UNIVERSITY OF CAPE COAST

MERCURY AND CADMIUM BUDGETS OF THE PRA ESTUARY IN THE WESTERN REGION OF GHANA

BY

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A Thesis submitted to the Department of Chemistry of the School of Physical Sciences, University of Cape Coast in partial fulfillment of the requirement for the award of Master of Philosophy (M.Phil) in Chemistry.

November 2007

DECLARATION

Candidate's Declaration

I herby declare that this thesis is the result of my own original work and that no part of it has been presented for another degree in this University or elsewhere.

Candidate's Signature..... Date.....

Name: ARKOFUL SAM

Supervisor's Declaration

We hereby declare that the preparation and presentation of this thesis were supervised in accordance with the guidelines on supervision of thesis laid down by the University of Cape Coast.

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ABSTRACT

Instrumental Neutron Activation Analysis was employed for the determination of mercury and cadmium in soil, water and fish samples from eight different sites of the Pra and Ayensu estuaries in Ghana. Mercury and cadmium was identified in μg/g levels and values correlated with the pH's of water column and soil samples. Substrates like the Blue tilapia and the European Green Crab were used as bioaccumulation indicators for the mercury levels in the various samples. A summary of the mean, minimum and maximum soil / water mercury and cadmium concentrations detected for the eight (8) sites, with 10km geographical distribution; river bank at Beposo showed a maximum of 3.95μg/g of mercury, which is below the Environmental Protection Agency's permissible limit of 0.134mg/g(134.0μg/g). Insignificantly low levels of mercury concentrations was analyzed in shoulder soils and water samples over the period of the study. A significantly high levels of concentrations of mercury existed in the riverbed sediments compared to that for the riverbank sediments, water and the shoulder soils. The degree of concentrations of mercury showed that, mercury and cadmium concentrations decreased significantly and gradually as one moved from Beposo to the Shama Beach through Bosomdo and Krobo.

The study showed a maximum mercury and Cadmium residue of both the European green Crab and the Blue tilapia. While the maximum mercury and

cadmium residue reported for the Tilapia were not consistently as high as those for the Crab. The maximum residue in the European Green Crab in three (3) reporting sites did not exceed the EPA action level (0.134 mg/g). The Crab had a higher body burden of mercury because it is a bottom –dwelling/feeding and predatory species.

ACKNOWLEDGEMENTS

It is my pleasure to acknowledge the valuable assistance and criticism received from friends, especially Nutifafa Doe and Christian Adokoh and other colleagues in the compilation of this work. Special acknowledgement and appreciation for information, help and guidance with sincere gratitude to my co-supervisor Mr. David Kofi Essumang who assisted in the preparation and research of this work.

I am indebted to the following for information: Mr. Opata, Mr. Adotey Dennis and all the staff in the Chemistry Department of the Ghana Atomic Energy Commission.

Throughout the preparation of this thesis, I have been loyally sustained and assisted by my Principal supervisor: Rev. Prof. D.K. Dodoo, of Chemistry Department of the University Of Cape Coast. I say thank you for your help and encouragement.

DEDICATION

To my wife, Christy, and my boys: Kobby and

K.K Arkoful-Sam

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LIST OF ACRONYMS

MCA- Multi Channel Analyser

IAEA- International Atomic Energy Commission

GAEC- Ghana Atomic Energy Commission

NNRI- National Nuclear Research Institute

Am-Be- Americium- Beryllium

WHO- World Health Organisation

EPA- Environmental Protection Agency

USDOE- United States Department of Energy

TWA- Time Weighted Average

ATL- Alanineaminotransferase

VOC's- Volatile Organic Compounds

AAS- Atomic Absorption Spectrometry

IUPAC- International Union of Pure and Applied Chemistry

NIST- National Institute of Standards and Technology

NAA- Neutron Activation Analysis

CHAPTER ONE

INTRODUCTION

General environmental issues

While there are many things to appreciate and celebrate about the world in which we live many pressing environmental problems cry out for our attention.

Food shortages and famines already are too familiar in many places and may increase in frequency and severity of population growth. Soil erosion and soil nutrient depletion continue at the same rate in the present as they have been in the past and we expect them to continue if nothing is done about them. Water shortage and contamination of existing water supplies threaten to be a critical environmental issue for agricultural production as well as for domestic and industrial uses. Many countries already have serious water contamination problems and more than one billion people lack access to clean water or adequate sanitation (Cunning, et al. 1997).

Toxic air and water pollutants, along with mountains of solid and hazardous wastes are becoming overwhelming problems in both the industrialized and developing countries. Mankind produces hundreds of millions of tons of dangerous materials annually, and much is disposed off in dangerous and irresponsible ways. No one wants this noxious stuff dumped in

his or her own backyard; but too often the solution is to export or transport it to someone else's compound. The health effects of pollution, toxic wastes, stress and other environmental ills of modern society have become a greater threat than infectious diseases for many of us in developing countries (Cunning, et al. 1997).

Local environmental issues

Ghana's 2.4% population growth rate and doubling time of 29 years, puts her among the countries with fast expanding population. With a population of about 21 million, it is expected that Ghana's population would hit more than 50 million by the middle of this century. Dependency on free-access natural resources for sustenance is the only hope for survival for a large proportion of the population. The resultant effects of this are massive over-exploitation of environmental resources such as the Forests and Savannah, Wildlife and Coastal resources (Asabere-Ameyaw, et al 2004).

The coastal waters are being degraded as a result of increasing organic and inorganic pollution from a variety of land-based and maritime activities. An important land-based activity that has the potential of polluting water is mining. Chemicals such as cyanide and mercury used in extracting gold are injurious to biotic life. Such chemicals eventually enter water systems to pollute them. Also illegal mining activities such as "galamsey," which use mercury to extract gold, pollute nearby water bodies.

The mercury used in the mining operations just upstream enter rivers, such as the Pra River, trickling down gradually through the estuary into the sea, if natural adsorption processes do not take place(Asabere-Ameyaw, et al 2004).

Natural occurrence of mercury

Mercury is a naturally occurring element that is found in air, water and soil. It exists in several forms: elemental or metallic mercury, inorganic and organic mercury compounds. Mercury is a shiny, silver-white metal and is a liquid at room temperature. At room temperature, exposed elemental mercury can evaporate to become an invisible, odourless and toxic vapour (www.epa.gov/mercury/faq.htm)

Inorganic mercury compounds take the form of mercury salts and are generally white powder or crystalline, with the exception of mercuric sulphide (cinnabar). Inorganic mercury compounds have been included in products such as fungicides, antiseptics or disinfectants. Some skin lightening and freckle creams, as well as some traditional medicines, do contain some mercury compounds (www.epa.gov/mercury/faq.htm).

Organic mercury compounds, such as methyl mercury are formed when mercury combines with carbon. Microscopic organisms convert inorganic mercury into methyl mercury, which is the most common organic mercury compound found in the environment. Methylmercury accumulates up the food chain (www.epa.gov/mercury/faq.htm).

Occurrence of mercury in the environment

Mercury is a naturally occurring element that can be found throughout the environment. Human activities, such as burning of coal and use of mercury to produce gold "galamsey", have increased the amount of mercury in many parts of the environment including the atmosphere, the abiosphere and the hydrosphere.

Exposure of mercury to human beings and/wildlife

The primary way by which the people in Ghana are exposed to mercury is by consuming fish containing methylmercury or through "galamsey" operations. Mercury in the atmosphere is eventually deposited to the earth's surface, either through dry deposition or by wet deposition (rain). When mercury falls from the air or runs off from the ground into water bodies, certain microorganisms in soils and sediments convert some part of it into methyl mercury, a highly toxic form of mercury.

Small organisms take up methylmercury as they feed. When animals higher up the food chain eat the smaller ones, they also take in the methylmercury. As this process (Known as bio-magnification), continues, the level of methyl mercury increases up the food chain. Fish that are higher in the trophic level of the food chain, such as sharks and swordfish, tend to concentrate higher methylmercury concentrations than fish in the lower trophic level on the food chain. This is true for both saltwater and freshwater fish. People and fish-eating wildlife therefore become exposed when they eat fish

and shellfish that contain methyl mercury (www.epa.gov/mercury/exposure.htm).

Mercury and human health

High levels of mercury in the blood stream of unborn babies and young children may harm the developing nervous system. Whether an exposure to mercury will harm a person's health depends on a number of factors. Almost all people have at least trace amounts of mercury in their tissues, reflecting mercury's widespread presence in the environment (www.epa.gov/mercury/faq.htm). People may be exposed to mercury in any of its forms under different situations. The factors that determine the severity to health effects from mercury exposure include:

* The dose – how much
* The duration of exposure – how long
* The route of exposure – eating, breathing, injecting, touching
* Other chemical exposures
* The specific characteristics of the person – age, health, weight etc.

Mercury compounds are much more readily absorbed through the gastrointestinal tract and skin. High exposures to mercury compounds can damage the gastrointestinal tract, the nervous system and the kidneys (www.atsdr.cdc.gov/toxprofiles/).

Uses of mercury

Mercury is used in dentistry in dental amalgam. Dental amalgam is a direct filling material used in restoring teeth. It is made up of approximately 40

– 50% mercury, 25% silver and 25-35 a mixture of copper, Zinc and tin. Amalgam use is declining because the incidence of dental decay is decreasing and because improved substitute materials are now available for certain applications. In Ghana, mercury use is prominent in the mining areas, where the "galamsey" operators use it to extract gold. Mercury is also used in thermometers, fluorescent light bulbs and some electrical switches. Inorganic mercury and organic mercury compounds such as phenyl mercury, ethyl mercury has been included in products such as fungicides, antiseptics or disinfectants. Some skin lightening and freckle creams also contain mercury (www.epa.gov/mercury/exposure.htm).

Cadmium (Cd)

Cadmium is a metallic element belonging, together with zinc and mercury, to group IIb of the periodic table. Some cadmium salts, such as the sulphide, carbonate, and oxide, are practically insoluble in water; these can be converted to water-soluble salts in nature. The sulphate, nitrate, and halides are soluble in water.

The average cadmium content of sea water is about 0.1µg/litre or less. River water contains dissolved cadmium at concentrations of between < 1 and 13.5ng/litre. In remote, uninhabited areas, cadmium concentrations in air are usually less than 1ng/m^3. In areas not known to be polluted, the median cadmium concentration in soil has been reported to be in the range of 0.2 to 0.4 mg/kg. However, much higher values, up to 160mg/kg soil, are occasionally found (EHC, 135:1992)

Environmental factors affect the uptake and, therefore, the toxic impact of cadmium on aquatic organisms varies. Increasing temperature increases the uptake and toxic impact, whereas increasing salinity or water hardness decreases them. Freshwater organisms are affected by cadmium at lower concentrations than marine organisms. The organic content of the water generally decreases the uptake and toxic effect by binding cadmium and reducing its availability to organisms. However, there is evidence that some organic matter may have the opposite effect (Gottofrey, 1988; Block, 1991).

Importance of the study

Most rivers can be found close to regional and district capitals, which serve as a tourism destination for many visitors. As such the gradual pollution and contamination of the rivers must be of prior concern to all well meaning Ghanaians and not only the central government.

This study is expected to provide (the Central Government, Environmental Protection Agency, Shama Ahanta District Assembly, Ghana Tourist Board and various Research Institutions) the following:

(1) Baseline levels of Mercury and Cadmium in the Pra Estuary.

(2) Give detailed information of mercury and Cadmium distribution in the Estuary.

(3) Recommend in the implementation of environmental sustainable policies on river Pra

(4) Update data in this study area.

Statement of the problem

The activities of the people living and working around the Pra basin contribute much to the pollution and contamination of the river. At the various communities up-stream such as Dunkwa-on- Offin, there are clusters of "galamsey" operators who have cited their indigenous companies close to the river. Almost all the mercury used in their extraction process settles into water bodies and affects the water quality (Adimado, A.A, et.al. 2001). The mercury falls to the ground in raindrops, in dust, or simply by gravity (known as air disposition) as fallout. The mercury droplets/aerosols end up in streams, lakes and estuaries; where it's transformed to methyl mercury through microbial activity. The processes of complexation, precipitation, adsorption etc. also lead to its transformation into methylmercury.

There is therefore an urgent need to study the amount of mercury brought from upstream, into the sediments, water column and the soils at the shoulders of the Pra basin as a results of the activities of these operators and the erstwhile Offin Continental Goldfields. The harmful nature of mercury and cadmium in the environment has therefore called for this work and as far as the biota and sustenance of the river is concerned.

Objectives of the study

The objectives of this work are to: (i) determine the budgets and the distribution of mercury and cadmium in the Pra Estuary using the Neutron Activation technique.

(ii) Study the bio-concentration of mercury and cadmium in fish and crab and the contribution of pH to the bio-concentration.

(iii) Make recommendations to official Government bodies for effective regulation on the use of mercury in the Ghanaian environment. Also, to provide a source of contamination data if any for the Ghana Water Company, tapping the Pra River for domestic and industrial production of water.

CHAPTER TWO

LERATURE REVIEW

Health effects of mercury

Mercury is a metal that occurs naturally in the environment. Metallic or elemental mercury (Hg^0) is the main form of mercury released into the air by natural processes. Mercury bound to other chemicals may have valence states of either +1 (Hg^{+1}) or +2 (Hg^{2+}). Mercury with a valence state of +1 is referred to as mercurous mercury, and mercury with a valence state of +2 is referred to as mercuric mercury. Many inorganic and organic compounds of mercury exist from the mercuric (divalent) cation (Hg^{2+}).

. The general public is most commonly exposed to mercury primarily from two sources:

(1) Eating of fish (e.g. tilapia and crabs) and marine mammals (e.g., whales, seals) that may contain some methylmercury in their tissues or

(2) From the release of elemental mercury from the dental amalgam used in tooth fillings. It is not known how much of the elemental mercury released from dental amalgam is inhaled as a mercury vapour, how much is breathed out, how much is swallowed in a liquid form, or how much is converted into a mercuric salt that is either swallowed directly absorbed into the oral mucosa. Animals at the top of the food chain tend to accumulate the most

methylmercury in their bodies. Any type of mercury released into the environment may, therefore, lead to increased levels of methylmercury in tissues of large fish and mammals. Occupational exposures are primarily to metallic mercury vapour.

Accidental exposures to mercury are more common than accidental exposures to many hazardous substances, because liquid mercury has been used in many electrical, mechanical and thermometers. Accidental exposures, even to small amounts of mercury, may be harmful. Liquid mercury is poorly absorbed by the skin and from the intestines, but vapours that are released from liquid mercury are readily absorbed through the lungs and are very harmful when inhaled. The literature on the health effects of mercury is extensive. However, the human and animal data are generally limited to inhalation exposure to metallic mercury vapours and oral exposure to inorganic and organic mercury compounds. There is limited dermal exposure information on adverse effects from ointments and creams that contain inorganic mercury compounds. Once absorbed, metallic and inorganic mercury enter the oxidation-reduction cycle.

Metallic mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs of humans and animals. Evidence from animal studies suggests that the liver is an additional site of oxidation. Absorbed divalent cation from exposure to mercuric compounds can, in turn, be reduced to the metallic or monovalent form and released as exhaled metallic mercury vapour. In the presence of protein sulfahydroyxl groups, mercurous mercury (Hg^{+})

disproportionate to one divalent cation (Hg^{2+}) and one molecule at the zero oxidation state (Hg^0). The conversion of methylmercury and phenyl mercury into divalent inorganic mercury can probably occur soon after absorption (www.atsdr.cdc.gov/toxprofiles/).

Discussion of health effects by route of exposure

To help public health professionals and others address the needs of persons living or working near hazardous waste sites, the information in this section is organized first by route of exposure — inhalation, oral, and dermal; and then by health effect — death, systemic, immunological, neurological, reproductive, developmental, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods — acute (14 days or less), intermediate (15–364 days), and chronic (365days or one year).

Inhalation exposure

Most of the studies on inhalation exposure concern exposure to metallic mercury vapour. For this reason, the term "metallic mercury" will be used in this section instead of "inorganic mercury." Other forms of inorganic mercury do not pose a risk by the inhalation pathway. Inhalation of sufficient levels of metallic mercury vapour has been associated with systemic toxicity in both humans and animals. The major target organs of metallic mercury-induced toxicity are the kidneys and the central nervous system.

At high exposure levels, respiratory, cardiovascular, and gastrointestinal effects also occur. Some metallic mercury vapour may condense or in the case of vapours from dental amalgam, may dissolve in saliva and be ingested

(WHO, 1991). Condensed droplets are more likely to be ingested than inhaled (resulting in a lower absorbed dose than would be expected for a given concentration in air). Mercury vapour concentrations in the general work environment may also be lower than those in the microenvironment immediately surrounding workers.

No studies were found concerning effect levels following inhalation exposure to inorganic salts of mercury (e.g., mercuric or mercurous salts, oxides). Also, much of the information regarding effects of metallic mercury vapours or volatile organic compounds (VOCs) come from studies with significant limitations. Information on inhalation exposure to organic mercury compounds (e.g., alkyl mercury compounds) in humans is limited to case reports and includes only qualitative data on gastrointestinal, renal, muscular, and neurological effects. In many cases, it is difficult to determine whether effects observed in exposed persons were directly attributable to mercury exposure. In addition, a great deal of the information on effects associated with inhalation exposure to metallic mercury vapour come from studies conducted several decades ago, when methods for determining exposure levels were less precise than current methods (WHO, 1991).

Respiratory effects

Metallic Mercury: In humans, respiratory symptoms are a prominent effect of acute-duration high-level exposure to metallic mercury vapours. The most commonly reported symptoms include cough, and tightness or burning pains in the chest (Bluhm et al. 1992a; Gore and Harding, 1987). The decreased

vital capacity observed by Lim et al. (1950) persisted for 1 month after

exposure. In the more severe cases, respiratory distress, pulmonary edema (alveolar and interstitial), lobar pneumonia, fibrosis, and desquamation of the bronchiolar epithelium have been observed. The ensuing bronchiolar obstruction by mucus and fluid results in alveolar dilation, emphysema, pneumothorax, and possibly death (Gore and Harding, 1987).

Little information is available regarding exposure levels resulting in the above symptoms. Very little information regarding respiratory effects associated with intermediate-duration exposures. However, two studies noted chronic coughs in subjects exposed to metallic mercury vapour for several weeks (Schwartz et al. 1992). No respiratory symptoms and no abnormalities were noted upon examining chest X-rays or the results of pulmonary function tests in a group of chloroalkali workers exposed for an average of >6 years to levels of mercury ranging from near 0 to 0.27 mg/m^3 (85% of the group was exposed at or below 0.1 mg/m^3) (Smith et al. 1970).

Respiratory effects in animals have been observed following acute inhalation exposure of metallic mercury vapours. Rats exposed to 27 mg/m^3 of elemental mercury vapours for 2 hours then observed for 15 days displayed dyspnea and death due to asphyxiation (Livardjani et al. 1991b). Respiratory tract lesions included lung, edema, necrosis of the alveolar epithelium and hyaline membranes, and occasional lung fibrosis.

Exposure to 28.8 mg/m^3 of mercury vapour lasting from 1 to 20 hours produced effects ranging from mild to moderate pathological changes (unspecified) (Ashe et al. 1953). For exposures lasting 30 hours, marked cellular degeneration and some necrosis were observed in the lungs of 1 rabbit. Less severe respiratory changes (unspecified mild-to-moderate pathological changes) were reported in rabbits following exposure to metallic mercury vapour at 6 mg/m^3 for 7 hours a day, 5 days a week for 1–11 weeks (Ashe et al. 1953). The usefulness of these results is limited because the study did not specify the pathological changes nor distinguished between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Congested lungs were observed in rats exposed to 1-mg/m^3 metallic mercury vapours for 100 hours continuously per week for 6 weeks (Gage, 1961). In rats exposed to 3-mg/m^3 mercury vapours for only 3 hours a day, 5 days a week for 12–42 weeks, pathological examination revealed no significant changes in the respiratory system (Kishi et al. 1978). The potential for oral exposure was not quantified in these studies; however, it is likely that most of the exposure was via inhalation.

Organic Mercury: Dyspnea, respiratory depression, and respirations frequently obstructed by mucus were observed in a farmer who had treated grain with phenylmercuric acetate for several seasons (Brown, 1954).

No studies were located regarding respiratory effects in animals after inhalation exposure to organic mercury.

Metallic Mercury: Increases in heart rate and blood pressure have been reported following inhalation exposure to metallic mercury in humans. Acute inhalation exposure to high concentrations of metallic mercury vapour generated by heating metallic mercury resulted in increased blood pressure (Snodgrass et al. 1981) and heart rate/palpitations (Bluhm et al. 1992a; Jaffe et al. 1983). In one of these cases, the increase in heart rate was characterized as a sinus trachycardia (Soni et al. 1992). Exposures of longer durations due to spills or occupational exposures have also been reported to result in increased blood pressure (Fagala and Wigg 1992; Foulds et al. 1987; Karpathios et al. 1991; Taueg et al. 1992) and increased heart rate (Fagala and Wigg 1992; Foulds et al. 1987). A single case report was located regarding cardiovascular effects resulting from inhalation of mercury vapours released from a paint that contained a high level of phenylmercuric acetate (Aronow et al. 1990). The affected child was diagnosed with acrodynia and exhibited a rapid heartbeat and hypertension.

Chronic-duration occupational exposures, however, have given mixed results regarding effects on blood pressure and heart rate. Two studies of workers exposed to relatively low levels of mercury (near 0–0.27 mg/m^3 in one study and an average of 0.075 mg/m^3 in the other) for an average of greater than 6 or 7 years showed no effects on blood pressure or electrocardiography (Schuckmann 1979; Smith et al. 1970).

