The results are also consistent with a range of 93.6 to 107.8% reported for Cr, Cd, Mn, Ni and Pb in whole blood, urine, saliva and hair (Olmedo *et al.*, 2010). Appendix III shows the percent recoveries of Pb, Cd and Cr in blood, urine, nail and hair samples.

Percentages of between 94.73 – 99.26% obtained show good recoveries and are within acceptable values, and thus indicate the suitability of the digestion method and certification of the AAS as reliable for the analyses.

5.1 Concentrations of Pb, Cd and Cr Analyses in Blood Samples of the Subjects

The distribution profile of the metals in the blood samples as depicted by figure 4.2 shows Pb metal being generally highest in the blood samples except for subjects 3 and 4 where the Cr levels instead were highest, thus the general trend of the metals in the blood can be said to be Pb>Cr>Cd. The mean Pb concentration in the blood samples of the subjects was observed to be 29.33 ± 4.80 µg/dL, with a minimum of 23.40 µg/dL observed in subject 7 and a maximum of 39.5 µg/dL in subject 3. Several surveys have shown that people who work in lead-related industries have higher blood lead levels than other people (Bener *et al*., 2001, Suwnsaksri *et al.*, 2002, Whittaker, 2003, Bagci *et al.*, 2004, Njoku and Ibe, 2009), Fatoki and Ayoade (1996) in Nigeria, reported the mean blood levels for the occupationally exposed group as varying between 28.5 ± 17.3 (SD), µg/100 ml, (28.5 ± 17.3 µg/dL) and 35.5 ± 15 (SD), µg/100 ml (35.5 ± 15 µg/dL) . Report by Babalola *et al.*, (2005) showed the mean blood Pb level of auto mechanics in Abeokuta-Nigeria to be 48.50 ± 9.08 µg/dL.

The mean Cd concentration in the blood samples was observed to be 7.78 ± 10.57 µg/dL with a minimum of 0.50 µg/dL observed in subject 8 and maximum of 27.30 µg/dL in subject 3. Musa *et al.* (2011) reported a similar value for exposed population in Zaria, Kaduna-Nigeria. A mean Cr concentration of 24.78 ± 21.77 µg/dL was observed, the minimum of 0.90 µg/dL observed in subject 8 and maximum of 63.40 µg/dL in subject 3.

The peak values of Pb, Cd and Cr observed in subject 3 may not be unconnected to the smoking habit of the subject, the low level of education and awareness coupled with the long period in the job as gathered from the questionnaire issued. Smoking increases the risk

of heavy metals concentration in the body and cigarette brands in Nigeria have been linked to high Cd content as reported by Iwegbue (Iwegbue *et al.*, 2009). Njoku and Ibe (2009) reported a blood Cd range of 0.058-0.0072 $mgdm^{-3}$ (equivalent to 0.72-5.8 µg/dL) for electronic technicians in Imo state -Nigeria. Appendix IV shows the concentrations of Pb, Cd and Cr in the blood samples of the test subjects.

Correlation coefficients of the metal pairs in the blood samples as shown in table 4.1 above show a significant positive correlation between Pb/Cd ($r=0.765506$, at $P<0.01$), positive correlations were also observed for Pb/Cr pair ($r= 0.838648$, at $P<0.001$) and Cd/Cr ($r= 0.853709$, at $P<0.01$). These high positive correlation values indicate the metals are likely from the same source (i.e. pollution source).

5.2 Concentrations of Pb, Cd and Cr Analyses in Urine Samples of the Subjects

The concentrations of Pb, Cd and Cr in the urine samples of the subjects are shown in Appendix V. As can be observed from figure 4.3 above, the distribution shows Pb metal being the highest in all subjects except for subjects 3 and 10 where Cr concentrations were peaked against Pb concentrations, thus the trend can generally be represented as Pb>Cr>> Cd. Subject 3 showed the highest level of Pb of 31.1 µg/dL while subject 2 showed the least level of Pb of 21.4 µg/dL, the mean value of Pb observed was 24.18 ± 2.98 µg/dL. Pb in urine (urine-Pb) is considered to reflect Pb that has diffused from plasma and is excreted through the kidneys (Barbosa *et al.*, 2005). A mean value of 6.81 ± 10.05 µg/dL was observed for Cd metal; the highest in subject 4 with a value of 26.10 µg/dL and the least value of 0.50 µg/dL observed in subject 2.

