UNIVERSITY OF CAPE COAST

STUDIES ON LEVELS OF MERCURY, ZINC AND CADMIUM IN SOME CROPS IN THE MINING COMMUNITIES OF TARKWA, PRESTEA, BANKYIM AND AGONA IN THE SOUTH WESTERN PART OF GHANA

BY

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DECLARATION

CANDIDATE DECLARATION

"I hereby declare that this thesis is as a result of my own original research and that no part of it has been presented for another degree in this university or elsewhere"

Student: ELIZABETH AHADZI

Signature..... Date... 16th Jan 2009

SUPERVISOR'S DECLARATION

We hereby declare that the preparation of this thesis was supervised in accordance with the guidelines on supervision of thesis laid down by the university of Cape Coast.

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DEDICATION

This work is dedicated first to the glory of God for making it possible for me to successfully complete this work. Secondly I dedicate this work to my mother Peace and my siblings Comfort, Vero and Francis.

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ABSTRACT

This research was conducted to determine the levels of mercury, cadmium and zinc in the leaves of *Xanthosoma sagittifolium* (kotombre), *Manihot esculenta* (cassava leaves) and the corm of Colecasia *esculenta* (taro corm) growing in the mining communities of Tarkwa, Prestea, Bankyim and Agona Junction. The Random sampling method was used to collect the samples and the wet digestion method was used to digest the samples for AAS analysis.

A statistical analysis of the results of the research reveals that the concentrations of Hg, Zn and Cd in samples from mining communities were higher than samples from non mining communities and that there is a significant difference in the means at 95% confidence level.

When the results obtained were compared with WHO and EPA permissible values, zinc and cadmium concentrations in the samples were within the normal concentration found in plant leaves and corms. The levels of mercury in the samples were however in the phytotoxic concentration range. There is therefore a cause for concern regarding the levels of Hg, in the selected vegetable crops from the mining district of Tarkwa because heavy metals can bioaccumulate as they move up the food chain.

TABLE OF CONTENTS

LIST OF PLATES

LIST OF FIGURES

LIST OF TABLES

CHAPTER ONE

INTRODUCTION

Whilst support for an increased production and consumption of fresh vegetables is an important goal, citizens have the right to safe food and to be assured that the vegetables available to them are not contaminated beyond acceptable safe limits. Chemical contamination from sources such as industries, vehicles, application of pesticides and mining activities can affect the safety of food.

Heavy metals are one of a range of important types of contaminants that can be found on the surface and in the tissue of fresh vegetables. Prolonged human consumption of unsafe concentrations of heavy metals in foodstuffs may lead to the disruption of numerous biological and biochemical processes in the human body. Heavy metal accumulation give rise to toxic concentrations in the body, while some elements (e.g. Arsenic, Cadmium, Chromium) act as carcinogens others (e.g. Mercury and Lead) are associated with developmental abnormalities in children. These heavy metals contaminate the soil and our water bodies too.

The term heavy metal refers to any metallic element that has a relatively high density and is toxic or poisonous even at low concentrations (Defreitas et al. 1981). They cannot be degraded or destroyed. To some extent they enter the human body via food, drinking water and air. As trace elements some heavy

metals (e.g. Zinc, Copper, and Selenium) are essential to maintain the metabolism of the human body. However, at higher concentrations they can lead to poisoning. They are dangerous because they tend to bioaccumulate. Bioaccumulation means an increase in the concentration of a chemical in a biological organism over time, compared to its concentration in the environment. Compounds accumulate in living things any time they are taken up and stored faster than they are broken down (metabolized) or excreted. Plants also absorb these toxic metals posing serious health problem to man since they are source of food to man. These plants include cassava, maize, plantain, rice cocoyam and many foodstuffs and even cocoa the cash crop of Ghana. Most of these environmental pollutants from the mining activities have harmful effects on man. The effect depends on the accumulated amounts of these pollutants. The uptake of metals from both the atmosphere through leaf surfaces and from the soil through the roots account for the elevated levels of metals in food crops grown in mining areas. Metals find their way into humans either by direct inhalation and absorption via the air or ingesting food and water.

Vegetable crops grown in mining areas are often contaminated with heavy metals (i.e. Mercury, Cadmium, and Zinc) due to the use of mercury and zinc for the extraction of gold. Cadmium is an impurity in zinc. There is therefore significant cause for concern regarding contamination.

Hypothesis

1. The levels of mercury, cadmium and zinc in *Manihot esculenta* leaves (cassava leaves) and *Xanthosoma sagittifolium* leaves (cocoyam leaves or

kotombre) grown in mining communities are higher than the normal concentrations found in plant leaves.

2. The levels of mercury, cadmium and zinc in *Colecasia esculenta* corms (taro corms) grown in mining communities are higher than the normal concentrations found in corms.

Objectives of the Study

1. Measure the levels of Mercury (Hg), Cadmium (Cd) and Zinc (Zn), in some vegetables grown in some mining communities in Ghana.
2. Compare the amount of Hg, Cd and Zn in vegetables grown in mining communities with those grown in non-mining communities.
3. Compare the levels of Hg, Cd and Zn in the selected vegetables from the study area with published data.
4. Recommend interventions in terms of policies and programmes that could reduce Hg, Cd and Zn contamination of vegetables grown in mining communities.

Statement of the Problem

Modern gold mining is machine and chemical intensive endeavor in which hundreds of tons of rock are moved and processed for every once of gold to be extracted. These massive operations bring about environmental degradation, including soil erosion, deforestation and chemical contamination of water bodies and food crops.

A large number of small-scale miners employ mercury for processing gold. In November 1990, there was about 40kg of mercury lost per month into the environment from the dredges in Dunkwa Goldfields (Tenkorang, 2000). It is also estimated that 5 tones of mercury is released from small-scale mining operations each year (Hilson, 2001).

Mercury is therefore a major pollutant in the mining environment. Mercury vaporizes easily at room temperature and the lost mercury, which may get into the atmosphere, could fall back in rain or dew or other precipitation to contaminate the soils, vegetation, human beings and livestock.

Though cadmium is not used directly in gold extraction processes, it is an impurity in zinc. There is therefore the likelihood that appreciable levels of cadmium will be released alongside zinc. Toxicity of zinc is attributed to cadmium impurity in zinc. Cadmium is one of the most toxic metals in the environment hence its selection for this work.

In the light of the above points, a concerted effort in research direction needs to be undertaken on vegetables grown in mining communities to assess the levels of contamination.

Justification

Gold is the major mineral produced in Ghana. The exploitation of gold should be done in an environmentally friendly manner so that it could be sustainable. The quest for wealth need not destroy the people, living things and our environment.

This study will help the mining companies, the small-scale miners, the government, farmers and the population around the mines in the following respect:

- It will provide data on the selected heavy metals into the staple vegetable crops selected.
- It will help advice policy makers in the agriculture sector on farming practices close to mining areas.
- It will help in the implementation of environmental sustainable policies for the mining areas.

The research will involve:

1. Collection of samples of *Xanthosoma sagittifolium* leaves (Cocoyam leaves also known as kotombre), *Manihot esculenta* leaves (Cassava leaves) and corms of *Colocasia esculenta* (Taro).
2. Pretreatment of samples of *Xanthosoma sagittifolium* leaves, *Manihot esculenta* leaves and corms of *Colocasia esculenta.*
3. Acid digestion of samples of *Xanthosoma sagittifolium* leaves, *Manihot esculenta* leaves and corms of *Colocasia esculenta.* Analysis of digests using flame and flameless Atomic Absorption Spectrophotometry.

***Manihot esculenta* (Cassava)**

Cassava is a tall semi-woody perennial shrub or tree with big palmate compound leaves. The dark green leaves are a foot or more across and have 5-9 lobes. The petioles are very long, up to 61cm long and they are red as are the stems. The tuberous edible roots are 20-76cm long and 2.5-7.6cm in diameter.

They grow in outward pointing clusters from the base of the stem just below the soil surface. There are several named cultivars available. The primitive "bitter cassavas" contain large amounts of cyanide and need a great deal of processing to make their roots edible. The modern "sweet" cultivars require only peeling and cooking. Vegetative propagation is done by cuttings from the stem.

Plate 1: Cassava plant

Indeed, cassava is the third largest source of carbohydrates for human food in the world, with Africa as its largest center of production (Claude Fauquet 1990). Cassava roots are very rich in starch, and contain significant amounts of calcium (50 mg/100g), phosphorus (40 mg/100g) and vitamin C (25 mg/100g) (FAO, 2003). However, they are poor in protein and other nutrients. In contrast, cassava leaves are a good source of protein.Young tender leaves of cassava can be used as a potherb, containing high levels of protein (8-10% F.W.) (Cock, 1985). Prepared in a similar manner as spinach, the leaves are pounded to fine chaff and cooked as a palaver sauce in Sierra Leone, usually with palm oil but vegetable oil can also be used. The leaves are also used to treat hypertension, headache, and pain (Cock, 1985).

Xanthosoma sagittifolium (New cocoyam)

Plate 2: Cocoyam plant

Xanthosoma sagittifolium is a herbaceous perennial of the tropical rain forest and, although in their natural habitat they grow under the forest canopy, under cultivation they are usually sown with full exposure to sunlight. They require well-drained soils and do not tolerate the permanent presence of water. It has a corm or main underground stem in the form of a rhizome from which swollen secondary shoots, or corms, sprout. Several large leaves also sprout from the main stem, which are sagittate and erect with long, ribbed petioles. The leaves are evergreen. The growth cycle lasts from nine to 11 months, during the first six months the corms and leaves develop; in the last four months, the foliage remains stable and, when it begins to dry, the plants are ready for the corms to be harvested (Abruña, 1997).

The edible parts of cocoyam are the subterranean tuberous corms which, contain between 15% and 39% of carbohydrates, 2.5% to 9.4% of protein and 70% to 77% of water (Tandehnjie, 1990). Leaf proteins are significantly higher than tuber proteins and ranged from 11.5% to 25.6% crude protein (Tandehnjie, 1990). Younger leaves have even higher protein content than the older ones (Tandehnjie, 1990). The corms have nutritional values comparable to potato and are probably easier to digest. A secondary use is the consumption of the young leaves which have nutritional values similar to spinach. Cocoyam leaves are preferred to taro leaves because they taste better.

Cocoyam has been displacing taro because of its better yield and because it can replace yams for preparing fufu, a very popular food in tropical Africa. The cocoyam has traditionally been a subsistence crop and any produce which is not consumed by producers' families goes to the market.

***Colocasia esculenta* (Taro)**

Taro (*Colocasia esculenta* (Linn) Schott), a member of the Araceae family, is an ancient crop grown throughout the humid tropics for its edible corms and leaves, as well as for its traditional uses. Taro root is a starchy tuber vegetable that looks like, and can be used similar to, a potato. Taro roots can be used as an alternative to potatoes. It does, however, have a hairy outer coating on its surface that is similar to the coating on a coconut. Today the plant is widely used throughout the world, in Africa, Asia, and South America. It is commonly grown amongst small scale farmers who operate within the subsistence economy in Ghana.

