ASSESSMENT OF HEAVY METALS CONCENTRATION IN ASH FROM INCINERATORS AND ITS ENVIRONMENTAL IMPLICATION IN NAIROBI, KENYA

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A thesis submitted in partial fulfilment of requirements for the award of degree of Master of Science (MSc) in Pharmacology and Toxicology

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DECLARATION

This Thesis is my original work and has not been presented for a degree in any other University.

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DEDICATION

I dedicate this work to my late loving Dad Kipkoros, My dear Mum Margret and my loving Wife and daughter Tapkigen for their inspiration in all my endeavours.

ACKNOWLEDGEMENTS

I would like to express my sincere appreciation and gratitude to the respective individuals and organizations:

- The study supervisors, J. M. Mbaria and L.W. Kanja for their guidance during the research and their positive critique from the conceptualization, undertakings and final comments.

- Mr.C.F Mbakaya for the hours spent discussing pertinent aspects of the project and for his contributions in improving the study design.

- Dr. M.O Nanyingi for his extraordinary scientific insight and discussions concerning the logistics and analytical aspects of the research and for spending enormous hours in reviewing the final write up.

- The entire technical and administrative staff of the Department of Public Health, Pharmacology and Toxicology, University of Nairobi.

- The Director, Kenya Medical Research Institute and the Director, Center for Public Health Research (CPHR, KEMRI) for granting me the permission to undertake this research.

- My wife Obot Tapkigen and daughter Tapkigen for their love and support during my entire study period.

- The study participants who voluntarily and generously provided vital socio-demographic parameters.

- The Department of Geology and Mines for facilitation of samples analysis, National Environmental Management Programme for permission to undertake the research.

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LIST OF ABBREVIATIONS

ATSDR	Agency for Toxic Substances and Disease Registry
ALA	δ-aminolevulinic acid
ALAD	Aminolevulinate delta-dehydratase
BCF	Bioconcentration factor
BSC	Board of Scientific Counselors
CEL	Cancer Effect Level
cm	Centimeter
CNS	Central Nervous System
DOL	Department of Labor
EDTA	Ethylenediaminetetraacetic acid
EPA	Environmental Protection Agency
GC	Gas Chromatography
ILO	International Labor Organization
Kd	Adsorption Ratio
LC50	Lethal Concentration, 50% Kill
LD50	Lethal Dose, 50% Kill
LOAEL	Lowest-Observed-Adverse-Effect Level
LSE	Levels of Significant Exposure
MRL	Minimal Risk Level
MS	Mass Spectrometry
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No-Observed-Adverse-Effect Level
NOHS	National Occupational Hazard Survey
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PbB	Blood lead level

PEL	Permissible Exposure Limit
ppm	Parts per million
REL	Recommended Exposure Limit
RfD	Reference Dose
STEL	Short Term Exposure Limit
TBARS	Thiobarbituric acid reactive substance
TLV	Threshold Limit Value
TSCA	Toxic Substances Control Act
WHO	World Health Organization

ABSTRACT

Incineration is used to destroy and reduce the amount of hazardous chemical and biological wastes consequently lowering their potential infectious and toxic properties. Incineration by-products like ash are a potential risk to human health and the environment. The problem is growing with an ever-increasing number of hospitals, clinics, diagnostic laboratories and research laboratories in the City of Nairobi, Kenya. The objectives of this study were to assess the heavy metals concentration in bottom ash and their ecotoxicological effects in twelve major incinerators in Nairobi.

A baseline survey on the environmental impacts of heavy metals and incineration methods was conducted between June and August 2007 at twelve major incinerators in Nairobi. Empirical field observation and field-level data collection through inventory, questionnaire survey and formal and informal interviews were administered from October to December 2007 after prior consenting of about 100 respondents. A structured questionnaire was designed to collect information on the respondents' socio-demographics, duration of stay in the proximity of incineration, attributable symptoms of heavy metal toxicity. A number of in-depth interviews were conducted to elaborate understanding the existing management practice of wastes, the potential hazards and morbidity due to heavy metal exposure. Quantitative data was analyzed, mainly with simple descriptive statistics.

A total of 36 bottom ash samples were collected during the study period in three different visits from all the incinerators. Ethical approval from respective institutional and government regulatory authorities was granted. The ashes were analyzed in triplicate for the presence of

Arsenic, Cadmium, Chromium, Copper, Mercury and Lead using Atomic absorption spectrometry (AAS) and detectable levels of all were found in bottom ash. One sample t- test (ANOVA) analysis revealed highly significant toxicity in all the elements. Chromium had the highest concentrations t=11.718, mean difference (MD) = 94.83mg/kg, CI (77.0206-112.6460) at p <0.05(Table 3) and Copper had the lowest concentrations but showed greatest variation among the sites, t=2.819, mean difference (MD) = 95.67 mg/kg, CI (20.9852- 170.3481) at P<0.05(Table 3) while Mercury had the smallest variation among all the sites, t = 8.529, mean difference (MD) = 31.42mg/kg, CI (23.3095-39.5238). Heavy metal concentrations exceeded the permitted values of GB 18918-2002

Ecotoxicological impacts and apparent toxicology indices on various body systems of the respondents were scored as Musculosketal system (21%), Dermatological (19%), Respiratory (18%), Gastrointestinal (13%), Cardiovascular (11%), Reproductive (9%) and Nervous (9%) respectively. Open dumping was the main disposal method in use.

Comprehensive evaluation of the environmental impacts of incineration pollution is necessary. Improvement on the design of incinerators is paramount with better management practices on proper disposal of bottom ash is needed to minimize the adverse environmental impact.

CHAPTER 1

1.0: INTRODUCTION

Incineration is used to destroy hazardous chemical and biological wastes which reduce the volume of municipal solid waste (MSW) and medical waste consequently lowering their potential infectious and toxic properties. In Kenya, less than 50 facilities incinerate municipal solid waste and medical waste. There are also industrial kiln facilities, and many industrial boilers and furnaces which combust hazardous and non-hazardous waste (Hansen, 2000).

With the development of the social economy and improvement of living standards and medical establishments, increased amounts of medical, pharmaceutical are being produced in Kenya. Incineration, as a waste treatment alternative benefiting volume reduction, thorough stabilization, sanitation, and energy generating, is playing a more and more important role in management of these wastes, (NCC, 2001)

A major concern is the potential risk to human health and the environment that might result from the emission of pollutants generated by incineration; some of those pollutants have been found to cause various adverse health effects to animals, plants and damage water sources (EPA, 2005). Emissions from incineration facilities contain many potentially harmful substances, including particulate matter; oxides of nitrogen; oxides of sulfur; carbon monoxide; dioxins and furans; heavy metals, acid gases; volatile chlorinated organic compounds; and polycyclic aromatic compounds (Thompson ***et al.***, 2004).

Incinerations discharges can lead to occupational and environmental exposures to toxic chemicals and subsequent health risks affecting waste workers, the general public, and the

environment. Resources are extremely limited in many countries, especially in remote areas. Consequently, open pit burning is still widely practiced for health-care waste including sharps, though this practice is objectionable due to emissions, the incomplete disinfection and destruction of the waste, and community complaints (Thompson ***et al.***, 2004). The adequacy of such emissions data to characterize fully the environmental implication of incineration is uncertain for incinerators in Nairobi and its environs. Hospitals, research laboratories and mortuaries use a variety of chemical substances, papers, bandages and pharmaceuticals. All of these wastes either are discharged directly into drainage systems as effluents or incinerated to reduce the waste volume (Emmanuel ***et al.***, 2005).

Heavy metals poisoning are associated with a variety of health problems in humans. Lead has a wide range of biological effects including; enzyme inhibition, neurological, metabolic, morphological and behavioral defects. Chronic cases can lead to cardiovascular complications, renal failure and reproductive deformities. Chromium toxicity is majorly produces respiratory syndromes, in chronic cases, it affects the immune system and it is a potential carcinogen. Mercury is a major CNS, cardiovascular and respiratory poison that causes neurological impairment, reproductive effects, liver damage and significant decreases in intestinal absorption. Cadmium causes renal failure and it is carcinogenic in chronic cases. Exposure to inorganic arsenic causes gastrointestinal irritation, immunological disturbances,cancer, heart disease, nerve injury and brain damage.

Improper disposal of health-care wastes, syringes and needles that are scavenged and reused may lead to significant numbers of infectious diseases such as hepatitis B, hepatitis C, HIV and possibly other infections in the developing world (Thompson and Anthony, 2005).

Incinerator emissions and associated risks may be reduced by implementing emission standards, operational controls, and enhanced management practices. Emission rates from incinerators are highly variable due to a number of reasons including poor incinerator designs, poor operation and inadequate maintenance.

The evaluation of the ecotoxicity of metals to environment is essential to predict changes in metal behavior in response to these environmental conditions. Therefore, the feasibility of the disposal and utilization of the bottom ash in Nairobi mainly depends on two factors: the speciation of heavy metals in the bottom ash and their environmental characteristics.

1.1 Justification:

Currently there is a global concern about environmental implication of incineration. Incinerator emissions are a major source of fine particulates of toxic metals and numerous organic chemicals. In Kenya there are inefficient safety measures designed to avoid acute environmental and human toxic effects due to incineration. Few scientific studies have attempted to assess the environmental implication providing insufficient toxicological evidence. Monitoring of incinerators has been unsatisfactory due the lack of rigorous legislative policies. Therefore there is an urgent need to assess concentration of heavy metals as a result of incineration in-order to develop and implement good combustion practice (GCP) and policy formulations to minimize environmental contamination by incineration of wastes containing heavy metals.

1.2 Research Hypotheses

Incineration of wastes containing heavy metals poses serious environmental implication.

1.3 Objectives:

1.3.1 General objective

To generate data on concentration of heavy metals in bottom ash after waste incineration to assist in formulation of sound environmental policies.

1.3.2 Specific objectives

i. To determine the concentration of Arsenic, Cadmium, Chromium, Copper, Mercury and Lead in bottom ash from twelve incinerator sites in Nairobi.

ii. To assess the types of incinerators used for incineration in the sites.

iii. To establish the methods of ash disposal from incinerators in Nairobi and implementation of mitigation strategies.

iv. To evaluate the morbidity due to exposure to heavy metals from the incinerators in Nairobi.

CHAPTER 2
LITERATURE REVIEW

2.0: Background Information

Thousands of tons of solid waste are generated daily in Africa. Most of it ends up in open dumps and wetlands, contaminating surface and ground water and posing major health hazards. Generation rates, available only for select cities and regions, are approximately 0.5 kilograms per person per day—in some cases reaching as high as 0.8 kilograms per person per day. While this may seem modest compared to the 1–2 kg per person per day generated in developed countries, most wastes in Africa are not collected by municipal collection systems because of poor management, fiscal irresponsibility or malfeasance and equipment failure (Johannessen *et al*,.1999a)

Majorly seventy percent (70%) of the waste is in organic form which could be converted to compost or used to generate biogas, but in situations where rudimentary solid waste management systems barely function, it is difficult to promote innovation, even when it is potentially cost-effective to do so. In addition, hazardous and infectious materials are discarded along with general waste in most of the countries in Africa (Johannessen ***et al***, .1999a)

Only in a few countries wastes are disposed off in sanitary landfills but most are deposited in open dumps or semi-controlled unlined landfills with no groundwater protection, leachate recovery, or treatment systems. The larger dumps are located on the edges of cities, towns, and villages, sometimes in ecologically sensitive areas, or areas where groundwater supplies are

threatened. They serve as breeding grounds for rats, flies, birds and other organisms that serve as disease vectors (UNEP-GEO-Team, 1999)

In Kenya the generation of waste has become an increasing social, economic health problem, due to both waste volumes and toxic elements release as a result of incineration. In Kenya's current waste management practice, economic instruments are generally not extensively used. One reason is that, in general, waste is easy to dispose of illegally as illustrated by uncontrolled dumping activities in urban centers and medical establishments. Another reason is that waste management is a complex process that contains many potentially negative loopholes to circumvent the intended incentive system (NCC, 2001)

About 1,500 tonnes of solid waste generated daily in Nairobi gets collected, the bulk of solid wastes are generated from, Pesticide repackaging, formulation and distribution, where there are over 30 manufacturing companies, Plastics industry, where there are about 100 producing thermo setting, flimsy packaging, Soap, Perfumes, Cosmetics, Toiletry, Cement and Lime, Ceramics, glass, petroleum and Pharmaceuticals and medical waste companies (NCC, 2001)

The National Environment Management Authority (NEMA) is a body that was established by an Act of parliament as the principal agency in charge of coordination, monitoring and supervision of all environmental management issues in the country. This it does in coordination with the district Environmental officers resident in every district in the country.

It is a requirement under regulation 13 of the National Environment (waste management) Regulations 1999 to obtain a license for operating a waste treatment/disposal facility from NEMA so as to increase environmental monitoring and awareness which will possibly lead to adherence to environmental laws and regulations, thus contributing to conservation of environment.

2.1: Combustion and Metal Concentration in the ash

The different in metals total concentration is as a result of different in combustion temperatures which are major parameter is determining the metals volatility, since the extent of evaporation of the metals and metal compounds in the furnace is directly related to combustion temperature. Although there might be a number of other factors having significant effects in waste incineration for example, highly volatile metals such as mercury and cadmium start to volatilize at temperatures as low as 200 ◦C. It is believed that operating temperature in an incinerator should be higher than 850 ◦C, however such high temperatures increase volatilization of hazardous metals and lead to adverse environmental pollution (Wei ***et al***, .1998).

Lower kiln temperatures causes all the metals except Pb, the most volatile one, to be retained mostly in bottom ash. Therefore, it could be said that the higher the amount of waste, the larger the fraction of metal that is retained in bottom ash at high temperatures during incineration process. Phases and possible secondary reactions of these compounds could have a significant role on metal partitioning (Bakoglu ***et al.***, 2002).

The presence of waste-derived Cl enhances volatilization by formation of metal chlorides that are typically more volatile than metal oxides, and therefore increases toxic metallic emissions. Chloride formation in incineration system depends on many factors which include Cl/metal

ratio, temperature and affinity of metal to Cl, presence of hydrogen, carbon and sodium. Sulfur, on the other hand, reduces the volatilization by formation of stable metal sulfates that displace the chlorides and oxides at low temperatures, while volatile sulfides may appear at high temperatures, particularly under reducing conditions (Trouvé ***et al***,. 1998)

Difficulties related with the incineration of solid wastes (poor mixing, requirement of long residence times for complete combustion, was also considered in this study. Since solid wastes contain most of the metals, penetration of the outside temperature into the furthest point inside the solid, i.e. the center, could take longer times than the theoretical ones, leading metals to be retained in bottom ash, even if the physical and chemical conditions are suitable for volatilization. Therefore, the part of metals retained in bottom ash is usually of larger concentration (EPA/NEMA, 2005).

In incinerators combusting hazardous wastes, high temperatures are desirable for the complete destruction of organic matter to minimize the formation of toxic organics during incineration, but they also enhances the metal volatility, resulting in an increase in the release of volatile metallic compounds, which could be toxic to the atmosphere. Therefore, from the public health point of view an efficient metal removal system is indispensable in incinerators to (Trouvé ***et al***,. 1998)

2.2: Environmental effects of heavy metals incineration

Incinerator discharges are released into air, water and soil. These discharges can lead to occupational and environmental exposures to toxic chemicals and subsequent health risks affecting waste workers, the general public, and the environment (Chimenos ***et al***., 2003). With poor management, infectious risks may also remain, largely in the occupational setting, e.g., waste handlers and incinerator operators. Incinerator emissions include both

"conventional" pollutants, e.g., particulate matter, sulfur oxides, nitrogen oxides, volatile organic compounds and carbon monoxide, as well as dioxins, furans, arsenic, lead, cadmium, chromium, mercury, and hydrochloric acid. In the aggregate, incinerators can emit significant quantities of gaseous and particulate pollutants to the atmosphere and incineration of health-care waste in small and poorly controlled incinerators is a major source of heavy metals, dioxins and furans. (Hansen, 2000).

Incineration itself is a controversial topic these days but it has its advantages and disadvantages; as waste volume reduction, destruction of combustible toxins, destruction of pathogenically contaminated material and energy recovery (Dempsey and Oppelt, 2003). The disadvantages of it are air pollution problems, ash must be landfilled and may be hazardous, high capital and operation costs and contamination of water bodies. Incineration thermally decomposes matter through oxidation, thereby reducing and minimizing the wastes, and destroying their toxicity (Hansen, 2000).