In contrast, workers exposed to an estimated 0.03 mg/m^3 of mercury vapour (estimate based on blood levels) for at least 5 years reported an increased incidence of palpitations, and cardiovascular reflex responses were slightly reduced compared to unexposed matched controls (Piikivi 1989). Also, workers in a thermometer plant had a high incidence of hypertension (5 of 9 workers) (Vroom and Greer 1972). A morbidity and mortality study of chloralkali workers showed an increased likelihood of death due to ischemic heart and cerebrovascular disease (Barregard et al. 1990). These studies are limited, however, because exposure to other chemicals may have contributed to the effects observed, exposure levels may have been estimated from only a few actual determinations, and other risk factors were not consistently considered.

Significant increases in systolic blood pressure and diastolic blood pressure were found in volunteers with dental amalgam containing mercury when compared to a control group (matched for age and sex) that had no amalgam fillings (Siblerud, 1990). However, the length of time that the individuals had the dental amalgams was not reported. Furthermore, the blood pressure levels of the amalgam group were closer than those of the nonamalgam group to "normal" blood pressure levels reported for the general population. The authors suggested that the populations from which such normal values are drawn are likely to include many people with amalgam dental fillings, but without additional data to determine which control group would best represent "normal," these results have limited use.

In animals, cardiovascular effects were noted following inhalation exposure to mercury vapour. Marked cellular degeneration with some necrosis of heart tissue was observed in rabbits following acute intermittent exposure to 28.8-mg/m^3 metallic mercury vapour for periods ranging from 4 to 30 hours (Ashe et al. 1953). Mild-to-moderate pathological changes (unspecified) were seen for 1–4-hour exposures. Exposures to lower concentrations (0.86–6 mg/m^3) of mercury vapour for periods ranging from 2 to 12 weeks also resulted in mild-to-moderate pathological changes (unspecified) in the hearts of rabbits. The usefulness of these results is limited because the study did not specify the pathological changes nor distinguish between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Organic Mercury: Only two case histories were found regarding cardiovascular effects in persons exposed by inhalation to organic mercury compounds. No cardiovascular effects were reported in four men hospitalized for neurological symptoms after inhaling an unspecified concentration of methylmercury dust for at least several months (Hunter et al. 1940). Elevated blood pressure was reported in two men exposed occupationally to methylmercury compounds (dose not known) (Hook et al. 1954).

No studies were found regarding cardiovascular effects in animals after inhalation exposure to organic mercury.

Gastrointestinal effects

Metallic Mercury: Many instances of gastrointestinal effects have been reported in humans following acute inhalation exposure to metallic mercury

vapour. A classical sign of mercury intoxication is stomatitis (inflammation of the oral mucosa). Accordingly, a number of case studies have reported stomatitis after acute-duration exposure to high concentrations of metallic mercury vapours (Bluhm et al. 1992a; Garnier et al. 1981; Snodgrass et al. 1981). Occasionally, the stomatitis is accompanied by excessive salivation (Karpathios et al. 1991) or difficult swallowing. Other gastrointestinal effects observed after acute-duration exposure to high levels of mercury include abdominal pains (Bluhm et al. 1992a, nausea and/or vomiting (Kanluen and Gottlieb 1991; Lilis et al. 1985; Rowens et al. 1991; Snodgrass et al. 1981; Soni et al. 1992; Taueg et al. 1992), and diarrheoa (Bluhm et al. 1992a; Kanluen and Gottlieb 1991; Rowens et al. 1991; Taueg et al. 1992).

Intermediate-duration exposures to mercury spills have also resulted in similar gastrointestinal effects. A case study reported that teenage girls exhibited anorexia, intermittent abdominal cramps, mild diarrhoea, painful mouth, and bleeding for 2 weeks after a spill of metallic mercury in their home (on carpet) resulted in the release of metallic mercury vapour (Sexton et al. 1976). Air levels in the home were measured 6 months after the initial spill and ranged from 0.02 to 1 mg Hg/m^3, depending upon the degree of ventilation and proximity to the spill. Fagala and Wigg (1992) reported a case of colicky abdominal pain and diarrhoea in a 12-year-old girl exposed to mercury vapours for approximately 6 months after a spill in her home. Limited information was located regarding gastrointestinal effects in persons who are chronically exposed to elemental mercury vapours. Stomatitis was observed in 22 of 72

workers exposed to mercury vapours in the manufacture of thermometers in the 1940s (Bucknell et al. 1993).

Two animal studies assessed the gastrointestinal effects from mercury vapour exposure. In rabbits, effects ranging from mild pathological changes to marked cellular degeneration and some necrosis of the colon were observed following exposure to 28.8 mg/m^3 mercury vapour for 4–30 hours (Ashe et al. 1953). A single exposure to 28.8 mg/m^3 for 1–2 hours or multiple exposures of 6 mg/m^3 for 7 hours a day, 5 days a week for up to 11 weeks resulted in either no changes or mild pathological changes. The usefulness of these results is limited because the study did not specify the pathological changes nor distinguish between primary and secondary effects (i.e., pathological changes secondary to induced shock).

Organic Mercury: Gastrointestinal effects were reported in several case studies of humans exposed to organomercurial compounds. A 39-year-old farmer who had dressed his seeds for several seasons with phenylmercuric acetate exhibited a swollen mouth, reddened and tender gums, carious teeth, a thin blue line at the gums, and an infected and swollen posterior pharyngeal wall (Brown, 1954). Similarly, two women who died following 3–5 months of occupational exposure to diethylmercury vapours exhibited inflammation of the mouth and gums, excessive salivation, and unspecified gastrointestinal disorders. Marked salivation was observed in one man and nausea was observed in another occupationally exposed to alkyl mercury compounds used for dressing seeds (Hook et al. 1954). Gastrointestinal effects were not,

however, observed in four men after inhalation of dust containing methylmercury for several months (Hunter et al. 1940).

No studies were found regarding gastrointestinal effects in animals after inhalation exposure to organic.

Haematological effects

Metallic Mercury: Initial exposure to high concentrations of elemental mercury vapours produces a syndrome similar to "metal fume fever," which is characterized by fatigue, fever, chills, and elevated leukocyte count. Evidence of moderate-to-high leukocytosis with neutrophilia was reported following acute inhalation exposure to metallic mercury vapour (Jaffe et al. 1983; Lilis et al. 1985; Rowens et al. 1991).

Similarly, an elevated white cell count was observed in a 12-year-old girl with a 6-month exposure to mercury vapours from a spill of metallic mercury in her home (Fagala and Wigg, 1992). Thrombocytopenia and frequent nosebleeds were reported in two of four family members exposed to mercury vapours in their home as a result of a spill of metallic mercury (Schwartz et al. 1992). The authors considered this to be a unique reaction to the mercury exposure. In volunteers with dental amalgam, significantly decreased haemoglobin and haematocrit increased mean amalgams (Siblerud, 1990). δ-Aminolevulinic acid dehydratase activity in erythrocytes was decreased in workers exposed to elemental mercury in the manufacture of tungsten rods (Wada et al. 1969). The decreases correlated with increases in urinary mercury. The estimated exposure level to mercury in the plant was slightly less than 0.1

mg/m^3. In workers exposed to 0.106–0.783 mg/m^3 mercury vapour, there was a significant increase in α2-macroglobulin and ceruloplasmin (a α-globulin protein active in the storage and transport of copper) compared to unexposed workers (Bencko et al. 1990).

No studies were found regarding haematological effects in animals after inhalation exposure to inorganic mercury (Bencko et al. 1990).

Organic Mercury: No studies were found regarding haematological effects in humans or animals after inhalation exposure to organic mercury (Bencko et al. 1990).

Musculoskeletal effects

Metallic Mercury: A number of studies have reported increases in tremors, muscle fasciculations, myoclonus, or muscle pains after acute (Adams et al. 1983; Bluhm et al. 1992a; Karpathios et al. 1991) exposure to metallic mercury vapour.

No studies were found regarding musculoskeletal effects in animals after inhalation exposure to metallic mercury.

Organic Mercury: Exposure to unspecified alkyl mercury compounds have caused muscular effects (e.g., muscle fasciculations, absence of deep reflexes in arms, Babinski reflex) (Brown, 1954; Hook et al. 1954).

No studies were located regarding musculoskeletal effects in animals after inhalation exposure to organic mercury.

Metallic Mercury: A case study described the acute poisoning of a young child who was exposed to mercury vapours that were produced from heating an unknown quantity of mercury (Jaffe et al. 1983). Hepatocellular effects were characterized by biochemical changes (e.g., elevated serum alanine aminotransferase [ALT]), ornithine carbamyl transferase, and serum bilirubin levels) and evidence of a decrease in the synthesis of hepatic coagulation factors. Similarly, hepatomegaly and central lobular vacuolation were observed in a man who died following acute-duration exposure to high levels of elemental mercury vapours (Kanluen and Gottlieb, 1991; Rowens et al. 1991). Serious liver effects have been noted in animals at high exposure concentrations. Acute inhalation exposure of rabbits to metallic mercury vapour concentrations of 28.8 mg/m^3 for 6–30 hours resulted in effects ranging from moderate pathological changes (unspecified) to severe liver necrosis (Ashe et al. 1953). These effects were less severe (mild effects to degeneration) at shorter exposure durations and following exposure to 6 mg/m^3 mercury vapours for 7 hours a day, 5 days a week for 1–5 weeks (Ashe et al. 1953). Effects ranging from moderate pathological changes to marked cellular degeneration and some necrosis were seen at mercury concentrations of 6 mg/m^3 for 7 hours a day, 5 days a week for 6–11 weeks (Ashe et al. 1953). The studies by Ashe et al. (1953) were deficient in quantitative data, and used a small number of animals. However, available human and animal data suggest that metallic mercury vapours can cause liver effects following acute exposures.

Hepatic effects

Metallic Mercury: A case study described the acute poisoning of a young child who was exposed to mercury vapours that were produced from heating an unknown quantity of mercury (Jaffe et al. 1983). Hepatocellular effects were characterized by biochemical changes (e.g., elevated serum alanine aminotransferase [ALT]), ornithine carbamyl transferase, and serum bilirubin levels) and evidence of a decrease in the synthesis of hepatic coagulation factors. Similarly, hepatomegaly and central lobular vacuolation were observed in a man who died following acute-duration exposure to high levels of elemental mercury vapours (Kanluen and Gottlieb, 1991; Rowens et al. 1991). Serious liver effects have been noted in animals at high exposure concentrations. Acute inhalation exposure of rabbits to metallic mercury vapour concentrations of 28.8 mg/m^3 for 6–30 hours resulted in effects ranging from moderate pathological changes (unspecified) to severe liver necrosis (Ashe et al. 1953). These effects were less severe (mild effects to degeneration) at shorter exposure durations and following exposure to 6 mg/m^3 mercury vapours for 7 hours a day, 5 days a week for 1–5 weeks (Ashe et al. 1953). Effects ranging from moderate pathological changes to marked cellular degeneration and some necrosis were seen at mercury concentrations of 6 mg/m^3 for 7 hours a day, 5 days a week for 6–11 weeks (Ashe et al. 1953). The studies by Ashe et al. (1953) were deficient in quantitative data, and used a small number of animals. However, available human and animal data suggest that metallic mercury vapours can cause liver effects following acute exposures.

Organic Mercury: Mid-zonal necrosis in the liver was observed during the autopsy of a farmer who died after treating grain with phenylmercuric acetate for several seasons (Brown, 1954). No conclusions can be drawn from this study, however, because other factors may have contributed to the hepatic effects in this subject.

No studies were found regarding hepatic effects in animals after inhalation exposure to organic mercury (Brown, 1954).

Renal effects

Metallic Mercury: The kidney is a sensitive target organ of toxicity following inhalation exposure to metallic mercury. This sensitivity may be, in part, because of the relatively high accumulation of mercury in the kidneys. Acute high-concentration inhalation exposure in humans has resulted in effects ranging from mild transient proteinuria or syndrome has been reported light changes in urinary acid excretion (Bluhm et al. 1992b; Soni et al. 1992); to frank proteinuria, hematuria, and or oliguria (Snodgrass et al. 1981); to acute renal failure with degeneration or necrosis of the proximal convoluted tubules (Jaffe et al. 1983; Kanluen and Gottlieb 1991; Rowens et al. 1991). Actual exposure concentrations are unknown in these cases, but urinary mercury excretion as high as 59–193 µg/hour has been reported (Bluhm et al. 1992b). The nephrotic syndrome was characterized by edema and proteinuria with albumin and hyaline casts in the urine. These changes usually abated within a few months following termination of exposure. Among a group of 10 patients who reported adverse effects associated with dental amalgams (the route of

exposure in dental amalgams is probably a mixture of inhalation exposure to mercury vapour released from the amalgams, absorption of the vapour through the oral mucosa, and ingestion), a decrease in the ability to concentrate the urine and elevated urinary albumin were observed (Anneroth et al. 1992).

Removal of one amalgam resulted in a significant decrease in urinary albumin (it is unknown whether other amalgams remained). In a study of renal function in 10 healthy volunteers having an average of 18 amalgam-filled tooth surfaces both before and after amalgam removal (Sandborgh-Englund and Nygren, 1996), no signs of renal toxicity were found in conjunction with mercury released from the amalgam fillings. Although plasma mercury levels increased significantly one day after removal of the fillings (all removals were accomplished in one dental session), glomerular filtration rates were similar both before and after mercury exposure (amalgam removal). Blood, plasma, and urine mercury concentrations were significantly lower 60 days after amalgam removal.

The results from a number of studies show renal toxicity in workers chronically exposed to mercury vapour (Cardenas et al. 1993; Ehrenberg et al. 1991).

Endocrine disruptive effects

Metallic Mercury: A 13-year-old boy exposed to mercury vapours for 2 weeks developed a thyroid enlargement with elevated triiodothyronine, and thyroxin; and low thyroid-stimulating hormone levels (Karpathios et al. 1991). Serum-free thyroxine (T4) and the ratio of free thyroxine to free 3,5,3'-

triiodothyronine (T3) were found to be slightly, but significantly, higher in workers with the highest exposure concentrations in a study of chloroalkali workers exposed to an average of 10 years to metallic mercury vapour (Barregard et al. 1994a, 1994b).

Further, serum-free T3 was inversely associated with cumulative mercury exposure, suggesting a possible inhibitory effect of mercury on 5'-deiodinases, which is responsible for the conversion of T4 to the active hormone T3. In this study, serum total testosterone (but not free testosterone) was positively correlated with cumulative mercury exposure, while prolactin, thyrotrophin, and urinary cortisol concentrations were not associated with exposure. However, two other occupational studies found no relationship between mercury exposure (unspecified concentration) and endocrine function (i.e., testicular, thyroid, and pituitary) (Erfurth et al. 1990; McGregor and Mason 1991). Biochemical indices that were measured in the occupational study by McGregor and Mason, 1991) to assess endocrine effects included serum testosterone, sex-hormone binding globulin, thyroid-stimulating hormone, and prolactin. Erfurth et al. (1990) measured both basal serum concentrations of thyrotropin, thyroxine, triiodothyronine, and cortisol, as well as the response to a thyrotropin challenge.

No studies were found regarding endocrine effects in animals after inhalation exposure to metallic mercury.

Organic Mercury: No studies were found regarding endocrine effects in humans or animals after inhalation exposure to organic mercury.

Dermal effects

Metallic Mercury: Inhalation exposure of individuals to elemental mercury vapours for acute and intermediate durations has resulted in erythematous and pruritic skin rashes (Aronow et al. 1990; Bluhm et al. 1992a; Foulds et al. 1987; Karpathios et al. 1991; Schwartz et al. 1992). Other dermal reactions to mercury exposure include heavy perspiration (Aronow et al. 1990; Fagala and Wigg 1992; Karpathios et al. 1991) and reddened and/or peeling skin on the palms of the hands and soles of the feet (Aronow et al. 1990; Fagala and Wigg 1992; Karpathios et al. 1991).

No studies were found regarding dermal effects in animals after inhalation exposure to metallic mercury.

Organic Mercury: No studies were found regarding dermal effects in humans or animals after inhalation exposure to organic mercury.

Ocular effects

Metallic Mercury: Ocular effects observed following acute exposure included red, burning eyes and conjunctivitis (Bluhm et al. 1992a). These case studies contained insufficient quantitative data for dose-response assessment.

No studies were found regarding ocular effects in animals after inhalation exposure to metallic mercury.

Organic Mercury: No studies were found regarding ocular effects in humans or animals after inhalation exposure to organic mercury.

Other systemic effects

Metallic Mercury: Initial exposure to high concentrations of elemental mercury vapours produces a syndrome similar to "metal fume fever," which is characterized by fatigue, fever, chills, and an elevated leukocyte count. Accordingly, several studies have reported fever and/or chills in humans after exposure to high concentrations of elemental mercury vapours (Aronow et al. 1990; Bluhm et al. 1992a; Garnier et al. 1981; Lilis et al. 1985; Schwartz et al. 1992; Snodgrass et al. 1981).

Organic Mercury: No studies were found regarding other systemic effects in humans or animals after inhalation exposure to organic mercury.

Immunological and Lymphoreticular effects

Metallic Mercury: The immune reaction in humans to mercury exposure appears to be idiosyncratic, with either increases or decreases in immune activity depending on individual genetic predisposition.

Therefore, it is not surprising that several studies of workers exposed to elemental mercury vapour have failed to show consistent or marked changes in immune function parameters in large populations. For example, no effect on serum immunoglobulins (IgA, IgG, or IgM) and no increase in autoantibody titres were observed in a group of chloroalkali workers exposed for an average of 13.5 years (Langworth et al. 1992b). Similarly, no increases in antilaminin antibodies were observed in workers exposed for an average of 7.9 years (Bernard et al. 1987), and no increase in antiglomerular basement membrane antibodies or IgE was seen in workers exposed for between 1.5 and 25 years

(Cardenas et al. 1993).No significant differences in the concentrations of immunoglobulins or complement components were found in a study of 76 chloralkali workers previously exposed to mercury vapour for an average of 7.9 years (range, 1.1–36.2 years) (Ellingsen et al. 1994). No increase in the prevalence of autoantibodies was observed between the formerly exposed worker group and a control group of 53 age-matched referents. The average time elapsed since the cessation of occupational exposure was 12.3 years (range, 1–35 years).

Evidence of a human autoimmune response has been obtained in a few studies. Examination of the kidneys of two workers with proteinuria revealed granular deposition of IgG and the complement C3 in the glomeruli (Tubbs et al. 1982). Among a group of 10 patients who reported adverse effects associated with dental amalgams (the route of exposure is probably a mixture of inhalation exposure to mercury vapour released from the amalgams and dermal exposure to the amalgams), 3 had increased antiglomerular basement membrane antibodies and 2 had elevated antinuclear antibodies (Anneroth et al. 1992). After removal of one amalgam, there was a significant decrease in IgE (it is unknown whether other amalgams remained). Also, 1 of 89 workers examined by Langworth et al. (1992b) showed a weak reaction to antiglomerular basement membrane, and 8 of 44 workers examined by Cardenas et al. (1993) showed an abnormally high anti-DNA antibody titre. Only two studies have shown increases in immune parameters in exposed populations. Increases in IgA and IgM were observed in workers in a mercury producing plant (Bencko

et al. 1990). The study is limited by a lack of information on daily dose levels, duration of employment and potential confounding factors (smoking, alcohol). An increase in anti-DNA antibodies was observed in workers from a chloralkali plant (Cardenas et al. 1993).

Other experimental evidence suggests that mercury can alter a number of parameters of the host's immune system and lead to increased susceptibility to infections, autoimmune diseases, and allergic manifestations. A positive correlation was found between the T-helper cell count and the duration of exposure ($p<0.05$). The combined stimulation of the T-cell line and an observed decrease in the helper/suppressor ratio were suggestive of an autoimmune response.

In a mercury-producing plant, neutrophil function was found to be significantly reduced in workers with mean exposure duration of 8 months (range, 0.5–46 months) (Perlingeiro and Queiroz, 1995). In this study, both chemotactic and chemical-specific reducing activities of the neutrophils of exposed workers were found to be affected. While improved industrial hygiene practices over a 6-month period resulted in a decrease in urine mercury concentration in the workers, it did not result in the return of neutrophil migration activity to within the normal range. There was, however, no observed increase in the incidence of infections in the mercury-exposed group compared to controls. Based on their observations, Perlingeiro and Queiroz (1995) suggested that even exposures to levels of mercury considered "safe" in some industrial settings might lead to impairment of neutrophil function.

Exposure of genetically susceptible mice to mercury vapour for a period of 10 weeks resulted in an autoimmune response similar to that seen in similar mice after treatment with mercuric chloride by subcutaneous injections and in drinking water (Warfvinge et al. 1995). This response was manifested as a syndrome, which included a general stimulation of the immune system, with hyperimmunoglobulinemia, anti-nucleolar-fibrillarin autoantibodies, and glomerular disease accompanied by vascular immune complex deposits. Actual inhalation exposure times for the 0.3–1 mg Hg/m^3 exposure concentrations varied from 0.5 to 19 hours a day (5 days a week), but doses for individual exposure groups were also expressed in µg/kg/week units.

Organic Mercury: No studies were found regarding immunological and lymphoreticular effects in humans or animals after inhalation exposure to organic mercury.

Neurological effects

Metallic Mercury: The central nervous system is probably the most sensitive target organ for metallic mercury vapour exposure. Nervous system disorders following exposure to metallic mercury vapours are both consistent and pronounced. Acute-, intermediate-, and chronic-duration exposures elicit similar neurological effects. Symptoms intensify and may become irreversible as exposure duration and/or concentration increases. Most occupational studies discuss chronic-duration exposure to a time-weighted average (TWA) concentration or to a concentration range, thereby preventing the assessment of

dose response relationships within the populations studied. However, the average exposure levels for affected groups are similar in many of these studies.