Plate 3: Taro plant

Plate 4: Taro corms

Taro is a herbaceous plant, often with large leaves and bearing one or more underground stems or corms. It is a succulent, glabrous, perennial herb. The aboveground portion of the plant is composed of large leaf laminae on long erect petioles. The laminae are 25 to 85 cm long and 20 to 60 cm wide. Their shape is entire and ovate to sagittate with an acuminate apex and rounded basal lobes. Taro possesses enlarged, starchy, underground stems which are properly designated corms. These have been found to be highly variable with respect to hydration, size, color, and chemistry (Wang, 1999).

Taro contains greater amounts of vitamin B-complex than whole milk (Miller, 2001). The cooked leaves have the same nutritional value of spinach (Miller, 2001). The Corm can be fried, boiled or rosted. The broad leaves can be used as green vegetables for preparing stew and soup; the leaves can also be boiled and mashed and use as source to eat boiled cocoyam, cassava or plantain.

A whole corm can be cut into sections for planting. The section or corm should not be too big or too small. Most taro varieties mature in about 8 months from planting (Stephens, 1994). Harvesting is done by shaking up the plant and uprooting it and bringing out the corms while those corms that remain in the soil are dug out.

How plants and humans take in trace metals

The uptake of metals from both the atmosphere through leaf surfaces and from the soil through, the roots may account for the elevated level of metals in food crops grown in mining areas. Some metals (e.g. zinc, selenium, cobalt, and copper) can act as nutrients and are essential for health, while others (e.g. mercury, cadmium, lead) have no known beneficial health effects. All may be harmful if excessive amounts are consumed.

The major source of human exposure to trace metals (as well as heavy metals) from the environment is from food (Goyer, 1991). Metals can also find their way into humans either by direct absorption via air or drinking water. Metals and other elements are present in foods either naturally, or as a result of human activities that is anthropogenic (e.g. agricultural practices, industrial emissions, mining, car exhausts).

A Brief History of Gold Mining in Ghana

Ghana has been endowed with diverse mineral resources such as gold, diamond and to a lesser extent bauxite and manganese dioxide. Mining had been

the single industry attracting overseas investors to Ghana. Gold occurs in various regions of Ghana. Some of the metal occurs as the metal in alluvial sand, others as oxides, pyrites and arsenopyrites. It was the metal in the alluvial sand washed down to the coast by rivers, which gave the former name Gold Coast.

Gold, the major mineral produced in Ghana, has been mined and exported for over hundreds of years long before the arrival of the Europeans. Gold was being picked from the sediments deposited along the banks and on the beds of rivers. Gold is believed to be the first commodity to be traded in at the beginning of the European contact. History records that in 1471, the Portuguese exchanged their items for gold at the estuary of the Pra River (Hug, 1959).

Dating back from 1880, both alluvial deposits and hard rocks mining started in Tarkwa (Hug, 1959). This modern mining was extended to Obuasi, where the gold deposits were richer than those in Tarkwa deposits in 1898. In the middle of the nineteenth century, gold mining had taken industrial scale dimension to become the major source of foreign exchange earner for Ghana. Between 1985 and 1999, foreign exchange earning from the minerals recorded about 700 per cent increase (Hug, 1959).

Ghana had fourteen gold mines, one bauxite mine, one manganese dioxide and one diamond mine as at April 2000. Out of these, only few are underground mines. In addition to these, there are a large number of small-scale miners who are mostly engaged in gold and diamond mining (Laing, 1994).

The Case of Tarkwa, Prestea and Surrounding Villages

Until about a century ago when the first gold mining concession was granted to the Frenchman, Mr. Bonnat, in the Tarkwa area all gold mining activities were carried out by the indigenous people by means of laborious and inefficient digging methods. Because they lack the mechanical equipment to pump out water when the water table was reached, most indigenous mining efforts were, of necessity, restricted to gold deposits which were discovered above the water table.

It is quite obvious that, our ancestors were merely scratching the surface of potentially rich ore bodies. This type of mining is not capital intensive and can be easily undertaken by indigenous entrepreneurs. All that is required to improve upon this type of mining are portable generators, water pumps and other digging equipments.

The Prestea Gold Belt, stretching for more than 30 miles from the Opon River north-west of Insu to the Fura River south-west of Prestea, is of importance owing to the great strength and persistence of the ore channels and Iodes and the payable content of gold in many of the ores.

Gold digging has been an indigenous activity in the Prestea area long before the first European Company started operation there in 1887, when the company known as Gie Appanto Gold Mining Company was incorporated to undertake the task. Prior to the incorporation of the Gie Appanto Company, another company (the Essaman Gold Mining Company) was already operating in the area. In 1990, the two companies change their names respectively to Appanto

Mines and Prestea Mines Limited. In 1925, a capital reconstruction of the two companies resulted in the emergence of the Ariston Gold Mines. This company continued its operations until 1961 when it was absorbed into the State Gold Mining Corporation group (Anin, 1989).

The Study Area

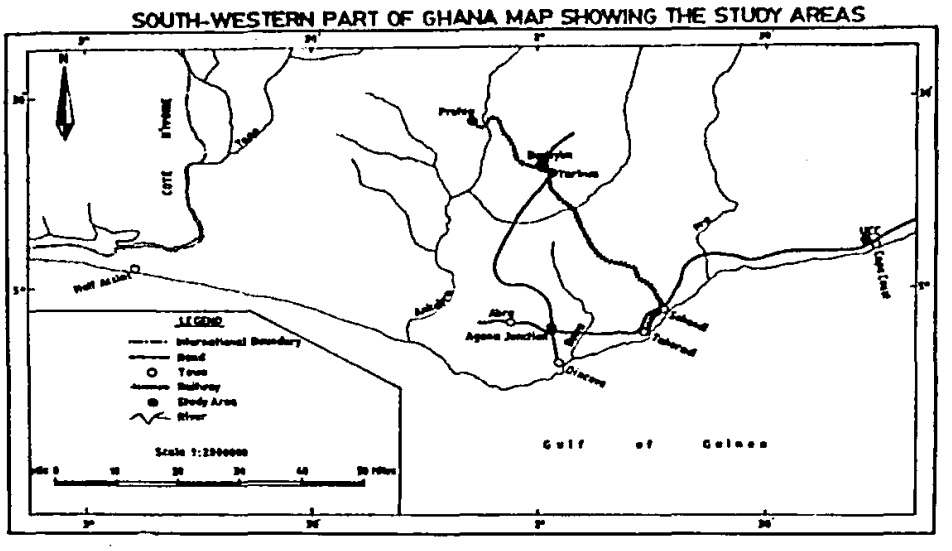

Fig 1: A map of the south-western part of Ghana showing the study area.

The area under study is Agona, Tarkwa, Bankyim and Prestea in the Wassa West District of the Western Region of Ghana. The area has over a century of gold mining history and has the largest concentration of gold mines in a single district on the continent of Africa with virtually all the mines doing surface mining. Tarkwa is in fact the district capital. The district is defined by latitudes

4°N and 5° 40"N and longitudes 1° 45'W and 2° 10'W. It has a total land area of 92354km^2.

Climate: The area falls within the equatorial climate zone. Rainfall peaks during two periods, namely middle of April-July and October-November with a mean annual rainfall of 1933mm. Relative humidity for the area ranges from 70%-90%. Mean monthly temperature ranges from 24°C to30°C.

The high, frequent and prolonged raining season has two effects on the environment. It contributes to the stagnant water in the mine craters resulting from mined out pits and therefore promotes the spread of mosquitoes. Where sulphide ores are being mined, it promotes acid mine drainage, which is a serious environmental problem. However, the prolonged raining season also has an attenuating effect on total dust in the atmosphere, which is positive.

Vegetation: The vegetation of the area consists of tropical rain forest with rich undergrowth of climbers. In primary forest areas, trees reach heights of about 15-45 meters. But these are at the summit of hills where mining has not yet reached.

Demographics: The population of the Wassa West District is put at 260,000 (1994 estimates) with an estimated annual growth rate of 3.0%. It is believed that 70 percent of this figure resides in the Tarkwa mining region where the population growth is said to be above the national 3.1%. This is due mainly to migration of people to the area in search of jobs in the mining sector. Although the Wassa people are natives of the area, the ethnic mix is highly varied due to the mining activities. The growing influx of people into the area in search of jobs and

the drift of unemployed youth from other regions in the country to the area for galamsey mining are major contributory factors to the growing population. Male-Female ratio for the area is estimated at 1: 9; children between the ages of 0-14 years constitute 24.8% of the population, compared to a national average of 45%. Old people (65 years and above) constitute 4.6% of the population while the working group 15-64 years account for 70% of the population. This unusual population structure is due to the general high rate of migration of labour to the centre in search of jobs in the mining sector particularly the males.

Agona Junction: Agona Junction has series of plantation along the road leading to Tikobo. There is a first class road connecting the area to Tarkwa. Apart from the oil palm plantation the indigenous people are peasant farmers. The area has a history of gold mining between 1878 and 1920 by European companies. Unfortunately majority of the old workings have collapsed or are covered with water and little can now be seen of the reefs that were worked.

Tarkwa: Tarkwa is the administrative capital of the Wassa West District. It is located north of Takoradi, the capital city of the Western Region. Tarkwa Township has a cluster of goldfields which are Ghana Australia Goldfields, Teberebie, Iduapriem Goldfields and Tarkwa Goldfields. Mining activities around Tarkwa dates back to the late 19th century when several small mining companies operated in an area known as Abontiakoon Concession (Gold Fields Gh., 2002). The township is generally within mountain ranges covered by thick forest. The soil of highlands of Tarkwa is clayey rocky while the lower ones are clayey

sandy. The study area is predominantly cultivated into food crops and in a few cash crops. Tarkwa is linked to Kumasi and Takoradi by road and rails.

Bankyim: Bankyim is a small village near Tarkwa the soil type is like that of Tarkwa but the vegetation is greener than that of Tarkwa. There are no mines in the area except that there is a major road through the village leading to Anglogold.

Prestea: Prestea is purely a mining town. The town has two main mining companies; they are Barnex (Prestea) Ltd. and Sankorfa Gold Ltd. It has a population of about 16300 but provides very few urban services.

Vegetable production in the study area

Vegetable farming in mining communities is mainly conducted by farmers with low socio-economic status cultivating small or marginal landholdings. Smallholder farmers have preference for growing cocoyam, cassava, taro and plantain and all family members that is women, children and men contribute. This type of vegetable cultivation hence supports livelihoods primarily through food provision, income generation and employment.

Vegetable crops are often grown in polluted and degraded environmental conditions in the mining communities and are subject to further pollution from vehicles and effluent from the mining industries. The people have little choice but to farm in polluted areas and have limited access to advice and support. There is therefore significant cause for concern regarding contamination.