Cr metal had a mean value of 14.78 ± 14.20 µg/dL with a minimum of 0. 00 µg/dL (BDL) in subject 8 and a maximum of 40.50 µg/dL in subject 3. It could be suggested also that, the slightly higher value of Cd observed in the urine sample of subject 4 may also be linked to the smoking habit of subject 4, however, the higher value of Cd observed in the urine sample of subject 4 over subject 3 (inspite of subject 3 having a higher blood Cd level) may be related to the nature of diet and the effectiveness of the excretion of Cd from the body through urine since urinary levels are a reflection of how much metal is being cleared from blood by the kidneys during a relatively brief interval (Orlowski *et al.*, 1998).

Correlation coefficients of the metal pairs in the urine samples as shown above in table 4.2 show a significant positive correlation between Pb/Cd ($r=0.6794$, at $P<0.01$), positive correlations were also observed for Pb/Cr pair ($r= 0.6606$, at $P<0.001$) and Cd/Cr ($r= 0.8071$, at $P<0.01$). These positive correlation values indicate the availability of the metals are related and are likely from the same pollution source.

5.3 Concentrations of Pb, Cd and Cr Analyses in Nail Samples of Subjects

The table of the concentrations of Pb, Cd and Cr in the hair samples of the subjects is shown in Appendix VI. The values obtained from the analyses show Pb having the highest figures in all test subjects as captured pictorially in figure 4.4 above. The trend of the concentrations of the three metals in the nail samples can be generally represented as Pb>Cr>>Cd. The mean Pb concentration was 37.13 ± 4.08 µg/g, the minimum observed value was of 31.61 µg/g in subject 10 and maximum of 44.12 µg/g in subject 4, the result is in agreement with the reported value of Pb by Abdulrahman *et al.*, (2012) in fingernails of

welders of 0.78 to 30.44 µg/g. Nail-Pb is considered to reflect long-term exposure because this compartment remains isolated from other metabolic activities in the body (Takagi *et al.*, 1988).

Furthermore, the mean Cd concentration was 1.00 ± 1.21 µg/g with a minimum observed value of 0.09 µg/g and a maximum of 3.01 µg/g in subjects 8 and 3 respectively while the mean Cr concentration was 18.49 ± 12.71 µg/g with a minimum of 7.95 µg/g and a maximum of 41.71 µg/g observed in subjects 8 and 3 respectively, similar values were reported by Abdulrahman *et al.* (2012). The peak value of Pb in the nail samples observed in subject 4 where as peak values of Cd and Cr were observed in subject 3 could be attributed to the slightly longer duration in the job of subject 4 (22 years) over subject 3 (20 years), thereby implying a greater accumulation of Pb in subject 4. The peak values of Cd and Cr in subject 3 is likely due to the smoking habit of the subject as discussed in sections 5.1 and 5.2 above.

Correlation coefficients of the metal pairs in the nail samples as shown in table 4.3 show a significant positive correlation between Pb/Cd ($r=0.7756$, at $P<0.01$), positive correlations were also observed for Pb/Cr pair ($r= 0.8510$, at $P<0.001$) and Cd/Cr ($r= 0.9540$, at $P<0.01$). These high positive correlation values indicate the metals are very likely also from the same pollution source.

5.4 Results of Pb, Cd and Cr Analyses in Hair Samples of Subjects

The concentrations of Pb, Cd and Cr in the nail samples of subjects are shown in Appendix VII. Figure 4.5 shows Pb metal having the highest concentration in the hair samples of all the subjects. The general trend of the metals in the hair samples was observed to be similar to that observed in the blood, urine and nail samples already discussed (Pb>Cr>>Cd). The highest Pb concentration was observed in subject 4 with a concentration of 50.39 µg/g and the least in subject 10 with 33.26 µg/g. Subsequently the mean Pb concentration was computed as 39.41 ± 5.63 µg/g, the mean tallies with Babalola *et al*., (2005) figure of 17.75 ± 5.16 µg/g in auto mechanics in Nigeria.