Currently, most concern has focused on direct exposure pathways through inhalation of air emissions affecting incinerator workers and individuals living or working in the proximity. Air pollutants deposited in soil, vegetation and water can lead to 'indirect' exposures through ingestion of locally-produced foods or water, and dermal absorption due to contact with contaminated dusts, soil, water and plants, (Dempsey and Oppelt, 2003). Indirect exposures pathways can far exceed direct exposures, where individuals are exposed through a different mix of pathways for persistent and/or bioaccumulative pollutants, e.g., polycyclic aromatic hydrocarbons. dioxins, polychlorinated biphenyls, mercury, cadmium, etc., that undergo

chemical and physical transformations, cycling in and out of soil, vegetation, and surface water (Thompson and Anthony, 2005).

The waste stream from incinerators also includes solid and liquid wastes, namely, bottom ash and residues from pollution control equipment. Typically, solid wastes are disposed in soils (typically landfills or pits). Liquid wastes containing heavy metals (e.g., wet scrubber effluent, boiler blow-down, etc.) from some incinerators may be discharged to a sanitary sewer. Disposal of waste, ash, liquid or other residues containing heavy metals in unlined pits or other improperly managed facilities may contaminate groundwater, which may be used for drinking water (Goyer, 1999; Robert ***et al.***, 2001).

2.3: ARSENIC

Arsenic is released into the environment by human activities among incineration, arsenical pesticide and preservative use, metal smelting, waste incineration, and coal combustion . Major sources include; contaminated foods (especially seafoods), water or medications, ore smelting/refining/processing plants, galvanizing, etching and plating processes. Tailings from or river bottoms near gold mining areas. Insecticides, rodenticides and fungicides (Na-, K-arsenites, arsenates, also oxides are commercially available). Commercial arsenic products include: sodium arsenite, calcium arsenate, lead arsenate and "Paris green" (cupric acetoarsenite) a wood preservative (Barton ***et al***, 2002).

During waste incineration, arsenic (As) is emitted to the air inform of various inorganic compounds or in elemental form. Arsenic has valance states of -3, 0, +3, or +5, Trivalent

arsenic compounds are quite soluble at ambient temperatures. Pentavalent arsenic is more soluble than trivalent. It is indicated that incineration of waste containing arsenic is typically the greatest source of exposure for the general population (Barton ***et al***, 2000).

2.3.1: Toxicokinetics

Both arsenates and arsenites are well absorbed by both the oral and inhalation routes. The rate of absorption of arsenic in highly insoluble forms (e.g., arsenic sulfide, lead arsenate) is much lower than that of more soluble forms via both oral and inhalation routes. Once absorbed in the intestines, arsenites are oxidized to arsenates and methylated. This process may then be repeated to result in dimethylated arsenic metabolites. The As (+3) form undergoes enzymic methylation primarily in the liver to form MMA and DMA. Most arsenic is promptly excreted in the urine as a mixture of As (+3), As(+5), MMA, and DMA; DMA is usually the primary form in the urine. Smaller amounts are excreted in feces. Some arsenic may remain bound to tissues, depending inversely on the rate and extent of methylation (Kitchin, 2001)

It is becoming increasingly evident that the toxicity and carcinogenicity of arsenic is likely to be closely associated with metabolic processes. Absorbed pentavalent arsenic is rapidly reduced to trivalent arsenic (AsIII) at least partially in the blood. Much of the formed trivalent arsenic is distributed to tissues and taken up by cells particularly hepatocytes (Florea and Büsselberg 2008). Many cell types appear to accumulate trivalent arsenic more rapidly than pivalent arsenic. Because trivalent arsenic (as arsenite) is known to be more highly toxic than pivalent arsenic (as arsenate), the reduction step may be considered bioactivation rather than

detoxification. Glutathione appears to play a role in the reduction of pivalent arsenic to trivalent arsenic, which is required prior to methylation (Kitchin, 2001)

Methylation of arsenic ultimately forms relatively less toxic MMA and DMA; this process is accomplished by alternating between the reduction of pivalent arsenic to trivalent arsenic and the addition of a methyl group; S-adenosylmethionine is considered to be the source of the methyl group. The methylation process appears to include multiple intermediates, some of which are more reactive than inorganic arsenic. Additional in vitro studies have demonstrated genotoxic and DNA damaging properties of both MMAIII and DMAIII (Lantz and Hays, 2006).

Arsenite inhibits pyruvate dehydrogenase (PDH), a complex that oxidizes pyruvate to acetyl-CoA, a precursor to intermediates of the citric acid cycle that provides reducing equivalents to the electron transport system for ATP production. This property may explain the depletion of carbohydrates in arsenite-treated rats (Rossman, 2003; Thomas ***et al.***, 2007)

2.3.2 Toxicodynamics

Dermal Effects

The most characteristic effect of long-term exposure to arsenic compounds is the development of skin lesions (pale and milky) which are mostly associated with chronic arsenicosis, include hyperkeratinization on the palms and soles skin, multiple hyperkeratinized corns or warts, whitelines over nails and hyperpigmentation of the skin (Ahsan ***et al.***, 2006; Chakraborti ***et al*** ., 2003b).

Altered dermal pigmentation and hyperkeratosis have also been reported in studies of humans exposed to arsenic by inhalation. Usually, the effects are mild (erythema and swelling), but may progress to papules, vesicles, or necrotic lesions in extreme cases (Haque ***et al.***, 2003; Rossman ***et al.***, 2004)

Cardiovascular Effects

The cardiac effects of arsenic include altered myocardial depolarization, cardiac arrhythmias, and ischemic heart disease. These are acute and chronic effects and from intravenous therapy with arsenic trioxide for acute promyelocytic leukemia (Mumford ***et al.***, 2007).

Chronic exposure to arsenic has also been shown to lead to effects on the vascular system. The most dramatic of these effects is "Blackfoot Disease," a disease characterized by a progressive loss of circulation in the hands and feet, leading ultimately to necrosis and gangrene (Wang *et al.*, 2003). Changes in cardiac rhythm and in some vascular end points have also been reported in animal studies of arsenicals (Wang ***et al.***, 2003; Tseng ***et al.***, 2005).

Respiratory Effects

Exposed workers often report irritation of the mucous membranes of the nose and throat, which may lead to laryngitis, bronchitis, or rhinitis.Bronchitis and sequelae (bronchiectasis, bronchopneumonia) have been observed in patients chronically exposed to arsenic (Tseng ***et al.***, 2005)

Gastrointestinal Effects

Both short-term and chronic exposures to arsenicals have been reported to result in weight loss due to irritant effects on gastrointestinal tissues including nausea, vomiting, diarrhea, and abdominal pain, (Chakraborti ***et al.***, 2003a).

Similar gastrointestinal effects have been reported after occupational exposures to arsenicals, although it is not known if these effects were due to absorption of arsenic from the respiratory tract or from mucociliary clearance resulting in eventual exposure (Vantroyen ***et al.***, 2004).

Neurological Effects

Chronic exposure to inorganic arsenic compounds may lead to the development of peripheral neuropathy and encephalopathy, with signs and symptoms such as headache, lethargy, mental confusion, hallucination, seizures, and coma are common (Vantroyen ***et al.***, 2004; Baker ***et al.***, 2005).

Developmental Effects

Arsenic has been shown to produce developmental effects by inhalation exposure in laboratory animals, although it is unclear whether or not the effects occur only at maternally toxic doses. Mice exposed to 22 mg As/m^3 for 4 hours on days 9–12 of gestation had serious developmental effects (significant increases in the percentage of dead fetuses, skeletal malformations, and the number of fetuses with retarded growth), while those exposed to 2.2 mg As/m^3 had only a 10% decrease in average fetal body weight, and those exposed to 0.20 mg As/m^3 had no effects (Milton ***et al.***, 2005).

Carcinogenic potential

Numerous studies on workers exposed to arsenic trioxide have reported an increased risk of lung cancer. Increased incidence of lung cancer has also been observed at chemical plants where exposure was primarily to arsenate. In general, studies reporting long-term exposure to 0.07 mg As/m^3 or greater have shown an increased incidence of lung cancer (Bean Freeman ***et al.***, 2004).

There is convincing evidence from a large number of epidemiological studies and case reports that exposure to arsenic increases the risk of developing skin cancer. The most common tumors seen are squamous cell carcinomas, which may develop from the hyperkeratotic warts or corns commonly seen as a dermal effect .There is increasing evidence that long-term exposure to arsenic can result in the development of bladder cancer, with transitional cell cancers being the most prevalent (Chen ***et al.***, 2007).

Genotoxicity

Collectively, in vitro and in vivo genotoxicity assays have demonstrated that arsenics cause single strand breaks, formation of apurinic/apyrimidinic sites, DNA base and oxidative base damage, DNA-protein crosslinks, chromosomal aberrations, aneuploidy, sister chromatid exchanges, and micronuclei. Chromosomal aberrations, characterized by chromatid gaps, breaks and fragmentation, endoreduplication, and chromosomal breaks, are dose-dependent and arsenite is more potent than arsenate (Bean Freeman ***et al.***, 2004 ; Florea and Büsselberg 2008)

2.3.3 Prophylaxis and management of Arsenic poisoning.

Arsenic must be methylated using methyl donors such as trimethylglycine, dimethylglycine, methionine. Some arsenic is bound to sulfur groups such as glutathione and excreted in the urine or bile. In cases of reported poisoning the individual should be removed from sources of arsenic and treated for anemia if indicated. Sulfurated methyl groups and foods rich in sulfurated amino acids (garlic, eggs, beans) are recommended. Supportive therapy with magnesium, B-vitamins, vitamin C, vitamin E, selenomethionine, lipoic acid is also recommended. Gastric Lavage by drinking enormous amounts of water should be encouraged (an adult's urine volume should be > 2 liters/day). Cysteine is contraindicated. Aggressive therapy for excess arsenic can employ sulfhydryl-group type conjugating agents. Therapy should be continued until urinary arsenic levels are consistently below those stated above (Vantroyen ***et al.***, 2004; ASDR, 2009).

2.4 CADMIUM

Cadmium emissions arise from two major source categories, natural sources and man-made or anthropogenic sources. Emissions occur to the three major compartments of the environment - air, water and soil, but there may be considerable transfer between the three compartments after initial deposition. Emissions to air are considered more mobile than those to water which in turn are considered more mobile than those to soils (Campbell, 2002). Higher levels of cadmium may accumulate in sedimentary rocks, and marine phosphates and phosphorites as high as 500 ppm beyond the normal earth's crust 0.1 and 0.5 ppm (EPA, 2005). Man-made cadmium emissions arise either from the manufacture, use and disposal of products

intentionally utilising cadmium, or from the presence of cadmium as a natural but not functional impurity in non-cadmium containing products (Thun ***et al***, 1985)

2.4.1Toxicokinetics

Following absorption from any route of exposure, cadmium widely distributes throughout the body, majorly ending up in the liver and kidney (Wester ***et al***., 1992). Most cadmium that is ingested or inhaled and transported to the gut via mucociliary clearance is excreted . Of the cadmium that is absorbed into the body, most is excreted very slowly, with urinary and fecal excretion being approximately equal (Wester ***et al***., 1992).

The cadmium (+2) ion readily bind to anionic groups (sulfhydryl groups) in proteins (e.g., albumin and metallothionein). Cadmium interacts with the protein metallothionein. Initially cadmium in plasma circulates primarily bound to albumin (Nordberg ***et al***., 1985). Cadmium enters the liver where it becomes bound to metallothionein and is released to the blood stream. Metallothionein-bound cadmium is readily filtered by the renal glomerulus and reabsorbed from the glomerular filtrate by the proximal tubule cells (Nordberg ***et al***., 1985)

Renal damage is believed to occur if there is a localization of free cadmium or an excessive concentration of cadmium that remains unbound to metallothionein. Metallothionein metabolism in liver and kidney is relatively independent of the exposure route; inhalation exposure also induces metallothionein in the lung and oral exposure induces metallothionein in the intestine (Nordberg ***et al***., 1985).

2.4.2 Toxicodynamics

Respiratory effects

Acute inhalation exposure to cadmium at concentrations above about 5 mg/m3 may cause destruction of lung epithelial cells, resulting in pulmonary edema, tracheobronchitis, and pneumonitis in both humans and animals. Studies in animals confirm that inhalation exposure causes severe, multifocal interstitial pneumonitis, diffuse alveolitis with hemorrhage, increased lung weight, inhibition of macrophages, focal interstitial thickening, edema, and necrosis and hyperplasia of alveolar cells (Andersen ***et al.*** 1988).

Gastrointestinal effects

The main symptoms following ingestion of cadmium at doses above about 0.07 mg/kg in humans are nausea, vomiting, and abdominal pain (Andersen ***et al.***, 1988).

Hematological effects

Both oral and inhalation exposure to cadmium can cause anemia in humans and animals. It is likely that cadmium transported to the gastrointestinal system from the lung following inhalation exposure would also reduce iron uptake and absorption (Davison ***et al.*** 1988).

Musculoskeletal effects

Prolonged inhalation or ingestion exposure of humans to cadmium at levels causing renal dysfunction can lead to painful and debilitating bone disease. Chronic exposure causes alternations in renal metabolism of vitamin D, which then may cause osteoporosis (Shigematsu, 1984; Nogawa ***et al.*** 1987).

Hepatic effects

Exposure to high levels of cadmium can cause liver damage in animals (necrosis of hepatocytes, metabolic changes, membrane peroxidation) (Andersen ***et al.***, 1988).

Renal effects

The first manifestation of kidney damage is decreased reabsorption of filtered low molecular-weight proteins, indicating damage to the renal tubules. Production of tubular proteinuria is a relatively specific effect of cadmium. Proteinuria , glycosuria and aminoaciduria from tubular injury are the most frequent renal effects caused by cadnium (Andersen ***et al.***, 1988).

Immunological and Lymphoreticular effects

Inhalation exposures have been shown to suppress the primary humoral immune response to be cytotoxic to spleen lymphocytes causing splemomegally and thoracic lymphadenopathy (Andersen ***et al.***, 1988).

Developmental Effects

Occupationally exposed pregnant workers to cadmium at concentrations ranging from 0.02 to 35 mg/m3 had offspring with decreased birth weights compared to unexposed controls. Developmental toxicity in offspring of female rats exposed to cadmium oxide at 0.02 mg Cd/m3 manifest by delayed ossification, decreased locomotor activity, and impaired reflexes in offspring (Andersen ***et al.***, 1988).

Genotoxic and Carcinogenic effects

Examination of lymphocytes from workers occupationally exposed to cadmium have shown statistically significant increases in chromosomal aberrations. The relationship between occupational exposure to cadmium and increased risk of cancer (particularly lung and prostate cancer) has been explored in a number of epidemiologic studies following prolonged inhalation of cadmium. Studies in rats provide strong evidence of the lung carcinogenic potential of chronically inhaled cadmium (Thun ***et al.***, 1985).

2.4.3 Prophylaxis and management of Cadmium poisoning.

Cadmium poisoning is managed like other heavy metals toxicosis. Hemodialysis may be used to remove circulating cadmium from the bloodstream. Addition of a chelating agent, particularly ethylenediamine tetraacetic acid, will increase the amount of cadmium removed by the dialysate. Dimercaptosuccinic acid an oral chelating agent), has been recommended for removal of cadmium from the blood (ASDR, 2009).

2.5: CHROMIUM

Chromium is a naturally occurring element found in animals, plants, rocks, and soil and in volcanic dust and gases. Chromium has oxidation states (or "valence states") ranging from chromium(-II) to chromium(VI). Elemental chromium (chromium(0)) does not occur naturally. Chromium compounds are stable in the trivalent state and occur in nature in this state in ores, such as ferrochromite. The hexavalent (VI) form is the second-most stable state. However, chromium (VI) rarely occurs naturally, but is usually produced from anthropogenic sources (EPA, 2005).

2.5.1: Toxicokinetics

Following exposure, hepatic and renal effects are most prevalent, with effects being generally lesser in other tissues (ASDR, 2009). The toxicity of chromium is dependent on the oxidation state of the chromium atom. Once it is taken into cells, chromium (VI) has been shown to undergo a reduction to chromium(III), with chromium(V) and chromium(IV) as intermediates (Liu ***et al.***, 1998).

Chromium (VI), chromium (V), and chromium (IV) have all been shown to be involved in fenton-like oxidative cycling, generating oxygen radical species. The formation of these radicals may be responsible for many of the deleterious effects of chromium on cells, including the formation of DNA strand breaks. The interaction of free radicals with DNA can result in structural DNA damage, functional damage, and cellular effects. The types of structural damage include DNA strand breaks, DNA-protein crosslinks, DNA-DNA interstrand crosslinks, chromium-DNA adducts, and chromosomal aberrations (Costa ***et al.*** 1997; Chen ***et al.***, 2007).

Functional damage includes DNA polymerase and RNA polymerase arrest, mutagenesis, and altered gene expression. Chromium can also interact with DNA to form adducts/complexes and DNA-protein crosslinks that interfere with DNA replication and transcription, and can promote the expression of regulatory genes such as nuclear factor-κβ, or may inhibit regulatory genes such as GRP78 having potential carcinogenesis. The structural and functional damage can lead to growth arrest and apoptosis (Carlisle ***et al.*** 2000; Chen ***et al.***, 2007).