Table 1: Mining Companies Operating in the Study Area

Company	Location	Start	Type of Mining Mining/Processing Method
Gold Fields (Gh) Ltd	Tarkwa	1993	UG/Open cast/heap leach
Teberebie Goldfields	Teberebie	1990	Open cast/heap leach
Bogoso Gold Limited	Tarkwa	1990	Open cast/CIL
Ghana Australian.	Iduapriem	1992	Open cast/CIL/heap leach
Barnex Prestea Ltd	Prestea	1997	UG/Open pit/CIL
Sankofa Gold Ltd.	Prestea	1995	Tailings Treatment/CIL
Abosso Goldfields	Abosso	1997	Open pit/CIL
Satellite Goldfields Ltd.		1999	Open pit/CIL

The Cost of Mining on the Environment and Livelihood

Mining is of fundamental importance to the economies of a number of countries including Ghana. The industry is however associated with serious environmental and health impacts. Very often, the people most impacted by mining have received few tangible benefits. The mining industry has been a focus of criticisms.

According to the International Labour Organization (ILO), mining is one of the world's most hazardous sectors, and is associated with about 15,000 deaths each year (ILO, 1997). In South Africa for instance, each ton of gold mined cost 1 life and 12 serious injuries (ILO, 1997).

Ghana needs to exploit its mineral wealth in an environmentally friendly manner so as to meet her commitments for national development. However the quest for wealth should not lead us to destroy our environment, through problems such as chemical pollution, soil degradation and pollution of our soils, water

bodies and the air, and deforestation and the destruction of human life. Life, Land, Clean Water and Air are more precious than Gold. All people depend on nature for life. The right to life is a guaranteed human right. It is therefore our responsibility to protect all of nature for present and future generations. Large scale gold mining violently uproots and destroys the spiritual, cultural, political, social and economic life of people as well as entire ecosystems. Historic and current destruction created by gold mining is greater than any value generated.

Currently, Bogoso Gold Limited (BGL) is carrying out active mining in Prestea town and has almost covered Prestea Government Hospital with mine waste. The company's activities are less than 30 meters from the Prestea Government Hospital and shocks from blasting traumatise patients. The force of blasting has damaged houses, many television sets, DVDs Video decks and other appliances of people in Prestea. The town has no filling station because BGL has mined the Shell filling station-the only one in Prestea and drivers in Prestea travel to Bogoso for fuel. Installations like the power sub-station are so close to the mine that communities are afraid that they would loose the station. When community people decided to embark on a peaceful demonstration in June 13th 2005, security forces shot seven demonstrators.

Environmental Effects of Mining

The main problems associated with surface mining in Ghana include devastation of our tropical rain forests, soil degradation, water and air quality changes. Gold processing also has deleterious environmental effects. The use of

Mercury, Cyanide and Zinc as the main chemicals for processing eventually leads to the pollution of rivers and water bodies. Underground mining has, on its part, given rise to problems with change in water quality change and potential for subsidence, especially if underground mine water is pumped to the surface. The Ghana government is aware that poor mining practices can cause excessive environmental degradation and contamination of our food crops and even livestock.

A study conducted by Friends of the Earth-Ghana at Obuasi and its surrounding settlements in 1996, showed that the Kwabrafo River at Obuasi in the Ashanti region, had 38 times more arsenic than World Health Organization (WHO) permissible levels whilst the Jimi river at Akrofrom also in the Ashanti region has 36 times more arsenic. Small scale mining operations also contribute significantly to the pollution of water bodies in the mining communities. Since most of the rivers in such communities are the source of drinking water for the people, failure by mining companies to provide alternative sources means a burden on women and children who provide water to the household in rural communities.

Due to the excessive environmental degradation caused by mining practices the Ghana government has made a concerted effort by setting up an Environmental Action Plan (EAP) (Laing, 1994).

The large-scale mining companies are required by EPA laws to manage the environment. Although the government has regularized the operations of small-scale miners in 1989, until recently they were not under the EPA laws. A

large proportion of these small-scale miners has very little knowledge about environmental hazard and hence causes most of the environmental damage.

Health Effects Associated with Mining

The health and safety hazards to miners are numerous and may include accidents (rock falls, mine fires, haulage accidents, shaft accidents etc), gas poisoning, high temperature and humidity effects and various occupational diseases (Laing, 1994). The air pollution in mining areas resulting from the emission of substances such as nitrogen oxides, sulphur dioxides and other atmospheric particles can cause Upper respiratory tract Infections (URTIs0) in people especially, women and children. The noise and the vibration resulting from the blasting of ore in mining operation is a serious problem for nearby residents.

There have been observed incidents in Tarkwa where nursing mothers have had to carry their sleeping babies at their backs whenever rocks are blasted by the mining companies to avoid sound vibrations and being hit by rock splinters. Within the small -scale mining sub-sector, several pits created by miners are left uncovered after use. These pits pose a danger in that, rain water accumulates in them and becomes breeding ground for disease vectors such as mosquitoes which cause malaria. Small -scale miners stand the risk of being trapped to death in the pits when they cave in. In July 1998, a chief fell into one of such pits at Tarkwa and died instantly.

In a study undertaken on small scale mining operations at Tarkwa and its surroundings in 1998, women with babies at their backs were found pounding

gold-bearing rocks without any protective clothing (Agyapong,1998). The high silica -content rocks however generate a lot of dust in the process and prolonged exposure to this dust according to medical experts can cause silicosis and silico-tuberculosis.

Poisoning from Chemical

Mercury is used for extraction of gold by amalgamation by both large-scale and small-scale miners. Small-scale miners with their little knowledge of dangers associated with the use of mercury, employ it for the extraction of gold. They evaporate off the mercury and have no good retorting facilities. Mercury has high vapor pressure and vaporizes easily into the atmosphere. The mercury may condense and become attached to particulate matter in the atmosphere. The condensed mercury may fall back to earth in rainfall or some other process. Zinc dust is used, instead of mercury, to precipitate the gold by some large-scale companies. Zinc is a major metal pollutant with cadmium as impurity. Cadmium is one of the most toxic metals in the environment. Mercury and cadmium are toxic. Mercury has been responsible for a number of poisoning calamities in a number of countries; Minamata in Jintus Valley in Japan in 1952 and 1960; and in Northern Iran in 1972, and still kills. (Miessler et al, 1991). Cadmium is an impurity in zinc. Cadmium is toxic at very low concentrations. It has been responsible for the "ouch-ouch" or "itai-itai" diseases in Japan (Alloway and Ayres, 1993). Cadmium causes hypertension and is antagonistic metabolically to zinc. Zinc is non-toxic and required for metabolism, reproduction and healing of

wounds. Toxic effects of large amounts of zinc are due to impurities of cadmium in zinc.

A Study conducted in Tarkwa and its surroundings revealed that mercury poisoning of small scale miners is a serious health hazard (Agyapong, 1998). This is a result of indiscriminate use as well as improper application of mercury in the processing of gold by small scale miners. Nursing mothers as well as their children stand a great risk of poisoning. According to medical experts, mercury poisoning can lead to birth defects in women when it enters the placenta and may lead to death.

It will be of interest to measure the levels of these metals in food crops grown in the environs of these gold mines since these metals can bioaccumulate as they move up the food chain.

Gold Production in Ghana

Gold production involves the mining of gold ore from the earth and the treatment of the ore to extract gold. Both small (traditional) and large-scale gold mining activities are carried out in Ghana.

Small Scale Mining (Galamsey)

'Galamsey' is the name given to the activity of non-professional small-scale mining in Ghana. 'Galamsey' operations are normally carried out in gold and diamond mining areas.

In gold mining areas, the gold bearing ores are dug from the ground or sometimes the tailing of the gold treatment plants and are washed several times with clean water in a bucket or a pan to remove the slime. Usually an inclined table is set up and the surface is covered with an old jute sack or a piece of woolen carpet or any woolen material that can hold heavy particles.

The gold bearing material in the pan is then poured onto the covered table and further washed to remove the light material. The heavy materials are trapped in the sack covering the table. This operation is continued until the sack is saturated with the concentrate and the initial material is reduced to a very small lump but very rich in gold.

The gold on the piece of sack or cloth is then washed into a pan. The water is decanted and mercury is added to the very small concentrate obtained. The mercury is rubbed hard into the concentrate until an amalgam (Au/Hg) is formed leaving behind the gangue. The gold amalgam is put in a clean white handkerchief and tied. Excess mercury is then squeezed out of the amalgam.

The amalgam which is grey in colour is roasted and in the process the mercury vapourises leaving the gold behind. The gold is collected and sold.

Large Scale Mining

Raw Materials: The main raw materials for the production of gold are the gold bearing ores obtained through underground and surface or open pit mining. The ores are classified as:

- Underground Ore: This consists predominantly of sulphides-pyrites (FeS_2) and arsonopyrites (FeAsS) mined from underground.
- Surface Ore: This consist of;

  Oxides- mainly as iron oxides-haematite (α-$Fe2O_3$) and magnetite (Fe_3O_4).

  Sulphides – mainly as pyrites and arsonopyrites.

  Transition- partially oxidized sulphide forms a transition between the above two types of surface ores.
- Old tailing: these are low-grade ores, which consist of dump tailings from the processing of gold.

In all the three raw materials, the gold is found trapped or entangled in the crystal structure of the sulphides and oxides.

The raw materials used in processing the ores include:

(1) Copper sulphate, ($CuSO_4$) used as a conditioner in flotation.

(2) Potassium or sodium pental (or amyl) xanthate $C_5H_{11}OCS_2^-$ (Kor Na) used as a collector in flotation.

(3) Sodium hydrogen sulphide (NaHS) to modify the pH.

(4) Sodium cyanide (NaCN) used in dissolving gold.

(5) Hydrated lime [$Ca(OH)_2$] provides an alkaline medium when dissolving gold with sodium cyanide.

(6) Borax (anhydrous sodium tetraborate) ($Na_2B_4O_7$), silica (SiO_2) and manganese dioxide, (MnO_2) act as fluxes to reduce melting point during the smelting and to produce slag.

(7) Zinc to precipitate gold.

(8) Activated carbon used to adsorb the dissolved gold.

(9) Limestone used for neutralization in the bio-oxidation process.

(10) Hydrochloric acid used to remove gold from activated carbon.

Underground mining

There are two broad classes of mining methods employed in underground gold mining: (a) those that require some form of support such as pillar. These include open stopping, and cut and fill. (b) Those that require no support. These include long wall mining, sub-level caving mining.

The choice of a mining method depends on characteristics such as the width, dip, strength, the ore body and the host rock, depth of deposit, cost of support, the ore body value (grade), the spatial distribution or the value, safety and cost of mining. The recovery of mineral from sub-surface rock involves the development of physical structures such as shafts and ramps which provides access to the mineralized zone, the liberation of the ore from the gold bearing rock, and the transportation and hoisting of the material to the mine surface. The liberation of the ore involves drilling and blasting the rock, cleaning and mucking and supporting and/or filling.

Surface mining

Surface mining is also known as open-pit mining. This is a mining technique used when the ore is close to the earth's surface. Open-pit mining is best when the soft weathered rocks make mining easy and little or no blasting is

needed. It is economical for ore grades as low as 2g/ton. Open-pit deposits often have a relatively short life span. This mining method may be either surface channel sampling, using a continuous trenching machine, or face sampling. The removal of ore and waste takes place-scrapes, backhoes and trucks may be used to remove the or that results in a great deal of control over ore extraction with ultimate control being a function and the size of a backhoe-scoop (www.personal.psu.edu.,assessed 26/10/2003).

