LEAD AND ARSENIC INADVERTENT OCCUPATIONAL HEALTH RISK ASSESSMENT IN INSTRUCTIONAL LABORATORIES IN MOI UNIVERSITY AND UNIVERSITY OF ELDORET, KENYA

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DECLARATION

DECLARATION BY THE STUDENT

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DECLARATION BY THE SUPERVISORS

This research thesis has been submitted with our approval as university supervisors.

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DEDICATION

To my dear father the late Daniel Salee who succumbed to cancer shortly before completion of this thesis.

ABSTRACT

Settled surface indoor dust is of environmental importance since it can act as a medium of human exposure to heavy metals. Universities laboratories are involved in varied activities some of which may expose arsenic (As) and lead (Pb) to workers. The objective of this study was to estimate potential health risks due to inadvertent occupational exposure to As and Pb in select instructional laboratories within Uasin Gishu County, Kenya. The research employed a cross-sectional study design. Sampled indoor settled dusts in ten sampling stations from Moi University and University of Eldoret were analyzed for Pb and As concentrations using F-AAS and XRF, respectively, alongside a descriptive study on laboratory safety and hygiene. Univariate data analysis, one-way ANOVA and t-test were done to describe and ascertain variations and the results compared with internationally stipulated standards. Estimation of occupational health risk was done in accordance with risk assessment models as described by U.S. EPA. The study found out that there were no vacuum cleaning equipment, waste collection schedules and occupational injuries and illness form. The facilities lacked a risk assessment tool, electronic inventories of safety data sheets (SDS) and personal protective equipment (PPE) were not only inadequate but also poorly maintained. Mean Pb levels ranged from 344.890±12.267-754.438±76 mg/kg, which were significantly above WHO/FAO: EU: U.S. EPA (95% CI: $p = 0.000$: $p = 0.000$-0.01197: $p = 0.000$-0.0991) recommended standards, respectively. Mean As levels ranged from 0.42-131.73 mg/kg, which were significantly lower ($p = 0.0121$-0.998) in most (80%) sampling stations than EU/FAO/WHO standards while 60% of the stations significantly ($p = 0.024$-0.795) surpassed U.S. EPA standards. Non-Carcinogenic risk for Pb HQ results in the entire study area were found to be above unit ($p = 0.048607$; $p = 0.00413$). These results were in agreement with both central tendency exposure (CTE) and reasonable maximum exposure (RME) non-carcinogenic risks. However, As HQ results in the entire study area were found to be less than unit ($p = 0.243459$; $p = 0.20453$) for men and women, respectively. Aggregate HI were significantly above unit ($p = 0.053234$ and $p = 0.004819$). Comparison with U.S. EPA's acceptable excess lifetime cancer risk (ELCR) of $1x10^{-6}$ - $1x10^{-4}$ in the entire area found out that men Pb risk was within acceptable levels ($p = 0.382236$) while women risk was significantly higher ($p = 0.035785$). The CTE and RME carcinogenic risks for Pb were both within ELCR levels. Arsenic cancer risk was within acceptable ELCR levels ($p = 0.180078$; $p = 0.155792$), however, CTE and RME cancer risks were above ELCR acceptable levels for men and women, respectively. Aggregate risks were all above acceptable risks. The study concluded that RMD work-unit was found to be the most exposed work-unit for both Pb and As cancer and non-cancer risks and that instructional laboratories are not entirely safe from Pb and As exposure risks. The study recommends that universities adhere to laboratory safety rules, come up with chemical hygiene plans (CHP) and process-specific risk assessments.

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LIST OF ABBREVIATIONS, ACRONYMS AND SYMBOLS

ABSder	Dermal absorption fraction
ACGIH	American Conference of Governmental Industrial Hygienists
ADI	Average Daily Intake
As	Arsenic
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
BW	Body Weight
CDI	Chronic Daily Intake
CDCP	Centre for Disease Control and Prevention
CDPH `	California Department of Public Health
CF	Contact Frequency
CSF	Cancer Slope Factor
CTE	Central Tendency Exposure
EASHW	European Agency for Safety and Health at Work
ED	Exposure Duration
EF	Exposure Frequency
ELCR	Excess Lifetime Cancer Risk
EPC	Exposure Point Concentration
EU	European Union
fdo	fraction transferred from dermal to oral
fgi	Gastro-intestinal absorption fraction
HI	Hazard Index
HIF	Human Intake Factor
HQ	Hazard Quotient

HUD	Housing and Urban Department of the US
IPCS	International Programme on Chemical Safety
IR	Intake Rate (also ingestion or inhalation rate)
kg	Kilogram
LADI	Lifetime Average Daily Intake
NIOSH	National Institute of Occupational Safety and Health of the US
NOAEL	No-Observable Adverse Effect Level
NORD	National Organization for Rare Disorders
MU	Moi University
MUMC	Moi University Main Campus
OSHA	Occupational Safety and Health Administration of the US
REL	Recommended Exposure Levels
RfC	Reference Concentration
RfD	Reference Dose
RME	Reasonable Maximum Exposure
SA	Surface Area
SBPS	School of Biological and Physical Sciences of Moi University
SES	School of Environmental Studies; University of Eldoret
TE	Transfer Efficiency
UCF	Unit Conversion Factor
UoE	University of Eldoret
U.S. EPA	United States Environmental Protection Agency
WHO	World Health Organization

OPERATIONAL DEFINITION OF TERMS

Absorption Fraction: unitless relative quantity of a chemical or percentage absorbed that dermally penetrates into the body

Average Daily Intake: Dose rate averaged over a pathway-specific period of exposure expressed as a daily dose on a per-unit-body-weight basis. The ADI is used for exposure to chemicals with non-carcinogenic non-chronic effects and usually expressed in terms of mg/kg-day or other mass/mass-time units, generally presented as an administered dose, not an absorbed dose**.**

Cancer slope factor: estimated chance that a person will develop cancer upon ingesting 1 mg/kg-day of a carcinogen for a 70 year lifetime

Central Tendency Exposure (CTE): refers to those individuals in a population who are typically or averagely exposed to a chemical in an environmental medium.

Chronic reference dose (RfD): An estimate of a daily oral exposure for a chronic duration (up to a lifetime) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Excess Lifetime Cancer Risk: a contaminant contained in a specific environmental medium whose chronic dose concentration exposed over a 70-year lifetime, corresponds to a maximum risk level of one excess cancer in a million people

Exposure: the contact of a physical, chemical or biological agent with the body of an organism. It is quantified concentration of the agent in the environmental medium in contact with the organism integrated over time duration of the contact.

Exposure assessment: The process of estimating or measuring the magnitude, frequency and duration of exposure to an agent.

Exposure duration: Length of time in years in which the organism is in contact with the chemical.

Exposure pathway: the inhalation, dermal or oral process by which an organism is exposed to a contaminant present in an environmental medium.

Exposure scenario: A set of assumptions, facts and inferences about how exposure takes place to aid in estimating, evaluating or quantifying exposures.

Inadvertent exposure: Accidental or unintentional contact of a chemical, physical, or biological agent with the outer boundary of an organism.

Indoor dust: settled indoor material, consisting of both soil and non-soil derived materials

Industrial hygiene: is a science and art of keeping people safe and healthy at work and in their devoted to the anticipation, recognition, evaluation, prevention and control of those environmental factors or stresses arising in or from the workplace which may cause sickness, impaired health and wellbeing, or significant discomfort among workers

Instructional laboratory/workshop: A room or place with appropriate equipment for teaching science where a group of students simultaneously receive instruction in and perform experimental procedures associated with a formally approved university academic course in Kenya

Lifetime average daily intake: Dose/intake rate expressed in terms of mg/kg-day or other mass/mass-time units for chemicals with chronic carcinogenic effects usually averaged over 70 years lifetime duration.

Minimal Risk Level: an estimate dose of a substance that on daily human exposure is unlikely to have an appreciable non-carcinogenic risk of adverse health effects over a specified duration of exposure.

Occupational tenure: aggregate number of years a person has worked in his or her current occupation, irrespective of the interruptions in employment, the number of employers or time spent in other occupations.

Reasonable Maximum Exposure (RME): Also known as "high end" exposure refers to people who are at the high end of the exposure distribution (approximately the 95^{th} or 90^{th} percentile). The RME scenario is intended to assess exposures that are higher than average, but are still within a realistic range of exposure.

Risk: the chance that a substance once taken up in the body of the organism in the conditions of its use, will likely cause deleterious health effects or illness. The higher the frequency, duration, amount used and severity of the hazard of exposure, the higher the risk to health.

Risk characterization: also known as risk estimation, is the process of quantifying risk after considering the exposure in order to make conclusion on the effect

Soil adherence: The quantity of contaminated soil that comes into contact and adheres to the skin and from which chemical contaminants are available for uptake at the skin surface.

Work unit: groups of workers believed to be exposed to similar health risks due to their work predisposition: performing similar tasks in a similar workplace and are exposed to the similar chemicals which pose similar risk to health.

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CHAPTER ONE

INTRODUCTION

1.1 Background information

Heavy metal refers to any naturally occurring element whose atomic density is greater than 4g/cm^3 or with atomic weights higher than 40.04 g mol^{-1} or is 5 or more times greater than that of water. The term has been particularly applied to metals such as cadmium(Cd), mercury(Hg), Arsenic(As) and lead (Pb), all of which are listed in the World Health Organization's (WHO, 2010a) top ten chemicals of priority to public concern. Others such as zinc(Zn), chromium(Cr), copper(Cu), manganese(Mn), cobalt(Co), nickel(Ni), selenium(Se), antimony(Sb) and thallium(Tl) are also regarded as heavy metals (Banfalvi, 2011; Ferguson *et al*., 2018). Exposure to heavy metals poses varying health challenges to humans and other life forms. Heavy metals present in soil are known to exhibit a relatively low mobility. Because of their both anthropogenic and natural origin, heavy metals are ubiquitous in the environment; therefore exposure to humans occurs through many pathways (Ali *et al*., 2019).

Dust is an important exposure pathway of heavy metals in humans. Dust can be described as a solid material comprising of soil, anthropogenic metallic components as well as natural biogenic materials (Gorman *et al.*, 2016). Once in the soil, metals tend to have a relatively low mobility but may enter and attach to dust particles. Past studies (Sahu *et al.*, 2018; Othman *et al*., 2019; Wei *et al*., 2018; Ali *et al*., 2012; Rohra *et al*., 2018; Darus *et al*., 2017; Mercier *et al*., 2011; Etim and Onianwa, 2012; Bijkerk, *et al*., 2006) on indoor dust have indicated that it has been in use as an environmental medium for

assessing human exposures to a constellation of various indoor chemical compounds. These contaminants include metals, persistent organic pollutants, radionuclides and allergens. Based on these studies, dust can be considered as an important medium of health hazards to populations in varied settings.

Indoor dust has been recognized as one locus of pollutants containing contaminants that have adverse human health effects. Trace elements, in particular heavy metals have been found in levels that may significantly pose adverse human health impacts (Kathryn *et al*., 2016; Hochstetler *et al*., 2011; Aucott and Caldarelli, 2012). Indoor settled dust has been found to be a composite of particulate matter that originates from both indoor and outdoor sources. Further, the presence of heavy metals in settled indoor dust have several sources in most cases are depended on the anthropogenic activities that take place in the interior environment, as well as exterior sources which find their way into buildings in addition to the location and condition of a building (Jaradat *et al*., 2004; Latif *et al*., 2009; Lucas *et al*., 2014. Pekey *et al*., 2012).

Although settled surface indoor dust has often been found to function as a reservoir of hazardous particulate contaminants including trace metals, the bulk transport of outdoor soil adhering to clothing and shoes could also contribute to indoor dust. According to Krupnova *et al.,* (2019) once in the indoor dust, humans' within the vicinities can accumulate these metals in their bodies through various routes of exposure posing potentially deleterious health effects. These exposure routes include dermal contact absorption, direct ingestion and inhalation.

The overall importance of heavy metals cannot be gainsaid because they are of greatest concern in any physical, chemical and biological environment. In particular exposure to cadmium, arsenic, mercury and lead heavy metals have been found to pose the main adverse effects to human health (Durand *et al*., 2015). These heavy metals have attracted most research interests and their implications on human health regularly reviewed by renowned international bodies such as the World Health Organization (WHO). Despite this, these heavy metals have been in use for quite a long time in human history. Although several adverse health effects of heavy metals have been not only known for a long time, but also the information widely disseminated, exposure to these heavy metals continues.

Arsenic as an element occurs naturally in the environment. Its concentration in the earth's crust has been reported to be approximately 0.0002%. Arsenic which has been ranked number one on the Agency for Toxic Substances and Disease Registry (ATSDR) "Top 20 List" is the most known cause of acute heavy metal poisoning in adults. Inorganic arsenic has been classified as a known human carcinogen by U.S. EPA (ATSDR, 2015). Inorganic arsenic for instance has been widely used in the wood industry where it is used as a preservative in the form of chromate copper arsenate (CCA). Arsenic has also been used in soaps, metals semi-conductors, paints, glassware, dyes, drugs, agricultural products and applications, as well as in industrial and electrical utilities.

Arsenic as an element does not easily break down therefore it's persistent in the environment with more than 45 years in soil, but it can change from inorganic to organic forms. Besides chemical reactions such as oxidation-reduction reactions, other various

natural processes such as bio-transformations and ligand exchange reactions affect its transport and fate in water and soil. In air, arsenic may disperse but eventually settles out and deposits in outdoor soils or indoor dust (Singh *et al*., 2015).

Lead element naturally occurs in the environment and is known to account for 0.0013% of the earth's crust. Lead ranks number two on the ATSDR's "Top 20 List" and has been reported to account for most of the cases of pediatric heavy metal poisoning (ATSDR, 2019). Its inorganic salts account for most of the lead emitted into the atmosphere. Exposure to the inorganic form of lead may occur through ingestion and drinking of lead contaminated food and water. Moreover, exposure via paint chips, air, soil and dust significantly contribute to the overall exposure. Direct inhalation of lead has been found to account for a small percentage of the aggregate human exposure. When it is airborne, lead along with dust settle onto clothing, water, food and other indoor and outdoor surfaces, and may subsequently be transferred to the perioral area (Check and Marteel-Parrish, 2013). Lead has been classified as a human mutagen and probable carcinogen (ATSDR, 2019).

Common sources of lead exposure in recent years include residual pollution, occupational settings or environmental contamination. Metallic lead has been widely used in the manufacture of cables, storage batteries, ammunition, steel and solder products, electronic equipment and computers circuit boards, radiation and x-rays shielding appliances and superconductor and optical technology. Inorganic lead salts have been extensively used in ceramics, plastics, pigments, enamels, insecticides, glass, paints and rubber products (Tchounwou *et al*., 2012).

Dust though mostly ignored in exposure studies as a significant environmental medium, can however, provide important information on the distribution and fate of chemical substances present on the surface environment as well as their concentrations. The composition of settled dust has been shown to be similar to atmospheric suspended particulates implying it can therefore be used as an indicator of pollutants such as heavy metal pollution in the atmosphere. In the indoor environment, evaluation of the settled dust may give the level of heavy metal concentrations and extrapolations done for human exposure assessment (Check and Marteel-Parrish, 2013).

In dusty environments, several past studies gave an estimate of up to 100 mg as the amount of dust that adults could ingest in a day (Calabrese *et al*., 1990; Leung *et al*., 2008; U.S. EPA, 1996, 1997). However, many of the U.S. EPA risk assessment studies for industrial settings have assumed that an adult could ingest 50 mg/day while 100 mg/day has been assumed for residential and agricultural scenarios. Thus, 50 mg/day is the recommended value in the U.S. EPA (2011) handbook for indoor workers besides representing a central tendency estimate of adult soil/dust ingestion.

Earlier investigations into work related exposures to hazardous substances in dust have put more emphasis on inhalation route exposure (IPCS, 1998). However, research in later years took into consideration the importance of other routes of exposure. Considerable amount of research has been conducted on inadvertent ingestion and dermal exposure. For example, it has been realized that Pb could pose health risks as a result of the oral exposure pathway for a myriad of chemical substances such as pesticides and pharmaceuticals. This has led to the birth of occupational hygiene programmes which

were designed to minimize and prevent contaminants from spreading. However, this did not take into consideration the complexity and mechanisms by which inadvertent ingestion exposures occur and its significance to the overall exposure (Cherrie *et al.*, 2006; Gorman *et al*., 2017).

Virtually, all occupations have some level of inherent of hazards inherent in them. Studies on occupational exposures and risk characterization to heavy metals have mainly concentrated on industrial settings. Owing to the nature of instructional laboratories activities in universities, occupational exposure to heavy metals cannot be ruled out. This study therefore was aimed to contribute to the knowledge pool by seeking to address the potential of settled indoor dust in instructional laboratories as a medium of As and Pb heavy metals occupational exposure and health risks.

1.2 Statement of the problem

Instructional institutions including universities have both dry and wet laboratories. These laboratories often present a wide range of activities some of which pose heavy metal exposure and health risks to staff and students working in these environments. Suspected arsenic poisoning for instance has been reported at the University of Southampton's chemistry department building (https://sotontab.co.uk/). Besides instructional laboratory sessions, these laboratories are heavily involved in research which may require the use of products with the potential to release heavy metals in the work environment.

In the recent past, research on heavy metals pollution in various environmental scenarios has seen an increasing trend (Radaideh *et al.,* 2017; Ogidi *et al*., 2017, Ackova, 2018). Documented health effects include cancer, anaemia, encephalopathy, ataxia,

hyperkeratosis, nephropathy and peripheral neuropathy (NORD, 2015). However, many studies on occupational exposure to heavy metals have basically measured industrial indoor air quality and blood levels, indoor dust only being a concern in residential buildings and children's playing grounds (Middleton *et al*., 2018; Junaid, 2017; Ondayo *et al*., 2016; Lu *et al*., 2014; Latif *et al*., 2014).

Whether there has been occupational health and safety monitoring in Kenyan public university's instructional laboratories and especially in indoor dust cannot be ascertained by any documented evidence. Thus occupational exposure to these heavy metals cannot be ruled out. This study therefore sought to determine the concentration of As and Pb in Moi University and University of Eldoret instructional laboratories indoor dust and assess the potential occupational health risks arising from inadvertent chronic exposure to these heavy metals.

1.3 Objectives

1.3.1 General Objective

To determine heavy metals (As and Pb) in selected instructional laboratories settled indoor dust and estimate the potential occupational exposure health risks in Kenyan public universities within Uasin Gishu County.

1.3.2 Specific Objectives

The study was undertaken with the following specific objectives:

1. To determine laboratory hygiene and safety practices at Moi University and University of Eldoret instructional laboratories.

2. To determine As and Pb concentrations in Moi University and University of Eldoret instructional laboratories settled indoor dust
3. To estimate non-carcinogenic occupational exposure to As and Pb in Moi University and University of Eldoret instructional laboratories
4. To characterize occupational As and Pb exposure risks among instructional laboratories staff
5. To determine As and Pb central tendency exposure (CTE) and reasonable maximum exposure (RME) risks due to work predisposition in the selected instructional laboratories.

1.4 Research questions

1. How is the laboratory hygiene? What are the safety practices in place in the instructional laboratories?
2. What are the concentrations of As and Pb in settled indoor dust in the instructional laboratories?
3. Are there non-carcinogenic occupational exposures to As and Pb from indoor dust in the selected laboratories?
4. Are the instructional laboratories workers occupationally exposed to Pb and As risks?
5. What are the As and Pb CTE and RME risks due to work predisposition in the selected instructional laboratories?

1.5 Justification

In the absence of studies that characterize risks from the potential occupational exposure to heavy metals and in indoor settled dust in instructional laboratories, it is envisaged that the research findings study from this may be utilized as a screening study or a baseline survey to monitor and evaluate workers health in the studied institutions. The study may also be pivotal in policy formulation and implementation with regard to laboratories safety policy in research institutions. Further the instructional laboratories workers may benefit from the research findings and the safety recommendations therein.

1.6 Scope of the study

This study focused only on instructional laboratories even though many other universities activities may pose occupational heavy metals exposure risks. For the purpose of this study, only Moi University Main Campus (MUMC), Moi University Rivatex and the University of Eldoret (UoE) were used for the study. This study considered exposure to heavy metals Pb and As through inadvertent dust ingestion, dermal contact, and dermal contact with subsequent inadvertent ingestion. Inhalation exposure pathway was not considered since direct inhalation in indoor environments accounts for only a small part of the total human exposure ((ATSDR, 2007).

1.7 Assumptions of the study

The study assumed that workers remained in their designated duty stations for 8 hours a day 5 days a week as required. The weights of adult men and women have been assumed to be 70 kg and 60 kg, respectively. Further, it was assumed the individual employee would have an average occupational tenure of 30 years before retirement age.

1.8 Limitations of the study

No similar research on the same environmental scenario and medium was found hence comparison of results to previous research was not possible. As is common with many occupational exposure studies, the data on ingestion and/or dermal contact was not accompanied with biological monitoring hence associations could not be ascertained.

CHAPTER TWO

LITERATURE REVIEW

2.1 Background information

The World Health Organization (WHO, 2010a) estimated that environmental factors accounted for more than 25% of the global burden of disease; this includes exposures to hazardous chemicals. Worldwide, exposure to Pb for instance has been reported to account for 2% of the ischemic heart disease burden and 3% of the cerebrovascular disease. Further, approximately 5% of the global burden of lung cancer has been attributed to outdoor air pollution while 9% is attributed to occupational exposure to toxic substances. The WHO further reported that an estimated 355, 000 people succumb each year to unintentional poisonings. Two thirds of these deaths occur in developing countries, as a result of immoderate exposure to, and lack of appropriate use of environmentally unfriendly chemicals especially pesticides; these figures were anticipated to change positively with time.

The potential adverse risks to the public that can be caused by chemical contaminants are not only well known but have also been documented for many years, however, these problems remain scantily addressed. In the developing world for instance, it is projected that utilization of toxic chemicals is bound to increase which will likely lead to increased adverse health effects unless it is accompanied with chemical sound management programmes. Due to the typical lack of resources for chemical risk management, these problems persist especially in developing countries such as Kenya (Pruss-Ustun *et al*., 2017).

Because heavy metals have both anthropogenic and natural sources, they are therefore ubiquitous in the environment. According to Berasaluce *et al*., (2019), human exposure to heavy metals occurs via many pathways and various environmental media. Dust has been recognized as a very significant environmental medium of metal human exposures. Dust has been described as a solid matter comprising of majorly soil, anthropogenic metallic constituents as well as natural biogenic materials. Dusts are usually solid particles, with a range of below 1μm up to at least 100μm in size (Gorman *et al.*, 2016). Dusts have been reported to become airborne but this depends on their source, physical characteristics and prevailing environmental conditions. Dust emanates from various sources which include soil, pesticides, asbestos, bacteria, pollen, abrasion of materials, shed skin, cigarette smoke and dust mites and is found either settled onto surfaces or suspended in the air (Morawska and Salthammer, 2006).

Indoor as well as outdoor dust is known to make an instrumental contribution to environmental heavy metal contamination. Indoor dust has commonly been found to contain a mixture of contaminants in the particulates which may form an invisible toxic hazard and this should be of great concern (Al-Momani, 2007). Since most people prefer to spend as much as over 80% of their time in built in environments, heavy metal indoor contamination occupational and non-occupational through indoor source emissions, deposition to indoor surfaces and filtration has seen an increase in concern (Hussein *et al*., 2013).

Therefore, As and Pb being contaminants of concern, several countries globally and regulatory bodies such as Food and Agriculture Organization (FAO), WHO, U.S. EPA as

well as individual countries have established maximum allowable limits of heavy metals in soils as shown in Table 2.1.

Table 2.1: Maximum Allowable Limits of Heavy Metals Concentrations in Soil (mg/kg) for Different Countries

Country	As	Pb	References
Germany	50	70	Lee and Lee, 2011
Poland	n.a	100	Mtunzi *et al*., 2015
U.K	32	450	http://www.yara.co.uk/6_Heavy_Metals
Australia	20	300	Environment Protection Authority, Australia, 2007
Taiwan	60	300	Lee and Lee, 2011
Bulgaria	10	26	Atanassov, 2007
Canada	20	200	Canadian Min. of Envt, 2009
China	30	80	Envtal. Protection Min. of China, 2015
Tanzania	1	200	He *et al*., 2015
FAO/WHO Guidelines	20	100	Chiroma *et al*., 2014; WHO, 2010b; WHO, 2012
EU Guidelines	20	300	European Comm. on Envt, 2002
South Africa	5.8	20	Dept of Envtal Affairs, SA, 2010
U.S. EPA Guidelines	7	400	U.S. EPA, 2018
New Dutch List	29	85	Adaramodu *et al*., 2012

(Source: Author, 2019)

2.2 Occupational laboratory safety and hygiene

In Kenya, the Occupational Safety and Health Act (2007) provides guidelines for the management of occupational safety and health matters, specifically to safeguard worker safety, health and welfare. Enacted in 2007, the Act is complemented by several subject specific subsidiary legislations in form of legal notices with rules on first aid, hazardous substances, safety committees, medical examinations, plant and equipment inspections among the few.

Sections 14, 21 and 22 of the Act, specify that accidents, diseases and dangerous occurrences need to be recorded on the general register and gives guidelines for reporting of the same to the Directorate of Occupational Safety and Health Services. On general health provisions, section 47 specifically requires that every workplace be kept clean. On chemical safety, there are clear guidelines in sections 83 to 90 ranging from handling, transportation, labeling as well as prevention of exposure through provision of exhaust systems. General welfare requirements stipulated in sections 91 to 96 include the provision of appropriate PPE and their accommodation facilities, potable drinking water, first aid facilities and permit to work system for hazardous tasks.