2.5.2: Toxicodyamics

Respiratory Effects

Acute exposure to chromium (VI) compounds may lead to asthma and other signs of respiratory distress, Dyspnea, cough, and wheezing. These have been reported among individuals who had inhaled "massive amounts" of chromium (VI) trioxide. Intermediate- to chronic-duration occupational exposure to chromium (VI) may cause an increased risk of death due to noncancer respiratory disease. High incidences of nasal septum perforation, septal atrophy and ulcerations, sinusitis, pharyngitis, and bronchitis have been found to be caused by its exposure (Kuo ***et al.***, 1997).

Gastrointestinal and Hematological Effects

Gastrointestinal effects have been associated with occupational exposure of humans to chromium compounds. In a report of two cases of acute exposure to "massive amounts" of chromium trioxide fumes, there were complains of abdominal or substernal pain (Mancuso, 1997).

Other reported symptoms are stomach pain, duodenal ulcer, gastritis, stomach cramps. Hematological evaluations of workers occupationally exposed to chromium compounds have found to cause leucopenia which is related to primarily to monocytosis and eosinophilia, decreases in hemoglobin concentrations and slight increases in bleeding has also been observed (Kuo ***et al.***, 1997).

Hepatic effects

Chromium (VI) has been reported to cause derangement of the cells in the liver, necrosis, lymphocytic and histiocytic infiltration, and increases in Kupffer cells. Abnormalities in tests for hepatic dysfunction included increases in sulfobromophthalein retention, gamma globulin, icterus, cephalin cholesterol flocculation, and thymol turbidity (Mancuso, 1997; Kuo ***et al.***, 1997).

Renal Effects

Some studies of workers exposed to chromium(VI) and chromium(III) in the chromate production industry have found increased urinary levels of low molecular weight proteins indicative of renal damage, such as retinol binding protein and antigens, and white blood cell and red blood cell casts, in the urine (Liu ***et al.***, 1998).

Severe renal impairment, renal failure, and necrosis of renal tubules have been reported in cases of fatal or near fatal ingestion of chromium(VI) compounds by humans and Acute nephritis has also been reported in cases of dermal exposure of chromium(VI) compounds. Numerous studies in animals exposed to compounds has reported renal effects. In acute experiments, necrosis of the proximal tubules and alterations of kidney enzymes and altered kidney function (Liu ***et al.***, 1998).

Immunological and Lymphoreticular effects

Acute reactions have been observed in chromium sensitive individuals exposed to chromium via inhalation and there have been complained of frequent skin eruptions, dyspnea, and chest

tightness, anaphylactoid reaction, characterized by dermatitis, facial angioedema, bronchospasms and urticaria. Similar anaphylactoid reactions have been observed with decreased forced expiratory volume, facial erythema, nasopharyngeal pruritus, nasal blocking, cough, and wheezing (Liu, 1998; EPA, 2005).

Genotoxicity effects

Studies of workers exposed to an average level of 0.008 mg chromium (VI)/m3 had increases in chromosomal aberrations and sister chromatid exchanges (EPA, 2005)

Carcinogenicity

Occupational exposure to chromium (VI) compounds has been associated with increased risk of respiratory system cancers, primarily bronchogenic and nasal (Mancuso, 1997; EPA, 2005)

Dermal and Ocular effects

These include irritation, burns, ulcers, an allergic type of dermatitis. In addition, tonsillitis, pharyngitis, atrophy of the larynx, and irritation and ulceration of mouth structures and buccal mucosa can occur from exposure to high levels of chromium (VI) compounds (EPA, 2005)

Ocular effects can occur because of direct contact of eyes with chromium compounds. These include corneal vesication congestion of the conjunctiva, discharge, corneal scar, and burns in chromate production workers because of accidental splashes (EPA, 2005)

2.5.3: Prophylaxis and management of Chromium poisoning.

Treatment in cases of acute, high-level chromium exposure is usually supportive and symptomatic. Supportive measures may include ventilatory support, cardiovascular support, and renal and hepatic function monitoring. When renal function is compromised, urine alkalinization and maintenance of adequate urine flow are important (ASDR, 2009).

Progression to anuria is associated with poor prognosis. Copious amounts of water are used to if the eyes and skin are directly exposed. Topical ascorbic acid has been successfully used to prevent chromium dermatitis and dermal burns caused by dichromate. The ulcers heal in several weeks without specific treatment. Ethylenediaminetetraacetic acid (EDTA) ointment 10% might facilitate removal of chromate scabs. Gastric lavage with magnesium hydroxide or another antacid might be useful in cases of chromium ingestion. Fluid and electrolyte balance is critical. Induction of vomiting is contraindicated (EPA, 2005; ASDR, 2009)

In low-dose exposure, resulting to chronic toxicosis, no specific treatment is needed. The mainstay of management is removing the patient from further exposure and relying on the urinary and fecal clearance of the body burden. Although normal urinary excretion is quite rapid, forced diuresis has been used (EPA, 2005)

2.6: LEAD

Lead in the environment is mainly particulate bound with relatively low mobility and bioavailability. Lead does not, in general, bioaccumulate and there is no increase in concentration of the metal in food chains. Lead is not essential for plant or animal life

(Dempsey, 2003). Lead is used in storage batteries, industrial paint, solder, electric cable covering, pottery glaze, rubber, toys, gasoline (tetraethyl lead), and brass alloys (Campbell, 2002)

2.6.1: Toxicokinetics

Absorption

Gastrointestinal absorption of inorganic lead occurs primarily in the duodenum this may involve active transport and/or diffusion through intestinal epithelial cells (transcellular) or between cells (paracellular), and may involve ionized lead (Pb^{+2}) and/or inorganic or organic complexes of lead (Oomen ***et al.***, 2003b).

Distribution

Lead in blood: it is rapidly taken by red blood cells, where it binds to several intracellular proteins. The major proposed pathway is an anion exchanger that is dependent upon HCO3 and is blocked by anion exchange inhibitors (Bannon ***et al.***, 2000).

Aminolevulinate, delta-, dehydratase (ALAD) is the primary binding ligand for lead in erythrocytes. Lead binding to ALAD is saturable; the binding capacity has been estimated to be approximately 850 µg/dL red blood cells (or approximately 40 µg/dL whole blood) and the apparent dissociation constant has been estimated to be approximately 1.5 µg/L (Bergdahl ***et al.***, 2006).

ALAD is a polymorphic enzyme with two alleles (ALAD 1 and ALAD 2) and three genotypes: ALAD 1, 1, ALAD 1, 2, and ALAD 2, 2. Higher PbBs were observed in individuals with the ALAD 1, 2 and ALAD 2, 2 genotypes compared to similarly exposed individuals with the ALAD 1, 1 genotype (Schwartz ***et al.***, 2000).

Lead in Blood Plasma -Lead in plasma exists in four states: loosely bound to serum albumin or other proteins with relatively low affinity for lead, complexed to low molecular weight ligands such as amino acids and carboxylic acids, tightly bound to a circulating metalloprotein, and as free Pb2+. Lead may also bind to γ-globulins. Lead in serum that is not bound to protein exists largely as complexes with low molecular weight sulfhydryl compounds (e.g., cysteine,homocysteine). Other potential low molecular weight lead-binding ligands in serum may include citrate, cysteamine, ergothioneine, glutathione, histidine, and oxylate (Schwartz ***et al.***, 2000).

Lead in Bone: Approximately 95% of lead in adult tissues, and approximately 70% in children, resides in mineralized tissues such as bone and teeth. A portion of lead in bone readily exchanges with the plasma lead pool and, as a result, bone lead is a reservoir for replenishment of lead eliminated from blood by excretion (Dietert, 2004)

Lead forms highly stable complexes with phosphate and can replace calcium in the calcium-phosphate salt, hydroxyapatite, which comprises the primary crystalline matrix of bone. As a result, lead deposits in bone during the normal mineralization process that occurs during bone

growth and remodeling and is released to the blood during the process of bone resorption (Healey *et al.*, 2008).

The distribution of lead in bone reflects these mechanisms; lead tends to be more highly concentrated at bone surfaces where growth and remodeling are most active. This also gives rise to an age-dependence in bone lead distribution. During infancy and childhood, bone calcification is most active in trabecular bone, whereas in adulthood, calcification occurs at sites of remodeling in cortical and trabecular bone (Healey *et al.*, 2008).

Excretion

Measurement of the renal clearance of ultrafilterable lead in plasma indicates that, in dogs and humans, lead undergoes glomerular filtration and net tubular reabsorption. Renal clearance of blood lead increases with increasing blood lead concentrations above 25 μg/dL. In humans, absorbed inorganic lead is excreted in feces, however, pathways of excretion may include secretion into the bile, gastric fluid and saliva (Diamond, 2005).

2.6.2: Toxicodynamics

Respiratory effects

Very limited information is available regarding respiratory effects in humans associated with lead exposure. Few studies have reported significant alterations in tests of pulmonary function among the workers exposed to lead (Bagci *et al.*, 2004).

Cardiovascular effects

The greatest concern for humans at low exposures may predispose people to glomerular disease. Changes in cardiac conduction and rhythm, which may be secondary to Lead-induced

impairment of peripheral nerve conduction have been noted (Vaziri and Sica, 2004; Böckelmann ***et al,***. 2002).

Hypertensive effects

Meta-analyses of the epidemiological findings have found a persistent trend in the data that supports a relatively weak, but significant quantitative association that amounts to an increase in systolic blood pressure of approximately 1 mmHg with each doubling of PbB, However, studies conducted in animal models support the plausibility of blood pressure effects of lead in humans. These studies have shown that long-term lead exposure can elevate blood pressure in nutritionally replete rats (Carmignani ***et al.***, 2000; Schwartz ***et al.***, 2000 ; Nawrot ***et al.***, 2002).

Gastrointestinal effects

Colic is a consistent early symptom of lead poisoning in occupationally exposed cases or in individuals acutely exposed to high levels of lead. Although gastrointestinal symptoms typically occur at PbBs of 100–200 μg/dL, they have sometimes been noted in workers whose PbBs were between 40 and 60 μg/dL (Rosenman ***et al.***, 2003).

Hematological effects

Lead causes microcytic and hypochromic anemia. Lead inhibits with the activities of δ-aminolevulinic acid dehydratase (ALA-D) and ferrochelatase. Heme biosynthesis is decreased and the activity of the rate-limiting enzyme of the pathway,δ-aminolevulinic synthetase (ALAS) is subsequently increased. These causes increased urinary porphyrins,

coproporphyrin,and δ-aminolevulinic acid (ALA); increased blood and plasma ALA; and increased erythrocyte protoporphyrin (EP) (Gurer-Orhan ***et al.***, 2004).

Musculoskeletal effects

A recent study in mice reported that lead delays fracture healing at environmentally relevant doses and induces fibrous non-unions at higher doses by the progression of endochondral ossification. In studies in cultured osteoblast-like cells, lead disrupted the modulation of intracellular calcium by 1, 25-dihydroxyvitamin D in a biphasic manner (Rosenman ***et al.***, 2003 ; Carmouche ***et al.***, 2005).

Hepatic effects

In children, exposure to lead has been shown to inhibit formation of the heme-containing protein cytochrome P-450, as reflected in decreased activity of hepatic mixed-function oxygenases. These biochemical transformations are mediated by hepatic mixed-function oxygenases (Carmouche ***et al.***, 2005).

Renal effects

Lead nephrotoxicity is characterized by proximal tubular nephropathy, glomerular sclerosis and interstitial fibrosis. Functional deficits in humans that have been associated with excessive lead exposure include enzymuria, low- and high-molecular weight proteinuria, impaired transport of organic anions and glucose, and depressed glomerular filtration rate. The functional changes are thought to be related to an effect of lead on mitochondrial respiration and phosphorylation (Muntner ***et al.***, 2003; Diamond, 2005).

Endocrine effects

Occupational studies provide evidence for an association between high exposures to lead and changes in thyroid, pituitary and testicular hormones. Changes in circulating levels of thyroid hormones, particularly serum thyroxine (T4) and thyroid stimulating hormone (TSH), generally occurred in workers having mean PbB≥40 –60 µg/dL. Altered serum levels of reproductive hormones, particularly follicle stimulating hormone (FSH), luteinizing hormone (LH), and testosterone, have been observed at PbB ≥30–40 µg/dL (Diamond, 2005).

Ocular effects

Lead is known to affect visual evoked potentials in adults and children and there is disruption the lens redox status by inducing oxidative damage to lens epithelial cells (Schaumberg ***et al.***, 2004).

Neurological effects

The most severe neurological effect of lead is encephalopathy which is more common in children than adults. Early symptoms that may develop within weeks of initial exposure include dullness, irritability, poor attention span, headache, muscular tremor, loss of memory, and hallucinations. The condition may then worsen, sometimes abruptly, to delirium, convulsions, paralysis, coma, and death (Rosenman ***et al.***, 2003).

Neurobehavioral effects in adults

Occupational exposure to lead has often been associated with signs of neurotoxicity. The incidence of these symptoms, including malaise, forgetfulness, irritability, lethargy, headache,

fatigue, impotence, decreased libido, dizziness, weakness, and paresthesia at PbBs that range from approximately 40 to 120 μg/dL (Lucchini ***et al.***, 2000; Rosenman ***et al.***, 2003).

Developmental toxicity

In addition to inducing neurobehavioral alterations in developing organisms, exposure to lead has been associated in some studies with reduced birth weight and gestational age, reduced stature in children and delayed sexual maturation in girls (Jelliffe-Pawlowski ***et al.***, 2006)

Immunotoxicity

Altered immune parameters have been described in workers handling substances containing lead. Reported effects have included changes in some T-cell subpopulations altered response to T-cell mitogens and reduced chemotaxis of polymorphonuclear (Dietert ***et al.***, 2004)

Three studies of children reported significant associations between PbB and increases in serum IgE levels. It is suggestion has been raised that *in utero* exposure to lead may be a risk factor for childhood asthma (Dietert ***et al.***, 2004; Karmaus ***et al.***, 2005).

Genotoxicity

The potential genotoxic effects of lead have been studied in lead workers as well as in *in vitro* cultures of mammalian cells and microorganisms. Although not always consistent, the results suggest that lead is a clastogenic agent, as judged by the induction of chromosomal aberrations, micronuclei, and sister chromatid exchanges in peripheral blood cells (James ***et al.***, 1994).

2.6.3: Prophylaxis and management of Lead poisoning.

The treatment is dependent on the form of lead involved. Elemental and inorganic lead can be treated effectively with chelators, but in organic Lead poisoning the compounds have already formed strong ligands with organic compounds in the body, and initial chelation is ineffective. At this stage, the patient is given supportive treatment (ASDR, 2009)

Eventually, if the patient is found to have a normal blood lead level and is generally well, no further treatment is needed. However, if the patient has a raised blood lead level, then chelating agents are used for treatment. If the patient is not acutely ill, oral agents can be used, and the new treatment DMSA is more effective than penicillamine (EPA, 2005; ASDR, 2009). For more acutely ill patients, calcium EDTA is used, which is administered intravenously Care must be taken during treatment, as the chelating agents cause the loss of much zinc as well as lead. Also dimercaprol, D-penicillamine is used (EPA, 2005; ASDR, 2009)

2.7: COPPER

Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and, at low levels, air. Its average concentration in the earth's crust is about 50 parts copper per million parts soil (ppm). Copper also occurs naturally in all plants and animals. It is an essential element for all known living organisms including humans and other animals at low levels of intake. At much higher levels, toxic effects can occur (Borak ***et al.***, 2000).

2.7.1: Toxicokinetics

Physiologically normal levels of copper in the body are held constant by alterations in the rate and amount of copper absorption, compartmental distribution, and excretion.The ability of copper to cycle between an oxidized state, Cu (II), and reduced state, Cu (I), is used by cuproenzymes involved in redox reactions. However, it is this property of copper that is also potentially toxic because the transitions between Cu (II) and Cu (I) can result in the generation of superoxide radicals and hydroxyl radicals (Camakaris ***et al***. 1999).

Copper homeostasis involves regulation of absorption, cellular uptake, intracellular transport, sequestration/storage, cellular efflux, and excretion from the body. *In vitro* studies provide evidence that copper uptake into intestinal cells appears to be saturable (Arredondo ***et al***., 2000).

Copper oxide was observed in alveolar capillaries 3 hours after albino rats were exposed to a welding dust aerosol generated from pure copper wires. The half time of copper sulfate in the lungs was estimated to be 7.5 hours after intratracheal instillation of 20 µg copper per Wistar rat (Saenko ***et al***., 1994).