Cd and Cr concentrations were highest in subject 3 with 3.11 µg/g and 40.92 µg/g respectively; the least values of Cd and Cr were seen in subject 8 with 0.10 µg/g and 8.25 µg/g respectively. The mean values of Cd and Cr were 1.09 ± 1.14 µg/g and 19.13 ± 11.61 µg/g respectively. The mean values of Cd is in agreement with Abdulrahman *et al.*, (2012) reported value of 0.67 to 2.06 µg/g Cd in hair samples of welders in Maiduguri-Nigeria. Smoking may be attributed to high values of Cd and Cr recorded in subjects 3 and 4 as have been reported (Creason *et al*., 1975 and Chattopadhyay *et al*., 1990). Hair and nail Pb concentrations in occupationally exposed subjects was reported to range between 1.020-409.726 and 8.130-765.306 µg/g and in environmentally unexposed subjects 0.123-25.160 and 1.076-65.613 µg/g, respectively. Similarly, hair and nail Cd concentrations in occupationally exposed subjects ranged between 0.014-22.086 and 0.214-35.714 µg/g and in environmentally unexposed subjects 0.113-1.627 and 0.028-8.108 µg/g, respectively (Mehra and Juneja, 2004).

Correlation coefficients of the metal pairs in the hair samples as shown in table 4.4 show a significant positive correlation between Pb/Cd ($r=0.9579$, at $P<0.01$), positive correlations were also observed for Pb/Cr pair ($r= 0.8974$, at $P<0.001$) and Cd/Cr ($r= 0.9238$, at $P<0.01$). The close correlation values are an indication that the metals are from same source.

5.5 Concentration of Each Metal Across the Blood, Urine, Nail and Hair Samples

The trend observed generally for each metal across the biological samples collected was; hair > nail > blood > urine, as can be clearly seen in the appendix VIII. This is as expected, as over time the bioaccumulation of the metals in the body with a corresponding low excretion will result in the high values in the hair and nail samples as observed and corresponding low values for urine samples. But blood metal concentrations is representative of the metals recently absorbed, hours to days and in some cases even weeks though may also be a reflection of metal mobilization from bone back to blood (Gulson *et al*., 1996).

For the bioavailability of Pb, the mean concentration in the blood samples was observed to be significantly higher than that in the urine samples ($t=5.781$ and $p<0.001$ at 95% CI) as expected while the mean concentration of the lead in the hair samples was observed to be also significantly higher than that in the nail samples ($t=-2.375$ and $p=0.049$ at 95% CI). Positive Pearson correlation values of $r=0.889$ and $r=0.854$ were obtained for the blood/urine and nail/hair pairs indicating their suitability as indices for bioavailability and accumulation of lead in the body. Positive correlations have been reported for metals

(Cd and Pb) in blood and urine (Shimbo *et al.*, 2000), this means both the concentration in the blood and the concentration in the urine can be used as reliable biomarkers of exposure to these metals.

Though the trend for Cd metal was similar to that of Pb, the difference observed between the mean blood Cd and mean urine Cd concentrations was not significant (t=0.417 and p=0.688 at 95% CI). So also the difference between the mean nail Cd levels and hair Cd levels was not significant (t=-0.638 and p=0.544 at 95% CI). Positive Pearson correlation values of r=0.922 and r= 0.995 for the blood/urine pair and nail/hair pair respectively show a good, strong correlation, hence indicating the suitability of the pairs as measures for the bioavailability and accumulation of cadmium in the body.

The mean concentration of Cr in the blood was much significantly higher than the mean Cr in the urine samples (t=2.941 and p=0.019 at 95% CI) but the mean Cr concentrations in the nail and hair samples were not significantly different (t=-0.441 and p=0.672 at 95% CI); blood>>urine, nail ≈ hair. The higher concentration of chromium in the blood than urine shows a low excretion of this metal from the body as the major route of excretion is through urine , it as well agrees with the high level of chromium observed accumulated in the nail and hair samples. The Pearson correlation values were strongly positive; r=0.888 and r=0.992, showing a good correlation between the blood and urine Cr concentrations and between the nail and hair Cr levels respectively, hence indicating their suitability as indices of bioaccumulation and exposure of chromium in the body.