In humans, several case studies have reported adverse neurological effects following acute inhalation of high concentrations of mercury vapour. A wide variety of cognitive, personality, sensory, and motor disturbances have been reported. The most prominent symptoms include tremours (initially affecting the hands and sometimes spreading to other parts of the body), emotional liability (characterized by irritability, excessive shyness, confidence loss, and nervousness), insomnia, memory loss, neuromuscular changes (weakness, muscle atrophy, muscle twitching), headaches, polyneuropathy (paresthesia, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities), and performance deficits in tests of cognitive function (Adams et al. 1983; Bluhm et al. 1992a; Hallee, 1969; Jaffe et al. 1983; Karpathios et al. 1991; Lilis et al. 1985; Snodgrass et al. 1981). A few individuals have also noted hearing loss, visual disturbances (visual field defects), and/or hallucinations (Bluhm et al. 1992a). Three and one-half months after exposure to high levels of mercury vapour during 2 days of an industrial liquid mercury salvaging operation, a 54-year-old man exhibited a syndrome resembling amyotrophic lateral sclerosis, characterized by slowed conduction velocities (suggestive of peripheral nerve damage). Urinary mercury levels were 100-µg/g creatinine at the time of the exam; after an additional 2 months (no treatment administered), levels dropped to less than 30 µg/g creatinine and symptoms disappeared (Adams et al. 1983). In contrast,

chelation therapy (2.3 dimercaptosuccinic acid [DMSA] or – acetyl-D, L-penicillamine [NAP]) and lowering of urinary mercury levels did not result in improvement in depression, anxiety, phobias, psychotic-like behaviour, interpersonal sensitivity, and hostility observed in another group of workers exposed to high concentrations of mercury vapour for up to 16 hours (Bluhm et al. 1992a).

Reproductive effects

Metallic Mercury: No acute-duration exposure data were found regarding reproductive effects in humans after inhalation exposure to metallic mercury. However, several studies found no effect on fertility following intermediate or chronic inhalation exposure to metallic mercury in humans (Cordier et al. 1991). A retrospective cohort study reported that male workers in a U.S. Department of Energy (DOE) plant exposed for at least 4 months had urinary mercury concentrations of 2,144–8,572 µg/L (Alcser et al. 1989). This sample population showed no significant difference in fertility compared to controls (unexposed workers); however, they were never monitored for elemental mercury exposure. In a questionnaire study assessing the fertility of male workers exposed to mercury vapour from various industries (i.e., zinc-mercury amalgam, chloroalkali, or electrical equipment product plants), there was no statistically significant difference in the number of children of the exposed group compared to a matched control group (Lauwerys et al. 1985). The concentration of mercury in the urine of these exposed workers ranged from 5.1-to 272.1-µg/g creatinine. No correlation was observed between

prolactin, testosterone, luteinizing hormone, and follicle stimulating hormone levels and blood or urine mercury levels in male workers exposed to mercury vapours (Erfurth et al. 1990; McGregor and Mason 1991). Also, no effect on the response of these hormones to challenge with gonadotropin releasing hormone was observed (Erfurth et al. 1990).

Although no effect on fertility was observed in exposed workers, an increase in the rate of spontaneous abortions was reported in association with increased mercury concentrations in the urine of the fathers exposed to metallic mercury in chloralkali plants before the pregnancy (Cordier et al. 1991). There was a significantly increased risk of spontaneous abortion, at a rate of 18.4%, when fathers had more than 50 µg/L mercury in the urine, compared to a rate of 8.6% when fathers were unexposed.(Sikorski et al. 1987) reported that women occupationally exposed to metallic mercury vapours (dentists and dental assistants) had more reproductive failures (spontaneous abortions, stillbirths, congenital malformations) and irregular, painful, or hemorrhagic menstrual disorders than a control (unexposed) group of women. The reproductive difficulties and menstrual disorders were correlated with mercury levels identified in scalp and pubic hair collected from the women. It should be noted that this study has been recently severely criticized for what Larsson (1995) calls "erroneous interpretation of results and distortion of conclusions." The Sikorski et al. (1987) paper is nonetheless presented in this toxicological profile as part of the available published data on reported human mercury exposure. Its presence here is based upon its publication in a credible peer-reviewed

international journal and is intended neither as endorsement nor condemnation of the data or conclusions in the 1987 paper. Rowland et al. (1994) report that 418 women with high exposure to mercury (i.e., female dental assistants) were less fertile than unexposed controls. In this study, the probability of conception with each menstrual cycle (called "fecundability" by the authors) in women who prepared 30 or more amalgams per week and who were evaluated as having 4 or more poor mercury-hygiene practices was 63% of the fecundity of the unexposed controls. Rowland et al. (1994) noted that occupational groups with roughly the same potential for exposure often contain subjects whose actual exposures are quite different, depending on their particular work environment and their work practices within that environment. For example, 20% of the women in the final sample in this study reported preparing more than 30 amalgams per week with 4 or poorer hygiene factors. Among the women preparing the same number of amalgams, this study found differences in "fecundability," based upon each dental assistant's reported number of poor mercury-hygiene factors.

One peculiar observation, however, was that women determined to have had low exposure to mercury in their dental occupation were found to be more fertile than unexposed controls. The reason(s) for the observed U-shaped dose-response curve were not known (Sikorski et al. 1987).

In addition, oestrous cycles during mercury exposure were longer than normal oestrous cycles in the same animals prior to exposure. Although the initial phase of the cycle was protracted, complete inhibition of the cycle did

not occur. During the second and third weeks of exposure, these rats developed signs of mercury poisoning including restlessness, seizures, and trembling of the entire body. The authors speculated that the effects on the oestrous cycle were caused by the action of mercury on the central nervous system (i.e., damage to the hypothalamic regions involved in the control of oestrous cycling)(Rowland et al. 1994).

Organic Mercury: No studies were found regarding reproductive effects in humans or animals after inhalation exposure to organic mercury.

Developmental effects

Metallic Mercury: No association was demonstrated between inhalation exposure of the father and increased rates of major fatal malformations or serious childhood illnesses (Alcser et al. 1989).

It is unclear whether the reproductive toxicity experienced by the woman was due to the mercury exposure. However, after recovery from overt mercury poisoning, she gave birth to a healthy child. A woman occupationally exposed to mercury vapours for 2 years prior to pregnancy and throughout pregnancy was reported to have delivered a viable infant at term (Melkonian and Baker, 1988). Urinary mercury in the woman at 15 weeks of pregnancy was 0.875 mg/L (normal levels are approximately 0.004 mg/L). Also, a case report of a woman exposed to mercury vapours in her home during the first 17 weeks of pregnancy reported that the woman delivered a normal child who met all developmental milestones (although the child was not formally tested for psychological development) (Thorpe et al. 1992). Although mercury exposure

was not measured, the child was born with hair levels of 3 mg/kg (3ppm) of mercury. This hair level is comparable to that observed in populations consuming fish once a week (WHO, 1990) and suggests that exposure in this case may have been relatively low.

Exposure of neonatal rats to metallic mercury vapour at 0.05 mg/m^3 for 1 or 4 hours a day for 1 week during a period of rapid brain growth (postpartum days 11–17) resulted in subtle behavioural changes when the rats were tested at 4 and 6 months of age (Fredriksson et al. 1992). Offspring of rats exposed for 1 hour/day showed increases in the time necessary to finish a task in the radial arm maze (spatial learning). Offspring of rats exposed for 4 hours a day showed increases in both the time to finish the task and in the number of errors committed. When tested for locomotor activity at 2 months, an increase in rearing was observed in the 4-hour/day groups, but repeat testing at 4 months showed lower locomotor, rearing, and total activity than controls. The 1-hour/day exposure group showed no difference from controls at 2 months, and increased activity and decreased rearing at 4 months when compared to controls.

Three groups of 12 pregnant Sprague-Dawley rats were exposed by inhalation to 1.8-mg/m3 metallic mercury vapour on gestation days (Gd) 11–14 and 17–20 for 1 hour ("low dose") or 3 hours ("high dose"). At postpartum day 3, each litter was reduced to 4 male and 4 female offspring. No significant differences between the mercury-treated offspring and the controls were observed for surface righting, negative geotaxis, pinna unfolding, and tooth

eruption. Tests of spontaneous motor activity (locomotion, rearing, rearing time, and total activity) showed that the mercury-treated offspring were hypoactive at 3 months of age; at 14 months, only total activity differed between exposed and control groups. In spatial learning tasks, exposed offspring showed retarded acquisition in the radial-arm maze but no differences in the circular-swim maze. A simple test of learning, habituation to a novel environment (activity chambers), indicated a reduced ability to adapt. The authors conclude that prenatal exposure to mercury vapour results in behaviour changes in the offspring similar to those reported for methylmercury. On postpartum days 3–4, the mercury contents in the brain, liver, and kidneys were 0.001, 0.004, and 0.002 mg Hg/kg, respectively, for control offspring; 0.005, 0.053, and 0.033 mg Hg/kg, respectively, for animals exposed for 1 hour a day; and 0.012, 0.112, and 0.068 mg Hg/kg, respectively, for animals exposed for 3 hours a day (Danielsson et al. 1993).

Four groups of 12 pregnant Sprague-Dawley rats were exposed to methylmercury or elemental mercury alone or in combination as follows: (1) administered 2 mg/kg/day methylmercury via gavage during Gd 6–9; (2) exposed by inhalation to 1.8 mg/m^3 metallic mercury (elemental mercury) vapour for 1.5 hours per day during Gd 14–19; (3) exposed to both methylmercury by gavage (2 mg/kg/day, Gd 6–9) and elemental Hg vapour by inhalation (1.8 mg/m3, Gd 14–19) (methylmercury + elemental mercury); or (4) given combined vehicle administration for each of the 2 treatments (control). The inhalation regimen corresponded to an approximate dose of 0.1 mg

Hg/kg/day. At postpartum day 3, each litter was reduced to 4 male offspring. There were no differences between any of the groups in maternal body weight gain before parturition. No differences in body weight, pinna unfolding, tooth eruption, surface righting reflex, and negative geotaxis were observed in the offspring. Offspring of dams exposed to elemental Hg showed hyperactivity in the spontaneous motor activity test chambers over all three parameters: locomotion, rearing, and total activity; this effect was potentiated in the animals of the methylmercury + elemental Hg group. In the swim maze test, the methylmercury + elemental mercury and elemental mercury groups evidenced longer latencies to reach a submerged platform, which they had learned to mount the day before, compared to either the control group or the methylmercury group. In the modified enclosed radial-arm maze, the methylmercury + elemental Hg and elemental Hg groups showed more ambulations and rearings in the activity test prior to the learning test. During the learning trial, the same groups (i.e., methylmercury + elemental Hg and elemental Hg) showed longer latencies and made more errors in acquiring all eight pellets. Generally, the results indicate that prenatal exposure to elemental mercury causes alterations to both spontaneous and learned behaviours, suggesting some deficit in the adaptive functions of the rats. Co-exposure to methylmercury, which by itself did not alter these functions at the dose given in this study, served to aggravate the changes significantly. Brain mercury concentrations in offspring were 1 ng/g w/w in the controls, 4 ng/g in the

methylmercury group, 5 ng/g in the elemental Hg group, and 12 ng/g in the methylmercury + elemental Hg group (Fredriksson et al. 1996).

A decrease in the number of living foetuses was observed in these dams compared to unexposed controls, and all pups born to the exposed dams died by the sixth day after birth. However, no difference in the occurrence of developmental abnormalities was observed between exposed and control groups. The cause of death of the pups in the mercury-exposed group was unknown, although the authors to a failure of lactation in the dams attributed an unspecified percentage of the deaths. Death of pups was also observed in another experiment in which dams were only exposed to the same dose level prior to fertilization, supporting the conclusion that high mortality in the first experiment was due, at least in part, to the poor health of the mothers.

Genotoxic effects

There is inconclusive evidence that occupational exposure to metallic mercury and to organic and inorganic mercury compounds, primarily through inhalation, causes structural and numerical chromosome aberrations in human lymphocytes. In one study, significant increases in the frequency of eccentric fragments (chromosome breaks) occurred in 4 workers exposed to high concentrations of metallic mercury and in 18 workers exposed to a mixture of mercuric chloride, methylmercuric chloride, and ethylmercuric chloride (Popescu et al. 1979). Mercury concentrations in the workplace ranged from 0.15 to 0.44 mg/m^3; the urinary excretion level of mercury for both exposed

groups was 890 µg/L. The findings of this study are suspect because the control group was not matched for sex, smoking habits, or sample size.

Additionally, one of the four individuals in the metallic mercury group had a history of benzene poisoning, which was reflected in the unusually high frequency of abnormal chromosome morphology seen in this individual. No difference in the incidence of aneuploidy was found between the exposed workers and the controls. In an earlier study, an apparent association between increased chromosome aberrations and workplace exposure to mercury (as measured by urinary mercury levels) was reported (Verschaeve et al. 1976).

However, the study was not well controlled (i.e., not matched for sex, smoking habits, or sample size), and the only significant increase in structural aberrations occurred in the three workers exposed to ethylmercury. Significant increases in aneuploid were also noted for the exposure groups compared to the control subjects. However, these data should also be interpreted with caution since age has an influence on aneuploidy, and in this study, there was a general trend toward a higher incidence of aneuploidy in the older exposed workers (ages 36–63 years). It is noteworthy that in a subsequent study performed by these investigators (Verschaeve et al. 1979), no adverse effects on the structure or number of chromosomes were demonstrated in 28 subjects exposed to moderate levels of metallic mercury (urinary levels of 50 µg/L).

The authors concluded that the results from their 1976 study, showing an association between increased chromosomal aberrations and occupational exposure to mercury, might have been affected by factors other than exposure

to mercury compounds. No increased frequency of structural aberrations was found in 22 workers exposed to mercury vapours; no information was provided on numerical aberrations (Mabille et al. 1984). The mean duration of exposure was 4 years, and the mean urinary and blood mercury levels in the exposed group contained 117-μg/g creatinine and 0.031μg/mL, respectively. More recently, peripheral lymphocytes from 26 male chloroalkali workers exposed to mercury vapours (25–50 $\mu g/m^3$), for a mean exposure time of 10 years, were analyzed for micronucleus induction. The results were compared to results obtained from 26 unexposed subjects (Barregard et al. 1991). Groups were matched for age (±7 years) and smoking habits; plasma, erythrocyte, and urine mercury levels were determined. Parallel lymphocyte cultures from each donor group were incubated in the presence of pokeweed mitogen, which stimulates both B- and T lymphocytes, and phytohemagglutinin, which primarily activates T-cells. The analysis showed no significant increase in the frequency or the size of micronuclei in the exposed versus the control group. Nor was there a correlation between micronuclei induction and plasma, erythrocyte, or urinary levels of mercury. Within the exposed group, however, there was a significant correlation between micronuclei induction in phytohemagglutinin stimulated lymphocytes and cumulative exposure (whole-blood mercury level over employment time); the response was independent of age or smoking habits. The authors stated that the evidence of a genotoxic response confined to T-lymphocytes could have been a random finding but hypothesized that long-term exposure to mercury may cause an accumulation of cytogenetic effects.

Similarly, there was no correlation between urinary mercury levels (60–245 µg/L) or the duration of exposure (11–34 years) and increased frequency of structural aberrations and micronuclei in the lymphocytes of 29 male workers exposed to mercury fulminate (Anwar and Gabal, 1991). From the overall results, the authors concluded that mercury in the manufacturing process might not have been the clastogen.

Cancer

Metallic Mercury: There is no evidence from epidemiological studies that indicates inhalation of metallic mercury produces cancer in humans (Cragle et al. 1984; Kazantzis, 1981). No evidence of an association between metallic mercury exposure and cancer mortality was found in a group of workers employed in a facility utilizing the metal in a lithium isotope separation process (Cragle et al. 1984). Overall mortality in the mercury-exposed group was less than that of the standard white male population and that of a control group of men who were not exposed to mercury. Similarly, no excess of cancer of the kidneys or nervous system was found among a cohort of 674 Norwegian men exposed to mercury vapours for more than 1 year at 2 chloroalkali plants (Ellingsen et al. 1993). Excess in lung cancer (type not specified) was found in Swedish chloroalkali workers 10 years after the end of long-term, high-level exposure to metallic mercury (Barregard et al. 1990). However, these workers had also been exposed to asbestos. Furthermore, no data on smoking status was provided, although the study implied that the workers did not smoke much.

No studies were found regarding cancer in animals after inhalation exposure to metallic mercury.

Organic Mercury: Associations were reported between the uses of mercury-containing fungicides (i.e., mercury levels in hair) and leukaemia in farmers and between the use of mercury-containing seed dressings and leukaemia in cattle (Janicki et al. 1987). However, the study was limited in reporting methodology used to conduct this study. Furthermore, the study did not adequately address exposure to other chemicals, or adjust for other leukemia risk factors.

No studies were found regarding cancer in animals after inhalation exposure to organic mercury.

Oral exposure

The bulk of the information regarding toxicity resulting from oral exposure to inorganic mercury comes from studies of mercuric chloride. However, a few studies are also available on the effects of oral exposure to mercuric acetate, mercurous chloride (calomel), and mercuric sulphide (cinnabar) (Janicki et al. 1987).

Neurological effects

Inorganic Mercury: The oral absorption of metallic mercury is negligible, and even massive doses have not resulted in neurological effects. The two case histories identified are unusual in that the dose levels could be reasonably well quantified. The first case history reported ingestion of 15mL (204 g) of metallic mercury by a 17-year-old male storekeeper who swallowed

mercury from the pendulum of a clock (apparently out of curiosity rather than as a suicide attempt). On admission, and 24 hours later, he was symptom free, and physical examination was normal. The patient complained of no gastrointestinal symptoms, and was treated with a mild laxative and bed rest. The results of serial daily urine mercury estimates were normal (all less than 15 µg) and did not suggest significant absorption. The radiological investigation illustrated a characteristic pattern of finely divided globules of mercury in the gastrointestinal tract (Wright et al. 1980).

The second and massive incidence of ingestion involved a 42-year-old man who had spent much of his life (since the age of 13) repairing instruments that contained mercury. He intentionally ingested an estimated 220 mL (or about 3,000 g) while repairing a sphygmomanometer (Lin and Lim, 1993). Upon admission, the patient presented with significantly elevated mercury blood levels (103 µg/L, normal <10 µg/L) and urine levels (73 µg/L, <20 µg/L). In the previous 2 years he had developed mild hand tremors, forgetfulness, irritability, and fatigue. The occupational exposures made it difficult to determine any additional neurological effects from the acute exposure. There was no history of peripheral neuropathy, vertigo, insomnia, or muscular weakness. Neuropsychiatric and psychology evaluations indicated poor concentration and a defect in recent memory (Lin and Lim, 1993).

Most case studies of neurotoxicity in humans induced by oral exposure to inorganic mercury salts have reported neurotoxic effects as the result of ingestion of therapeutic agents that contain mercurous chloride (e.g., teething

powders, ointments, and laxatives). Several children treated with tablets or powders containing mercurous chloride exhibited irritability, fretfulness, sleeplessness, weakness, photophobia, muscle twitching, hyperactive or hypoactive tendon reflexes, and/or confusion (Warkany and Hubbard 1953). A 4-year-old boy who had been given a Chinese medicine containing mercurous chloride for 3 months developed drooling, dysphagia, irregular arm movements, and impaired gait (Kang-Yum and Oransky 1992). Davis et al. (1974) reported that two women developed dementia and irritability due to chronic ingestion of a tablet laxative that contained 120 mg of USP-grade mercurous chloride (0.72 mg Hg/kg/day, assuming an average body weight of 70 kg). One woman had taken 2 tablets daily for 25 years, and the other woman took 2 tablets daily for 6 years. Both patients died from inorganic mercury poisoning, and at autopsy, low brain weight and volume and a reduced number of nerve cells in the cerebellum were seen. Light microscopic analysis revealed granules of mercury within neuronal cytoplasm. Electron microscopy revealed mercury deposits in some neurons.

Death

Inorganic Mercury: A case study described the death of a man who had been receiving treatment for a wound with daily applications for approximately 2 months of a Chinese medicine containing mercurous chloride (Kang-Yum and Oransky, 1992). The patient was reported to have died from renal failure.

An early study conducted by (Schamberg et al. 1918) reported death in rabbits after an ointment containing 50% mercury was "rubbed" into the skin for 5 minutes; however, inadequate experimental methodology and an absence of study details prevent a determination of the amount of mercury involved.

Organic Mercury: No studies were found regarding death in humans or animals after dermal exposure to organic mercury.

Elimination and Excretion

Elimination of metallic mercury occurs through the urine, faeces, and expired air, while inorganic mercury is excreted in the urine and faeces in humans. Animal data on excretion are limited but indicate that excretion is species and dose dependent. The faeces are a major elimination route for inorganic mercury compounds, but high acute doses increase the percentage of excretion via the urine. Excretion of organic mercury is predominantly thought to occur through the faecal (biliary) route in humans. In animals, phenylmercury is excreted initially though the bile and then shifts to urine, whereas methylmercury is primarily excreted in the bile and then the faeces. Age is a factor in the elimination of mercury in rats following inorganic and organic mercury exposure, with younger rats demonstrating significantly higher retention than older rats. Both inorganic and organic mercury compounds can be excreted in breast milk. There is no data suggesting that the route of exposure affects the ultimate elimination of inorganic and organic mercury that is absorbed into the body.

Health Effects of Cadmium

Human uptake of cadmium takes place mainly through food. Foodstuffs that are rich in cadmium can greatly increase the cadmium concentration in human bodies. Examples are liver, mushrooms, shellfish, mussels, cocoa powder and dried seaweed. An exposure to significantly higher cadmium levels occurs when people smoke. Tobacco smoke transports cadmium into the lungs. Blood will transport it through the rest of the body where it can increase its effect by potentiating cadmium that is already present from cadmium-rich food.

Other high exposures can occur with people who live near hazardous waste sites or factories that release cadmium into the air and people that work in the metal refinery industry. When people breathe in cadmium it can severely damage the lungs (DEFRA, 2002), this may even cause death.