There is some evidence that albumin plays a passive role in copper transport, carrying a large portion of the exchangeable copper in the circulation and releasing this to other carriers for actual cell-specific uptake and transcuprein is another plasma protein carrier, Thus, dietary copper is transported to, and enters, the liver and kidney (Saenko ***et al***., 1994).

Copper then reemerges into the plasma bound to the ceruloplasmin. Ceruloplasmin, which tightly binds six or seven copper atoms. Copper, probably as Cu (I) rather than Cu (II) enters the cell via a carrier-mediated process. The membrane-bound copper transporting adenosine triphosphatase (Cu-ATPase), which selectively binds copper ions, transports copper ions into and out of cells (Borak ***et al.***, 2000).

The metabolism of copper consists mainly of its transfer to and from various organic ligands, most notably sulfhydryl and imidazole groups on amino acids and proteins. In the liver and other tissues, copper is stored bound to metallothionein and amino acids and in association with copper-dependent enzymes where it is incorporated into the molecule, and it is released from the liver (Saenko ***et al.***, 1994).

A considerable fraction of fecal copper is of endogenous biliary origin and the rest is derived from unabsorbed copper and copper from desquamated mucosal cells. Normally, 0.5–3.0% of daily copper intake is excreted through the urine (Camakaris ***et al.***, 1999). Excess copper is sequestered within hepatocyte lysosomes where it is complexed with metallothionein. However, this protective mechanism is saturable and liver lesions can develop above the saturation limit. In copper loaded rats, lysosomes become enlarged and more fragile with decreased membrane fluidity (Tao ***et al.***, 2003). Excess copper results in oxidative damage, including lipid peroxidation. Increases in the level of thiobarbituric acid reactive substance (TBARS), a measure of lipid peroxidation, have been found in copper-loaded rats (Tao ***et al.***, 2003).

2.7.2: Toxicodynamics

Copper is an essential nutrient that is incorporated into a number of metalloenzymes involved in hemoglobin formation, drug/xenobiotic metabolism, carbohydrate metabolism, catecholamine biosynthesis, the cross-linking of collagen, elastin, and hair keratin, and the antioxidant defense mechanism. Copper-dependent enzymes, such as cytochrome c oxidase, superoxide dismutase, ferroxidases, monoamine oxidase (MOA), and dopamine β-monooxygenase, function to reduce activated oxygen species or molecular oxygen (Borak ***et al.***, 2000).

Copper is readily absorbed from the stomach and small intestine. Excess copper absorbed into gastrointestinal mucosal cells induces the synthesis of and binds to metallothionein Copper that eludes binding to intestinal metallothionen is transported to the liver. It is stored in the liver bound to liver metallothionen, from which it is ultimately released into bile and excreted in the feces. Although copper homeostasis plays an important role in the prevention of copper toxicity, exposure to excessive levels of copper can result in liver and kidney damage, anemia, immunotoxicity, and developmental toxicity. Many of these effects are consistent with oxidative damage to membranes or macromolecules (Borak ***et al.***, 2000).

Other Toxic effects

These include gastrointestinal, hematological, endocrine, ocular, hepatic and neurological effects, anorexia, nausea,and occasional diarrhea has been reported with exposure levels ranging from 111 to 434 mg Cu/m3 over a 3-year period (ASDR, 2009). Also hepatomegaly

has been observed in workers exposed to copper dust; the exposure levels ranged from 111 to 434 mg Cu/m3 (ASDR, 2009).

Enlargement of the sella turcica, nonsecretive hypophyseal adenoma, accompanied by obesity, arterial hypertension, and "red facies" has been observed in workers exposed to 111–434 mg Cu/m3 as copper dust. They noted that there was a possibility that the clinical manifestations of hypophyseal adenoma or of Cushing's syndrome may have been the result of a disturbance of copper metabolism (Borak ***et al.***, 2000, ASDR, 2009)..

Workers exposed to copper dust have reported eye irritation. Headache, vertigo, and drowsiness have been reported in workers exposed to 111–434 mg/m3 copper dust (Borak ***et al.***, 2000).

2.7.3: Prophylaxis and management of copper poisoning.

Primary treatment in human toxicity- use of ammonium molybdate and 1g anhydrous sodium sulphate injection of ammonium tetrathiomolybdate, (EPA, 2005; ASDR, 2009)

2.8: MERCURY

Mercury occurs naturally in the environment and exists in several forms: metallic mercury (also known as elemental mercury), inorganic mercury, and organic mercury. At room temperature, some of the metallic mercury will evaporate and form mercury vapors. The higher the temperature, the more vapors will be released from liquid metallic mercury (Clarkson, 1989).

2.8.1:Toxicokinetics

Absorption is high (approximately 70–80%) for inhaled metallic mercury vapor, and negligible for oral exposure to liquid metallic mercury because of its high lipophilicity and be transferred readily through the placenta and blood-brain barrier (Bennett ***et al.***, 2008). The oxidation of metallic mercury to inorganic divalent cation in the brain can result in retention in the brain (Warfvinge and Bruun, 2000; Bennett ***et al.***, 2008).

Metallic mercury can be oxidized to inorganic divalent mercury by the hydrogen peroxidase-catalase pathway. The inorganic divalent cation can, in turn, be reduced to metallic mercury. The mercurous ion is unstable in the presence of sulfhydryl groups, and undergoes disproportionation into one atom of metallic mercury and one ion of mercuric mercury. For inorganic mercuric compounds, the low absorption in the lungs is probably due to the deposition of particles in the upper respiratory system that should be cleared rapidly (Wright ***et al.***, 1998; Clarkson ***et al.***, 2007).

The elimination of mercury can occur via the urine, feces, and expired air. Following exposure to inorganic mercury (mercuric), mercury is eliminated in the urine and feces. Organic mercury compounds are excreted predominantly via the feces in humans. In animals, methylmercury is excreted in the feces, and phenylmercury compounds are initially excreted in the feces and then in the urine. Organic mercury compounds are excreted predominantly in the inorganic form. Both inorganic mercury and methylmercury are excreted in breast milk (Warfvinge and Bruun, 2000).

2.8.2: Toxicodynamics

Respiratory Effects

Inhalation of metallic mercury vapor may result in clinical respiratory symptoms (e.g., chest pains, dyspnea, cough, reduced vital capacity. On more severe cases lead to respiratory distress, pulmonary edema, lobar pneumonia, fibrosis, desquamation of the bronchiolar epithelium, and death due to respiratory failure (Soni ***et al.***, 1992).

Cardiovascular Effects

Inhalation of metallic mercury may affect the cardiovascular system in humans, producing elevations in blood pressure and/or heart rate (Bluhm ***et al.***, 1992). Studies of workers chronically exposed to elemental mercury vapor have shown increased incidences of palpitations, high incidences of hypertension, and increased likelihood of death due to ischemic heart and cerebrovascular disease (Barregard ***et al.***, 1990; Bluhm ***et al.***, 1992).

Gastrointestinal Effects

Both inhalation and oral exposures to mercury have resulted in gastrointestinal toxicity. Mercurial stomatitis is a classic symptom of mercury toxicity and has been observed following inhalation exposure to both inorganic and organic mercury. Mercuric chloride is caustic to the tissues causing blisters, ulceration, hemorrhages and necrosis accompanied by shock and circulatory collapse throughout the gastrointestinal tract (Bencko *et al.*, 1990).

Hematological effects

Leukocytosis associated with a metal fume fever-like syndrome has been observed in persons exposed to high concentrations of metallic mercury vapor. Decreased hemoglobin and decreased δ-aminolevulinic acid dehydratase activity in erythrocytes or increased serum proteins involved in the storage and transport of copper in workers exposed to mercury vapor has been reported (Bencko ***et al.***, 1990).

Musculoskeletal effects

Increases in tremors, muscle fasciculations, myoclonus, or muscle pains are symptoms experience with high exposure to mercury (Bencko ***et al.***, 1990).

Hepatic Effects

Elevated serum glutamic pyruvic transaminase (SGPT), ornithine carbamyl transferase, and serum bilirubin, as well as evidence of decreased synthesis of hepatic coagulation factors. Similarly, hepatomegaly and hepatocellular vacuolation have been observed (Soni ***et al.***, 1992).

Renal effects

The nephrotic syndrome in humans associated with the ingestion, inhalation, or dermal application of mercury is primarily identified as protenuria , hematuria, oliguria, urinary casts, edema and inability to concentrate the urine. Hypercholesterolemia may also be observed (Soni ***et al.***, 1992)

Dermal effects

The predominant skin reaction is erythematous and pruritic skin rashes. Other dermal reactions characteristic of acrodynia include heavy perspiration (Bluhm ***et al.***, 1992).

Ocular effects

Mercury vapors cause red and burning eyes, conjunctivitis, and a yellow haze on the lenses of the eye. The yellow haze was associated with longterm occupational exposures (Bluhm ***et al.***, 1992).

Immunological effects

The human data are very limited, and only decreased IgG production has been observed in workers chronically exposed to metallic mercury vapor (Bencko ***et al.***, 1990).

Neurological effects

The elemental and methylmercury-induced toxicity and the specific neurotoxic symptoms include tremors, irritability, excessive shyness, confidence loss, and nervousness, insomnia, memory loss, neuromuscular changes, headaches, polyneuropathy and performance deficits (Bluhm ***et al.***, 1992).

Reproductive effects

Metallic mercury vapor does may cause an increase in the rate of spontaneous abortions. Inorganic (mercuric chloride) and organic (methylmercuric chloride) mercury decreased the percentage of motile spermatozoa in vitro (Bluhm ***et al.***, 1992; ASDR, 2009).

Developmental effects

Animal studies suggest that both inorganic mercury and organic mercury cause developmental toxicity. Metallic mercury vapor may be transferred across the placenta, suggesting developmental toxicity in offspring of mothers that ingest sufficient amounts of organic mercury (Barregard ***et al.***, 1990).

Genotoxic Effects

There is evidence showing that the induction of primary DNA damage in mammalian and bacterial cells and weak mutagenesis in mammalian cells suggests that mercury compounds has some genotoxic potential (Barregard ***et al.***, 1990; ASDR, 2009.).

2.8.3: Prophylaxis and management of Mercury poisoning.

Inhalation of metallic mercury should be treated with Intra venous hydrocortisone (a steroid) for pulmonary complications. Acute ingestion of mercury salts should be treated initially with induced emesis and gastric lavage. Oral administration of polythiol resins may also be useful as they bind mercury in the GI tract, which helps to prevent absorption into the body. Chelating agents which may be useful are dimercaprol, succimer and penicillamine (EPA, 2005; ASDR, 2009.)

Acute intakes of inorganic mercury should be treated with chelating agents, and dimercaprol and D- penicillamine are the most effective compounds. Chronic inorganic mercury poisoning

is best treated with penicillamine. Organic mercury poisoning should not be treated with the chelating agent dimercaprol as it may increase mercury levels in the brain;

Treatment in animals: Primary: sodium thiosulfate orally and parenterally, BAL by injection (EPA, 2005)

CHAPTER 3

MATERIALS AND METHODS

3.1 Study area

3.1.1 Geographical position

The research was conducted within Nairobi metropolis covering eight public and private hospitals, 2(two) research laboratories and 2(two) mortuaries that incinerates medical wastes, animal carcasses and pathological wastes. Nairobi is the capital and largest town in Kenya, it is located at 1°16S, 36°48′E and occupies 684 km² with an estimated urban population of between 3 to 4 million. It is situated at 1661 meters (5450 ft) above sea level. The majority of the populations are slum dwellers (CBS, 1999).

There are numerous hospitals and private clinics, laboratories and few mortuaries that are major sources of hazardous wastes to the neighboring residential areas.

3.1.2 Criteria of choice of study sites

A reconnaissance survey was conducted between May and July 2007 to investigate the characteristics of the sites: Type of wastes handled, amount of waste, disposal methods, incineration techniques, environmental impacts and morbidity associated with the distance of incinerators from residential areas. Twelve facilities with incinerators handling industrial, medical and municipal wastes were sampled. Using a ranking index the sites were profiled and chosen according to the level of overall impact based on the above criteria. The design of incinerators was considered based on criterion and good combustion practices set by EPA/NEMA. The hospitals were based on the profound environmental impact as results of

the quantity of the waste they generate and incinerate were majorly included in the sites, followed by research laboratories and mortuaries.

3.1.3 Ethical considerations

Before conducting the research permission was obtained from the Kenyas' national regulatory body- National Environmental Management Agency (NEMA). Prior consenting was done with respective respondents where the objectives of the study were elaborated and authorization was granted from administrative officers. Administrative permissions was sought from each study site with agreements of provision of the findings to the benefit of the public and research institutions. All the research was conducted according to institutional, national and international environmental regulations standards which are University of Nairobi, National Environmental Management Authority and EPA.

Due to confidentiality purposes and institutional protection laws all sites were coded accordingly to eliminate any unintentional conflict of interest as the results will be shared publicly. The facilities were therefore coded as; Hospitals (H), Research Laboratories (L) and Mortuaries.(M).

Figure 1: Standard height of an incinerators' chimney discharging fumes from study site (H3).

3.2 Data collection

3.2.1 Qualitative data collection

The population sample was obtained by random selection from eligible individuals living /staying near the incinerator sites and those living away from the incinerator sites as controls (about 10 Km away). 100 respondents were purposively selected based on age, sex, Occupation, Level of education and duration of stay near the study site in years who latter responded to the structured questionnaire on heavy metals sensitivity.

Prior consenting was done before preparation of semi-structured questionnaires at CPHR-KEMRI from pre-existing morbidity questionnaires which was adapted from UNEP. They were pretested and administered in two languages (English, Kiswahili) in a standardized format in Appendix (I). It had 20 questions on acute and chronic heavy metal toxicity. A hazard index was based on individuals scoring "yes" in five or more of the questions, which covered all major human body systems that is on attributable signs/symptoms of heavy metals toxicity. Face to face, interviews were used to verify the results of the questionnaires (Appendix (I))

To establish the esthetic quality of the study areas and to establish a basis for evaluating how study facts and figures correlate, visual assessment and field investigations were conducted in all study sites. Generated waste was also followed through the various management practices, and visual inspection and field investigations were similarly done. The assessment and investigations were also conducted to aid in the selection and design of the measurement procedure for the quantities generated and incinerated in each site

3.2.2 Quantitative data collection and Sample preparation

The sampling of bottom ashes was carried out in accordance with the recommendations of the International Ash Working Group (IAWG, 1997). Homogeneous steps were applied to the materials in order to derive representative laboratory samples. Bottom ash samples were collected from the selected sites on three weekly different visits from October to December 2007. About 5 kg of ash was collected and dried at room temperature for 5 days. All the visible metals and glass objects were removed; ash samples were pulverized and passed through a mesh sieve. The ash was then made into fine powder using a mechanical grinder. Whole samples were mixed properly using a blender before analysis.

3.3 Heavy metal analysis

The collected samples were analyzed for a multi-element suite including, Arsenic (**Ar**), Cadmium (**Cd**), Cobalt (**Co**), Copper (**Cu**), Chromium (**Cr**), Mercury (**Hg**) and lead (**Pb**) by Flame Atomic Absorption Spectrophotometry.

3.3.1 Digestion of samples

A half a gram (0.5 g) of sample were weighed into a glass 100 ml boiling tube and to this 10 ml of deionised water was added, followed by 7.5 ml of concentrated hydrochloric acid (HCl) and 2.5 ml of concentrated nitric acid (HNO_3). The samples were digested at room temperature overnight prior to being placed onto a Gerhardt Kjeldatherm digestion block connected to a Gerhardt Turbosog scrubber unit (filled with 10% w/v sodium hydroxide). The samples were then refluxed at 130 °C for four (4) hours. After cooling to ambient temperature,

the digests were filtered into volumetric flasks, diluted with deionised water, made up to a volume of 50 ml and mixed.

3.3.2 Calibration curve and standardization of sample.

Accuracy of the method was calculated by preparing samples containing the same quantity of placebo/ blank, all the components (trace elements in a sewage sludge amended soil) except the analyte to be determined as the real sample. All were prepared in 15% v/v hydrochloric acid and 5% v/v nitric acid. Mean recoveries (% *R*) of each element for triplicates prepared samples with identical spike ranged from 98.9 to 100.8%.

The detection limit was calculated as $[(Y_b+3\ \text{S.D.})-b]a^{-1}$ and the quantification limit as $[(Y_b+10\ \text{S.D.})-b]a^{-1}$, where Y_b is the mean of the blank signal, S.D. is the standard deviation of the blank signal, b is the intercept of the calibration straight line, and a is the slope of the calibration straight line (Miller J.C and Miller J.N, 1993).