Old tailing

These are low-grade ores, which consist of dump tailings from the processing of gold. Old tailings have an amount of gold in them. The dumps tailing from the gold processing plants are further milled in ball mills. These are then subjected to gravity separations. The overflow is sent to floatation tank for it to be concentrated. The remaining ground tailing are then leached with cyanide in the presence of lime, oxygen and activated carbon (Anamuah-Mensah et al, 1996).

The carbon adsorbs the gold complex. The loaded carbon is then washed, and desorbed and regenerated. From here, the gold is liberated by the electrolytic cell onto the steel wool cathode. The cathode is calcined and smelted to obtain the bullion gold (Anamuah-Mensah et al, 1996).

Gold treatment and recovery process at Tarkwa Gold Fields

The Tarkwa Gold Mine currently utilizes heap leach techniques to recover

gold. The operations consist of two separate heap leach circuits, the Tarkwa plant (North Plant) and the Teberebie Plant (South Plant). The heap leaching technique is a cost effective way of treating low-grade ores. The process involves crushing, agglomeration, leaching, filtration, precipitation, calcining and smelting.

Treatment of gold bearing ore is started with crushing the ore into smaller sizes of about 25mm. The smaller sized gold bearing clayey ore is then agglomerated with cement to enhance leaching with NaCN. Most of the ores treated are clayey in nature and for heap leaching to be successful; the ore must have good permeability to achieve uniform distribution of cyanide leach solution throughout the heap. After agglomeration the ore is stacked into a heap and left to cure for at least 48 hours to form the Calcium Silicate bridging (Anamuah-Mensah et el, 1996)

The ore is stacked on a pad lined with high-density polyethylene material that prevents cyanide solution from leaching into the environment. The heap is continuously sprayed with dilute sodium cyanide solution. The solution percolates through the heap and as it does so the gold in the ore is gradually leached out. This is collected into a pond as concentrated gold solution. The reaction of the heap leaching is:

$$4Au_{(s)} + 8NaCN_{(aq)} + O_{2(g)} + 2H_2O_{(l)} \rightarrow 4NaAu(CN)_{2(aq)} + 4NaOH_{(aq)}$$

The slurry is a solution containing much of the gold and solids solids also containing gold. This slurry is filtered to give a filtrate, which is the gold concentrate. The concentrated solution is then treated with zinc, which displaces the gold from the cyanide complex and precipitates it. The equation is as follows:

$$2NaAu(CN)_{2\,(aq)} + Zn_{(s)} \rightarrow 2Au_{(s)} + Na_2Zn(CN)_{4\,(aq)}$$

The product is calcined and smelted at the gold house to obtain the gold bullions.

CHAPTER TWO

LITERATURE REVIEW

Heavy Metal

The term **heavy metal** refers to any metallic chemical element that has a relatively high density and is toxic or poisonous even at low concentrations. Examples of heavy metals include Mercury (Hg), Cadmium (Cd), Arsenic (As), Chromium (Cr), Thallium (Tl), and Lead (Pb). Heavy metals are natural components of the Earth's crust. They cannot be degraded or destroyed. To a small extent they enter our bodies via food, drinking water and air. As trace elements, some heavy metals (e.g. Copper, Selenium, and Zinc) are essential to maintain the metabolism of the human body. However, at higher concentrations they can lead to poisoning. Heavy metal poisoning could result, for instance, from drinking-water contamination (e.g. lead pipes), high ambient air concentrations near emission sources, or intake via the food chain.

Heavy metals are dangerous because they tend to **bioaccumulate.** Bioaccumulation means an increase in the concentration of a chemical in a biological organism over time, compared to its concentration in the environment (Defreitas et al. 1981). Compounds accumulate in living things any time they are taken up and stored faster than they are broken down (metabolized) or excreted.

Heavy metals can enter a water supply by industrial and consumer waste, or even from acidic rain breaking down soils and releasing heavy metals into streams, lakes, rivers, and groundwater.

Toxic heavy metals have no function in the body and can be highly toxic. If heavy metals enter and accumulate in body tissue faster than the body's detoxification pathways can dispose of them, a gradual buildup of these toxins will occur. High-concentration exposure is not necessary to produce a state of toxicity in the body tissues and, over time, it can reach toxic concentration levels.

Environmental contamination and exposure to heavy metals such as mercury, cadmium and lead is a serious growing problem throughout the world. Human exposure to heavy metals has risen dramatically in the last 50 years as a result of an exponential increase in the use of heavy metals in industrial processes and products. Many occupations involve daily heavy metal exposure; over 50 professions entail exposure to mercury alone. In today's industrial society, there is no escaping exposure to toxic chemicals and metals. In general, heavy metals are systemic toxins with specific *neurotoxic, nephrotoxic, fetotoxic* and *teratogenic* effects. Heavy metals can directly influence behavior by impairing mental and neurological function, influencing neurotransmitter production and utilization, and altering numerous metabolic body processes.

Systems in which toxic metal elements can induce impairment and dysfunction include the blood and cardiovascular, eliminative pathways (colon, liver, kidneys, skin), endocrine (hormonal), energy production pathways, enzymatic, gastrointestinal, immune, nervous (central and peripheral),

reproductive, and urinary. Heavy metals alter pro-oxidant/antioxidant balance and bind to free *sulfhydryl groups*, resulting in inhibition of *glutathione* metabolism, numerous enzymes and hormone function. Nutritionally, heavy metals are directly antagonistic to essential trace elements and compete with nutrient elements for binding sites on transport and storage proteins, *metalloenzymes* and receptors.

Breathing heavy metal particles, even at levels well below those considered nontoxic, can have serious health effects. Virtually all aspects of animal and human immune system function are compromised by the inhalation of heavy metal particulates. In addition, toxic metals can increase allergic reactions, cause genetic mutation, compete with "good" trace metals for biochemical bond sites, and act as antibiotics, killing beneficial bacteria. Much of the damage produced by toxic metals stems from the proliferation of *oxidative free radicals* they cause. Heavy metals can also increase the acidity of the blood. The body draws calcium from the bones to help restore the proper blood pH. Further, toxic metals set up conditions that lead to inflammation in arteries and tissues, causing more calcium to be drawn to the area as a buffer, contributing to hardening of the artery walls with progressive blockage of the arteries and osteoporosis. Even minute levels of toxic elements have negative health consequences, affecting nutritional status, metabolic rate, the integrity of detoxification pathways. The biological half-lives for heavy metals are variably long; the half-life for cadmium in the kidney is decades. Most heavy metals are readily transferred across the placenta, found in breast milk, and are well known to have serious detrimental effects on behavior, intellect and the developing nervous system in children. For

adults, silent symptoms of chronic, low level heavy metals accumulation in tissues can progress from a steady decline in energy, productivity and quality of life to accelerated cardiovascular disease, premature dementia and total debilitation. Unfortunately, the possibility of heavy metals burden is often not considered and patients continue to suffer needlessly.

Chronic symptoms frequently associated with excessive accumulation of heavy metals include fatigue, musculoskeletal pain, neurological disorders, depression, failing memory, and allergic hypersensitivity. Disruption of the metabolism and balance of nutrient elements results in marked aberrations in the metabolism of carbohydrate, protein/amino acids, lipids, neurotransmitters and hormones. Mercury is well known for its direct, destructive effects on neuronal function while cadmium has direct adverse effects on cells in the arterial wall.

Metals and other elements are present in foods either naturally, or as a result of human activities (e.g. agricultural practices, industrial emissions, mining, car exhausts), from contamination during manufacture/processing and storage, or may be added directly. Many cases of heavy metals burden are associated with industrial exposure, but our food, drinking water and environment do not appear to be getting any purer.

In this chapter we shall discuss three of these heavy metals that are normally found in high concentrations in mining communities. These are Mercury, Cadmium and Zinc. Mercury and Zinc are often employed in the extraction of gold. Cadmium is however a contaminant of Zinc.

Mercury

Mercury is a naturally occurring heavy metal. At ambient temperature and pressure, mercury is a silvery-white liquid that readily vaporizes and may stay in the atmosphere for up to a year. When released to the air, mercury is transported and deposited globally. Mercury, along with cadmium and zinc, falls into Group IIb of the Periodic Table. In addition to its elemental state, mercury exists in the mercury (I) and mercury (II) states in which the mercury atom has lost one and two electrons, respectively. The most important ore of mercury, cinnabar (mercuric sulfide), has been mined continuously since 415 BC (Clarkson and Marsh, 1982).

The element mercury (Hg) and its compounds have no known normal metabolic function. Their presence in the cells of living organisms represents contamination from natural and anthropogenic sources; all such contamination must be regarded as undesirable and potentially hazardous (NAS, 1980).

In the period before the industrial revolution, Hg was used extensively in **gold extraction** and in the manufacture of felt hats and mirrors; in the 1800's, it was used in the chloralkali industry, in the manufacture of electrical instruments, and as a medical antiseptic; and since 1900, it has been used in pharmaceuticals, in agricultural fungicides, in the pulp and paper industry as a slimicide, and in the production of plastics (Clarkson and Marsh, 1982). Current world use of mercury is estimated at 10,000 to 15,000 metric tons annually (Boudou and Ribeyre, 1983), of which the United States accounts for about 18% (Clarkson and Marsh, 1982).

Inorganic mercury (metallic mercury and inorganic mercury compounds) enters the air from mining activities as well as other industrial processes and waste sites. Methyl mercury may be formed in water and soil by small organisms called bacteria. Methyl mercury builds up in the tissues of plants and other organisms. Its levels in tissues increase as we go up the food chain. Environmental Protection Agency (EPA) drinking water limit is 2 ppb.

Chronic, low-level mercury (Hg) exposure is a problem that goes well beyond the controversial issue of dental amalgams. Mercury can deliver a one-two punch that can cause significant oxidative damage in the body. Two primary mechanisms for the toxic effects of Hg are: 1) Hg is a pro-oxidant which catalyzes the production of *peroxides* and enhances the subsequent formation of *hydroxy radicals* and *lipid peroxides*, and 2) Hg interferes with the body's capacity to quench highly reactive oxygen species. By virtue of its affinity for free sulfhydryl groups, Hg binds to glutathione (GSH) and can inhibit enzymes involved in GSH metabolism; e.g. Hg forms a tight bond with selenium (Se) thereby "displacing" Se from its critical role as an obligatory constituent of glutathione peroxidase. Hg^{++} can directly bind to 1 or 2 GSH molecules resulting in irreversible removal of this key constituent from our anti-oxidative armory.