Cadmium is first transported to the liver through the blood. There, it is bond to proteins to form complexes that are transported to the kidneys. Accumulation of cadmium in the kidney may affect vitamin D metabolism and may increase the excretion of calcium and phosphorus into the urine. This may lead to a disruption of the calcium balance, resulting in osteomalacia, osteoporosis and spontaneous fractures. A number of reports have documented disorders of calcium metabolism and bone effects amongst men occupationally exposed to cadmium, and decreased bone density and increased risk of fractures were reported in women in the Cadmibel study (DEFRA, 2002). Bone disease resulting from exposure to cadmium in the general environment has only been reported in people from a highly contaminated region in Japan (Itai-itai

disease), characterized as osteomalacia, osteoporosis, increased fractures and renal tubular dysfunction (DEFRA, 2002). This causes the excretion of essential proteins and sugars from the body and further kidney damage. It takes a very long time before cadmium that has accumulated in kidneys is excreted from a human body.

Other health effects that can be caused by cadmium are: Diarrhoea, stomach pains and severe vomiting, Bone fracture, Reproductive failure and possibly even infertility Damage to the central nervous system, Damage to the immune system, Psychological disorders and Possibly DNA damage or cancer development (www.lenntech.com).

Identity, Physical and Chemical Properties

Cadmium is a lustrous, silver-white, ductile, very malleable metal. Its surface has a bluish tinge and the metal is soft enough to be cut with a knife, but it tarnishes in air. It is soluble in acids but not in alkalis. It is similar in many respects to zinc but it forms more complex compounds

It is rarely found in a pure state. It is present in various types of rocks and soils and in water, as well as in coal and petroleum. Among these natural sources, zinc, lead, and copper ore are the main sources of cadmium. Cadmium can form a number of salts. Its mobility in the environment and effects on the ecosystem depend to a great extent on the nature of these salts. Since there is no evidence that organocadmium compounds(EHC, 135:1992), where the metal is covalently bound to carbon, occur in nature, only inorganic cadmium salts will be discussed. Cadmium may occur bound to proteins and other organic

molecules and form salts with organic acids, but in these forms, it is regarded as inorganic.

Cadmium has a relatively high vapour pressure. The vapour is oxidized quickly to produce cadmium oxide in air. When reactive gases or vapour, such as carbon dioxide, water vapour, sulphur dioxide, sulphur trioxide or hydrogen chloride, are present, the vapour reacts to produce cadmium carbonate, hydroxide, sulphite, sulphate or chloride, respectively. These salts may be formed in stacks and emitted to the environment.

Some of the cadmium salts, such as the sulphide, carbonate or oxide, are practically insoluble in water. However, these can be converted to water-soluble salts in nature under the influence of oxygen and acids; the sulphate, nitrate, and halogenates are soluble in water (www.epa.gov/cadmium.faq.htm).

Natural occurrence

Cadmium is widely distributed in the earth's crust at an average concentration of about 0.1 mg/kg and is commonly found in association with zinc. However, higher levels are present in sedimentary rocks: marine phosphates often contain about 15 mg/kg (GESAMP, 1984).

Weathering and erosion result in the transport by rivers of large quantities of cadmium to the world's oceans and this represents a major flux of the global cadmium cycle; an annual gross input of 15,000 tones has been estimated (GESAMP, 1987). In background areas away from ore bodies, surface soil concentrations of cadmium typically range between 0.1 and 0.4 mg/kg (Page, 1981). The median cadmium concentration in non-volcanic soil

ranges from 0.01 to 1 mg/kg, but in volcanic soil levels of up to 4.5 mg/kg have been found (Korte, 1983).

Volcanic activity is a major natural source of atmospheric cadmium release. The global annual flux from this source has been estimated to be 100-500 tonnes. (Nriagu, 1989). Deep sea volcanism is also a source of environmental cadmium release, but the role of this process in the global cadmium cycle remains to be quantified.

The average cadmium content of sea water is about 0.1µg/litre or less (Korte, 1983), while river water (Mississippi, Yangtze, Amazon, and Orinoco sampled between 1976 and 1982) contains dissolved cadmium at concentrations of between 1.1-13.5 ng/litre (Shiller, & Boyle, 1987). Cadmium levels of up to 5 mg/kg have been reported in river and lake sediments and from 0.03 to 1 mg/kg in marine sediments (Korte, 1983).

Current measurements of dissolved cadmium in surface waters of the open oceans give values of < 5 ng/litre. The vertical distribution of dissolved cadmium in ocean waters is characterized by a surface depletion and deep water enrichment, which corresponds to the pattern of nutrient concentrations in these areas (Boyle, et al, 1976) This distribution is considered to result from the absorption of cadmium by phytoplankton in surface waters and its transport to the depths, incorporation to biological debris, and subsequent release.

Environmental Transport, Distribution and Atmospheric Deposition

Cadmium is removed from the atmosphere by dry deposition and by precipitation. In rural areas of Scandinavia, annual depositions rates of 0.4-0.9

g/ha have been measured (Laamanen, 1972; Andersson, 1977). Similarly, in a rural region of Tennessee, USA, a deposition rate of 0.9 g/ha was observed (Lindberg, et al 1982). In Ghana not much data have been recorded so far as Essumang et al 2005 showed in their studies of fallout from vehicular activities in the Kumasi metropolis.

Transport from Water to Soil

Rivers contaminated with cadmium can contaminate the surrounding land, either through irrigation for agricultural purposes, by the dumping of dredged sediments, or through flooding (Forstner, 1980; Sangster, 1984). For example, agricultural land adjacent to the Neckar River, Germany, received dredged sediments to improve the soil, a practice that produced soil cadmium concentrations in excess of 70mg/kg (Forstner, 1980). Much of the cadmium entering fresh waters from industrial sources are rapidly adsorbed by particulate matter, where it may settle out or remain suspended, depending on local conditions. This can result in low concentrations of dissolved cadmium even in rivers that receive and transport large quantities of the metal (Yamagata, & Shigematsu, 1970).

Cadmium Exposure and Human Health

It has been well established that excess cadmium exposure produces adverse health effects on human beings. For virtually all chemicals, adverse health effects are noted at sufficiently high total exposures. For certain elements such as copper and zinc which are essential to human life, a deficiency as well as an excess can cause adverse health effects. Cadmium is not regarded as

essential to human life. The relevant questions with regard to cadmium exposure are the total exposure levels and the principal factors which determine the levels of cadmium exposure and the adsorption rate of the ingested/inhaled cadmium by the individual, in other words, the pathways by which cadmium enters the food chain, the principal pathway of cadmium exposure for most human beings.

Much of the cadmium which enters the body by ingestion comes from terrestrial foods. This is to say, from plants grown in soil or meat from animals which have ingested plants grown in soil contaminated with cadmium. Thus, directly or indirectly, it is the cadmium present in the soil and the transfer of this cadmium to food plants together with the cadmium deposited out of the atmosphere on edible plant parts which establishes the vast majority of human cadmium intake, 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).

General Methods on Mercury and Cadmium Analysis Neutron Activation Analysis

This activation method or analysis is based upon the measurement of radioactivity that has been induced in samples by irradiation with neutrons or charged particles, such as hydrogen, deuterium or helium ions.

Neutrons and Neutron Sources

Three sources of neutrons are employed in neutron activation analysis: reactors, radionuclide, and accelerators. All three produce highly energetic neutrons (in the MeV range), which are usually passed through a moderating material that reduces their energies to a few hundredths of an electron volt.

Most activation methods are based upon thermal neutrons, which react efficiently with most elements of analytical interest.

Nuclear rectors are sources of copious thermal neutrons and are, therefore, widely used for activation analyses. A typical research reactor will have a neutron flux of 10^{11}to10^{14}ncm^{-2}s^{-1}. These high neutron densities lend to detection limits that for many elements range from 10^{-3} to 10mg.

Radioactive Neutron Source

Radioactive isotopes are convenient and relatively inexpensive sources of neutrons for activation analyses. Their neutron flux densities range from 10^5 to 10^{10} n cm^{-2} s^{-1}. As a consequence, detection limits are generally not as good as those in which a reactor serves as a source.

Accelerators

Bench top charged particle accelerators are commercially available for the generation of neutrons. A typical accelerator consists of an ion source that delivers deuterium ions to an area where they are accelerated through a potential of about 150kv to a target containing tritium absorbed on titanium or zirconium. The reaction is:

$^{2}{}_{1}H + {}^{3}{}_{1}H \rightarrow {}^{4}{}_{1}He + {}^{1}{}_{0}D$

Neutrons produced in this way have energies of about 14 MeV and are useful for activating the lighter elements.

Interactions of Neutrons with Matter

Free neutrons are not stable, and they decay with a half –life of about 10.3 min to give protons and electrons. Free neutrons do not, however, generally exist long enough to disintegrate in this way because of their great tendency to react with ambient material. The high reactivity of neutrons arise form their zero charge that permits them to approach charged nuclei without interference from columbic forces. Neutron Capture is the most important reaction for activation analysis. Here, a neutron is captured by the analyte nucleus to give an isotope with the same atomic number, but with a mass number that is greater by one.

An example of a reaction that produces prompt gamma rays is:

$^{23}{}_{11}Na + {}^{1}{}_{0}n \rightarrow {}^{24}{}_{11}Na + {}^{0}{}_{0}\gamma$

The prompt gamma rays formed by capture reactions are of analytical interest in special cases; but the radionuclide produced (^{24}Na) is generally of far greater utility.

Theory of Activation Analysis

When the sample is exposed to a flux of neutrons, thereto of formation of radioactive nuclei from a single isotope can be shown to be $dN^*/dt = N\Phi$ 1

Where dN^*/dt is the formation rate of active particles in neutrons per second (n/s), N is the number of stable target atoms, Φ is the average flux in $cm^{-2}s^{-1}$

and σ is the capture across section in cm^2/target atom. The last is the measure of the probability of the nuclei reacting with a neutron at the particle energy employed.

Once formed, the radioactive nuclei decay at a rate $-dN^*/dt$ given by equation 1. That is $-dN^*/dt = \lambda . N^*$.

Thus during irradiation with a uniform flux of neutrons, the net rate of formation of active particles is:

$dN^*/dt = N \Phi \sigma - \lambda . N^*$ 2

When this equation is integrated from 0 to t one obtains

$N^* = N \Phi \sigma / \lambda . [1 - \exp(-\lambda . t)]$.....3

If $\lambda = 0.693/t_{½}$ then 3 yields

$N^* = N \Phi \sigma / \lambda . [1 - \exp(-0.693\ t/t\ ½)]$.....4

Rearranging 4 yields

$A = \lambda . N^* = N \Phi \sigma [1 - \exp(-0.693t/t½)] = N \Phi \sigma S$.....5

Where A = activity, S saturation factor = $[1 - \exp(-0.693t/t½)]$

The foregoing equation can be rewritten in terms of experimental rate measurements as:

$R = N\Phi \sigma c S$ 6

In many analyses irradiation of sample and standards is carried out for a long enough period to reach saturation. Under this circumstance, all of the

terms except N on the right side of equation 6 are constant, and the number of analyte radionuclides is directly proportional to the counting rate. If the parent or target nuclide is naturally occurring, the weight of the analyte w can be obtained from N by multiplying it by Avogadro's number N, the natural abundance of the analyte isotope, and the chemical weight. Since all of these are constants, the weight of analyte is directly proportional to the counting rate. Thus, if we use the subscripts x and s to represent sample and standard, respectively, we may write

$R_x = K\, w_x$.....7

$R_x = K\, w_s$ .....8 (Skoog et al 2001).

Where k is proportionality constant. Dividing one equation by the other and rearranging leads to the basic equation for computing the weight of analyte in an unknown:

$Wx = (R_x/R_s)^* w_s$

Application of Neutron Activation

Neutron activation methods offer several advantages including high sensitivity, minimal sample preparation and ease of calibration. Often these procedures are non destructive and for this reason are applied to the analysis of art objects, coins, forensic samples and archaeological specimens. The major disadvantages of activation methods are the need for large and expensive equipment and special facilities for handling and disposing of radioactive

materials. Another handicap is the long time required to complete analyses when longlived radionuclide is used.

Scope

The neutron activation is potentially applicable for the determination of 69 elements. In addition, four of the inert gases form active isotopes with thermal neutrons and thus can also be determined. Finally, three additional elements – oxygen, nitrogen and yttrium – can be activated with fast neutrons from an accelerator. A list of types of materials to which the method has been applied is impressive and includes metals, alloys, archaeological objects, semiconductors, biological specimens, rocks, minerals and water.

Accuracy

The principal errors that arise in activation analysis are due to self-shielding, unequal neutron flux at sample and standard, counting uncertainties, and errors in counting due to scattering, absorption and differences in geometry between sample and standard. The errors from these sources can usually be reduced to less than 10% relative: uncertainties in the range of 1% to 3% are frequently obtainable.

Sensitivity

The most important characteristic of the neutron activation method is its remarkable sensitivity for many elements. As little as 10^{-5} mg of several elements can be detected.

The efficiency of chemical recovery, if required prior to radio assay, may limit the sensitivity of an activation analysis. Other factors include the sensitivity of

the detection equipment for the emitted radiation, the extent to which the activity in the sample decays between irradiation and assay, the time available or counting, and the magnitude of the background count with respect to that for the sample. A high rate of decay is desirable from the standpoint of minimizing the duration of the counting period. Concomitant with high decay rates, however, is the need to establish with accuracy the time lapse between the cessation of irradiation and the commencement of counting. A further potential complication is associated with counting rates that exceed the resolving time of the detecting system; under these circumstances, a correction must be introduced to account for the difference between lapsed clock and live (real) counting times (Skoog et al 2001).

Cold – Vapour Atomic Absorption Spectrometry

The cold-vapour technique is an atomization method applicable only to the determination of mercury because it is the only metallic element that has an appreciable vapour pressure at ambient temperature. The method of choice for this analysis is cold vaporizations followed by atomic absorption spectrometry. In performing analysis of this type, mercury is converted to Hg^{2+} by treatment of the sample with an oxidizing mixture of nitric and sulphuric acids followed by reduction of the Hg^{2+} to the metal with $SnCl_2$.

The equation for the reaction is giving below:

$$Hg^{+} + HNO_3/H_2SO_4 \longrightarrow Hg^{2+}$$

$$Hg^{2+} + SnCl_2 \longrightarrow Hg^{0}$$

The elemental mercury is swept into a long-pass absorption tube by bubbling a stream of inert gas through the mixture from which the element was formed. Measuring the absorbance at 253.7nm completes the analysis. Detection limits in the parts – per- billion ranges are realized.

Atomic Absorption Instrumentation

Instruments for atomic absorption spectrometry (AAS) are similar in design and consist of a radiation sources, a sample holder, a wavelength selector, a detector and a signal processor and readout. The sample holder in atomic absorption instruments in the atomizer cell that contains the gaseous atomized sample (Skoog et al 2001).

The Operational Definition of pH

The utility of pH as a measure of the acidity or alkalinity of aqueous media has made the potentiometer measurement of pH one of the most common techniques in all science.

The operational definition of pH endorsed by National Institute of standards and Technology (NIST), similar organizations in other countries and the IUPAC is based upon the direct calibration of the meter with carefully prescribed standard buffers followed by potentiometric determination of the pH of the unknown solutions.

Potentiometric pH Measurements with a Glass Electrode.

The glass electrode is a remarkably versatile tool for the measurement of pH under many conditions. The electrodes can be used without interference in solutions containing strong oxidants, reductant, gases and proteins (a calomel

reference electrode rather than a silver/silver reference electrode). The pH of viscous or even semisolid fluids can be determined.

Special Electrodes for pH Measurements

Among these include small electrodes for pH measurements in a drop (or less) of solution or in a cavity of a tooth micro- electrodes that permit the measurement of pH inside a living cell, systems for insertion in a flowing liquid stream to provide a continues monitoring of pH. A small glass electrode can be swallowed to indicate the acidity of the stomach contents (the reference in kept in the mouth), and a combination electrode contains both the indicator and reference electrodes in a single probe.

Summary of Errors Affecting pH Measurements with Glass Electrodes

The ubiquity of the ph meter and the general applicability of the glass electrode tend to lull the chemist into the attitude that any measurement obtained with such an instrument is surely correct. It is well to guard against this false sense of security because there are distinct limitations to the electrode system. The errors are:

1. The alkaline error: Modern glass electrodes become somewhat sensitive to alkali – metal ions at pH values greater than 11 to 12
2. The acid error: At a pH less than 0.5, causes obtained with a glass electrode tend to be somewhat high.
3. Dehydration: Dehydration of the electrode may cause unstable performance and errors

4. Errors in low Ionic strength: It has been found that significant errors (as much as 1 or 2 pH units) may occur when the pH or low ionic strength sample, such as lake or stream samples, are measured with a glass (calomel electrode system. The prime source of such errors has been shown to be non-reproducible junction potentials, which apparently result from partial clogging of the fritted plug or porous fibre that is used to restrict the flow of liquid from the salt bridge into the analyte solution.

5. Variation in junction potential:Variation in the junction potential between the standard and sample leads to a fundamental uncertainty in the measurement of pH for which a correction cannot be applied. Absolute values more reliable than 0.10-pH unit are generally unobtainable. Even reliability to 0.03-pH unit requires considerable solutions of pH changes in a single solution that are as small as 0.001 units. For this reason, many pH meters are designed to permit readings, less than 0.01 pH units.

6. Error in the pH of the standard buffer: Any in accuracies in the preparation of the buffer used for calibration, or changes in its composition during storage, will be propagated as errors in pH measurements. A common cause of deterioration is the action of bacteria on organic components of buffers (Skoog et al 2001).

The Blue Tilapia

The Blue Tilapia, *Tilapia zillii* constitute a large group of food fishes widely distributed in mainly the tropical and subtropical areas of the world. They are still useful in the aspect of pond management. Some aquaculturists use tilapias as both as feed for brood fish and carnivorous fishes and the control of unwanted aquatic vegetation.

Although they are not carnivorous, they are very aggressive, especially when nesting and raising young. Blue Tilapia has few diseases and parasites. Although they are basically freshwater fishes, most of the species can live in brackish water and some adapt readily to seawater. Blue tilapias are fast moving, up-feeding and non-predatory, with low contamination of pollutants. Tilapias occupy low trophic levels, eating phytoplankton, zooplankton, decaying organic matter, depending on the species (Spataru et.al 1978).

European Green Crab

The European Green Crab, *Carcinus maenas*, is a voracious predator that feeds on many types of organisms, particularly bivalve molluscs (example clams, oysters and mussels), polychaetes, and small crustaceans. The green crab is slow moving, bottom-dwelling/feeding, hence highly polluted contaminated with pollutants.

Despite its name, the European Green Crab occasionally is not green. The dorsal (top) shell or carapace is mottled, dark green in colouration, and has small, yellow patches. Its ventral surface (underside colour may change from

green to orange and then red during the molting cycle. The most distinctive characteristic separating it from other Pacific Northwest crabs is the array of 5 spines on either side of the eyes on the front end of the carapace. The 3 rounded lopes (bumps) between its eyes may also be used to help identify the European green crab. An adult European Green Crab is typically about 2.5 inches long, but can range up to 4 inches (Attrill et al 1996).

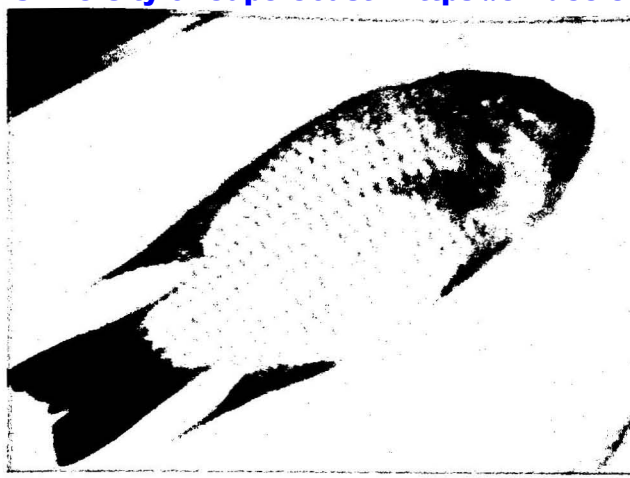


Diagram 1: Picture of the Blue Tilapia, *Tilapia zillii*

Diagram 2. Picture of the European Green Crab, *Carcinus maenas*

CHAPTER THREE

EXPERIMENTAL

Irradiation source

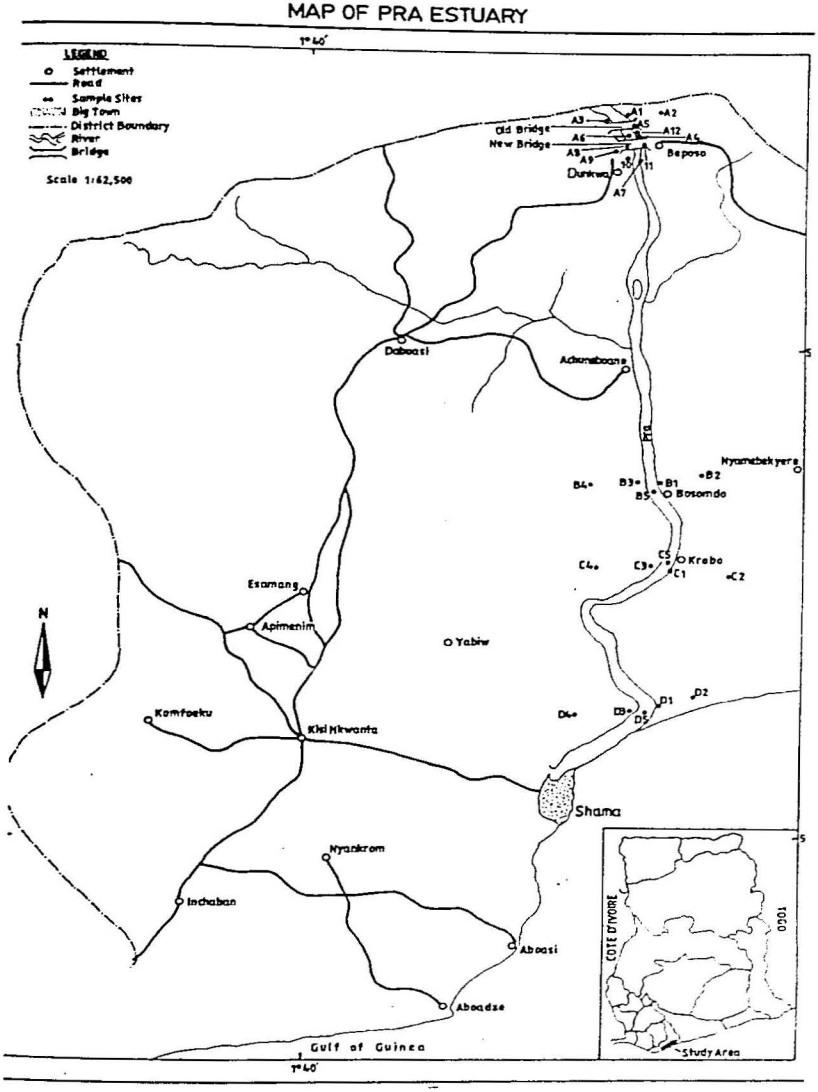
The irradiation source was a 20-Curie Americium – Beryllium (Am-Be) radioactive neutron source. It is cylindrically shaped and is fixed in a holder at the center of a fibre – glass tank, filled with de-ionised water. The de-ionised water serves a dual purpose of moderator and also absorber of neutrons. Concrete blocks arranged round the tank provide extra shielding. Transfer of sample to and from the neutron source was by means of a flexo-rabbit pneumatic transfer system operating under a pressure of 15 psi, giving a sample transfer time of 1.3 seconds (Tetteh, G.K. – 1989). The thermal neutron flux at the irradiation site was 5 x 10^{11} $ns^{-1}cm^{-2}$ by (Osae, E.K. et al – NNRI/GAEC/ 1989).