5.6 Concentration of Pb, Cd and Cr in the Subjects by Different Categories

5.6.1 Concentration of Pb, Cd and Cr in the test subjects by social habits (smoking)

The level of lead in smoking electronic repairers is significantly higher than that of non smoking electronic repairers as shown in table 4.5, but the cadmium and chromium levels in smoking electronic repairers were much higher than those of non smoking electronic repairers ($P<0.01$), with more than a ten-fold increase in some values observed. This may not be unconnected with the high level of heavy metals in common cigarette brands in Nigeria as reported by Akpoveta *et al.,* 2011 and Iwegbue *et al.,* 2009. The average content of cadmium in cigarette available in Nigeria is 1.28 μg per cigarette and a person who smokes 20 cigarettes per day is estimated to increase his daily cadmium retention by approximately 1μg/day (0.53 – 1.65 μg/day) (Nnorom *et al.,* 2005). Tobacco leaves naturally accumulate relatively high levels of cadmium, which is volatilized during burning, and contributes significantly to a smoker's exposure to cadmium. Tobacco leaves naturally accumulate and concentrate relatively high levels of Cu (7.1 – 68.8), Cd (5.73 – 7.96), Pb (0.04 – 14.4), and Ni (2.21 – 3.45) as compared to medicinal and edible plants Cu (0.686 – 0.953), Cd (0.072 – 0.12), Pb (0.374 – 0.615), and Ni (0.31– 0.523) (Sahito *et al.*, 2003 and Arain *et al.*, 2008). Each cigarette contains from 0.5 to 2.0 μg of cadmium, 10% of which is inhaled. Smokers exhibited significantly elevated scalp hair Cd and Pb when compared to non-smokers (Afridi *et al.,* 2012). Smoking is known to increase blood cadmium level; the American Conference of Governmental Industrial Hygienists notes that smoking is a significant source of cadmium exposure and median levels in blood of smokers are between 1.4 and 4.5 μg /L (0.14 μg/dL and 0.45) as reported by Kang (Kang *et*

al., 2013), the Cd values for smokers far exceeds the normal range of Cd for smokers who are not occupationally exposed, this points to a significant contribution of occupational Cd to the total blood Cd level. The result of this research is also in line with Kowal *et al.*'s report 'Cd levels increased with were higher in cigarette smokers than nonsmokers in urine, blood, and all tissues' (Kowal *et al.*, 1979). It could be concluded that smoking in electronic repairers further exposes the workers to these metals and therefore increases the burden of these metals on the body

5.6.2 Concentration of Pb, Cd and Cr in the test subjects by education

The test subjects were grouped with respect to education into three groups; those with higher education (diploma, degree, e.t.c), those with secondary school education (high school) and those with primary school education or less as shown in table 4.6. The mean values for the concentration of the metals in the subjects based on the level of formal education, show the concentrations of Pb, Cd and Cr significantly decreased as the level of education increased. This indicates, the higher the level of education the better the practice and adherence to safety measures and as well greater hygiene in the work place. The increased education translates into increased awareness and knowledge of the possible dangers of these metals and better knowledge of safe practice. Similar relationship between education and level of exposure awareness was established in a study conducted in Mombasa to assess the health effects of lead exposure among Jua Kali (informal sector) workers: out of those who were aware of lead toxicity, 3 out of a possible 50 were from primary school representing 5%. 3 out of a possible 44 were from secondary school

representing 6.8% and 2 out of a possible 11 were from college, representing 18% (Ashraph *et al.*, 2013). The situation among electronic repairers in Nigeria is no different from that of the general population as has been reported. Limited knowledge and awareness of domestic sources of lead exposure and its health effect among the populace in Nigeria have been reported (Adebamowo *et al.*, 2006).