3.3.3 Atomic Absorption Spectrophotometry (AAS)

This was conducted at the department of geology and mines in Nairobi. Following preparation, samples were analyzed by AAS (Perkin Elmer Elan, 6000). The technique of flame atomic absorption spectroscopy (AAS) requires a liquid sample to be aspirated, aerosolized, and mixed with combustible gases, such as acetylene and air or acetylene and nitrous oxide. The mixture was ignited in a flame whose temperature ranged from 2100 to 2800 °C. During combustion, atoms of the element of interest in the sample were reduced to free, unexcited ground state atoms, which absorb light at characteristic wavelengths. The

characteristic wavelengths are element specific and accurate to 0.01-0.1nm. To provide element specific wavelengths, a light beam from a lamp whose cathode is made of the element being determined is passed through the flame. Reproducibility of the method calculated after analysis of samples by the same analyst in different days (day-to-day fluctuation) and by two different analysts in different days (analyst-to-analyst fluctuation) gave relative standard deviation of 1.1 and 1.6%, respectively (Abarca ***et al.***, 2001).

3.4 Data and Statistical analyses

The data collected by questionnaire survey were analysed mainly with simple descriptive statistics; while the qualitative mode of analysis was mainly narrative. Completed questionnaires were screened daily for completeness and appropriateness of data. All qualitative data was first cleaned manually, coded and entered into Microsoft Excel ® (Microsoft Corporation 2007) for storage and later analysis.

Analyses were done by use of descriptive statistics analysis including, cross tabulations, graphs and linear regression (correlation). Qualitative data was compiled according to sources, analyzed and summarized under categories descriptively by use of the analysis outline. All statistical analyses were carried using Statistical Package for Social Science (SPSS v 12).

CHAPTER 4

RESULTS

4.1: Qualitative data

4.1.1: Morbidity and toxicology of systems.

The prevalence of symptoms according to body systems varied largely over the study population. The systemic of toxicity ranged from musculosketal system (21%), Dermatological (19%), Respiratory (18%), Gastrointestinal (13%), Cardiovascular (11%) Reproductive (9%) and Nervous (9%) systems respectively (Figure 2).

Signs of heavy metals toxicity were observable in most of the informants who scored 1 (≥5 signs of the morbidity questionnaire) see Appendix 1. A resident who had recently migrated near the study sites or had stayed for less than 5 years had less significant toxicity scores compared to those who had stayed for more than five years.

4.1.2: Socio-demographic characteristics.

One hundred (100) informants (63 males and 37 females) were interviewed(Appendix II). 52.4% (33) of male scored 1, while 47.6% had less significant toxicity index. 56.7% of females had significant toxicity index, while 43.3% scored 2.

The effect of heavy metal poisoning was significant among the respondents residing within (≥1≤ 10Km) of the incinerator sites ($p<0.005$). The population residing far away from the sites was less diseased. There were more evident chronic signs in older individuals > 45 years.

The level of education and occupation of respondents influenced their understanding of the potential hazards of incinerators. Primary level (27%) and uneducated respondents (5%) lacked the awareness of the potential toxicity of dispersions and effluents from incinerators. An equal number of respondents (34%) who had secondary and tertiary education had significant knowledge on the risks of heavy metals from the incinerators.

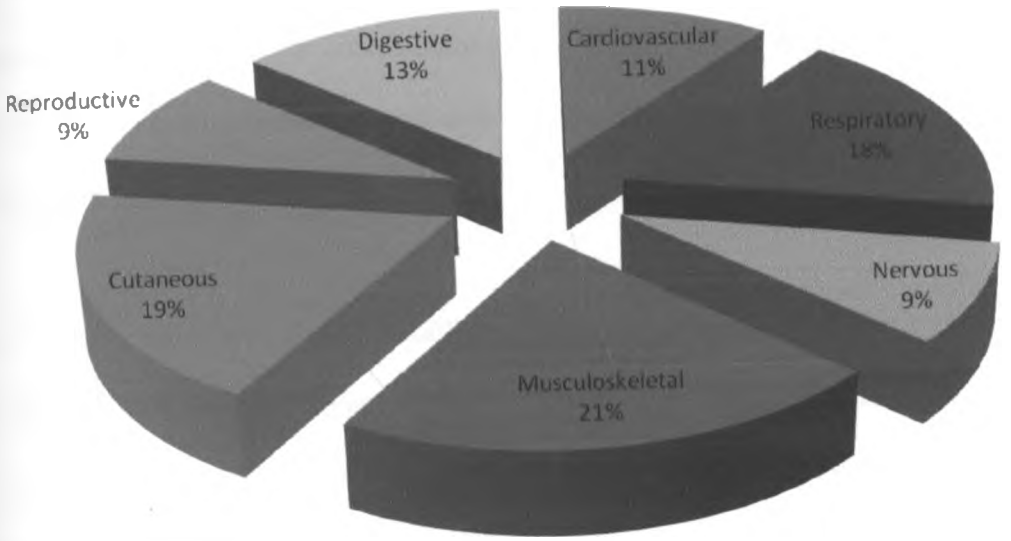


Figure 2: Percent distribution of systemic toxicosis

4.1.3: Ecotoxicology and environmental impact of heavy metals.

There was general poor growth and discoloration of vegetation which ranged from white brownish – yellow/red to others being black, the edges of the leaves exhibited a similar coloring near the incinerators with lower forms mostly affected due to soil contamination by fly ash dispersions and bottom ash leachates.

It was also evident that degradation of soil quality and texture near the sites due to contamination. Atmospheric contamination was evident due to dissipation of smoke clouds during the hours of incinerator operation in almost all the sites where the incinerator has been in operation for more than four years.

4.1.4: Ash handling and disposal methods

All sites followed the dry disposal technique. A major portion of the bottom ash in most of the facilities were collected as dry matter. This study documented that most of the incineration residues from both hospitals and research laboratories were openly dumped at sites close to the incinerators (Figure 3).

Both hospitals and research laboratories had openfills that were being used to dump all of the wastes and ashes. The dumping sites were located a few meters from the premises or within the sites perimeter fences.

Figure 3: Ash mounds disposed near incinerator at site L2

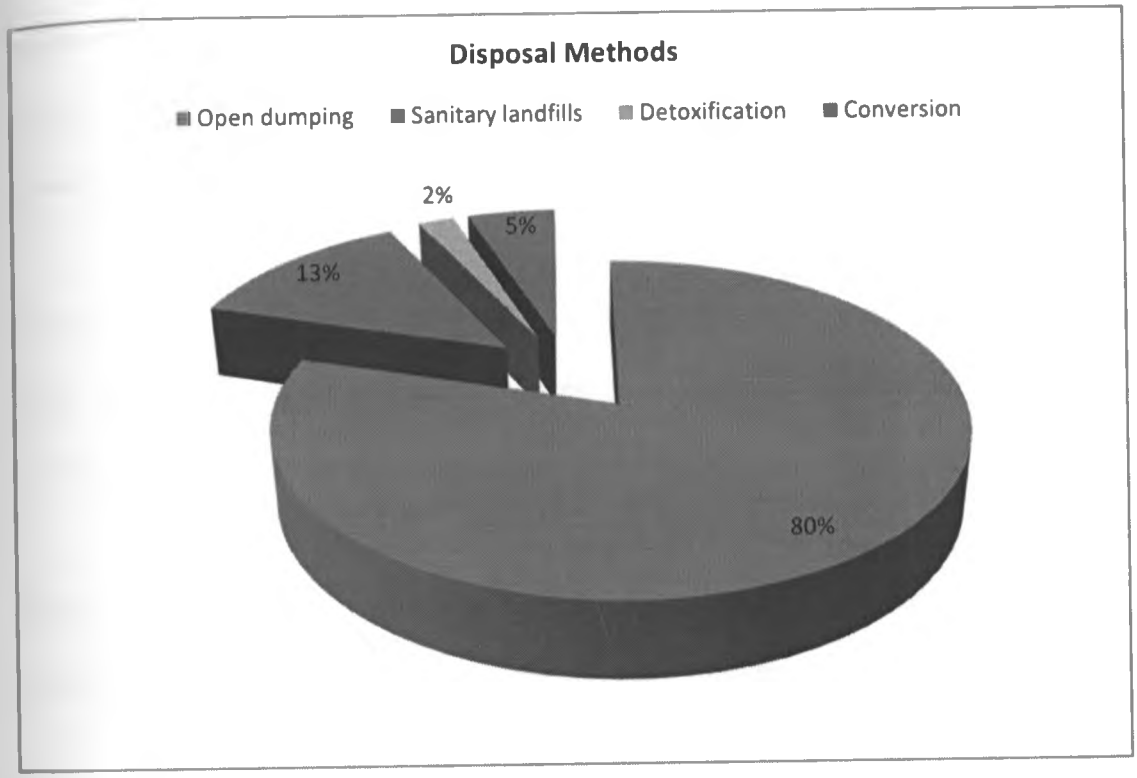


Figure 4: Modes of waste disposal in the study sites.

4.1.5: Types of wastes

Table1 gives the classification of the waste generated at the eight hospitals, two research laboratories and the mortuaries. Waste were classified as general, medical and sharps. General waste which include packaging materials such as cardboard, office paper, leftover food, cans that do not pose any immediate danger to humans or the environment. Pathological waste contained tissues, organs, placentas and other body parts especially from mortuaries.

Infectious waste contained "pathogens" in sufficient quantity that when exposed to it could result in diseases. Examples; culture plates, drainage bags, surgical and theatre wastes, contaminated plastic items etc.

Sharps were anything that could cause a cut or puncture leading to wound. Items like needles, syringes, scalpels, knives, broken glass, etc. form part of sharp wastes.

Hospital waste was categorized as any waste that is produced from healthcare facilities such as general hospitals, medical centres, medical laboratories or animal hospitals. This therefore includes both non-hazardous and hazardous waste constituents.

Type of wastes	Toxicology	Composition	Sites
General		Packaging materials (mostly cardboard), office paper, leftover food, cans, plastics bags and containers, etc.	All
Medical	Infectious waste	Clinical specimens, culture plates, drainage bags, surgical waste, autopsy waste, blood, blood products and body fluids	Hospitals Laboratories
Medical	Pathological waste	Human tissues, organs, fetuses, placentas, amputated body parts and other body parts	Hospitals Mortuaries
Medical	Solid chemicals and pharmaceutical waste	Spilled or expired drugs and chemicals	All
Sharps		Needles, syringes, blades, broken glass, scalpels etc.	Hospitals Laboratories

Table 1: Classification of the different types of wastes generated and incinerated at the study sites.

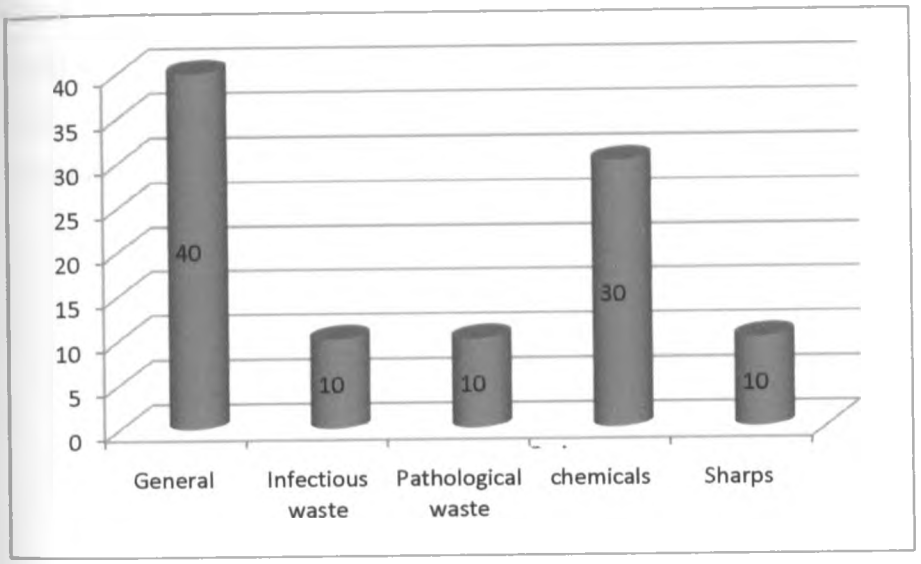
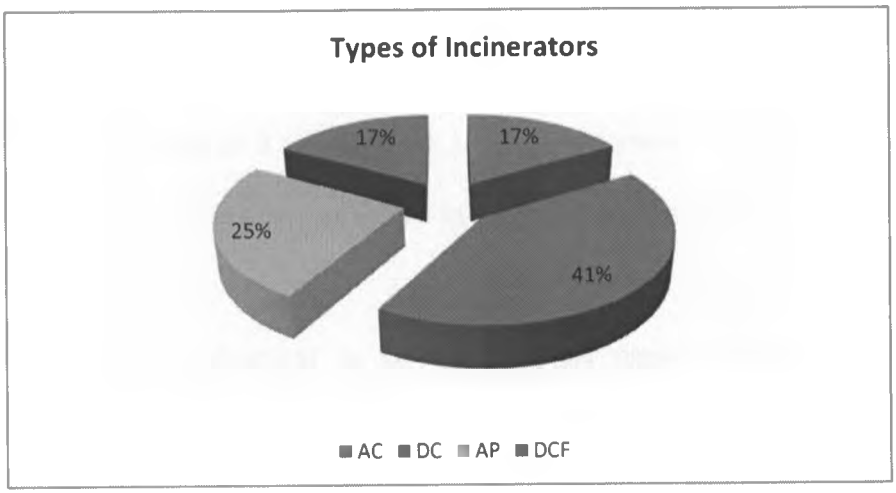


Figure 4: Percent distribution of the different types of wastes generated and incinerated at the study sites.

4.1.6: Incinerator designs and types

Most of the incinerators used in these facilities were refractory furnace type and the other type was a rotary kiln type of incinerators. Chimneys of most incinerators studied were short and didn't required height as shown in figure 1. The temperature range of most incinerators was observed to be fluctuating at lower ranges depending on the condition in which the incineration was carried out. The maximum temperature at which most waste was incinerated was between 600^0C to 900^0C. Only few incinerators had their maximum temperature in the combustion chamber set to 1600^0C.

Most incinerators operated 2-4 hours per day. Some were only used after it get fill up (3 days a week) and some incinerators are operated during night. Incinerators used in these facilities were Autocombustion incinerators, Double combustion incinerators, Air propulsed incinerators and Double combustion with filtration incinerators as shown in figure 4 below.



AC = Autocombustion incinerators
DC= Double combustion incinerators
AP = Air propulsed incinerators
DCF = Double combustion with filtration incinerators

Figure 5: Percent distribution of designs/types of incinerators from the sites.

4.2: Heavy metal composition

The data contained in figure 5 indicates the concentration of heavy metals in bottom ash from all the study sites. Mean concentrations of heavy metals (mg/kg) were Hospital 3 (70.3) >Hospital 5 (70.2) >Hospital 6 (70.0) >Hospital 7 (65.0) >Hospital 4 (64.5) >Lab 1 (62.0) >Hospital 8 (60.0) >Hospital 2 (55.8) >Lab 2 (54.3) >Mortuary 1 (42.8) >Hospital 1 (40.5) and Mortuary 2 (29.0) respectively as shown in (Figure 4 and Table 2).

Mercury was the least abundant in all the samples, present within the range of 11–58(31mg/kg). Considerable variation was found in the heavy metal concentration between the twelve different facilities.

Ash samples from hospital H3 (70.3 mg/kg) had the highest cumulative mean concentration of heavy metals, followed by H5 (70.2) >H6 (70.0) >H7 (65.0) >H4 (64.5>H8 (60.0) >H2 (55.8) and H1 (40.5) respectively (Figure 5).

Chromium (95mg/kg) had the highest bottom ash mean concentration in all the sites followed by Cu (62mg/kg) >Pb (60mg/kg) >Cd (58mg.kg) >Ar (35mg/kg) and Hg (31mg/kg) respectively (Figure 6 and Table 2).

There was variable distribution of mean element concentrations in all the sites. Arsenic, Cadmium and Chromium had evident Gaussian distribution while Lead, copper and Mercury had skewed distributions ($P<0.05$) (Figure 7).

85% of the facilities did not maintained record of waste generated and waste production for incineration ranges from 200 to 1400 kg/day in 60% of the facilities who were keeping records. And regarding waste disposal guidelines Seventy percent (70%) of the facilities did not have it, while 30 % had it, while 50% of the facilities were found to be giving out various protective gears to the incinerator operators.