Study Area (Sites)

The target area was the Pra Estuary in the Western region of Ghana. Pra is in the Eastern Part of Shama Ahanta District Assembly. Sampling started from the Beposo Township, where the Bridge separates the northern section of the river and stretches from Beposo Township –to about 10km through the following villages.

(i) Bosomdo (ii) Krobo and (iii) Kedzi (Shama Beach)

Below is the map of the sampling area:



Sample Collection

Seventy-seven soils, nineteen water samples, sixteen tilapias and fourteen crabs, were collected from eight (8) sites (A. Beposo, B. Bosomdo, C. Krobo, D. Shama Beach. Control: Samples were also obtained from Dawukwa, Okyereko, Atakyedo and Sankor). Before transporting them to the laboratory, the pH of the water samples were taken.

The rubber sample containers were pre-treated by washing them with detergent, well-cleaned with de-ionised water and dried in an oven prior to sampling date. The soil samples were taken by hand with gloves into the rubber sample containers. Long bamboo stick, with one end open, such that there is short distance between a node and an opening, was used to scoop the sediment from the river bed. The sediment was transferred into the sample rubber containers.

Two species of fish (*Tilapia zillii, Carcinus maenas)* were used for this analysis. Four prominent fishing shores along the estuary of the river Pra were also chosen as the sampling sites. Fishing nets were set in all the sampling sites a day before the sampling date and this was left in the hands of caretakers. Fish samples were taken by hand with gloves, from the net, kept in rubber container and stored in an ice chest until further treatment.

Diagrams 3, 4, 5 and 6: Pictures of the Researcher sampling

Diagram 3

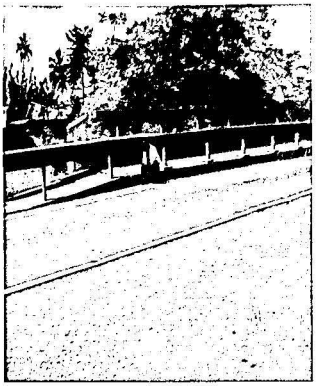


Diagram 4

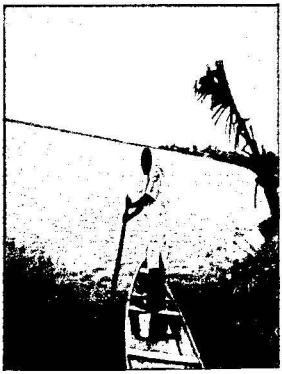


Diagram 5

Diagram 6

From the eight sites, samples were taken at a two week interval for two (2) months. Selected, composite soil and water samples each weighing 100grams, 500mL respectively were loaded into rabbit plastic containers for irradiation. Each of the samples was sent by the pneumatic transfer system into the Am-Be source for irradiation. This is because the system allows only one sample irradiation at a time. The irradiation schemes were chosen so as take into account the half-life of the radionuclide. In this regard, the following irradiation times were chosen: I hour, 24 hours, 600 sec medium counting. At the end of each irradiation the sample was returned for counting, with the appropriate delay (cooling) time allowed where necessary.

Sample Preparation and Storage

Soil samples collected were air-dried for 5 days in order to remove moisture. Dried soils were ground in an agar mortar and well sieved. About 100g of the samples were weighed, sealed and well wrapped into a clean polyethylene material. The capsule together with the IAEA Standard Reference materials (SARM-7 and SARM-9 and Hg, Cd) were packed in a large irradiation vial (rabbit capsule) and heat-sealed. Satisfied standard for Hg and Cd were prepared by IAEA (TECDOC-1443, 2005).

The water samples were kept in a refrigerator for further analysis. About 500mL of water samples were pipetted into hard polyethylene vials. 0.5mL of the Standard Material (i.e. Mercury and Cadmium Standards) was pipette into an acetone-washed vial and encapsulated with sucrose. The vials were heat-sealed with a soldering rod and placed in a larger vial or rabbit capsule. The

samples were doubly encapsulated for irradiation owing to inherent leakage and heat from the reacting neutrons.

Each fish sample was dissected using a rubber knife and all the flesh isolated for further analysis. About 200mg of each fish sample was weighed into a clean polyethylene film, using Mettler Balance AE 163- BDH. The films were wrapped and heat sealed. These samples were labelled according to their site. The samples were packed into 7mL volume rabbit capsules for irradiation.

Data Processing

The detector type used for the counting of signals was an ENERTEC, High Purity Germanium (HPGe) detector of 3000 (+ve) bias and a resolution of 2.55 KeV for 1332 KeV photo peak of Co-60. The associated electronics are: high voltage supply (Canberra model 3105), Spectroscopy amplifier (Canberra model 2010) and Canberra Multi-Channel Analyser (MCA) series 35 – plus.

The signals from the detector were passed through the spectroscopy amplifier and then accumulated by MCA for a present time. The spectra from MCA were transferred to a DEL 350 microcomputer for analysis, using Gamma Spectrum Analysis software MAESTRO 32 supplied by the IAEA. This software identifies the various photo peaks and works out the areas under them.

By means of Excel spread sheet, each delayed time, Td was calculated and hence the concentration of mercury in each sample determined. The validation of the analytical procedure was undertaken by irradiating an IAEA Standard reference material of mercury, cadmium and (SARM 7 and 9), satisfied standard for Hg and Cd, under same experimental conditions.

Chemicals and Reagents

The following chemicals, reagents and standards were used:

1. Standard Reference Material, 1646a estuarine sediment, supplied by US Dept. of Commerce, National Institute of standards and Technology, Gai Thersberg, MD 20899
2. Standard Reference Material, 3133 Mercury standard solution, (contains HNO_3). Lot No: 991304, Supplied by US Dept. of Commerce, National Institute of Standard, and Technology.
4. Acetone, AnalaR Analytical reagent, Refractive index N_D^{20} – 1.3580 – 1.3600. Weight per/mol at 20^0C – 0.789 – 0.791g, BDH chemicals Ltd – Poole England
5. Sucrose, sigma Ultra, 99.5%. GC supplied by sigma-Aldric Inc. USA.
6. Adsorbent Pure Cotton Wool by, Cotton Dressing Industry Ltd., Accra-Ghana.

Equipment

The equipment used:

1. Soldering Rod, Ersa TE 40, 24v, 60w/350°C, BDH Chemicals Ltd – Poole England
2. Mesh [Din 4188] Prufsieb

   Min 0.5 mm- Max 500mm

   BDH Chemicals Ltd- Poole England

3. Eppendorf Research pH meter

BDH Chemicals Ltd- Poole England.

Identification, Quantification and Data Reduction

A modern Multi channel Analyser (MCA) containing about 4096-8192 channels of data, spanning the range of 60-2000Kev was used. The evaluation of gamma spectra involved several steps, such as locating peaks in the spectrum, determination of peak energies and net areas and calculating the elemental concentrations. Because of the large amount of data, these procedures were performed in a set of sophisticated computer program (MAESTRO 32). Once peak search algorithms detected the peak, the peak area is determined by various methods, which required clean and well – resolved peaks.

Normalisation of the weights between standard reference materials (mercury, cadmium and (SARM 7 and 9) and the unknowns was done by an excel software. The overall equation was as follows:

$C_{sam}=C_{std}\ (A_{sam}/A_{std})\ (D_{std}/D_{sam})\ (C_{std}/C_{sam})\ (W_{std}/W_{sam})$ where W_{sam} and W_{std} are the weights of the sample and the standard respectively.

C_{sam} is the unknown concentration of the element in the sample, C_{std} is the known concentration of the element in the standard, A_{sam} is the activity of the sample and A_{std} is the activity of the standard. D_{std} and D_{sam} are the delayed times also (td) of the standard and samples respectively.

Validation and Quality Control Analysis

Validation of the analytical procedure was undertaken by irradiating an IAEA standard reference material: Hg- 9.94μg/g and Cd- 9.5μg/g, with 5% uncertainty and counting under identical experimental conditions.

Standard Material	Concentration of Mercury and Cadmium in μg/g						
	Std	1	2	3	4	Mean	% Recovery
Hg	9.94	9.81	9.82	9.84	9.85	9.83	98.89
Cd	9.5	9.4702	9.4779	9.4700	9.4710	9.4727	99.70

pH Determination

Determination of pH of Soil Sample

About 1.00g of dried soil sample was exactly weighed and suspended in 100mL of distilled water for 24hours. The supernatant solution was filtered to get a clear solution. The pH of the subsequent solution was determined using the Eppendorf Research pH meter.

Determination of pH of Water Samples

The frozen water samples were allowed to attain the normal room temperature of about 26^0C. The pH's of the water samples was determined with Eppendorf Research pH meter.

Sample Trail Calculations

Beposo

Sample number	Number of samples measured	Hg content in µg/g	Mean, $\bar{x}$, µg/g of Hg	Sum of squares of Deviation from means $x10^{-6}$
A1	4	0.00146,0.0041,0.0038,0.00401	0.0033425	82.296
A4	4	0.00319,0.0061,0.00246,0.00256	0.0035775	8.7973
A5	4	0.00358,0.0043,0.00365,0.00367	0.0038	0.3322
A7	4	0.0059,0.00366,0.00367,0.0064	0.0049075	6.3005
AW	2	0.00006,0.0001	0.00008	0.0008
No 0f measurements	=18		Sum of squares	=97.7268

The values in columns 4 and 5 for sample 1 were computed as follows:

X_1	$\lvert x_1 - \bar{x} \rvert$	$\lvert x_1 - \bar{x} \rvert^2 \times 10^{-6}$
0.00146	0.0018825	3.54
0.0041	0.007575	57.38
0.0038	0.004575	20.93
0.00401	0.0006675	0.446
0.01337	sum of squares	=82.296

$\bar{x} = \underline{0.01337} = 0.0033425$

4

The other data in columns 4 and 5 were obtained similarly, then

$$\sigma_{pooled} = \sqrt{\frac{\sum_{i=1}^{N1}\left(x-\bar{x}\right)^2 + \sum_{i=2}^{N2}\left(x_2-\bar{x}\right)^2 + \ldots\ldots\ldots + \sum_{i=5}^{N5}\left(x_5-\bar{x}\right)^2}{N1+N2+N3+N4+N5+\ldots.-nt}}$$

$$\sigma_{pooled} = \sqrt{\frac{(82.296+8.7973+0.3322+6.3005+0.0008)x10^{-6}}{18-5}}$$

$$= \sqrt{\frac{(97.716)x10^{-6}}{13}}$$

$$= \sqrt{7.517446x10^{-6}}$$

$$= 2.74179 \times 10^{-6} = 0.00274179$$

$$\Rightarrow Z = \frac{x-\mu}{\sigma} = \frac{0.01337 - 0.0033425}{0.00274179}$$

$$= \frac{0.0100275}{0.00274179} = 3.66$$

Confidence level,%	Z	Confidence level,%	Z
50	0.67	96	2.00
68	1.00	99	2.58
80	1.29	99.7	3.00
90	1.64	99.9	≤3.29
95	1.96		

CHAPTER FOUR

RESULTS AND DISCUSSION

The results from the sampling areas is presented below during the sampling period

TABLE 1: MEAN Hg AND Cd CONC. OF SHOULDER SOILS AT VARIOUS SITES			
Site	Hg/µg/g	Cd/µg/g	pH
Beposo	nd	0.026±0.0035	8.32
Bosomdo	nd	0.013±0.002	8.02
Krobo	nd	0.0094±0.00014	8.15
Shama Beach	nd	0.0267±0.004	8.35

TABLE 2: MEAN Hg AND Cd CONC. IN RIVER BED SEDIMENT AT THE VARIOUS SITES			
Site	Hg/µg/g	Cd/µg/g	pH
Beposo	3.95±0.55	0.0379±0.0017	5.56
Bosomdo	1.86±0.29	0.018±0.0027	6.89
Krobo	0.9±0.15	0.01±0.0016	7.81
Shama Beach	0.21±0.03	0.029±0.0044	8.17

TABLE 3: Hg AND Cd CONC. µg/g IN SHOULDER SOILS AND RIVER BED SEDIMENT AT BOSOMDO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
Sites	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Shoulder 2	nd	0.0503±0.007	8.26	nd	0.0062±0.0032	8.38	nd	0.0021±0.0003	8.02	nd	nd	8.29
shoulder 3	nd	0.0254±0.0038	8.37	nd	nd	8.22	nd	nd	8.31	nd	0.0043±0.0006	8.27
shoulder 8	nd	0.042±0.0063	8.28	nd	0.0025±0.0006	8.31	nd	nd	7.61	nd	0.0106±0.0018	8.33
sediment	3.58±0.54	0.0068±0.001	5.46	4.3±0.6	0.013±0.0019	5.32	3.65±0.55	0.012±0.0018	5.31	3.78±0.17	0.012±0.0018	5.33

TABLE 4: Hg AND Cd CONC. µg/g IN SHOULDER SOILS AND RIVER BED SEDIMENT AT BOSOMDO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Shoulder 1	nd	nd	8.37	nd	nd	8.21	nd	nd	8.33	nd	0.013±0.002	8.34
shoulder 4	nd	0.0054±0.0003	8.22	nd	0.0076±0.00012	8.32	nd	0.0101±0.00152	8.2	nd	0.0103±0.0015	8.23
sediment	1.1±0.2	0.020±0.003	6.92	2.46±0.37	0.016±0.0025	5.98	1.3±0.2	nd	6.81	2.58±0.39	nd	5.82

TABLE 5: Hg AND Cd CONC. µg/g IN SHOULDER SOILS AND RIVER BED SEDIMENT AT KROBO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Shoulder1	nd	0.006±0.0009	8.38	nd	0.014±0.002	8.24	nd	nd	8.04	nd	0.0082±0.0001	8.36
shoulder 4	nd	0.018±0.0009	8.42	nd	0.0018±0.00027	8.33	nd	0.002±0.0003	8.02	nd	nd	8.25
sediment	0.2±0.05	0.001±0.002	8.14	2.13±0.32	0.007±0.0004	6.22	0.11±0.02	0.023±0.0035	7.98	1.2±0.2	nd	6.84

TABLE 6: Hg AND Cd CONC. µg/g IN SHOULDER SOILS AND RIVER BED SEDIMENT AT SHAMA BEACH FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Shoulder 1	nd	nd	8.27	nd	0.009±0.0003	8.27	nd	0.042±0.007	8.94	nd	0.024±0.003	8.3
shoulder 4	nd	0.0041±0.0006	8.2	nd	0.0153±0.0023	8.32	nd	0.021±0.00315	8.24	nd	0.001±0.0005	8.25
sediment	0.12±0.02	0.029±0.0044	8.19	0.51±0.08	nd	8.02	0.1±0.02	nd	8.13	0.11±0.02	nd	8.12

TABLE 9: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND WATER COLUMN AT KROBO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
sediment	0.2±0.05	0.001±0.002	8.14	2.13±0.32	0.007±0.0004	6.22	0.11±0.02	0.023±0.0035	7.98	1.2±0.2	nd	6.84
Water Column	nd	nd	7.61	0.022±0.003	0.002±0.0002	7.86	nd	0.0004±0.00006	7.02	nd	0.0007±0.00009	7.82

TABLE 10: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND WATER COLUMN AT SHAMA BEACH FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
sediment	0.12±0.02	0.029±0.0044	8.19	0.51±0.08	nd	8.02	0.1±0.02	nd	8.13	0.11±0.02	nd	8.12
Water Column	nd	0.0044±0.0001	7.08	0.07±0.001	0.0005±0.00008	7.21	nd	0.0012±0.0002	6.97	nd	0.002±0.0003	7.02

TABLE 11: Hg AND Cd CONC. µg/g IN WATER COLUMN AND SHOULDER SOILS AT BEPOSO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
Sites	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Shoulder 2	nd	0.0503±0.0.007	8.26	nd	0.0062±0.0032	8.38	nd	0.0021±0.0003	8.02	nd	nd	8.29
shoulder 3	nd	0.0254±0.0038	8.37	nd	nd	8.22	nd	nd	8.31	nd	0.0043±0.0006	8.27
shoulder 8	nd	0.042±0.0063	8.28	nd	0.0025±0.0006	8.31	nd	nd	7.61	nd	0.0106±0.0018	8.33
Water Column	nd	nd	7.97	0.6±0.01	0.0005±0.00006	7.98	0.1± 0.02	0.0007±0.00001	7.15	nd	0.00043±0.00019	7.22

TABLE 12: Hg AND Cd CONC. µg/g IN WATER COLUMN AND SHOULDER SOILS AT BOSOMDO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Water Column	nd	0.0003±0.00005	7.72	0.0004±0.00006	0.0005±0.00006	7.81	nd	nd	7.22	nd	0.0033±0.0005	7.32
Shoulder 1	nd	nd	8.37	nd	nd	8.21	nd	nd	8.33	nd	0.013±0.002	8.34
shoulder 4	nd	0.0054±0.0003	8.22	nd	0.0076±0.00012	8.32	nd	0.0101±0.00152	8.2	nd	0.0103±0.0015	8.23

TABLE 13: Hg AND Cd CONC. µg/g IN WATER COLUMN AND SHOULDER SOILS AT KROBO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Water Column	nd	nd	7.61	0.022±0.003	0.002±0.0002	7.86	nd	0.0004±0.00006	7.02	nd	0.0007±0.00009	7.82
Shoulder 1	nd	0.006±0.0009	8.38	nd	0.014±0.002	8.24	nd	nd	8.04	nd	0.0082±0.0001	8.36
shoulder 4	nd	0.018±0.0009	8.42	nd	0.0018±0.00027	8.33	nd	0.002±0.0003	8.02	nd	nd	8.25

TABLE 14: Hg AND Cd CONC. µg/g IN WATER COLUMN AND SHOULDER SOILS AT SHAMA BEACH FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
Water Column	nd	0.0044±0.0001	7.08	0.07±0.001	0.0005±0.00008	7.21	nd	0.0012±0.0002	6.97	nd	0.002±0.0003	7.02
Shoulder 1	nd	nd	8.27	nd	0.009±0.0003	8.27	nd	0.042±0.007	8.94	nd	0.024±0.003	8.3
shoulder 4	nd	0.0041±0.0006	8.2	nd	0.0153±0.0023	8.32	nd	0.021±0.00315	8.24	nd	0.001±0.0005	8.25

TABLE 15: MEAN Hg AND Cd CONC. µg/L IN WATER COLUMN AT THE VARIOUS SITES

Site	Hg/µg/L	Cd/µg/L	pH
Beposo	0.0175±0.075	0.00054±0.00009	7.58
Bosomdo	0.03±0.005	0.00123±0.0002	7.52
Krobo	0.0055±0.00075	0.001±0.00016	7.58
Shama Beach	0.018±0.0025	0.002±0.00031	7.02

TABLE 16: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND RIVER BANK SEDIMENT AT BEPOSO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
River Bed	3.58±0.54	0.0068±0.001	5.46	4.3±0.6	0.013±0.0019	5.32	3.65±0.55	0.012±0.0018	5.31	3.78±0.17	0.012±0.0018	5.33
River Bank 1	1.46±0.22	0.036±0.0054	6.92	4.1±0.6	0.002±0.00032	5.86	3.8±0.6	0.002±0.00032	5.98	4±0.6	nd	5.92
River Bank 7	5.9±0.9	0.093±0.014	5.21	6.4±0.92	0.027±0.004	5.02	3.66±0.6	0.0018±0.0003	5.3	3.67±0.16	0.00211±0.00032	5.42

TABLE 17: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND RIVER BANK SEDIMENT AT BOSOMDO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
River Bed	1.1±0.2	0.020±0.003	6.92	2.46±0.37	0.016±0.0025	5.98	1.3±0.2	nd	6.81	2.58±0.39	nd	5.82
River Bank 2	1.38±0.21	0.012±0.002	7.03	1.2±0.2	0.04±0.006	6.87	nd	nd	7.08	1.28±0.2	0.0119±0.0018	6.79
River Bank 3	1.26±0.2	0.012±0.002	6.76	1.23±0.2	0.039±0.0059	6.81	nd	0.003±0.0001	7.11	1.38±0.2	0.012±0.002	7.04