Two other major anti-oxidative enzymes that are inhibited by Hg are *superoxide dismutase* (SOD) and *catalase*. By acting as pro-oxidant and inhibiting anti-oxidative processes, Hg could result in excess levels of free radicals, which are particularly disruptive to mitochondrial function and the nervous system. Hormones, the master regulators of metabolism, are also

vulnerable to Hg. Hg inhibits the formation of active thyroid hormone (T3), presumably by binding to and "wasting" Se, which is an obligatory co-factor for the *iodinase* enzyme. *Progesterone* uptake by cells is inhibited when Hg binds to an important free sulfhydryl group on the progesterone receptor. *Testosterone* production and adrenal function may also be compromised with Hg burden. Considering the potential effects of Hg on hormone metabolism, it is not surprising that major *chronic fatigue* is a hallmark symptom of Hg burden. Hg-induced peripheral neuropathy, tremor, depression, irritability and sleep disturbance may be related to adverse effects of Hg on amino acid status. It is well documented that Hg disrupts intracellular transport in neurons by inhibiting *microtubule polymerization* and assembly. To add insult to injury, Hg can also decrease the production of neurotransmitters. For example, *taurine* is a neurotransmitter that is derived from *cysteine*. Cysteine is the rate-limiting amino acid for GSH synthesis and is frequently deficient in Hg burdened patients. Hence, Hg induced depletion of the precursor of taurine (cysteine) might contribute in part to the adverse neurological effects of Hg. Urine amino acid analysis of Hg toxic patients may also reveal deficiencies in *phenylalanine* and *tyrosine* (precursors to *catacholamines* and *thyroxine*), *tryptophan* (precursor to serotonin) and *glutamate* (precursor to GABA). Such deficiencies may be related to the general malabsorption, which is commonly associated with *candidiasis* in Hg burdened patients.

Chemical Properties of Mercury

Mercury, a silver-white metal that is liquid at room temperature and is

highly volatile, can exist in three oxidation states: elemental mercury (Hg^0), mercurous ion (Hg_2^+), and mercuric ion (Hg^{2+}). It can be part of both inorganic and organic compounds (EPA 1980; Clarkson et al. 1984). The mercuric species is the most toxic inorganic chemical form, but all three forms of inorganic Hg may have a common molecular mechanism of damage in which Hg^{2+} is the toxic species (Clarkson and Marsh, 1982).

When pure, mercury does not tarnish on exposure to air at ordinary temperature but when heated to near the boiling point slowly oxidizes to HgO. Mercury forms alloys with most metals except iron and combines with sulfur at ordinary temperature. Insoluble in and not attacked by H_2O; soluble in dilute HNO_3; not attacked by HCl or cold H_2SO_4; converted by heating with concentrated H_2SO_4 into mercurous or mercuric sulfate, depending on the excess of the acid and time of heating.

Mercury salts when heated with Na_2CO_3 yield metallic Hg and are reduced to metal by H_2O_2 in presence of an alkali hydroxide. Cu, Fe, Zn and many other metals form precipitate with metallic Hg from neutral or slightly acid solutions of mercury salts. Soluble ionized Mercuric salts give a yellow precipitate of HgO with NaOH and a red precipitate of HgI_2 with alkali iodide. Mercurous salts give a black precipitate with alkali hydroxides and a white of precipitate calomel with HCl or soluble chlorides. They are slowly decomposed by sunlight.

Sources of Mercury

The major source of mercury is the natural degassing of the earth's crust

and amounts to between 25 000 and 125 000 tones per year (Wood, 1975). Anthropogenic sources are probably less than natural sources. World production of mercury by mining and smelting was estimated at 10 000 tones per year in 1973 and has been increasing at an annual rate of about 2 % (Wood, 1975). The chloralkali, electrical equipment, and paint industries are the largest consumers of mercury, accounting for about 55% of the total consumption.

Natural Sources of Mercury

Mercury from natural sources enters the biosphere directly as a gas, in lava (from terrestrial and oceanic volcanic activity), in solution, or in particulate form; cinnabar (HgS), for example, is a common mineral in hot spring deposits and a major natural source of mercury (Das et al. 1982). The global cycle of Hg involves degassing of the element from the Earth's crust and evaporation from natural bodies of water, atmospheric transport (mainly in the form of Hg vapor), and deposition of Hg back onto land and water. Oceanic effluxes of Hg are tied to equatorial upwelling and phytoplankton activity and may significantly affect the global cycling of this metal. If volatilization of Hg is proportional to primary production in the world's oceans, oceanic phytoplankton activity represents about 36% of the yearly Hg flow to the atmosphere, or about 2,400 tons per year (Kim and Fitzgerald, 1986). Mercury finds its way into sediments, particularly oceanic sediments, where the retention time can be lengthy, and where it may continue to contaminate aquatic organisms (Lindsay and Dimmick, 1983). Estimates of the quantities of Hg entering the atmosphere from degassing of the surface of the planet vary widely, but a commonly quoted figure is 30,000 tons annually

(Clarkson et al. 1984). In aquatic ecosystems, removal of the source of anthropogenic Hg results in a slow decrease in the Hg content of sediments and biota (NAS, 1978). The rate of loss depends, in part, on the initial degree of contamination, the chemical form of Hg, physical and chemical conditions of the system, and the hydraulic turnover time (NAS, 1978).

Anthropogenic Sources of Mercury

As a direct result of human activities, mercury levels in river sediments have increased fourfold since pre-cultural times, and twofold to fivefold in sediment cores from lakes and estuaries (Das et al. 1982). During the past 100 years, it has been estimated that more than 500,000 metric tons of Hg entered the atmosphere, hydrosphere, and surface soils, with eventual deposition in subsurface soils and sediments Das et al. (1982). Several activities that contribute significantly to the global input of Hg include the combustion of fossil fuels; **mining** and **reprocessing of gold**, copper, and lead; operation of chloralkali plants; and disposal of batteries and fluorescent lamps NAS 1978, Das et al. (1982). The atmosphere plays an important role in the mobilization of Hg, 25% to 30% of the total atmospheric Hg burden is of anthropogenic origin (NAS, 1978). The major use of mercury has been as a cathode in the electrolytic preparation of chlorine and caustic soda (Birge et al, 1979). In 1968 this use accounted for about 33% of the total U.S. demand for Hg (EPA, 1980). Mercury, however, is no longer registered for use in antifouling paints, or for the control of fungal diseases of bulbs EPA (1980).

Cycling

There are two cycles involved in the environmental transport and distribution of mercury. One is global in scope and involves the atmospheric circulation of elemental mercury vapor from sources on land into the oceans. However, the mercury content of the oceans are so large, at least 70 million tons that the yearly increases in concentration due to deposition from the global cycle are not detectable (Ferrara, et al. 1982).The other cycle is local in scope and depends upon the methylation of inorganic mercury mainly from anthropogenic sources. Many steps in this cycle are still poorly understood, but it is believed to involve the atmospheric circulation of di-methyl mercury formed by bacterial action.

Uses of Mercury

The chloralkali, electrical equipment, and paint industries are the largest consumers of mercury, accounting for about 55% of the total consumption. Mercury has a wide variety of other use in industry, agriculture, military applications, medicine, and dentistry.

Agriculture: Methyl mercury is used in Fungicides and pesticides to treat grains and seeds worldwide.

Cosmetics: Mercury is added to decrease bacterial growth in some cosmetics.

Dental fillings: Mercury is widely used, though many dentists no longer employ the silver-mercury amalgam, as they feel that it leads to a variety of problems. The American Dental Association, however, still claims that there is no

proven mercury toxicity due to dental amalgams.

Medicines: Organic mercurial diuretics have been the most common, though these are less used these days. Mercury-containing cathartics, anthelminetics, and teething powders were also employed in the past.

Industry: Mercury is used in the manufacture of mirrors, latex paints, fabric softeners, felt, floor waxes and polishes, sewage sludge, laxatives containing calomel, cinnabar jewelry, tattoo dyes, **and extracting gold** and silver from ores.

Scientific equipments: Mercury is used in thermometers because its coefficient of expansion is nearly constant; the change in volume for each degree of rise or fall in temperature is the same. It is also used in other types of scientific apparatus, such as barometers, thermostats, hydrometers, pyrometers; in mercury arc lamps producing ultraviolet rays, fluorescent lamps, mercury vapor lamps, and electric rectifiers.

Chemistry: In the manufacture all mercury salts, as catalyst in oxidation of organic compounds; making amalgams, in determining Nitrogen by Kjeldahl method, for Millon's reagent; as cathode in electrolysis, electro analysis, and many other uses.

The Chemical Compounds of Mercury

The chemical compounds of mercury (II) are much more numerous than those of mercury (I). In addition to simple salts, such as chloride, nitrate and sulfate, mercury (II) forms an important class of organometllic compounds. These are characterized by the attachment of mercury to either one or two carbon atoms

to form compounds of the type RHgX and RHgR' where R and R' represent the organic moiety. The most numerous are those of the type RHgX. X may be one of a variety of anions.

The carbon-mercury bond is chemically stable. It is not split in water or by weak acids or bases. The stability is not due to the high strength of the carbon-mercury bond but to the very low affinity of mercury for oxygen.

The organic moiety, R, takes a variety of forms, some of the most common being the alkyl, the phenyl, and methoxy-ethyl radicals. If the anion X is nitrate or sulfate, the compound tends to be "salt-like" having appreciable solubility in water; however, the chlorides are covalent, non-polar compounds that are more soluble in organic solvents than in water.

From the toxicological standpoint, the most important of these organometallic compounds is the subclass of short-chain alkyl mercurials in which mercury is attached to the carbon atom of a methyl, ethyl, or propyl group.

Chemical Speciation of Mercury

Chemical speciation is probably the most important variable influencing eco-toxicology of Hg, but Hg speciation is difficult, especially in natural environments (Boudou and Ribeyre, 1983). Mercury compounds in an aqueous solution are chemically complex. Depending on pH, alkalinity, redox, and other variables, a wide variety of chemical species are liable to be formed, having different electrical charges and solubility. For example, $HgCl_2$ in solution can speciate into $Hg(OH)_2$, Hg^{2+}, $HgCl^+$, $Hg(OH)^-$, $HgCl_3^-$, and $HgCl_4^{2-}$; anionic forms predominate in saline environments (Boudou and Ribeyre, 1983).

The following speciation among mercury compounds has been proposed by Lindquist et al. 1984, where V stands for volatile, R for water-soluble or particle-borne reactive species, and NR for non-reactive species (Hg° is elemental mercury):

V: Hg°, $(CH_3)_2Hg$

R: Hg^{2+}, HgX_2, HgX_3^-, and HgX_4^{2-},

$X = OH^-$, Cl^- and Br^-.

HgO on aerosol particles.

Hg^{2+} complexes with organic acids.

NR: CH_3Hg^+, CH_3HgCl, CH_3HgOH and other organomercuric compounds, $Hg(CN)_2$. HgS and Hg^{2+} bound to sulfur in fragments of humic matter.

The main volatile form in air is elemental mercury but di-methyl mercury may also occur (Slemr et al., 1951).

Uncharged complexes, such as $HgCl_2$, CH_3HgOH etc., occur in the gaseous phase, but are also relatively stable in fresh water (snow and rain as well as standing or flowing water). $HgCl_4^{2-}$ is the dominant form in sea water.

Mercury and Health

The health hazards of mercury and its compounds have been recognized for quite some time. In the early part of the 19th century, hat makers used a solution of mercury salts to soften animal hairs in the production of felt. Hat makers were known to exhibit bizarre behavior and terms such as "hatter's shakes" arose due to the neurological symptoms of chronic (long-term) mercury

poisoning. Consequently, the Mad Hatter in "Alice in Wonderland" got his name because hatters in Lewis Carroll's day often displayed quite erratic behavior (www.ec.gc.ca/MERCURY).

