

**Heavy Metal Concentrations in the Environment and in Selected Staple Foods
Consumed Around Kisumu Region, Kenya.**

Peter Kahenya Kinyanjui

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Food Science and Technology in the Jomo Kenyatta University of Agriculture and
Technology

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DECLARATION

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

Signature

Date.....

Peter Kahenya Kinyanjui

This thesis has been submitted for examination with our approval as University Supervisors.

Signature

Date.....

Dr. A. O. Makokha

JKUAT, Kenya

Signature

Date.....

Prof. G. M. Kenji.

JKUAT, Kenya

Signature

Date:

Dr. A. Onyango

JKUAT, Kenya.

DEDICATION

I dedicate this work to my parents, Lucy and John Kinyanjui. You made sure I grew up to be whom I am today.

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ABBREVIATIONS AND ACRONYMS

PTWI	Permitted Tolerable Weekly Intake
IAEA	International Atomic Energy Agency
CNS	Central Nervous System
GI	Gastro Intestinal
PNS	Peripheral Nervous System
CV	Cardiovascular
MeHg	Methyl Mercury
Hg	Mercury
Pb	Lead
As	Arsenic
Cd	Cadmium
RfD	Reference Dose
EPA	Environmental Protection Agency
FDA	Federal Drugs Administration
MPC	Maximum Permissible Concentrations
UK	United Kingdom
ppm	Parts Per Million
OECD	Organization of Economic Cooperation and Development
AMAP	Arctic Monitoring and Assessment Program.

ABSTRACT

The objective of this study was to determine the level of mercury, lead, cadmium and arsenic in the environment and in selected staple foods consumed around Kisumu region of Kenya and to determine if those levels were within the maximum allowable limits by WHO. The heavy metal content of the samples was determined using AAS after wet ashing. The lead content in the water and soil ranged between 0.00 - 8.6 $\mu\text{g}/100\text{ ml}$ and 0.11 - 1.69 $\text{mg}/100\text{ g}$, respectively. In the maize and beans it was 0.00 - 0.23 $\text{mg}/100\text{ g}$, and in the fruits and vegetables it was 0.00 - 0.38 $\text{mg}/100\text{ g}$, while in the fish it was 0.01 - 0.50 $\text{mg}/100\text{ g}$. The mercury content in the water and soil ranged between 0.01 - 0.03 $\mu\text{g}/100\text{ ml}$ and 1.00 - 4.05 $\mu\text{g}/100\text{ g}$, respectively. In the dry maize and beans it was 1.38 - 2.20 $\mu\text{g}/100\text{ g}$, and in the fruits and vegetables was 0.00 - 3.41 $\mu\text{g}/100\text{ g}$, while in the fish it was 1.36-3.80 $\mu\text{g}/100\text{ g}$. The cadmium content in the water and soil ranged between 0.04 - 0.11 $\mu\text{g}/100\text{ ml}$ and 0.07-0.15 $\text{mg}/100\text{ g}$, respectively. In maize and beans it was 0.07 - 0.15 $\text{mg}/100\text{ g}$, while in the fruits and vegetables it was 0.00 - 0.14 $\text{mg}/100\text{ g}$. In fish it was 0.09 - 0.16 $\text{mg}/100\text{ g}$. The arsenic content in the water and soil ranged between 0.00 - 8.30 $\text{ng}/100\text{ ml}$ and 12.39 - 24.36 $\mu\text{g}/100\text{ g}$, respectively. In the maize and beans it was 5.21 - 7.03 $\mu\text{g}/100\text{ g}$, while in the fruits and vegetables it was 2.89 - 7.34 $\mu\text{g}/100\text{ g}$. In fish it was 4.31 - 7.66 $\mu\text{g}/100\text{ g}$. The results of this study show that the lead, mercury, cadmium and arsenic content in water and soil from Kisumu region were within the WHO maximum limits. However, there were significant differences in the heavy metals concentrations due to geographical location. There significant differences in heavy metal concentrations between species. It is recommended that further research be done to

determine the effect of food preparation on the heavy metals content in the foods, monitor the levels of the heavy metals in human tissue and also determine other sources of heavy metals contamination apart from soil and water and continuous monitoring of heavy metals since there may be seasonal variations in their levels.

CHAPTER 1:

INTRODUCTION

1.1 Background information

Heavy metal toxicity represents a significant public health problem world wide. If unrecognized or inappropriately treated, heavy metal toxicity can result in significant morbidity and mortality (WHO, 1995). At ppm and sub-ppm levels, toxic heavy metals such as arsenic, cadmium, lead and mercury can affect human health, regardless of whether they are present in substances we ingest or in paint, fabric, plastic, and other materials we get in contact with (WHO, 1993). Various regulatory bodies around the world strictly regulate the maximum concentrations of heavy metal contaminants in foodstuffs, drugs and cosmetics. Most regulations have set the maximum limits for these trace metal contaminates in foods and water (IAEA, 1994).

The periodic table contains 105 elements, of which 80 are considered as metals. Toxic effects in humans have been described for less than 30 of these metals. Many metals are essential to biochemical processes, and others have found therapeutic uses in medicine. However, occupational exposure to heavy metals has accounted for the vast majority of poisonings throughout human history (WHO, 1993). A heavy metal is a member of an ill-defined subset of elements that exhibit

metallic properties, which would mainly include the transition metals, some metalloids, lanthanides, and actinides. Many different of heavy metals definitions have been proposed; some based on density, some on atomic number or atomic weight and some on chemical properties or toxicity (Duffus, 2002). The term heavy metal has been called "meaningless and misleading" in an IUPAC technical report due to the contradictory definitions and its lack of a "coherent scientific basis" (Duffus, 2002). There is an alternative term; toxic metal, for which no consensus of exact definition exists. Depending on context, a heavy metal can include elements lighter than carbon and can exclude some of the heaviest metals. One source defines a heavy metal as one of the "common transition metals, such as copper, lead, and zinc. These metals are a cause of environmental pollution (heavy-metal pollution) from a number of sources, including lead in petrol, industrial effluents and leaching of metal ions from the soil into lakes and rivers by acid rain" (Oxford University Press, 2000). Another common definition is based on the weight of the metal (hence the name heavy metal), classifying all metals weighing more than 5000kg/m^3 , such as lead, zinc and copper as heavy metals (Zevenhoven and Kilpinen, 2001). In medical usage, heavy metals are loosely defined and include all toxic metals irrespective of their atomic weight. Heavy metal poisoning can possibly include excessive amounts of iron, manganese, aluminium or beryllium (the fourth lightest element), or a semimetal such as arsenic. This definition excludes bismuth, the heaviest of stable elements, because of its low toxicity (Duffus, 2002). Heavy metals occur naturally in the ecosystem

with large variations in concentration. Nowadays anthropogenic sources of heavy metals, i.e. pollution, have been introduced to the ecosystem. Waste derived fuels are especially prone to contain heavy metals so they should be a central concern in a consideration of their use (Zevenhoven and Kilpinen, 2001).

Hippocrates described abdominal colic in a man who extracted metals, and the pernicious effects of arsenic and mercury among smelters were known even to Theophrastus of Erebus who lived in the period 370-287 BC (Jarup, 2004). Toxic effects from chronic exposure to heavy metals are far more common than acute poisonings. The chronic exposures may lead to a variety of conditions depending on the route of exposure and the metabolism and storage of the specific element in question (IAEA, 1994). Many of the heavy metals have been implicated as carcinogens in the setting of chronic exposure. The most common heavy metals implicated in acute and/or chronic toxicity are lead, arsenic, and mercury (WHO, 1993). For the most part, heavy metals bind to oxygen, nitrogen, and sulfhydryl groups in proteins, resulting in alterations of enzymatic activity. This affinity of heavy metal species for sulfhydryl groups serves a protective role in heavy metal homeostasis as well. Increased synthesis of metal binding proteins in response to elevated levels of a number of metals is the body's primary defense against poisoning. For example, the metalloproteins which are induced by many metals are rich in thiol ligands, which allow high-affinity binding with cadmium, copper, silver, and zinc among other elements (Silva *et al.*, 2005). Other proteins involved

in both heavy metal transport and excretion through the formation of ligands are ferritin, transferrin, albumin, and hemoglobin (IAEA, 1994). Although ligand formation is the basis for much of the transport of heavy metals throughout the body, some metals may compete with ionized species such as calcium and zinc to move through membrane channels in the free ionic form. For example, lead follows calcium pathways in the body, hence, its deposition in bone and gingivae (IAEA, 1994). Nearly all organ systems are affected by heavy metal toxicity. However, the most commonly affected organ systems include the central nervous system (CNS), peripheral nervous system (PNS), gastrointestinal (GI), hematopoietic, renal, and cardiovascular (CV) (Silva *et al.*, 2005) . To a lesser extent, lead toxicity involves the musculoskeletal and reproductive systems (WHO, 1987). The organ systems affected and the severity of the toxicity vary with the particular heavy metal involved, the age of the individual, and the level of toxicity (IAEA, 1994).

Generally, children are more susceptible to the toxic effects of the heavy metals and are more prone to accidental exposures. Inorganic lead salts enter the body by way of ingestion or inhalation (WHO, 1987). For adults only about 10% of the ingested dose is absorbed. In contrast, children may absorb as much as 50% of an ingested dose. The percentage of absorbed lead is increased with deficiencies of iron, calcium, and zinc (WHO, 1989). It is also increased with a predominantly

milk diet, possibly due to the high lipid content of the milk. Children and infants are prone to developmental delays that are secondary to lead toxicity (IAEA, 1994).

Lake Victoria, with a total catchment area of approximately 194 200 km², is located between latitudes 0°20N, 3°0S and longitudes 31°39E, 34°53E and is shared by Kenya, Uganda and Tanzania. Lake Victoria basin has a total catchment's area of approximately 12,300 km² and a mean run-off of approximately 7,300,103 m³. In Kenya, it comprises of a low-lying, dry lakeshore basin surrounded by Kericho, Nandi and Kisii highlands, with an annual rainfall of approximately 2000 mm. The area has a short rainy season from November to December and a long rainy season from March to May. The main river basins include Nyando (3450 km²), Sondu-Miriu (3489 km²) and Nzoia. Rivers Yala, Kibos and Awach form the other basins. The area is under extensive sugar, tea and coffee plantations. Associated agricultural industries include Panpaper mills (Webuye), sugar factories (Muhoroni, Miwani, Chemilil, Nzoia, Sony and Mumias) and tea factories (Kericho and Kisii regions). Most of the rivers in the lake basin drain areas overlain by Precambrian and tertiary volcanics. Past documented hydrological data show variable annual mean suspended loads and discharge rates of some rivers in the area (Campbell *et al.*, 2006).

Kisumu town is the headquarters of Nyanza province, Kenya, on the northeastern shore of Lake Victoria. It is the commercial, industrial, and transportation centre of western Kenya, serving a hinterland populated by almost four million people. Kisumu is an important link in the Lake Victoria–Mombasa trade because of its water and rail connections. This has led to a high population density in the town, which results in heavy vehicle population, high quantities of sewerage disposed and high human activities that leads to pollution. The heavy vehicle population has led to lead pollution. Many of these vehicles have questionable roadworthiness because the regulatory measures in the country are not strictly enforced and they emit heavy fumes, which pollute the air and foodstuff grown or vended along the roads (Mutuku, 2004). Studies have reported high levels of lead content in vegetation sampled near major highways (Finkelman, 1996; Isabella, 1997). Research has also shown that there is good correlation between average traffic counts and average soil and plant lead content at sites close to the roadside (Isabella, 1997). Kisumu is the major urban town within the basin with various small scale to large scale industries (fish processing industries, tannery, brewery and textiles). Waste is discharged from municipal and industrial activities into the lake through rivers, surface run-off and storm waters.

1.2 Statement of the problem and Justification

Over the past century, there has been an increasing awareness throughout the world of the health and developmental risks associated with environmental exposure to toxic metals, such as, lead (Pb), mercury (Hg), cadmium (Cd), and arsenic (As). While exposure to toxic levels of any of these environmental contaminants may result in impaired health in adults, the toxicological effects of these metals are often more devastating in the developing central nervous system and general physiological systems of children (Counter and Buchanan, 2004). In Kenya regulations on the concentrations of heavy metals in the foods have been established. However, despite the well established severity of the consequences of heavy metal poisoning, there is very little data on the magnitude of heavy metals contamination foods and environment in Kenya. This project is thus designed to avail information on the extent of heavy metals contamination in staple foods and the environment in Kisumu, around the Lake Victoria region of Kenya. Such information is important for designing intervention measures for minimizing heavy metal pollution. Lake Victoria is an important source of fish both for local consumption and export. In the industrialized countries, there are strict regulations on the concentrations of heavy metals in foods. Strict monitoring and enforcement measures have been put in place in these countries for these heavy metals (IAEA, 1994). Therefore, contamination of Lake Victoria fish with the heavy metals could lead to the affecting of very many people both economically and health wise in Kenya, regionally and internationally. Its safety should be guaranteed and

safeguarded at all times. The results of this study can thus be used in designing necessary corrective and preventive actions to prevent or eliminate the threat of heavy metals contamination. Kisumu is a major urban town with various small to large scale industries (fish processing industries, tannery, brewery and textiles). The industrial wastes are discharged into the lake through rivers, surface run-off and by storm waters. There is a likelihood of the discharges containing toxic metals; including lead, mercury, cadmium and arsenic that could contaminate the lake ecosystem. It is thus important to establish the level of heavy metals contamination in the Lake Victoria ecosystem in order to determine the appropriate intervention measures if need be.

The findings of this study, will enhance awareness among stakeholders on the severity of the heavy metals contamination of food and water, and thus on their safety, and consequently on human health. Such information will be disseminated to policy makers with the objective of influencing positive intervention policies on heavy metal pollution in Kenya. Information generated will also contribute towards proper management of the environment given the nutritional and health implications posed.

1.3 Objectives

1.3.1 Main objective

The main objective of this study was to determine the level of mercury, lead, cadmium and arsenic in the environment and in selected staple foods consumed in the Kisumu region of Kenya.

1.3.2 Specific objectives

The specific objectives are to:

- 1.3.2.1. Determine the level of mercury in staple foods, soil and water.
- 1.3.2.2. Determine the level of lead in staple foods, soil and water.
- 1.3.2.3. Determine the level of cadmium in staple foods, soil and water.
- 1.3.2.4. Determine the level of arsenic in staple foods, soil and water.
- 1.3.2.5. To compare the level of heavy metals in the staple foods, soil and water with the maximum safe limits.

1.4 Research questions

- 1.4.1 What are the lead, mercury, cadmium and arsenic concentrations in the environment (soil and water) in Kisumu region?
- 1.4.2 What are the lead, mercury, cadmium and arsenic concentrations in the selected staple foods consumed in Kisumu region?

1.4.3 Are the lead, mercury, cadmium and arsenic concentrations in the water, soil, fish, maize, beans, fruits and vegetables within the safe maximum health limits as given by WHO?

CHAPTER 2:

LITERATURE REVIEW

2.1 Introduction

Heavy metal pollution can arise from many sources but most commonly arises from the purification of metals, e.g., the smelting of copper and the preparation of nuclear fuels (Duffus, 2002). Electroplating is the primary source of chromium and cadmium. Through precipitation of their compounds or by ion exchange into soils and muds, heavy metal pollutants can localize and lay dormant. Unlike organic pollutants, heavy metals do not decay and thus pose a different kind of challenge for remediation. Currently, plants or microorganisms are tentatively used to remove some heavy metals such as mercury (Zevenhoven and Kilpinen, 2001). Motivations for controlling heavy metal concentrations in gas streams are diverse. Some of them are dangerous to health or to the environment (e.g. Hg, Cd, As, Pb, Cr), some may cause corrosion (e.g. Zn, Pb), some are harmful in other ways (e.g. Arsenic may pollute catalysts) (Zevenhoven and Kilpinen, 2001). Within European community the 13 elements of highest concern are As, Cd, Co, Cr, Cu, Hg, Mn, Ni, Pb, Sn, and Tl, the emissions of which are regulated in waste incinerators. Some of these elements are actually necessary for humans in minute amounts (Cu, Cr, Ni) whilst others are carcinogenic or toxic, affecting, among others, the central

nervous system (Hg, Pb, As), the kidneys or liver (Hg, Pb, Cd, Cu) or skin, bones or teeth (Ni, Cd, Cu, Cr) (Zevenhoven and Kilpinen, 2001).

2.2 Lead

2.2.1 General description

Lead is the commonest of the heavy elements, accounting for 13 mg/kg of the earth's crust. Several stable isotopes of lead exist in nature, including, in order of abundance, ^{208}Pb , ^{206}Pb , ^{207}Pb , and ^{204}Pb . It is a soft metal with a melting point of 327 °C (WHO, 2003).