Under the US Occupational Safety and Health Act (1970), every employer is required to provide a safe and healthful workplace. Often, instructional laboratories have numerous researches involving many researches and this often requires the sharing of common space, and at times chemicals and equipment. As such it is good that such facilities establish, communicate, document and comply with laboratory-specific manuals and rules. These rules should, however, be all inclusive and detailed as opposed to chemical hygiene plans.

2.2.1 Indoor Surface Contamination Criteria

The Occupational Safety and Health Administration (OSHA) of the US standards describes housekeeping provisions which among other things addresses surface contamination issues and mandates that “surfaces be maintained as free as practicable” of accumulations of the regulated substances (OSHA, 2008). The National Institute of Occupational Safety and Health (NIOSH) recommended exposure levels (RELs) do not

address indoor surface contamination either, nor does American Conference of Governmental Industrial Hygienists (ACGIH). Since general quantitative relationships between surface contamination and air pollutant concentrations have not been ascertained, the use surface dust wipe samples can therefore be applied to determine if surfaces of concern are as 'clean as practicable'. According to NIOSH (2008), a totally insignificant inhalation dose is associated with ordinary cleanliness, based on surface contamination remaining after ordinarily thorough and appropriate cleaning of the surfaces.

Federal standards are yet to identify and adopt an exposure limit for lead contamination especially on occupational related surfaces. With reference to a letter (Fairfax, 2013), OSHA's Directorate of Compliance Programs indicated the requirements of OSHA's standard for lead (29 CFR 1926.62(h) (1), 1926.62(i)(2)(i) and 1926(i)(4)(ii) interpreted the level of lead-contaminated dust allowable on workplace surfaces. The agency recommended "that all surfaces shall be maintained as free as practicable" of accumulations lead and other contaminants of concern. Employers were further directed to ensure their staff have access to clean changing areas especially for employees whose airborne exposure to lead was found to be above the permissible exposure limit. Clearly delineated lunchroom facilities and eating areas are also supposed to be provided and maintained "as free as practicable" from any lead contamination.

Further, in exceptional cases where workers may directly come into contact with indoor surfaces such as, storage facilities, worktop surfaces, lunchroom and eating facilities, floors in change rooms, known to be contaminated with lead, OSHA does not expect their

surfaces to be any clean. OSHA therefore indicated that for other surfaces, it was rather difficult to set a specified level to describe “how clean is clean", nor the level of Pb contamination that could suitably define strive to address specific but different challenges with an aim to keep lead-surface contamination to a minimum hence the term ‘practicable’.

It was also OSHA’s view that a housekeeping program which is as rigorous as ‘practicable’ is necessary in many jobs to keep airborne lead levels below permissible exposure conditions at a particular site. With particular regard to attic contaminated surfaces indicated that they must be cleaned (or alternative methods used such as sealing the lead in place), as necessary to mitigate lead exposures. OSHA emphasized that the intention of the provision was to keep a check on employers’ frequency of cleaning and other appropriate housekeeping activities.

DiBiasio and Kimko (2013) further recommended on frequent cleaning order to prevent avoidable lead exposure and the likely potential exposure that can be caused by re-entrainment of lead dust. Overall, the intention of the "as-free-as-practicable" provision was meant to ensure that lead accumulation in dust does not in any way become a potential source of occupational lead exposure. OSHA therefore has stated that any appropriate method of housekeeping that achieves this provision is acceptable. No information was found specifically touching on lead contamination exposure limits of surfaces in universities instructional laboratories.

2.2.2 Chemical Safety and Personal Hygiene

Laboratories are required to possess a documented specific Chemical Hygiene Plans (CHP). All laboratory personnel are therefore supposed to undertake and complete all required training, be conversant with the laboratory specific CHP and a risk assessment conducted before for the use of the chemical. The risk assessment should in effect evaluate the need and proper application and usage of occupational hazard and risk control measures such as engineering and administrative controls as well as appropriate PPE (personal protective equipment). OSHA encourages that the risk assessment be documented in a laboratory notebook or in any other written appropriate procedure (OSHA, 2012).

The House Safety Executive (HSE, 2010) of the UK has also documented on occupational health and safety in engineering workshops describing how most frequent and serious occupational hazards arise, how to carry on risk assessments in addition to how to control and/or eliminate them. Further, several universities the world over have documented laboratory safety and CHP's. Among many others, these include the University of Chicago (https://researchsafety.uchicago_Chemical_Hygiene_Plan accessed on March 16, 2019), Indiana University (https://lab-safety-chemical-hygiene accessed on March 19, 2019) and the University of New Mexico (https://employee-safety/chemical-safety accessed on March 20, 2019). Of essence the basis of a chemical safety and hygiene should encompass adherence to chemical-specific handling requirements which can be accessed by referencing the chemical's safety data sheet (SDS).

Personal hygiene is of paramount importance when working in a laboratory. As per the CHP's, all lab personnel are required to observe personal hygiene measures such as washing of their hands always after handling chemicals, before any snacking and leaving for lunch and at the end of each workday. Further, consumption and storage any form of food or drink is absolutely prohibited in laboratories. It is also advisable that long hair as well as loose clothing be confined in order to prevent any accidental contamination. Further, any form of cosmetics including lip balm or even skin lotions should not be used in the laboratory (HSE, 2010)

2.3 Use of dust for heavy metal exposure assessments

Among the environmental media of human exposure that are used to trace metal contaminants in the indoor environment, dust has largely been ignored. However, dust may comprise of sinking airborne particles, soil dust, vehicle exhausts, house dust and aerosols that maybe airborne or transported by water hence making a significant contribution to environmental pollution. Most studies of heavy metals pollution via dust have focused largely on dust deposited on roads (Mishira *et al*., 2018; Soltani *et al*., 2015; Faiz *et al*., 2012; Liu *et al*., 2014; Shinggu *et al*., 2010). A study by Hejami and Ahmed (2014) on settled indoor dusts in Toronto, Canada reported that highest level of heavy metals were in the schools laboratory dusts as when compared to household, office and classroom dusts.

Besides, many studies on street dust in the past have mainly concentrated on the concentrations of the elements and identifying their sources (Zglobicki *et al*., 2018; Mohammed and Crump, 2013). Studies have shown that soil particles can indirectly or

directly be transformed into interior house dust subsequently being ingested by children and adults through inadvertent hand-mouth contact, dust inhalation or even geophagia (Gorman *et al.*, 2017).

Indoor settled dust presents as a composite of particulate matter derived from both indoor and outdoor sources (Mohammed and Crump, 2013). The settled surface dust has been reported to often function as a reservoir of hazardous particulate contaminants including trace metals. A study on heavy metals in public primary schools classrooms dust in Nigeria for instance showed that, Cr, Cd and Pb were found in the investigated samples however the metals concentrations detected in classroom dust were found to be much lower when compared to some levels reported concentrations for roads dust of some renowned world cities (Popoola *et al*., 2012).

In spite of this, occupational exposure criteria have mainly put more emphasis on airborne concentrations of several heavy metals. Settled surface soils and dusts can, however, be of particular importance as environmental media of human exposure to heavy metals since eventually both media act as repositories for the air-borne metal particulates. In this case, human exposures via the settled surface dust or soil exposure scenarios have been known to occur long after emissions of airborne trace metals have ceased. There is great evidence that there has been human exposure to metals in soil and surface dust via oral, skin contact with dust and soil and also by inhaling airborne dust particles. Though the ingestion route majorly accounts to risk at sites contaminated with metals, the importance of the dust ingestion route is not specific to only metals. However, due to uncertainties in estimates of surface dust ingestion that exists though this pathway,

this can contribute significantly to the uncertainties in risk and exposure assessments of metals in dust (Wilson *et al.,* 2013).

In occupational settings, surface indoor dust samples have often provided vital information in two occasions; first, hands of the employees can inadvertently come into contact with settled dust on a surface and then be subsequently orally taken up when transferred from hand-to-mouth; and secondly, when the contaminant on the surface can be dermally absorbed if the skin comes into contact frequently with the contaminated surface dust (Gorman *et al.*, 2016).

Once exposed to heavy metals in the dust, humans can accumulate them in their bodies via direct dermal contact absorption, inhalation or ingestion with the potential to pose deleterious effects. Studies have reported adults to exhibit lower risk when compared to children. This is because, children are known to engage more in hand to mouth activities, while their neurological system is still developing and have been reported to have a much higher absorption rate of heavy metal as compared to adults (Moya and Phillips, 2014).

According to Lioy *et al*., (2002), subject to the frequency of cleaning, the sampled materials found on the surfaces can be indicative of accumulations resulting from many varied activities that may have taken place over a long range of time periods. Frequently cleaned surfaces (such as kitchen worktops) often in most cases indicate most recent deposition whereas surfaces that remain undisturbed for long periods (such as tops of refrigerators) reflect materials deposits over a significant long period of time. Common play area surfaces and toys for instance, can be indicative of surfaces for objects which are most frequently in contact by either adults or children. Deposits on wells and

windowsills can also provide vital information on substances that find their way from exterior sources to interior environments or may also be indicative of materials as a result of flakes of paint from the old surfaces around the windows.

Further, Shraim *et al*. (2016) indicated that surface sampling can provide a wealth of material that can be harnessed for estimation of potential contact with any levels of contaminants of concern. For instance, it can provide information on acute and chronic health effects, identification of sources of the toxicants as well as aggregate or cumulative exposure assessments. However, because of the low amount of dust normally found on many surfaces under study, this may result into inability to identify sources of the contaminants.

Previous studies have also reported settled dusts to have been found as a suitable surrogate for indoor air pollution. Thus, it can be collected for monitoring of human health since it also constitutes a direct route of human exposure to re-suspended airborne contaminants in particulates through inhalation (Morawska, 2004) of settled dust as well as ingestion through contaminated food or by hand-to-mouth contact (Schripp, 2008).

Because of their risk factors, many past and recent studies on heavy metal exposure via indoor dust in educational institutions have mainly targeted elementary school going children (Tan *et al*., 2018; Popoola *et al*., 2012; Latif *et al*., 2014; Ondayo *et al*., 2016). Heavy metals have also been reported in residential buildings. In a study done in Malaysia for instance, the results showed that the range of heavy metals observed in residential buildings at Seberang Prai Tengah were in the range of 2.20-14.00 mg/kg, 1.50-32.70 mg/kg, 1.50-76.80 mg/kg and 14.60-54.40 mg/kg for Cu, Ni, Pb and Zn

respectively, while the heavy metal concentrations in the investigated areas followed the order: Pb > Zn > Ni > Cu (Abdul-Wahab, 2012).

2.4 Occupational exposure to lead and arsenic

Dinman and Dinman (2000), have documented that for chemicals to have a toxic effect on the body they must first pass across a functional barrier separating the environment from the internal organs. Human exposure studies clearly describe how chemicals in the environment enter into the human body, commonly: inhalation with the barrier being the lining of the lung; dermal absorption with the *stratum corneum* as the barrier and ingestion with the wall of the gastrointestinal tract as the barrier. With regard to potential toxicity effects, the inhalation route of exposure has been reported to be the significant route with regard to potential toxicity effects, then skin contact with chemicals with the ingestion route ranked last (WHO, 2010a).

In the workplace, harmful substances may come into contact with and enter the workers bodies through breathing (inhalation route), by contacting contaminated surfaces and subsequently passing through the skin (dermal route) as well as sometimes by swallowing (ingestion route). The dermal route is the exposure pathway of interest when it comes to exposure to agents on surfaces. Contaminated surfaces including walls, large objects to small particles and floors within reach are capable of dermal contact. Besides the skin in itself is a surface likely to be contaminated, therefore it can be the source that contaminates. When pollutants are adsorbed to particles, they can become suspended from the contaminated surfaces, in which case then the inhalation route becomes a significant pathway of exposure (Roberts and Ott, 2007). Oral exposure may also occur

in situations whereby, for example, small particles with contaminants adsorbed to their surfaces are ingested. A contaminant could also be ingested if it comes into contact with contaminated food, hands and other objects (WHO, 2000).

Earlier assessments of exposure in the workplace tended to focus primarily the inhalation route of exposure targeting mainly manufacturing processes known traditionally for producing large quantities of airborne dusts such as cotton mills, mines, smelters to name a few. These processes also produced many other vapours and gases that lacked proper control and were often reported to damage workers respiratory system besides absorption through the lungs into the blood hence inducing other target organ toxicity. Lately, emphasis on occupational exposure has tended to focus more on the potential for chemicals to pass through the unbroken skin (dermal route) and this therefore stems the need for greater understanding and control of dermal exposure (Omrane *et al*., 2018; Romero-Zarazua *et al*., 2015).

Normally, exposures to particular contaminants were understood to mean external exposure. This therefore means that exposures are more to do with the amount of the substance ingested, amount inhaled which is represented by the airborne concentration of the substance in the breathing zone of a worker and/or the amount that comes into contact with the skin. It had nothing to do with the concentrations within the body, normally ascertained by the amount of the substance that is absorbed by the digestive system (ingestion route), respiratory system (inhalation route) or entering the body through the skin (dermal route) (ECHA, 2010).

2.4.1 Inadvertent occupational ingestion of lead and arsenic

Gorman *et al.*, (2017) has defined inadvertent ingestion as ingestion that arises from contact between the mouth or the perioral region (the area surrounding the mouth) and contaminated hands or objects, which results in ingestion of which the individual may be oblivious. Within occupational hygiene, in most occupational scenarios, the ingestion pathway has been least prioritized. This has been due to the perception that this route accounts the least amount to the aggregate exposure levels (Christopher, 2008). A study in the United Kingdom, however, estimated that up to 4,500,000 workers could have had some regular none-trivial intake of hazardous substances by inadvertent ingestion route of exposure (Cherrie *et al.*, 2006).

Further, according to Cherrie *et al*., (2006), the importance of the ingestion route of exposure has tended to be considered insignificant. This could be attributed to;

1. The commonly accepted belief that ingestion of chemicals hence can be avoided since it results from only acts of gross negligence or intentional means

2. The recognition that many materials they have low bioavailability hence are maybe poorly absorbed from digestive system thus are unlikely to produce toxic effects especially when swallowed in very small quantities

3. The presumption that the amount of material taken into the workers body by ingestion route may be small when compared with the other routes such as inhalation and dermal contact.

In addressing occupational hygiene, all of the above three assumptions maybe applied. An obvious example is when workers involved in handling hazardous chemicals may inadvertently ingest harmful substances without the intention to inflict self-harm.

Heavy metals have been highly linked with the oral route of exposure hence receiving some considerable attention in occupational settings. Besides, the ingestion route has been well studied leading to availability of well defined exposure assessment methodologies and therefore its toxic effects are more understood. The removal of lead paint in old buildings for instance has the potential to cause some ingestion exposure via food contamination or hand to mouth contact (Abdul-Wahab, 2012).

In cases of inadvertent ingestion of hazardous chemicals, the processes leading to this exposure must involve transferring the substance from the medium into *peri-oral* area or directly to the mouth. This proposition can only be realistic if the contaminant or the mixture containing it must be a relatively solid, liquid or non-volatile. This will ensure the availability of the contaminant during the transfer processes, which must of essence include dermal contact of the area around the mouth with the contaminated objects or hands (the *peri-oral* area) or contact of the contaminated objects or hands into the mouth. This then must be followed by migration of the contaminant into the mouth. More often than not, cases of splashing onto the face or into the mouth may also relevant mechanisms; however they're of less significance (Christopher, 2008)

There exist significant unexplained variations in the mouthing habits between individuals. This has been attributed to human behaviour and personality traits which are also pertinent in determining who could be at more risk from inadvertent ingestion

exposure route depending on the frequency, duration of contacts as well as the type of contacts. When considering personal traits, some subjects for instance, were found to be more likely to often touch their faces while others were more likely to bite their finger nails than others. The frequency of these cases were also found to significantly vary depending on individual response to some situations (Nwudu *et al*., 2018). For instance, in some people the frequency of touching their faces significantly increases when they are more nervous or anxious. Further, some people tend to greatly exhibit repetitive personal habits which include biting of nails and sucking of fingers that increase the significance of inadvertent ingestion exposure.

Eating in the workplace has also been reported to exacerbate the transfer of contaminants by hand-to-mouth contact. This has been well exemplified in a past comparative study between Chinese and Malay workers in a lead battery production plant by Chia *et al.*, (1991). In the study, the Malay workers were found to have increased lead levels in blood. Unlike the Chinese who normally feed with chopsticks, the Malay's culturally tend to eat food using their hands; hence the elevated lead could be attributed to this habit. Hwang and Chen, (2000) also reported increased urinary arsenic levels which was mostly attributed to ingestion exposure from contaminants on the hands during maintenance in a semiconductor manufacturing plant.

The ingestion of dust was found to be the major pathway for As in a risk assessment study done in Pakistan by Subhani *et al*., (2015). This was followed by dermal and inhalation dermal contact. Arsenic was subsequently reported to accumulate in human biological tissues such as nails and hair. The study further reported that total As in soil

dust was two to three times less than indoor dust and arsenic bio-accessibility ranged from 13.8% to 20.2% in soil dust while that of indoor dust ranged from 75.4% to 83.2%.

In another study of Pb refinery workers in Japan, lead levels blood highly correlated with lead in fingernails and also in facial wipes $r = 0.59$ and $r = 0.73$, respectively. The study therefore exemplified that the works elevation of blood lead levels could be attributed to lead contaminated face and fingers through ingestion. The study by Hwang and Chen (2,000) also reported a high correlation between the mass of lead detected on the lips of workers and workers blood lead levels.

2.4.2 Inadvertent dermal exposure of lead and arsenic

In the past lead and other toxic metals dermal contamination by dusts attracted insignificant attention. This was generally from the assumption by many that this exposure pathway posed insignificant risk from percutaneous penetration and any toxic effect and therefore was due to systemic uptake. However, a past study by Stauber *et al* (1994) suggested that lead applied to the skin as lead acetate or lead nitrate, was rapidly absorbed through the skin and was detected in sweat, blood, and urine within 6 hours of application. The study used radiolabeled Pb acetate or Pb nitrate on volunteer people for 16 days and exemplified that there was skin absorption of this form of soluble lead. There was also found to be increased lead in urine, blood and sweat.

A study by Sun *et al.*, (2002) also confirmed skin penetration of lead metal powder in exposed workers and in rats. They found that Pb concentration in the *stratum corneum* from the hands to be correlated to Pb content in workers' blood and in urine. In rats, lead

oxide metal powder (PbO) was applied to the skin under wrap for 12 days and both resulted in significant elevations of Pb in urine.

A later study by Filon *et al*., (2006) further reported that lead oxide can pass through intact human skin with a median amount at 24 hours of 2.9 ng/cm^2 (25–75th percentiles 0.35– 6.0) and removing Pb after 30 minutes did not cause a reduction of Pb penetration in 24 hours but only caused a reduction in skin Pb content suggesting that removing Pb powder after 30 minutes is not sufficient to reduce the apparently rapid initial absorption that can occur during the first few minutes. This penetration increased nine fold when used on an abraded skin protocol. According to this study, in 30 minutes, perhaps a sufficient amount of Pb has already passed into the *stratum corneum* and created a concentration gradient or the decontamination with the cleansers was not complete and allowed penetration to continue. This rapid Pb skin absorption was also in accordance with the study by Sun *et al*., (2002).

The study further concluded that the dermal contact with commonly used cleansers could significantly increase Pb skin penetration within a day. The study therefore recommended that even if quickly followed by washing, at all times it was necessary at all costs to prevent the occurrence of skin contamination because skin content and penetration could be increased by a short duration of contact. According to Gorman *et al.*, (2017), lead ingestion exposure due to transfer from the skin to the mouth through the hands can be increased by the dermal uptake of lead which should be of importance.

The California Department of Toxics Substances Control (DTSC) in 2004 developed a lead risk assessment spreadsheet (California Lead Spread Model) that California agencies

use to estimate blood lead concentrations that might result from exposure to lead through soil and dust ingestion, dietary intake, inhalation and dermal contact. The DSTC default value is also consistent with information on dermal absorption fraction and the K_p values for lead (10^{-4}) in soil that is included in U.S. EPA guidance materials. Specifically, a dermal absorption fraction for lead (0.1) can be extrapolated from the cadmium values by assuming that relative absorption rates for the two substances in water are also applicable (on a relative basis) to contaminated soils.

In a past study by Wester *et al*., (1993), arsenic was found to be absorbed across the skin from soil. Gorman *et al*., (2017 in a recent study asserts that arsenic risk assessments have greatly underestimated the dermal uptake route. Their study was in support of a positive correlation between exposures on the hands and exposure on the perioral area as found by Christopher (2008). Regulatory agencies have, however, relied on soluble forms of arsenic dermal absorption data as the technical basis for specific absorption values. These values have been in the past used to calculate arsenic exposure in weathered soil (Lowney *et al*., 2010).

In addition to direct exposure through dermal contact followed by inadvertent hand-to-mouth ingestion, indirect arsenic exposure via dermal, ingestion exposure through contaminated soils and dust from CCA-treated wood has also been reported by the California Department of Public Health (CDPH, 2001).

2.5 Health impacts of heavy metals

2.5.1 Health impacts of arsenic exposure

Arsenic is ranked number one on the ATSDR's "Top 20 List." It has further been reported as the most common cause of acute heavy metal poisoning in both adults and children. Inorganic arsenic is classified as a known human carcinogen by the U.S. EPA (ATSDR, 2015). Inorganic As poses health risks even in low-dose chronic exposure. Arsenic contamination through diet, drinking water and by inhalation of polluted dust and air affects all populations including children and adults, pregnant women and the unborn babies (Ferguson *et al.,* 2018; Yunus *et al*., 2016).

Depending on the route of exposure, As will primarily target various organs in the human body. Adverse effects of the ingestion as well as the dermal routes of exposure for instance will most often manifest in skin causing lesions and discoloration, while diarrh0ea, nausea, and abdominal pains are manifestations of the gastrointestinal tract. Cancers of the bladder, skin, lung and liver have also been linked to ingestion exposure (Hong *et al*., 2014a). On the other hand, a rise in incidences of lung cancer and mucous membranes irritation have been linked to and the inhalation route of exposure. As listed by the World Health Organization (WHO, 2012), abdominal pain, vomiting, diarrhea with subsequent by numbness and muscle cramp are some of the immediate symptoms of acute As poisoning. In severe cases, mortality may occur.

The ATSDR (2015) arsenic toxicology profile characterized symptoms of chronic oral exposure. Primary symptoms included appearance of small warts or corns on torso, soles and palms, and also skin darkening. These symptoms were the result of changes in blood

vessels near the skin induced by As. Cancers of the skin, liver, bladder and lungs are also commonly attributed to prolonged As exposure (Durand *et al*., 2015; WHO, 2012). An association between cardiovascular diseases and high As exposure has also been reported (Chen *et al*., 2011; Tsuji *et al*., 2014), which may have been as a result of endothelial cells of the vasculature caused by As. Singh *et al*., (2011) also noted that inorganic As has a long standing association with cancers of internal organs, particularly the prostate.

In summary, parasthesia, cognitive deficits, muscular weakness, peripheral neuropathies, ataxia and fatigue have all been associated with chronic arsenic toxicity (NORD, 2015). Gastrointestinal effects include jaundice, hepatomegaly, nausea, anorexia, and vomiting. Further, As also affects the skin causing eczema, erythema, diffuse alopecia, pigmentation or arsenic melanosis, keratosis (especially of soles and palms), brittle nails, bands or white lines in the nails (Mee's lines), scaling and desquamation as well as localized subcutaneous edema. Even when arsenic concentrations in hair and urine may be within normal limits, arsenical polyneuritis may be diagnosed by the presence of white striae in the fingernails (WHO, 2018).

2.5.2 Health impacts of lead exposure

Lead is the first element that was characterized by its kind of toxicity since it affects many cellular processes and enzymes hence affecting all organs of the body to varying degrees depending on the duration and level of Pb exposure. The ATSDR (2019) has classified Pb as a group 2A carcinogen indicating it is probably carcinogenic to humans.

Key lead-induced health effects arising from chronic lead exposure include neurological effects (peripheral neuropathy, encephalopathy and fatigue); gastrointestinal effects

(dyspepsia, nausea, lead red line on gingival tissue); reproductive effects (reduced sperm count and motility, abnormal sperm, miscarriages/still births); heme synthesis (anemia), renal effects (hypertension, chronic nephropathy with proximal tubular damage). Other effects include arthralgia and myalgia (Zheutlin *et* al., 2018; Gildow, 2015; Ji *et al*., 2015).

Also according to Assi *et al*., (2016) and Emese (2008), mental retardation, birth defects, allergies, colic, autism, lack of concentration, dyslexia, psychosis, weight loss, arthritis, hyperactivity, mood swings, seizures, numbness paralysis (beginning in the forearms), shaky hands and muscular weakness have all been attributed to chronic exposure to lead.

Once in the body lead is finally majorly stored in the bones accounting for 80 – 95% of total body amounts where it can be released later to other body systems. Besides, lead also crosses the placental barrier affecting the foetus and children. Comparatively, lead exposure is more detrimental to the health of children than adults. Various studies have reported evidence that Pb exposure leads to various health effects in children including behavioural and developmental problems hence pitting children as the most vulnerable population (Tan *et al.*, 2018; Moya and Phillips, 2014).

2.6 Health risk assessment

The liquids people drink, the air they breathe, the surfaces they touch, the food they eat and the products they use in their day to day activities may be contaminated hence exposing them to a variety of potentially harmful agents. The reduction or prevention of human exposure to harmful environmental agents that account for indirect or direct

increase in the rates of disease, disability or discomfort and premature death occurrences, is an important aspect of public health protection (WHO, 2000).