5.6.3 Concentration of Pb, Cd and Cr in the test subjects by type of electronics

All but one subject do repairs in general electronics (TV, Radio, pressing iron, household electrical appliances, e.t.c), one of the subjects previously worked on general household electronics but has since switched his practice to coupling and servicing of inverters for about six years prior to this study. As observed from table 4.7, the values for the concentrations of Pb, Cd and Cr obtained from the category specialized in inverters when compared with those specialized in general electronics to date were observed to be lower ($p<0.05$). Plausible explanation for the decrease in the available and accumulated Pb level may be due to the fact as gathered from the field that dealing in inverters meant less handling and use of solders however increased risk of cadmium from cadmium in batteries. The Pb levels may agree well with this explanation, but the decreased levels of cadmium and chromium observed could not be explained using this argument.

5.6.4 Concentration of Pb, Cd and Cr by use of protective devices

The level of safe practice was found to significantly affect the concentration of the metals observed as can be seen from table 4.8 above. The subjects who imbibe some safety measures were found to have reasonably lower Pb values and much lower Cd and Cr values ($p<0.05$). The values of Pb observed in users of safety devices may not be much lower compared to non users, as they leave their hands bare unprotected while working but with gas mask to protect them leads dust, the risk of lead ingestion from hands is still present

5.6.5 Concentration by length of period in the job

The distribution of Pb, Cd and Cr based on the length of period in the job is shown in table 4.9. As expected the concentrations of the metals in the blood, urine, nail and hair samples were in direct relation with the number of years in the job; the more the number of years in the job the more frequent and longer the exposure to these metals, hence, the higher the concentrations, especially in the hair and nail samples (for example, the affinity for SH-groups by Cd makes Cd likely to accumulate in keratin-rich tissues like nails and hair and consequent re-introduction into the blood). Buchancova *et al.* (1993) in their work reported 'Cd levels in blood, urine, and hair significantly increased in relation to duration of exposure to industrial metals'.

5.6.6 Concentration by source of food eaten at work and by age

From the questionnaire issued to the subjects, it was seen that all the subjects ate at least a meal at work, however, the source was different. The research attempted to analyze the concentrations of the metals as observed to investigate the influence of the source of food eaten. Source of food eaten at work was classified into three; home, restaurant and vendor as shown in table 4.10. Eating at work has generally been considered to be a cause for increased exposure to metals at work, this is basically due to the risk of ingestion of these metals while eating, for instance, workers among bridge painters who smoked and ate at work regularly had a significant increased risk of having an elevated BLL (>40 μg/dL in males and >30 μg/dL in females) (Chuang *et al.*, 1999). The values obtained with respect to the source of food eaten at place of work, show no relationship between the concentrations of the metals and source of food eaten at work. The metals primarily get into the food irrespective of source at the workshop, through the handling of the food with contaminated hands and metal dust in the workshop.

The range of the ages of the repairers was between 25 and 45, hence the subjects were grouped into four groups: 25-30, 31-35, 36-40 and 41- 45. As seen in table 4.11, Pb concentration in nails has been reported to depend on the subject's age (Nowak and Chmielnicka, 2000). From the result, a weak relationship which gets stronger with the age is observed between the concentrations of the metals and the age of the subjects i.e. there is a greater tendency of the retention (bioaccumulation) of the metals in the system with age. This may be explained by the fact that with age the function of the various systems declines, hence the ability of the body to excrete the ingested metals decreases. Kowal *et al.*

(1979) reported similar result for Cd, in the report Cd levels increased with age in urine and all tissues.

CHAPTER SIX

SUMMARY, CONCLUSION AND RECOMMENDATION

6.1 Summary and Conclusion

The results of the research show the level of exposure of electronic repairers to lead, cadmium and chromium to be of concern. The bioavailability and accumulation figures of lead, cadmium and chromium were high in the repairers; the mean values for all the metals were higher than the World Health Organization and ILO standards. The trend of the metals in the biological samples was generally found to be Pb>Cr>Cd. Hence the null hypothesis was discarded and alternate hypothesis accepted.

Personal and social habits like smoking were found to affect the concentrations of these metals. The level of education, use of safety devices, period of exposure (length of period in the job), the nature of electronics and the age of the electronic repairers were also found to remarkably affect the concentrations of the metals. However the source of food eaten at work was found to have insignificant effects on the levels of the metals present.