2.2.2 Occurrence

Lead is used in the production of lead acid batteries, solder, alloys, cable sheathing, pigments, rust inhibitors, ammunition, glazes, and plastic stabilizers (WHO, 1989). Tetraethyl and tetramethyl lead are important because of their extensive use as antiknock compounds in petrol, but their use for this purpose has been almost completely phased out in many developing countries including Kenya (WHO, 2003). From a drinking-water perspective, the almost universal use of lead compounds in plumbing fittings and as solder in water-distribution systems is important. Lead pipes may be used in older distribution systems and plumbing (Quinn and Sherlock, 1990). Atomic absorption spectrometry and anodic stripping voltammetry are the methods most frequently used for determining the levels of

lead in environmental and biological materials. Detection limits of less than 1 µg/litre can be achieved by means of atomic absorption spectrometry (ISO, 1986).

With the decline in atmospheric emissions of lead since the introduction of legislation restricting its use in fuels, water has assumed new importance as the largest controllable source of lead exposure in the USA (Levin *et al.*, 1989). Lead is present in tap water to some extent as a result of its dissolution from natural sources but primarily from household plumbing systems in which the pipes, solder, fittings, or service connections to homes contain lead. PVC pipes also contain lead compounds that can be leached from them and result in high lead concentrations in drinking-water. The amount of lead dissolved from the plumbing system depends on several factors, including the presence of chloride and dissolved oxygen, pH, temperature, water softness, and standing time of the water, soft, acidic water being the most plumbosolvent (Schock, 1989; 1990). Although lead can be leached from lead piping indefinitely, it appears that the leaching of lead from soldered joints and brass taps decreases with time (Levin *et al.*, 1989). Prepared food contains small but significant amounts of lead. Lead content is increased when the water used for cooking or the cooking utensils contain lead, or the food, especially if acidic, has been stored in lead-ceramic pottery ware or lead-soldered cans. The intake of lead from lead-soldered cans is declining as the use of lead-free solders becomes more widespread in the food processing industry (Quinn and Sherlock, 1990; Galal-Gorchev, 1991).

Soils and household dust are significant sources of lead exposure for small children (Clausing *et al.*, 1987), but the levels are highly variable, ranging from <5 µg/g to tens of milligrams per gram in contaminated areas. As lead is immobile, levels in contaminated soil will remain essentially unchanged unless action is taken to decontaminate them (CDC, 1985). The highest lead concentrations usually occur in surface soil at depths of 1–5 cm. Studies in inner-city areas in the USA have shown that peeling paint or dust originating from leaded paint during removal may contribute significantly to children's exposure to lead (Mushak and Crocetti, 1989).

2.2.3 Health effects

Adults absorb approximately 10% of the lead contained in food (USEPA, 1986), but young children absorb 4–5 times as much (Alexander, 1974). Absorption is increased when the dietary intakes of iron or calcium and phosphorus are low (Blake, 1983a;b). The principal vehicle for the transport of lead from the intestine to the various body tissues is the red blood cell (Moore, 1988), in which lead is bound primarily to haemoglobin and has a special affinity for the beta, delta and, in particular, fetal gamma chains (Ong and Lee, 1980). Following its absorption, lead appears both in a soft tissue pool, consisting of the blood, liver, lungs, spleen, kidneys, and bone marrow, which is rapidly turned over, and in a more slowly turned over skeletal pool. (Holtzman, 1978; Barry, 1978). Inorganic lead is not

metabolized in the body. Unabsorbed dietary lead is eliminated in the faeces, and lead that is absorbed but not retained is excreted unchanged via the kidneys or through the biliary tract (ATSDR, 1990).

Lead is a cumulative general poison, infants, children up to 6 years of age, the fetus, and pregnant women being the most susceptible to adverse health effects. Its effects on the central nervous system can be particularly serious (WHO, 2003). Overt signs of acute intoxication include dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucinations, and loss of memory, encephalopathy occurring at blood lead levels of 100–120 µg/dl in adults and 80–100 µg/dl in children. Signs of chronic lead toxicity, including tiredness, sleeplessness, irritability, headaches, joint pain, and gastrointestinal symptoms, may appear in adults at blood lead levels of 50–80 µg/dl. After 1–2 years of exposure, muscle weakness, gastrointestinal symptoms, lower scores on psychometric tests, disturbances in mood, and symptoms of peripheral neuropathy were observed in occupationally exposed populations at blood lead levels of 40–60 µg/dl (USEPA, 1986).

Lead interferes with the activity of several of the major enzymes involved in the biosynthesis of haem (USEPA, 1986). The only clinically well-defined symptom associated with the inhibition of haem biosynthesis is anaemia (Moore, 1988), which occurs only at blood lead levels in excess of 40 µg/dl in children and 50

µg/dl in adults (FAO, 1987). Lead-induced anaemia is the result of two separate processes: the inhibition of haem synthesis and an acceleration of erythrocyte destruction (Moore, 1988). Enzymes involved in the synthesis of haem include δ-aminolaevulinate synthetase (whose activity is indirectly induced by feedback inhibition, resulting in accumulation of δ-aminolaevulinate, a neurotoxin) and δ-aminolaevulinic acid dehydratase (δ-ALAD), coproporphyrinogen oxidase, and ferrochelatase, all of whose activities are inhibited (USEPA 1986; Moore, 1988). Reproductive dysfunction may also occur in females occupationally exposed to lead (USEPA, 1986; IARC, 1980). In 1986, JECFA established a provisional tolerable weekly intake (PTWI) of 25 µg of lead per kg of body weight (equivalent to 3.5 µg/kg of body weight per day) for infants and children (FAO, 1987). This PTWI was reconfirmed by JECFA in 1993 and extended to all age groups (WHO, 1993). On the assumption of a 50% allocation to drinking-water for a 5-kg bottle-fed infant consuming 0.75 litres of drinking-water per day, the guideline value is 0.01 mg/litre (WHO, 2003). As infants are considered to be the most sensitive subgroup of the population, this guideline value will also be protective for other age groups.

2.3 Arsenic

2.3.1 General description

Arsenic exists in oxidation states of -3, 0, 3 and 5. It is widely distributed throughout the Earth's crust, most often as arsenic sulfide or as metal arsenates

and arsenides. In water, it is most likely to be present as arsenate, with an oxidation state of 5, if the water is oxygenated. However, under reducing conditions (<200 mV), it is more likely to be present as arsenite, with an oxidation state of 3 (IPCS, 2001).

2.3.2 Occurrence

The different forms in nature are Arsenic (As), Arsenic trioxide (As_2O_3), Arsenic pentoxide (As_2O_5), Arsenic sulfide (As_2S_3), Dimethylarsinic acid (DMA) - $(\text{CH}_3)_2\text{AsO}(\text{OH})$, Monomethylarsonic acid (MMA) - $(\text{CH}_3)\text{AsO}(\text{OH})_2$, Lead arsenate (PbHAsO_4), Potassium arsenate (KH_2AsO_4), Potassium arsenite ($\text{KAsO}_2\text{HAsO}_2$). Arsenicals are used commercially and industrially as alloying agents in the manufacture of transistors, lasers and semiconductors, as well as in the processing of glass, pigments, textiles, paper, metal adhesives, wood preservatives and ammunition. They are also used in the hide tanning process and, to a limited extent, as pesticides, feed additives and pharmaceuticals. Arsenic is introduced into water through the dissolution of rocks, minerals and ores, from industrial effluents, including mining wastes, and via atmospheric deposition (IPCS, 1981; Nadakavukaren *et al.*, 1984; Hindmarsh and McCurdy, 1986). In well oxygenated surface waters, arsenic(V) is generally the most common arsenic species present (Irgolic, 1982; Cui and Liu, 1988); under reducing conditions, such as those often found in deep lake sediments or groundwater, the predominant form

is arsenic(III) (Lemmo *et al.*, 1983; Welch *et al.*, 1988). An increase in pH may increase the concentration of dissolved arsenic in water (Slooff *et al.*, 1990).

Arsenic concentrations measured in remote or rural areas range from 0.02 to 4 ng/m³ (US NRC, 1999). In urban areas, arsenic concentrations of 3–200 ng/m³ have been measured. Much higher concentrations (>1000 ng/m³) are present in the vicinity of industrial sources (Ball *et al.*, 1983; WHO, 1987; US NRC, 1999). The level of arsenic in natural waters, including open ocean seawater, generally ranges between 1 and 2 µg/litre (Hindmarsh and McCurdy, 1986; US NRC, 1999). Concentrations may be elevated, however, in areas with volcanic rock and sulphide mineral deposits (Hindmarsh and McCurdy, 1986); in areas containing natural sources, where levels as high as 12 mg/litre have been reported (Grinspan and Biagini, 1985); near anthropogenic sources, such as mining and agrochemical manufacture (US NRC, 1999); and in geothermal waters (mean 500 µg/litre, maximum 25 mg/litre) (US NRC, 1999). Mean arsenic concentrations in sediment range from 5 to 3000 mg/kg; the higher levels occur in areas of contamination (US NRC, 1999) but are generally unrelated to arsenic concentrations in water. The total estimated daily dietary intake of arsenic may vary widely, mainly because of wide variations in the consumption of fish and shellfish. Most data reported are for total arsenic intake and do not reflect the possible variation in intake of the more toxic inorganic arsenic species. Limited data indicate that approximately 25% of the arsenic present in food is inorganic, but this is highly dependent upon the type

of food (Hazell, 1985; US EPA, 1988; IPCS, 2001). Fish and meat are the main sources of dietary intake of arsenic (Gartrell *et al.*, 1986a); levels ranging from 0.4 to 118 mg/kg have been reported in marine fish sold for human consumption, and concentrations in meat and poultry can be as high as 0.44 mg/kg (Health and Welfare Canada, 1983). The mean daily intake of arsenic in food for adults has been estimated to range from 16.7 to 129 µg (Hazell, 1985; Gartrell *et al.*, 1986a; Dabeka *et al.*, 1987; Zimmerli *et al.*, 1989); the corresponding range for infants and children is 1.26–15.5 µg (Nabrzyski *et al.*, 1985; Gartrell *et al.*, 1986b). In preliminary studies in North America, the estimated daily intake of arsenic from the diet was 12–14 µg of inorganic arsenic (Yost *et al.*, 1998).

2.3.3 Health effects

Ingested elemental arsenic is poorly absorbed and largely eliminated unchanged. Soluble arsenic compounds are rapidly absorbed from the gastrointestinal tract (Hindmarsh and McCurdy, 1986); arsenic(V) and organic arsenic are rapidly and almost completely eliminated via the kidneys (Buchet *et al.*, 1981a; Luten *et al.*, 1982; Tam *et al.*, 1982). Inorganic arsenic may accumulate in skin, bone, liver, kidney and muscle (Ishinishi *et al.*, 1986); its half-life in humans is between 2 and 40 days (Pomroy *et al.*, 1980). Inorganic arsenic is eliminated from the body by the rapid urinary excretion of unchanged arsenic in both the trivalent and pentavalent forms and by sequential methylation (Buchet and Lauwerys, 1985; Lovell and Farmer, 1985). Limited short-term studies on humans indicate that the

capacity to methylate inorganic arsenic is progressively, but not completely, saturated when daily intake exceeds 0.5 mg (Buchet *et al.*, 1981b). In humans, inorganic arsenic does not appear to cross the blood–brain barrier; however, transplacental transfer of arsenic in humans has been reported (Gibson and Gage, 1982).

A number of studies have attempted to show that arsenic is an essential element, but a biological role has not been demonstrated so far (US NRC, 1999, 2001). Arsenic has not been demonstrated to be essential in humans (IPCS, 2001). The acute toxicity of arsenic compounds in humans is predominantly a function of their rate of removal from the body. Arsine is considered to be the most toxic form, followed by the arsenites (arsenic(III)), the arsenates (arsenic(V)) and organic arsenic compounds. Lethal doses in humans range from 1.5 mg/kg of body weight to 500 mg/kg of body weight (Buchet and Lauwerys, 1982). Acute arsenic intoxication associated with the ingestion of well water containing 1.2 and 21.0 mg of arsenic per litre has been reported (Feinglass, 1973; Wagner *et al.*, 1979). Early clinical symptoms of acute intoxication include abdominal pain, vomiting, diarrhoea, muscular pain and weakness, with flushing of the skin. These symptoms are often followed by numbness and tingling of the extremities, muscular cramping and the appearance of a papular erythematous rash (Murphy *et al.*, 1981). Within a month, symptoms may include burning paraesthesias of the extremities, palmoplantar hyperkeratosis, Mee's lines on fingernails and

progressive deterioration in motor and sensory responses (Fennell and Stacy, 1981; Murphy *et al.*, 1981; Wesbey and Kunis, 1981). Signs of chronic arsenicism, including dermal lesions such as hyper- and hypopigmentation, peripheral neuropathy, skin cancer, bladder and lung cancers and peripheral vascular disease, have been observed in populations ingesting arsenic contaminated drinking-water (Tseng *et al.*, 1968; Borgoño and Greiber, 1972; Hindmarsh *et al.*, 1977; Tseng, 1977; Zaldivar, 1980; Zaldivar and Ghai, 1980; Valentine *et al.*, 1982; Cebrian *et al.*, 1983). Dermal lesions were the most commonly observed symptom, occurring after minimum exposure periods of approximately 5 years. Effects on the cardiovascular system were observed in children consuming arsenic-contaminated water (mean concentration 0.6 mg/litre) for an average of 7 years (Zaldivar, 1980; Zaldivar and Ghai, 1980). In a large study conducted in Taiwan, a population of 40 421 was divided into three groups based on the arsenic content of their well water (high, >0.60 mg/litre; medium, 0.30–0.59 mg/litre; and low, <0.29 mg/litre) (Tseng, 1977). The role of these metabolites with regard to arsenic carcinogenicity remains unknown.

2.4 Mercury

2.4.1 General description

Mercury (with the chemical symbol of Hg) is a naturally occurring element found in air, water, and soil. It is distributed throughout the environment by both natural and anthropogenic (human) processes. Mercury is found in various inorganic and

organic forms and is persistent in the environment namely elemental mercury (with the chemical symbol of Hg⁰), ionic mercury (also known as inorganic mercury with the chemical symbol of Hg (II) or Hg²⁺) exists in nature as Hg (II) mercuric compounds or complexes in solution; and organic mercury with methylmercury (with the chemical formula MeHg) being the most important (US EPA, 1997c).

2.4.2 Occurrence

Various studies indicate that anthropogenic releases of mercury from industrial and combustion sources contribute to the levels of methylmercury in fish, especially in freshwater and immediate coastal environments. However, also contributing to these fish methylmercury concentrations are existing background concentrations of mercury, which may consist of mercury from natural sources, as well as historic anthropogenic mercury which has been re-emitted from the oceans or soils (US EPA, 1997a, 1997c; UNEP, 2002). Methylmercury bioaccumulates (higher concentration than the surroundings) in marine and fresh water fish and mammals. The older the fish or mammal, the higher the methylmercury concentration. It also biomagnifies, that is, the higher the organism is in the food chain, the higher its trophic level and the higher its methylmercury concentration. Therefore, bigger predatory fish are more likely to have higher levels of methylmercury (Storelli, 2003). High methylmercury concentration has also been observed in fish at lower levels in the food web, likely due to higher background

levels of mercury in the environment. Occupational exposures have been reported from (among others) chloralkali plants, mercury mines, mercury-based small-scale gold and silver mining, refineries, thermometer factories, dental clinics with poor mercury handling practices, and production of mercury-based chemicals (ATSDR, 1999; UNEP, 2002). Many workers may still be exposed to elevated mercury levels and therefore may be at risk (UNEP, 2002). Small-scale or artisanal mining, using gold-mercury amalgamation to extract gold from ore is a significant source of exposure for the workers and nearby populations (UNEP, 2002). Miners burn the gold-mercury amalgam to vapourize the mercury and recover the gold; thus the miners and local populations can have high exposure to mercury vapours. Consumption of contaminated fish by community residents can result in the intake of high levels of methylmercury (UNIDO, 2003). Some fatalities and severe poisonings have been associated with acute exposures to elevated air levels resulting from heating metallic mercury and mercury-containing objects (ATSDR, 1999; UNEP, 2002). Dental fillings made with mercury amalgam can be a source of human exposure to elemental mercury (UNEP, 2002). Exposures to elemental mercury or inorganic mercury forms can also occur due to use of some skin-lightening creams and soaps and the presence of mercury in some traditional medicines (such as certain traditional Asian or Chinese remedies). Exposures to organic mercury may result from the use of thimerosal (ethylmercury thiosalicylate) as a preservative in some vaccines and other pharmaceuticals (ATSDR, 1999; UNEP, 2002). However, the use of thimerosal in vaccines is being

phased out, or significantly reduced, in many countries, especially in vaccines intended for children.