Today, the main effects of mercury exposure to humans are understood to be neurological, renal (kidney), cardiovascular and immunological impacts (Goldwater and Clarkson, 1972) Mercury is toxic by ingestion, inhalation and skin absorption with acute and chronic exposure effects including central nervous system and kidney damage. Acute exposure includes nausea, blurred vision, painful breathing, excessive salivation and pneumonitis, while chronic or longer-term exposure includes memory disturbance, hypertension, vision problems, hallucinations, tremors and personality changes.

Because mercury can cross the blood-brain barrier, and because it can affect brain development (Goldwater and Clarkson, 1972), its effects are of special concern to pregnant or lactating women and young children. For this reason, many states, including North Carolina, as well as the U.S. EPA, have issued fish consumption advisories. Many of these advisories are directed towards pregnant or lactating women and young children, or are stricter for these populations, due to concerns over developmental disabilities in infants from mercury exposure.

Whether an exposure to the various forms of mercury will harm a person's health depends on a number of factors. Almost all people have at least trace amounts of methyl mercury in their tissues (epa.gov/mercury/effects.htm), reflecting methyl mercury's widespread presence in the environment and people's

exposure through the consumption of fish and contaminated food. People may be exposed to mercury in any of its forms under different circumstances.

The factors that determine how severe the health effects are from mercury exposure include (Baldwin and Marshall, 1999): the chemical form of mercury (methyl mercury is more toxic than elemental mercury), the dose, the age of the person exposed (the fetus is the most susceptible), the duration of exposure, the route of exposure (inhalation, ingestion, dermal contact, etc.) and the health of the person exposed.

Metabolism of Mercury

When an individual is exposed to mercury, a certain percentage is absorbed, depending on the route of exposure and the form of the mercury. About 80% of elemental mercury is absorbed when inhaled, however less than 1% of ingested liquid mercury is absorbed (www.ec.gc.ca/MERCURY). Methyl mercury, on the other hand, is readily absorbed irrespective of the exposure pathway. Approximately 95% of ingested methyl mercury is absorbed, and absorption through the lungs and skin (Taylor and Francis, 1995) is also believed to be quite high. Both elemental and methyl mercury can cross the blood-brain and placental barriers. The critical target organ for elemental mercury is the adult and fetal brain, and the critical target organs for methyl mercury are the brain and the kidneys.

Inorganic mercury compounds do not readily migrate through the blood-brain or placental barriers, but do accumulate in the kidneys. Absorption of

inorganic mercury varies with the type of inorganic salt. Within the body, the kidneys accumulate the highest concentrations of all forms of mercury, yet mercury can also concentrate in the brain, the central nervous system, the liver, and indeed in most organs in the body.

Mercury is predominantly excreted from the body in urine and feces, but usually at a slower rate than that of uptake, leading to the accumulation of mercury in living tissue. Mercury is deposited in hair as it grows, and it may also be found in breast milk. This may result in high concentrations in infants whose mothers are heavily exposed. The unborn child also receives some of the maternal mercury body burden because mercury compounds cross the placental barrier, yielding equal or higher blood concentrations in the fetus than in the mother. (www.ec.gc.ca/MERCURY).

Some Facts about Mercury

Most authorities on Mercury ecotoxicology agree on six points.

First, Hg and its compounds have no known biological function, and its presence in living organisms is undesirable and potentially hazardous.

Second, forms of mercury with relatively low toxicity can be transformed into forms with very high toxicity through biological and other processes.

Third, methyl mercury can be bioconcentrated in organisms and biomagnified through food chains, returning mercury directly to man and other upper trophic level consumers in concentrated form.

Fourth, mercury is a mutagen, teratogen, and carcinogen, and causes

embryocidal, cytochemical, and histopathological effects.

Fifth, high body burdens of mercury normally encountered in some species of fish and wildlife from remote locations emphasize the complexity of natural mercury cycles and human impacts on these cycles.

And finally, the anthropogenic use of mercury should be curtailed, because the difference between tolerable natural background levels of mercury and harmful effects in the environment is exceptionally small.

These, and other aspects of mercury and its compounds in the environment as a result of anthropogenic or natural processes, have been the subject of many reviews, including those by Jernelov et al. (1972, 1975), NAS (1978), Birge et al. (1979), Nriagu (1979), EPA (1980, 1985), Clarkson and Marsh (1982), Das et al. (1982), Boudou and Ribeyre (1983), Elhassani (1983), Clarkson et al. (1984).

Cadmium

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. The most common oxidation state of cadmium is +2; though rare examples of +1 can be found. Cadmium can mainly be found in the earth's crust. It always occurs in combination with zinc. Cadmium also consists in the industries as an inevitable by-product of zinc, lead and copper extraction. After being applied it enters the environment mainly through the ground, because it is found in manures and pesticides. It was first discovered in Germany in 1817 (Van Assche and Ciarletta,

1992). The name is derived from the Latin *cadmia* and the Greek *kadmeia.* Cadmium enters air from mining, industry, and burning coal and household wastes.

Cadmium is also a contaminant in zinc. Cadmium particles in air can travel long distances before falling to the ground or water. It enters water and soil from waste disposal and spills or leaks at hazardous waste sites. It binds strongly to soil particles. Some cadmium dissolves in water. It doesn't break down in the environment, but can change forms. Fish, plants, and animals take up cadmium from the environment. Cadmium stays in the body a very long time and can build up from many years of exposure to low levels. Environmental Protection Agency (EPA) drinking water limit: 5 ppb. EPA also limits how much cadmium can enter lakes, rivers, waste sites, and cropland and forbids cadmium in pesticides. Cadmium has direct adverse effects on cells in the arterial wall. It is well documented that cadmium disrupts intracellular transport in neurons by inhibiting *microtubule polymerization* and assembly. Eating food or drinking water with very high levels (metal and compounds) increases salivation, severely irritates the stomach, leading to vomiting and diarrhea. Skin contact with cadmium is not known to cause health effects in humans or animals. Long term exposure to lower levels of cadmium in air, food, or water leads to a build up of cadmium in the kidneys and possible kidney disease. Other potential long term effects are lung damage and fragile bones, abdominal pain, choking and tenesmus. Acute effects of cadmium occur by breathing high levels dust or fumes and may cause throat dryness, cough, headache, vomiting, chest pain, extreme restlessness and

irritability, pneumonitis, possibly bronchopneumonia and can cause death due to severe lung damage. Cadmium salts are more toxic than those of zinc. The Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds may reasonably be anticipated to be carcinogens.

Cadmium has no essential biological function and is extremely toxic to humans. In chronic exposure, it accumulates in the body, particularly in the kidneys and the liver (Baldwin and Marshall, 1999, Williams et al. 1999). As cadmium and zinc are found together in natural deposits, so are they similar in structure and function in the human body. Cadmium may actually displace zinc in some of its important enzymatic and organ functions; thus, it interferes with these functions or prevents them from being completed (Baldwin and Marshall, 1999). The zinc-cadmium ratio is very important, as cadmium toxicity and storage are greatly increased with zinc deficiency, and good levels of zinc protect against tissue damage by cadmium. The refinement of grains reduces the zinc-cadmium ratio, so zinc deficiency and cadmium toxicity are more likely when the diet is high in refined grains and flours.

Cadmium and solutions of its compounds are toxic, particularly in soluble and respirable forms, being more easily absorbed through inhaled dusts and fumes. Chronic dust or fume exposure can irreversibly damage the lungs, producing shortness of breath and emphysema. Acute poisoning from inhalation of fumes and ingestion of cadmium salts can also occur and at least one death has been reported from self-poisoning with cadmium chloride (Baldwin and Marshall, 1999). The risks of absorption via dermal contact are negligible. The

International Agency for Research on Cancer lists cadmium metal and several of its compounds as **carcinogens**. Because of its toxicity, the use of cadmium is regulated by the Environmental Protection Agency (EPA) and other regulatory control agencies.

Cadmium has a wide variety of sources in the environment and from industry. One source is from ingestion of grown foodstuffs, especially grain and leafy vegetables, which readily absorb cadmium from the soil. The cadmium may occur naturally or as a contaminant and the contaminants include sewage sludge, fertilizers, polluted groundwater and mining effluents. Cadmium may also contaminate fish (Hu, 1998, Williams et al. 1999)

About three-fourths of cadmium is used in Ni-Cd batteries, most of the remaining one-fourth is used mainly for pigments, coatings and plating, and as stabilizers for plastics. Cadmium has been used particularly to electroplate steel where a film of cadmium only 0.05 mm thick will provide complete protection against the sea. Cadmium has the ability to absorb neutrons, so it is used as a barrier to control nuclear fission.

Chemical Properties of Cadmium

Cadmium and its compounds are stable. In water some of the compounds will be quite soluble (cadmium chloride) and others will be insoluble (cadmium oxide). As fine powder cadmium metal will burn, releasing toxic fumes of cadmium oxide. Cadmium is most often found combined with other elements, which produces compounds such as Cadmium chloride, Cadmium oxide, and Cadmium sulfite.

Natural sources of Cadmium

Cadmium is a naturally occurring element in the crust of the earth. Coal and other fossil fuels contain cadmium and their combustion releases the element into the atmosphere. Cadmium is found naturally in various ores: lead and copper containing zinc, some iron ores, and in sulfide ore. These can result in emissions to water. Volcanic emissions contain cadmium-enriched particles.

Cadmium-containing ores are rare and when found they occur in small quantities. Greenockite (CdS), the only cadmium mineral of importance, is nearly always associated with sphalerite (ZnS). Consequently, cadmium is produced mainly as a byproduct from mining, smelting, and refining sulfide ores of zinc, and to a lesser degree, lead and copper. Small amounts of cadmium, about 10% of consumption, are produced from secondary sources, mainly from dust generated by recycling iron and steel scrap (Baldwin and Marshall, 1999). Production in the United States began in 1907 but it was not until after World War I that cadmium came into wide use (Hu, 1998).

The naturally occurring presence of cadmium in the environment results mainly from gradual phenomena, such as rock erosion and abrasion, and of singular occurrences, such as volcanic eruptions. Cadmium is therefore naturally present in air, water, soil and foodstuffs.

Anthropogenic Sources of Cadmium

There are many sources from which our environment and our bodies can be contaminated with cadmium. Cigarette smoke, refined foods, water pipes, coffee and tea, coal burning, and shellfish are all definite sources. Cadmium is

also a component of alloys, used in electrical materials, and is present in ceramics, dental materials, and storage batteries.

During the growth of grains such as wheat and rice, cadmium (from the soil) is concentrated in the core of the kernel, while zinc is found mostly in the germ and bran coverings (Taylor and Francis, 1995). With refinement, zinc is lost, increasing the cadmium ratio. Refined flours, rice, and sugar all have relatively higher ratios of cadmium to zinc than do the whole foods.