6.2 Recommendations

To maintain an adequate hygienic level and reduce greatly the risk of exposure to heavy metals, the following basic measures should be imbibed by electronic repairers:

- No smoking, eating or drinking in polluted areas

- There should be a separated area where eating and drinking can be done without the risk of contamination
- A protective working cloth should be used by the workers and should be kept within the working premises.
- Working clothing and street clothes should be stored separately
- It is essential to reach the electronic repairers' community with occupational health and safety training programmes specifically tailored to their needs, in order to reduce their health risks
- Occupational health and safety training must also be an integral part of electrical/electronic education in schools.
- More health care personnel should be trained on occupational health hazards
- The development of safer materials and user friendly Material Safety Data Sheet (MSDS) information by the industry should also be a priority

Substitution of a mixed diet with a vegetarian diet for 3 months is advised, such results in a tendency to increased elimination of cadmium and lead in the faeces as reported by Vahter *et al*. (1992). Rich diets in protein, iron and calcium should be eaten regularly by electronic repairers as low dietary concentrations of calcium and protein has been found to promote absorption of cadmium from the intestinal tract of experimental animals (Min, *et al*., 2008; Washko and Cousins, 1977 and Friberg *et al.*, 1975). Low iron status in laboratory animals and humans has also been shown to result in greater absorption of cadmium (Hamilton and Valberg, 1974; Flanagan *et al*., 1978).

There is the need for the Government to increase sensitization and awareness on the hazardous effects of Pb, Cd and Cr in electronic repairers and a wider approach to assess heavy metals exposure in artisan trades should be encouraged and undertaken.

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APPENDICES

Appendix I

List of Apparatus and Equipment

1. Analytical balance
2. Beaker – 50 ml, 100 ml
3. Conical flask – 50 ml, 100 ml, 250 ml
4. Cotton wool
5. EDTA tubes – 5 ml
6. Filter paper – Whatman no. 42 grades
7. Fume cupboard
8. Funnel
9. Glass rod
10. Hand gloves
11. Hot plate and Oven
12. Masking tape
13. Measuring cylinder – 5 ml, 10 ml, 100 ml
14. Methylated spirit

15. Pipette

16. Plastic bags

17. Plastic bottles for digest – 120 ml

18. Polyethylene tubes

19. Pyrex flask

20. Spatula

21. Stainless scissors

22. Syringe – 5 ml

23. Thermometer

24. Varian AA240FS Fast Sequential Atomic Absorption Spectrometer

25. Volumetric flask – 10 ml, 50 ml, 100 ml

26. Wash Bottles

List of reagents and Chemicals (Analar grade)

1. Conc. Nitric acid
2. Triton X-100
3. Acetone

4. Conc. Perchloric acid
5. Conc. Hydrogen peroxide
6. Conc. Hydrochloric acid
7. Cadmium nitrate
8. Lead nitrate
9. Sodium dichromate

Appendix II: A sample of the questionnaire issued to the subjects

Personal Data

Participant ID Number ……. Gender …….. Age …….…….....

Location……

……

Age bracket (years): < 25 ……. 25-30 ……. 31-35 …… 36-40 ……. > 40 ……..

Level of education: 1. No formal education ……. 2. Primary school……

3. Secondary school…… 4. Diploma/ degree……..

Occupational Data

Brand of electronics specialized in: Radio ……..Video CD & DVD Players ……..

Television…… Others (specify) …….

Length of period in the job (years): 6-10 …… 11-15 …….. 16-20 ……21-25……...……

26-30…….30 and above ……

Do you use lead solder: Yes …….. No ……

If no what type of solder do you use ……

What protective devices do you use at work ……..

…….

Do you have special working clothes yes ……. No……

Do you keep your dirty working clothes at your home? No …… Yes……

Are you exposed to vehicular emission at: Home ……..Work …….....

Are you exposed to dust at: Home…… Work…….

Personal Habits

Do you smoke? Never……. Rarely…… (0-10 cigarettes per day) Medium….. (10-20 cigarettes per day) Lots…… (more than 20 cigarettes per day)

Do you drink alcohol? Never…..... At least once a month…….. At least once a week…… At least once a day……

Are you exposed to vehicular emission at: Home ……..Work …….....