In spite of its potential risks, mercury continues to be used in a variety of products and processes all over the world. Elemental mercury is used in artisanal and small-scale mining of gold and silver; chloralkali production; manometers for measurement and control; thermometers; electrical switches; fluorescent lamp bulbs; back lights of computers; and dental amalgam fillings. Mercury compounds are used in batteries; biocides in the paper industry, pharmaceuticals, paints, and on seed grain; and as laboratory reagents and industrial catalysts (ATSDR, 1999). Mercury can be released to air, water bodies, and soils during production (or other uses) or after disposal of the mercury-containing products and wastes. Mercury is also present in various raw materials (such as coal, oil, wood, and various mining deposits) and can be released to the air or other media when these materials are burned, processed, or disposed. Among human activities, combustion of fossil fuels is the most important in terms of both volume and distribution. Moreover, large amounts of mercury that remains in mine tailings, landfills, sediments, and stockpiles present a threat of future release (UNEP, 2002). Mercury emissions can be, however, distributed over long distances in the atmosphere and oceans. Therefore, even countries with minimal mercury emissions, and other areas situated remotely from dense human activity, may be affected. For example, high mercury exposures have been observed in the Arctic, far from any significant

sources of anthropogenic releases (ATSDR, 1999; UNEP, 2002; AMAP, 2002; AMAP, 2003, Van Oostdam *et al.*, 2005).

2.4.3 Health effects

The primary targets for toxicity of mercury and mercury compounds are the nervous system, the kidneys and the cardiovascular system. It is generally accepted that developing organ systems (such as the fetal nervous system) are the most sensitive to toxic effects of mercury. Fetal brain methylmercury levels are higher than in maternal blood (SACN, 2004), and the developing central nervous system of the fetus is currently regarded as the system of highest concern as it demonstrates the greatest sensitivity (WHO, 2004). It should be noted, however, that in humans the nervous system continues to develop through adolescence. Other systems that may be affected include the respiratory, gastrointestinal, hematologic, immune, and reproductive systems. As described below, the health effects caused by elevated exposures to elemental mercury, inorganic mercury compounds, and organic mercury compounds (methylmercury) differ (ATSDR, 1999; UNEP, 2002). At higher concentrations, adverse kidney and thyroid effects, pulmonary dysfunction, changes in vision and deafness may also be observed (ATSDR, 1999; US EPA, 1997d; UNEP, 2002). Short-term exposure to high concentrations of Hg₀ vapour damages the lining of the mouth, irritates lungs, cause tightness of chest, coughing, nausea, vomiting, diarrhea, and increased blood pressure (ATSDR, 1999). A few studies suggest that Hg₀ may cause reproductive

toxicity. However, most human studies indicate that long-term exposure to Hg⁰ does not affect the ability to have children (ATSDR, 1999). Studies with rats suggest that Hg⁰ exposure may result in behavioural developmental effects (US EPA, 1997d). Findings from genotoxicity assays are limited and do not provide supporting evidence for a carcinogenic effect of Hg⁰ (US EPA, 1997d). Nonetheless, as noted above, noncancer effects on the nervous system appear to be the most sensitive effect. Damage to the kidneys is the key end-point in exposure to inorganic mercury compounds. Accidental ingestion of mercuric chloride by children resulted in cardiac effects (increased heart rate and blood pressure) (ATSDR, 1999; US EPA, 1997d). Accidental drinking or ingestion of inorganic Hg can also cause considerable damage to the digestive tract and kidney even with the limited absorption rate. In addition, dermal exposures to ionic mercury can lead to adverse effects to the skin such as dermatitis (ATSDR, 1999; MPP and NRDC, 2005). There are some data indicating that mercuric chloride may be a germ cell mutagen. Positive results have been obtained for chromosomal aberrations in multiple systems, and evidence suggests that mercuric chloride can reach female gonadal tissue (ATSDR, 1999; US EPA, 1997d). Nonetheless, as noted above, noncancer damage to the kidney is considered the most sensitive effect. Neurotoxicity is the most sensitive endpoint of exposure to inorganic mercury compounds. In humans, the indices of neurotoxicity include neurobehavioral deficits, neuronal loss, ataxia, visual disturbances, impaired hearing, paralysis and death (WHO, 2004). Offspring born of women exposed to

high levels of methylmercury during pregnancy have exhibited a variety of developmental neurological abnormalities similar to cerebral palsy, including the following: delayed onset of walking, delayed onset of talking, altered muscle tone and deep tendon reflexes, and reduced neurological test scores (ATSDR, 1999; NRC, 2000; US EPA, 1997d; UNEP, 2002).

Both the central and peripheral nervous systems show signs and damage (WHO, 2004; Eto *et al.*, 2002). Deficits in acts of daily living were observed in Minamata disease adult patients significantly increased and were further aggravated by aging (Kinjo *et al.* 1993). Neurological subjective complaints as well as nonspecific complaints in a population in a polluted area in Minamata could also be influenced by past methylmercury exposure (Fukuda *et al.* 1999). The study of about 900 Faroese children showed that prenatal exposure to methylmercury resulted in neuropsychological deficits at seven years of age (Grandjean *et al.*, 1997). Developmental delays were significantly associated with the methylmercury exposures, even excluding the children whose mothers had higher hair mercury levels (above 10 µg/g) (Grandjean *et al.*, 1997). Within the low exposure range, each doubling of the prenatal methylmercury exposure level was associated with a developmental delay of 1-2 months (UNEP, 2000; ATSDR, 1999; UNEP, 2002).

Various studies also suggest that exposures to methylmercury may cause adverse effects to the cardiovascular system, including increased risk of acute myocardial

infarction and elevated blood pressure (Salonen *et al.*, 2000, Rissanen *et al.*, 2000, Guallar *et al.*, 2002, Yoshizawa *et al.*, 2002, Virtanen *et al.*, 2005, Grandjean *et al.* 2004). These studies suggest that even small increases in methylmercury exposures may cause adverse effects on the cardiovascular system, thereby leading to increased mortality (UNEP 2002). WHO has concluded that the available evidence for the potential cardiotoxicity of methylmercury was, however, not conclusive, but noted that further studies were needed (WHO, 2004). The Joint FAO/WHO Expert Committee on Food Additives (JECFA), which also evaluates chemical contaminants in the food supply, has established provisional tolerable weekly intakes (PTWIs) for total mercury at 5 µg/kg body weight (WHO, 1987) and for methylmercury at 1.6 µg/kg body weight (WHO, 2004). The PTWI for methylmercury of 1.6 µg/kg body weight was reconfirmed by JECFA at its 67th meeting in 2006 (WHO, 2006). The US EPA has developed Reference Doses (RfDs) for mercuric chloride of 0.3 µg/kg/day and methylmercury 0.1 µg/kg/day. At exposures increasingly greater than the RfD, the potential for adverse health effects increases (US EPA, 1995a, 1995b, 2001).

2.5 Cadmium

2.5.1 General description

Cadmium is a metal with an oxidation state of +2. It is chemically similar to zinc and occurs naturally with zinc and lead in sulfide ores. It is a soft white solid with the density of 8.64 g/cm³, melting point of 320.9 °C, boiling point of 765 °C at 100

kPa and is soluble in dilute nitric and concentrated sulfuric acids (IARC, 1976; Ros and Slooff, 1987; Ware, 1989).

2.5.2 Occurrence

Cadmium metal is used mainly as an anticorrosive, electroplated onto steel. Cadmium sulfide and selenide are commonly used as pigments in plastics. Cadmium compounds are used in electric batteries, electronic components and nuclear reactors (Friberg *et al.*, 1986; Ros and Slooff, 1987). Fertilizers produced from phosphate ores constitute a major source of diffuse cadmium pollution. The solubility of cadmium in water is influenced to a large degree by its acidity; suspended or sediment-bound cadmium may dissolve when there is an increase in acidity (Ros and Slooff, 1987). In natural waters, cadmium is found mainly in bottom sediments and suspended particles (Friberg *et al.*, 1986). Cadmium can be determined by atomic absorption spectroscopy using either direct aspiration into a flame or a furnace spectrometric technique. The detection limit is 5 µg/litre with the flame method and 0.1 µg/litre with the furnace procedure (ISO, 1985, 1986; Ware, 1989). Cadmium is present in ambient air in the form of particles in which cadmium oxide is probably an important constituent (Friberg *et al.*, 1986). Annual average concentrations in four cities in Germany in 1981–1982 were 1–3 ng/m³. Cigarette smoking increases cadmium concentrations inside houses. The average daily exposure from cigarette smoking (20 cigarettes a day) is 2–4 µg of cadmium

(Ros and Slooff, 1987). Rivers containing excess cadmium can contaminate surrounding land, either through irrigation for agricultural purposes, dumping of dredged sediments or flooding. It has also been demonstrated that rivers can transport cadmium for considerable distances, up to 50 km, from the source (WHO, 1992). Nonetheless, studies of cadmium contamination in major river systems over the past twenty to thirty years have conclusively demonstrated that cadmium levels in these rivers have decreased significantly since the 1960s and 1970s (Cook and Morrow, 1995; Elgersma *et al.*, 1992; Mukunoki and Fujimoto, 1996; Van Assche and Ciarletta, 1992). Cadmium concentrations in unpolluted natural waters are usually below 1 µg/litre (Friberg *et al.*, 1986). Median concentrations of dissolved cadmium measured at 110 stations around the world were <1 µg/litre, the maximum value recorded being 100 µg/litre in the Rio Rimao in Peru (WHO/UNEP, 1989). Average levels in the Rhine and Danube in 1988 were 0.1 µg/litre (range 0.02–0.3 µg/litre) (ARW, 1988) and 0.025 µg/litre (AWBR, 1988), respectively. In the sediments near Rotterdam harbour, levels in mud ranged from 1 to 10 mg/kg dry weight in 1985–1986, down from 5–19 mg/kg dry weight in 1981 (Ros and Slooff, 1987). Contamination of drinking-water may occur as a result of the presence of cadmium as an impurity in the zinc of galvanized pipes or cadmium-containing solders in fittings, water heaters, water coolers and taps. Drinking-water from shallow wells of areas in Sweden where the soil had been acidified contained concentrations of cadmium approaching 5 µg/litre (Friberg *et al.*, 1986). In Saudi Arabia, mean concentrations of 1–26

µg/litre were found in samples of potable water, some of which were taken from private wells or cold corroded pipes (Mustafa *et al.*, 1988). Levels of cadmium could be higher in areas supplied with soft water of low pH, as this would tend to be more corrosive in plumbing systems containing cadmium.

Cadmium in soils is derived from both natural and anthropogenic sources. Natural sources include underlying bedrock or transported parent material such as glacial till and alluvium. Anthropogenic input of cadmium to soils occurs by aerial deposition and sewage sludge, manure and phosphate fertilizer application. Cadmium is much less mobile in soils than in air and water. The major factors governing cadmium speciation, adsorption and distribution in soils are pH, soluble organic matter content, hydrous metal oxide content, clay content and type, presence of organic and inorganic ligands, and competition from other metal ions (OECD, 1994). The use of cadmium-containing fertilizers and sewage sludge is most often quoted as the primary reason for the increase in the cadmium content of soils over the last 20 to 30 years in Europe (Jensen and Bro-Rasmussen 1992). Atmospheric cadmium emissions deposition onto soils has generally decreased significantly over that same time period (Cook and Morrow, 1995; Mukunoki and Fujimoto, 1996). Indeed, recent studies in Europe have documented that atmospheric emissions do not presently have a significant impact upon the cadmium content of soils (Bak *et al.*, 1997). Fossil fuels contain 0.5 to 1.5 ppm cadmium, but phosphate fertilizers contain from 10 to 200 ppm cadmium (Cook and Morrow, 1995).

Food is the main source of cadmium intake for non-occupationally exposed people. Crops grown in polluted soil or irrigated with polluted water may contain increased concentrations, as may meat from animals grazing on contaminated pastures (IARC, 1976). Animal kidneys and livers concentrate cadmium. Levels in fruit, meat and vegetables are usually below 10 µg/kg, in liver 10–100 µg/kg and in kidney 100–1000 µg/kg. In cereals, levels are about 25 µg/kg wet weight. In 1980–1988, average cadmium levels in fish were 20 µg/kg wet weight. High levels were found in shellfish (200–1000 µg/kg) (Galal-Gorchev, 1991). Based on cadmium levels measured in 1977–1984, the estimated daily intake in food by the Netherlands population is 20 µg/person (IARC, 1976). The dietary daily intake of cadmium has also been estimated to be in the range 10–35 µg (Galal-Gorchev, 1991). In contaminated areas in Japan, daily intakes in 1980 were in the range 150–250 µg, based on measurements of cadmium in faeces (Friberg *et al.*, 1986).

2.5.3 Health effects

It has been well established that excess cadmium exposure produces adverse health effects on human beings. Humans normally absorb cadmium into the body either by ingestion or inhalation. Dermal exposure (uptake through the skin) is generally not regarded to be of significance (Lauwerys, 1988). It is widely accepted (WHO, 1992; ATSDR, 1997) that approximately 2% to 6% of the cadmium ingested is actually taken up into the body. Factors influencing cadmium absorption are the

form in which cadmium is present in the food, and the iron status of the exposed individual. In contrast, from 30% to 64% of inhaled cadmium is absorbed by the body, with some variation as a function of chemical form, solubility and particle size of the material inhaled. Thus, a greater proportion of inhaled cadmium is retained by the body than when cadmium is taken in by ingestion. For the non-occupationally exposed individual, inhalation exposure to cadmium does not usually contribute significantly to overall body burden. The exception to this generalization is the cigarette smoker.

For chronic cadmium exposure, effects occur mainly on the kidneys, lungs, and bones. A relationship has been established between cadmium air exposure and proteinuria (an increase in the presence of low molecular weight proteins in the urine and an indication of kidney dysfunction) (WHO, 1992; OECD, 1994). Cadmium is known to accumulate in the renal cortex, and there is evidence that the level of cadmium in the renal cortex associated with increased urinary excretion is about 200 to 250 $\mu\text{g/g}$ (wet weight). Depending upon exposure level and other sources of cadmium, this level might be reached after 20 years occupational exposure. However, recent work has demonstrated that these effects are reversible at low exposure levels once the cadmium exposure has been removed or reduced (Roels *et al.*, 1997). With today's low occupational exposure standards, coupled with required biological monitoring of cadmium exposure levels (e.g. cadmium-in-blood and cadmium-in-urine) and kidney function parameters (e.g. B-2 microglobulin, a low molecular weight protein), there is every assurance that

kidney dysfunction or other effects do not develop in occupationally exposed workers as they did in the past. Ellis *et al.*, (1981, 1983) were able to determine kidney and liver cadmium concentrations by in-vivo neutron capture γ -ray spectroscopy. Bruckner and Linsel (1977) reported a 140-fold increase in the hair cadmium concentrations of exposed workers. The general consensus appears to favour the use of hair cadmium measurements as indices to cadmium exposure (Hammer *et al.*, 1971; Baker *et al.*, 1977; Milosevic *et al.*, 1980; Takeuchi *et al.*, 1990), but this is not universally endorsed (Kim, 1982; Watanabe *et al.*, 1974; Imahori *et al.*, 1979).

The World Health Organization (WHO) has established a provisional tolerable weekly intake (PTWI) for cadmium at 7 $\mu\text{g}/\text{kg}$ of body weight. This PTWI weekly value corresponds to a daily tolerable intake level of 70 μg of cadmium for the average 70-kg man and 60 μg of cadmium per day for the average 60-kg woman. Clearly, the daily cadmium intake for the general population from food, which is by far the dominant source of cadmium, is well below the guidelines established by the World Health Organization. The average daily cadmium intake for the general population in the Western World has shown a distinct downward trend from 1970 through 1992 (Van Assche and Ciarletta, 1992), a reduction presumed to be due to the marked decreases in direct atmospheric deposition of cadmium onto crops and soils. Other studies have suggested that, over the timeframe of 1980 - 1985, levels of cadmium intake have been relatively constant (OECD, 1994). At

an absorption rate of 5% from ingestion, the average person is believed to retain about 0.5 to 1.0 μg of cadmium per day from food.

2.6 WHO heavy metal limits in water, soil, fish, maize, beans, fruits and vegetables

The WHO limits for lead, mercury, cadmium and arsenic in various food samples, water and soil are shown in Table 2.1.