In order to sustain development, increase agricultural productivity, control and prevent many diseases and other numerous human activities, harmful chemicals have become part and parcel of human life. When not used properly, despite their many benefits, chemicals have the potential to cause adverse effects on environmental quality hence human health. With increase in human development both in developed as well as in developing countries, which have been predicted to continue increasing growth of chemical industries, stems the need for chemicals widespread, worldwide application thus increasing the potential of adverse environmental effects. In this context, in order to pursue and attain the principles of sustainable development, of priority is the recognition and the assessment and management of human risks from exposure to harmful chemicals (WHO, 1999).

Dourson *et al*., (2013), noted that field of human risk assessment has continued to receive widespread recognition within both the scientific and regulatory contexts since about 1970's. Risk assessment is therefore a conceptual framework with an aim to provide the mechanisms for a structured review of information with relevance to estimating environmental or human health outcomes. As early as in the 1980's, the risk assessment paradigm as advanced by the National Academy of Sciences has proven to be a useful tool in conducting risk assessments (National Research Council, 1983). According to this paradigm, the risk assessment process is divided into four distinct steps: hazard identification, the dose-response assessment, the exposure assessment and finally the risk

characterization. This is the paradigm that has continued to be in use to date (Berasaluce *et al*., 2019; Omrane *et al*., 2018; Yu-Mei *et al*., 2018).

i) Hazard identification

Hazard identification is the first step in risk assessment which based on assessment of all available data on mode of action and toxicity, aims to evaluate the weight of evidence for adverse human effects. This step is meant to address two primary issues: (1) whether an agent may adversely affect human beings, and (2) the scenario under which the identified hazard occurs. The assessment of available data maybe evaluated based on analysis of structure-activity relationships as well as observations in humans. This step therefore requires scientific judgment as to whether under given exposure conditions; the chemical evaluated can pose harmful health effect in humans. In general terms, toxicity of the harmful agent is normally observed in either one organ or in some cases many target organs in the body. In most cases, following exposure to a known chemical, multiple end-points are observed, with the first significant adverse effect to occur with increase in dose, also referred to as the critical effect being determined (WHO, 2010a).

ii) Dose-response assessment

This step involves characterization of the relationship between the incidence of an adverse health effect as determined from the dose of an agent received or administered. For most cases of adverse effects (i.e. neurological/behavioural, organ-specific, immunological, reproductive, developmental or non-genotoxic carcinogenesis), there is generally a threshold which is considered as the dose or concentration below which adverse effects are not anticipated to occur. However, for other types of toxic effects, a

no threshold exists which is the assumption that at any level of exposure, there is a probability some harm will occur. For studies on mutagenesis and genotoxic carcinogenesis, this latter assumption is generally applied.

Based on a NOAEL/LOAEL (no/lowest-observed adverse effect level) and uncertainty factors in cases where a threshold has been assumed, a level of exposure is traditionally assumed below which it is believed that there are no adverse effects. On the other hand, the magnitude by which the estimated exposure (for instance the margin of safety) is exceeded is considered alongside various sources of uncertainty. This approach has in the past often been described as a "safety evaluation". This dose has therefore been considered critical since it is first approximation of the threshold dose. However, as opposed to the NOAEL/LOAEL, a model-derived estimate (or its lower confidence limit) also referred to as the "benchmark dose" of a particular incidence level for the critical effect, has increasingly been proposed for use in quantitative assessment of the dose-response effects (U.S. EPA, 2011).

It should, however, be noted that, for the risk assessment of chemicals for which the critical effect may not have a threshold (genotoxic carcinogens, germ cell mutagens) a clear consensus is lacking on the appropriate methodology. A number of approaches have therefore been adopted for risk assessment based largely on characterization of dose-response for such cases. Therefore, the NOAEL, which is regarded as the first approximation of a threshold have largely been replaced by critical data points which define the slope of the dose-response relationship (WHO, 2010a).

iii) Exposure assessment

The exposure assessment step aims at: (1) identifying potentially exposed populations; (2) identifying the potential exposure conditions and exposure pathways; (3) quantifying the potential doses/chemical intakes. The routes of exposure are inhalation, ingestion or the dermal absorption routes. The U.S. EPA, (2011) describes exposure as the contact of visible external physical boundaries (for example mouth, nostrils, skin) with a chemical agent. Exposure therefore is directly dependent on the frequency, intensity and contact duration. The intensity of contact is typically expressed in terms of the concentration of contaminant per volume or unit mass (mg/m^3, μg/g, ppm μg/L, among the many) in the media of human exposure.

Exposure assessments are of essence used to determine whether populations of interest may be potentially exposed to hazardous chemicals in the environment. If they are found to be exposed, a further determination on the quantity, the route of exposure, type of media as well as duration of exposure. The exposure point concentration (EPC) is normally the quantified level of the chemical of concern in a medium with which the person may be in contact. The environmental media of exposure normally include water, air, outdoor and indoor soil and dust in locations frequented by a population. Consumer products or food with which people come in contact also act as other media of exposure. The exposure point concentrations that are as representative as possible of the potential human contact with a chemical of concern are evaluated for media, durations and their physical locations. This exposure assessment step therefore requires information on: the environmental media that is anticipated to contain the potential chemical of concern, the

relevant exposure route pathways and the appropriate exposure duration (IPCS, 2009; WHO, 2010a).

The media of exposure must be found to contain the chemical of interest with the potential to contain cause adverse human effects. These exposure scenarios may occur as non-occupational (or community exposures) occupational settings or while using products. Generally, the exposure assessment step therefore while considering phase associations and chemical forms, the exposure route pathways, and subsequently expressing the exposure point concentration in a way to best reflect the bioavailable amount of metal (U.S. EPA, 2011).

An exposure pathway may either be direct or indirect, whichever the case the variables involved such as frequency, intensity and contact duration of the contact with the contaminated media are necessary. Indirect exposures which in most cases are inadvertent for instance could be via by soil/dust pollution and dermal contact with the soil and its subsequent ingestion. When necessary, they use a bioavailability factor for the intake route in determining this indirect exposure entails assessment of concentrations in intake media for example in dust (Muralikrishna and Manickam, 2017).

Whereas exposure routes entail ingestion, ingestion and inhalation, pathways on other hand describe clearly the specifics of any exposure, for instance, inhalation of Pb in dust by children during remediation of a nearby lead-smelter Superfund site (Khoury and Diamond, 2003). When air serves as the primary medium of contact, then the direct major exposure pathways for human intakes of metals in are mostly found to be inhalation and dermal.

However, there exists an indirect pathway in which air may also serve as an antecedent medium. These include: the deposition of metals suspended in air to sediment and surface water and subsequent intake via dermal contact and ingestion; the deposition of suspended metals onto indoor and outdoor surface dusts and subsequent intake via ingestion, dermal contact or inhalation and finally the uptake of deposited metals into the terrestrial and/or aquatic biota that enters into the human food chain and subsequent intake via ingestion. Generally, the ingestion exposure pathway normally associated with contaminants in the environmental media such as in water, food and in both indoor and outdoor soil (Yu-Mei *et al*., 2018).

Chemicals must be present in the air for inhalation to occur. Further, depending on their solubility and vapour pressures, chemicals with low solubility and with moderate to high vapour pressures can be inhaled on volatilizing from water or soil. The chemical trichloroethene which is an organic solvent for instance can readily volatilize from potable water and be inhaled once available in the air. Besides, the inhalation route can also pay an important role in the exposure of less volatile chemicals. The PCB's (polychlorinated biphenyls) which are less volatile for instance can be inhaled when it occurs at elevated levels in soil or in other solid substrates (WHO, 2010a).

Finally, the dermal absorption route of exposure requires that there's contact between a harmful chemical and the skin. This for instance can occur in water during bathing or swimming. The earlier assumption that upon exposure to airborne inorganic forms of metals, the dermal pathway plays an insignificant role in contributing to internal doses has been refuted since it bore no empirical basis especially for certain metals. Past studies

have in the past made efforts to quantify the extent of or the kinetics involving how metals deposited on the skin penetrate (Stauber *et al*., 1994; Hostynek *et al*., 1998); in order to quantify the dermal contribution when estimating risks. Yu-Mei *et al*., (2018) in a recent study noted that all chemical contaminants and their exposure pathways should be considered in order to come up with an aggregate assessment.

Exposure assessment requires the determination of doses which refers to the amount of chemical to which individuals are exposed that crosses the external boundary. Dose is therefore directly dependent upon the contaminant levels in the environmental media and the intake rate (inhalation, ingestion or dermal absorption). The potential dose is the amount of contaminant which could be taken into the human body through ingestion, inhalation or deposition on the skin. Further, the absorbed dose is the amount of contaminant absorbed upon intake into the human body via the lungs, the gastrointestinal tract (GIT) or the skin. Findings from pharmacokinetic studies followed by intra-peritoneal or other injected delivery into the test animal on either the potential dose from the absorbed dose or animal feeding studies typically form the basis for toxicological risk assessments. According to WHO (2000), potential dose (PD) may be calculated as follows:

$$\mathbf{PD = C \times IR} \qquad \mathbf{Eqn\ 2.1)}$$

Where:

PD = potential dose (mg/day);

C = contaminant concentration in the environmental media of interest (mg/L, mg/m^3, mg/cm^2, mg/g,); and

IR = intake or contact rate with the environmental media (cm^2/day, m^3/day, g/day, L/day).

The exposure concentration term is usually based exclusively on chemical and site specific data which is relevant to the population and/or site of interest. The exposure concentration may also be based on measured concentration in the media (for example, water, soil, air) of study interest or on a chemical - and site - specific modeled concentration. The contact rate which in practice is the rate of inhalation, ingestion or dermal contact may at times be expressed as the aggregate of more than one term (for instance., the dermal contact rate for dust/soil may be expressed as the surface area in cm^2/day multiplied by the soil adherence factor in mg/cm^2) as:

$$\mathbf{ADD_{POT} = (PD \times ED \times EF)/(BW \times AT)} \qquad \textbf{(Eqn.2.2)}$$

Where:

ADD_{POT} = potential average daily dose (mg/kg/day);

ED = exposure duration (days/year);

EF = exposure frequency (years);

BW = body weight (kg); and

AT = Averaging time (days).

The aim of exposure assessment is to determine the extent and nature of contact with chemical contaminants anticipated or experienced under varying conditions. Multiple approaches have been used in the past to conduct exposure assessments. In general terms, the approaches include direct and indirect techniques, which involve the measuring of

environmental chemical levels in the media and also personal exposures, in addition to biomarkers. Models and questionnaires have also been used often (U.S EPA, 2008).

According to (WHO, 2010a), in order to estimate the concentrations to which environmental life support systems (soil, water and air) or human populations are exposed, exposure assessment studies also require that emissions, their pathways and rates of movement of substances and their degradation or transformation be determined. The expected numerical output may be an estimation of the rate, intensity, duration or frequency of contact exposure or even the dose (resultant amount that will actually cross the barrier) which is dependent on the purpose of the exposure assessment.

In exposure assessment studies, the term "worst case exposure" has been used to historically mean the maximum possible exposure, or when everything happens that can plausibly happen to maximize exposure. However, in actuality, this “worst case exposure” at times falls on the uppermost point of the population distribution, in most of the cases, it is usually somewhat higher than the individual in a population that has the highest exposure. The worst case exposure is a hypothetical individual representation and therefore an extreme set of environmental conditions which usually is not observed in actual populations (U.S. EPA, 2011).

Bridges (2003) asserts that in most risk assessment studies, exposure assessment is therefore the "weakest link". The calculation of chemical residues in water, soil or air leads into uncertainty in human exposure assessments since often it is not a good indicator in terms of bioavailability. According to Arain and Neitzel (2019), though the use of biomarkers may at times provide information about the environmental pollution, it

should, however, be noted that as a result of point sources, pollution may often occur in hotspots. It is therefore inadequate for risk assessments to assume that an entire population is exposed to the same or maybe an average, chemical concentration. The source-pathway-receptor paradigm advances that the spatial distribution of soil contaminant levels and receptors, if possible be maintained in order to calculate a more realistic contaminant intake and subsequent risks (Gay and Korre, 2006). Based on physical location specific exposure land-uses and pathways, site-specific risk assessments though often limited to high-profile studies and large projects enables to get more realistic results (Lester *et al*., 2007).

iv) Risk characterization

Risk characterization usually the final step in risk assessment has often been described to act as the bridge that links risk assessment to risk management. This is because it provides the basis that enables all the calculations, that uncertainties inherent in the evaluation are understood and that the results of the risk assessment are also well understood and interpreted. This step therefore serves to integrate information from the exposure assessment and toxicity assessment besides synthesizing an overall conclusion that is expected to be informative, complete and useful for decision making (U.S. EPA, 2012).

In order to evaluate the human health risk the HQ (hazard quotient), HI (hazards index), and CRA (carcinogenic risk assessment) are normally applied. The excess lifetime cancer risk (ELCR) is used to calculate the carcinogenic risk in carcinogenic risk assessment (CRA) studies. An ELCR higher than the acceptable range of one individual in one

million to one individual in ten thousand persons (1×10^{-6} - 1×10^{-4}) implies increased probability of an individual developing cancer over their lifetime hence indicates potential carcinogenic risk. Thus, U.S. EPA (2012) recommends that if the CRA value is less than one individual in one million ($CRA < 1\times10^{-6}$), can be regarded as negligible, whereas if the CRA value is greater than one in ten thousand ($CRA > 1\times10^{-4}$) the risk is regarded as likely to be harmful to human beings.

For non-carcinogenic risks, a HI refers to total risk value for a single metal and indicates the aggregate value of the HQ for a substance through different pathways. If the value of HI is less than one ($HI < 1$) then it indicates that no significant risk of non-carcinogenic effects is anticipated to occur. However, if the HI is more than one ($HI >1$), there is an indication that non-carcinogenic effects may occur, the probability therefore increases directly with increasing HI value (U.S. EPA, 2011).

Risk characterization therefore provides the essential rationale and scientific evidence about risk that is designed to support risk managers in decision-making. The estimates of the risk to human health under relevant exposure scenarios are provided. Thus, while including attendant uncertainty, risk characterization evaluates and integrates the available scientific evidence for use in estimating the importance, nature and more often the magnitude of environmental and/or human risk so as to reasonably estimate the result from an exposure under specific circumstances to a particular environmental agent (WHO, 2000).

v) Risk management

The term risk management attempts to encompass all the activities that are required to reach decisions regarding the action to be taken on an associated risk such as necessary reduction or elimination. The risk management options/strategies can therefore be broadly classified as non-regulatory/regulatory, advisory or technological, economic, all these regarded as not mutually exclusive. The population size, the scientific quality of the risk assessment, costs of meeting the targets, the resources, as well as the arising managerial decisions are key decision factors that vary enormously depending on one decision context to another (WHO, 2000).

It has also been recognized that the risk management phase is most often unstructured but seldom codified or uniform hence is viewed as a complex multidisciplinary procedure which should be expected to respond to evolving inputs from numerous sources. Both risk perception and risk communication have increasingly received recognition as pertinent elements, which if risk management decisions are expected to broadly receive public acceptance must also be considered (Aven, 2016; WHO, 2000).

2.6.1 Central tendency and reasonable maximum exposure risks

For any specific agent or site, different individuals within a population often exhibit a wide variability in the amount of chemical contact. This implies there is a range in the level of exposures that is actually experienced by individuals in a population. While some individuals may be exposed to a lower degree of chemical contact but for a shorter duration (for instance, individuals using a recreational facility not only sited near but also downwind of the factory), others may have a high degree of contact for an extended

duration (for example, occupational exposure to an agent). Thus, human contact with contaminated environmental media is not regarded as a specific value but is rather best viewed as distributions of all possible values (UNDP, 2000).

The U.S. EPA (2006) policy on exposure assessment therefore requires that a range of possible exposure scenarios be considered as opposed to a specific value. Both reasonable maximum exposure (RME) estimates or high end and central tendency exposure (CTE) estimates risk assessments should thus be included. The CTE refers to an estimate of the average contamination level experienced by the affected population, based on the amount of agent present in the environment and the frequency and duration of exposure while RME is the highest dose estimated to be experienced by some individuals, commonly stated as approximately equal to the 90^{th} or 95^{th} percentile exposure category for individuals. Since CTE and RME risks are calculated using single or discrete numbers (also known as point estimates) for each input value, this approach is also commonly referred to as the "point estimate method" (Boffetta *et al*., 2011).

The Oregon State for instance requires that both CTE and RME be considered in the risk assessments. For the calculation the CTE, the arithmetic mean is usually used to represent environmental contamination levels and mean estimates of all exposure factors are also used (Oregon Department of Environmental Quality, 2010).

The use of both CTE and RME calculations provides a platform for the semi-quantitative measure of the range of anticipated risks that may occur under particular specified exposure scenarios. The RME and CTE are regarded as simple probabilistic forms of risk assessment that can provide risk managers with an estimate of the upper percentile and

mean estimates of exposure, respectively. At all times, to characterize both the CTE and RME scenarios, the concentrations are supposed to be as representative as possible. In most scenarios, when the number of samples is large enough, representative concentrations are determined using 95% upper confidence limits (UCLs) on the mean as RME estimates while means as used as CTE estimates (U.S. EPA, 2007).

On the other hand, when the number of detected values is small, the approach for calculating representative CTE and RME concentrations for detect samples sizes, *n*, from 1 to 4 is:

n = 1; there is no RME, the result is CTE;

n = 2; there's no RME, maximum detect is CTE,

n = 3 or 4; maximum detect is RME, the mean is CTE.

2.7 Risk estimation of lead and arsenic heavy metals

Risk estimation also known as or risk characterization is usually the final step in human risk assessment aimed at gathering, comparing, reviewing and organizing the output of an exposure and toxicity assessments exposure duration, frequency and magnitude, pathways and receptors and toxicity values. For heavy metals just like other chemicals, risk is calculated for each of the heavy metal by utilizing toxicity and exposure data. The calculated risk is thus expressed in numerical forms for both carcinogenic risks and non-carcinogenic hazards (WH0, 2010a).

The reference dose (RfD) also called the toxicity value for non-cancer effects from ingestion exposure is an estimate of the highest dose that can be taken in every day without causing adverse non-cancer health effects. The U.S. EPA (2006) for instance, has

determined that the reference dose (RfD) for inorganic arsenic as 0.0003 mg kg^{-1} day^{-1}. Non-carcinogenic effects are thus evaluated by comparing an exposure dose with the predetermined toxicity value (RfD) and expressed as a hazard quotient (HQ). The hazard quotient assumes that a level of exposure exists below which an adverse non-carcinogenic human health effect is unlikely to be experienced. The HQ is therefore the ratio of the exposure concentration in an environmental medium to the reference dose expressed as;

$$\mathbf{HQ} = \frac{\textbf{site exposure (for instance ADI)}}{\textbf{Reference dose}} \qquad \textbf{(Eqn. 2.3)}$$

Where: ADI is the average daily exposure

Hazard quotient values are normally variable, with values more than unit in general terms considered as indicative of an unacceptable hazard while values equal to or less than unit indicate acceptable levels.

The U.S. EPA (2011) has already developed toxicity values in their exposure handbooks that are used to estimate the risk of getting adverse health effects in addition to cancer as a result of exposure to lead and inorganic arsenic. For instance, toxicity values exist based on studies of workers involved in the application of arsenical pesticides, workers occupationally exposed to arsenics as well as populations exposed to arsenic via consumption of drinking water with high levels of arsenic. The estimation of the chance of a person developing cancer from ingesting 1 mg/kg-day for a lifetime (usually approximated as 70yrs) is referred to as the cancer slope factor (CSF). As with all other chemical contaminants, the potential for carcinogenic risk for heavy metals is expressed

by estimation of the probability of developing cancer by an individual in a population for a specific lifetime. Thus,

Cancer risk = exposure (ADI) x CSF (Eqn 2.4)

Results obtained from computations of risk are then used to evaluate the degree of risk in that values that can vary from extremely high increased risk (10^{-1}) to very low increased risk (10^{-6}) (Florida Department of Health, 2015). Cancer risk is thus expressed as a unitless probability of an individual developing cancer in a population over a lifetime, quantified and expressed as shown in Table 2.2.

Cancer risks can also and are usually presented as one significant digit in order to avoid confusion in information precision. An ELCR (excess lifetime cancer risk) from exposure an individual carcinogen of for instance 1.4×10^{-6} can be presented as 1×10^{-6}, and would be indicative of an acceptable risk. Further, an ELCR of 1.5×10^{-3} from an aggregate exposure to multiple carcinogens can be presented as 2×10^{-3}, and would be indicative an unacceptable risk. Just like with other chemicals, cumulative risk of exposure is determined by summing risks for each individual chemical in all the pathways and calculated risk values compared to applicable risk goals (U.S. EPA, 2011).

Table 2.2: Quantifying and Interpreting Carcinogenic Risk

Value	Expression	Quantity	Risk level
$1x10^{-1}$	1.0E-1	One in ten	"very high" increased risk
$1x10^{-2}$	1.0E-2	One in a hundred	"high" increased risk
$1x10^{-3}$	1.0E-3	One in a thousand	"moderate" increased risk
$1x10^{-4}$	1.0E-4	One in ten thousand	"low" increased risk
$1x10^{-5}$	1.0E-5	One in a hundred thousand	"very low" increased risk
$1x10^{-6}$	1.0E-6	One in a million	"extremely low" increased risk
$1x10^{-9}$	1.0E-9	One in a billion	No risk

(Source: WHO, 2011)

CHAPTER THREE

MATERIALS AND METHODS

3.1 Introduction

This chapter describes the location of the study area and highlights of the work units. The research design and sampling procedures are also described including detailed laboratory sample treatment and analysis for the studied heavy metals (Pb and As).

3.2 Study area

This study targeted instructional laboratories in the two public universities within Uasin Gishu County. The study area thus comprised of Moi University (MU) and University of Eldoret (UoE) located approximately 36 kilometers South East and 10 km to the North of Eldoret town, in Uasin Gishu County, respectively. The study area is bound by laasdtitudes 0º 30’ S and 0º 35’ N and longitudes 35º 30’ E and 35º 37’ W (Figure 3.1).

Moi University, with a total of fourteen (14) schools and University of Eldoret, with nine (9) schools have several well established instructional laboratories hosted by the schools offering science/technical degree programmes which were the target of this study. The instructional laboratories/workshops of Moi University’s Town campus located approximately 3 km to the South of Eldoret town within Eldoret Municipality which hosts the Rivatex East Africa facility and mostly used by the university’s engineering students for workshop attachment, was also included in this study.

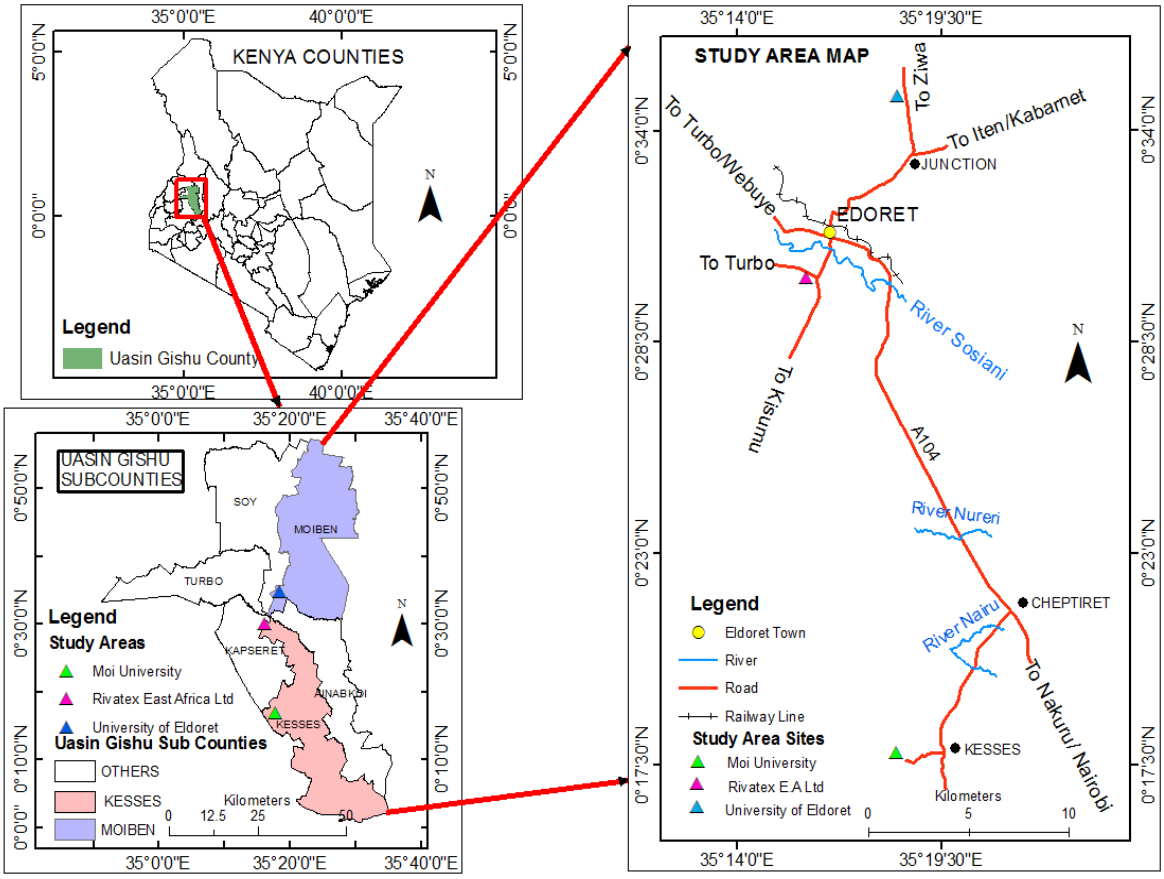

Figure 3.1: Location of study sites in Uasin Gishu County, Kenya

(Source: Author, 2019)

The student population was approximately 38,700 with Moi University having the highest population of approximately 24,500 (www.mu.ac.ke) and UoE handling about 14,200 students (www.uoeld.ac.ke). Of the student population, approximately 38% (14,600) undertake science related (laboratory based) courses therefore regularly using the laboratories.