TABLE 18: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND RIVER BANK SEDIMENT AT KROBO FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
River Bed	0.2±0.05	0.001±0.002	8.14	2.13±0.32	0.007±0.0004	6.22	0.11±0.02	0.023±0.0035	7.98	1.2±0.2	nd	6.84
River Bank 2	1.04±0.16	0.013±0.0019	7.02	nd	0.0042±0.00062	7.32	nd	0.0021±0.00032	7.89	1.04±0.16	0.012±0.002	7.16
River Bank 3	1.12±0.17	0.018±0.009	6.9	nd	0.0018±0.00027	7.98	nd	0.002±0.0003	8.1	nd	nd	8.12

TABLE 19: Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND RIVER BANK SEDIMENT AT SHAMA BEACH FOR THE SAMPLING PERIOD

	SAMPLING DATES											
	10/05/2006			24/05/2006			08/06/2006			29/06/2006		
SITES	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH	Hg	Cd	pH
River Bed	0.12±0.02	0.029±0.0044	8.19	0.51±0.08	nd	8.02	0.1±0.02	nd	8.13	0.11±0.02	nd	8.12
River Bank 2	nd	nd	8.03	nd	nd	7.69	nd	0.014±0.0022	8.42	nd	0.002±0.0003	8.36
River Bank 3	nd	0.0041±0.0006	8.13	nd	0.015±0.0023	8.17	nd	0.021±0.00315	8.21	nd	0.001±0.0005	7.92

TABLE 20: MEAN Hg AND Cd CONC. µg/g IN RIVER BANK SEDIMENT AT THE VARIOUS SITES

Site	Hg/µg/g	Cd/µg/g	pH
Beposo	2.9±0.435	0.0133±0.002	5.71
Bosomdo	0.97±0.146	0.0213±0.0066	6.94
Krobo	0.4±0.06	0.0078±0.0012	7.57
Shama Beach	nd	0.008±0.00012	8.12

TABLE 21: MEAN Hg AND Cd CONC. IN RIVER BED SEDIMENT AND SHOULDER SOILS AT THE VARIOUS SITES

	River Bed		Shoulder Soils	
Site	Hg/µg/g	Cd/µg/g	Hg/µg/g	Cd/µg/g
Beposo	3.95±0.55	0.0379±0.0017	nd	0.026±0.0035
Bosomdo	1.86±0.29	0.018±0.0027	nd	0.013±0.002
Krobo	0.9±0.15	0.01±0.0016	nd	0.0094±0.00014
Shama Beach	0.21±0.03	0.029±0.0044	nd	0.0267±0.004

TABLE 22: MEAN Hg AND Cd CONC. IN RIVER BED SEDIMENT AND WATER COLUMN AT THE VARIOUS SITES

	River Bed		Water Column	
Site	Hg/µg/g	Cd/µg/g	Hg/µg/L	Cd/µg/L
Beposo	3.95±0.55	0.0379±0.0017	0.0175±0.075	0.00054±0.00009
Bosomdo	1.86±0.29	0.018±0.0027	0.03±0.005	0.00123±0.0002
Krobo	0.9±0.15	0.01±0.0016	0.0055±0.00075	0.001±0.00016
Shama Beach	0.21±0.03	0.029±0.0044	0.0175±0.0025	0.002±0.00031

TABLE 23: MEAN Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND RIVER BANK SEDIMENT AT THE VARIOUS SITES

	River Bed		River Bank	
Site	Hg/µg/g	Cd/µg/g	Hg/µg/g	Cd/µg/g
Beposo	3.95±0.55	0.0379±0.0017	2.9±0.435	0.0133±0.002
Bosomdo	1.86±0.29	0.018±0.0027	0.97±0.146	0.0213±0.0066
Krobo	0.9±0.15	0.01±0.0016	0.4±0.06	0.0078±0.0012
Shama Beach	0.21±0.03	0.029±0.0044	nd	0.008±0.00012

TABLE 24: MEAN Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND CRAB AT VARIOUS SITES OVER THE SAMPLING PERIOD

	RIVER BED		CRAB	
SITE	Hg	Cd	Hg	Cd
Beposo	3.95±0.55	0.0379±0.0017	-	-
Bosomdo	1.86±0.29	0.018±0.0027	5.86±0.879	0.01009±0.0015
Krobo	0.9±0.15	0.01±0.0016	4.27±0.641	0.00895±0.00013
Sh. Beach	0.21±0.03	0.029±0.0044	3.23±0.4845	0.00779±0.0012

TABLE 25: MEAN Hg AND Cd CONC. µg/g IN RIVER BED SEDIMENT AND TILAPIA AT VARIOUS SITES OVER THE SAMPLING PERIOD

	RIVER BED		TILAPIA	
SITE	Hg	Cd	Hg	Cd
Beposo	3.95±0.55	0.0379±0.0017	0.57±0.086	0.00293±0.00044
Bosomdo	1.86±0.29	0.018±0.0027	0.51±0.0765	0.00274±0.000411
Krobo	0.9±0.15	0.01±0.0016	0.47±0.0705	0.00254±0.000381
Sh. Beach	0.21±0.03	0.029±0.0044	0.44±0.066	0.00266±0.000399

CONTROL: MEAN Hg CONC. IN µg/g AT THE SITES

SITE	Sh.Soil	R. Bed	River Bank	Water Column	Tilapia	Crab
Dawukwa	nd	nd	nd	nd	nd	nd
Okyereko	nd	nd	nd	nd	nd	nd
Atakyedo	nd	nd	nd	nd	-	nd
Sankor	nd	nd	nd	nd	nd	nd

TABLE 26: MEAN Hg AND Cd CONC. µg/g OF SUBSTRATE AND pH

	CRAB		TILAPIA		Water (pH)	Soil (pH)
SITE	Hg	Cd	Hg	Cd		
Beposo	-	-	0.57±0.086	0.00293±0.00044	7.58	5.56
Bosomdo	5.86±0.879	0.01009±0.0015	0.51±0.0765	0.00274±0.000411	7.52	6.89
Krobo	4.27±0.641	0.00895±0.00013	0.47±0.0705	0.00254±0.000381	7.58	7.81
Sh. Beach	3.23±0.4845	0.00779±0.0012	0.44±0.066	0.00266±0.000399	7.02	8.17

Figure 1

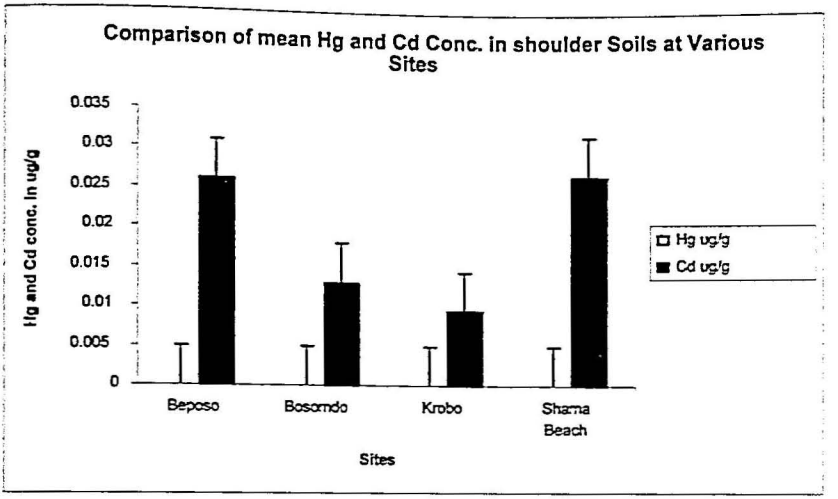
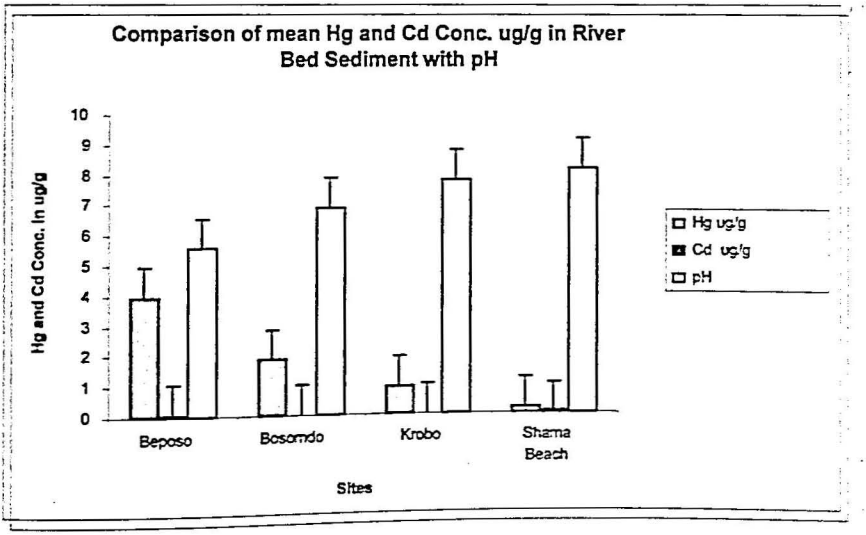


Figure 2



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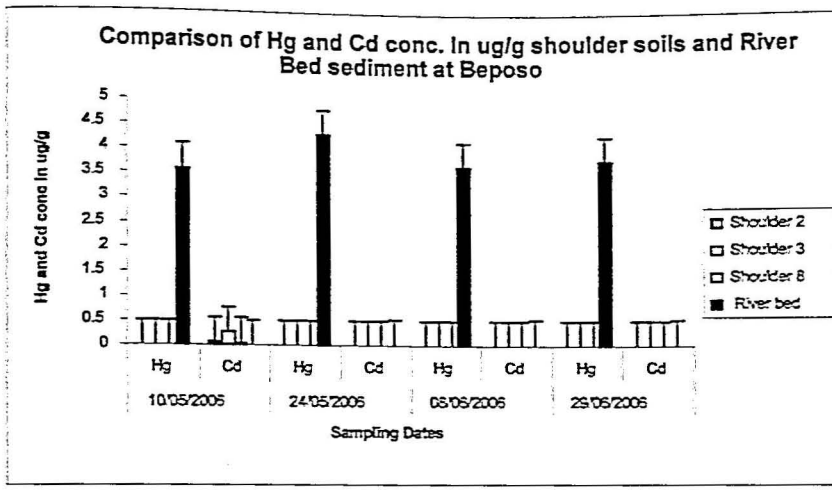

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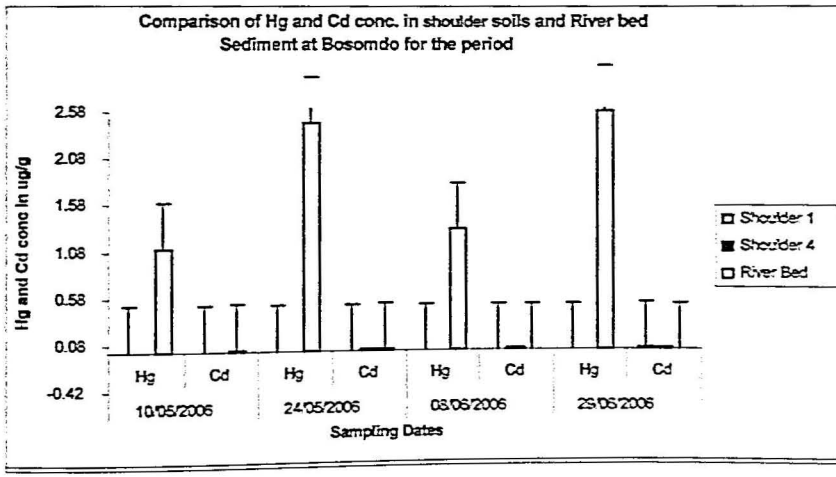

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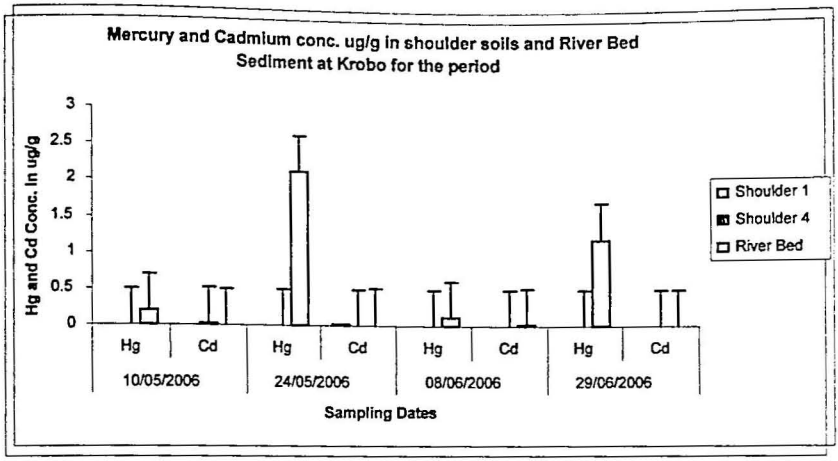

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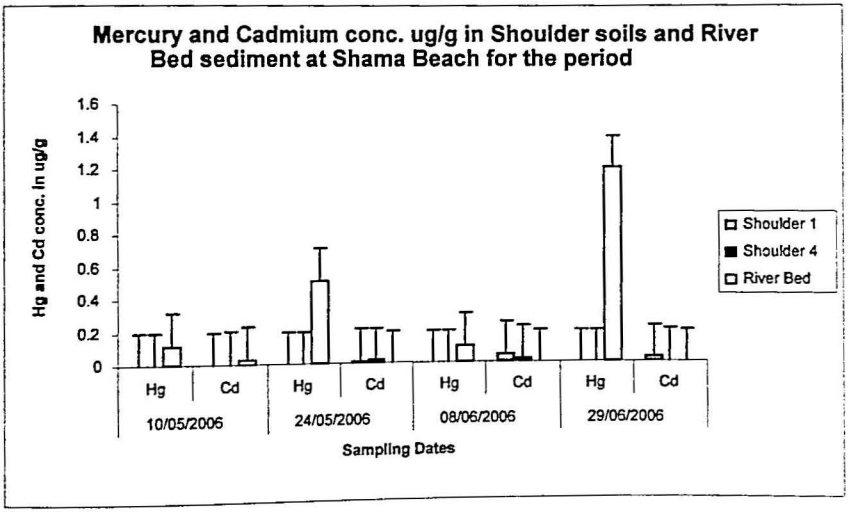

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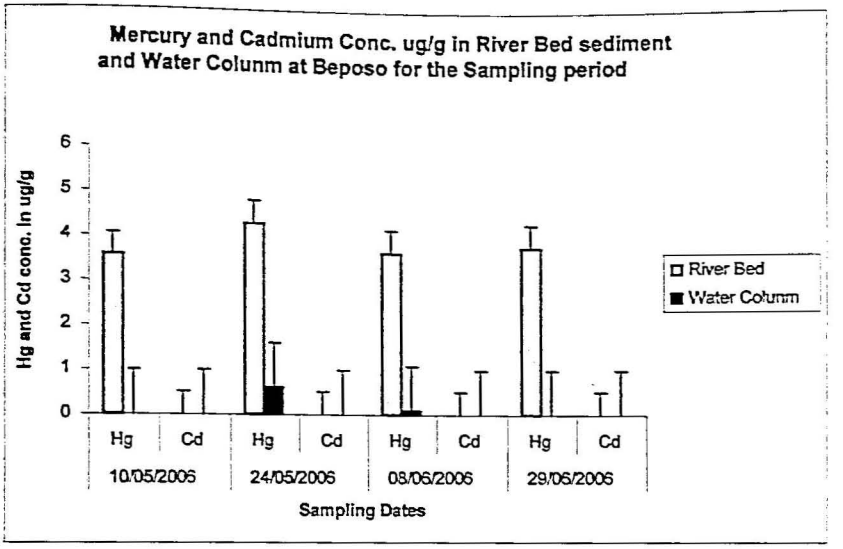

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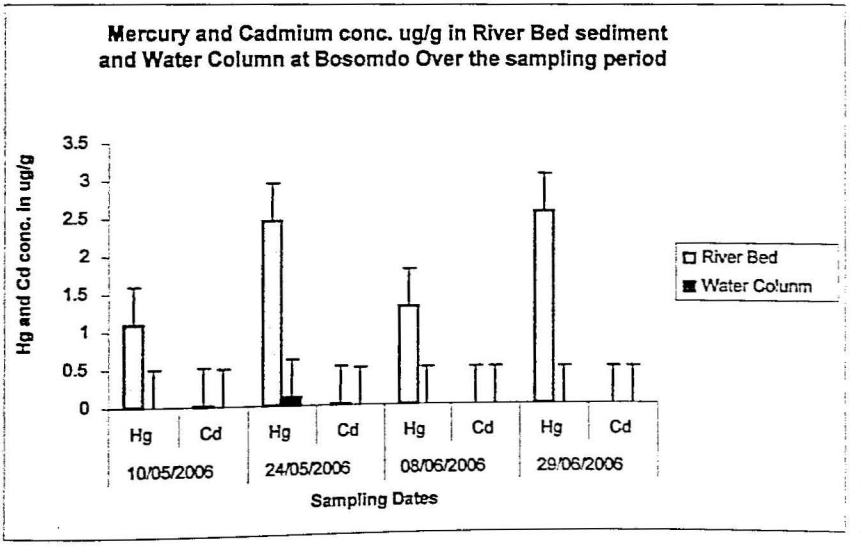

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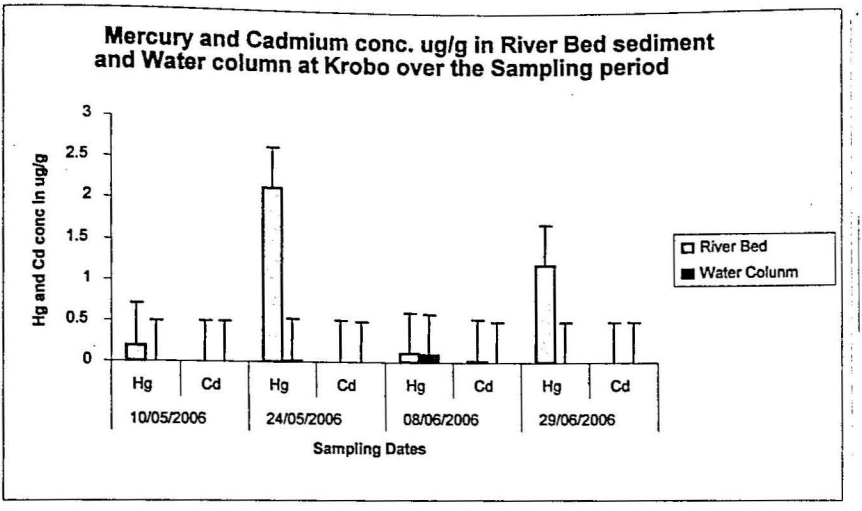

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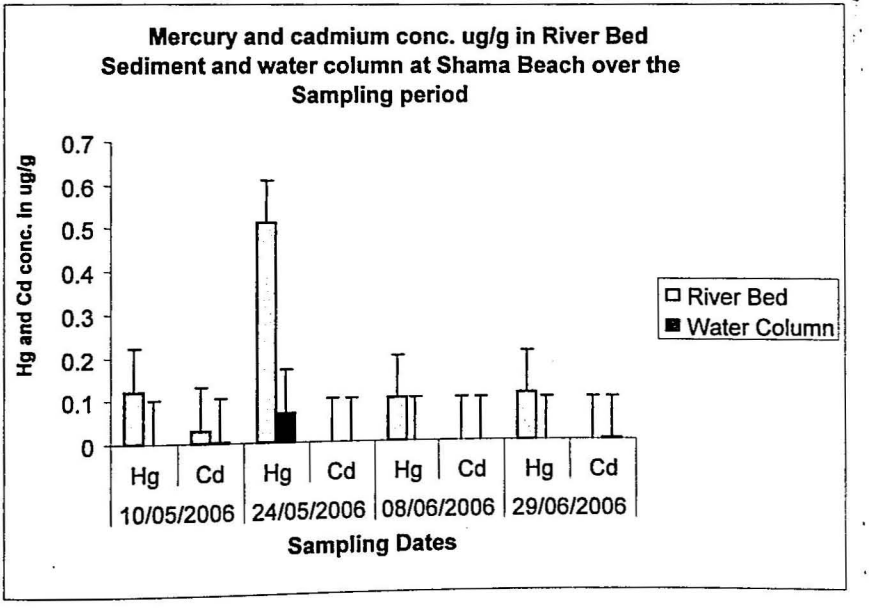

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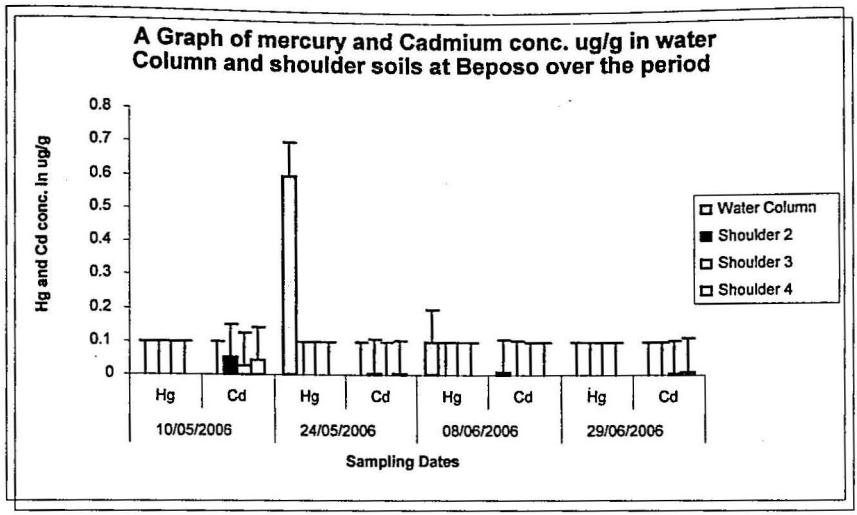