One pack of cigarettes contains about 20μg of cadmium, or about 1μg per cigarette (Taylor and Francis, 1995). About 30 percent of that goes into the lungs and is absorbed, and the remaining 70 percent goes into the atmosphere to be inhaled by others or to contaminate the environment. With long-term smoking, the risk of cadmium toxicity is increased. Though most of it is eliminated, a little bit is stored every day. Marijuana may also concentrate cadmium, so regular smoking of cannabis may also be a risk factor for toxicity from this metal (Williams et al. 1999).

Water pipes can be a source of cadmium concentration (Hu, 1998)). Cadmium is often used to protect metals from corrosion. Galvanized (zinc) pipes usually contain some cadmium, as does the solder used to hold them together. Soft or acid water is corrosive and causes the metals in the pipes to break down, releasing cadmium and other minerals from them. Hard water containing calcium and magnesium salts actually coats the pipes and protects against the leaching of other minerals. Soil levels of cadmium are increased by, cadmium in water, by sewage contamination, by cadmium in the air, and by high-phosphate fertilizers.

Coffee and tea may contain significant cadmium levels. Root vegetables such as potatoes may pick up more cadmium, and the grains can concentrate cadmium. Seafood, particularly crustaceans, such as crab and lobster, and mollusks, such as clams and oysters, have higher cadmium levels (Hu, 1998). The anthropogenic sources of Cadmium can be grouped under the following:

Industrial sources of Cadmium: Air pollution with cadmium comes from zinc mining and refining, and from the burning of coal. Cadmium is also an industrial contaminant from the steel-making process.

Cadmium is obtained as a by-product from the treatment of zinc, copper, lead, and iron ores, therefore facilities that treat these ores may emit cadmium compounds to the environment (mainly water). Coal and oil burning power plants may emit cadmium compounds to the air.

Transport sources of Cadmium: The combustion of motor fuels (petrol) in cars, trucks, and planes result in emissions to air, and particles from tire wear may result in emissions to air, land and water.

Consumer products that emit Cadmium: Small industrial domestic use of cadmium products will emit low levels of cadmium to the environment. Tobacco smoke will be an indoor source of cadmium.

Cadmium is found in many domestic products, e.g. tobacco products, phosphate fertilizers, polyvinyl chloride (PVC) products, photocells, petrol, oils, tyres, automobile radiators, some textile dyes and colors, electronic components, heating elements in electric kettles and hot water systems, batteries, and ceramic glazes.

Uses of Cadmium

About three-fourths of cadmium is used in batteries (especially Ni-Cd batteries) and most of the remaining one-fourth is used mainly for pigments, coatings and plating, and as stabilizers for plastics.

Cadmium also finds use in the following areas:

1. It is used in some of the lowest melting alloys. 2. Due to a low coefficient of friction and very good fatigue resistance, it is used in bearing alloys. 3. 6% of cadmium finds use in electroplating. 4. Many kinds of solder contain this metal. 5. As a barrier to control nuclear fission. 6. Compounds containing cadmium are used in black and white television phosphors and also in the blue and green phosphors for color television picture tubes. 7. Cadmium forms various salts, with cadmium sulfide being the most common. This sulfide is used as a yellow pigment. Cadmium selenide can be used as red pigment, commonly called *cadmium red.* 8. Used in some semiconductors such as cadmium sulfide, cadmium selenide, and cadmium telluride, this can be used for light detection or solar cells. HgCdTe is sensitive to infrared. 9. Some cadmium compounds are employed in PVC as stabilizers. 10. Used in the first neutrino detector. 11. Used to block voltage-dependent calcium channels from fluxing calcium ions in molecular biology.

Cadmium Levels in Foodstuffs

Cadmium levels can vary widely in various types of foodstuffs. Leafy vegetables such as lettuce and spinach and certain staples such as potatoes and

grain foods exhibit relatively high values from 30 to 150 ppb. Peanuts, soybeans and sunflower seeds also exhibit naturally high values of cadmium with seemingly no adverse health effects. Meat and fish normally contain lower cadmium contents, from 5 to 40 ppb. Animal offal such as kidney and liver can exhibit extraordinarily high cadmium values, up to 1,000 ppb, as these are the organs in animals where cadmium concentrates (WHO 1992, ATSDR 1997). The cadmium contents of foodstuffs may vary widely with the agricultural practices utilized in the particular areas such as phosphate fertilizers, sewage sludge and manure application, the types of crops grown, and atmospheric cadmium deposition from natural or anthropogenic sources. Since various studies have shown that man's cadmium intake, as least for non-smokers, comes principally (approximately 95%) from the ingestion of foods rather than from inhalation of cadmium in air, it is the cadmium levels of foods which most affect the general population.

There are strong indications that cadmium levels in foodstuffs have substantially decreased during the past several decades due to the progressive control of cadmium emissions to the environment (Van Assche and Ciarletta 1993, Watanabe et al. 1993, Watanabe et al. 1994). Recent studies have further documented that the cadmium content of food crops in Europe and many other countries are now stable and not increasing with time (Chaudri et al. 1995).

Human Intake of Cadmium

Ingestion: 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. 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).

As noted earlier, the cadmium content of terrestrial foods varies significantly as a function of the type of food crop grown, the agricultural practices pursued, and the atmospheric deposition of cadmium onto exposed plant parts. Cadmium levels in the soil principally derived from natural sources, phosphate fertilizers and sewage sludge will naturally impact upon this cadmium uptake. However, this effect is secondary to the type of crop grown and the agricultural practices followed with respect to tillage, Aiming and crop rotation.

Levels of Cadmium Intake from Foods: Many studies have attempted to establish the average daily cadmium intake resulting from foods; In general, these studies show that the average daily diet for non-smokers living in uncontaminated areas is at present at the low end of the range of 10 to 25µg of cadmium (Elinder 1985, OECD 1994, ATSDR 1997). This general trend is confirmed by decreasing blood cadmium levels in the general population in several countries during this time period (Ducoffre 1992). In a rather recent evaluation, the International Programme on Chemical Safety (IPCS) assessed the average daily intake at the lower end of this range (WHO, 1992).

The World Health Organisation (WHO) has established a provisional tolerable weekly intake (PTWI) for cadmium at 7µg/kg of body weight This PTWI weekly value corresponds to a daily tolerable intake level of 70µg of cadmium for the average 70kg man and 60µg of cadmium per day for the average 60kg woman. Clearly, the daily cadmium intake for the general population from food, which is by far the dominant source of cadmium, is well below the guidelines established by the World Health Organisation. The average daily cadmium intake for the general population in the Western World has shown a distinct downward trend from 1970 through 1992 (Van Assche and Ciarletta, 1992), a reduction presumed to be due to the marked decreases in direct atmospheric deposition of cadmium onto crops and soils. Other studies have suggested that, over the timeframe of 1980 - 1985, levels of cadmium intake have been relatively constant (OECD, 1994). At an absorption rate of 5% from ingestion, the average person is believed to retain about 0.5 to 1.0µg of cadmium per day from food.

There is considerable information in the literature regarding the cadmium contents of foods grown in contaminated areas (Elinder 1985, WHO 1992, OECD 1994). Detailed studies have indicated that only a small percentage of these contaminated areas were actually utilized for growing foods which were subsequently consumed with the exception of rice fields in Japan where considerable cadmium did find its way into the average person's diet through rice grown on contaminated rice fields (Elinder, 1985). In specific cases, management measures to reduce the transfer of cadmium from historically contaminated soils

into the local food chain have proven successful (Staessen et al. 1991).

Inhalation: Cadmium inhalation is a far smaller contributor to total cadmium body burden except, as previously noted, in the cases of smokers or some highly exposed workers of the past. Today, the inhalation route is well controlled in the occupational setting, and is well-controlled from point sources such as those which directly pertain to the non-ferrous, cadmium or cadmium products industries. Ambient air emissions from fossil fuel power generation plants, the iron and steel industry and other major industries where cadmium may be present as a low concentration impurity, on the other hand, may be substantial because the volumes of the waste gases generated are substantial.

Cadmium Intake From Cigarette Smoking - Smokers absorb amounts of cadmium comparable to those from food, about 1 to 3μg of cadmium per day, from the smoking of cigarettes. It has been reported that one cigarette contains about 1 - 2μg of cadmium and that about 10% of the cadmium content is inhaled when the cigarette is smoked (WHO, 1992). Cigarette construction, the use of filters and variations in the cadmium contents of tobaccos could decrease cadmium exposure by this route, but in general cigarette smoking is habits which can more than double the average person's daily cadmium intake. Cigarette smokers who are also occupationally exposed may increase their total cadmium intake even further.

Cadmium Intake From Occupational Exposure - Up to the 1960s, very elevated cadmium in air exposure levels were measured in some workplaces, sometimes as high as 1mg/m^3. Since that time, workplace exposures and standards

have decreased markedly so that most occupational exposure standards today are in the range from 2 to 50μg/m³. The result has been that occupational exposures today are generally below 5μg/m³, and most cadmium workers are exposed at levels which are considered to be safe (ATSDR, 1997). In rare cases where cadmium air levels are higher, the use of personal protective equipment is obligatory. Extensive preventative hygiene programs and medical follow-up programs have been developed to control the risk related to cadmium exposure at the workplace (ACGIH 1996, OSHA 1992, Lauwerys 1986, Cadmium Council 1986). Considering present levels of occupational exposure cadmium intake, general dietary intake, and cigarette smoking intake, it still would appear, however, that the average daily cadmium intake is well below the values recommended by the World Health Organisation.

Sources of Cadmium to the General Population: Ingestion of cadmium in food is the major source of cadmium for non-smokers. Average daily intakes from food in non-contaminated areas is at the lower end of the 10 to 25μg range of which approximately 0.5 to 1.0μg is actually retained in the body. Uptake of cadmium from smoking could more than double that amount.

The actual level of intake which results from food ingestion varies as a function of multiple factors. For example, certain crops (e.g., sunflowers) and shellfish contain naturally elevated amounts of cadmium. Individuals who consume large amounts of these materials might thus at first seem to be at increased risk. However, recent studies have demonstrated that foods which are naturally enriched in cadmium are also enriched in substances which inhibit the

uptake of cadmium into the body. Thus, individuals who ingest large amounts of sunflower seeds may ingest up to 100μg cadmium per day, yet these individuals do not have levels of cadmium in blood or urine which are higher than individuals with far lower levels of cadmium intake (Reeves et al. 1997). Similarly, consumption of a diet rich in shellfish can double the intake of dietary cadmium without producing significant impacts upon blood cadmium (Vahter et al. 1996) These studies illustrate that the cadmium content of food is just one of a number of factors which determines the actual uptake of cadmium into the body.

Indeed, recent studies have suggested that overall nutritional status is a more important determinant of cadmium uptake into the body than is the actual amount of cadmium ingested (Vahter et al. 1996). For example, women subsisting upon a vegetarian diet and with reduced iron stores have increased uptake of ingested cadmium. For these women, iron deficiency is a more important determinant of cadmium uptake than is the actual amount of cadmium ingested.

The present levels of cadmium intake in most European countries are far below the PTWI recommended by WHO. Indeed, as a result of numerous public health policies implemented over the past several decades, the cadmium body burden of the general population appears to be rapidly declining (Friis et al. 1998). Present exposure levels in many European countries are now comparable to, or lower than, those which characterize 'unadulterated populations' residing in the jungles of South America (Hecker et al. 1974).