Table 2.1: WHO limits for the heavy metals in µg/100 g (or ml)

Sample	Lead	Mercury	Cadmium	Arsenic
Water	1.5	0.6	300	1
Soil	500	200	5000	50
Fish	250	5	250	10
Maize	20	1.5	700	10
Beans	20	1.5	700	10
Fruits	30	1.5	700	10
Vegetables	30	1.5	700	10

Source: WHO, 1999; WHO, 2003; WHO, 2004

CHAPTER 3:

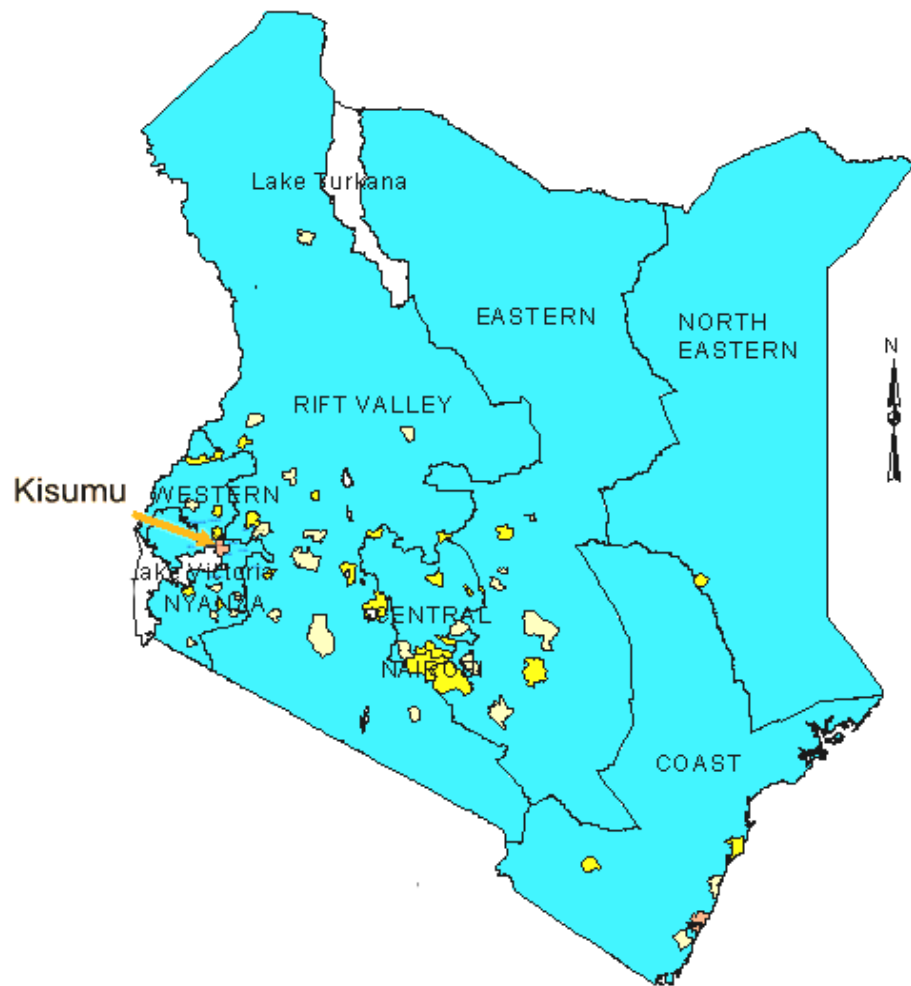
MATERIALS AND METHODS

3.1 Research design

This research was a cross sectional descriptive study where heavy metals (lead, mercury, cadmium and arsenic) levels were determined in soil, water and foods and compared with the maximum safe WHO limits (WHO 1999; 2003; 2004).

3.2 Research area

The study was carried out within a 10 kilometer radius in and around Kisumu City. Waters from the Lake Victoria, river Kisiat, and taps were collected. The foods were collected from the local farmers and from the market. The soils were taken from the adjacent areas to the lake and from the environs of Kisumu City. The site of the study was chosen because of the human and industrial activities that may result in discharge of effluent into the lake and its environs occasioning heavy metal pollution. Below, is a map of Kenya showing the Kisumu region, where the study was carried out.



Source: <http://www.unhabitat.org>

Figure 3.1: A map of Kenya showing Kisumu region



Source: <http://www.travelpod.com/travel-blog-city/Kenya/Kisumu/tpod.html>

Figure 3.2: A map of Kisumu region showing the sampling sites.

3.3 Sampling

The sampling was done three times during the study period, namely in April, September and November 2007. April was chosen to represent the long rains season, September for the dry season and November for the short rains season. Water and soil were sampled along the Lake Victoria shore and within a 10 km radius. Foods were collected from local farmers and also from the Kisumu Municipal market.

3.4 Collection of samples

3.4.1 Food samples

3.4.1.1 Fish – fish of different sizes were purchased at the Dunga and Tilapia landing sites on Lake Victoria. This was done early in the morning when the fishermen were landing after overnight fishing. The fish varieties analyzed included:

- Nile tilapia (*Oreochromis niloticus* (L)),
- Nile perch (*Lates (lates) niloticus* (L)),
- Butter catfish (*Schilbe intermedius* (Ruppell)),
- Migori haplo (*Hapochromis* species).
- Dagaa (Lake Victoria sardine (*Rastrineobola argentea* (Pellegrin)),

Three samples of each fish species were purchased from randomly chosen fishermen at each of the two landing sites. Dry dagaa was purchased from the Kisumu Municipal market.

3.4.1.2 Maize and beans

- Maize (*Zea mays* L) – 500 g of dry maize was purchased from five randomly selected vendors in the Kisumu Municipal market and used to make a homogeneous sample.
- Beans (*Phaseolus* spp) – 500 g of dry beans were purchased from five randomly selected vendors in the Kisumu Municipal market and used to make a homogeneous sample.

3.4.1.3 Vegetables and fruits – several farmers were randomly selected around Kisumu and three replicates of edible parts of vegetables and fruits, each weighing approximately 500 g purchased from them. Similar samples of the fruits and vegetables were purchased from the Kisumu Municipal market. The vegetables and fruits purchased included:

- Bananas (*Musa paradisiaca*)
- Spiderplant (akeyo/deck) (*Cleome guanadra* L)
- Oranges (*Citrus spp*)
- Spinach (*Spinacia oleracea*)
- Tomato (*Lycopersicon esculentum*)
- Custard apple (*Annona reticulate* L)
- Kales (*Brassicca oleracea*)
- Black nightshade (*Solanum nigrum*)
- Amaranth (*Amaranth amaranth*)
- Arrow roots (*Colocasia antiquorum*)
- Cow peas (*Vigna unguiculata* L)

3.4.2 Water

The sampling sites for the water samples were purposively chosen based on anthropological activities, whereby the areas with higher anthropological activities were preferred. The lake Victoria water was sampled at the Dunga and Tilapia Beaches. At each sampling point, five samples of 500 ml were collected. The specific sampling points were at the shore, and 200 metres inshore at depths of 30

cm. The river water was sampled from River Kisiat near the Nyamasaria Bridge, on the Nairobi-Kisumu highway. Five river water samples each of 500 ml were collected. The tap water samples were collected from five sampling points and the sample size was each 500 ml.

3.4.3 Soils

Soils were sampled from purposively selected sampling points, from where grid sampling was adopted for three samples each of 500g. The samples were collected at the depth of 0-15 cm (IAEA, 1994). The soil samples were obtained from among other areas, the Nairobi-Kisumu highway and at point of about 2 km away from the Nairobi-Kisumu highway. The sampling was done as per the IAEA protocols for soil sampling (IAEA, 1994).

3.5 Sample handling and preparation

3.5.1 Water

Immediately after sampling, 5 ml of concentrated HCl was added to the water samples to stabilize them, and then transported to the Food Science and Technology Department at JKUAT for analysis. The transport conditions were as recommended by Iqbal *et al.* (2006).

3.5.2 Soils

After collection, the soil samples were air-dried, ground, and screened to pass through a 0.5 mm sieve and then stored in plastic jars for further laboratory analysis according to AOAC (1984).

3.5.3 Fruits and vegetables

All the fruit and vegetable samples were cleaned to remove adhering soil particles, washed three times with de-ionized water and 0.05 M HCl, and then rinsed with de-ionized water three times to ensure dislodging and removal of dust particles (Turzen, 2003). The samples were then dried in a fan-forced oven at 60 ± 5 °C for 48 hours, ground using a stainless steel grinder, sifted through a 0.2-mm sieve, and stored in plastic jars for further analysis (AOAC, 1984).

3.5.4 Maize and beans

After delivery to the Food Biochemistry Laboratory at JKUAT, the food samples were stored under refrigeration until analysis. The samples were ground and dried at 65 °C for 10 hours prior to analysis (AOAC, 1984).

3.6 Laboratory analyses

For the soil and food samples, three replicate samples of one gram each were analyzed as per the AOAC method (AOAC, 1984). The samples were treated with 5 ml of a mixture of nitric acid, sulphuric acid and hypochloric acid in the ratio of

6:3:1. They were then allowed to stand for five minutes after shaking thoroughly (Turkekul *et al.*, 2004). The samples were digested on a hot plate starting at 70 °C through to 120 °C until the volume reduced to approximately 1 ml, and the suspended white fumes of SO₃ were clearly observed. The samples were then allowed to cool to room temperature after which approximately 20 ml of 5% hydrochloric acid solution was added. The samples were heated on a hot plate at approximately 75 °C for 15 minutes and then cooled. They were filtered through Whatman filter paper number 42 into a 100 ml volumetric flask, and filled to the mark using 5% HCl. A blank sample was also prepared in the same way (Turzen, 2003; WHO, 2006).

The water samples were put into a 250 ml beaker and placed on a hot plate. The water was heated and while about to boil, 2 ml of nitric acid-water mixture (50:50) and 10 ml hydrochloric acid-water mixture (50:50) were added. The samples were evaporated until the mixture reduced to approximately 25 ml. The 25 ml residues were transferred into 100 ml volumetric flasks and filled to the mark with distilled water (Iqbal *et al.*, 2006).

The lead, cadmium, mercury and arsenic contents in the samples were determined using a Shimadzu Atomic Absorption Spectrophotometer, Model AA-6200 according to Osborne and Voogt, (1978) and Turkebul *et al.*, (2004). Commercial samples of lead, cadmium, mercury and arsenic standards were used as reference

compounds (Wako Pure Chemical Industries Ltd., Japan). They were analyzed using Flame Emission Spectrophotometer. Mercury standard and samples were analyzed without the flame. Arsenic analysis was preceded by hydride vapour generation using the hydride vapour generator, while mercury analysis was preceded by mercury vapour generation using the mercury vapour generator (Turzen, 2003).

3.7 Data analysis and interpretation

Statistical analysis of the data was done using SAS (SAS version 9.2) (Mutuku, 2004) to compare the lead, mercury, cadmium and arsenic concentrations in the different samples of foods, soils and water. Quantitative values such as the mean, range, standard error and correlations were used (Schartz, 1993). The obtained results were compared with the maximum permissible concentrations (MPC) by WHO (1993; 2004).

CHAPTER 4:

RESULTS AND DISCUSSION

4.1 Lead

4.1.1 Lead content in the water samples from Kisumu region

The results of lead content in water samples from Kisumu region are shown in Figure 4.1. The lead content of the tap water was significantly lower than the water from Lake Victoria and River Kisiat ($p < 0.05$). Water treatment involves a flocculation and sedimentation step to clarify the water. This step could have contributed to the reduction in the lead content in the tap water as lead could have been trapped in the floccules. There was no significant difference ($p < 0.05$) in lead content between the water samples from the lake and the river. This observation was expected since the rivers feed the lake and thus their lead content is expected to be similar. The water from the lake shores at the two different sites (Dunga and Tilapia beaches) were not significantly different ($p < 0.05$) from each other. The lake waters (200 m in the lake) were also not significantly different ($p < 0.05$) at the two different sites (Dunga and Tilapia beaches). However, it is worth noting that the results showed a significant difference between the shoreline waters and the water sampled from 200 m inshore ($p < 0.05$), with the shoreline waters containing significantly higher lead content as compared to the inshore waters. Sources of lead in surface water or sediment could include deposits of lead containing dust

from the atmosphere, wastewater from the industries that handle lead, urban runoff and mining piles (UNEP, 2000). The observation made above indicates that the entry of lead into the lake water is at the shoreline, thus explaining why it was highest there. It was lower inshore due to the dilution effect and also due to the lower extent sedimentation of the suspended matter in the water.

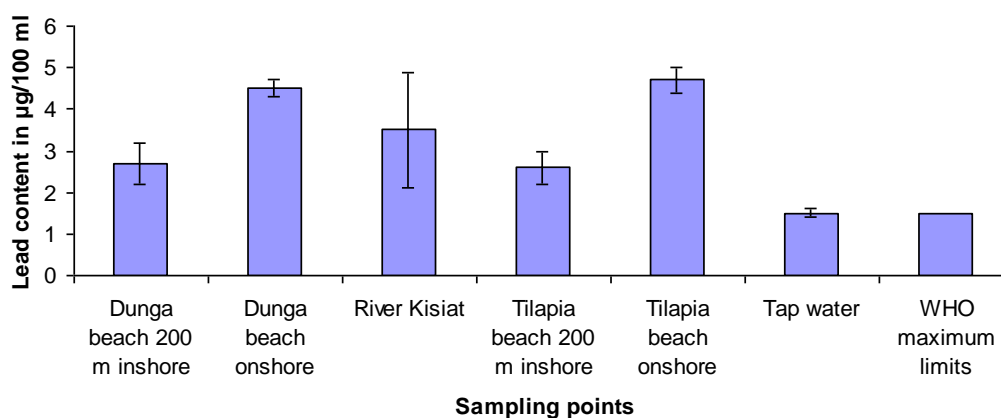


Figure 4.1: Lead content in waters from different sites in Kisumu region ($\mu\text{g}/100\text{ ml}$)

The WHO maximum lead limits in drinking water are 0.015 mg/litre or 1.5 $\mu\text{g}/100\text{ ml}$ (WHO, 1989). The mean lead content of the tap water analyzed (1.5 $\text{g}/100\text{ ml}$) was within this limit. It can thus be concluded that the tap water supplied in Kisumu city was within the maximum WHO limits for lead in drinking water. However, the lake and river waters did not meet this set standard and would thus not be fit for human consumption without prior treatment.

During the rainy season, lead could be leached from soils found next to busy highways into the water system posing a serious health problem (USEPA, 2003).

Once lead from vehicle exhaust fall onto the soil, it usually sticks to the soil, depending on the particulate size, from where small amounts may enter rivers and lakes and streams as the soil particles are moved by rainwater (Mahaffey, *et al.*, 1982). Lead may remain adsorbed to the soil particles in water for many years. Movement of lead from soil to water also depends on lead salt or compound, and on the physical and chemical characteristics of the soil (USEPA, 2003). In general, very little lead occurs in lakes, rivers, or ground water (WHO, 1989). Man's exposure to lead through water is low compared to exposure through air and food (WHO/FAO, 1995). The concentrations of lead in water in most American cities as determined in 1962, ranged from trace to 6.2 $\mu\text{g}/100\text{ ml}$ (Duggan and Inskip, 1985). McBe (1970) found lead concentrations of more than 5.0 $\mu\text{g}/100\text{ ml}$ in 41 out of 2595 samples and 25% of them contained no measurable amount of lead (McBe, 1970). The lead content in the tap water analyzed in this study was 1.0 to 1.9 $\mu\text{g}/100\text{ ml}$. This amount is much lower than the lead content reported in the two earlier studies in the American cities. Lead in drinking water occurs mainly through contamination at various points in the delivery system such as lead pipes, solder and water faucets (UNEP, 2000). The American cities studied were more likely to have lead plumbing, thus contributing to the high lead levels in the tap waters. The water supply systems in Kenya are relatively new and thus are mostly free of lead, thus explaining the low lead content in the Kisumu region waters as compared to the American cities waters. Lead concentration has also been found to be high in plastic pipes when left standing overnight (WHO, 1987). The sources of

lead in plastic pipes are probably lead stearate which is used as a stabilizer in the manufacture of polyvinyl plastics.

4.1.2 Lead content in the soil samples from Kisumu region

The results of the lead content of the soils from Kisumu region are shown in Table 4.1. The Nairobi-Kisumu roadside soil had significantly higher lead contents as compared to the soil obtained 2 km away from the road ($p < 0.05$). The major source of lead pollution could have been due to vehicular emissions. Previous studies have reported high levels of lead content in soils and vegetation sampled near major highways (Finkelman, 1996; Isabella, 1997). Research has shown that there is good correlation between average traffic counts and average soil and plant lead content at sites close to the roadside (Isabella, 1997). The significantly higher lead content in the soil from the roadsides may be attributed to the accumulation of lead in soil emanating from the vehicular emissions since of use leaded petrol in Kenya has only recently been partially eliminated. However, the lead content of the soils from Dunga beach and 2 km from the roadside had significantly lower lead content ($p < 0.05$). The two being far from the source of lead were expected to contain lower lead content which was found to be the case. There was no significant difference in the lead content in the soil from the Tilapia beach and R. Kisiat ($p < 0.05$). Both sites experienced relatively high vehicular traffic since River Kisiat sampling point was near the highway and Tilapia beach had a lot of anthropological activities including car washing business.