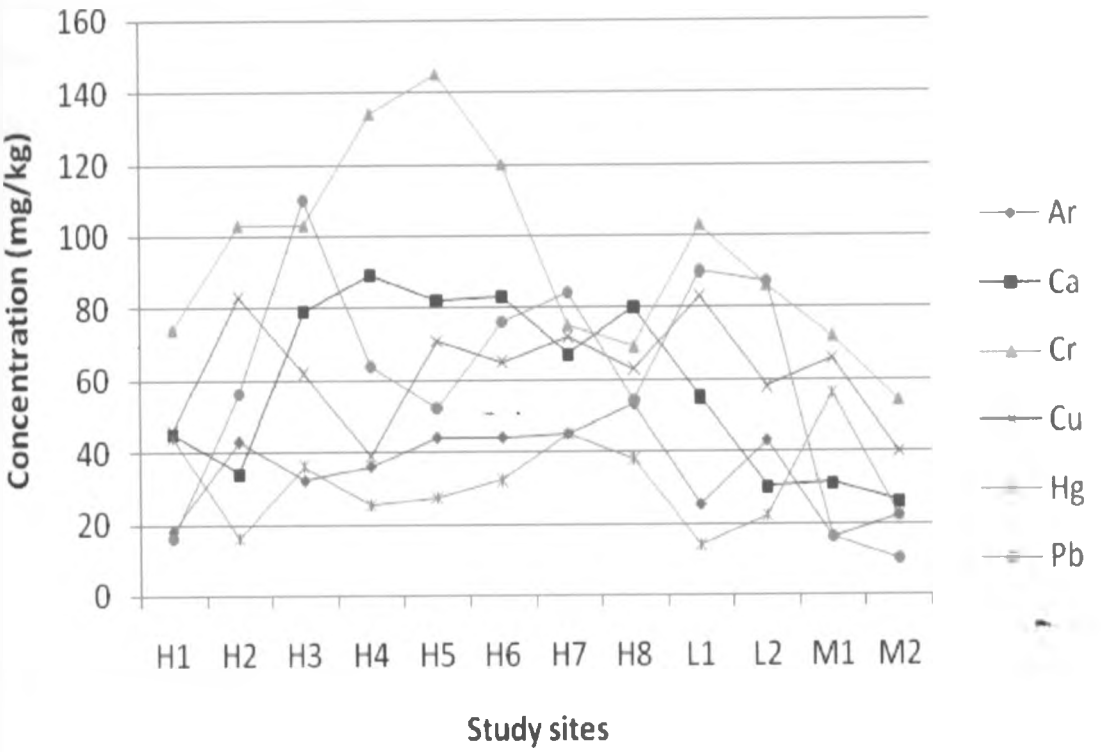


Figure 6: Comparison of concentration of elements in bottom ash from the twelve (12) sites.

Research laboratories had moderate detectable levels of heavy metals with L1 (62.0) > L2 (54.3), while mortuaries had the lowest detectable concentrations M1 (42.8) > M2 (29.0)

One sample t- test (ANOVA) analysis revealed that heavy metals concentration (toxicity) were highly significant across all study sites. Chromium had the highest concentrations t=11.718, mean difference (MD) = 94.83mg/kg, CI (77.0206-112.6460) at p <0.05(Table 3)

Copper had the lowest concentrations but showed greatest variation among the sites, t=2.819, mean difference (MD) = 95.67 mg/kg, CI (20.9852- 170.3481) at P<0.05(Table 3)

Mercury had the smallest variation among all the sites, t = 8.529, mean difference (MD) = 31.42mg/kg, CI (23.3095-39.5238).

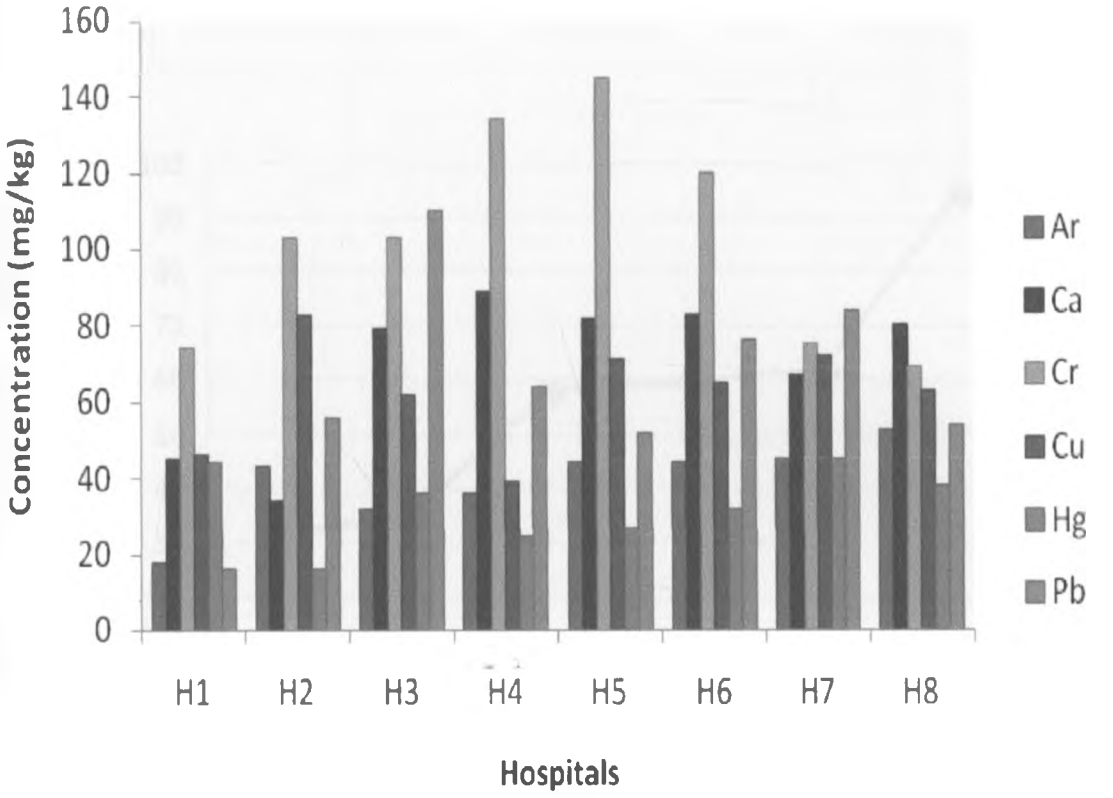


Figure 7: Comparison of concentration of elements in bottom ash from the hospital incinerators.

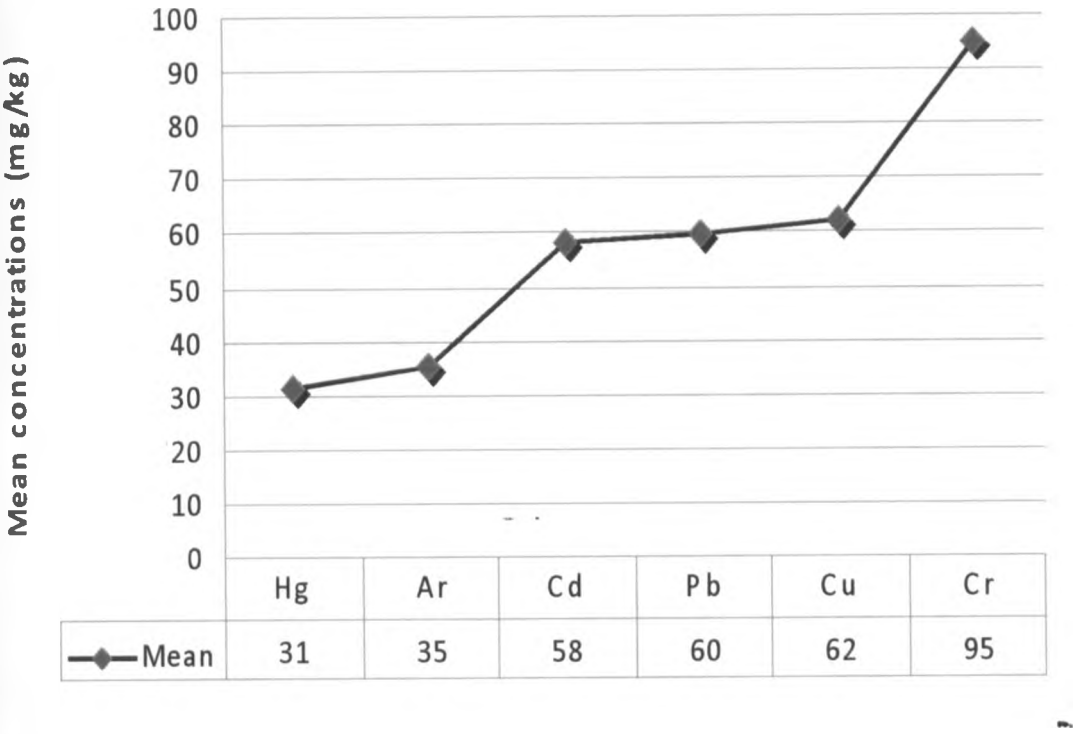


Figure 8: Elemental mean concentrations in mg/kg from all the sites.

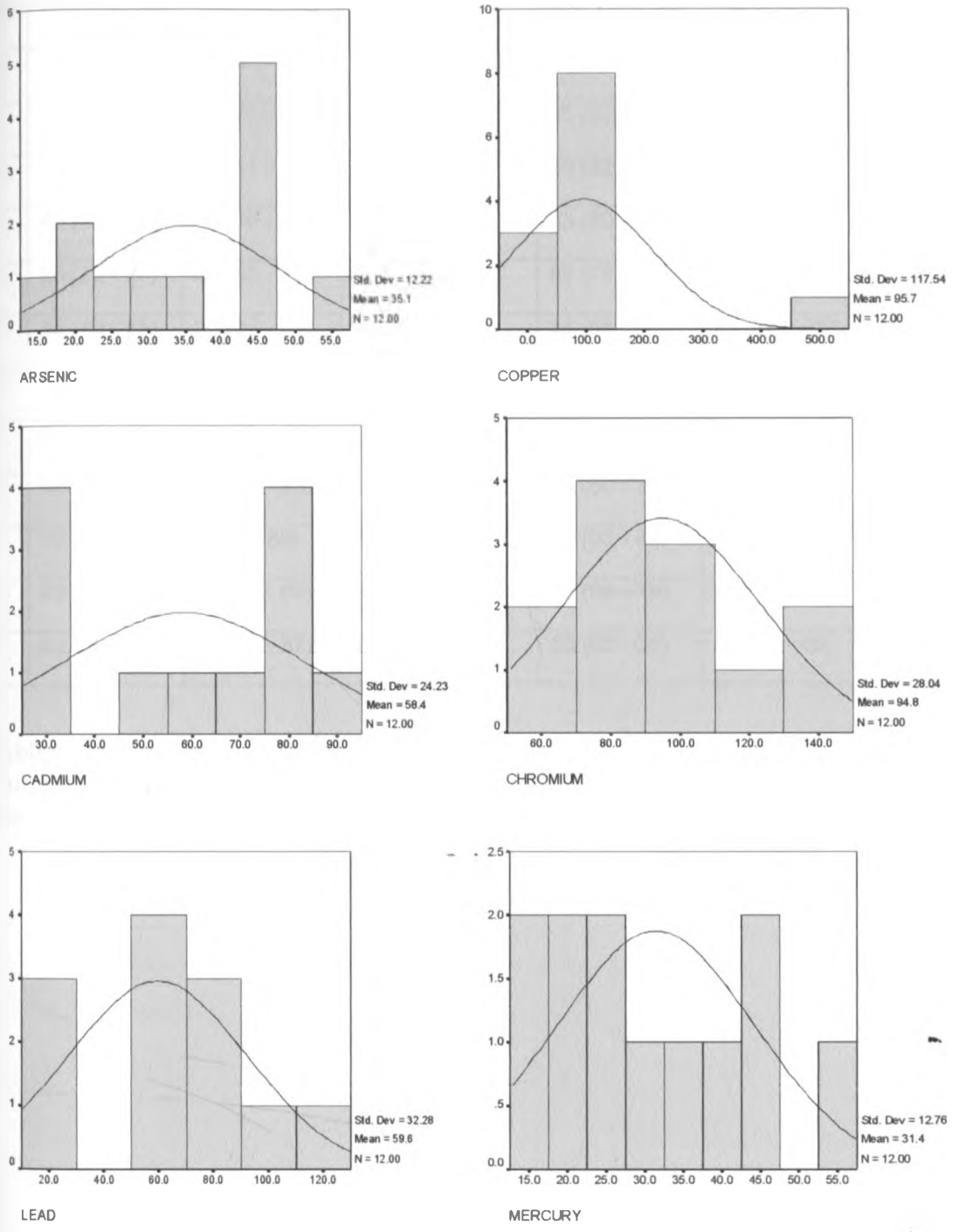


Figure 9: Normalized distribution plots/curves of mean element concentrations ±SD over the sites

Sites	Ar (mg/kg)	Cd(mg/kg)	Cr (mg/kg)	Cu (mg/kg)	Hg (mg/kg)	Pb (mg/kg)
H1	18 (17- 19)	45 (37 - 50)	74 (70 -76)	46 (45 - 48)	44 (41 - 47)	16 (11 - 19)
H2	16 (13 - 20)	31 (25 - 33)	72 (70 - 74)	65 (62 - 68)	56 (53 - 58)	16 (14 - 18)
H3	22 (15 - 26)	26 (21 - 29)	54 (50 - 56)	40 (32 - 45)	22 (18 - 25)	10 (6 – 15)
H4	43 (42 - 45)	34 (30 - 36)	103 (98 - 111)	83 (80 - 85)	16 (14 - 18)	56 (55 – 58)
H5	25 (23 - 27)	55 (50 - 58)	103 (95 -109)	83 (79 - 88)	14 (11 - 16)	90 (87 - 94)
H6	43 (40 - 45)	30 (27 - 35)	86 (82 - 92)	58 (55 - 60)	22 (17 - 25)	87 (81 – 91)
H7	32 (30 - 34)	79 (74 - 85)	103 (97 - 115)	62 (57 - 65)	36 (28 - 42)	110 (95 - 120)
H8	36 (35 - 38)	89 (88 - 90)	134 (129 - 135)	39 (35 - 42)	25 (22 - 30)	64 (62 – 68)
L1	44 (41 –46)	82 (79 - 88)	145 (138 - 149)	71 (60 - 78)	27 (21 - 33)	52 (50 – 54)
L2	40 (38 - 42)	83 (8 - 86)	120 (118 - 126)	65 (63 - 68)	32 (29 - 34)	76 (70 – 80)
M1	45 (44 -46)	67 (64 - 69)	75 (70 - 79)	72 (68 - 75)	45 (43 - 47)	84 (83 – 86)
M2	53 (50 -55)	80 (75 - 87)	69 (64 - 72)	63 (60 - 65)	38 (31 - 46)	54 (50 - 56)

Table 2: Mean concentration of elements in bottom ash from twelve incinerator facilities in Nairobi.
Key: H = Hospital; L= Research Laboratory; M= Mortuary

Metals	Detection limit (EPA/NEMA)	Mean[a,b] ± S.D[c]	Max
Ar	20	35 ± 12	53
Ca	10	58 ± 24	89
Cr	50	95 ± 28	145
Cu	50	62 ± 15	83
Hg	10	31 ± 13	56
Pb	30	60 ± 32	110

[a] Detected concentrations only.
[b] Arithmetic mean.
[c] Arithmetic SD.

Table 3: NEMA elemental detection limits (mg/kg)

		Test Value					
		t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
	Std. Deviation					Lower	Upper
Arsenic	12.22113	9.944	11	.000	35.0833	27.3184	42.8483
Cadmium	24.22793	8.352	11	.000	58.4167	43.0230	73.8104
Cobalt	24.22793	8.352	11	.000	58.4167	43.0230	73.8104
Chromium	28.03515	11.718	11	.000	94.8333	77.0206	112.6460
Copper	117.54019	2.819	11	.017	95.6667	20.9852	170.3481
Mercury	12.75973	8.529	11	.000	31.4167	23.3095	39.5238
Lead	32.28132	6.394	11	.000	59.5833	39.0728	80.0939

Table 4: Statistical output of the T-tests and analysis of variance (ANOVA) of elements in bottom ash

CHAPTER 5

DISCUSSION

5.1: Disposal methods and potential eco-toxicological impacts

Disposal methods that are employed in different study sites are major contributors to environmental contamination. The current study revealed that land filling is still the main management option for bottom ash disposal (BA) in Kenya concurring with Thompson and Anthony, (2005). This poses a potential health risk to humans and animals due to soil and groundwater contamination (Bipp ***et al.***, 1998; Bruder-Hubscher ***et al.***, 2002).

Out of the twelve (12) facilities studied, they managed their waste by burning it in different kind of incinerators. Two different classes of waste incineration practiced involved: class 1 (Uncontrolled batch type combustion, no Air Pollution Control Devices) and Class 2 (Controlled, batch type combustion, no or minimal Air Pollution Control Devices), reflecting that the incinerators are comparatively polluting types. Most of them apply the uncontrolled batch type combustion, which is estimated to contribute 97.33 % of the *total emission per annum*.