Major activities in the selected institutions entailed teaching and learning through lectures and practical modes of content delivery. Practicals are carried out in laboratories and workshops in the case of engineering related courses. Practical activities that may expose

the students and staff working in these facilities may include but not limited to pure Pb and As handling and stock/spiking spillages, wood glue and lead adhesive tapes, sealants, wood stains, sanding, primers, paints, greasing, welding metal dust and fumes, fabrication and soldering. Others include handling textile dyes/pigments, wood preservation and treatment with chromated copper arsenate (CCA), analysis of pesticides, as well as electrotype metal cutting, and repair of electric motors, bearings and other semi-conductors and devices (Appendix III).

3.3 Research design

In total, 20 laboratories/workshops were identified which comprised of 7 at MU (Main Campus), 5 at MU (Rivatex East Africa) and 8 at UoE. The research employed a cross-sectional study design in two phases; the first phase was descriptive whereby the work-units were interviewed regarding their workplace hygiene and safety practices. The second phase was analytical phase involving sample collection from the environmental medium, sample preparation, laboratory procedures and subsequent quantification of Pb and As concentrations for risk assessment.

3.4 Sampling design

3.4.1 Sampling of laboratories

The campuses included in this study were chosen because they host basically all the technical and science oriented academic programmes offered at MU and UoE. A walk-through survey was done to identify potential health and safety hazards the study units as per the objectives of this study. The selected laboratories were open and accessible 8 hrs

a day five days a week. In total, 20 laboratories were identified which comprised of 7 at MU (Main Campus), 5 at MU (Rivatex East Africa) and 8 at UoE.

The identified instructional laboratories/workshops were further stratified based on the hosting universities resulting in 12 (60%) and 8 (40%) from MU and UoE, respectively. Proportionate stratified random sampling was then applied to select 10 (50%) laboratories for exposure and health risk assessment. These therefore comprised of 4 from UoE, 3 from MU Main campus and 3 from MU Rivatex East Africa facility. Table 3.1 shows the selected sampling stations.

Table 3.1: Sampling Stations

Facility name	Code	University	Host school	Location
Chemistry Lab 1	ECA	University of Eldoret	Science	Administration building
Wood science workshop	EWW	Ditto	Natural Resource Management	New site
Chem Lab 3	EC	Ditto	Science	Old site
Technology Education workshop	ETD	Ditto	Education	Administration building
Chem Lab	MC	Moi University	Biological and physical sciences	Mackay building
Welding shop	MMW	Ditto	Engineering	Engineering block
Sheet metal shop	MSM	Ditto	Ditto	ditto
Electronics workshop	REW	Ditto	Ditto	Rivatex East Africa
Motor rewinding shop	RMR	Ditto	Ditto	ditto
Mechanical shop	RMD	Ditto	Ditto	ditto

(Source: Author, 2019)

3.4.2 Sampling of work units

Based on the objectives of this study, the employees working in the 10 sampled facilities were purposively sampled. Work units from the employees were identified by getting the list of job title groups and interviewing supervisors at each laboratory regarding practical information about work practices and procedures on the various routine and non-routine tasks carried out by the various workers in normal operations. The main activities for each facility were outlined and where the tasks were similar for the identified staff, they were grouped together and considered as a single work unit.

Since the technical employees in each sampling station were found to be performing similar activities with negligible or lack thereof of other categories of staff, the possibility of similar exposure to Pb or As was inferred. Purposive sampling method was then applied to select the work-units and each sampling station therefore comprised a work unit implying 10 work units were studied as identified in Table 3.1.

3.5 Data collection

3.5.1 Interview schedule

For each of the 10 work units sampled, the supervisor was purposively sampled and an interview scheduled (Appendix IV). The interview schedule comprised of three parts; Section A collected data on general facility information such as job titles of the workers, level of training, nature of duties and general housekeeping of the facility. Section B aimed to collect data on laboratory/workshop hygiene and safety practices such as safe handling, storage and disposal of wastes from the facilities, knowledge of safety data sheets (SDS) formerly referred to as material safety data sheets (MSDS), health

surveillance and records on occupational injuries and illnesses, in order to collect data on work practices that may exacerbate exposure to contaminants.

Section C collected supplementary data on personal traits of the worker. In order to evaluate worker-individual traits that may affect inadvertent exposure some select personal dispositional traits which can be traced to the individual's personality and how it predisposes the individual to occupational exposure, individual traits were studied. These included occupational mobility indicative of the rate at which workers change from one area of occupation to another and occupational tenure or aggregate number of years a person has worked in his or her current occupation, irrespective of the interruptions in employment, the number of employers or time spent in other occupations. Further, the presence of hair on the head and/or face, smoking and hand washing habits were also evaluated.

3.5.2 Sampling of indoor settled dust

Composite settled dust samples were collected fortnightly for a period of 6 months using new pre-cleaned polyethylene brush and dustpan as recommended by U.S. EPA's (2008) guidance for the sampling and analysis of heavy metals in indoor residential dust and according to Ardashiri and Hashemi (2017). Sampling was done by gently sweeping the floors, corners and wiping of visible dust on equipment tops and raised areas such as windowsills and sash areas with dry ash less filter paper (Whatman No. 42).

Upon thoroughly mixing the settled dust, approximately 100 g of composite settled dust samples were then collected by quartering sampling method fortnightly for each laboratory/workshop in duplicate. In order to avoid sample contamination, pre-cleaned

disposable hand gloves were worn and a disposable plastic spoon was used to scoop each composite sample. A total of 222 composite sweep dust samples were thus collected for Pb analysis in this study.

For As analysis, simple random sampling was applied to select 5 composite sweep samples in duplicate from within the 222 samples for each of the 10 sampling stations. A total of 100 dust samples were thus collected for As analysis. All indoor dust samples were then transferred into pre-cleaned re-sealable plastic bags and further wrapped in clearly labeled plain brown paper bags awaiting laboratory analysis.

3.5.3 Laboratory sample preparation and analysis

a) Lead analysis

The 222 samples were oven dried in a drying furnace overnight at 70ºC and the composite samples passed through a 0.2 mm aperture sieve. For Pb analysis, 0.3 g of a well homogenized composite sweep sample was accurately weighed in duplicate into digestion (conical) flasks. An 8 ml of freshly prepared aqua regia (2 ml HNO_3 and 6 HCl that is, ratio of 1:3, both analytical grade) was then added and shaken for approximately 2 minutes. The conical flask was then covered and the contents heated for 2 hours on medium heat of a hot plate until all bubbling ceased. Nitric acid was added whenever necessary to avoid the samples running dry. The heating was continued until a pale brown colour resulted indicating digestion was complete.

The digests were then allowed to cool and filtered through a 0.45µm Whatman filter paper into pre-washed 50 ml standard volumetric flask. The residue was then washed three times with de-ionized water and the filtrate filled to the mark with de-ionized

distilled water. Further, 8 blank water samples were also analyzed. The digests were then transferred into correctly labeled acid pre-cleaned plastic bottles awaiting analysis using Flame atomic absorption spectrophotometer (F-AAS, Model Spectra AA/20).

Equipment calibration standard solutions were prepared according to equipment specification by dissolving 1 g of pure Pb metal (99.9 % Pb) in a volume of 1:1 nitric acid (HNO_3): water, and diluting the solution to 1000 ml (cm^3) in a one litre volumetric flask to give a concentration of 1000 mg/L of the metal. This was used as stock solution to prepare 1, 5, 10, 15 and 20 ppm's of Pb metal for instrument calibration.

Blank water samples were prepared by treating deionized/distilled water in the same way as the samples. The concentrations of Pb in the digests were then measured by the duly calibrated Flame atomic absorption spectrometer (F-AAS).

Data for Pb metal concentration in composite dust samples was attained by extrapolation from the standard curve and conversion from the mg/L results to mg/kg calculated according as;

Metal concentration (mg/kg) was calculated as:

$$\text{Metal concentration (mg/kg)} = \frac{\text{A} \times \text{B}}{\text{g (sample weight)}} \qquad \textbf{(Eqn 3.1)}$$

Where: A is concentration of metal in digested solution (mg/l) and

B is final volume of digested solution (mls)

b) Arsenic analysis

Arsenic analysis was carried out using the portable S1 Titan XRF (X-ray fluorescence) Spectrometer (Bruker Model) at the Mines and Geological laboratory as shown in Appendix I (a-e). Completely dry dust samples were sieved to < 250 μm using standard testing sieve. Dust samples were then screened using a XRF analyzer.

The XRF was calibrated using standard procedures as per the user manual prior to use. Approximately 10 g from each of the 50 previously dried and sieved dust duplicate samples was scooped into the sample cup up to ¾ full and placed in the XRF directly to the detector. The S1 Titan XRF was then mounted on a stand and interfaced with a computer and once the detection trigger was placed, the detected As levels were read directly from the interfaced computer in parts per million (ppm).

3.5.4 Quality control and quality assurance

In order to assess the accuracy of the data that were obtained by the methods used in this study, a blank solution using distilled water was prepared and underwent the same processes as the field samples and analyzed together with the samples in the F-AAS. The blank water samples analyzed had undetectable Pb concentrations implying it's unlikely that there was contamination in the samples laboratory preparation and analysis procedures. Instrument calibration and recalibration was done before analysis and after every 10 samples.

All the glassware that were used in the digestion as well as in the filtration procedures were initially rinsed with tap water, cleaned with soap, then washed thoroughly with tap water, rinsed again with distilled water and then soaked in 1% HNO_3 overnight to remove

any anticipated contamination by heavy metals and finally was rinsed thoroughly with deionized and distilled water.

At each sampling station, the dust samples were collected using pre-cleaned brush and dust pan and samples placed into new sealed and well-labeled ziploc bags wrapped in clearly labeled brown paper bags to prevent contamination and to assure sample quality. In order to remove any moisture the dust samples were left to dry in a desiccator. Cross contamination of samples during XRF analysis was minimized by pre-cleaning the scooper and sampling cup before analyzing sample.

3.5.5 Occupational exposure assessment

Estimation of exposure rate was indirectly calculated as the exposure point concentration (EPC) which describes the concentration of a chemical in an exposure medium (settled indoor dust in this case) multiplied by human intake factors (HIF) or the average amount of an environmental medium contacted by the exposed person each day per body weight according to U.S. EPA exposure factors handbooks (U.S. EPA, 1997; 2011). For the purpose of this study, the resultant data typically referred to as chronic average daily intake (ADI), was used to explore two routes of exposure that is dermal and ingestion pathways for both As and Pb.

a) Dermal exposure

For dermal exposure, data for two exposure paths were evaluated;

1) Dermal contact with a contaminated surface and subsequent transdermal uptake

2) Dermal contact with a contaminated surface followed by inadvertent ingestion of contaminants transferred to skin

For dermal contact with chemicals in dust/soil, dermally absorbed average daily dose was estimated using U.S. EPA (2011) models: Average daily intake (ADI) for non-carcinogenic effect via dermal contact was determined by (ADIder) in mg/kg/bw as:

$$\textbf{ADI}\text{der} = \frac{\boldsymbol{EPC_s \times SAd \times CF \times TE \times EF \times ED \times ABSder}}{\boldsymbol{BW \times AT \times UCF}} \qquad \textbf{(Eqn. 3.2)}$$

Where EPC_s - mean heavy metal (Pb, As) concentration in settled indoor dust in mg/kg.

Average daily intake (ADI) for non-carcinogenic effect via dermal contact and subsequent incidental ingestion (ADIing):

$$\textbf{ADI}\text{der/ing} = \frac{\boldsymbol{EPC_s \times SAi \times CF \times TE \times fdo \times fgi \times EF \times ED}}{\boldsymbol{BW \times AT \times UCF}} \qquad \textbf{(Eqn. 3.3)}$$

Lifetime Average Daily Intake (LADI) for a carcinogen via dermal contact (LADIder):

$$\textbf{LADI}\text{der} = \frac{\boldsymbol{EPC_S \times SAd \times CF \times AF \times EF \times ED \times ABSder}}{\boldsymbol{BW \times AT_C \times UCF}} \qquad \textbf{(Eqn. 3.4)}$$

Lifetime Average Daily Intake for carcinogenic effect via dermal contact and subsequent incidental ingestion (LADIing):

$$\textbf{LADI}\text{der/ing} = \frac{\boldsymbol{EPC_S \times SAi \times CF \times ABSder \times fdo \times fgi \times EF \times ED}}{\boldsymbol{BW \times AT_C \times UCF}} \qquad \textbf{(Eqn. 3.5)}$$

b) Exposure by ingestion of dust

Non-carcinogenic average daily intake (CDI) of dust by oral ingestion for occupational exposure was determined using the equation (U.S. EPA, 2011);

$$\textbf{ADI}\text{ing} = \frac{\boldsymbol{EPC_S \times CR \times EF \times ED}}{\boldsymbol{BW \times AT \times UCF}} \qquad \textbf{(Eqn. 3.6)}$$

Whereas carcinogenic lifetime ADI was determined using equation;

$$\textbf{LADI}\text{ing} = \frac{\boldsymbol{EPC_S \times CR \times EF \times ED}}{\boldsymbol{BW \times AT_C \times UCF}} \qquad \textbf{(Eqn. 3.7)}$$

The exposure parameters used for different exposure pathways especially by U.S EPA (2011) in health risk assessment for standard indoor exposure scenario are as shown in Appendix II.

3.5.6 Health risk characterization

i) Non-Carcinogenic risk characterization

The non-carcinogenic risk was characterized using a hazard quotient (HQ), which is the ratio of the average daily intake (ADI) to the reference dose (RfD) (U.S. EPA, 2011; IRIS, 2000).

Non-carcinogenic risk was therefore determined as the Hazard Quotient (HQ) as;

$$\textbf{HQ} = \frac{\text{ADI non}-\text{carcinogenic}}{\text{RfD}} \qquad \textbf{(Eqn. 3.8)}$$

Where:

RfD is the reference dose factor (chemical specific). The oral chronic reference doses are 3.00E-04 mg/kg-day and 3.5E-03 mg/kg-day; dermal chronic reference doses 1.23E-03 mg/kg-day and 5.25E-04 mg/kg-day for As and Pb respectively.

HQ is a dimensionless quantity, that is expressed as the probability of an individual suffering an adverse effect and the RfD values that go in its denominator are such that the critical value for *HQ* is unit: If HQ is bigger than 1, then the ADI of a particular metal exceeds the RfD, indicating that there is a potential risk associated with that metal.

For n number of heavy metals, the non-carcinogenic effect to the population is as a result of the summation of all the HQs due to individual heavy metals. The sum is referred to as the Hazard Index (HI) expressed as:

$$\mathbf{HI} = \sum_{k=1}^{n} \boldsymbol{HQk} = \sum_{k=1}^{n} \frac{\boldsymbol{ADIk}}{\boldsymbol{RfDk}} \qquad \textbf{(Eqn. 3.9)}$$

Where HQk, ADIk and RfDk are values of heavy metal k

ii) Carcinogenic risk characterization

For carcinogenic risk characterization, according to U.S. EPA (2011) the risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen.

The equation for calculating the excess lifetime cancer risk was:

$$\boldsymbol{Risk_{pathway}} = \sum_{k=1}^{n} \boldsymbol{LADI_k\, CSF_k} \qquad \textbf{(Eqn. 3.10)}$$

Where:

Risk is a unit less probability of an individual developing cancer over a lifetime. $LADI_k$ in mg/kg/day and CSF_k (chemical specific; oral slope factor 1.5 mg/kg/day and 0.085

mg/kg/day; dermal slope factor 3.66 mg/kg/day and 0.0085 mg/kg/day for As and Pb, respectively) are the lifetime average daily dose and the cancer slope factor, respectively for the kth heavy metal, for n number of heavy metals through the ingestion pathway. The slope factor converts the estimated daily intake of the heavy metal averaged over a lifetime of exposure directly to incremental risk of an individual developing cancer.

The total excess lifetime cancer risk (ELCR) for an individual was finally calculated from the average contribution of the individual heavy metals for all the pathways using the following equation:

Risk (total) = Risk (der/ing) + Risk (der) + Risk (ing) **(Eqn. 3.11)**

Where: Risk (der/ing), Risk (ing) and Risk (der) are risks contributions through dermal and subsequent ingestion, ingestion and dermal pathways.

Resultant data from the calculated exposure assessment levels and risk characterization for both lead and arsenic heavy metals were then compared with U.S. EPA's acceptable excess lifetime cancer risk (ELCR) of 1×10^{-6} - 1×10^{-4} risk levels or one individual in 10,000 – one individual in 1,000,000 persons developing cancer.

3.5.7 Central tendency and reasonable maximum exposure risks

This was done by the point estimate method range of exposure levels that the central tendency exposure (CTE) and reasonable maximum exposure (RME) scenarios. According to U.S. EPA (2010) and Boffetta *et al*., (2011), a RME scenario assesses risk to individuals whose exposure characteristics may result in much higher potential exposure than seen in the average individual while a CTE scenario assesses potential risk to an average member of the population. As for most scenarios, for large number of

samples as is the case with Pb in this study, representative concentrations were calculated using means as CTE estimates while the 95% upper confidence limits (UCLs) on the mean were used to calculate RME estimates (U.S. EPA, 2007).

Lead and arsenic CTE and RME non-carcinogenic risks were characterized using a hazard quotient (HQ), while carcinogenic risks were characterized by multiplying the LADI with chemical specific cancer slope factors. The results were then compared with U.S. EPA (2010) default CTE and RME value for Pb and As.

Whenever the number of detected values was small, as is the case with As results in this study, the approach for "calculating representative CTE and RME concentrations for detect sample sizes, *n*, from 1 to 4 was:

n = 1; there is no RME, the result is CTE;

n = 2; there's no RME, maximum detect is CTE,

n = 3 or 4; maximum detect is RME, the mean is CTE".

3.6 Data analyses and presentation

Data collected on Pb and As concentration levels were entered, collated and managed using Predictive Analytic Software for windows (version 23.0). Univariate analysis was used to determine means, range and percentages for each sampling station. Results were then compared with stipulated acceptable lead and arsenic risk based concentrations from several countries as well stipulated levels from internationally recognized bodies such as WHO, Food and Agriculture Organization (FAO), European Union (EU) as well as U.S. EPA to ascertain heavy metal contamination factors and inferences made from resultant data.

T-test (Microsoft Excel, 2016) analysis was done to ascertain significant differences between the concentrations of heavy metals in the samples collected from the different sampling stations.

Heavy metal results were further extrapolated to estimate lifetime and average daily intake (LADI and ADI) for metal exposure, characterize health risks as well as to ascertain the CTE and RME risks of the studied work-units. T-test was further subjected to test for significant variations.

One-way analysis of variance (ANOVA; $p<0.05$) was conducted for multi-comparison of the concentrations with stipulated world standards such as the WHO as well as extrapolated ADI and LADI risk assessment results. Further, Tukey's HSD test of significance was carried out to ascertain the sources of variations. Tables and figures were used to present the results.

CHAPTER FOUR

RESULTS

4.1 Laboratory hygiene, safety practices and worker traits

4.1.1 Laboratories hygiene and safety practices

Workplace hygiene and the safety practices in place were studied for each of the 10 work-units. The nature of each facility's activity is as shown in Appendix III. Results from the interview guide (Appendix IV) indicated that workplace hygiene and safety occupationally exposed Pb and As to staff working in these facilities. A summary of the results are as shown in Appendix V.

A facilities walk-through survey found out that in all the facilities there was visible settled dust on flat surfaces, sampling of settled indoor dust was therefore possible as per the objectives of this study. Results of the interview schedule indicated that the facilities housekeeping in all the sampling stations mainly involved brush and pan dry sweeping of the floor surfaces except for the chemistry laboratories (EC, ECA, MC) which employed both wet and dry cleaning practices on weekly basis on average. Housekeeping for 6 (60%) of the laboratories was therefore done without any observable routine as per the discretion of the concerned staff with only REW (10%) being dry swept on a daily basis. It was notable that RMD workshop was not cleaned for the entire sampling period. None of the facilities employed vacuum cleaning method.

Seven (70%) of the facilities studied employed local ventilation methods which mainly involved opening the windows. Only the 3 chemistry laboratories had fitted extract fume

cupboards (hoods) which were found to be inadequate due to the high number of students involved. Results from the interview guide further found out that in all the work units, there was no program for regular or periodical monitoring of fumes, dust, gases or vapours. Besides the preparation rooms which doubled up as storage rooms, this study further found out that there was no clear delineation of areas with toxic (including Pb and As) containing solutions and other wastes in their safe handling, storage, management of spills and disposal.

Availability and proper use of required personal protective equipment (PPE) was also assessed. It was found that all the 10 work-units had been provided with only dust coats notably navy blue coats for the workshops and white for the chemistry laboratories, as the required PPE's. Though some activities required other PPE's such as respirators, overalls, gloves, safety boots, these were not provided for all the work units. The dust coats were hardly washed with clearly noticeable dirt especially for the workshops work units. Cleaning of the dust coats was done in their homes for all the studied work-units.

In essence, all the studied facilities lacked waste collection schedules, safety boots, staff periodical training, a risk assessment tool, electronic inventories of safety data sheets (SDS), hazard warnings, medical surveillance nor occupational injuries and illness form (OIIF). This should therefore be a concern to the occupational safety of the employees engaged in these facilities.

4.1.2 Worker - individual traits and behaviour

Reports of the interview schedule reported that occupational mobility as an indicator of worker-individual traits that may affect exposure was generally absent in the skilled

permanent staff with their occupational tenure ranging from 5-36 years. Further, all staff both male and female had no noticeable facial hair while all had noticeable hair on their heads. Hand washing habits varied with all admitting to at some point snacking in the workplace without prior washing of hands. Only three male smokers were reported in all the facilities and they never washed their hands before smoking. All the overalls and dust coats were left in the facility premises.

Results of micro-activity data in the different work-units were collected by closely observing workers as they went about their routine tasks, though this activity was not explicitly revealed to the worker. The results showed that in some occasions, a worker would bring his hand into contact with various surface areas in the facility as well as his/her face including the perioral area. From the fore-going therefore, it was necessary to study the possibility of inadvertent exposure to selected heavy metals as per the objectives of this study.

4.2 Heavy metal concentrations in settled indoor dust

4.2.1 Lead concentrations

Figure 4.1 below illustrates results of the lead concentrations in dust samples collected from the study area. Concentrations ranged from 165.533 mg/kg to 921.400 mg/kg in samples from RMR and RMD, respectively. The mean lead concentrations in settled dust ranged from 344.890±12.267 mg/kg to 754.438±76 mg/kg at REW and RMD stations, respectively.

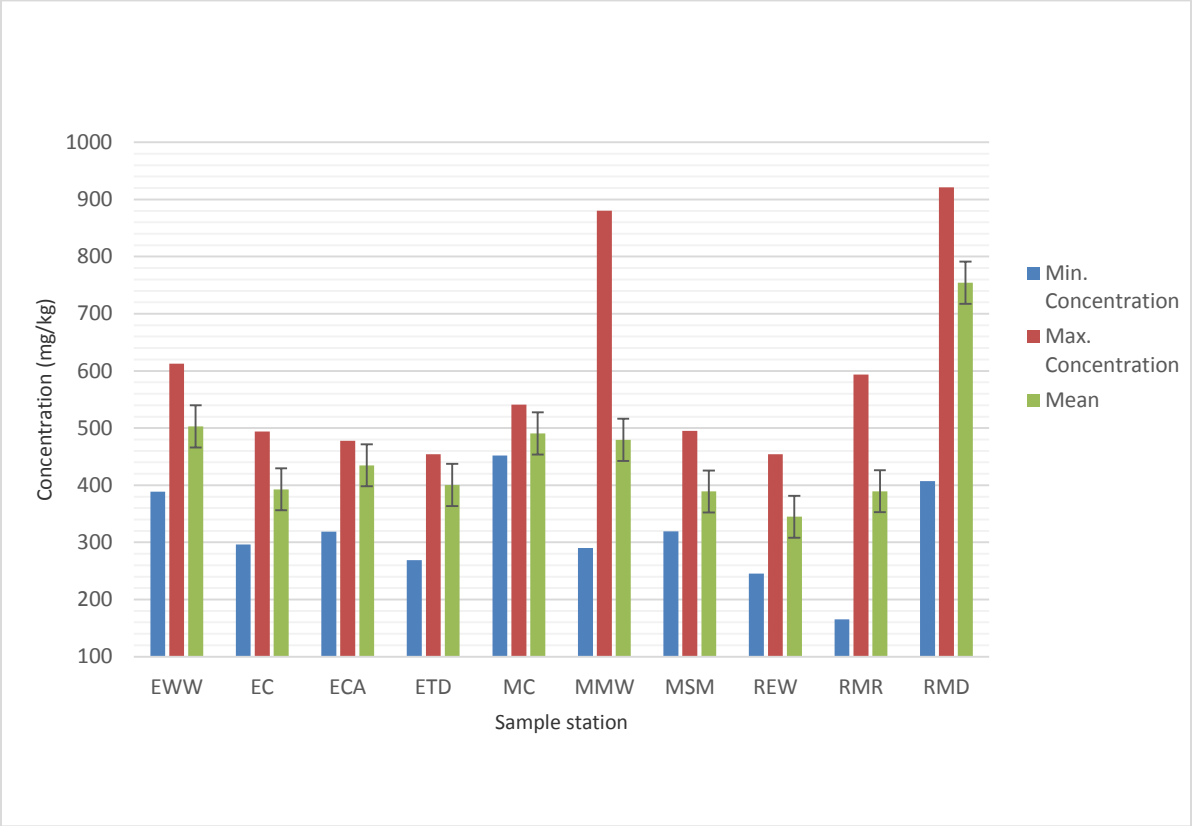

Figure 4.1: Pb Concentrations in the Sampling Stations

At 95% (CI), one sample t-test analysis for comparison of mean lead concentrations with EU (European Commission on Envt, 2002; 300 mg/kg); U.S. EPA (2011; 400 mg/kg) and WHO (2010b; 100 mg/kg) recommended standards in uncontaminated dust are as indicated in Table 4.1. The results showed that dust in studied samples in most sampling stations mean lead levels were significantly above the recommended standards. All mean lead levels significantly surpassed WHO/FAO (p = 0.000) and EU (p < 0.05) recommended standards. With an exception of RMR sampling station which was significantly lower (p =0.38) when compared to U.S. EPA standards, all the studied stations therefore had significantly elevated lead levels (p < 0.05).