Table 4.1: Lead content in soil from Kisumu region (mg/100 g)

.Sample	Mean	Range	
		Minimum	Maximum
Dunga beach	0.15±0.01 ^c	0.11	0.22
River Kisiat	0.27±0.03 ^b	0.12	0.45
Tilapia beach	0.38±0.03 ^b	0.39	0.17
Roadside	0.76±0.13 ^a	0.26	0.68
2 km from the roadside	0.16±0.01 ^c	0.12	0.21
WHO limits	0.5		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

An inverse relationship between the distance from the road and lead content of the soil has been observed in various soils and vegetables (Tyroller, 1988). The soil from Tilapia beach showed significantly higher average lead levels than those from Dunga beach ($p < 0.05$) which could be attributed to the human activities difference at the two different landing sites. While Dunga beach only serves as a landing site for the fishermen with very few other human activities, Tilapia beach boasts of a thriving fast food business and car washing facilities. The higher lead levels in the soil from Tilapia beach could then be attributed to the car washing business and its higher vehicle traffic which raises the lead levels both from the vehicle exhaust and possible gasoline leakages. The results of this study fall within

the range established in an earlier study in Uasin Gishu District of Kenya where the typical values were found to be 0.1 to 0.3 mg/100 g in soils along the highway (Kunguru and Tole, 1994). The recommended maximum WHO level for lead in agricultural soil is 0.50 mg/100 g (USEPA, 2003). From the results obtained, the soil from the Dunga beach, Tilapia beach and River Kisiat were within the set limits. However, the mean lead content in the soil from the roadside (0.76 ± 0.13 mg/100 g) exceeded this limit. This could be explained by the high traffic densities experienced in Kisumu City.

4.1.3 Lead in the fruits and vegetables from Kisumu region

The results of lead contents in fresh foods from Kisumu region are shown in Table 4.2. The cow pea's leaves, kales, custard apple and bananas had below detectable levels of lead. They had thus significantly lower ($p < 0.05$) lead content as compared to the other fruits and vegetables. Amaranth, arrow roots and onions had significantly higher ($p < 0.05$) lead content compared to the other fruits and vegetables. The two ground crops analyzed (arrow roots and onions) had significantly higher lead content ($p < 0.05$) as compared to other crops suggesting a strong possibility of the soil being a source of lead. Particles originating from the air can be found in vegetables particularly green leafy ones growing or vended along roads (Isabela, 1997). Lead also gets into the foods from plants absorbing lead that is in the soil.

Table 4.2: Lead content in fruits and vegetables from Kisumu region (mg/100 g)

Sample	Mean	Range	
		Minimum	Maximum
Arrow roots	0.29±0.02 ^a	0.18	0.38
Onions	0.28±0.01 ^a	0.26	0.26
Cow peas leaves	ND ^c	ND	ND
Kales	ND ^c	ND	ND
Amaranth	0.29±0.02 ^a	0.19	0.37
Black nightshade	0.17±0.02 ^b	0.14	0.22
Guava	ND ^c	ND	ND
Tomatoes	0.11±0.01 ^b	0.10	0.13
Custard apples	ND ^c	ND	ND
Oranges	0.06±0.01 ^c	0.05	0.08
Spiderplant	0.12±0.05 ^b	ND	0.31
Black berries	0.17±0.03 ^b	0.12	0.25
Banana	ND ^c	ND	ND
WHO limits in foods	0.03		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

The higher lead content in amaranth could have been caused by bioaccumulation of lead, a phenomenon that has been observed in other crops as reported in other studies (Countera and Buchananb, 2004). The concentration of lead in various food items is varied. Karlwoski and Wojciechowska (1991) found a range of 0 - 1.3 mg/kg (0 - 0.13 mg/100 g) for vegetables. This is consistent with some of the results obtained from this study with cow peas' leaves, kales and traditional vegetables falling within this range. The amaranth and black night shade however had higher lead levels than those reported in this study. Kehoe (1961) found lead in every food in both industrial and non-industrial communities. Some of the vegetables and fruits analyzed in the current study did not contain detectable levels of lead. The detection limit for lead in this study was 0.01 µg/100 g thus any samples with lead below this limit were not detected.

Leelhaphunt *et al.* (1994) found lead levels of 210 µg/100 g (0.21 mg/100 g) in tomatoes purchased from Thailand markets. The lead content in tomatoes reported in this study was on average 0.11 mg/100 g ranging from 0.10 to 0.13 mg/100 g. This was lower than that reported in the 1994 study in Thailand. However, these figures are much higher than those reported by Denmark National Food Agency (Benko *et al.*, 1995). This may be due to the successful phasing out of leaded gasoline in the European countries. In Japan, Muramatsu *et al.* (1994) found lead concentrations of 184 µg/100 g (0.184 mg/100 g) in tomatoes. This is higher than the concentrations reported for tomatoes in this study (0.13 mg/100 g). Studies by

Sadjirun (1994) in Jakarta Indonesia established lead levels of 9.26 $\mu\text{g/g}$ (0.926 mg/100 g) in cabbage (*B. oleracea* var. capitata), 1.02 $\mu\text{g/g}$ (0.102 mg/100 g) in carrots and 10.74 $\mu\text{g/g}$ (1.074 mg/100g) in cauliflower. These were generally higher than the results of this study. WHO Codex Alimentarius Commission minimum levels in fruits is 30 $\mu\text{g}/100\text{ g}$ (0.03 mg/100 g) (WHO, 1999). Most of the fruits analyzed fell below this minimum level with the exception of tomatoes and black berries. The vegetables however, had significantly higher levels of lead as compared to the maximum allowable limit.

4.1.4 Lead in maize and beans from Kisumu region

The results of lead content in maize and beans from Kisumu region are shown in Figure 4.3. The maize had significantly lower ($p < 0.05$) lead content than the beans since it had non-detectable levels. However, the two beans varieties analyzed had no significant difference ($p < 0.05$) in lead content. Karlwoski and Wojciechowska (1991) found a range of 0-0.139 mg/100 g for grains. The maize analyzed fell within this range. However, the beans had slightly higher levels than these. Airborne lead can be deposited on the foods during distribution and display in the market (Isabella, 1997). In an earlier study in Uasin Gishu (Kunguru and Tole, 1994) the lead content in maize grown along the highway was found to be 13 to 25 $\mu\text{g}/100\text{ g}$, which was much higher than the values from the current study. The lead content of the maize analyzed in the current study had lower lead content than the maize grown away from the highway which had an average lead content of 6

µg/100 g (Kunguru and Tole, 1994). The maize lead levels in this study are very low compared to studies done in other parts of the world. According to Yang *et al.* (1994), cereals in China had lead levels of 6.3 µg/100 g.

Table 4.3: Lead content in maize and beans from Kisumu region (µg/100 g)

Sample	Mean	Minimum	Maximum
Green/yellow beans	0.18±0.02 ^a	0.15	0.23
Maize	ND ^b	ND	ND
Red/white beans	0.15±0.01 ^a	0.13	0.17
Maximum WHO limits	20		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.001$) from each other.

In Denmark, the National Food Agency established lead levels of 3.3 µg/100 g in cereals (National Food Agency of Denmark, 1992; Andersen *et al.*, 1996). Both studies reported lead levels above those established in this study. Another study by Urieta *et al.* (1996) found mean lead levels of 2.0 µg/100 g in cereals from Spain. In the United Kingdom, Ysart (1994) reported mean lead levels of 2.0 µg/100 g in cereal products. In Poland, Krelowska-Kula (1991) found lead levels of 6.7 µg/100 g in cereals while Sapuner- Postrunzic *et al.* (1996) reported mean lead levels of 7.6 µg/100 g in cereals. The maize lead levels established in this study were thus lower or within the ranges of lead established all over the world. Leelhaphunt *et al.*

(1994) in Thailand found mean lead levels of 570 $\mu\text{g}/100\text{ g}$ in baby corn (*Zea mays*). The Thailand study showed lead levels, which were much higher than those observed in this study.

In a related study in Thailand, Leelhaphunt *et al.* (1994) found varying lead levels in various bean varieties. In kidney beans, 118.3 $\mu\text{g}/100\text{ g}$, mung beans, 241.2 $\mu\text{g}/100\text{ g}$, and cowpeas 223.9 $\mu\text{g}/100\text{ g}$. The values reported in this study are lower than those reported in the Thailand study. Other studies by Zhang *et al.* (1998) in China, found lead levels of 2.6 $\mu\text{g}/100$ in pulses. These levels are higher than those established in this study (0.15- 0.18 $\mu\text{g}/100\text{ g}$). Muller and Anke (1995) in a German study found mean lead levels of 2.2 $\mu\text{g}/100\text{g}$ in white bean while Urieta *et al.* (1996) reported levels of 1.0 $\mu\text{g}/100\text{ g}$ of lead in Spain. The lead levels observed in the current study are lower than those reported in the European studies. It is important to note that phasing out of leaded gasoline in European countries has successfully been implemented as compared to Kenya and other developing countries (UNEP, 2000) but the number of vehicles on Kenyan roads is much lower than those in the European cities. The permissible levels by WHO (1999) in cereal is 20 $\mu\text{g}/100\text{ g}$ on dry weight basis. The contents of lead in maize reported in this study are generally within the permissible maximum levels by WHO. In pulses, the WHO (1999) permissible maximum limits are also 20 $\mu\text{g}/100\text{ g}$. The levels reported in this study for beans are thus within the WHO limits.

4.1.5 Lead content in fish samples from Kisumu region

The results of lead content in fish collected from Kisumu region are shown in Figure 4.2. The butter fish and Migori haplo (kamongo) had significantly higher levels of lead as compared to the other fish analyzed ($p < 0.05$). Nile perch on the other hand had significantly lower lead levels than the other fish analyzed ($p < 0.05$). There was no significant difference ($p < 0.05$) in lead content between the dried sardines and fresh sardines (dagaa). This indicates the the drying process of the dagaa was done away from sources of environmental contamination like dust. The tilapia of the two different sizes (small and medium) were not significantly different ($p < 0.05$) in lead content. Previous studies have indicated that the heavy metals content in fish is dependent on the size and the trophic level due to bioaccumulation with the predatory fish containing higher levels (Storelli, 2002). This was observed in the current study with the butter fish and Migori haplo registering higher lead content than the sardines. The lead content in the tilapia fishes of the different sizes was not significantly different since their diet is similar. The lead content in the fish analyzed ranged from a low of 0.11 to a high of 0.25 mg/100 g. In other studies, Karlwoski and Wojciechowska (1991) found a range of 0.02-0.25 mg/100 g for fish and seafood. The lead content of the fish analyzed in the current study thus fell within this range.

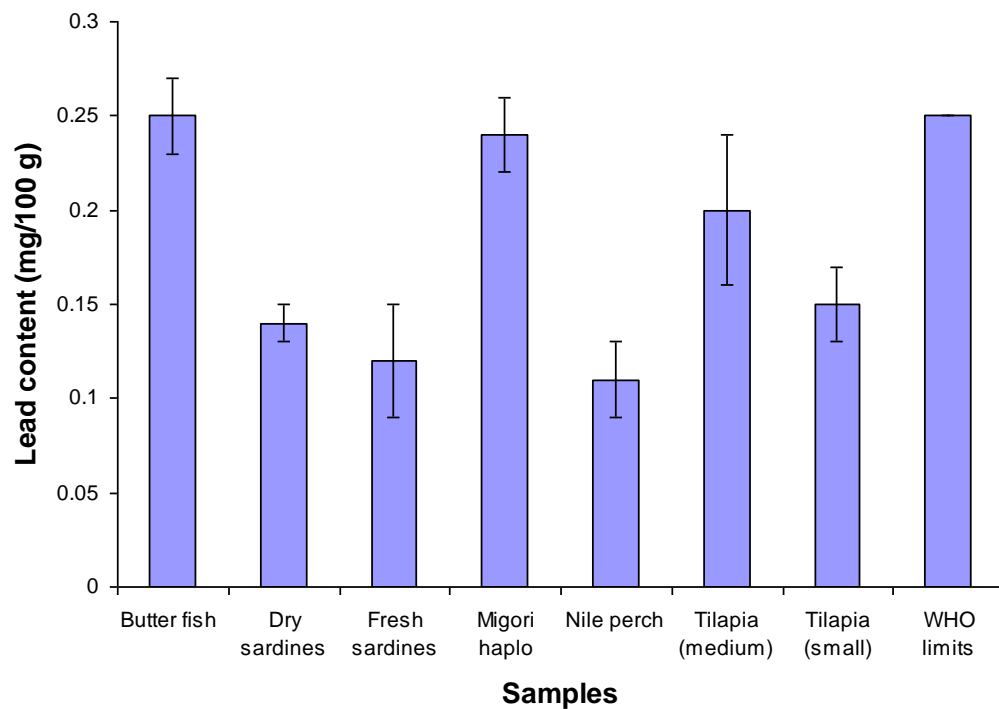


Figure 4.2: Lead content in fish from Kisumu region (mg/100 g)

The WHO set limits for lead in fish is 0.25 mg/100 g (WHO/FAO, 1995). The fish analyzed in this study fell within this limit.

4.2 Mercury

4.2.1 Mercury content in water samples from Kisumu region

The results of mercury content of water from Kisumu region are shown in Table 4.4. Tilapia beach shoreline water had significantly higher mercury content ($p < 0.05$) as compared to the other waters. With the exception of the Tilapia beach waters, there was no significance difference ($p < 0.05$) between the other water samples. Earlier studies of water total mercury concentrations have found a range

of 0.7 to 4.0 ng/litre, in northern L. Victoria (Amyot *et al.* 2000). Another study by Campbell *et al.* showed that the water total mercury concentrations ranged from 0.7 to 5.8 ng/litre (Campbell *et al.*, 2003). The results of this current study have indicated a mercury content of 0.01 to 0.03 µg/100 ml which is much higher than the earlier figures. However, the earlier studies had further found that total mercury concentrations were consistently elevated immediately near the cities of Jinja and Kisumu, where untreated sewage effluents are common in these shallow near shore environments (Campbell *et al.*, 1998). This could thus explain the higher mercury levels found during the current study since the sites samples were in close proximity of Kisumu City.

Table 4.4: Mercury content in water from Kisumu region (µg/100 ml)

	Mean	Range	
		Minimum	Maximum
Dunga beach 200m inshore	0.02±0.00 ^a	0.01	0.02
Dunga beach shoreline	0.01±0.00 ^a	0.01	0.01
River Kisiat	0.02±0.00 ^a	0.02	0.03
Tilapia beach shoreline	0.03±0.00 ^b	0.01	0.02
Tilapia beach 200m inshore	0.01±0.00 ^a	0.02	0.03
Tap water	0.02±0.00 ^a	0.01	0.02
WHO maximum limits	0.6		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

In the Kisumu region environment, the mercury contamination of the lake water could have arisen from the untreated sewage effluents and wash downs from gold mining activities upstream in Kakamega which fall under the lake's catchments area. Anthropogenic total mercury sources have likely increased when agricultural and developmental activities escalated in the African Great Lakes region (Campbell *et al.*, 2003.) Anthropogenic influences in the L. Victoria basin has already been implicated in excessive nutrient (C, N, P) inputs, significant shifts in diatom and chironomid species, increased concentrations of polyaromatic hydrocarbons from wood and fuel combustion, and the introduction of persistent organic pollutants (DDT, toxaphene) in recent short cores (Hecky, 1993; Lipiatou *et al.*, 1996; Verschuren *et al.*, 2002). Improper use or disposal of mercury and mercury containing objects can cause mercury exposure, for example after spills of elemental mercury or improper disposal of fluorescent light bulbs (Goldman and Shannon, 2001). However, the majority of mercury pollution comes from industrial emissions, especially from the burning of fossil fuels particularly high sulphur coal (Robertson, 2004). Other industrial sources include mercury mining and smelting, chloralkali process plants, and organic mercurial pesticide (Robertson, 2004). Other sources of mercury vapour include crematoriums, waste incinerators, and volcanoes (Goldman and Shannon, 2001). The Environmental Protection Agency (EPA) has limited the level of inorganic mercury in rivers, lakes, and streams to 0.0144 µg/100 ml. The shoreline water from the two beaches meets this requirement while the rest do not. Of greater concern are especially the

tap and river waters which are used for direct consumption by the residents of the city. The Food and Drug Administration (FDA), limits levels of mercury to no more than 0.2 µg/100 ml in bottled water. The waters analyzed contained mercury levels much lower than this limit. The WHO (1999) limits for mercury in drinking water is 0.6 µg/100 ml. All the water analyzed had mercury levels way below this limit.