Bottom ashes are toxic, but none of the facilities were found to bother about the toxic ash they produced through incineration treatment option. The ash management ranges from Open dumping being widely used and has long been recognized as a potential source of public health and environmental problems. Impacts to human health is substantiated by the fact that open dumping of waste releases toxic substances into the soil and groundwater and other eco-system. Impact associated to carcinogen emission as a result of heavy metals is higher in open

dumping,Wind easily blows over the dumped waste, dispersing these pollutants to nearby communities having a direct influence (Morselli ***et al.***, 2008).

Dry collection and disposal of bottom ash in form of ash mound not only saves the use of land and water but minimizes the chances of water pollution, by minimizing leaching. However proper measures like lining at the bottom of the ash mound, stabilization and covering of the completed mound is indeed paramount to minimize environmental damage. In this study it was apparent that all the dumping sites lacked lining and the reason for this was majority of these facilities point out the cost of putting it as the issue. NEMA generally accept landfill as the final option of resting hazardous solid waste but it was not being applied in most of the facilities.

With regard to the existence of waste disposal guidelines in these facilities, Seventy percent (70%) revealed that they hadn't waste disposal guidelines while 30 % hospitals had waste disposal guidelines. In response of knowledge about the existence of any guidelines on medical waste disposal (WHO, MOH), 70 % of the facilities (8 out of 12) weren't aware existence of the guidelines. However, 30 % (i.e. 4 out of 12) were aware of Waste disposal Guidelines. This shows a gap in personnel regarding waste disposal guidelines in place. In regard to awareness about relevant legal provision (national and international) of disposal of ash; 40% of the facilities under the study weren't aware of any regulation in place on incineration while 60% were aware of NEMA regulation.

The study also revealed that most facilities' personnel receive no formal training and are unaware or misinformed about many aspects of medical waste management. Some believe it is safe to burn mercury and any plastics (including PVC) in the incinerator. Others mistakenly believe that the law requires all waste from their facilities be burned. Others have been told that the incinerator is smoke-less, that it does not require a permit to operate. None are aware of potential adverse health effects of incinerator emissions or ash.

The presence of the incinerator undermines good waste segregation and minimization practices. As the field investigation confirms, the incinerator becomes a convenient storage chamber to accumulate waste and is used to burn all wastes. Moreover, because these incinerators are erroneously promoted as smoke-less and heavy metals-free, some facility staff assume that regulations do not apply, hence undermining enforcement of environmental laws.

Few of these facilities had in-house training and instruction from the senior staffs on incineration. Some of the personnel's had participated in waste management training/workshops. 50% of the facilities were found to be giving out various protective gears to incinerator operators e.g. mask, gloves, apron, clothes, boots but majority of these personnel were not using the devices. In addition to these protective gears, non of the facility vaccinate personnel against hepatitis and tetanus.

It was also evident in this study that few of the facilities had well maintained records of waste generated and incinerated representing 45%, while 55% didn't maintain records and hence

exact volume/weight of waste could not obtained during the study. However, waste incinerated ranges from 200 to 600 kg/day.

In terms of wastes being incinerated, almost all kinds of waste viz: needles, syringes, surgical waste, lab waste, sharps, pathological wastes, gloves, infectious waste, infected body parts. All the facilities were found to incinerate plastic, plastic bottles, syringes, gloves, tubes vaccine campaigning syringes, plastic sheets. It is very important to note here that, the syringes, gloves, tubes are made of PVC plastics which is favorable to produce Dioxin and Furan and some heavy metals. The mean percentage composition of the waste was found in the following decreasing order: General 40%, infectious waste10%, Pathological waste 10%, chemicals waste 30% and Sharps 10%.

Most sixty percent (60%) of the facilities revealed they had receive community complains regarding incineration operation while forty percent (40%) had not experienced community complain. The community complains was nuisance from incineration smoke. One facility were planning to relocated its incineration due to community complain. Some were in the process of replacing the smoky brick kiln types of incinerators with comparatively better to reduce smoke emission, while some facilities were operating at night to avoid the community complain.

In sitting of incinerators, 7 out of 12 facilities didn't consider siting as a major factor when they were constructing, most of these incinerators were located near residential facilities and this is a major contributor to the contamination of soils, air and water bodies as it was

evidence that there was general poor growth and discoloration of vegetation near the incinerators with lower forms mostly were affected due to soil contamination by fly ash dispersions and bottom ash leachates. There was degradation of soil quality and texture near the sites due to soil contamination. Atmospheric contamination was evident due to dissipation of smoke clouds during the hours of incinerator operation.

Leaching is the most likely path by which bottom ash constituents would become mobile environmental contaminants. Sitting is recommended not close to tall buildings and in an area free from air turbulence, Positioning within buildings needs appropriate flue arrangements with adequate ventilation but these facilities did not meet all these requirements.

5.2: Incinerator designs

Proper design and operation of incinerators should achieve desired temperatures, residence times, and other conditions necessary to destroy pathogens, minimize emissions, avoid clinker formation and slagging of the ash (in the primary chamber), avoid refractory damage destruction, and minimize fuel consumption. It appears that the temperature, residence time and other recommendations were rarely achieved by these incinerators.

A lot of bottom ash was generated in most facilities due to incomplete combustion of wastes, Poor incinerator designs determined the amount and quality of ashes released. Additionally, few facilities utilize air pollution control equipment. This study indicated visible smoke from the stack; smoke emission from the chamber doors and air inlets; commingling of sharps and non-infectious waste, despite some source segregation; large quantities of unburned materials

(sometimes plastics, syringes, glass, paper and gauze) in the ash; deficient ash disposal practices as seen in figure 3. Height of at least 4 – 6 m high chimneys needed for both adequate dispersion plus draft for proper air flow, but most of the chimneys studied were less than 4m high in height thus not meeting the required standards as in figure 1 and, depending on wind direction, emitted gases might have been dispersed to nearby communities being a potential cause of systems toxicities as evident by the prevalence of musculosketal system (21%), Dermatological (19%), Respiratory (18%), Gastrointestinal (13%), Cardiovascular (11%), Reproductive (9%) and Nervous (9%) systems respectively. Therefore, community living near the open landfill therefore acts as passive samplers through inhalation of polluted air.

The effect of heavy metal poisoning was significant among the respondents residing within ($\geq 1 \leq 5$KM) of the incinerator sites ($p<0.005$). The population residing far away from the sites was less diseased. There were more evident chronic signs in older individuals > 45 years, because of age and time – exposure related toxicity and cumulative effect of heavy metals.

The level of education and occupation of respondents influenced their understanding of the potential hazards of incinerators. Both primary level (27%) and uneducated respondents (5%) lacked the awareness of the potential toxicity of dispersions and effluents from incinerators. An equal number of respondents (34%) who had secondary and tertiary education had significant knowledge on the risks of heavy metals from the incinerators.

The high population growth in Nairobi area has led to residential homes being built close to the landfill sites and incineration sites. Depending on the wind direction, when the waste is burned, smoke reaches these homes, dispersing toxic air pollutants.

Most of the incinerators used in these facilities were Autocombustion incinerators, Double combustion incinerators, Air propulsed incinerators and Double combustion with filtration incinerators.

5.3: Temperature range of Incinerator and actual operation practice

Incinerators for combusting hazardous wastes of various industries and clinical wastes, desires high temperatures for the complete destruction of organic matter to minimize the formation of toxic organics during incineration, but they also enhances the metal volatility, resulting in an increase in the release of volatile metallic compounds, which could be toxic to the atmosphere. As seen in study site H1, H3 and H6, incinerators had a capacity to burn the waste upto 966 °C, But while operating these incinerators, temperature is set between 400-600°C. In H2, M1 and M2 the temperature in chamber is set in 700°C. The actual operation temperatures in these chambers were observed to be fluctuating at lower ranges depending on the condition in which the incineration was carried out. The maximum temperature at which waste was incinerated in H4, H5 and L2 were set at 966°C. But the control panel is set to incinerate waste at 600°C in primary chamber and 1600°C in secondary chamber. The incinerators at H7 and H8 at 600°C were all set at 900°C in while incinerating the waste. The maximum temperature the combustion chamber was heated on only 600°C in the incinerator at L1. The temperature range of 200–600°C is the most suitable condition for greater formation of emission of environmental pollutants. Thus most of the incinerators operated in these facilities were found not meeting the required temperature to completely incinerate the waste feed into them.

5.4: Operation hours of Incinerator

Incinerators must be fully heated up before wastes are added, requiring about 30 min or longer, depending on ambient temperature, type of fuel, fuel moisture content, etc. However, most of the 12 facilities surveyed were not being operated in this fashion, rather, were loaded prior to lighting, Destruction of organic compounds and microorganisms in incinerator off-gas is related to residence time. Secondary chamber residence time is the biggest factor in achieving low emission levels. A minimum residence time of 2 seconds at high temperatures is a common standard.

Sixty percent (60%) of the incinerators are mostly operated 2-4 hours per day. Some were only used after it get fill up (3 days a week). Some incinerators are operated during night time to avoid public complain and conflict.

5.5: Toxicology of body systems

The body systems toxicity index was variable according to the different toxicokinetic and toxicodynamic profiles of the elements. There was an association between the age of informants and toxicity score, suggestive of time-exposure dependence. There was a positive correlation between distance of stay from the incinerators and the magnitude of exposure; the results strongly suggest the eco-toxicological potential of the heavy metals since heavy metals tend to bioaccumulate and this means an increase in the concentration of a chemical over time, compared to the chemical's concentration in the environment. Compounds accumulate in living things any time they are taken up and stored faster than they are broken down (metabolized) or excreted.

The high population growth in Nairobi area has led to residential homes being built close to the landfill sites and incineration sites. Depending on the wind direction, when the waste is

burned, smoke reaches these homes, dispersing toxic air pollutants. Dermatological signs were suggestive of the direct exposure due to dispersal of fly ashes and use of contaminated waters.

Studies of metal exposure have shown that women often have higher blood levels of metals than men in the same families or communities. The mechanisms are not clear. Nor are such data typically corrected for hematocrit or albumin level. If the higher blood levels influence body burden or half-life, they would reflect susceptibility, but at present I found no studies to clarify this.

Many studies shows that metals behave differently in males and females of various species for instance in some studies women have shown to have higher levels of metals in hair than in men but this likely reflect a protective mechanism (increased excretion) as a susceptibility (increased absorption) and this can also be discussed that in this study females had high significant toxicity index than males.

As a general rule immature humans, are more susceptible to metal toxicity than mature animals, and elderly often shows heightened susceptibility. Immature humans often have much greater uptake (e.g., 50% for lead in the immature intestine vs 10% for adults), and their organs may be more vulnerable as well. This is especially true of neurotoxic chemicals that can produce significant impairment in the developing nervous system.

Metallothionein levels vary with age in which most organs have high levels that decrease to adult levels. Other studies also have shown that heavy metals intakes vary with age, infants

having higher levels than older people in the same family or community as it was evident in this study that there were chronic signs in older individuals > 45 years than in those who middle age groups. Whether Metallothionein are protective or not, it may influence the response of any organ to a heavy metal.

The effect of heavy metal poisoning was significant among the respondents residing within (≥1≤ 5Km) of the incinerator sites ($p<0.005$). The population residing far away from the sites was less diseased. The level of education and occupation of respondents influenced their understanding of the potential hazards of incinerators. Primary level (27%) and uneducated respondents (5%) lacked the awareness of the potential toxicity of dispersions and effluents from incinerators. An equal number of respondents (34%) who had secondary and tertiary education had significant knowledge on the risks of heavy metals from the incinerators.

The bioavailability and eco-toxicity of metals mainly depends on their speciation in matter. Heavy metals that are distributed in acid soluble/exchangeable fraction and reducible fraction are readily to be absorbed in plants or in water system causing pollution. Cadmium, copper lead, mercury and arsenic ions, dissolved in water easily hence easy for these elements to cause systemic toxicosys or to provoke systems damage. They are associated with carcinogenic and toxic substances in the environment in a long perspective in places where bottom and fly ashes are disposed (ASDR, 2009).

5.6: Total elemental concentration in bottom ash

Statistical analysis showed that there was significant difference in total concentration of metals in all the incineration plants. This different in total concentration is as a result of different in combustion temperatures which is major parameter determining the metals volatility, since the extent of evaporation of the metals and metal compounds in the furnace is directly related to combustion temperature. It is believed that operating temperature in an incinerator should be higher than 850 ◦C, however such high temperatures increase volatilization of hazardous metals and lead to adverse environmental pollution (Wei ***et al***, .1998), the findings of this study clearly demonstrated that the site with incinerators with lower combustion temperatures had high ash metals content. Mean concentrations of heavy metals (mg/kg) in all the study sites were high as it is in H3 (70.3), H6 (70.0), H7 (65.0), L1 (62.0) and H8 (60.0) respectively.

Lower kiln temperatures causes all the metals except Pb, the most volatile one, to be retained mostly in bottom ash. Therefore, it could be said that the higher the amount of waste, the larger the fraction of metal that is retained in bottom ash at high temperatures during incineration process as seen in H4, H5,H6, H7 and H8. It is also evident in this study that Phases and possible secondary reactions of these compounds during combustion could have a significant role on metal partitioning as seen in H4, H5 and L2 where temperature was set approximately at 1600°C while incinerating the wastes which relate to Bakoglu ***et al***.,2002 findings.

Difficulties related with the incineration of solid wastes (poor mixing, requirement of long residence times for complete combustion, should also be considered as a major factor for high concentration of heavy metals in this study as it is manifested; Chromium (95mg/kg) had the highest mean concentration followed by Cu (62mg/kg) >Pb (60mg/kg) >Cd (58mg.kg) >Ar

(35mg/kg) and Hg (31mg/kg) in which these values exceeded the permitted values of GB 18918-2002 (EPA/NEMA, 2005) as shown in table 3, the reason for this as shown in the literature review is that solid wastes contain most of the metals, penetration of the outside temperature into the furthest point inside the solid, i.e. the center, could take longer leading metals to be retained in bottom ash. This is suggestive why the concentrations of metals were above the recommended standards in this study.

It is also suggestive in this study that the high concentration of chromium resulted from incineration of stainless steel "sharps" which may have contained a significant amount of chromium.

CHAPTER 6

CONCLUSIONS AND RECOMMENDATIONS

6.1: Waste management

It's imperative that proper waste management criteria be instituted through a coordinated mix of practices that includes source reduction, recycling (including composting), and disposal. The most environmentally sound management of ash wastes is achieved when these approaches are implemented according to EPA's/NEMA preferred order: source reduction first, recycling and composting second and disposal in landfills or waste combustors last. In Kenya land filling is opted for open dumping after incineration of hazardous wastes. Modern landfills should be well-engineered facilities that are located, designed, operated, and monitored to ensure compliance with NEMA regulations. Solid waste landfills (SWL) must be designed to protect the environment from contaminants which may be present in the solid waste stream. The landfill siting plan should prevent the siting of landfills in environmentally-sensitive areas as well as on-site environmental monitoring systems which monitor for any sign of groundwater contamination.

It's therefore recommended that Solid waste landfills (SWL) should be in agreement with NEMA/EPA standards which include:

- **Location:** implementation of location restrictions to ensure that landfills are built in suitable geological areas away from faults, wetlands, flood plains, or other restricted areas.

- **Lining:** Composite liners that include a flexible membrane (geomembrane) overlaying two feet of compacted clay soil lining the bottom and sides of the landfill protect groundwater and the underlying soil from leachate releases (Figure 8).
- **Operating practices**: There is need for adaptation of proper operating mechanisms at all these sites these include compacting and covering waste frequently with several inches of soil help reduce odor; control litter, insects, and rodents; and protect public health.
- **Closure and post closure of Landfills**: Should be done to provide long-term care of closed landfills.
- **Financial assurance**—provides funding for environmental protection during and after landfill closure (i.e., closure and postclosure care).
- **Bioreactors**: These should be designed to quickly transform and degrade organic waste. The increase in waste degradation and stabilization is accomplished through the addition of liquid to enhance microbial processes.