One-way ANOVA analysis showed significant variations (P = 0.000) in mean Pb concentrations. Tukey's post hoc analysis showed that mean lead concentrations in samples from EWW, EC, ECA, MMW, MSM, ETD, REW and RMR were homogenous indicating insignificant variation. Variation was observed between RMD and all other sampling stations (p = 0.0001), and between sampling stations MC and REW (p = 0.048). The mean Pb concentrations were considered to be elevated and thus were further used to calculate average daily intakes for Pb non-carcinogenic and carcinogenic risk assessment.

Table 4.1: One Sample T-test *p* Values for Mean Pb Levels against International Standards in Soil

S. Station	EU (*p* values)	U.S. EPA (*p* values)	WHO/FAO (*p* values)
EWW	2.5 E-09	2.5E-06	1.54E-12
EC	0.000169	0.002739	2.68E-09
ECA	4.72E-08	0.00439	3 E-12
ETD	4.29E-05	0.008523	1.49E-09
MC	5.13E-10	3.42E-07	8.41E-13
MMW	0.00509	0.00991	2.07E-05
MSM	9.31E-06	0.006821	5.36E-11
REW	0.01197	0.00402	4E-09
RMR	0.01164	0.3826	1.13E-06
RMD	0.00028	0.00116	2.87E-05

4.2.2 Arsenic concentrations

Figure 4.2 illustrates results of arsenic concentrations in dust samples collected from the study area. Concentrations ranged from 0.04 ppm to 349.24 ppm in samples from ETD and RMD, respectively, while mean As concentrations ranged from 0.424 ppm to 131.73 ppm at ETD and RMD stations, respectively.

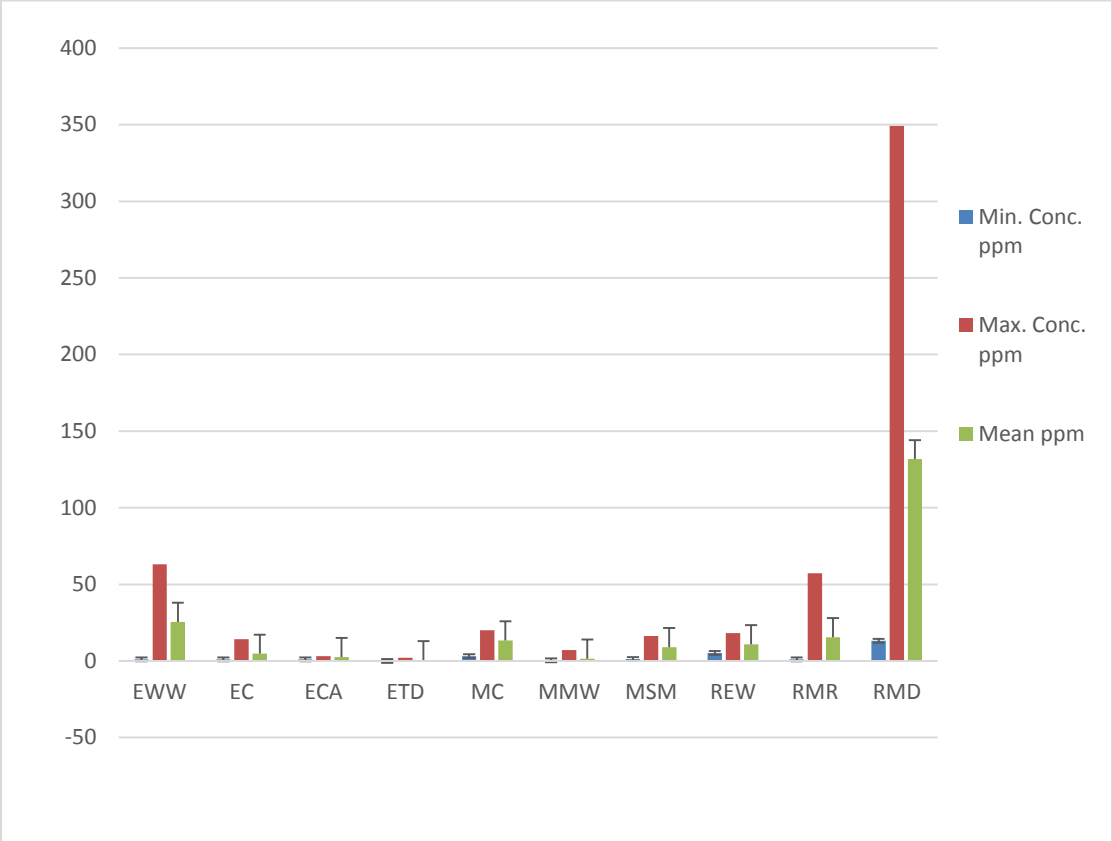

Figure 4.2: Arsenic Concentrations in the Sampling Stations

At 95% confidence level (CI), t-test analysis for comparison of the results with EU (European Comm. on Envt, 2002; 20 mg/kg), FAO/WHO (2012; 20 mg/kg) and U.S. EPA (2011; 7 mg/kg) recommended standards in uncontaminated dust in studied samples mean arsenic levels are as shown in Table 4.3. The results indicated that mean dust levels were significantly lower ($p > 0.05$) in most (80%) sampling stations than EU and FAO/WHO standards while 60% of the stations significantly surpassed ($p < 0.05$) U.S. EPA standards.

One-way ANOVA analysis showed significant variation ($p = 0.004709$) in mean As concentrations. Tukey's HSD post hoc analysis shows that samples from EWW, EC,

ECA, MC, MMW, MSM, ETD, REW and RMR were homogenous indicating insignificant variation while RMD work-unit showed significant variation with all other work-units. The concentrations were used to estimate average daily intakes for As non-carcinogenic and carcinogenic risk assessment.

Table 4.2: One Sample t-test *p* Values for Mean As levels against International Standards in Soil

S. Station	EU (*p* values)	WHO/FAO (*p* values)	U.S. EPA (*p* values)
EWW	0.032	0.032	0.088
EC	0.998	0.998	0.795
ECA	0.300	0.300	0.402
ETD	0.380	0.380	0.128
MC	0.952	0.952	0.054
MMW	0.217	0.217	0.390
MSM	0.100	0.100	0.079
REW	0.992	0.992	0.026
RMR	0.354	0.354	0.024
RMD	0.0121	0.0121	0.010

4.3 Estimation of non-carcinogenic occupational exposure

Non-carcinogenic average daily intake (ADI) for dermal contact with a contaminated surface and subsequent transdermal uptake (ADIder); dermal contact with a contaminated surface followed by inadvertent ingestion of contaminants transferred to skin ((ADIder/ing) and for inadvertent occupational ingestion (ADIing) were calculated using equations 3.2, 3.3 and 3.6, respectively, from mean Pb and As concentrations at each sampling station.

4.3.1 Occupational lead non-carcinogenic exposure assessment

Mean total lead ADI results for men and women are as shown in Table 4.3. The findings from this study indicated that RMD work-unit recorded the highest total ADI for Pb at 6.7744×10^{-3} mg/kg/day and 7.8866×10^{-3} mg/kg/day body weight for men and women workers, respectively, while REW work-unit recorded the least calculated ADI at 3.0970×10^{-3} mg/kg/day and 3.6130×10^{-3} mg/kg/day body weight for men and women, respectively.

Table 4.3: Lead Total ADI Results for Men and Women

	Mean Total ADI $\times10^{-3}$		**P value (1-tailed) (RfD)**		**P value (2-tailed) between M and W**
W.U	**M**	**W**	**M**	**W**	
EWW	4.5161	5.2688	0.0000	0.000	0.0003
EC	3.5266	4.1143	0.4332	0.004	0.0285
ECA	3.9039	4.5541	0.0009	0.000	0.0003
ETD	3.5950	4.1941	0.0001	0.0085	0.0120
MC	4.3364	5.0590	0.0000	0.000	0.0000
MMW	4.3364	5.0213	0.0113	0.0236	0.3307
MSM	3.4934	4.0756	0.4770	0.0005	0.0026
REW	3.0970	3.6130	0.1512	0.3270	0.3436
RMR	3.4973	4.0801	0.4879	0.0670	0.2274
RMD	6.7744	7.8866	0.0010	0.0004	0.3005

W.U – Work-unit M – Men W – Women

At 95% confidence interval (CI), one sample t-test results for comparison of lead total ADI's for both men and women in the work-units with reference doses are also as shown in Table 4.3. The results indicate that 60% of the work-units had significantly higher ADI levels than the reference dose. Further, comparison of lead total ADI's for men in the

entire study area, results indicate that they were found to be above Pb reference doses (p = 0.0493), while those for women were also found to be higher (p = 0.00421).

Comparison for both men and women total ADI using two sample two tailed t-test (p = 0.05) indicate that there was no variation in lead total ADI between men and women (p = 0.19595) in the entire study area. However, there were significant variations between men and women Pb ADI exposures in the same work-units in 60% of the work-units as shown in Table 4.3.

One-way ANOVA analysis showed significant variation for lead total ADI in men (p =0.000) and women (p =0.000) within the work-units. Tukey's HSD post hoc analysis indicated total ADI's for EWW, EC, ECA, MMW, MSM, ETD, REW and RMR work-units were homogenous indicating insignificant variation while there was significant variations between RMD and all other work-units (p = 0.0001), between MC and ETD (p = 0.005) and between MC and REW (p = 0.025) for both men and women, respectively.

4.3.2 Occupational arsenic non-carcinogenic exposure assessment

Similarly for arsenic as shown in Table 4.4, RMD work-unit had the highest calculated total ADI at 31.701×10^{-4} mg/kg/day and 36.9845×10^{-4} mg/kg/day body weight for men and women, respectively. On the other hand, ETD work-unit had the least calculated ADI at 0.0992×10^{-4} mg/kg/day and 0.1158×10^{-4} mg/kg/day body weight for men and women, respectively.

At 95% (CI), a one sample t-test for total ADI variation between individual work-units from arsenic reference doses indicate that only EWW, RMR and RMD work-units were

significantly ($p < 0.05$) exposed to arsenic non-carcinogenic effects for both men and women while MC work-unit was significantly exposed for women only. The results are as shown in Table 4.4.

At 95% (CI), a two sample t-test for arsenic variations between men and women in the work-units are as shown in Table 4.4. The results show that there was no observed significant variation between men and women total ADI exposure in all the work-units ($p > 0.05$).

Table 4.4: Arsenic Total ADI results for men and women

	Mean Total ADI $\times 10^{-4}$		P value (1-tailed) (RfD)		P value (2-tailed) between M and W
W.U	**M**	**W**	**M**	**W**	
EWW	6.1481	7.1729	0.0089	0.0077	0.765561
EC	1.1133	1.3218	0.9667	0.0800	0.716948
ECA	0.6088	0.7103	0.9998	0.9995	0.602685
ETD	0.0992	0.1158	1.0000	1.0000	0.603801
MC	3.2156	3.7516	0.0808	0.0500	0.643743
MMW	0.3446	0.4020	0.9998	1.0000	0.747385
MSM	2.1627	2.5286	0.3890	0.2612	0.746074
REW	2.6211	3.0603	0.1414	0.0751	0.604066
RMR	3.6250	4.3574	0.0074	0.0045	0.379076
RMD	31.701	36.9845	0.0533	0.0500	0.816552

One-way ANOVA analysis showed significant variations for arsenic total ADI in men ($p = 0.000704$) between the work-units. Tukey's HSD post hoc analysis showed total ADI's for EWW, EC, ECA, MC, MMW, MSM, ETD, REW and RMR work-units were homogenous indicating insignificant variation. However, there was significant variations due to significant differences between RMD and EC ($p = 0.002$), ETD ($p = 0.001$), MC

(p = 0.005), ECA (p = 0.002), MMW (p = 0.001), MSM (p = 0.003), REW (p = 0.004) and RMR (p = 0.006) in men.

Further, one-way ANOVA analysis indicated significant variations for arsenic total ADI in women (p = 0.00708). Variations were due to significant differences between RMD and EC (p = 0.0019), ETD (p = 0.0008), MC (p = 0.0053), ECA (p = 0.0024), MMW (p = 0.0011), MSM (p = 0.003), REW (p = 0.0042) and RMR (p = 0.0055)**.** Thus, significant variation in arsenic non-carcinogenic exposure was also attributed to elevated ADI levels from the RMD work-unit that had relatively higher ADI than the rest.

One sample t-test results for arsenic total ADI for both men and women in the entire study area were found to be significantly below (p = 0.05993; p = 0.307817) the RfD's, respectively, with both exhibiting a high variance due to the effect of high recorded mean As levels at EWW and RMD work-units. Further, the results indicate that there was no significant variation in arsenic total ADI between men and women (p = 0.851545) in the entire study area.

4.4 Occupational non-carcinogenic risk characterization

The non-carcinogenic risk for dermal contact with a contaminated surface and subsequent transdermal uptake (HQder); dermal contact with a contaminated surface followed by inadvertent ingestion of contaminants transferred to skin (HQder/ing) and for inadvertent occupational ingestion (HQing) of dust were calculated separately for both Pb and As for each work-unit as per equation 3.8. The total risk (HI) was then ascertained by summing up the resultant risks as per equation 3.9.

4.4.1 Occupational lead non-carcinogenic risk characterization

The calculated HI's using mean Pb indoor settled dust concentrations and the U.S. EPA (2007) reference doses for the considered pathways for both male and female employees scenarios at the various sampling locations were as presented in Table 4.5. The mean HQ's for all the work-units ranged from 0.8849 - 1.9356 and 1.0322 - 2.2533 for men and women, respectively. The highest HQ was recorded at RMD work-unit with the lowest recorded at REW work-unit.

Table 4.5: Lead Non-Carcinogenic Risk Characterization

	Mean HQ		P value (1-tailed) (HQ =1)		P value (2-tailed) between M and W
W.U	**M**	**W**	**M**	**W**	
EWW	1.2903	1.5053	0.0000	0.0000	0.0003
EC	1.0076	1.1756	0.0432	0.0018	0.0126
ECA	1.1153	1.3013	0.0909	0.0000	0.0004
ETD	1.0271	1.1959	0.0319	0.0451	0.1531
MC	1.2589	1.4687	0.0000	0.0000	0.0000
MMW	1.2297	1.4346	0.0159	0.0236	0.3193
MSM	0.9981	1.1645	0.4776	0.0005	0.0026
REW	0.8849	1.0322	0.4420	0.0273	0.0399
RMR	0.9993	1.1657	0.4966	0.0543	0.2045
RMD	1.9356	2.2533	0.0010	0.0004	0.3006

At 95% (CI), one sample t-test results for comparison of lead HQ's for the considered pathways for both men and women in the work-units with unit (1) are as shown in Table 4.5. The results indicate that women in all the work-units had significantly higher HQ's than unit ($p < 0.05$) implicating they could be potentially at risk of lead non-carcinogenic exposure. However, for men, 30% of the work-units (RMR, REW and MSM) had HQ's

significantly lower than unit ($p > 0.05$) implying they could be potentially safe. Comparison for both men and women unit using two sample two tailed t-test indicate that there were variations in 60% of the work-units (EWW, EC, ECA, MC, MSM and REW).

One-way ANOVA analysis showed there was significant variation in lead HQ in men ($p = 0.0000$) between the work-units. Further, Tukey's HSD post hoc analysis showed there was significant variation in men Pb HQ between the work-units ($p = 0.0000$). Besides RMR work-unit, RMD indicated similar significant variations ($p = 0.0010053$) with all the other work-units. Further, significant variations were also found between EWW and REW ($p = 0.00361$), MC and REW ($p = 0.02211$).

Similarly, women also exhibited significant variations ($p = 0.000472$) in Pb HQ between the work-units. Tukey's HSD post hoc analysis showed there was significant variation in women Pb HQ between the work-units ($p = 0.0001$). Significant variations were found only between RMD and other work-units EC ($p = 0.00749$), ECA (($p = 0.03646$), MMW ($p = 0.04199$), MSM ($p = 0.003599$), REW ($p = 0.00101$) and RMR ($p = 0.00339$).

At 95% (CI), two sample t-test indicate that there was significant variation ($p = 0.0193816$) between men Pb-HQ and women Pb-HQ in the entire study area. Lead mean HQ results for men in the entire study area were found to be above unit ($p = 0.048607$). Similarly, those for women were also found to be higher ($p = 0.00413$). Further, it was observed that there was no variation in lead non-carcinogenic risk between men and women ($p = 0.851545$) in the entire study area.

4.4.2 Occupational arsenic non-carcinogenic risk characterization

As indicated in Table 4.6, RMD work-unit recorded the highest mean HQ with ETD recording the least. Theoretical mean arsenic HQ for all the work-units ranged from 0.0340 - 10.5670 and 0.0397-12.3281 for men and women, respectively.

At 95% (CI), one sample t-test results for comparison of As HQ for the considered pathways for both men and women in the work-units with unit (1) are as shown in Table 4.6. The results indicated that only 40% of the work-units had significantly higher HQ than unit ($p < 0.05$) for both men and women implicating they could be at risk of arsenic exposure. Comparison for both men and women unit using two-sample two tailed t-test indicate that there were no significant variation ($p = 0.854724$) between men As-HQ and women As-HQ.

Table 4.6: Arsenic Non-Carcinogenic Risk Characterization

	HQ		P value (1-tailed) (HQ =1)		P value (2-tailed) between M and W
W.U	**M**	**W**	**M**	**W**	
EWW	2.0493	2.3910	0.010899	0.008766	0.76544
EC	0.3777	0.4406	0.946386	0.919724	0.81773
ECA	0.2029	0.2367	0.999806	0.999524	0.60005
ETD	0.0340	0.0397	0.999996	0.999997	0.31845
MC	1.0719	1.2506	0.048084	0.041761	0.64350
MMW	0.1149	0.116	0.999837	0.999698	0.74833
MSM	0.7224	0.8428	0.388923	0.261137	0.74593
REW	0.8744	1.0201	0.141294	0.07506	0.60447
RMR	1.2450	1.4525	0.007425	0.0045	0.37726
RMD	10.5670	12.3281	0.05337	0.052715	0.34683

One-way ANOVA analysis showed there was significant variation in As HQ in men (p = 0.000443) between the work-units. Further, Tukey's HSD post hoc analysis showed there was significant variation in men As HQ between the work-units (p = 0.0068). Variations were recorded between RMD and EWW; EC; ECA; ETD; MMW ((p = 0.0010), MSM (p = 0.00162), REW (p = 0.00200), and RMR (p = 0.00334).

Similarly to men, women exhibited significant variations (p = 0.000764) in As-HQ between the work-units. Tukey's HSD post hoc analysis showed there was significant variation in women As HQ between the work-units (p = 0.000764). Further, similarly with men, significant variations were found only between RMD and other work-units; ECA, ETD and MMW (p = 0.0010), EWW (p = 0.01239), EC (p = 0.00135), MC (p = 0.00349), REW (p = 0.00268) and RMR (p = 0.00438).

Contrary to lead, arsenic HQ results for men in the entire study area were found to be less than unit (p = 0.243459) with those for women also found to be less (p = 0.20453). Further, it was observed that there was no variation in arsenic non-carcinogenic effect between men and women (p = 0.851545) in the study area.

4.4.3 Cumulative occupational non-carcinogenic risk characterization

Assuming an additive effect from the considered pathways, cumulative mean heavy metal contributions to non-carcinogenic hazard index and their variations are as presented in Fig. 4.3. The ETD work-unit posed the least cumulative non-carcinogenic risk (HI) at >1.0611 and >1.2356 for both men and women, respectively. On the other hand, RMD work-unit recorded the highest non-carcinogenic risk (<12.5026; <14.5814) for both men and women, respectively.

At 95% (CI), two sample t-test indicate that there was no significant variation (p = 0.854123) between men As-HQ and women As-HQ in the entire study area. Further, there was neither significant variation (p = 0.590383) between men As-HQ and men Pb-HQ nor was there significant variation (p = 0.5991072) between women As-HQ and women Pb-HQ. Additionally, there was neither significant variation (p = 0.727732) between men As-HQ and women Pb-HQ nor was significant variation (p = 0.484382) recorded between women As-HQ and men Pb-HQ in the entire study area.

In the entire study area, at 95% (CI), one sample t-test indicated that Pb and As HI were significantly above unit (p = 0.053234; p = 0.004819) in men and women, respectively. General variations in lead and arsenic risks are as represented graphically in the clustered bar chart below (Fig. 4.3).

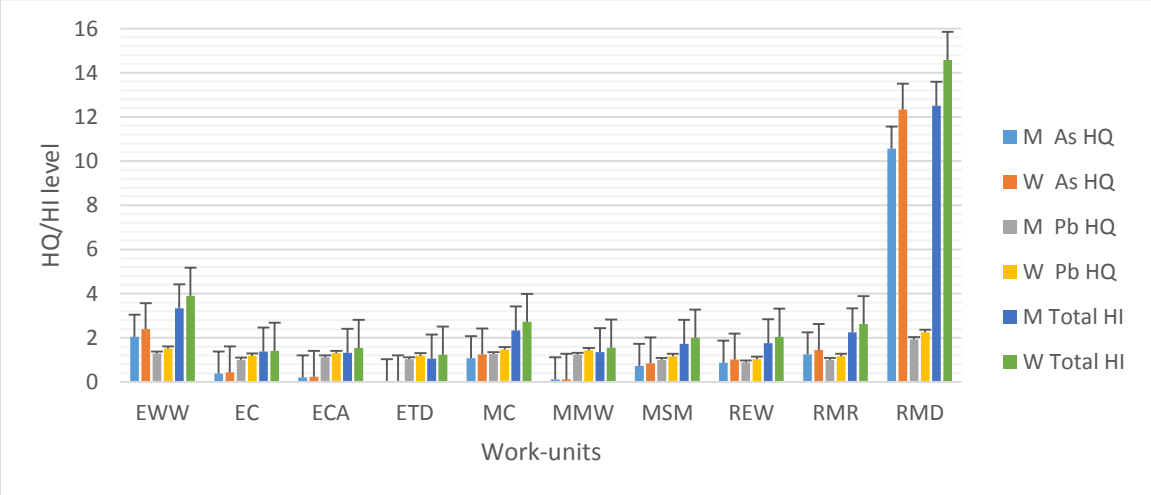

Figure 4.3: Lead, Arsenic and Cumulative Non-Carcinogenic Risk Characterization

4.5 Occupational carcinogenic risk characterization

Carcinogenic lifetime average daily intake (LADI) for dermal contact with subsequent transdermal uptake (LADIder), for dermal contact with a contaminated surface followed by inadvertent ingestion of contaminants transferred to skin (LADIder/ing) and for occupational ingestion were calculated as per equations 3.4, 3.5 and 3.7, respectively from mean Pb and As concentrations at each work-unit.

Total LADI results for excess lifetime cancer risks for both men and women were calculated as per equation 3.10 for each work unit. Assuming an additive effect, the total risk as per equation 3.11 for all the considered pathways were calculated for Pb and As in order to assess the average contribution of the individual heavy metals. The derived dosages were in essence multiplied by the cancer slope factors for both Pb and arsenic. The results were further evaluated for cancer-causing effects by comparing with U.S. EPA's acceptable excess lifetime cancer risk (ELCR) of 1×10^{-6} - 1×10^{-4} risk.

4.5.1 Lead carcinogenic risk characterization

Table 4.7 shows carcinogenic risk characterization for Pb concentrations. Mean risk results ranged from 7.7268×10^{-5} - 16.9026×10^{-5} and 8.9850×10^{-5} - 19.7197×10^{-5} for men and women, respectively. The REW and RMD work-units had the highest and least carcinogenic risk for both men and women, respectively.

At 95% confidence interval (CI), one sample t-test results for comparison of lead total LADI's from the considered pathways for both men and women in the work-units with lifetime carcinogenic risk (1×10^{-6} or one person in a million) are as shown in Table 4.5. The results indicate that all the work-units had significantly higher LADI levels than the

ELCR (p = 0.000) value. Comparison with acceptable ELCR 1 x 10^{-4} (or one person in ten thousand) levels indicated that half of the work-units (EWW, EC, MC, RMD, MSM) had significantly higher ($p < 0.05$) risks for both men and women. However, ECA work-unit also had a significantly higher risk (p = 0.000297) for women. The rest recorded theoretical carcinogenic risk within acceptable levels.