4.2.2 Mercury content in the soil samples from Kisumu region

The results of mercury content of soils Kisumu region are shown in Table 4.5. Tilapia Beach shoreline soil had significantly higher mercury content ($p < 0.05$) than the Dunga Beach shoreline soils. This could be due to the higher anthropological activities at the Tilapia Beach as compared to the Dunga Beach as discussed under 4.2 above. However there was no significant difference between the mercury content of the soils from Dunga Beach shoreline and 2 km off the highway ($p < 0.05$). This again can be attributed to their reduced anthropological activities. The soils from the road sides were significantly higher in mercury content ($p < 0.05$) than all the other soils. In another study, surface soil samples collected from various agricultural sites around Jinja, Napoleon Gulf, had total mercury concentrations between 1.27 and 4.84 µg/100 g (Campbell *et al.*, 2003). All the soils analyzed in the current study had their mercury contents falling within this range. The soils mercury content ranged from 1.39 to 4.05 µg/100 g.

In Tanzania near Mwanza Gulf, soil samples collected near gold mercury processing sites have shown extremely elevated total mercury concentrations of 25.0 to 156.0 $\mu\text{g}/100\text{ g}$, although the total mercury concentrations declined very quickly to 0.2 $\mu\text{g}/100\text{ g}$ with depth in the soil cores and with distance from the processing sites (van Straaten, 2000). This rapid decline was attributed to the presence of oxyhydroxides in the Tanzanian soils restricting mercury movement away from the mercury source (van Straaten, 2000). The results of the current study shows that the mercury content of the soils around Kisumu City are much lower than those reported in Mwanza gulf in Tanzania.

Table 4.5: Mercury content in soil from Kisumu region ($\mu\text{g}/100\text{ g}$)

Sample	Mean	Range	
		Minimum	Maximum
Dunga beach shoreline	1.41 \pm 0.06 ^c	1.00	1.70
2km off the roadside	1.41 \pm 0.06 ^c	1.00	1.70
River Kisiat	1.88 \pm 0.17 ^{bc}	1.39	2.57
Tilapia beach shoreline	2.38 \pm 0.18 ^b	1.60	3.43
Roadside	3.13 \pm 0.29 ^a	2.17	4.05
WHO maximum limits	200		

Each value is a mean \pm SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

A review of forested soils from around the world (Roulet *et al.*, 1998) indicates that the Lake Victoria surface soil has total mercury concentrations of 0.17–0.51 µg/100 g. This is much lower than the values obtained from the current study, however, this can be attributed to the fact that the earlier study had focused on forested areas of the lake Victoria catchments which have nil or minimal anthropological activities. The results of the current study however fall within the ranges for temperate soils in North America and Sweden of 0.1–32.5 µg/100 g, and also of the Amazon soils of 0.6–21.9 µg/100 g. Data on normal total mercury concentrations in African soils are very limited and there is a need to survey soil concentrations across East Africa (Campbell *et al.*, 2003). Lakes Ontario and Erie shores were found to contain mercury values between 14.0 to 480.0 µg/100 g (Mudroch *et al.*, 1988). This was much higher than the mercury levels found in the soils from L. Victoria shores. A study in southern L. Victoria found that wetland sediments near gold mines had elevated total mercury concentrations of 17.0–535.0 µg/100 g dry weight prior to entering the Igonozela wetlands (van Straaten, 2000). These are much higher than those found in the current study. The WHO limits for mercury in soil are 200 µg/100 g. All the soils analyzed in the current study had mercury levels much lower than this limit.

4.2.3 Mercury content in maize and beans samples from Kisumu region

The results of mercury contents in maize and beans from Kisumu region are shown in Figure 4.3. Maize had significantly higher ($p < 0.05$) mercury levels as compared

to the beans. The two bean varieties were however not significantly different ($p < 0.05$). Total mercury sources to L. Victoria region are primarily anthropogenic (Campbell *et al.*, 2003). It can thus be postulated that the mercury found in the maize and beans could have arisen from environmental exposure to dust containing mercury compounds. The set WHO (2006) maximum limit for mercury in foods is $1.5 \mu\text{g}/100 \text{ g}$. The two bean varieties' mercury content fell within this range. However, the mercury content of the maize was slightly higher than this limit.

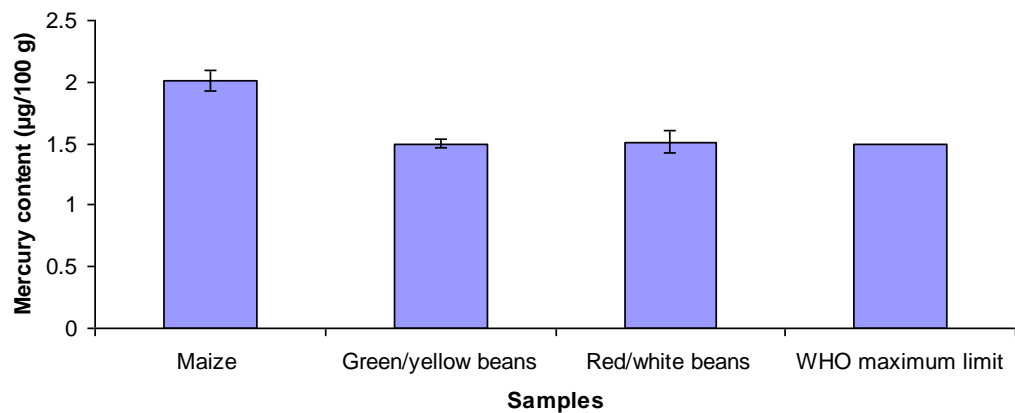


Figure 4.3: Mercury content in maize and beans from Kisumu region ($\mu\text{g}/100 \text{ g}$)

4.2.4 Mercury content in fruits and vegetables samples from Kisumu region

The results of mercury content of the fruits and vegetables from Kisumu region are shown in Table 4.6. Kales were found to have significantly higher ($p < 0.05$) mercury levels than the rest of the vegetables and fruits. Among the underground crops, the onions had significantly lower mercury content ($p < 0.05$) than the arrow roots. Among the fruits, the oranges, blackberries, bananas and guava had

significantly lower ($p < 0.05$) mercury content as compared to the tomatoes and custard apple.

Table 4.6: Mercury content in fruits and vegetables from Kisumu region ($\mu\text{g}/100\text{ g}$)

Sample	Mean	Range	
		Minimum	Maximum
Banana	0.86 ± 0.04^c	0.77	0.98
Tomatoes	1.97 ± 0.09^b	1.79	2.20
Black nightshade	0.99 ± 0.10^c	0.82	1.28
Onions	0.22 ± 0.02^e	0.16	0.26
Amaranth	1.87 ± 0.21^b	0.80	2.63
Cow peas leaves	0.72 ± 0.14^d	0.36	1.38
Kales	3.16 ± 0.10^a	2.59	3.41
Arrow roots	1.56 ± 0.05^b	1.33	1.76
Black berries	ND ^e	ND	ND
Custard apple	1.69 ± 0.13^b	1.47	2.05
Guava	0.65 ± 0.05^d	0.53	0.77
Oranges	ND ^e	ND	ND
Spiderplant	1.75 ± 0.28^b	0.90	2.61
WHO maximum limits	1.5		

Each value is a mean \pm SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

Among the leafy vegetables, there were no significant differences ($p < 0.05$) between amaranth, black night shade and traditional vegetables. These three vegetables tend to grow in the wild explaining their similarities. The set standard for mercury in foods is $1.5 \mu\text{g}/100 \text{ g}$. The cow peas' leaves, banana, black berries, black nightshade, guava, onions and oranges were found to be within this limit. The amaranth, arrow roots, custard apple, kales, tomatoes and traditional vegetable were found to have higher contents of mercury than the set limit.

4.2.5 Mercury content in fish samples from Kisumu region

The results of mercury content in fish from Kisumu region are shown in Figure 4.4. The Migori haplo (kamongo) was found to have significantly higher ($p < 0.05$) mercury content than the rest of the fish. The two different sizes of tilapia did not have a significant difference ($p < 0.05$) in mercury content. This is contrary to expectation since earlier studies had indicated that heavy metals content including mercury tend to increase in fish with increase in size due to bio-accumulation (Storelli *et al.*, 2002). This could have however be due to their similarity dietary intakes considering the fish analyzed were not very different in size most likely had a very similar diet. The fresh and dried sardines did not have a significant difference ($p < 0.05$) in levels of mercury which indicates that the drying process did not have a significant effect on the mercury content of the sardines. Monitoring of environmental and human exposure to mercury in the Nungwe Bay area of the Lake Victoria goldfields Tanzania, has revealed low mercury concentrations in

fish ranging between 1.8 and 16.9 $\mu\text{g}/100\text{ g}$ with a mean of 7 $\mu\text{g}/100\text{ g}$ (Campbell *et al.*, 2003). All the fish analysed in this study had their mercury content within this range. Mining has not produced a significant increase in environmental methylmercury that is available for bioaccumulation in aquatic food chains. Highest mercury concentrations in fish caught along the southern shores of Lake Victoria and in rivers draining from gold processing sites were detected in lungfish species (*Protopterus aethiopicus*), and lowest Hg concentrations in tilapia (*Oreochromis niloticus* and *Tilapia zillii*) (Campbell *et al.*, 2003).

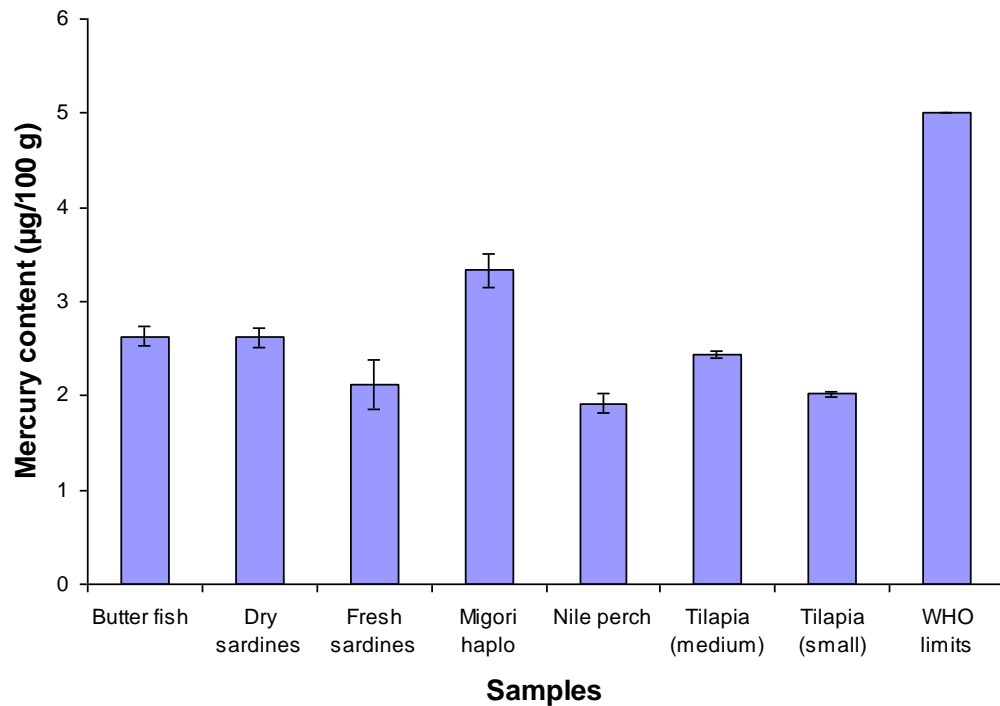


Figure 4.4: Mercury content in fish from Kisumu region ($\mu\text{g}/100\text{ g}$)

Lake Victoria fish contain lower levels of mercury than some other lakes in the region, such as Lake Malawi and Lake Albert (Campbell *et al.*, 2003). The earlier research suggested that most fish species caught in Lake Victoria are safe to eat, even if consumed often (Campbell *et al.*, 2003). This is good news for fish exporters, who must meet international guidelines for acceptable mercury levels. Nearly all fish contain trace amounts of methyl mercury, some more than others. In areas where there is industrial mercury pollution, the levels in the fish can be quite elevated (Campbell *et al.*, 2003). In general, however, methyl mercury levels for most fish range from less than 1 µg/100 g to 50 µg/100 g (Campbell *et al.*, 2003). The fish analyzed in the current study falls within this range. Certain species of very large tuna, typically sold as fresh steaks or sushi, can have levels over 100 µg/100 g (Campbell *et al.*, 2003) which is much higher than the levels of mercury in fish found in the current study. The WHO maximum limit of mercury in fish is 5 µg/100 g. This was not exceeded by any of the fish varieties analyzed in the current study.

4.3 Cadmium

4.3.1 Cadmium content of water samples from Kisumu region

The results of cadmium content in water from Kisumu region are shown in Figure 4.5. R. Kisiat and tap waters had significantly higher ($p < 0.05$) cadmium content as compared to the lake water. The lake waters from all the sampling points at both the Dunga and Tilapia Beaches were not significantly different ($p < 0.05$) in

cadmium content. The tap and river water are not significantly different from each other ($p < 0.05$) in cadmium content. Cadmium may enter aquatic systems through weathering and erosion of soils and bedrock, atmospheric deposition direct discharge from industrial operations, leakage from landfills and contaminated sites, and the dispersive use of sludge and fertilizers in agriculture (OECD, 1994). The most likely source of cadmium in the waters analyzed could be the use of fertilizers in agriculture which ends up in the water systems through soil erosion. Much of the cadmium entering fresh waters from industrial sources may be rapidly adsorbed by particulate matter, and thus sediment may be a significant sink for cadmium emitted to the aquatic environment (WHO, 1992). Some data shows that recent sediments in lakes and streams range from 0.2 to 0.9 ppm in contrast to the levels of generally less than 0.1 ppm cited for fresh waters (Cook and Morrow, 1995).

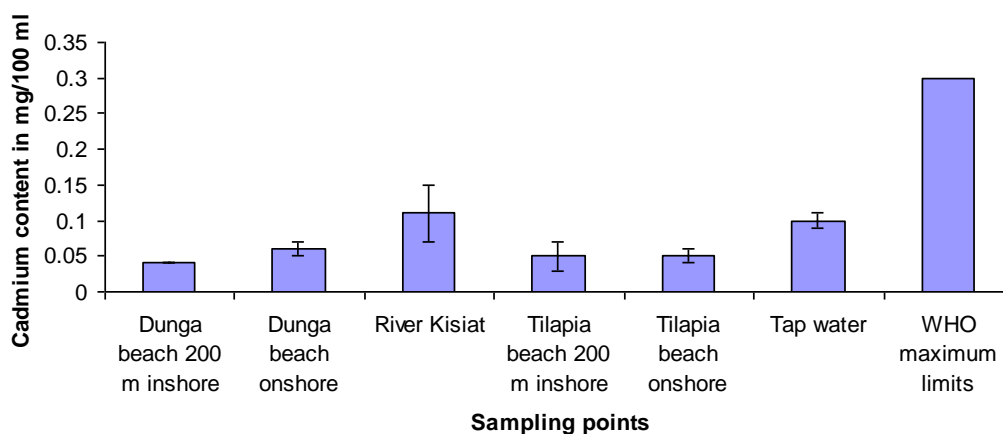


Figure 4.5: Cadmium content in water from Kisumu region (mg/ 100 ml).

The average cadmium content in the world's oceans has variously been reported as low as <5 ng/L (WHO, 1992) and 5-20 ng/L (OECD, 1994; Jensen and Bro-Rasmussen, 1992) to as high as 110 ng/L (CRC, 1996), 100 ng/L (Cook and Morrow 1995) and 10 to 100 ng/L (Elinder, 1985). Higher levels have been noted around certain coastal areas (Elinder, 1985) and variations of cadmium concentration with the ocean depth, presumably due to patterns of nutrient concentrations, have also been measured (WHO, 1992; OECD, 1994). Even greater variations are quoted for the cadmium contents of rainwater, fresh waters, and surface waters in urban and industrialized areas. Levels from 1 µg/100 ml to 400.0 µg/100 ml have been quoted in the literature depending on specific location and whether or not cadmium total or dissolved cadmium is measured (Elinder, 1985; WHO, 1992; OECD, 1994). The results of this study fall within this range being 40 to 110 µg/100 ml. It has been demonstrated that rivers can transport cadmium for considerable distances, up to 50 km, from the source (WHO, 1992). Nonetheless, studies of cadmium contamination in major river systems over the past twenty to thirty years have conclusively demonstrated that cadmium levels in these rivers have decreased significantly since the 1960s and 1970s (Cook and Morrow, 1995; Elgersma *et al.*, 1992; Mukunoki and Fujimoto, 1996; Van Assche and Ciarletta, 1992). Cadmium compounds are currently mainly used in rechargeable nickel–cadmium batteries. Cadmium emissions have increased dramatically during the 20th century, one reason being that cadmium-containing products are rarely re-cycled, but often dumped together with household waste.