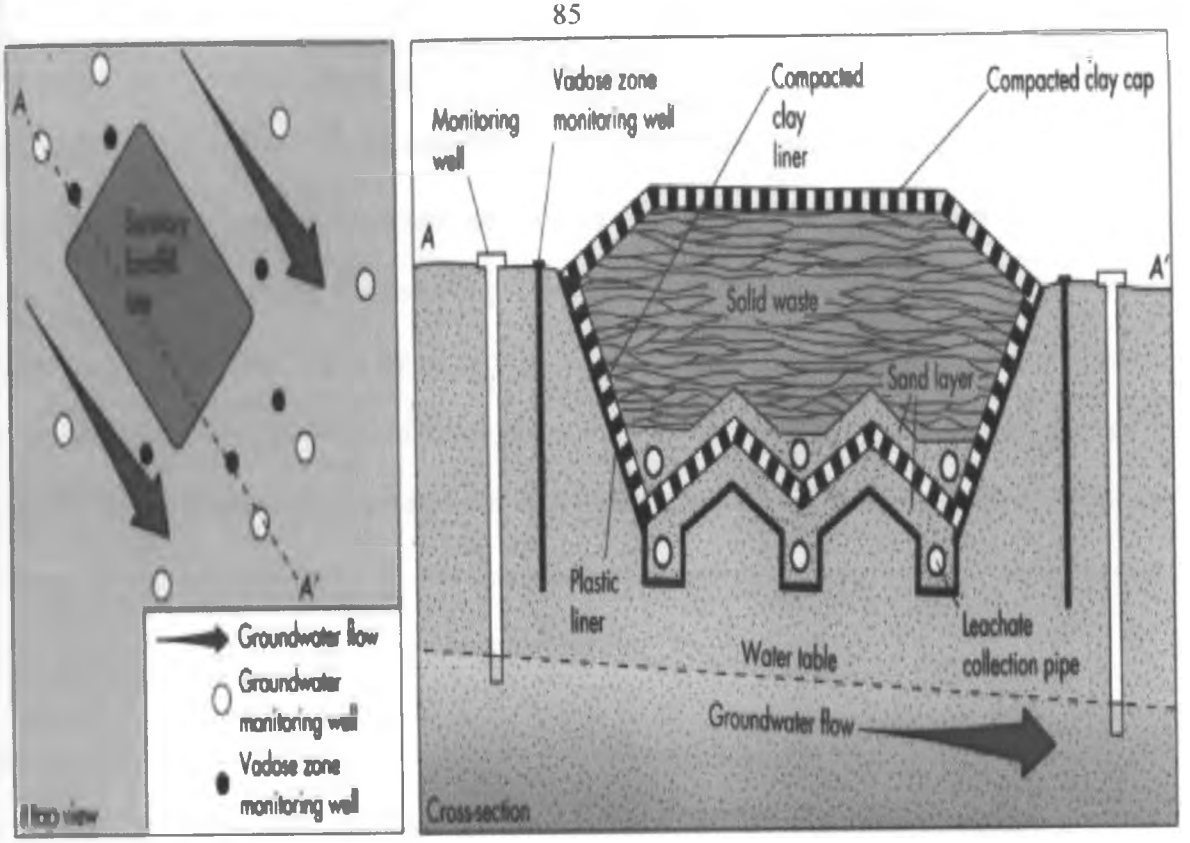


Figure 10: Modern landfill with plastic, clay liner and collection pipes to prevent leachate from entering the groundwater (Adopted from EPA, 2005).

6.2: Incineration

To reduce waste volume, local governments or private operators can implement a controlled incineration. In addition to reducing volume, combustors, when properly equipped, can convert water into steam to fuel heating systems or generate electricity. Incineration facilities can also remove materials for recycling. Burning of solid wastes can generate energy while reducing the amount of waste by up to 90% in volume and 75% in weight. Burning waste at extremely high temperatures also destroys chemical compounds and disease-causing bacteria. Regular testing ensures that residual ash is non-hazardous before being landfilled. About ten percent of the total ash formed in the combustion process can be used for beneficial use such as daily cover in landfills and road construction.

Improvement on the design of incinerators is paramount in all the study sites for efficient and complete combustion of solid wastes. These should include a variety of pollution control technologies significantly reduce the gases emitted into the air, including Scrubbers that use a liquid spray to neutralize acid gases and filters to remove tiny ash particles. Design and operation consistent with achieving good combustion conditions in waste incinerators and proper disposal of the ash residue. Minimum design and operating parameters for incinerator temperature, residence time and combustion air distribution are recommended to provide guidance to proponents in designing an incineration system that will achieve high combustion efficiencies. The installation of continuous monitors on all incinerators at these study sites is necessary; they should be located properly to measure the relevant parameters and should be equipped with recording devices for subsequent reference and analysis.

All the incinerators should be compliant with the national limits, through source emissions testing performed in accordance with the documented methods and procedures. NEMA should be primarily responsible for regulating combustors because air emissions from combustion pose the greatest environmental concern.

6.3: Laws and Regulations

The government of Kenya should institute legislations to protect human health and the environment from the potential hazards of waste disposal and incineration.

These should be geared towards reducing the amount of waste generated and ensuring that wastes are managed in an environmentally-sound manner.

National regulatory bodies and environmental lobbyists should establish distinct interrelated programs that will:

- Encourage the government to develop comprehensive plans to manage nonhazardous industrial solid waste and municipal solid waste, sets criteria for municipal solid waste landfills and other solid waste disposal facilities, and prohibits the open dumping of solid waste.

- Establish a system for controlling hazardous waste from the time it is generated units its ultimate disposal – in effect, from "**cradle to grave**".

- Provide general guidelines for the waste management program envisioned by parliament acts. They should include a legislative mandate directing NEMA to develop a comprehensive set of regulations to implement the law. These regulations, or rulemakings, issued by NEMA, translate the general mandate of the law into a set of requirements for the Agency and the regulated community.

This preliminary study attempts to provide evidence of the effects of environmental degradation and ecotoxicological implications due to incineration of medical, general and

pharmaceutical wastes. The results of this study will lead to the opinion that incineration facilities emitting substantial quantities of bottom ash, fine particulates, volatile heavy metals and hazardous organic pollutants.

These findings revealed a major policy implementation gap between the government regulatory body and the involved institutions. Government regulatory authorities should not approve facilities, which fail to comply with international standards of Minimum and Maximum Risk Levels of heavy metals environmental exposure.

Urgent measures should be taken to reduce the emissions from waste burning installations in Kenya and to apply rigorous biological monitoring until they can be taken out of service and safer methods of waste disposal brought into operation. Vigorous efforts should also be made to reduce the amount of waste produced, as there is presently no entirely satisfactory solution for its disposal at all these sites.

Taking into account these results and the difficulty in identifying causes of epidemics associated with incineration. It is a matter of considerable concern that incinerators be introduced with a comprehensive system to study their health effects of the local population.

Therefore, it is strongly suggested that the comprehensive evaluation of the environmental impacts of bottom ash is necessary before decisions can be made on its utilization, treatment or disposal. These results suggest that both chemical and biological analytical approaches are necessary for environmental impact assessment of heavy metals contamination.

Epidemiological surveillance of the general population in the proximity of the incineration facilities should be undertaken to estimate the population's heavy metals exposition, the morbidity and mortality indices. Risk assessment and risk management pertaining to toxic metals in the environment should be conducted for preparedness and policy implementation as a criterion for site remediation

Geographical mapping of exposure to heavy metal poisoning and spatiotemporal analysis by the use of geographic information systems (GIS) to develop incidence predictive risk maps can be vital in probabilistic risk modelling for future policy formulations and site location of the incinerators.

Biomonitoring and mapping of heavy metals exposure requires the existence of precise biomarkers which can be developed in local laboratories and implemented through inter- and intralaboratory.

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APPENDICES

Appendix I: Heavy Metal Sensitivity Questionnaire

NO : 00..... **DATE.... /10/2007**

APPENDIX 1: RESPONDENTS PERSONAL INFORMATION

Name of site----------
Name of respondent ----------
Duration of working/Living at sites ----------
Age in years ----------
Sex ----------
Marital Status Single----- Married -------
Level of formal Education ----------

This questionnaire should serve as a warning/alert to persons scoring "yes" in **five** or more of the questions below.

1. Have you had sore gums (gingivitis) often over the years?	Yes	No
2. Have you had mental symptoms such as confusion, forgetfulness and hallucinations?	Yes	No
3. Has severe depression aggressive behaviors, temper tantrums and social Withdrawal has been frequent problems?	Yes	No
4. Has ringing in the ears (tinnitus), hearing loss, difficulty hearing been present?	Yes	No
5. Have you had pains at the corner of your jaws (temporal mandibular joint)?	Yes	No
6. Have you had unusual shakiness (tremors) of your hands and arms or twitching of other muscles?	Yes	No
7. Do you have rashes "brown spots" or "age spots" under your eyes, palms, hands or elsewhere in the skin of your body?	Yes	No
8. Have you tended to have blurred vision or sensitivity to light?	Yes	No
9. Have you had loss of appetite/weight loss, nausea, vomiting, and diarrhea?	Yes	No
10. Do you have numbness or burning sensations in your mouth or gums?	Yes	No
11. Do you have numbness or unexplained tingling in your arms or legs?	Yes	No
12. Have you developed difficulty in walking (ataxia), swallowing or talking over the years?	Yes	No
13. Do you often have a "metallic" taste in your mouth, abdominal pain, stomach cramps; burning of the throat and mouth?	Yes	No
14. Do you have a lot of bad breath (halitosis) or white tongue (thrush)?	Yes	No
15. Have you had menstrual pains premature births, Spontaneous abortion or stillbirths?	Yes	No
16. Do you have heart irregularities or rapid pulse (tachycardia)?	Yes	No
17. Do you have unexplained joint pains?	Yes	No
18. Do you have unidentified chest pains?	Yes	No
19 Is your sleep poor or do you have frequent insomnia?	Yes	No
20. Are you extremely fatigued much of the time and never seem to have enough energy?	Yes	No

Appendix II: Informants socio-demographic parameters

Informants code	Age	Sex	Occupation	Level of education	Duration of stay(years)	Toxicity index
NB1	32	m	Security	T	12	1
NB2	24	m	Incinerator	S	7	1
NB7	34	f	Incinerator	S	6	1
NB9	42	m	Driver	S	5	1
NB12	62	f	Military	T	11	1
NB14	28	m	Mortuary	S	4	1
NB16	31	m	Incinerator	T	6	1
NB20	48	m	Incinerator	T	5	1
NB22	27	m	Business	P	4	1
NB26	20	f	Security	S	2	1
NB30	26	m	Incinerator	S	6	1
NB31	56	f	None	P	4	1
NB34	27	m	Incinerator	T	3	1
NB37	34	f	Incinerator	S	9	1
NB43	23	m	Incinerator	S	8	1
NB48	40	f	Mortuary	T	8	1
NB50	32	m	Incinerator	T	5	1
NB54	28	f	Military	S	5	1
NB57	47	f	Incinerator	P	8	1
NB59	54	f	None	P	5	1
NB61	51	m	Mortuary	S	7	1
NB64	53	f	Incinerator	P	8	1
NB67	30	m	Security	S	6	1
NB69	29	f	Incinerator	S	12	1
NB72	49	f	Housewife	T	10	1
NB75	38	m	Incinerator	T	8	1
NB78	32	f	Business	P	8	1
NB81	28	m	None	P	5	1
NB83	31	f	Incinerator	S	6	1
NB87	28	m	Military	P	8	1
NB88	29	m	Laborer	T	7	1
NB93	38	m	Incinerator	P	6	1
NB95	28	m	Mortuary	P	9	1
NB96	29	m	Incinerator	S	4	1
NB97	40	m	Electrician	T	10	1
NB99	36	m	Incinerator	T	6	1
NB3	27	m	Shoeshine	P	5	1
NB4	35	f	Business	N	3	1
NB5	38	m	Researcher	S	12	1
NB6	21	f	Doctor	S	2	1
NB8	39	m	None	P	6	1
NB10	24	m	Student	N	4	1
NB11	54	f	Security	T	2	1
NB13	30	m	Business	T	5	1
NB15	29	f	Ro	S	3	1
NB17	26	m	Nurse	T	2	1
NB18	24	m	Student	S	8	1
NB19	45	f	None	T	1	2
NB21	36	f	Military	T	6	1
NB23	43	m	Plumber	T	3	1

NB24	27	m	Business	T	5	1
NB25	25	m	Electrician	T	4	1
NB27	29	m	Accountant	S	3	2
NB28	32	m	Student	P	7	1
NB29	31	f	Business	P	4	1
NB32	36	m	Laborer	T	8	2
NB33	33	f	Security	P	2	2
NB35	22	f	Porter	S	5	2
NB36	32	m	Business	S	7	2
NB38	36	f	Military	T	2	2
NB39	34	m	Business	N	8	2
NB40	25	m	Student	T	10	2
NB41	30	f	Security	T	12	2
NB42	41	f	Fieldworker	P	2	2
NB44	27	m	Student	T	5	2
NB45	29	m	Business	S	9	2
NB46	25	f	Doctor	T	10	2
NB47	33	m	Nurse	P	4	2
NB49	31	m	None	N	9	2
NB51	34	m	Driver	N	7	2
NB52	25	f	Shoe shiner	T	8	2
NB53	28	m	Plumber	S	9	2
NB55	32	m	Electrician	P	2	2
NB56	46	m	Mortuary	S	4	2
NB58	49	m	Teacher	T	9	2
NB60	38	m	Dr	S	3	2
NB62	34	f	Incinerator	T	5	1
NB63	27	m	Fieldworker	T	5	2
NB65	34	f	Military	S	2	2
NB66	32	m	Researcher	P	3	2
NB68	26	m	Accountant	P	5	2
NB70	27	m	Cook	P	2	2
NB71	51	m	Nurse	P	4	2
NB73	41	m	Security	P	4	2
NB74	34	f	Teacher	P	2	2
NB76	43	m	Laborer	S	5	2
NB77	40	m	None	S	9	1
NB79	46	f	Housewife	P	5	2
NB80	27	m	Student	T	3	2
NB82	43	m	Security	T	4	2
NB84	43	f	Housewife	S	9	2
NB85	34	m	Business	P	7	2
NB86	38	m	Driver	S	3	2
NB89	35	f	Business	S	4	2
NB90	25	m	Teacher	T	9	2
NB91	27	m	Driver	S	6	2
NB92	29	f	Researcher	P	5	2
NB94	34	f	Housewife	T	5	2
NB98	54	f	Housewife	S	5	2
NB100	25	m	Nurse	S	4	2

KEY: Sex; m=Male, f= female: Level of education: P=Primary, S=Secondary, T=Tertiary.
Toxicity index score: 1= ≥5 signs, 2= ≤5 signs.

Appendix III: Information on Waste Disposal and Practices.

Name of site----------
Name of respondent ----------
Age in years ----------
Sex ----------
Marital Status **Single-----** **Married -------**

1. How do you manage the waste?

1. Open dumping
2. Sanitary landfills
3. Detoxification
4. Conversion

2. Do you have waste disposal guidelines in place?

1. Yes
2. No

3. Do you know of any guidelines on medical waste disposal in place?

1. Yes
2. No

4. Are you aware any legal provision/ regulation (national and international) of waste disposal in place?

1. Yes
2. No

5. Have you received any formal training regarding management of medical waste?

1. Yes
2. No

Is yes, Specify..... .....
If no, specify ....

6. Are there any in-house training on waste disposal and management?

1. Yes
2. No

7. Are you being provided with protective devices?

1. Yes
2. No

If yes, Specify.....

8. How often do you use them?

1. Not at all
2. Sometimes
3. All the times

9. Have you ever been vaccinated due to the kind of work you do and the type of wastes you handle?

1. Yes
2. No

10. Do you keep records of the waste you generate and incinerate?

1. Yes
2. No

11. What is the quantity of waste you generated and incinerated in this facility?

1. <100 kg
2. >100 kgs
3. 200-600 kgs
4. >600 kgs

12. Which types of waste do you incinerate? (Tick)

- Needles/syringes/surgical/pathological/gloves/infectious/plastic/plastic/chemicals

13. Is there a bottom linner at the dumping site?

1. yes
2. no

14. Does the site have liners on the sides?

1. yes
2. no

If yes, what kind of liner is it?

1. plastic membrane,
2. compacted clay soil

15. Does each platform or cell have a cap on is there a leachate control system on site?

1. yes
2. no

16. What type of leachate control system is in place?

1. basal drainage
2. pumping from wells

17. What is done with collected leachate?

1. re-circulated
2. treated on site
3. Pumped or taken away?

18. Has any analysis of the leachate been done?

1. yes
2. no

19. Do you monitor the flow of emission at the site?

1. Yes
2. No

20. Have you ever received any community complain regarding incineration operation?

1. Yes
2. No

If yes, what action has been undertaken? Specify.....

21. Siting of incinerators?

1. Good
2. Poor

If poor, specify the condition.....

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22. Examine the general environs next to the incinerator, Comment.....

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23. Details of incinerator, Specify.....

(i) Type
(ii) Size
(iii) Capacity
(iv) Fuel

24. Details of Pollution Control Systems

S. No		Existing	Proposed to be installed
1.	Air		
2.	Water		
3.	Noise		
4	Solid Waste		

25. What are the conditions of the Incinerators?

1. Satisfying
2. Not satisfying

If not satisfying. Comment on the practices and defects.....

26. When do you operate the incinerator?

1. Only day time.
2. Only at night
3. Both day time and night.

27. How many days of the week do you operate the incinerator?

1. One
2. Two
3. Three
4. All days

28. What are the temperature range of the incinerator and actual operation practice? Specify and comment

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