Comparison for both men and women risk using two sample two tailed t-test (p = 0.05) indicate that there were variations in lead total LADI between men and women in half (50%) of the work-units (EWW, ECA, ETD, MC and MSM) with the other half (EC, MMW, REW, RMR and RMD) indicating no variations.

Table 4.7: Lead Carcinogenic Risk Characterization

	Total RISK ×10^{-5}		**P value (1-tailed) (μ=1 x 10^{-4})**		**P value (1-tailed) (μ=1 x 10^{-6})**		**P value (2-tailed) M and W**
W.U	**M**	**W**	**M**	**W**	**M**	**W**	
EWW	11.2683	13.1465	0.00043	8.32E-07	2.58E-13	1.96E-13	0.000807
EC	8.7994	10.2657	0.0281	0.015396	2.11E-07	8.3E-06	0.733612
ECA	9.7405	11.3636	0.19058	0.000297	1.89E-13	3.73E-13	0.000368
ETD	8.9694	10.4649	0.3815	0.180774	1.63E-10	1.9E-10	0.009391
MC	10.9890	12.8267	0.00011	7.54E-08	2.36E-13	2.33E-13	1.19E-06
MMW	10.7391	12.5283	0.41598	0.129779	7.81E-07	7.12E-07	0.308456
MSM	8.7164	10.1694	0.00012	0.0378001	7.57E-13	3.06E-12	0.002147
REW	7.7268	8.9850	0.398	0.2422	8.83E-11	7.11E-08	0.497358
RMR	8.7264	10.1806	0.07855	0.421801	6.71E-08	5.22E-08	0.261433
RMD	16.9026	19.7197	0.00024	0.000889	1.18E-05	1.17E-05	0.300317

One-way ANOVA analysis showed significant variations (p =0.000) for lead carcinogenic risk in men, a similar trend also exhibited by women. Tukey's HSD post hoc analysis showed there was significant variation in men lead risk between work-units (p = 0.0000). Significant differences were due to variations between RMD and REW, EC, MSM, RMR, ETD, MMW, ECA and MC ($p < 0.0001$), and between MC and REW (p = 0.042). Similarly, there were significant differences in women's total LADI (p = 0.0000) with variations arising from differences between RMD and REW, EC, MSM, RMR, ETD, MMW, ECA ($P < 0.0001$), between RMD and MC ($p < 0.000$), and between MC and REW (p = 0.015).

One sample t-test results indicated there was no significant variation in means for lead carcinogenic risk between men and women (p = 0.1928) in the entire study area. Comparison with 1×10^{-4} risk value indicate that lead risk in men was significantly lower (p = 0.382236) but significantly above 1×10^{-6} risk value. (p = 0. 001). However, for women, risk was significantly higher when compared with both 1×10^{-4} risk value (p = 0.035785) and 1×10^{-6} risk value (p = 0.0004242) for the entire study area.

4.5.2 Arsenic carcinogenic risk characterization

Carcinogenic risk characterization for As for the considered pathways are shown in Table 4.8. Similar for Pb, RMD work-unit had the highest risk at 13.9584×10^{-4} and 16.2847×10^{-4} for men and women, respectively. Further, ETD work-unit recorded the least carcinogenic risk at 0.00689×10^{-4} and 0.00813×10^{-4} for men and women, respectively.

At 95% confidence interval (CI), one sample t-test results for comparison of As carcinogenic risk from the considered pathways for both men and women in the work-

units with acceptable ELCR values are as shown in Table 4.8. The results indicate that all the work-units had significantly higher risk levels than the ELCR value of 1 x 10^{-6} value ($p < 0.05$) except for men at ETD work-unit ($p = 0.097679$). Comparison with acceptable ELCR 1 x 10^{-4} levels indicated that 50% of the work-units (EWW, MC, REW, RMR, RMD) had significantly higher risks ($p < 0.05$) for both men and women with the other half having less. Comparison for both men and women total risk using two sample two tailed t-test indicate that there were no variations in As total risk between men and women in all the work-units as shown in Table 4.8.

Table 4.8: Arsenic Carcinogenic Risk Characterization

	Mean Risk ($\times 10^{-4}$)		**P value (1-tailed) (μ=1 x 10^{-4})**		**P value (1-tailed) (μ=1 x 10^{-6})**		**P value (2-tailed) M and W**
W.U	**M**	**W**	**M**	**W**	**M**	**W**	
EWW	2.7071	3.1582	0.011324	0.009868	0.043693	0.043682	0.81559
EC	0.4989	0.5819	0.11656	0.30832	0.012658	0.012547	0.715797
ECA	0.2682	0.3129	0.08533	0.08355	0.004284	0.009474	0.493698
ETD	0.00689	0.00813	0.79050	0.31601	0.097679	0.058856	0.49167
MC	1.4159	1.6519	0.013363	0.008057	0.006224	0.006203	0.656337
MMW	0.1518	0.1771	0.64550	0.14500	0.032137	0.036981	0.828219
MSM	0.9543	1.1134	0.461702	0.351318	0.017265	0.017862	0.713198
REW	1.1549	1.3475	0.026347	0.013995	0.00498	0.004267	0.660009
RMR	1.6446	1.9186	0.013879	0.007582	0.000518	0.00053	0.395494
RMD	13.9584	16.2847	0.006444	0.008088	0.0055342	0.0073102	0.703978

One-way ANOVA analysis showed there was significant variation for As carcinogenic risk in men ($p = 0.00207$) between the work-units. Similarly, women also exhibited

significant variations (p = 0.009594). Further, Tukey's HSD post hoc analysis showed there was significant variation in men As risk between work-units (p = 0.0000). Significant variation was found to be due to variations between RMD and ETD (p = 0.003), RMD and MWW (p = 0.004), RMD and ECA (p = 0.004), RMD and EC (p = 0.005), RMD and MSM (p = 0.007), RMD and REW (p = 0.008), RMD and MC (p = 0.010), and RMD and RMR (P = 0.012).

Similarly, there were significant differences in women's As risk (p = 0.0000). Significant variation was found to be due to variations between RMD and EC (p = 0.018), RMD and ECA (p = 0.016), RMD and ETD (p = 0.014), RMD and MWW (p = 0.015), RMD and MSM (p = 0.024), RMD and REW (p = 0.026), RMD and MC (p = 0.030), and RMD and RMR (p = 0.034).

Moreover, one sample t-test indicated As carcinogenic risk in the entire study area were found to be similar and significantly above (p = 0.0001) ELCR value (1×10^{-6}) for both men and women. However, risk was lower than 1 x 10^{-4} risk value for both men (p = 0.180078) and women (p = 0.155792).

4.5.3 Cumulative occupational carcinogenic risk characterization

Total carcinogenic risk arising from both mean As and Pb concentrations when considering risks from all the considered pathways for all work units were also as shown in Figure 4.4. As expected due to the high elevated levels for both As and Pb in RMD work-unit, this work unit theoretically posed the highest risk for both As and Pb carcinogenic risk with a total risk of 17.9750E-05 and 18.2567E-05 for men and women, respectively. Similarly for non-carcinogenic risks, ETD work-unit had the least

carcinogenic risk for both Pb and As with an aggregate risk of 0.8763E-05 and 1.0546E-05 for men and women, respectively in the entire study area.

At 95% (CI), two sample t-test indicate that there was neither significant variation (p = 0.358119) between men-Pb and men-As risk nor was there significant variation (p = 0.305781) between men-Pb and women-As carcinogenic risk in the entire study area. Further, there was neither significant variation (p = 0.358172) between women-Pb and women-As nor was there significant variation (p = 0.426545) between women-Pb and men-As. Additionally, no significant variation (p = 0.854123) was recorded between men and women cumulative risk in the entire study area.

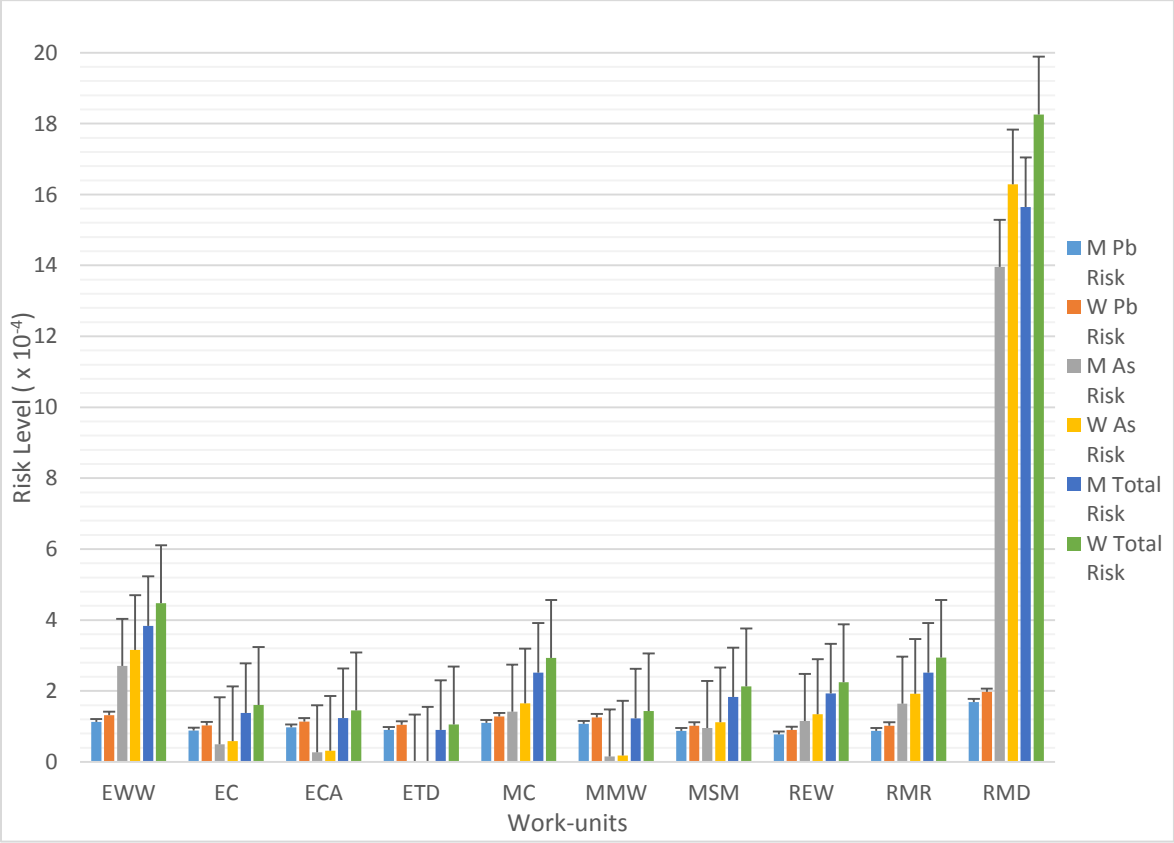

Figure 4.4: Lead, Arsenic and Cumulative Carcinogenic Risk Characterization

At 95% (CI), one sample t-test indicated that aggregate heavy metal carcinogenic risk from both Pb and As for men in the entire study area were found to be significantly above ELCR value of 1×10^{-6} in both men (p = 0.021539) and women (p = 0.021488). Additionally, they were above the 1×10^{-4} risk value at p = 0.0467124 and p = 0.052721 for men and women, respectively. General variations in lead and arsenic risks are as represented graphically in the clustered bar chart (Fig. 4.4).

4.6 Work units CTE and RME to non-carcinogenic risks

Assessment of work-units range of exposure for non-cancer risks were evaluated by comparing calculated CTE (central tendency exposure) and RME (reasonable maximum exposure) risks values as per U.S. EPA (2011) default values with unit. For non-carcinogenic exposure, hazard quotient (HQ) and hazard index (HI) greater than 1 indicated that the health-based guideline had been exceeded.

4.6.1 Non-carcinogenic central tendency exposure risk

Figure 4.5 presents the CTE non-carcinogenic risk characterization results for Pb and As non-cancer effects for both men and women. The CTE HQ's for Pb ranged from 0.885 – 1.935 and 1.032 – 2.252 with As CTE HQ's ranging from 0.17 – 10.567 and 0.198 – 12.328 for men and women, respectively.

At 95% (CI), one sample t-test CTE risk for Pb in the entire study area was significantly above unit in all the work-units (p = 0.049327; p = 0.004788) for both men women, respectively. On the contrary, arsenic CTE HQ's were significantly lower (p = 0.150897; p = 0.121806) for non-carcinogenic risk for men and women, respectively. Thus, as per the findings of this study, all the work-units were theoretically at risk to Pb CTE but were

not at risk to As CTE non-cancer risks. Aggregate risk from both Pb and As were thus significantly above unit (p = 0.0093936; p = 0.0093232) for men women, respectively.

Further, at 95% (CI) two sample t-test indicated that there were neither significant variations ($p > 0.05$) between Pb CTE HQ's and As CTE HQ's nor between aggregate HI (p = 0.754732) for men and women in the entire study area. Hence there was anticipated risk from CTE non-carcinogenic exposure accruing from As exposure.

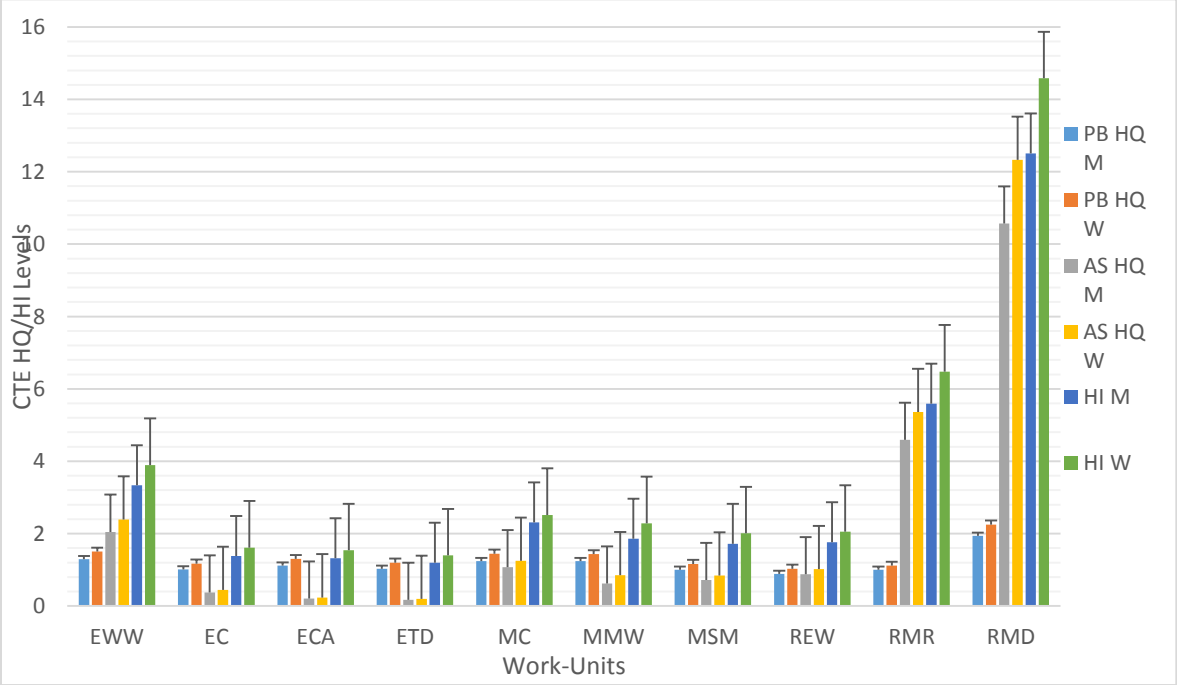

Figure 4.5: CTE for Lead and Arsenic Non-Carcinogenic Risk

4.6.2 Non-carcinogenic reasonable maximum exposure risk

Lead RME HQ's ranged from 0.916 – 2.131 and 1.061 – 2.486 while As RME HQ's ranged from 0.091 – 28.015 and 0.097- 32.682 for men and women, respectively. The findings are as shown in Fig. 4.6. At 95% (CI), similarly to CTE risk results, one sample t-test indicated that Pb RME risk for all the work-units were significantly higher (p =

0.035668; p = 0.004294) than 1 while that for As was significantly lower (p = 0.158235; p = 0.148912) for men and women, respectively, in the entire study area (Fig. 4.6).

Moreover, as was the case with CTE, the findings of this study indicated that all the work-units in the entire study area were theoretically at risk to Pb but were not at risk to As RME non-carcinogenic risks. However, aggregate risk from both Pb and As were thus significantly above unit (p = 0.033651; p = 0.027562) for men women, respectively.

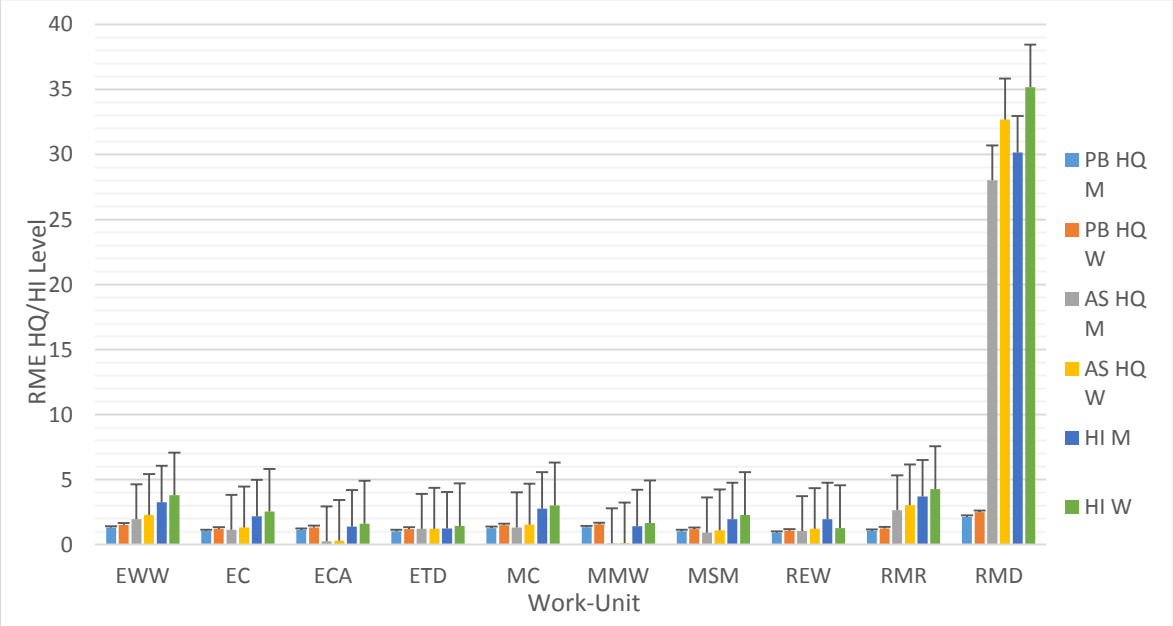

Figure 4.6: RME for Lead and Arsenic Non-Carcinogenic Risk

Further, as was the case with CTE risk results, at 95% (CI) two sample t-test indicated that there were neither significant variations (p > 0.05) between Pb RME HQ and As RME HQ nor between aggregate HI (p = 0.872168) for men and women in the entire study area. Thus, as per the findings of this study, all the work-units were theoretically at risk to aggregate Pb and As RME non-cancer risks.

4.7 Work units CTE and RME to carcinogenic risks

Assessment for CTE and RME cancer risks was done by comparing theoretical CTE and RME risks as per the findings of this study with U.S. EPA's (2011) excess lifetime cancer risk (ELCR) $1x10^{-6}$ - $1x10^{-4}$excess cancer risk for regulatory purposes.

4.7.1 Carcinogenic central tendency exposure risk

Figure 4.7 presents the CTE carcinogenic risk results. Central tendency exposure risks for Pb ranged from 7.7268×10^{-5} – 16.9026×10^{-5} and 8.985×10^{-5} – 19.7197×10^{-5} for men and women, respectively. Arsenic CTE risks ranged from 0.2246×10^{-4} – 37.0062×10^{-4} and 0.2621×10^{-4} – 43.1739×10^{-4} for both men and women, respectively with RMD posing the highest risk for both Pb and As.

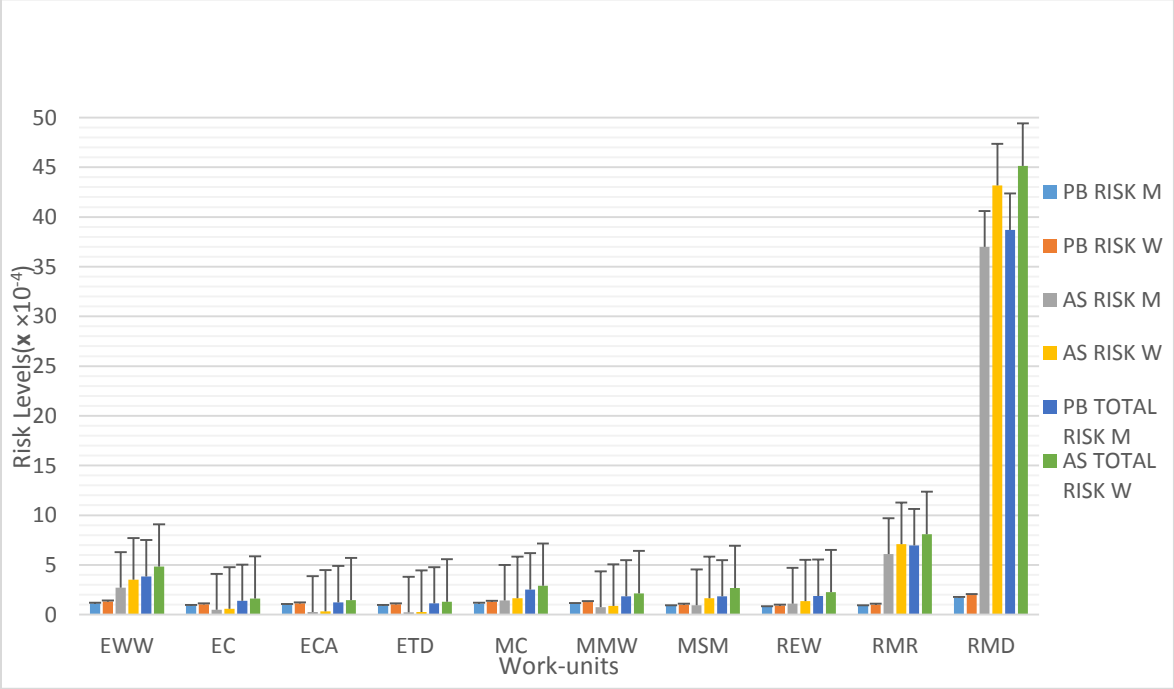

Figure 4.7: CTE for Lead and Arsenic Carcinogenic Risk

At 95% (CI), one sample t-test CTE risk for Pb in the entire study area were significantly above the lower bound acceptable $1x10^{-6}$ excess cancer risk (p = 0.000) for both men women. Similarly, arsenic CTE HQ's were significantly higher (p = 0.009464; p = 0.0091106) for carcinogenic risk for men and women, respectively.

However, when compared to the upper bound acceptable $1x10^{-4}$ excess cancer risk, both men (p = 0.3082) and women (p = 0.3785) were not significantly at risk to Pb CTE risk. Arsenic CTE risk were significantly higher than acceptable ELCR levels (p = 0.014104; p = 0.01288) for men and women, respectively. Aggregate CTE for carcinogenic risks were thus significantly above ELCR (p = 0.009729; p = 0.008858) for men and women, respectively.

Further, at 95% (CI), two sample t-test indicated that there were no significant variations ($p > 0.05$) between Pb CTE risk and As risk nor between aggregate risk (p = 0.845696) for men and women in the entire study area. Hence there was anticipated risk from aggregate Pb and As CTE carcinogenic exposure.

4.7.2 Carcinogenic reasonable maximum exposure risk

As shown in Fig. 4.8, reasonable maximum exposures for Pb cancer risk ranged from 8.002×10^{-5} – 18.6081×10^{-5} and 9.3356×10^{-5} – 21.171×10^{-5} for men and women, respectively, while those for As ranged from 0.3448×10^{-4} – 84.0451×10^{-4} and 0.4707×10^{-4} – 98.0526×10^{-4} men and women, respectively in the entire study area.

At 95% (CI), one sample t-test indicated that Pb RME risk for all the work-units were significantly above 1×10^{-6} (p = 0.000566; p = 0.0049) risk value while that for As was

also significantly higher (p = 0.010896; p = 0.010902) for men and women, respectively, in the entire study area. Aggregate risk from both Pb and As were significantly higher (p = 0.033651; p = 0.027562) than 1×10^{-6} risk value for men women, respectively as shown in Figure 4.8.