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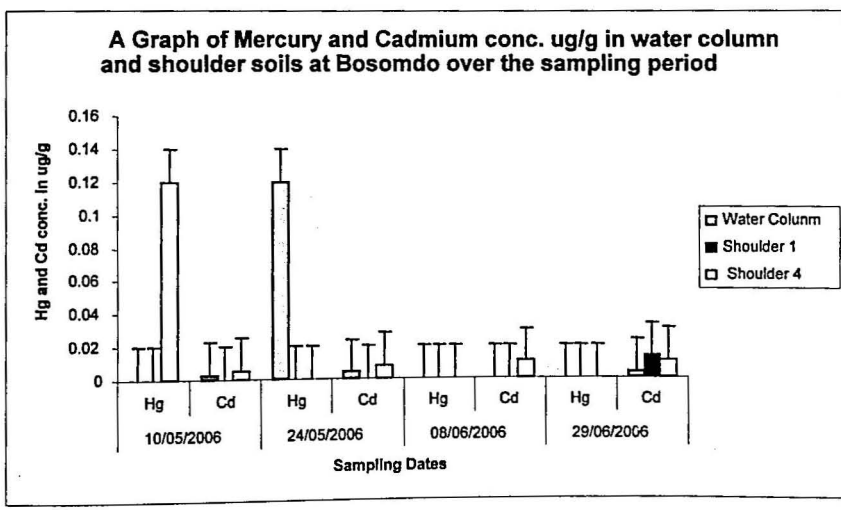

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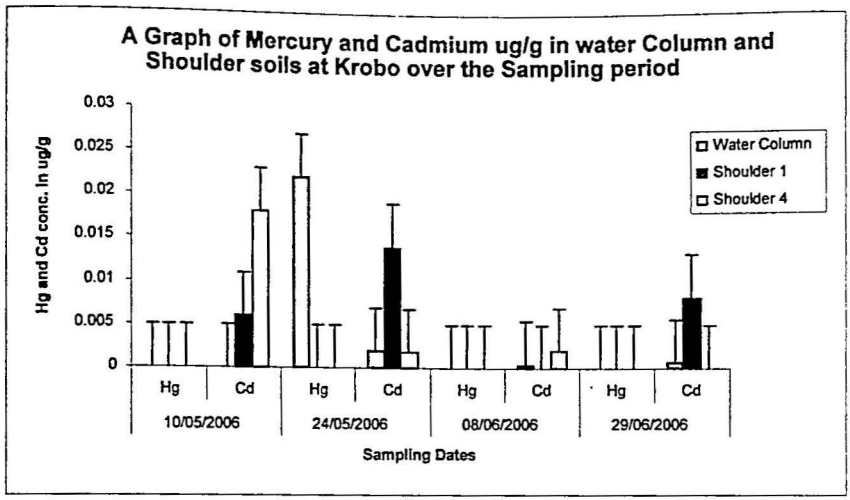

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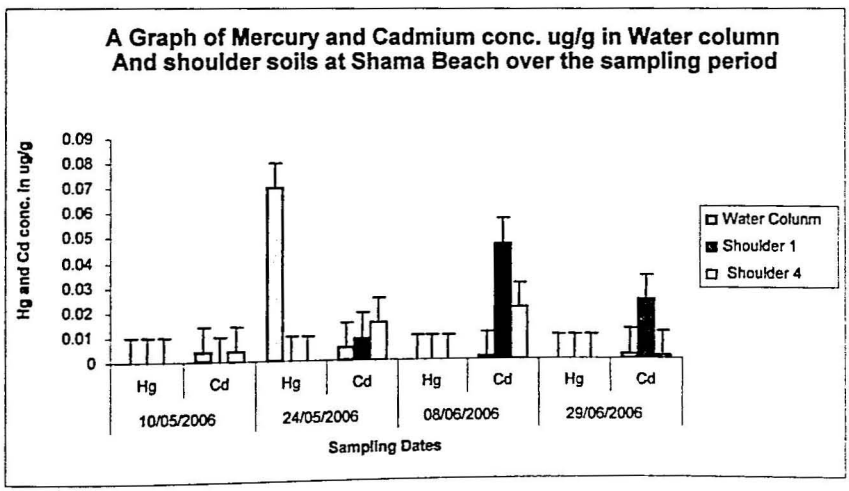

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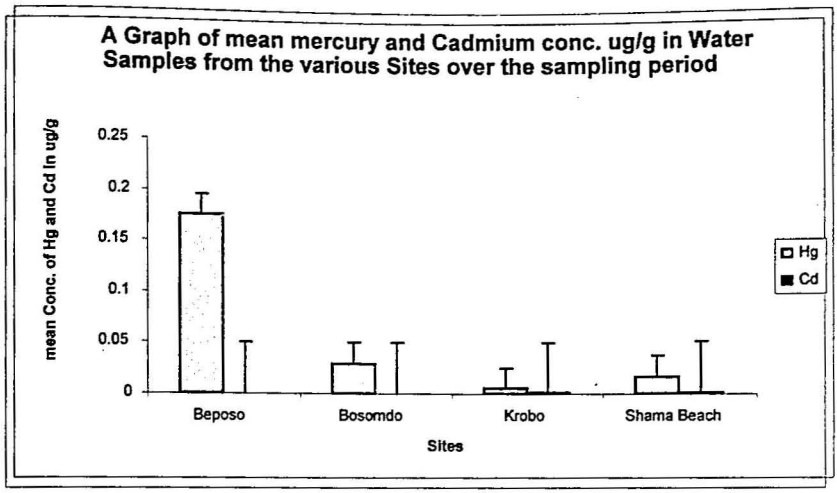

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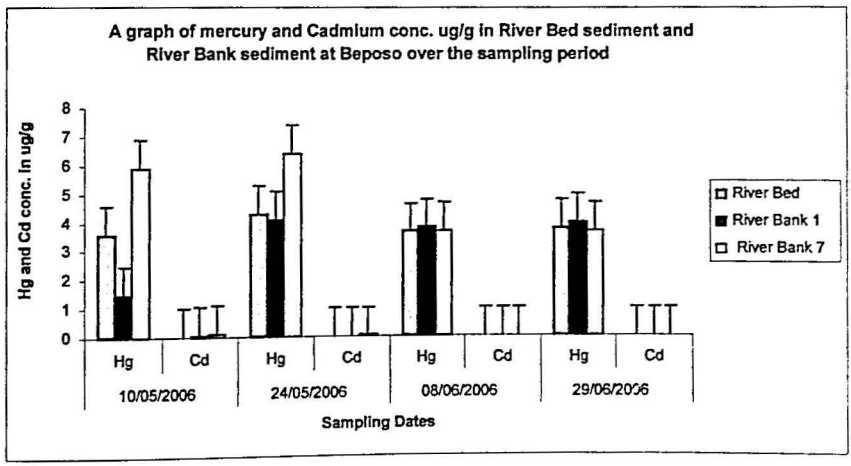


Figure 17

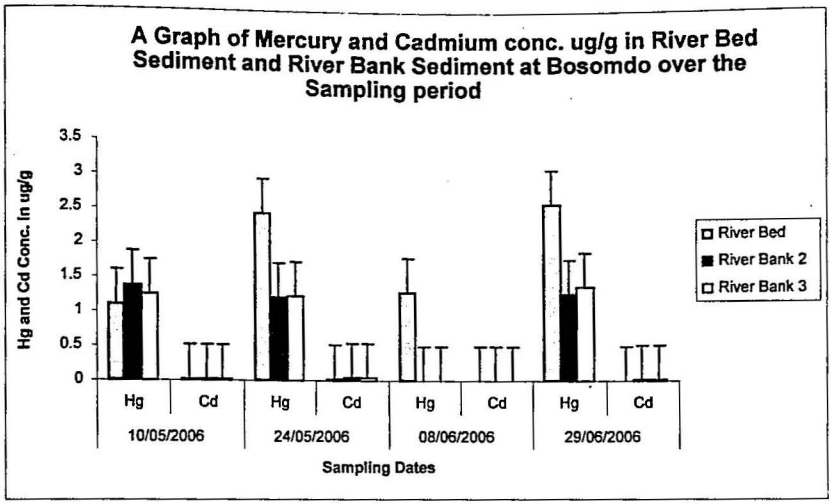
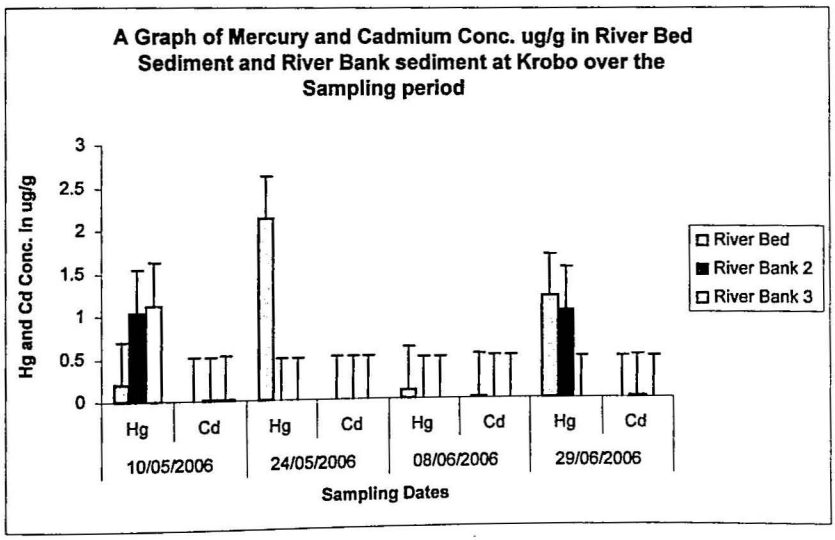


Figure 18



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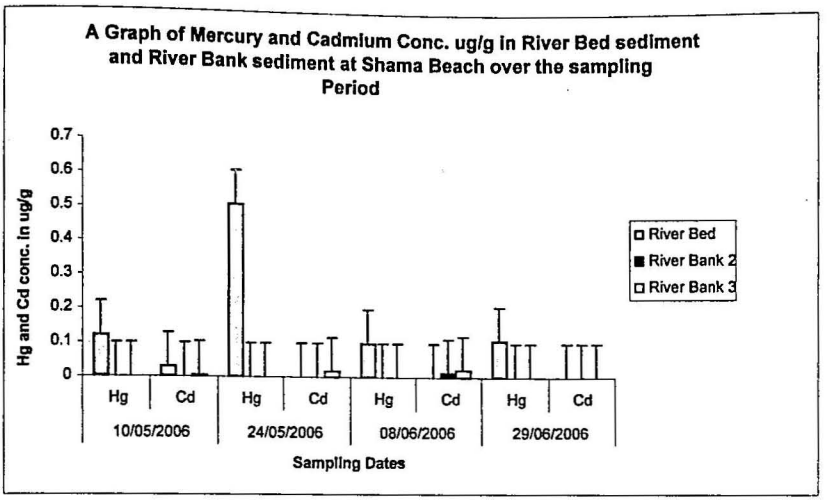

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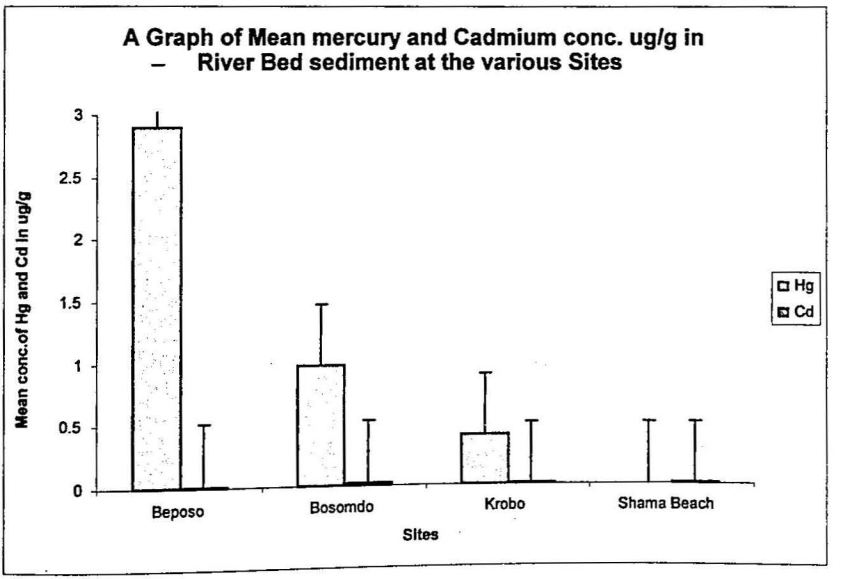

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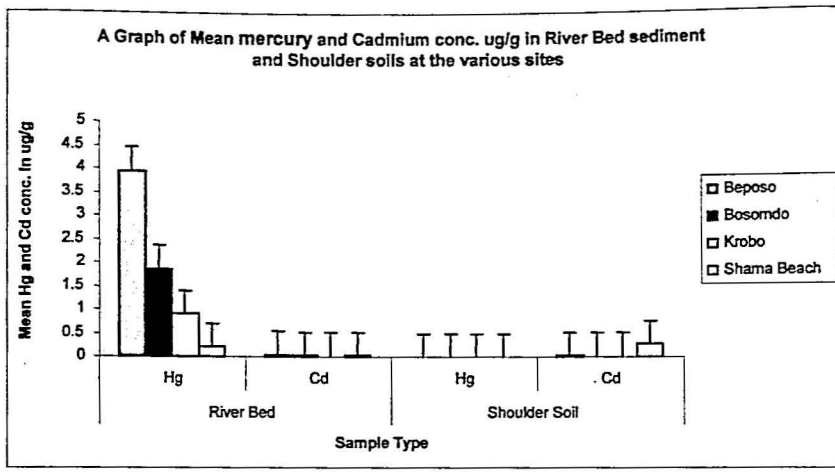


Figure 22

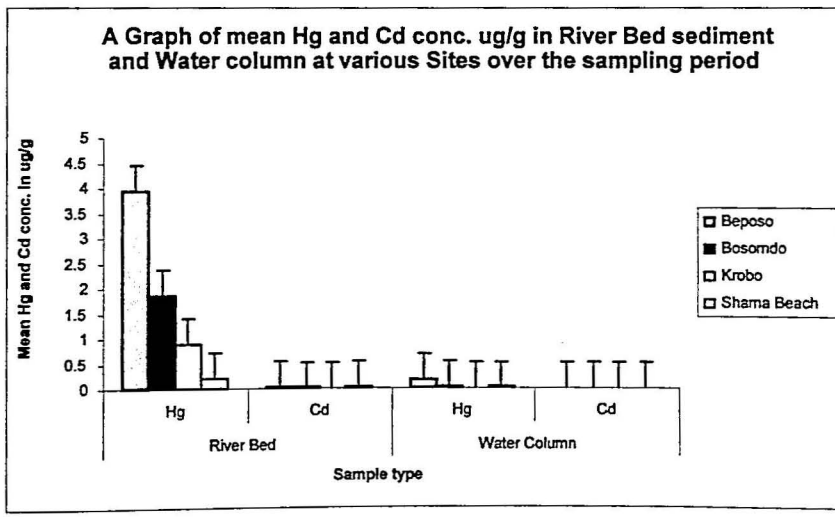


Figure 23

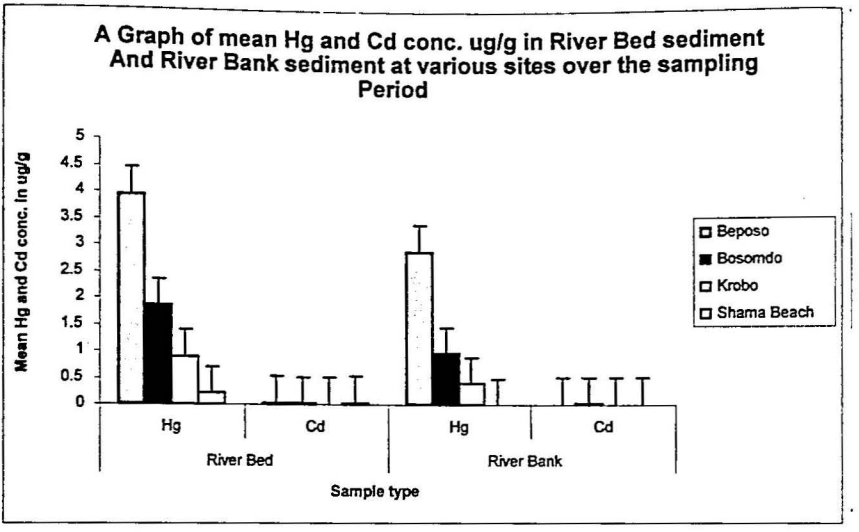


Figure 24

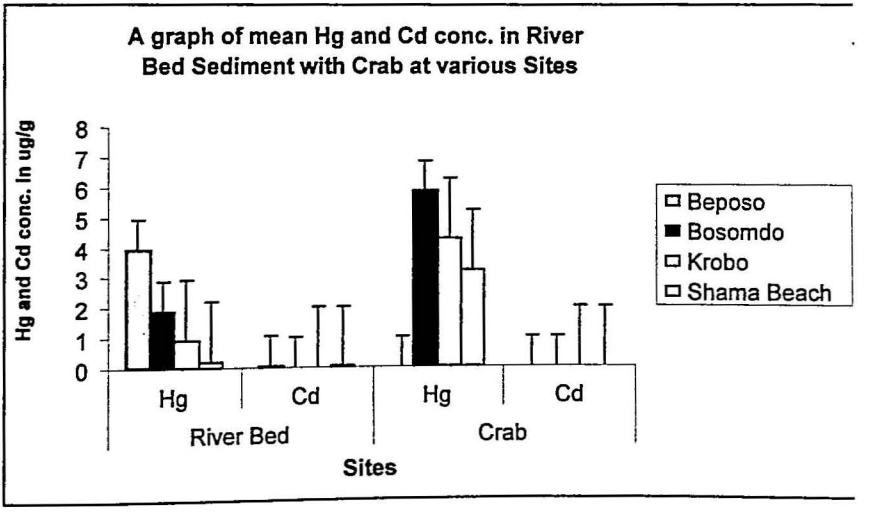


Figure 25

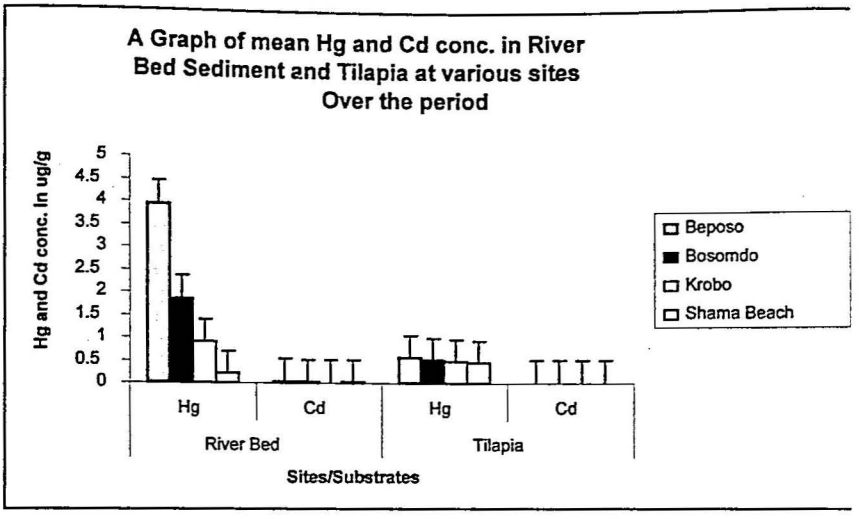
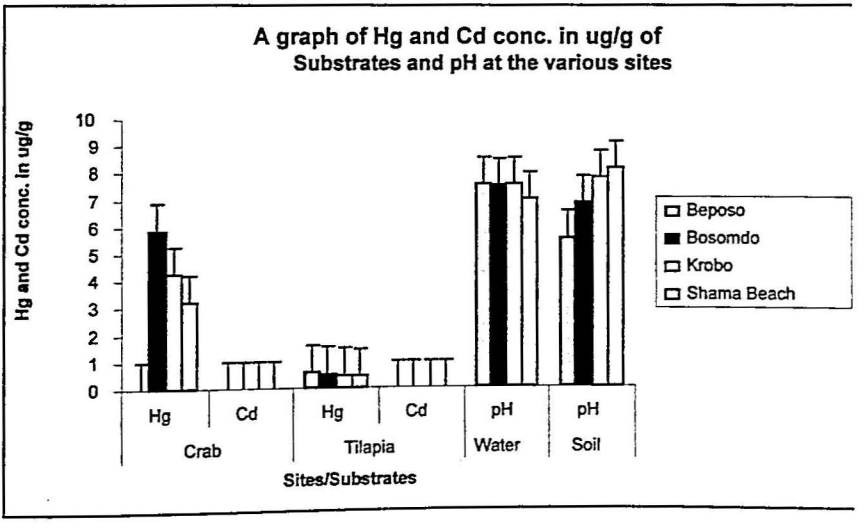


Figure 26



Discussion

Cadmium

The mean cadmium concentrations in the shoulder soils of Beposo, Bosomdo, Krobo, and Shama Beach proved that a significant amount of Cadmium existed. This is probably due to the emissions from vehicular exhaust (Essumang et.al 2005).