The WHO limits for cadmium in water is 0.3 mg/100 ml. All the water analyzed during this study fell within this limit.

4.3.2 Cadmium content in soil samples from Kisumu region

The results of the cadmium content in the soil from Kisumu region are shown in Table 4.7. The soils from the Tilapia Beach, River Kisiat and roadside were significantly higher ($p < 0.05$) in cadmium content as compared to the Dunga Beach and 2 km off the road soils. The River Kisiat soils were not significantly different ($p < 0.05$) from the roadside and Tilapia beach soils. Fossil fuels contain 0.5 to 1.5 mg/kg cadmium, but phosphate fertilizers contain from 10 to 200 mg/kg cadmium (Cook and Morrow 1995). The higher cadmium content in the roadside soil could be associated to the deposition of cadmium from fossil fuel combustion in vehicles; the same could be true of the River Kisiat and Tilapia Beach soils which also experience high traffic levels. The three areas also experience higher anthropogenic activities which could have led to the higher cadmium content. Cadmium in soils is derived from both natural and anthropogenic sources. Natural sources include underlying bedrock or transported parent material such as glacial till and alluvium. Anthropogenic input of cadmium to soils occurs by aerial deposition and sewage sludge, manure and phosphate fertilizer application. This could explain the higher levels of cadmium in the Tilapia Beach which has more agricultural activities as compared to the other areas sampled. Cadmium is much less mobile in soils than in air and water. The use of cadmium-containing

fertilizers and sewage sludge is most often quoted as the primary reason for the increase in the cadmium content of soils over the last 20 to 30 years in Europe (Jensen and Bro-Rasmussen, 1992).

Table 4.7: Cadmium content in soils from Kisumu region (mg/100 g)

Sample	Mean	Range	
		Minimum	Maximum
Dunga beach shoreline	0.13±0.05 ^b	0.12	0.16
2 km off the road	0.13±0.05 ^b	0.12	0.16
River Kisiat	0.09±0.01 ^c	0.07	0.13
Tilapia beach shoreline	0.15±0.00 ^a	0.13	0.18
Roadside	0.07±0.00 ^d	0.05	0.09
WHO maximum limits	5.00		

Each value is a mean ±SE of three analyses done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.001$) from each other.

Atmospheric cadmium emissions deposition onto soils has generally decreased significantly over that same time period (Cook and Morrow, 1995; Mukunoki and Fujimoto, 1996). Indeed, recent studies in Europe have documented that atmospheric emissions do not presently have a significant impact upon the cadmium content of soils (Bak *et al.*, 1997). Except in cases of extreme contamination, the concentration of cadmium in soils is not the primary determinant of cadmium in the human diet. For example, leafy vegetables and

potato tubers naturally accumulate higher levels of cadmium than do fruits and cereals (Mench, 1998). Moreover, tillage and crop rotation practices similarly have a greater impact upon the cadmium content of food than does the concentration of cadmium in soils (Mench, 1998). The WHO maximum allowable limits for cadmium in soil are 5 mg/100g. This limit was not exceeded by any of the soil samples analyzed.

4.3.3 Cadmium in fruits and vegetables from Kisumu region

The results of cadmium content in fruits and vegetables from Kisumu region are shown in Table 4.8. The tomatoes, kales and custard apple had significantly higher ($p < 0.05$) levels of cadmium as compared to the other fruits and vegetables analyzed. On the other hand, cow peas' leaves, arrow roots, and guava had significantly lower ($p < 0.05$) levels of cadmium. It was also observed that the cadmium levels in the leafy vegetables were very similar. Cadmium levels can vary widely in various types of foodstuffs (ATSDR, 1997). Leafy vegetables such as lettuce and spinach and certain staples such as potatoes and grain foods exhibit relatively high values from 3 to 15 $\mu\text{g}/100\text{ g}$ (WHO, 1992; ATSDR, 1997). The cadmium contents of foodstuffs may vary widely with the agricultural practices utilized in the particular areas such as phosphate fertilizer, sewage sludge and manure application, the types of crops grown, and atmospheric cadmium deposition from natural or anthropogenic sources.

Table 4.8: Cadmium content in fruits and vegetables from Kisumu region (mg/100 g)

Sample	Mean	Range	
		Minimum	Maximum
Spiderplant	0.12±0.05 ^a	0.00	0.31
Tomatoes	0.13±0.00 ^a	0.12	0.13
Black nightshade	0.07±0.01 ^b	0.05	0.07
Onions	0.07±0.00 ^b	0.07	0.08
Amaranth	0.04±0.00 ^b	0.02	0.06
Cow peas leaves	0.01±0.00 ^c	0.00	0.01
Kales	0.14±0.01 ^a	0.12	0.15
Arrow roots	ND ^c	ND	ND
Banana	0.05±0.00 ^b	0.05	0.06
Black berries	0.02±0.00 ^c	0.02	0.02
Guava	0.01±0.00 ^c	0.01	0.02
Oranges	0.02±0.00 ^c	0.01	0.03
Custard apple	0.13±0.00 ^a	0.12	0.13
WHO maximum limits	0.70		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

Therefore the cadmium content of terrestrial foods varies significantly as a function of the type of food crop grown, the agricultural practices pursued, and the

atmospheric deposition of cadmium onto exposed plant parts (ATSDR, 1997). This could explain the difference between the levels reported in the earlier study which were much lower than the levels reported in the current study. Earlier studies by Turzen (2003) indicated that the average level of cadmium in vegetables in Bangladesh was 0.18 mg/100 g. this was higher than the values reported in the current study. There are strong indications that cadmium levels in foodstuffs have substantially decreased during the past several decades due to the progressive control of cadmium emissions to the environment (Van Assche and Ciarletta, 1993; Watanabe *et al.*, 1993; Watanabe *et al.*, 1994). Recent studies have further documented that the cadmium content of food crops in Europe and many other countries are now stable and not increasing with time (Chaudri *et al.*, 1995). In earlier studies, levels in fruit and vegetables were reported to be usually below 1 µg/100 g (Galal-Gorchev, 1991). The level of cadmium reported in the fruits and vegetables in the current study were much higher than these. The WHO limit for cadmium in food is 0.7 mg/100 g. This was not exceeded by any of the fruits and vegetables analyzed.

4.3.4 Cadmium in maize and beans from Kisumu region

The results of cadmium content in maize and beans from Kisumu region are shown in Figure 4.6. The cadmium content in the maize was significantly higher ($p < 0.05$) than the beans. There was no significant difference ($p < 0.05$) in cadmium content in the different beans analyzed. Much of the cadmium which enters the body by

ingestion comes from terrestrial foods (Van Assche, 1998). This is to say, from plants grown in soil or meat from animals which have ingested plants grown in soil. Thus food plants together with the cadmium deposited out of the atmosphere on edible plant parts establishes the vast majority of human cadmium intake (Van Assche, 1998). Crops grown in polluted soil or irrigated with polluted water may contain increased concentrations, as may meat from animals grazing on contaminated pastures (IARC, 1976).

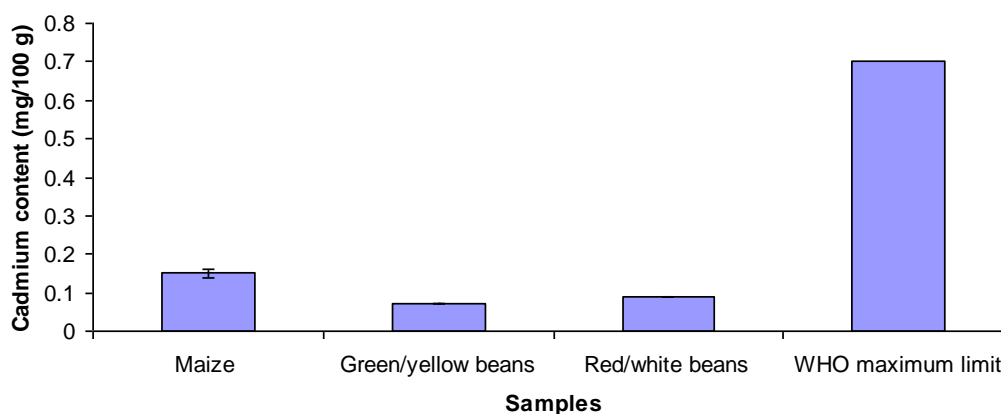


Figure 4.6: Cadmium content in maize and beans from Kisumu region (mg/100 g)

In cereals, levels are about 2.5 $\mu\text{g}/100\text{ g}$ wet weight have been reported (Galal-Gorchev, 1991). This is much lower than the levels reported in maize in the current study. There is considerable information in the literature regarding the cadmium contents of foods grown in contaminated areas (Elinder, 1985; WHO, 1992; OECD, 1994). Detailed studies have indicated that only a small percentage of these contaminated areas were actually utilized for growing foods which were subsequently consumed with the exception of rice fields in Japan where

considerable cadmium did find its way into the average person's diet through rice grown on contaminated rice fields (Elinder, 1985). In specific cases, management measures to reduce the transfer of cadmium from historically contaminated soils into the local food chain have proven successful (Staessen *et al.*, 1991). The WHO limit of cadmium in foods is 0.7 mg/100 g. this limit was not exceeded by the maize and beans analyzed in the current study.

4.3.5 Cadmium content in fish samples from Kisumu region

The results of cadmium content in fish from Kisumu region are shown in Figure 4.7. From the results of this study, the butterflyfish, Migori haplo and medium sized tilapia had significantly higher levels of cadmium ($p < 0.05$) as compared to the rest. The cadmium content was lowest in the small sized tilapia. However there was no significant difference between the sardines and the Nile perch ($p < 0.05$). It is however important to note that the levels of cadmium were generally very close. Some have estimated that 98% of the ingested cadmium comes from terrestrial foods, while only 1% comes from aquatic foods such as fish and shellfish, and 1% arises from cadmium in drinking water (Van Assche, 1998). In previous studies, average cadmium levels in fish were found to be 2 $\mu\text{g}/100\text{ g}$ wet weight. High levels were found in shellfish of 20–100 $\mu\text{g}/100\text{ g}$ (Galal-Gorchev, 1991). These levels are much lower than the levels reported in the current study. The WHO maximum allowable limit for cadmium in fish is 2.5 mg/100 g (WHO, 2004). This limit was not exceeded by any of the fish varieties analyzed.

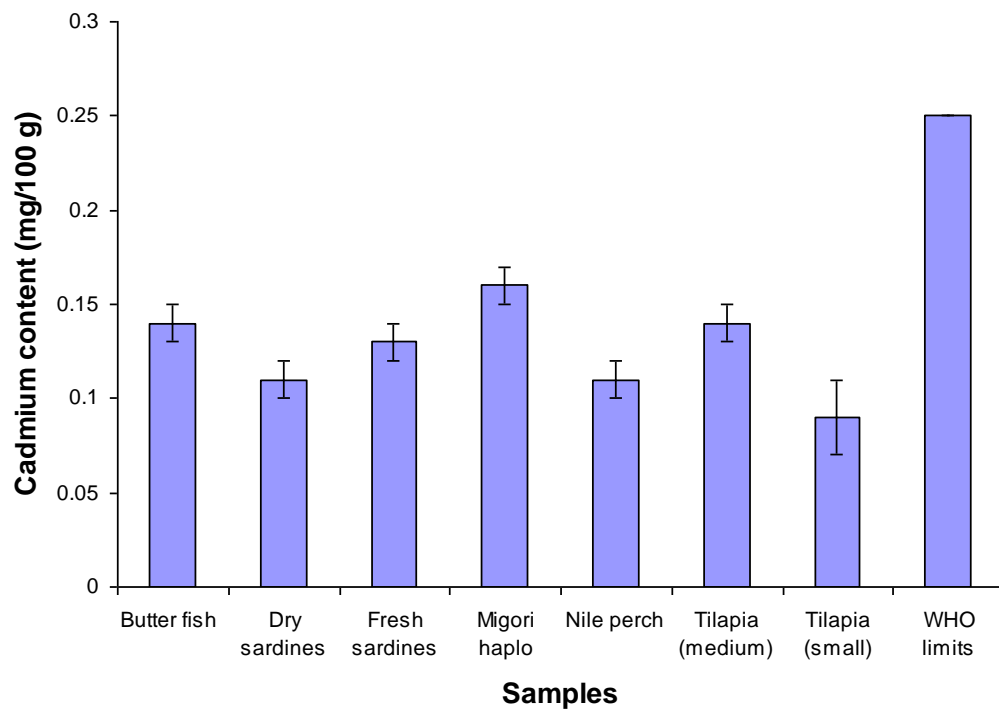


Figure 4.7: Cadmium content in fish from Kisumu region (mg/100 g)

4.4 Arsenic

4.4.1 Arsenic content in water samples from Kisumu region

The results of arsenic content in water from Kisumu region are shown in Table 4.9. The arsenic content of water from the tilapia beach shoreline was significantly higher ($p < 0.05$) from the other water samples. On the other hand the tap water did not contain any detectable amounts of arsenic and was significantly lower than the rest of the water samples ($p < 0.05$). This could be attributed to the water treatment process where much of the arsenic could have been removed together with the floccules during the sedimentation step. It was also noted that the arsenic content

in the waters from the beach and the lake inshore were significantly different ($p < 0.05$) at the two beaches (Tilapia and Dunga). The shoreline waters had significantly higher ($p < 0.05$) levels of arsenic than the inshore waters. This indicates that there is a reduction in arsenic content in the lake waters with increasing distance from the shoreline. This shows that the level of anthropogenic pollution is higher at the shorelines than in the lake.

Table 4.9: Arsenic content in water from Kisumu region (ng/100 ml)

Sample	Mean	Range	
		Minimum	Maximum
Dunga beach 200m inshore	2.0±0.10 ^d	1.60	2.70
Tap water	ND ^e	ND	ND
River Kisiat	1.7±0.60 ^d	0.00	3.90
Tilapia beach 200m inshore	4.2±0.50 ^c	2.70	5.80
Tilapia beach shoreline	13.1±1.10 ^a	9.10	16.80
Dunga shoreline	8.30±1.10 ^b	1.30	11.30
WHO minimum limits	1000		

Each value is a mean ±SE of three analyses each done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

Also noted was that the arsenic content in the River Kisiat waters was not significantly different from the arsenic content of the shoreline water from the two beaches (Dunga and Tilapia). This could be attributed to the fact that the water

from both sources had been exposed to pollution due to human activities at the two sources. The water from the Tilapia beach shoreline had significantly higher ($p < 0.05$) arsenic content than the Dunga beach shoreline waters. This could be attributed to the higher level of human activities at the Tilapia beach as compared to the Dunga beach. This is because the physical, chemical and biological processes occurring permanently in an aquatic environment should be considered to explain the inorganic elements and heavy metals concentrations (Nguyen *et al.*, 2005). External processes like discharging of pollutants and anthropogenic activities also affect the concentration and behavior of inorganic elements and heavy metals concentrations (Baeyens *et al.*, 1998). Human activities have increased the concentration of metals in many of the natural water systems which have raised concerns regarding metal bioaccumulation and human health hazards (Pan and Brugam, 1997).

In addition, conditions in the catchments and lake are important for the mobility and availability of metals in the water. Rivers and lakes are exposed to atmospheric deposition of anthropogenically derived trace elements. This can create harmful effects on environmental condition and human health due to their toxicity and bioaccumulation in various environmental compartments (Jarup, 2004, Silva *et al.*, 2005). WHO has recommended 10 parts per billion ($1 \mu\text{g}/100 \text{ ml}$) as the limit for arsenic in drinking water (WHO, 2003). From the results

obtained, all the water sources analyzed were within this limit ranging from 0.00 to 13.1 ng/100ml (0.00 to 0.013 $\mu\text{g}/100\text{ ml}$)

4.4.2 Arsenic content in soil samples from Kisumu region

The results of arsenic content of soils from Kisumu region are shown in Figure 4.8. From the results of this study, soil from R. Kisiat was found to have significantly higher ($p < 0.005$) levels of arsenic as compared to the rest of the soils. This could be due to higher anthropogenic activities around the river since there is bathing and washing of clothes taking place there. In soils contaminated through anthropogenic activity, arsenic contents may exceed 50 mg/kg (Ali *et al.*, 2003).