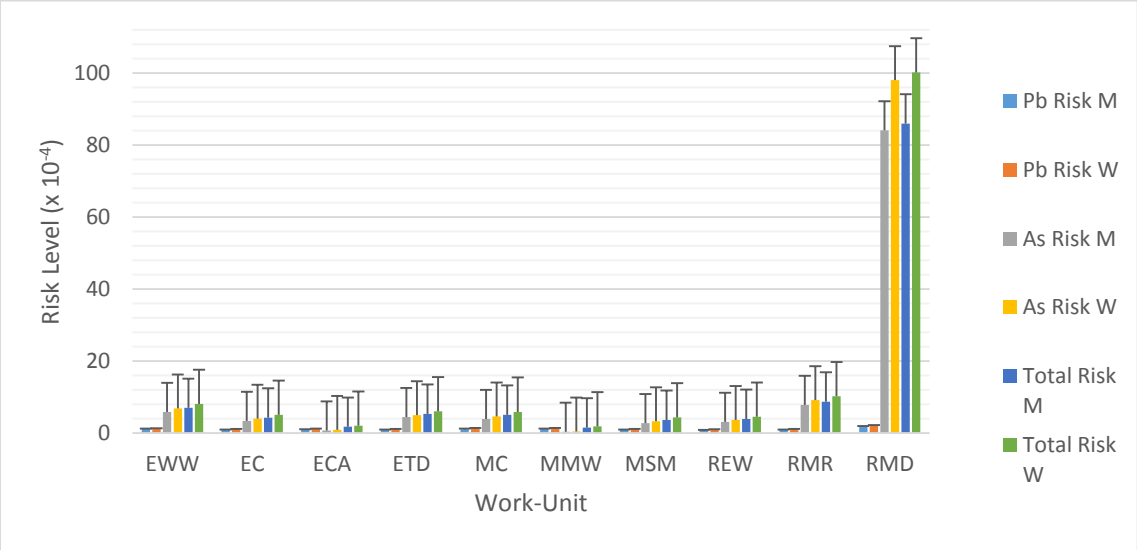

Figure 4.8: RME for Lead and Arsenic Carcinogenic Risk

Further, Pb RME risk for all the work-units in the entire study area were significantly below (p = 0.305079; p = 0.115597) ELCR 1×10^{-4} risk value while that for As was significantly above (p = 0.013018; p = 0.0128349) for men and women, respectively. Aggregate risk from both Pb and As was also significantly above (p = 0.0119696; p = 0.0118182) 1×10^{-4} risk value for men and women, respectively.

Further, at 95% (CI), two sample t-test indicated that there were no significant variations ($p > 0.05$) between Pb and As RME's risk nor between aggregate risk (p = 0.880937) for

men and women in the entire study area. Hence there was anticipated risk from RME carcinogenic exposure in the entire study area.

CHAPTER FIVE

DISCUSSION

5.1 Laboratories hygiene, safety practices and worker traits

5.1.1 Laboratories hygiene and safety practices

The study area did not conform to expected standards as per internationally accepted guidelines as well as Kenya's OSHA Act (2007). According to Occupational Safety and Health Administration (OSHA) Occupational Exposure to Hazardous Chemicals in Laboratories standard factsheet (29 CFR 1910.1450) also referred to as the Laboratory Standard, it is a requirement that employers, universities not withstanding strive to minimize risks in the research and teaching environment in which hazards and the associated risks are known. This can be by proper procedures, protocols and equipment with the intention of protecting the health and safety of students, employees and the public regarding the safe handling of chemicals and other hazards that are present in the work place (www.osha.gov/29 CFR 1910.1450 accessed on January 24, 2019).

In devoid of a program for regular or periodical monitoring of fumes, dust, gases or vapours, workers in the studied instructional facilities could be occupationally exposed to contaminants in the work-place. In particular, where the work processes generate air movement such as the machine workshops at RMD, ETD, EWW, MMW as well as MSM work-units, local exhaust ventilation should be installed close to the source of release and/or generation in order to remove the airborne contaminants. This will ensure they do not spread hence the worker's breathing zone airflow has sufficient airflow. The WHO (2010a) ascertains that, whenever people are occupationally exposed to dust, they are at risk of occupational dust related diseases. Further, in both developing and developed

countries, year after year, disease and permanent and temporary disabilities as well as deaths have been reported resulting from overexposure to contaminated dusts.

According to U.S. EPA (2003a) and Watts (2009), indirect occupational exposure may be experienced by family members who may be exposed to contaminants brought in the house by the worker as was the case in this study. Workers who are occupationally exposed to heavy metals may potentially expose their family members through bringing home contaminated shoes and clothing. Besides, unlike the case of this study, work clothing should therefore be tailored in such a way that they do not allow gathering of dust especially in their shoes and pockets. At all times, clothing suspected to be contaminated with toxic chemicals and should never find its way into workers homes. Besides, laundering of the same should be done safely in a designated area within the employers' facility under stringent conditions.

The use of proper personal protective equipment (PPE) as stipulated by European Agency for Safety and Health at Work (EASHW, 2010) encourages on mainstreaming occupational safety and health into university education to safeguard both staff and students. Unavailability, inconsistence and improper use of PPE's as found in this study has been linked with elevated occupational blood lead levels (Nwudu *et al*., 2018). PPE and especially RPE (respiratory personal equipment) are not only a necessity to further aid in mitigating heavy metal exposure but must always be kept clean by conscientiously cleaning them. This is in addition to properly maintaining them in order to remain effective in preventing exposure to workers. Though a costly affair, it should however be noted that poorly maintained PPE renders them ineffective.

Contrary to the findings of this study, universities in the world over have clear laboratory hygiene plans that state the policies, responsibilities and procedures that protect workers from the health hazards that maybe associated to the particular workplace. Examples include the University of Notre Dame (https://www.nd.edu accessed on February 11, 2019) and Indiana University (https://www.iu.edu/env accessed on February 15, 2019). Among other things, the plans require that an employer develops criteria that will be used to not only determine and implement but also put in place control measures aimed to reduce occupational exposure to hazardous chemicals. Such controls include engineering controls, use of personal protective equipment (PPE), and also appropriate hygiene practices.

The lack of awareness on occupational health risk management in the study area which was found to be generally weak besides many occupational health management regulations not being effectively implemented potentially leaves all personnel who work with hazardous chemicals exposed to the related risks. Further, the lack of exposure monitoring and medical surveillance as per requirements may too tend to exacerbate occupational exposure according to Rout and Sidkar (2017).

5.1.2 Worker - individual traits and behaviour

Personality traits as a determinant of human behaviour are particularly an important aspect in determining who may be at risk from inadvertent occupational exposure to contaminants. Since occupational mobility was generally absent in this study, these workers may thus be exposed to higher magnitudes of exposure as exemplified in a study on human habits on heavy metals human exposure by Lawal *et al*., (2015). The U.S. EPA

(2011) has further highlighted on exposure magnitudes as a function of time frame of exposure which is dependent on occupational mobility and tenure besides presenting recommended exposure factors for occupational mobility.

The habit of snacking without prior washing of hands as found in this study may too contribute to exposure risks by the inadvertent route as supported by Decharat *et al*., (2012) who observed that poor workers personal hygiene significantly correlated with blood lead levels though for a different environmental scenarios. The Technical Guidance Document on chemical risk assessment from the European Chemical Bureau (ECB, 2010) further asserts that exposure by the ingestion route is in most cases controlled by putting in place appropriate good hygiene practices which include the segregation of eating and working facilities and provision of adequate washing of hands with clean safe water prior to eating.

Besides cigarette smoke containing lead, not smoking can significantly lower a workers occupational exposure to lead. Studies by Hong *et al.,* (2014b); Ahmad *et al.,* (2014) have further highlighted the role of smoking and/or eating in potentially contaminated areas to inadvertent exposure via ingestion. Though smoking habit was generally absent with only three male smokers reported in all the facilities, hand washing is thus encouraged before this activity besides provision of a smoking zone.

The OSHA Laboratory Safety Chemical Hygiene Plan (www.osha.gov 1-800-321-6742 accessed on February 22, 2019) advocates that eating, smoking, drinking or any material is likely to exacerbate an ingestion hazard, in the workplace should be forbidden. In addition to ensuring there are adequate washing facilities in the workplace, the employer

should provide designated areas and restrict such activities in these areas. Whenever there is a possibility of occupational exposure to contaminated dust, adherence to personal care which includes proper washing of hands, brushing of teeth, cleaning of nails, washing of hair and showering and the habitual showering prior to eating and after the work are necessary measures.

Additionally, proper training of workers about the risks and hazards from the substances used is very vital. Further, periodical exposure monitoring and any other control measures by the employer are also necessary. Since the workers are often the people who are knowledgeable of their day-to-day happenings during work procedures, the employer should strive to seek their views on what may lead to exposure for effectiveness of control.

5.2 Heavy metal concentrations

5.2.1 Lead concentrations

Mean Pb concentrations in the sampling sites decreased in the order of RMD > EWW > MC > MMW > ECA > ETD > EC > RMR > MSM > REW (Fig. 4.1). There were significant variations attributable to relatively higher lead concentration in dust samples from RMD and MC sampling stations and REW that recorded the least mean Pb level. Besides the nature of activities in the individual work-units, variations in Pb occurrences could be attributed to housekeeping habits considering REW and RMR work-units though housed together with RMD work-unit recorded far less Pb levels (Appendix IV). The RMD work-unit which ranked first in terms of Pb levels was never cleaned in the entire study period while EWW which came second was cleaned only once.

Mean concentrations of Pb heavy metal were above the average earth crustal concentrations (14.8 mg/kg) in the continental crust in all the sampling stations as proposed by Wedepohl (1995). The elevated concentration of these metals may thus be attributed to anthropogenic activities as highlighted in Appendix III.

It was, however, difficult to compare the findings of this study with other studies since no study under similar environmental scenario was found for comparison in Kenya or the world over. Nevertheless, Pb analysis results for this study were found to be higher than for some studies on Pb in dust. Recent studies for instance by Ardashiri and Hashemi (2017); Hejami and Ahmed (2014) on Pb levels in school laboratories dust in Bushehr, Iran and Toronto, Canada, respectively, reported less Pb levels than the findings in this study. Additionally, studies by Adaramodu *et al*., 2012 and Latif *et al*., 2014 also reported less Pb levels.

However, other studies though done in different environmental scenario, have recorded much higher levels of Pb than found in this study. A study done on the vicinities of informal used lead-acid battery recycling operations in Nairobi, Kenya (Ondayo *et al*., 2016) for instance, recorded very high Pb levels. Further, results of a study in industrial settings (NIOSH, 2008; Huang *et al.,* 2017) reported higher Pb levels than the findings in this study. This therefore, implies that Pb levels may tend to increase especially in industrial settings as compared to indoor residential and instructional laboratory settings.

The EPA Office of Solid Waste (U.S. EPA, 2018) has put in place a detailed directive on risk assessment and cleanup of soil lead in residential areas. According to the directive, soils with lead levels less than 400 mg/kg have been recommended as generally safe for

residential use. For the purposes of soil lead screening therefore, 400 mg/kg for residential soils is the recommended maximum level. The findings of this study found Pb concentrations to be significantly above maximum allowable limits for Pb in soil in most individual countries guidelines the world over as shown in Table 2.1. Lead levels in the work-units were also significantly higher than internationally accepted standards such U.S. EPA, EU and WHO/FAO guidelines (Table 4.1). Chronic occupational exposure of high amounts Pb as found in this study may lead to increased accumulation in the body resulting in adverse health implications.

5.2.2 Arsenic concentrations

Mean As concentrations in the sampling sites decreased in the order of RMD > EWW > RMR > MC > REW > MSM > EC > ECA > MMW > ETD (Fig. 4.2). Significant variations were attributable to As in dust samples from RMD sampling station that was much higher than the rest. Arsenic concentrations were however significantly much lower than Pb concentrations for the same work-units. This exemplifies variations in occurrence of heavy metals in similar occupational environments. Mean concentrations of As were above the average earth crustal concentrations (1.7 mg/kg) in the continental crust in 80% of the sampling stations except at ETD and MMW according to Wedepohl (1995).

Besides house-keeping habits, elevated As at EWW sampling station could be attributed to the use of treated wood in the facility which is commonly used for practical lessons by the Wood Science and Technology students. As is a component of wood preservatives such as chromated copper arsenate (CCA) as reported in a study by Kwon *et al.*, (2004) on As contamination arising from use of CCA as a wood preservative. Further, Gribovich

(2012) reported higher mean As concentrations derived from CCA playgrounds as compared to those obtained for the non-CCA playgrounds with these results relatively lower compared to the findings for this study.

Similarly with Pb, since no guidelines or regulations for As heavy metal in settled indoor surface dust in Kenya were found, evaluation of the extent of As heavy metal contamination in the dust was therefore done by comparing the concentrations with recommended maximum allowable limits from other countries as shown in Table 2.1. Arsenic levels in RMD work-unit for instance surpassed set screening levels for As in soils (U.S. EPA, 2003b) and the maximum allowable limits for all the countries.

Further, no study under similar environmental scenario was found for comparison in Kenya or the world over. Therefore it was difficult to compare the findings with other studies. However, in some instances, As levels from the findings of this study were less when compared to results from other studies (Kamunda *et al.*, 2016; Liu *et al*., 2010; Kar *et al*., 2013; Huang *et al.,* 2017). These findings suggest the importance of universities instructional laboratories as environmental scenarios to occupational heavy metal exposure risks.

5.3 Estimation of non-carcinogenic occupational exposure

According to U.S. EPA (2018), an indoor worker as a receptor spends considerable amount if not all, of the working hours in the indoor environment. Thus, as is the case with laboratory staff in this study, an indoor worker may have no direct dermal contact with outdoor dust/soils thus any dermal exposure will in most cases emanate from indoor

dusts/soil dusts only. However, this worker will most likely be exposed to chemicals from contaminated dust/soils that have been incorporated into indoor dust/soil via the ingestion pathway. The U.S. EPA (2011) holds that extrapolated ADI levels for any potential contaminant of concern should not exceed the important pathway reference dose.

5.3.1 Occupational lead non-carcinogenic exposure assessment

Lead ADI values were compared with the pathway reference doses to ascertain non-carcinogenic risk. The findings of this study ascertained that Pb ADI were significantly above one (1), which is equivalent to the threshold value in 60% of the cases (EWW, ECA, ETD, MC, REW, RMD) for both men and women (Table 4.3). Besides, extrapolated ADI were also found to be higher for both men and women. Thus, non-carcinogenic occupational exposure to Pb in these work-units may occur.

On the other hand; EC, MMW, MSM and RMR work-units were found to be significantly lower. It was notable that all these work-units had mean Pb concentration levels below 400 mg/kg which also coincides with U.S. EPA's Guidelines (U.S. EPA, 2018) maximum allowable limit of heavy metals concentrations in soil. Due to elevated Pb levels as compared to the rest, the RMD work-unit was therefore the most exposed. Significant variations between the work-unit were attributed to ADI levels from RMD and MC work-units that were relatively higher than the rest.

Though difficult to compare the findings of this study since no earlier studies were found under the same environmental scenario, nevertheless, the ADI findings as per this study were higher than recent studies reported by Shabbaj *et al*., (2018) on Pb exposure to road

dust and also by Nkansah *et al*., (2017) in dust around fuel filling stations. Further, these findings were much lower when compared to a study done by Huang *et al*., (2017).

As per the findings in this particular study, ADI for dermal contact exposure with subsequent transdermal uptake was found to be the main route of occupational exposure to Pb for both men and women when compared to incidental ingestion and dermal contact with a contaminated surface followed by inadvertent ingestion of contaminants transferred to skin. These findings were in agreement with those by Kamunda *et al.*, (2016) on risk assessment study in soils from a gold mining basin and also by Ardashiri and Hashemi (2017) in residential indoor dust, who in their study also reported that the dermal pathway contributed the greatest to non-carcinogenic risk in adults. Further, these results were in agreement with studies by Cherrie *et al*., (2006); Dinman and Dinman (2000), who observed that in occupational settings involving chemicals, the dermal route in terms of potential toxicity was more significant as compared to the ingestion route.

Other studies have also found dermal exposure to other heavy metals to be the major exposure pathway to non-cancer effects for adults. Olujimi *et al*., (2015) in his study on heavy metals human health risk assessment at an illegal gold mining site, for instance, found dermal exposure to nickel as the major exposure route. Thus, it is possible for dermal exposure to reach a level of significance. However, other studies though done in different environmental scenarios have reported the ingestion pathway as the main route of exposure as obtained by Zheng *et al*., (2013) on street dust of heavily industrialized city.

The study found that the entire study area was exposed to Pb non-carcinogenic risk in both men and women. Lead is a neurotoxin. It affects the central nervous system (CNS) and causes behavioural disorders. Overt signs of intoxication include irritability, dullness, poor attention span, muscle tremor, headaches, hallucinations, and loss of memory. Chronic and ongoing exposure to lead may also lead to severe damage of the reproductive system, damage to the kidneys and brain, anaemia as well as increased blood pressure (CDCP, 2016; Armah *et al*., 2012).

5.3.2 Occupational arsenic non-carcinogenic exposure assessment

The study found that, the workers at EWW, RMR and RMD were theoretically exposed to As in both men and women. Variations in arsenic non-carcinogenic exposure was attributed to elevated ADI levels from RMD work-unit that had relatively higher ADI than the rest (Table 4.4). Thus 70% of the work-units were not occupationally theoretically exposed to As non-carcinogenic risks for both men and women. Nevertheless, chronic exposure to heavy metal as is the case in this particular study might lead to As build up in the body, instigating non-cancer effects. Similarly to lead, it was difficult to compare the findings of this study since no earlier studies were found under the same environmental scenario.

The characteristic difference in the body weight parameter of the exposure pathways for the two target groups (men and women) controlled the differences in the resultant exposures; indeed female calculated magnitude of exposure was higher than male exposure for all the work-units. When compared to Pb, as a result of the high recorded mean Pb concentration levels as compared to As, total ADI's for Pb thus surpassed that

of As by over 100% in all sampling work-units. This therefore, implied that exposure to Pb contributed a high burden of non-carcinogenic exposure.

As stated by Tchounwou *et al*., (2012), human exposure to high levels of As concentration as observed in RMD and EWW work-units can induce cardiovascular diseases, skin alterations, diabetes, hematologic disorders, hearing loss, neurologic and neurobehavioural disorders.

5.4 Occupational non-carcinogenic risk characterization

Extrapolated hazard quotients (HQ) and hazard index (HI) risks were used to ascertain non-carcinogenic risks. The HI value shows the sum of the value of the HQ for different substance through different pathways. The U.S. EPA (2011) further holds that if the HQ or HI for a chemical is equal to or less than one, there is no appreciable risk that non-cancer health effects will occur. If the HQ or HI exceeds one, there is some possibility that non-cancer effects may occur.

5.4.1 Occupational lead non-carcinogenic risk characterization

As per the results of this study, for women, all (100%) the work-units were found to be theoretically occupationally exposed to Pb non-carcinogenic risks, while for men, 30% were found to be potentially safe (Table 4.5). This variation was attributed to their differences in their weight with women having a lower weight. It should however be noted that not all adult men and women bear the assumed weights of 70 kg and 60 kg, respectively, and therefore risk of exposure may as well be an individual case based on this attribute.

When considering the entire study area, extrapolated HI levels were above 1 in all the work-units for both men and women indicating that they were all exposed to Pb non-cancer risks. Though not done in the same environmental scenarios, these HI findings were much lower when compared to a study done by Huang *et al*., (2017) in a lead-zinc mining area in China which reported a HI of 3.97×10^{-1} for Pb. Moreover, higher non-carcinogenic risk (HI= 2.4) was also found in Pb involving an earlier study performed by Al-Rajhi *et al*., (2006) but comparable to those by Hassan (2012), both in residential buildings. In the latter study in urban areas, lead HI values in the entryway of homes and the living room were 1.30 and 1.20, respectively. However, the aggregate HI for Pb risk for this study was higher than those reported by Sun (2017) HI= 0.436 and Zheng *et al*., (2010) HI= 0.144 both in street dust, Kong *et al*., (2011) HI=0.0021 in re-suspended dusts on building surfaces and Nkansah *et al*., (2017) HI= 0.023 in dusts around filling stations.

There is significant evidence that exposure to Pb has been attributed to many of the body systems. Once in the body, Pb is taken up and is known to be stored in bones whereby it affects calcium absorption and can disrupt skeletal development (Gildow, 2015). Further, during pregnancy and breast feeding, lead accumulated in the bones may be released and can pass the placental barrier from the mother to her vulnerable, developing foetus damaging the haematologic system hence causing reduction in children's intelligence quotient (IQ) thus affecting academic performance, cause memory loss and decrease sight and hearing ability of children and induce attention deficit disorders (Sanborn *et al.*, 2012). The workers in these facilities may thus be at risk these health implications.

5.4.2 Occupational arsenic non-carcinogenic risk characterization

Arsenic hazard quotients for this study were significantly above 1 in 40% (Table 4.6) of the work units (EWW, MMW, MC and RMR). Contrary to lead, arsenic HQ results for both men and women in the entire study area were found to be less than unit. The interpretation of the risk assessment extrapolated from calculated values for the considered pathways in this study therefore revealed that there was no significant evidence of anticipated occupational adverse health impacts according to the benchmarks as established by U.S. EPA (2011).

However, these values were higher than those reported by Han (2017) in dusts from parks and squares in China but lower than those reported by Rout and Sidkar (2017) in an iron ore pelletizing industry, both of which are different environmental scenarios than that studied in the present study.

Human chronic oral exposure to elevated levels of inorganic arsenic has been reported (WHO, 2012) to result in peripheral neuropathy, gastrointestinal effects, skin lesions, anaemia, hyperpigmentation, vascular lesions, gangrene of the extremities, and damages the kidney or liver.

5.4.3 Cumulative occupational non-carcinogenic risk characterization

While assuming an additive effect, total non-carcinogenic risk (HI) arising from both As and Pb when considering risks from all the considered pathways for all work units for both Pb and As, were all significantly above unit for this study because of the elevated HI's for Pb (Fig. 4.3). It is therefore likely that all work-units in all the sampling sites

were theoretically at risk to non-carcinogenic risks arising from cumulative Pb and As exposure.

Due to the high elevated levels for both As and Pb in RMD work-unit, this work unit was found to be the most potentially exposed for both As and Pb non-carcinogenic risk with a HI of 14.5814 while ETD work unit had the least with a HI of 1.2356. The findings of this study therefore deduce that workers occupational predisposition may expose them to As and Pb non-carcinogenic induced health effects.

5.5 Occupational carcinogenic risk characterization

Hazard risk assessment was subsequently done for both men and women using calculated life-time average daily intake (LADI) to ascertain the target indoor dust contaminant concentration that would represent a theoretical excess cancer risk of one-in-a million (1×10^{-6}) and one-in-a ten thousand (1×10^{-4}) for an individual chemical assuming no exposure from other sources. The cancer risk assessment for selected heavy metals was estimated using ingestion mode of exposure. Exposure to contaminant concentrations greater than the excess lifetime cancer risk implies that further investigation needs to be done on the contaminant of concern and thus is not indicative of people developing any health related problems.

The extrapolated results for carcinogenic (LADI) assumed the individual workers would be engaged in similar duties for 30 years. This exposure duration (employment period), however, affects the carcinogenic risk in that cancer risk would decrease with subsequent decrease in exposure duration. Length of exposure duration, however, has no effect on the non-carcinogenic hazard index (U.S. EPA. 2012).

5.5.1 Lead carcinogenic risk characterization

The RMD work-unit posed very high unacceptable risks of 1.69026E-04 (1 in every 5,916 individuals) and 1.97196E-04 (or 1 in every 5,071 individuals) for men and women, respectively. Lead theoretical carcinogenic risks were thus found to be above the excess lifetime cancer risk (1×10^{-6} - 1×10^{-4}) in 50% of the work-units (EWW, EC, MC, RMD, MSM). Hence, half of work-units could be considered to be safe from Pb related carcinogenic effects in their lifetime (Table 4.7).

When the entire area was extrapolated for Pb carcinogenic risks, men were found to be within the acceptable (1×10^{-6} - 1×10^{-4}) range and could therefore be generalized to be safe. However, women risks were found to be above the acceptable range and therefore could be generalized as unsafe. These cancer risk results were further found to be more significant than those of a study by Kamunda *et al.*, (2016) which reported Pb cancer risk for adults to be 3.51E-06 (1 in 294,800 individuals).

The (ATSDR, 2019) has determined and listed lead as a probable cancer-causing agent, or carcinogen when exposed to humans. Occupational exposures to lead have been linked to cancers, such as cancers of the colon, brain, bladder, rectum and the kidney. Several studies in lab animals have also reported that oral (by swallowing or other means) exposure to compounds of lead has been attributed to cause cancer. In particular, kidney tumors have been linked with lead exposure while different studies have also linked lead with other tumors such as lung, brain and some other organs.

5.5.2 Arsenic carcinogenic risk characterization

Arsenic theoretical carcinogenic risks in this study were found to be above ELCR level (1×10^{-6} - 1×10^{-4}) in 50% of the work-units (EWW, MC, REW, RMR, RMD) for both men and women. Notably was the RMD work-unit which posed very high As carcinogenic risk at or 1.39584E-03 (1 in every 716 individuals) and 1.62847E-03 (1 in every 614 individuals) for men and women, respectively, which is regarded as "moderate" increased risk hence unacceptable risk.

Moreover, As carcinogenic risk in the entire study area were found to be within the acceptable levels (1×10^{-6} - 1×10^{-4}), thus no carcinogenic effects were anticipated from long term exposure. However, this could be attributed to the extremely low risks indicated in some of the work-units. Arsenic carcinogenic risk characterization at ETD for instance was considered "extremely low" increased risk at 0.813E-06 (one in every 1,230,012 individuals) in men.

Chronic exposure to As has been linked with carcinoma, cancer of the skin, cancers in liver, lungs, kidney, urinary bladder and of the colon (Baker *et al*., 2018; Armah *et al*., 2012).

5.5.3 Cumulative occupational carcinogenic risk characterization

For cancer probabilities across the exposure scenarios, the risks ranged from "extremely low" (below 1 in a million chance) to "moderate" (above 1 in a thousand chance) increased risk. Total carcinogenic risk arising from both As and Pb exposure indicated that though total exposure point concentrations for Pb in all the samples were much higher than those for As for the same work-units, As contributed to much higher

occupational carcinogenic total risk as compared to Pb in 60% (EWW, MC, MSM, REW, RMR and RMD) of the work-units. In all the cases, RMD work-unit had posed unacceptable "low" increased risk from occupational exposure to both As and Pb heavy metals at 1 in every 5,563 individuals and 1 in every 5,477 individuals for men and women, respectively. This should be of great concern to the workers in these facilities.