Are you exposed to dust at: Home…… Work…….

Diet issues

Do you eat in your working area: Yes …….. No ……..

How many meals ……Source of food: Home …….Vendors …… Restaurant …….

Nature of serving…… ……

Do you eat fish: Yes …… No ……..

How frequently do you eat fish? At least once a **month…….** At least once a **week……..** At least once a **day ……..**

Type & Source……

Drinking water source: Tap …. Well …. Borehole …. Spring ….Bottled water …….

Medical History

Any history of lead poisoning: Yes ……. No ……..

If yes when and how severe……

Nature of treatment …….

Do you experience any of these symptoms: Headache…. Nausea ……. Catarrh….. Depression..... Loss of appetite..... Intermittent abdominal pain..... Diarrhoea..... Constipation..... Muscle pain.....

Appendix III: The percentage recoveries of Pb, Cd and Cr in blood, urine, nail and hair samples

ELEMENT	RECOVERY %			
	BLOOD	**URINE**	**NAIL**	**HAIR**
Pb	98.57	99.26	98.99	99.07
Cd	97.01	96.45	96.19	96.69
Cr	94.73	98.86	98.38	95.27

Appendix IV: Pb, Cd and Cr concentrations (µg/dL) in blood samples of test subjects

Subjects	**Pb**	**Cd**	**Cr**
1	29.5	1.2	18.1
2	28.2	8.2	21.3
3	39.5	27.3	63.4
4	33.5	25.9	61.1
5	29.3	0.9	23.5
6	27.7	11.1	10.5
7	23.4	0.8	1.9
8	24.1	0.5	0.9
9	32.2	0.8	16.7
10	25.9	1.1	30.4

Appendix V: Pb, Cd and Cr concentrations (µg/dL) in urine samples of test subjects

Subjects	**Pb**	**Cd**	**Cr**
1	25.4	0.9	9.2
2	21.4	0.5	13.6
3	31.1	21.5	40.5
4	25.5	26.1	25.3
5	23.9	1.3	10.9
6			
7	22.1	0.9	1.6
8	22.2	0.7	BDL
9	23.7	0.6	1.85
10	22.3	8.8	30.1

*BDL – beyond detection limit

Appendix VI: Pb, Cd and Cr concentrations (µg/g) in nail samples of test subjects

SUBJECTS	**Pb**	**Cd**	**Cr**
1	35.02	0.40	11.36
2			
3	42.56	3.01	41.71
4	44.12	2.98	39.25
5	37.39	0.23	16.15
6	35.43	1.50	15.14
7	33.18	0.21	10.92
8	36.73	0.09	7.95
9	38.11	0.13	11.57
10	31.61	0.47	12.34

Appendix VII: Pb, Cd and Cr concentrations (µg/g) in hair samples of test subjects

Subjects	**Pb**	**Cd**	**Cr**
1	38.252	0.418	13.411
2	37.269	0.709	15.016
3	46.312	3.111	40.915
4	50.394	2.732	37.186
5	36.382	0.31	16.007
6	41.4212	1.711	14.192
7	36.245	0.235	10.973
8	35.161	0.102	8.248
9			
10	33.264	0.512	16.204

Appendix VIII: The mean concentrations of each metal in all the four samples

METAL	SAMPLE	MEAN ± SD
LEAD Pb	BLOOD	29.33 ± 4.80 µg/dL
	URINE	24.18 ± 2.98 µg/dL
	NAIL	37.13 ± 4.08 µg/g
	HAIR	39.41 ± 5.63 µg/g
CADMIUM Cd	BLOOD	7.78 ± 10.57 µg/dL
	URINE	6.81 ± 10.05 µg/dL
	NAIL	1.00 ± 1.21 µg/g
	HAIR	1.09 ± 1.14 µg/g
CHROMIUM Cr	BLOOD	24.78 ± 21.77 µg/dL
	URINE	14.78 ± 14.20 µg/dL
	NAIL	18.49 ± 12.71 µg/g
	HAIR	19.13 ± 11.61 µg/g