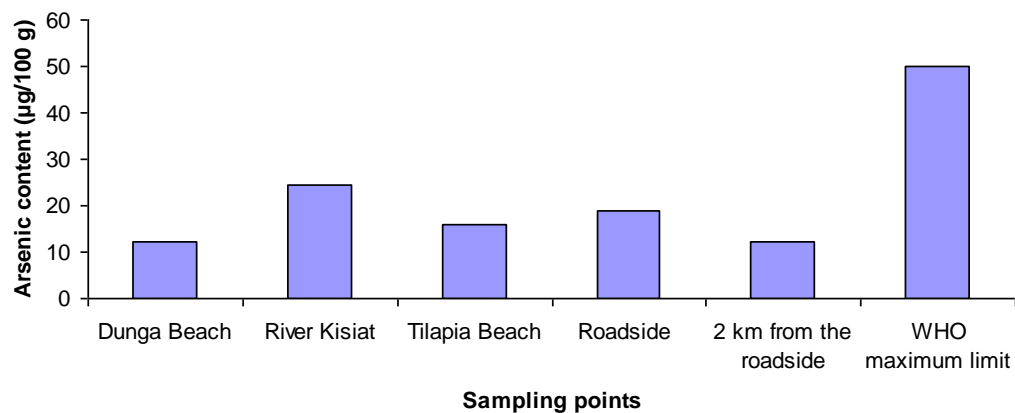


Figure 4.8: Arsenic content in soils from Kisumu region ($\mu\text{g}/100\text{ g}$)

The Dunga shoreline soil and 2 km from the highway were significantly lower ($p < 0.05$) in arsenic content than the roadside and river Kisiat soils. This is because both of them being further away from human activities, had lower anthropogenic

activities. It was also noted that there was no significant difference ($p < 0.05$) in the arsenic content in the soils from the two beaches (Dunga and Tilapia) which is an indicator of similarities in human activities at the two beaches. The roadside soils were not significantly different ($p < 0.05$) from the Tilapia beach soils. The two sites have high vehicular traffic thus their source of arsenic could be both anthropogenic as well as from the combustion of petroleum products whose combustion has been found to emit some arsenic (Ghelberg *et al.*, 1983). In a study in Bangladesh, concentration of arsenic in the irrigated soils varied from 0.32 to 2.75 mg/100 g. On the other hand, in the areas where irrigation-water did not contain arsenic, the soil arsenic varied from 0.01 to 0.28 mg/ 100g (Huq, 2006). The concentration of arsenic in the soils in the current study (0.012 to 0.024 mg/100 g) was much lower than those reported in the Bangladesh study with the water containing arsenic and within the same range with the water without arsenic. The WHO limit for arsenic in agricultural soils is 50 $\mu\text{g}/100\text{g}$ (WHO, 2003). This limit was not exceeded by any of the soil samples analyzed.

4.4.3 Arsenic content in maize and beans samples from Kisumu region

The results of arsenic contents of maize and beans from Kisumu region are shown in Figure 4.9. From the results obtained from this study, the arsenic content was significantly different ($p < 0.05$) between the maize and beans. Maize generally had significantly ($p < 0.05$) higher levels of arsenic. Among the two varieties of beans analyzed, there was a significant difference ($p < 0.05$) in the amount of arsenic.

Dietary arsenic represents the major source of arsenic exposure of most of the general population. The actual total arsenic concentrations in foodstuffs from various countries will vary widely depending on the food type, growing conditions (type of soil, water, geochemical activity, use of arsenical pesticides) and processing techniques (Khaliqzmann and Khan, 2003).

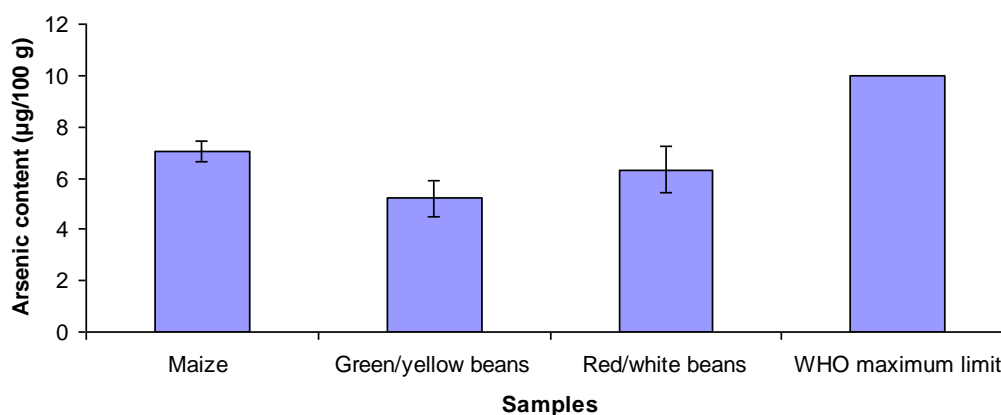


Figure 4.9: Arsenic content in maize and beans from Kisumu region (µg/100 g)

Yost *et al.*, 1998 reported arsenic content of 20 µg/100 g in cereals consumed in the Northern America diet. The findings of this study (5.2 to 7.0 µg/100 g) indicated a much lower level of total arsenic in the maize sold in the Lake Victoria region of Kenya. In the same North American study, Yost *et al.* (1998) found the total arsenic levels in pulses to be 20 µg/100g. This is much higher than the figures found in this study. In another study by Khaliqzmann and Khan (2003) in Bangladesh, the level of arsenic in both cereals and pulses was established as 20 µg/100g which is much higher than the levels found in this study. The WHO limit set for cereals is 10µg/100 g (WHO, 2003) which was not exceeded by the maize

purchased from Kisumu market. The WHO limit set for pulses is also 10 µg/100 g (WHO, 2003) which was also not exceeded by the two varieties of beans studied in this study. The beans are also safe for human consumption.

4.4.4 Arsenic content in fruits and vegetables samples from Kisumu region

The results of arsenic content in the fruits and vegetables from Kisumu region are shown in table 4.10. From the results obtained, the onions, oranges, black berries and black nightshade had significantly higher ($p < 0.05$) arsenic content than the rest of the fruits and vegetables analyzed. On the other hand amaranth, arrow roots, kales and traditional vegetables had significantly lower ($p < 0.05$) levels of arsenic as compared to the rest. The oranges had no significant difference ($p < 0.05$) with the bananas and guavas. Among the underground crops, the onions had significantly higher arsenic levels compared to the arrow roots ($p < 0.05$). There was no significant difference between the fruits and vegetables ($p < 0.05$) as they were represented evenly in all the arsenic ranges. Naidu (Naidu, 2001) reported 0.64 µg/100g arsenic content in fruits. This is also much lower than the arsenic content found in fruits in the Lake Victoria study which ranged from 4.20 µg/ 100 g to 7.19 µg/100 g. Other studies have shown that arsenic content in plants varied considerably with type of plants, type of soil, and arsenic content of irrigation-water. Generally, the highest concentrations of arsenic were always recorded in plant-roots, and this may be attributed to contamination from fine colloidal

particles (Abedin *et al.*, 2002; Hironaka and Ahamad, 2003; Alam and Rahman, 2003).

Table 4.10: Arsenic content in fruits and vegetables from Kisumu region ($\mu\text{g}/100\text{ g}$)

Sample	Mean	Range	
		Minimum	Maximum
Spiderplant	3.94 \pm 0.08 ^{de}	3.64	4.25
Tomatoes	4.56 \pm 0.06 ^{cd}	4.39	4.67
Black nightshade	7.34 \pm 0.07 ^a	7.16	7.50
Onions	6.90 \pm 0.10 ^a	6.71	7.17
Amaranthus	2.89 \pm 0.10 ^e	2.40	3.39
Cow peas leaves	5.96 \pm 0.32 ^{ab}	5.07	7.28
Kales	3.85 \pm 0.16 ^{de}	3.59	4.29
Arrow roots	2.90 \pm 0.51 ^e	1.43	4.55
Banana	4.43 \pm 0.04 ^{cd}	4.32	4.49
Black berries	6.96 \pm 0.09 ^a	6.77	7.20
Custard apple	5.51 \pm 0.06 ^{bc}	5.34	5.62
Guava	4.31 \pm 0.05 ^{cd}	4.20	4.43
Oranges	6.99 \pm 0.08 ^a	6.79	7.19
WHO limit	10.00		

Each value is a mean \pm SE of three analyses done in triplicates.

Means within columns followed by different letters are significantly different ($p < 0.05$) from each other.

Debeka *et al.*, 1993 carried out a survey of the arsenic intake by Canadian children and found out the vegetable consumed in Canada had arsenic content ranging from 0.28 $\mu\text{g}/100\text{ g}$ to 0.7 $\mu\text{g}/100\text{ g}$. The arsenic content of the vegetables analyzed in the current study were much higher than those reported in the Canadian study since they ranged from 1.43 $\mu\text{g}/100\text{ g}$ to 7.50 $\mu\text{g}/100\text{ g}$. In another study by Naidu (2001), figures similar to the Canadian study were found and these were also much lower than those found in the current study. In a Bangladesh study the arsenic content in vegetables was 2.8 to 7.0 $\mu\text{g}/100\text{ g}$. This was similar to the arsenic content in vegetables found in the current study. In another study in Bangladesh, the arsenic content of tomatoes was found to be 400 $\mu\text{g}/100\text{ g}$ which is 100-fold higher than those found in this study (4.5 $\mu\text{g}/100\text{ g}$). In the same study, the arsenic content of arrow roots was found to be 2000 $\mu\text{g}/100\text{ g}$ which is also much higher than those found in this study. In the same study the arsenic content of the onions was found to be 5 $\mu\text{g}/100\text{ g}$ which was similar to the arsenic content of onions in the current study (Huq *et al.*, 2006). The WHO limits for arsenic in foods including fruits and vegetables is 10 $\mu\text{g}/100\text{ g}$ (WHO, 2003). This limit was not exceeded by any of the fruits and vegetables analyzed in the current study.

4.4.5 Arsenic content in fish samples from Kisumu region

The results of arsenic content in fish from the Kisumu region are shown in Figure 4.10. The dry sardines (dagaa) had significantly higher levels ($p < 0.05$) of arsenic as compared to the rest of the fish samples. Most of the other fish samples were

not significantly different ($p < 0.05$) from each other. The tilapia of the two different sizes (small and medium) were not significantly different ($p < 0.05$) from each other. The fresh sardines had significantly lower levels of cadmium ($p < 0.05$) as compared to the dried sardines' sample. This could be attributed to the concentration of the arsenic during drying process. The difference between the fresh and dry sardines could also be attributed to exposure to anthropological contamination during drying and display in the market.

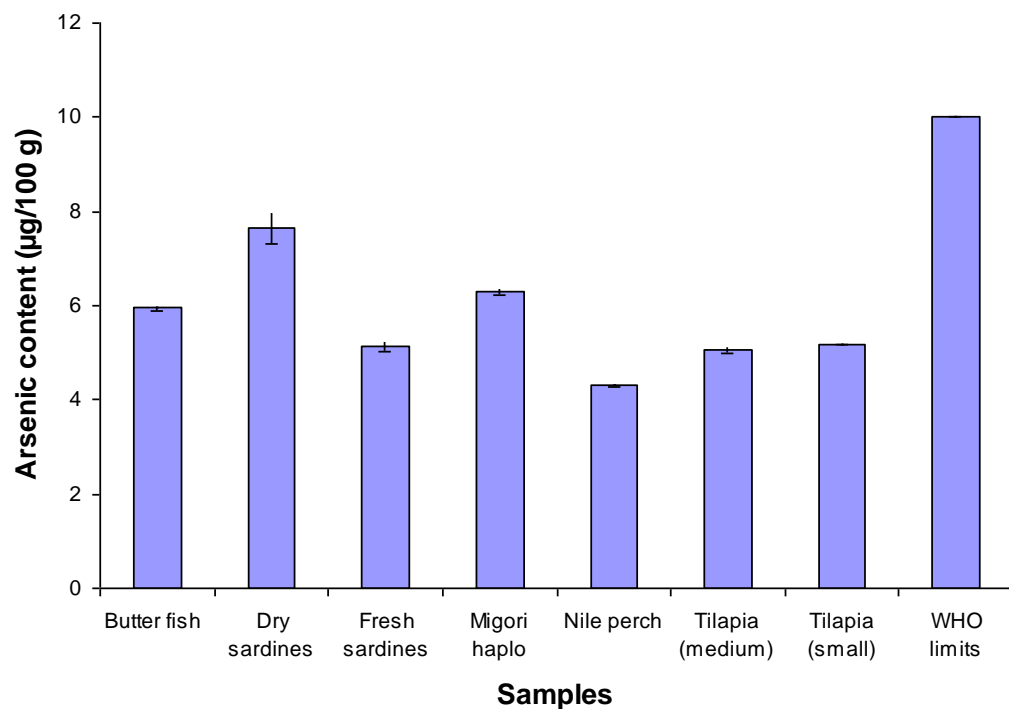


Figure 4.10: Arsenic content in fish from Kisumu region (µg/100 g)

In a North American study in 1993, Debeka *et al.* found arsenic average levels of 166.2 µg/100 g in fish and shell fish (Debeka *et al.*, 1993). This was much higher

than the average value of 1.43 to 7.60 $\mu\text{g}/100\text{ g}$ in fish found in this study. In another study in North America, Yost *et al.* found arsenic levels of 110 $\mu\text{g}/100\text{ g}$ in fish (Yost *et al.*, 1998). This was also much higher than the levels found in the current study. The Australian limit of 1 mg/kg (100 $\mu\text{g}/100\text{ g}$) dry weight was set with regard to the high seafood intake of Australian people, as seafood contains elevated amounts of arsenic. However, most arsenic in this seafood is organic (Leelphart *et al.*, 2004). This limit is also much higher than the findings of the current study. The WHO limit for arsenic in fish is 10 $\mu\text{g}/100\text{ g}$ (WHO, 2003). This limit was not exceeded by any of the fish types analyzed in the current study. The fish is thus safe for human consumption.

CHAPTER 5:

CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

The waters from Kisumu region, which were sourced from L. Victoria, River Kisiat and taps were within the WHO limits for mercury, cadmium and arsenic. The tap water samples were the only ones that met the WHO limits for lead. There were variations based on the sources. Geographical location was noted to cause variations especially with the L. Victoria water whose heavy metal contents were found to be different at the two beaches, Dunga and Tilapia Beach. The distance from the shoreline was also noted to cause variations, with the inshore waters containing lower heavy metal contents.

The soils from Kisumu region were found to be within the WHO limits for mercury, cadmium and arsenic. The roadside soils samples contained higher lead contents than the WHO limit. However, there were differences due to geographical location with the soils from areas with high anthropological activities containing higher heavy metal loads while those from areas with lower anthropological activities contained lower heavy metal loads.

The maize from Kisumu region was found to be within the WHO limits for lead, cadmium and arsenic in foods. However, it was found to contain slightly higher mercury content than the WHO limits for mercury in foods.

The beans from Kisumu region were found to be within the WHO limits for lead, mercury, cadmium and arsenic in foods. However, there were variations in the heavy metals contents from one heavy metal to another with some being significantly higher than others.

The fish from Kisumu region was found to be within the WHO limits for lead, mercury, arsenic and cadmium. However, there were variations in heavy metal contents between the species of fish studied.

The fruits and vegetables from Kisumu region were found to be within the WHO limits for arsenic and cadmium. However, there were differences in the arsenic and cadmium contents between the different species of fruits and vegetables studied.

Eight of the thirteen different fruits and vegetables studied had lead contents above the maximum WHO limit for lead in foods. The remaining five were within the limits. It was observed that the lead content variations were very large between the different fruits and vegetables studied.

Six of the thirteen different fruits and vegetables analyzed had higher mercury content than the maximum WHO limit for mercury in foods. The remaining seven were within the WHO limits. The mercury content variation were also very big between the different types of vegetables studied.

5.2 Recommendations

It is recommended that further research be done on the effect of food preparation on heavy metal levels in the foods. Such work should focus on the effect of common food preparation methods such as washing, soaking and boiling on heavy metal concentrations in the foods, to determine if they are significantly affected by these preparation methods.

It is recommended that a continuous monitoring of heavy metal levels of water, soil and foods be put in place since there could be seasonal variations in their levels. It is also recommended that a study be carried out to determine the levels of heavy metals in human tissue. It is also recommended that a study be carried out to determine other sources of heavy metals other than water and soils studied in the current study, especially air. Further studies should also be carried out to determine the parameters causing the apparent variations in the heavy metals content in the different species of fruits and vegetables analyzed.

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