Aggregate carcinogenic risk arising from both As and Pb when considering risks from all the considered pathways for all work units, were above U.S. EPA's target risk for cancer of 1×10^{-6} - 1×10^{-4}. From these findings, it can therefore be concluded that all work-units were theoretically at risk to carcinogenic aggregate risks arising from Pb and As exposure.

The theoretical cancer risk findings were higher than those reported by Ferguson *et al*., (2018) for all exposure scenarios where the risks ranged from "extremely low" (near 1 in a million chance) to "low" (near 1 in ten thousand) increased risk.

For this study, the dermal contact with a contaminated surface and subsequent transdermal uptake pathway seemed to be the major contributor to both non-cancer risks as well as excess lifetime cancer risk. Although the ingestion route has been pitted as the most important exposure pathway with the dermal exposure route generally considered a minor exposure pathway, the results of this study were in agreement with a screening study by Johnson and Kissel (1996). With arsenic as one of the primary soil contaminants, the study reported 37 sites out of 200 risk assessments for superfund sites had dermal contact with contaminated soil projected excess lifetime cancer risks (ELCR) greater than one individual out ten thousand individuals (ELCR > 1×10^{-4}). Moreover,

the study found dermal exposure to be the most dominant exposure route at 9 sites for the considered metals.

5.6 Work units CTE and RME to non-carcinogenic risks

5.6.1 Non-carcinogenic central tendency exposure risk

As per the results of this study, all women staff in all (100%) work-units were found to be potentially at risk to CTE Pb non-carcinogenic risks while male staff were found to be safe in only REW (HI>1) work-unit. This therefore implies that all workers exposed to Pb levels above stipulated soil screening concentrations (400 mg/kg) could be said to be theoretically at risk to Pb non-carcinogenic effects. Lead CTE risk in the entire study area was significantly above unit in all the work-units for both men and women implying they were exposed to Pb CTE non-carcinogenic risks.

When CTE exposure risks were considered for As non-cancer risks, 50% of the work-units (EC, ECA ETD, MMW, MSM and REW) for male staff were found to be safe. In addition to REW, similar work-units were also found to be exposed to As non-cancer risks. Contrary to lead, for the arsenic CTE HQ's were significantly below unit in the entire study area for both men and women, thus As non-carcinogenic effects may not be anticipated.

5.6.2 Non-carcinogenic reasonable maximum exposure risk

Similarly to CTE, all women staffs in all work-units were found to be potentially at risk to Pb RME non-cancer risks while male staffs were found to be safe in only REW work-unit. On the contrary when considering RME for As non-cancer risks for all pathways, fewer (40%) work-units were considered to be safe for both male (ECA, ETD, MMW,

RMR, MSM) and female staff (50%). This therefore exemplifies that employees exposed to high end exposure point concentrations (RME) are more at risk than those exposed to mean (CTE) concentrations.

Moreover, the findings of this study indicate that all the work-units in the entire study area were theoretically at risk to Pb but were not theoretically at risk to As RME non-carcinogenic risks for men and women in the entire study area. However, aggregate risk from both Pb and As were significantly above unit. Thus, as per the findings of this study, all the work-units were theoretically at risk to Pb and As RME non-cancer risks.

Based on RME's results, all work-units were exposed to Pb non-cancer risks. RMD work-unit as per this study was found to be the most exposed to both Pb and As non-cancer risks. The CTE and RME findings for both Pb and As were all below those reported by Armah and Obiri (2016) for the same heavy metals in Ghana and for arsenic by the US Dept. of Health and Human Services (2014) though for different environmental scenarios.

5.7 Work units CTE and RME to cancer risks

5.7.1 Carcinogenic central tendency exposure risks

Lead CTE risks were above U.S. EPA's excess lifetime cancer risk (ELCR) level of 1×10^{-6} - 1×10^{-4} in 30% of the work-units (EWW, MC, RMD). The highest Pb CTE risk was recorded at RMD work-unit (1 in 5,916 and 1 in 5,071 individuals for male and female staff, respectively, was regarded as "low" increased risk hence unacceptable risk since it was above U.S. EPA's excess cancer risk of 1×10^{-6} - 1×10^{-4}. Nevertheless, lead CTE cancer risk for the considered pathways in the entire study area was found to be

within the U.S. EPA's ELCR level. Thus, Pb CTE cancer risks were not anticipated. These results were lower than those reported by Armah *et al*., (2012) and Armah and Obiri (2016) in Ghana though for a different environmental scenario.

Though REW work-unit as per this study had the least arsenic CTE cancer risk (1 in 12,941 individuals and 1 in 11,129 individuals for male and female staff, respectively), this risk was slightly below U.S. EPA's excess lifetime cancer risk of 1×10^{-6} - 1×10^{-4}. Moreover, it is interpreted as "low" increased risk hence unacceptable. Arsenic CTE cancer risks were thus above excess cancer risk in all the work-units. Further, arsenic CTE risk was significantly considered to be above excess cancer risk for men and women in the entire study area. Aggregate CTE for carcinogenic risks were thus significantly above acceptable excess cancer risk for men and women.

The findings of the study were, however, way below those for As cancer health risk for Tamso (approximately 10 out of 100 individuals) and Prestea (approximately 1 out of 100 individuals) areas in Ghana as reported by Obiri *et al*., (2006), but higher than those reported by US Dept. of Health and Human Services (2014) though for different environmental scenarios. Moreover, these cancer risk CTE results for both Pb and As were all below those reported by Ted (2014) for construction and excavation workers.

5.7.2 Carcinogenic reasonable maximum exposure risk

Lead RME cancer risks were found to be above excess lifetime cancer risk of 1×10^{-6} - 1×10^{-4} for men in 40% (EWW, MC, MMW, RMD) of the work-units. Additionally, EC, ECA and ETD work-units were also found to be above excess cancer risk for women.

Nevertheless, lead RME risk for the entire study area was significantly found to be within U.S. EPA's excess cancer risk of 1×10^{-6} - 1×10^{-4} for both men and women.

Arsenic RME results indicated only ETD, MMW and RMR work-units to be theoretically safe from cancer risks, though this could be quite conservative owing to the limited number of samples for this study. More so, arsenic RME risk for the entire study area was significantly found to be above U.S. EPA's excess cancer risk of 1×10^{-6} - 1×10^{-4} for both men and women. Thus, As cancer effects may occur as per the results of this study.

Aggregate risk from both Pb and As was also significantly above the acceptable ELCR range (1×10^{-6} - 1×10^{-4}) for both men and women. Thus, as per the findings of this study, the entire study area was theoretically at risk to Pb and As RME cancer risks.

It was rather difficult to compare these findings with past studies since none was found under the same environmental scenario. Nevertheless, RME results for both Pb and As in this study were all below those reported by Ted (2014) though for a different environmental scenario.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

6.1.1 Laboratories hygiene and safety practices

The study concluded that the hygiene and safety practices did not conform to requirements as stipulated in OSHA Acts in Kenya and other countries as well as the WHO. Lack of proper cleaning as well as programs for periodical monitoring of fumes, dust, gases or vapours and clear delineation of areas with toxic solutions and other wastes in their safe handling, storage, management of spills and disposal could as well exacerbate Pb and As occupational exposure. Thus, workers were not entirely safe from risks that may arise from workplace hygiene and safety practices. Various mitigation procedures, which include but not limited to substitution in the use of materials that may be containing heavy metals, daily vacuuming of indoor settled dust and other administrative control measures were lacking, implementation of the same could minimize the occupational exposure of heavy metals.

The staffs were found to be snacking and smoking in their work areas without prior washing of hands, besides, designated eating areas were not provided in all the work-units. This exemplifies the lack of personal hygiene and adherence to stipulated laboratory safety standards at all times. Moreover, the universities management were found to be negligible in the provision, training, maintenance and monitoring of all the required PPE's. Workers clothing were taken home and laundered at the workers homes instead of safely at the workplace. The collection of toxic dusts/soils in the workplace

was uncontrolled and inappropriately disposed and this could lead to exposure of the concerned workers.

Access to information, instruction and training on risks to health arising from exposure to possible contaminants with heavy metals and warning signs though necessary was lacking. Workers should therefore strive to have knowledge of SDS or other appropriate references on hazards. Health surveillance and latest summary of occupational injuries and illness form should be availed in all the facilities.

Occupational exposure of indoor dusts should be of particular interest since they are have been associated with classical widespread of occupational, particularly lung diseases majorly pneumoconiosis. High levels of lead exposure has been associated with systemic intoxications More so, other dust-related illnesses attributed to much lower exposure levels include; asthma, cancer, irritation and allergic alveolitis as well as a whole range of non-respiratory illnesses.

6.1.2 Heavy metal concentrations

Mean concentrations of Pb decreased in the order of RMD > EWW > MC > MMW > ECA > ETD > EC > RMR> MSM > REW. Mean As concentrations also varied significantly and decreased in the order of RMD > EWW > RMR > MC > REW > MSM > EC > ECA> MMW > ETD. The RMD work-unit therefore recorded the highest Pb and As levels. The study established that there were significant variations between Pb and As heavy metals concentrations in the studied instructional laboratories. Mean Pb concentration in the entire study were majorly above the EU, FAO/WHO and U.S. EPA standards. The present study though no similar ones to compare with, has therefore

provided baseline data for metal concentrations in universities instructional laboratory indoor settled dusts. This study further exemplifies that indoor settled dusts can be used as an indicator for heavy metal pollution human exposure in indoor occupational settings.

6.1.3 Exposure, risk assessment and work-units CTE and RME

Lead HI for men and women in the entire study area were found to be above unit while those for arsenic were found to be lower implying that work-units maybe potentially exposed to non-carcinogenic Pb effects but may be safe from As cancer effects. These results were in agreement with CTE and RME non-carcinogenic risks. However, aggregate HI was found to be above unit implying that there was potential non-carcinogenic exposure arising from the studied elements.

Lead cancer risk in men was significantly within acceptable ELCR (1×10^{-6} - 1×10^{-4}) suggested by U.S. EPA. However, women risk was significantly higher. Further, these results were in agreement with CTE carcinogenic risks but contrasted with RME findings in that risk for both men and women were above acceptable levels. Thus, aggregate Pb and As carcinogenic risk for men and women in the entire study area were above ELCR levels. Arsenic cancer risk was within acceptable ELCR levels; cumulative risk was found to be above acceptable levels. However, As RME cancer risks were above acceptable levels for both men and women. The RMD work-unit was found to be the most exposed work-unit for both Pb and As cancer and non-cancer risks.

Based on the findings of this study, it can therefore be concluded that “average” and “high end” range of exposure of the work-units was significantly high for those employees exposed to high end exposure point concentrations (RME) than those with

central tendency exposure (CTE) for all the studied work-units. Thus, workers could be exposed differently depending on the range of exposure.

The present study deduced that there is potential cancer and non-cancer risk from Pb and As heavy metals in settled indoor dusts via the different studied exposure routes. Since the studied work-units exhibited low occupational mobility coupled with high occupational tenure, there is a possibility that these metals can accumulate and persist in their body tissues leading to deleterious health effects. Moreover, occupational exposure through indoor settled dust via the studied exposure routes is only one of the major human exposure pathways in which workers can be exposed to heavy metals contaminants, other routes of exposure (such as through diet) also exacerbate exposure. In devoid of comparable studies under similar environmental scenarios, the findings may therefore be utilized as a pilot study or a baseline survey to monitor and evaluate workers health in the studied institutions.

The study findings could however, be conservative in that, though the risk assessment used 50 mg of dust per day as the intake rate for indoor workers as recommended by U.S. EPA, in some work-units (EWW, RMD, MSM and MMW) which were characterized by too much dust in their operations, the applied intake rate could as well be lower hence reducing the extrapolated risk. Variations in men and women exposure risks for both non-carcinogenic and carcinogenic risks could be attributed to their differences in weight, with women having a lower weight. It should however be noted that not all men and women bear the assumed weights of 70 kg and 60 kg, respectively, and therefore risk of exposure may as well be an individual case based on this attribute.

6.2 Recommendations

1. Instructional laboratory workers should adhere to safety rules at all times
2. Universities to come up with chemical hygiene plans specific to the instructional laboratories
3. The possibility of characterizing the dusts in order to explore comprehensively potential occupational non-carcinogenic and carcinogenic exposure risks
4. Proper process-specific risk assessments should be conducted and reviewed periodically

6.3 Areas for further research

Based on the findings of this study therefore the following areas are recommended for further research;

1. Appropriate biomarkers such as urine be used to ascertain the magnitude of aggregate exposure
2. Modeling and/or the development of toxicological or epidemiological data necessary to increase certainty

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APPENDICES

Appendix I: XRF analyzer

(Source: Author, 2019)

Process of Mounting S1 Titan XRF for Sample Analysis: **a**. The portable S1 Titan XRF equipment; **b**. The XRF stand; **c**. Sample cup; **d**. Sample placed the XRF stage; **e**. Stage covered and system ready for sample analysis

Appendix II: Exposure Parameters used for Health Risk Assessment

Parameter	**Unit**	**Worker**	**References**
EPCs-Exposure point concentration	mg/kg		Present study
BW - Body weight	Kg	M-70; W- 60	U.S. EPA, 2011
EF – Exposure frequency	days/yr	250	U.S. EPA,2003a; 2011
ED – Exposure duration	yrs	30	U.S. EPA, 2011
CF – Contact frequency events/day	none	8	Michaud *et al*., 1994; Paull, 1997
CR = Contact rate (occupational dust ingestion)	mg/day	50	U.S. EPA, 1997; 2011
SAd – Skin surface area (dermal)	cm^2	3300	U.S.EPA, 1997; 2004
SAi – Skin surface area, ingestion	cm^2	790	U.S.EPA, 1997; 2003a
ABSder - Dermal absorption fraction	none	0.1	U.S.EPA, 2004; 2011
fdo – dermal-oral fraction transfer	none	0.04	Michaud *et al*., 1994; U.S. EPA, 2004; 2011
fgi – fraction GI absorption	none	1	U.S. EPA, 1997; 2011
AF – Adherence factor	mg/cm^2	0.2	U.S. EPA, 2011
AT – Averaging time for non-cancer	days	365×ED	U.S. EPA, 2011
AT_c – Averaging time for cancer	days	25,550 (365×70)	U.S. EPA, 2011
UCF - Unit conversion factor	kg/mg	10^{-6}	U.S. EPA, 2011

Appendix III: Probable Sources of Pb and As

Facility	Main activity	Probable Pb source	Probable As source
EC	Teaching and research	Pure lead handling and stock spillages, waste lead and lead compounds, glassware	Analysis of pesticides, herbicides and insecticides
EWW	Making select furniture for sale, woodwork teaching and research	Wood glue and lead adhesive tapes, sealants, wood stains, sanding, primers, paints, old wall paint, greasing	Wood preservation and treatment (CCA), wood glue, wood dust and chips
ECA	Teaching and research	lead testing practicals and research, AAS equipment, spillages, ceramic floors	Analysis of pesticides, herbicides and insecticides
ETD	Metal cutting, welding, folding, soldering, shaping	welding metal dust/fumes, fabrication, soldering, greasing, painted scrap metal handling, sanding, wall paint	Welding fumes, bearings, electrotype metal, soldering
MC	Teaching and research	lead spiking in research and spillages, glassware, paint	Analysis of pesticides, herbicides and insecticides
MMW	Cutting and welding of metals	Welding fumes, soldering, handling scrap painted metal, greasing	Welding and soldering fumes, electrotype metal cutting
MSM	Cutting, folding and fabrication of sheet metals, teaching and research	Welding fumes, soldering, handling scrap painted metal, greasing	electrotype metal cutting
REW	Assembling and repair of electronic parts from the factory, teaching and research	Soldering, repair of electronic devices, antifriction parts, electronic cables/wires	Electric semiconductors/devices e,g. transistors, capacitors and resistors, circuit boards
RMR	Dismantling, repair and winding of electric motors from the factory,	Blowlamp burning, insulation materials, electric wires,	Electric motors and other semi conductors/devices

	teaching and research	soldering	
RMD	Mechanical/auto parts assembling, repair, cutting and shaping metals, teaching and research	Textile dyes/pigments, greasing Worn out steel machinery, auto parts repair, soldering, welding fumes	Welding fumes, bearings, electrotype metal, soldering, semiconductor applications

Appendix IV: Interview Guide

A. General Facility Information

A1.Lab/workshop reference number _____ Date_____

A2. Job titles _____

A3. Sex_____

A4. Level of training_____

A5. Yrs of service _____

A6. Nature of duties

_____

_____

Number of shift hours per day/ week_____

Main activity in the lab_____

Lab area where most time is spent _____

A7. General lab ventilation; local exhaust _____

Extract (fume hoods) etc_____

A8. Frequency of cleaning per week_____

Cleaning method: Vacuumed _____ Wet/dry sweeping_____

Others

(Describe)_____

_____

_____

B. Laboratory/workshop Hygiene and Safety Practices

B1.Program under which facility regularly or periodically monitor for the presence of fumes, gases, mists, dusts or vapors?_____

_____

B2. Safe handling of As and Pb containing solutions (if any)_____

_____

_____

B3. Storage of As and Pb containing solutions (if any)

_____

_____

B4. Handling of spills containing toxics

_____

_____

B5. Disposal of fluids containing As and Pb metals (if any)

_____

_____

_____

B6. Electronic inventory of SDS or other appropriate references on hazards

_____

_____

B7. Demarcation of potentially contaminated areas and display of warning signs

_____

_____

B8. Is there a designated lunch room/area? _____

B9. Eating and drinking habits in the laboratory area _____

_____

_____

_____

B10. Washing and clothe wear changing habits_____

_____

B11. Are there areas in this facility in which personal protection devices or equipment are required or recommended?_____

B12. Availability and proper use of PPE:

(i)Respirators;_____

(ii)Gloves;_____

(iii)Overalls; _____

(iv)Boots_____

Others;_____

B13. Is there a visible presence of dust within the facility? _____

B14. Proper storage of floor wastes/materials _____

_____

B15. Information, instruction and training on risks to health arising from exposure to heavy

metals_____

_____

_____

B16. Health surveillance e.g. periodic health assessment or biological monitoring etc.

_____

_____

B17. Latest summary of Occupational Injuries and Illness

Form_____

_____

C. Worker - Individual traits and behavior

C1. Occupational mobility

_____

C2. Occupational tenure in yrs _____

C3. Facial hair: (Yes; No): (Long: Short)

C4. Smoker: (Yes; No)

C5. Washes hands before smoking: (Yes; No)

C6. Goes home with any item of clothes worn on site? (Yes; No)

State which items_____

C8. Other comments about the worker

_____

_____

Miscellaneous observations _____

_____

_____

General comments about the facility _____

_____

_____

_____

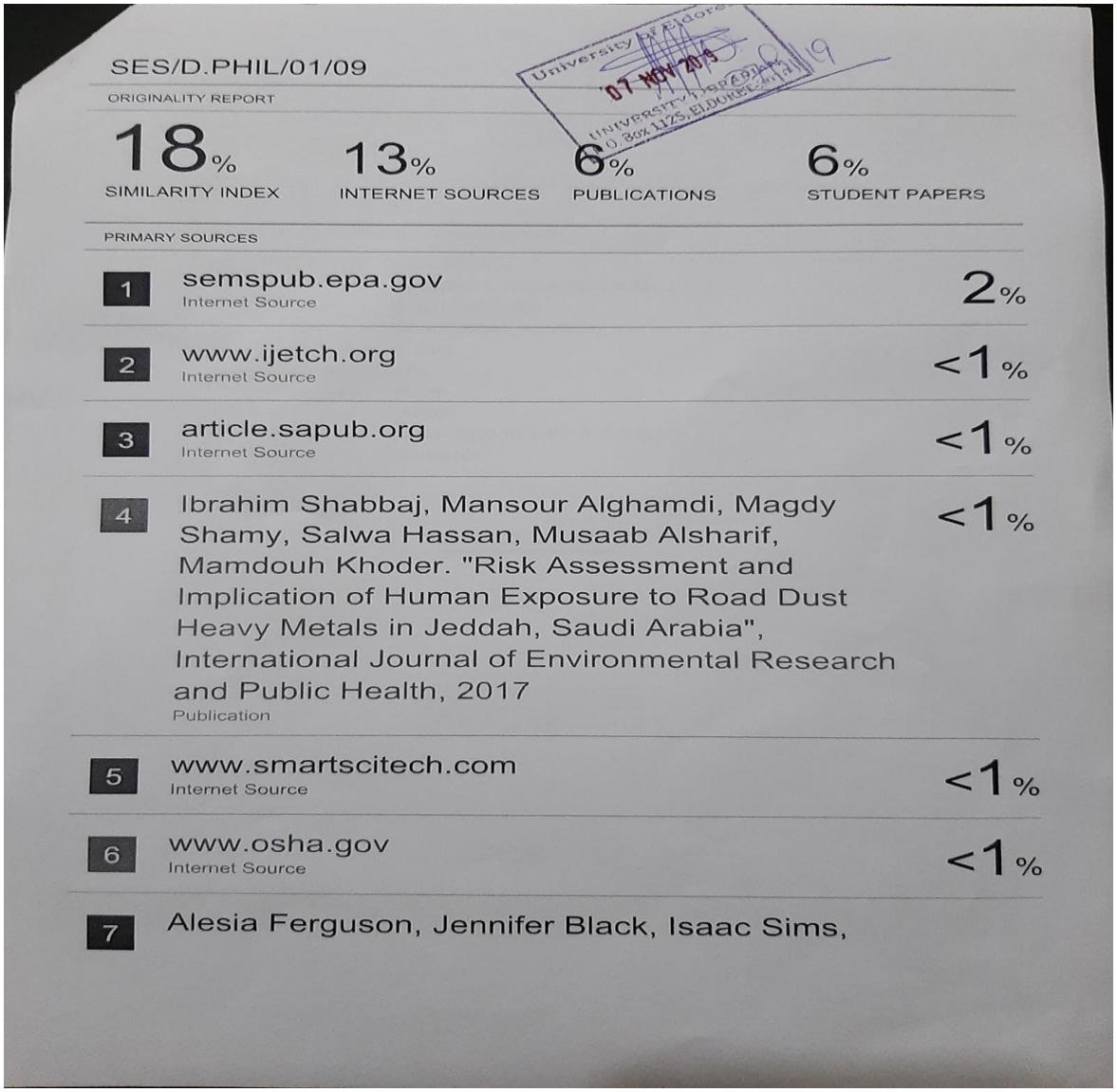
Appendix V: Results on Laboratories Hygiene and Safety Practices

Facility information	**EC**	**EWW**	**ECA**	**ETD**	**MC**
Dust presence	Yes	Yes	Yes	Yes	Yes
ventilation	Fume hood - inadequate	N/A	Fume hood	N/A	Fume hood
Housekeeping/ Frequency	Dry sweeping Weekly	Dry sweeping Rarely	Dry/wet Weekly	Dry Haphazardly	Dry/ rarely wet Weekly
Solid waste disposal bin/location	Dustbin	dumped on the interior periphery	dustbin	dumped on the interior periphery	Dustbin
Waste pick schedule	N/A	N/A	N/A	N/A	N/A
Chemical spill kit	N/A	N/A	N/A	N/A	N/A
PPE Goggles and Face Shield	N/A	Old inadequate goggles	N/A	Old goggles	N/A
Booties	N/A	N/A	N/A	N/A	N/A
Coveralls/ Lab coats	Lab coats	Lab coats	Lab coats	Lab coats	Lab coats
Hand gloves	Yes	N/A	Yes	N/A	Yes
Periodical training	N/A	N/A	N/A	N/A	N/A
Risk assessment tool	N/A	N/A	N/A	N/A	N/A
Eating/drinking	Infrequent	N/A	N/A	N/A	Frequent
Electronic SDS inventory	N/A	N/A	N/A	N/A	N/A
Hazard warnings	N/A	N/A	N/A	N/A	N/A
Medical surveillance	N/A	N/A	N/A	N/A	N/A
OIIF summary	N/A	N/A	N/A	N/A	N/A

Appendix V: Cont.

Facility information	**MMW**	**MSM**	**REW**	**RMR**	**RMD**
Dust presence	Yes	Yes	Yes	Yes	Yes
ventilation	N/A	N/A	N/A	N/A	N/A
Housekeeping Frequency	Dry sweeping haphazardly	Dry sweeping Haphazardly	Dry sweeping daily	Dry sweeping Haphazardly	Dry none
Solid waste disposal bin/location	Dustbin	dumped on the interior peripheries	dustbin	dumped on the interior peripheries	N/A
Waste pick schedule	N/A	N/A	N/A	N/A	N/A
Chemical spill kit	N/A	N/A	N/A	N/A	N/A
PPE Goggles and Face Shield	Old inadequate goggle	Old inadequate goggles	N/A	N/A	N/A
Booties	N/A	N/A	N/A	N/A	N/A
Coveralls/ Lab coats	Lab coats	Lab coats	Lab coats	Lab coats	Lab coats
Hand gloves	N/A	N/A	N/A	N/A	N/A
Periodical training	N/A	N/A	N/A	N/A	N/A
Risk assessment tool	N/A	N/A	N/A	N/A	N/A
Eating/drinking	Infrequent	Infrequent	N/A	N/A	N/A
Electronic SDS inventory	N/A	N/A	N/A	N/A	N/A
Hazard warnings	N/A	N/A	N/A	N/A	N/A
Medical surveillance	N/A	N/A	N/A	N/A	N/A
OIIF summary	N/A	N/A	N/A	N/A	N/A

Appendix VI: Similarity Index/Anti-Plagiarism Report



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