Beposo had a high cadmium concentration of 0.026μg/g. The cadmium concentration decreased as one moved from Beposo through to Krobo. About 0.0267μg/g of cadmium was detected at Shama Beach, the highest. This might be due to the contribution of Cadmium from the sea water [about 0.1μg/Litre or less] (EHC, 135:1992).

Table 2 and Figure 2, showed an increase of cadmium concentrations at all the sites. Much of the cadmium which entered the fresh water from industrial sources was rapidly adsorbed by particulate matter where they settled at the bed of rivers (Yamagata et.al 1970). The cadmium concentrations in shoulder soils at Beposo were significantly high over the sampling period. At Beposo, the first sampling gave a cadmium concentration of 0.0503μg/g, with a least of 0.0254μg/g. Subsequently sampling periods, had a decreased concentrations of cadmium with the last being an exception.

Tables 4, 5 and 6, indicated that much of the cadmium concentration settled at the river bed than in shoulder soils. Some of the cadmium salts, such as the sulphides, carbonates or oxides, are particularly insoluble in water. However, these can be converted to water-soluble salts under the influence of

oxygen and acids: the sulphates, nitrates and halogenates are soluble in water (EHC 135:1992). Tables 7, 8, 9, and 10 indicate that cadmium concentrations in water column were much smaller than in river bed sediment and in shoulder soils. This was also evident in Figure 11, 12, 13, and 14.

A review of the mean cadmium concentration in river bank sediments, showed values ranging from 0.008µg/g to a high of 0.0379µg/g at the various sites. Cadmium concentrations values in river bank sediment were quite low compared to concentrations in the bed sediment. An exceptionally higher value of 0.0213µg/g was realised at Bosomdo, a village next to Beposo. This was due to the topography of the site, which always experienced flooding during the sampling period.

The study proved clearly that much of the cadmium settled at the bed of the river rather than the bank as shown in Table 23 and Figure 23.

Mercury

The mean mercury concentrations in the shoulder soils of Beposo, Bosomdo, Krobo and Shama Beach were below the detection level. This is evident in Table 1 and Figure 1. The non-detectable nature of mercury in the shoulder soils may have been caused by the volatile forms of mercury (e.g. metallic mercury and dimethyl mercury) that are expected to evaporate to the atmosphere or solid forms partition to particulate soil or water column and later transported down in the water column to the sediments (Hurley et al. 1991). Samples collected form the riverbed at various sites gave a detectable mean mercury concentration.

Beposo had the highest mercury concentration of 3.95µg/g with a pH of 5.56. Mercury concentrations decrease as one moved from Beposo through to Shama Beach. At Shama Beach 0.21µg/g of mercury concentration was detected which was the least of all. Thus, fresh water and marine sediments are important repositions for inorganic forms of the element, and leaching is a relatively insignificant transport process in soils. The values for the riverbed indicated that most of the mercury deposited on soil is absorbed onto soil sediments and little in water (Krabbenhoft and Babiarz, 1992).

Table 2 and Figure 2; show a significant decrease of mercury concentrations and an increase in pH for Beposo to Shama Beach. This could be explained by the fact that soil sediment mercury levels decreases with distance. In Table 3, the mercury concentrations in shoulder soil were all non-detectable at Beposo over the sampling period. At Beposo, minimum of pH 7.61 was realized in shoulder soils, with 8.37 being the maximum. However mercury concentrations in the riverbed sediments were significantly high. The sampling gave a least mercury concentration of 3.58µg/g and a high of 4.3µg/g in the second sampling. This could be attributed to a high rainfall pattern during that period. Schuster (1991) stated that an adsorption of mercury in soil is decreased with increasing pH and vice versa. This was observed as shown in Figure 3.

At Bosomdo, Krobo and Shama Beach, the mercury concentrations in the riverbed sediments were all significantly higher than in shoulder soils. Over the period of sampling at the sites, there was no mercury in shoulder soils of the

Pra River. This could be explained by the fact that, volatile forms (example, metallic mercury and dimethylmercury) might have evaporated to the atmosphere, whereas solid forms to particulates in the soil or water column and are transported downward in the water column to the bed sediments (Hurley et al. 1991). This relationship is shown in Figures 4, 5 and 6.

A relationship between mercury concentrations in riverbed sediments and the water column sample is shown in Tables 7, 8, 9 and over the sampling period at the various sites (Hurley et al 1991). Mercury concentrations in water samples at Beposo gave a significantly low value of 0.1µg/L and in some cases even non-detected. At least pH of 7.15 was realized over the period with a high pH of 7.98, during the second sampling. At pH of about 7.00 in water column, insignificant levels of mercury concentrations were observed. As pH reduced to about 5.00-6.00, a significant mercury concentration was realised.

In all, water samples had low mercury concentrations at Bosomdo, Krobo and Shama Beach over the sampling period compared to concentration in riverbed sediments. This pattern is shown in Figures 7, 8, 9 and 10.

Mercury concentrations in water samples at Beposo over the period, even though as low as 0.1µg/L were significantly higher than concentrations in shoulder soils. Levels of mercury in water samples at Bosomdo, Krobo and Shama Beach followed almost the same pattern over the sampling period. This significant pattern was confirmed in Figures 11, 12, 13 and 14.

Surface waters may be saturated with volatile elemental mercury, whereas sediments are the primary source of mercury in surface waters (Bloom

and Effler, 1990). The pH range of 7.02 – 7.58 were realized in water samples from the various sites. The mean mercury concentration in water samples at Beposo was 0.0175µg/L. Concentration of mercury in water column samples collected at Bosomdo and Krobo was less than 0.04µg/L, but Shama Beach gave, a slight increase of 0.018µg/L. This slight increase might have been due to soil organic content and contribution from sea, which are variables, related to mercury concentration as determinable in Table 15 Figure 15 (Rada et al 1989).

Levels of mercury in riverbank sediments at Beposo were quite higher than concentration in the bed in Table 16. A high 6.4µg/g of mercury concentration was realised with a pH of 5.02. These high levels might be attributed to mining activities along the Pra River. The Pra River overflows its banks and during dry season dries up leaving metal traces at bank. Also, a good number of people wash and bath upstream. Apart from Beposo, Bosomdo, Krobo and Shama Beach had higher levels of mercury in the riverbed sediment than at the banks. These results are shown in Figures 17, 18 and 19.

At pH of 5.71 to 8.12, the mean mercury concentrations at the various sites were form 0-2.9µg/g. Mercury concentrations at Shama Beach were below detection limit. An estimated value of 2.9µg/g for Beposo was reported, as Bosomdo and Krobo gave values of 0.97µg/g and 0.4µg/g respectively. The reported mean mercury concentrations suggested that, concentrations of mean mercury in riverbank of the various sites reduced as one moved towards the estuary, as in Table 20.

The concentration of mercury in Crab sampled at the various sites had a range of 0-5.86µg/g. Crabs were not found at Beposo, but those of the other sites gave a substantial uptake of mercury and cadmium. Bosomdo, a village close to Beposo had a mean mercury concentration of 5.86µg/g in Crab, a value about 4 times that in the riverbed sediment as showed in Table 24. At pH 8.17, the average mercury concentration of Crab at Shama beach was 3.23µg/g (wet weight). Crabs tend to accumulate so much mercury and cadmium because they are bottom-dwelling species (May and McKinney, 1981). This significant high values could also be attributed to the feeding habit and slow movement of Crabs, which intend makes them accumulate much mercury and cadmium at the bed of the river. The mean concentration declined significantly from 5.86µg/g to 3.23µg/g in Crab over the few months. This decline was presumably due to curtailed production, use, and emissions of mercury and cadmium from the erstwhile Offin Continental Goldfields and 'galemsey' activities along the Pra River (Lowe et al 1985).

In Table 25, Tilapia were analysed as whole-body samples and all concentrations were reported on wet weight basis. A high mercury concentration of 0.57µg/g in Tilapia was observed at Beposo. Least of 0.44µg/g of mercury was reported at Shama Beach, while Bosomdo and Krobo had 0.51µg/g and 0.47µg/g respectively. The mercury and cadmium concentrations in Tilapia were lower than those of in the Crab. This could be as a result of the fast movement and surficial water-dwelling habits of the Tilapia (May and McKinney, 1981). The most common organic form of mercury, methyl

mercury, is soluble, mobile and quickly enters the aquatic food chain. This form of mercury is accumulated to a greater extent in biological tissue than are inorganic forms of mercury (Riisgard and Hansen, 1990). Fishes appear to accumulate methyl mercury from both food sources and the water column. However, (Hall et al 1997) found that food was the predominant source of methylmercury uptake by fish.

The total mercury concentrations in the riverbed sediment and river bank sediment ranged from 0 – 3.95µg/g at the various sites. Table 23, showed clearly, that the mean mercury concentration in the sediment was higher than in soils from the river bank. The results of 3.95µg/g in the bed at Beposo as compared to 2.9µg/g in the shoulder might have been caused by runoff water from the shoulder soil, which eventually settles at the river basin. At Shama Beach, the maximum level of mercury was 0.21µg/g. signifying a slight decrease from a nearby site (Krobo), with 0.9µg/g. A mean mercury concentration at the various sites, except Beposo was non-detectable. This trend confirms that most inert mercury in precipitation is bound to particulate matter, which is relatively immobile when deposited on to the sediments or water (Meile et al. 1991).

In Table 22, a mean mercury concentration in water ranged form 0.0055µg/L to 0.175µg/L. This gave a significant decrease as compared to the concentrations in riverbed sediment. The reasonable small amounts of mercury concentration might have been due to vapour-phase mercury volatilization from the surface waters, as cited by (Schroeder and Fanaki, 1988). However, the

dominant activity controlling the distribution of mercury compounds in the environment appeared to be the sorption of non-volatile forms to the bed sediment and particulate matter, with little resuspension from the bed sediments back into the water column, as cited by Bryan and Langston (1992).

A review of the mean mercury concentration, at riverbank sediments showed values ranging from 0 to 2.9µg/g at the various sites. The highest concentration was found at Beposo, a location upstream. Mercury and cadmium concentration values in the riverbank sediment were quite low compared to concentrations in bed sediment. Shama Beach, which is close to the estuary, had a non-detectable value, with Bosomdo and Krobo giving 0.97µg/g and 0.4µg/g respectively. The study showed clearly that much of the mercury and cadmium concentration settled at the bed of the river rather than at the bank, as shown in Table 23 and Figure 23.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

Conclusion

The mean, minimum and maximum soil / water mercury and cadmium concentrations detected for the four (4) sites, with the 10km geographical distribution; river bed at Beposo showed a maximum of 3.95µg/g of mercury and 0.0379µg/g of cadmium, which are far below the Environmental Protection Agency's permissible limit of 0.134mg/g(134.0µg/g). An insignificantly low level of mercury concentrations could be analyzed in shoulder soils and water samples over the period of the study. Low levels of mercury in water and shoulder soil showed up in peaks but could not be quantified using the Neutron Activation Analyser. High pH levels usually gave non – detectable limits of mercury concentrations.

High levels of concentrations of mercury and cadmium existed in the riverbed sediments compared to that analyzed in the riverbank sediments, water and the shoulder soils. The degree of concentrations of mercury shows that, mercury concentrations decreased significantly and gradually as one moved from Beposo to the Shama Beach through Bosomdo and Krobo. The maximum mercury and cadmium residue reported for the European green Crab and the Blue tilapia. While the maximum mercury residue reported for the Tilapia are

not consistently as high as those for the Crab, the maximum residue in the European Green Crab at three (3) reporting sites, still did not exceed the EPA action level (0.134 mg/g). The Crab has a higher level of mercury and cadmium because it is a bottom –dwelling/feeding and predatory specie.

The mercury and cadmium concentration in fishes can be at least an order of magnitude higher than in commercial fish purchased in a market. Therefore, recreational and subsistence fisher men, including the natives of Beposo, Krobo, Bosomdo and Shama Beach who consume locally caught fish from the Pra River or consume long –lived predatory species, can be exposed to some mercury and cadmium concentrations than individuals who consume similar amounts of commercially marketed fish from a variety of sources (Ebert et al 1996; EPA 1995 K). The exposure to mercury and cadmium will also be higher among people who regularly eat fish and other seafood products, compared to those who occasionally or never eat fish or other seafood products. The increased exposure has been demonstrated by blood mercury levels several times higher in people who regularly eat fish, compared to those who occasionally or never eat fish (Buzin et al, 1989; Capon and Smith 1982; Oskarssan et al 1996; Phelps et al, 1980).

Recommendations

The study hereby observed that the levels of mercury decreased as one moved to the estuary, it is therefore recommended that:

1. an extensive work is done upstream to analyze the mercury and cadmium budgets, since inhabitants of Dunkwa –on-Offin and it

environs are suspected to carry out most of the "Galamsey operations".

2. work be carried out to quantify the various forms of mercury and cadmium (speciation) that exist in the Pra estuary.
3. an extensive work be done on the transport and partitioning of mercury and cadmium in surface waters and soils influenced by the particular form of the compound.
4. a study be done on vaporization of mercury from the various soils controlled by temperature.
5. a study of the contribution of mercury and cadmium levels by the sea into the estuary if any be undertaken
6. apart from the substrates used that is water, fish, crustaceans, sediment and soil, the use of body fluids and hair be used as indicators to confirm mercury and cadmium–contamination levels.

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APPENDIX 1

Values of Concentrations of Mercury and Cadmium from Beposo over sampling period.

SAMPLING I	DATE: 10-05-2006			
TABLE 1:BEPOSO	SOIL	WATER	Soil	Water
SAMPLE	Hg/µg/g	Hg/µg/L	Cd/µ g/g	Cd/ µg/L
A1	0.00146±0.00022		0.036±0.0054	
A2	nd		0.0503±0.007	
A3	nd		0.0254±0.0038	
A4	0.00319±0.00048			
A5	0.00358±0.00054		0.0068±0.0001	
A6	nd			
A7	0.00590±0.00090		0.093±0.014	
A8	nd		0.042±0.0063	
A9	nd			
A10	nd			
A11	nd			
A12	nd			
AW		nd		nd
SAMPLING II	DATE:24-05-2006			
SAMPLE	SOIL	WATER		
2A1	0.0041±0.00060		0.002±0.00032	
2A2	nd		0.0062±0.0003	
2A3	nd		nd	
2A4	0.00610+0.00090			
2A5	0.00430±0.00060		0.013±0.0019	
2A6	nd			
2A7	0.00640±0.00092		0.027±0.004	
2A8	nd		0.0025±0.0006	
2A9	nd			
2A10	nd			
2A11	nd			
2A12	nd			
2AW		0.00060±0.00001		0.6±0.1
SAMPLING III	DATE: 08-06-2006			
SAMPLE	SOIL	WATER		
3A1	0.00380±0.00060		0.002±0.00032	
3A2	nd		0.0021±0.0003	
3A3	nd		nd	
3A4	0.00246±0.0.00037			
3A5	0.003650±0.00055		0.0012±0.0018	

APPENDIX 2

Values of Concentrations of Mercury and Cadmium from Bosomdo over the sampling period.

SAMPLING I		**DATE:10-05-2006**		
TABLE II:BOSOMDO	SOIL	WATER	SOIL	Water
SAMPLE	Hg/µg/g	Hg/µg/L	Cd/µ g/g	Cd/µ g/L
B1	nd		nd	
B2	0.00138±0.00021		0.012±0.002	
B3	0.00126±0.0002		0.012±0.002	
B4	nd		0.0054±0.0003	
B5	0.00110±0.00020		0.02±0.003	
BW		nd		0.0003±0.00005
SAMPLING II		**DATE:24-05-2006**		
SAMPLE	SOIL	WATER		
2B1	nd		nd	
2B2	0.00120±0.0002		0.04±0.006	
2B3	0.00123±0.0002		0.039±0.0059	
2B4	nd		0.0076±0.00012	
2B5	0.00246±0.00037		0.016±0.0025	
2BW		0.000120±0.00002		0.0005±0.00006
SAMPLING III		**DATE:08-06-2006**		
SAMPLE	SOIL	WATER		
3B1	nd		nd	
3B2	nd		nd	
3B3	nd		0.003±0.0001	
3B4	nd		0.0101±0.00015	
3B5	0.00013±0.00002		nd	
3BW		nd		nd
SAMPLING IV		**DATE:29-06-2006**		
SAMPLE	SOIL	WATER		
4B1	nd		0.013±0.002	
4B2	0.00128±0.00002		0.0119±0.0018	
4B3	0.00138±0.00002		0.012±0.002	
4B4	nd		0.0103±0.0015	
4B5	0.00258±0.00039		nd	
4BW		nd		0.0033±0.0005
Definition of sample codes				

B1-Shoulder soil at Bosomdo
B2-Sediment from river bank at Bosomdo
B3-Sediment from river bank at the opposite side
B4-Shoulder soil from opposite side of river
B5-Sediment from river bed
Bw-Water samples
nd-Not Detected

APPENDIX 3

Values of the concentration of Mercury and Cadmium from Krobo over the sampling period.

SAMPLING I		DATE:10-05-2006		
TABLE III:KROBO	SOIL	WATER		
SAMPLE	Hg/µg/g	Hg/µg/L	Cd/µg/g	Cd/µg/L
C1	nd		0.006±0.0009	
C2	0.00104±0.00016		0.0013±0.0019	
C3	0.00112±0.00017		0.018±0.009	
C4	nd		0.018±0.009	
C5	0.00020±0.00005		0.001±0.002	
CW		nd		nd
SAMPLING II		DATE: 24-05-2006		
SAMPLE	SOIL	WATER		
2C1	nd		0.014±0.002	
2C2	nd		0.0042±0.00062	
2C3	nd		0.0018±0.0002	
2C4	nd		0.0018±0.0002	
2C5	0.00213±0.00032		0.001±0.0002	
2CW		0.000022±0.000003		0.002±0.0003
SAMPLING III		DATE: 08-06-2006		
SAMPLE	SOIL	WATER		
3C1	nd		nd	
3C2	nd		0.0021±0.0032	
3C3	nd		0.002±0.0003	
3C4	nd		0.002±0.0003	
3C5	0.00011±0.00002		0.023±0.0035	
3CW		nd		0.0004±0.00006
SAMPLING IV		DATE: 29-06-2006		
SAMPLE	SOIL	WATER		
4C1	nd		0.0082±0.0001	
4C2	0.00104±0.00016		0.012±0.002	

4C3	nd		nd	
4C4	nd		nd	
4C5	0.00120=0.00020		nd	
4CW		nd		
				0.0007=0.0009

Definition of sample codes
C1-Shoulder soil at Krobo
C2-Sediment from river bank
C3-Sediment from opposite side of river bank
C4-Shoulder soil taken opposite side of bank
C5- River bed sediment
Cw- water samples
nd-Not Detected

APPENDIX 4

Values of Concentrations of Mercury and Cadmium from Shama Beach over the sampling period.

SAMPLING I	DATE:10-05-2006			
TABLE IV: SHAMA BEACH	SOIL	WATER	SOIL	WATER
SAMPLE	Hg/µg/g	Hg/µg/L	Cd/µg/g	Cd/µg/L
D1	nd		nd	
D2	nd		nd	
D3	nd		0.0041±0.0006	
D4	nd		0.0041±0.0006	
D5	0.000120±0.00002		0.029±0.0044	
DW		nd		0.0044±0.0001
SAMPLING II	DATE:24-05-2006			
SAMPLE	SOIL	WATER		
2D1	nd		0.009±0.0003	
2D2	nd		nd	
2D3	nd		0.015±0.0023	
2D4	nd		0.015±0.0023	
2D5	0.00051±0.00008		nd	
2DW		0.00007±0.000001		0.0005±0.00008
SAMPLING III	DATE:08-06-2006			
SAMPLE	SOIL	WATER		
3D1	nd		0.042±0.007	
3D2	nd		0.014±0.0022	
3D3	nd		0.021±0.00315	
3D4	nd		0.021±0.00315	
3D5	0.00010±0.00002		nd	
3DW		nd		0.0012±0.0002
SAMPLING IV	DATE:29-06-2006			
SAMPLE	SOIL	WATER		
4D1	nd		0.024±0.003	
4D2	nd		0.002±0.0003	
4D3	nd		0.001±0.0005	
4D4	nd		0.001±0.0005	
4D5	0.00011±0.00002		nd	
4DW		nd		0.002±0.0003

D1-Shoulder soil at Shama Beach
D2-Sediment from the bank of river at Shama Beach(Kedzi)
D3-Sediment from opposite side of river bank

D4-Shoulder soil taken from opposite side of river
D5- River bed sediment at Shama Beach
DW-Water samples
nd-Not detected

APPENDIX 5

Values of Concentrations of Mercury and Cadmium from the Ayensu River over the sampling period as Control.

SAMPLING V	DATE: 03-07-2006			
TABLE V: CONTROL(AYENSU)	SOIL	WATER	SOIL	WATER
SAMPLE	Hg/µg/g	Hg/µg/L	Cd/µg/g	Cd/µg/L
E1	nd		nd	
E2	nd		nd	
E3	nd		nd	
CTW 1		nd		nd
E4	nd		nd	
E5	nd		nd	
E6	nd		nd	
CTW2		nd		nd
E7	nd		nd	
E8	nd		nd	
E9	nd		nd	
CTW3		nd		nd
E10	nd		nd	
E11	nd		nd	
E12	nd		nd	
CTW4		nd		nd

Definition of sample codes

E1-Shoulder soil at Dawukwa

E2-Sediment from river bank

E3-River bed sediment at Dawukwa

CTW 1-Water samples at Dawukwa

E4-Shoulder soil at Okyereko

E5-Sediment from river Bank

E6-River Bed Sediment at Okyereko

CTW2- Water samples at Okyereko

E7-Shoulder Soil at Atakyedo

E8-Sediment from river Bank

E9-River Bed Sediment at Atakyedo

CTW3- Water samples at Atakyedo

E10- Shoulder Soil at Sankor

E11- Sediment from river Bank

E12-River Bed Sediment at Sankor

CTW4- Water samples at Sankor

nd- not detected