Health Effects of Cadmium

Cadmium and solutions of its compounds are toxic, particularly in soluble

and respirable forms, being more easily absorbed through inhaled dusts and fumes. Chronic dust or fume exposure can irreversibly damage the lungs, producing shortness of breath and emphysema. The risks of absorption via dermal contact are negligible. The International Agency for Research on Cancer lists cadmium metal and several of its compounds as **carcinogens** (Hazard Summary, 1992). Because of its toxicity, the use of cadmium is regulated by the Environmental Protection Agency (EPA) and other regulatory control agencies.

Breathing high levels of cadmium may severely damage the lungs and can cause death. Eating food or drinking water with very high levels of Cadmium severely irritates the stomach, causing vomiting and diarrhea. Cadmium mainly accumulates in the kidneys and liver and can lead to serious kidney failure, nephrotoxicity, renal stone formation, bone disease and persistent proteinuria at high exposures. Cadmium stays in the body for a very long time and can build up from many years of exposure to low levels. Other effects from acute cadmium exposures include: muscle cramps, salivation, sensory disturbances, liver injury, convulsions, shock and renal failure.

Other potential effects of long-term cadmium exposure include: high blood pressure, iron-poor blood, liver disease, nerve or brain damage, lung damage, fragile bones, intestinal damage.

A balanced diet can reduce the amount of cadmium taken into the body from food and drink. Animal studies suggest that more cadmium is absorbed into the body if the diet is low in calcium, protein, or iron, or is high in fat. Some epidemiological studies have suggested a link between drinking hard water and

some degree of protection from hypertension. In some studies, younger animals absorbed more cadmium and were more likely to lose bone and bone strength than adult animals. Animals exposed en-utero to high cadmium levels suffered behavior abnormalities, learning deficits, low birth weight, and skeletal abnormalities, but birth defect potential in humans is not well known. Small portions of cadmium can cross the placenta, and cadmium can be present in breast milk if the mother carries elevated levels.

The kidney is the critical target organ for the general population as well as for occupationally exposed populations. Cadmium is known to accumulate in the human kidney for a relatively long time, from 20 to 30 years, and, at high doses, is also known to produce health effects on the respiratory system and has been associated with bone disease. Most of the available epidemiological information on cadmium has been obtained from occupationally exposed workers or on Japanese populations in highly contaminated areas.

Most studies have centered on the detection of early signs of kidney dysfunction and lung impairment in the occupational setting, and, in Japan, on the detection and screening for bone disease in general populations exposed to cadmium-contaminated rice. More recently, the possible role of cadmium in human carcinogenesis has also been studied in some detail.

Zinc

Zinc is one of the commonest elements in the earth's crust. It's found in air, soil, and water, and is present in all foods. Zinc has many commercial uses including its use in extracting gold by the cyanide process. Most of the zinc in soil

stays bound to soil particles. It builds up in fish and other organisms, but it doesn't build up in plants.

Zinc can act as nutrient in the body and is essential for health, while others (e.g. mercury, cadmium, lead) have no known beneficial health effects. All may be harmful if excessive amounts are consumed for example even though zinc is good for human health high levels of zinc can result in a deficiency of copper, another metal required by the body. The importance of zinc and copper are illustrated by their roles as co-factors for SOD. Zinc is also very important in protein, nucleic acid, and energy metabolism and copper is required in the synthesis of *catacholamines*.

Zinc is an essential element in our diet. Too little zinc can cause health problems, but too much zinc is also harmful. Acute toxicity: Inhalation of fumes may result in sweet taste, throat dryness, cough, weakness, generalized aching, chills, fever, nausea and vomiting. Zinc chloride fumes have caused injury to mucous membranes and pale gray cyanosis. Ingestion of soluble salts may cause nausea, vomiting and purging. Breathing large amounts of zinc (as dust or fumes) can cause a specific short-term disease called metal fume fever. This is believed to be an immune response affecting the lungs and body temperature. Chronic toxicity: Harmful health effects generally begin at levels from 10-15 times the RDA ie recommended daily allowance (in the 100 to 250 mg/day range). Eating large amounts of zinc, even for a short time, can cause stomach cramps, nausea, and vomiting. Taken longer, it can cause anemia, pancreas damage, and lower levels of high-density lipoprotein cholesterol (HDL - the good form of

cholesterol).Pure zinc is a bluish-white, shiny metal. Powdered zinc is explosive and can burst into flames if stored in a damp place. Because it is an element, zinc does not degrade nor can it be destroyed.

Zinc is one of the most abundant trace elements in the human body. It is typically taken in by ingestion of food and water, although it can also enter the lungs by inhaling air, including that contaminated with zinc dust or fumes from smelting or welding activities. The amount of zinc that can pass directly through the skin is very small.

Absorption of zinc into the bloodstream following ingestion is normally regulated by homeostatic mechanisms, so the balance between zinc intake and excretion is controlled. Absorption from the gastrointestinal tract is 20 to 30% in people with diets containing adequate levels of zinc, but it can reach 80% in those with low levels of zinc in their diets or body tissues (Walsh et al. 1995). Zinc is normally excreted in the urine and feces, with small amounts excreted in sweat. About 90% of what is retained is found in muscles and bones (Fox, 1990). Zinc is an essential element in our diet, but too little or too much can be harmful.

Zinc has many commercial and industrial uses. Metallic zinc is used to coat iron and other metals to prevent rust, and it is also used in dry cell batteries. Zinc is mixed with other metals to form alloys such as brass and bronze, and pennies are made from a copper-zinc alloy. Zinc is also combined with other elements such as chlorine, oxygen, and sulfur to form zinc compounds used to make white paints, ceramics, rubber, wood preservatives, dyes, and fertilizers. Zinc compounds are also used in the drug industry as ingredients in common

products like sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and anti-dandruff shampoos.

It can be released by natural processes, but most results from human activities. Releases to air, water, and soil are common in areas where ores are mined, processed, and smelted for zinc. Because cadmium and lead are commonly present in zinc containing ores, they are also typically released during these processes and so areas are often jointly contaminated. Zinc can be released to the atmosphere during the production of steel and burning of coal or waste. Surface water can be impacted by discharges of metal manufacturing and chemical industry wastes, and also by run-off following precipitation on soils high in zinc, either due to the natural setting or human applications, including use of zinc fertilizer on agricultural soils.

Chemical Properties of Zinc

Zinc, an element with an atomic weight of 65, is classified as a Group IIB post-transition member of the periodic table. The Group IIB metals below zinc in the periodic table are cadmium and mercury, nonessential metals of greater toxicity. Key chemical characteristics of zinc are 1) a tendency to lose two electrons and as the +2 cation form salts of varying solubility in aqueous solution and 2) a tendency to form relatively stable coordinate bonds with electronegative ligands such as nitrogen, oxygen, and sulfur. Zinc, unlike other transition elements, is relatively stable in the divalent state and does not undergo redox changes.

Zinc in aqueous solution as the +2 cation becomes hydrated at low pH and

at high pH forms zincate anions, possibly Zn $(OH)_4^{-2}$. Various zinc compounds differ significantly in their aqueous solubility. For example, the solubility of zinc chloride (mw 136) in water at 25šC is 432 g/100 ml whereas that of zinc oxide (mw 81) at 29šC is 0.00016 g/100 ml (Lide ed. 1990).

Natural Sources of Zinc

Zinc is part of nature. Most rocks and many minerals contain zinc in varying amounts and zinc exists naturally in air, water and soil. The average natural level of zinc in the earth's crust is 70 mg/kg (dry weight), ranging between 10 and 300 mg/kg (Malle, 1992).

At some locations, zinc has been concentrated to much higher levels by natural geological and geochemical processes. Such concentrations, found at the earth's surface and underground, are being exploited as ore bodies. The most commonly found zinc mineral is sphalerite (ZnS). Zinc metal is produced both from ores and from recycled zinc products. In fact, 30% of the world zinc supply today comes from recycled zinc. Due to natural erosion processes like the weathering and abrasion of rock, soils and sediments by wind and water, a small but significant fraction of natural zinc is continuously being mobilized and transported in the environment. Volcanic eruptions, forest fires and aerosol formation above seas also contribute to the natural transport of zinc. These processes causes cycling of zinc in the environment, resulting in natural background levels in the air, surface waters and soil.

Just as the natural amount of zinc in soil varies, the zinc concentration in water depends on a multitude of factors such as the nature and age of the

geological formations through which the water flows, together with biological and physicochemical conditions (Sandstead, 1982). Seasonal variations also influence zinc concentration in water (Boutron et al. 1995). Nonetheless, some general categories of surface waters can be defined, which are characterized by a range of natural background zinc levels. These general categories, called habitat types, are where communities of organisms - ecosystems - dwell, which are conditioned to the zinc levels present. The European alluvial lowland rivers, the U.S. Rocky Mountain streams, and the Great Lakes in North America are examples of freshwater habitat - types with different natural ranges of zinc concentration (Boutron et al. 1995).

Anthropogenic Sources

Zinc is found throughout the environment in air, soil, and water, and it is present in all foods. It can be released by natural processes, but most results from human activities. Releases to air, water, and soil are common in areas where ores are mined, processed, and smelted for zinc. Because cadmium and lead are commonly present in zinc containing ores, they are also typically released during these processes and so areas are often jointly contaminated. Zinc can be releases to the atmosphere during the production of steel and burning of coal or waste. Surface water can be impacted by discharges of metal manufacturing and chemical industry wastes, and also by run-off following precipitation on soils high in zinc, either due to the natural setting or human applications, including use of zinc fertilizer on agricultural soils.

The average concentration of zinc in air (as fine dust particles) is typically

less than 1 microgram per cubic meter ($\mu g/m^3$) lthough concentrations of $5\mu g/m^3$have been measured near industrial sources (Argonne National Laboratory, 2005). In lakes and rivers, some zinc remains dissolved in water or as fine suspended particles, while other zinc settles to the bottom in association with heavier particles. Average concentrations range from 0.02 to 0.05 milligram per liter (mg/L) in surface water and 0.01 to 0.1 mg/L in drinking water (Argonne National Laboratory, 2005). Zinc generally remains in the upper layers bound to soil particles, but it can leach to groundwater depending on soil characteristics, moving more readily in sandy soil. Concentrations of zinc in sandy soil particles are about 200 times higher than in the water between the soil particles, and concentration ratios are even higher (over 1,000) in both loam and clay soils.

Some fish may accumulate zinc, but it does not build up in plants. The typical ratio of the concentration in plants to that in soil is estimated at 0.9 (or 90%). Zinc has been measured in food at concentrations ranging from 2 parts per million (ppm) in leafy green vegetables to 29 ppm in meat, fish, and poultry. On average, people ingest 7 to 163 mg of zinc every day (Argonne National Laboratory, 2005).

Levels of Zinc in the Environment

Airborne zinc particles are deposited on the land and surface waters. In the soil, zinc is bound to the soil complex (clays, organic matter...), depending on different physicochemical soil factors such as pH and organic matter content (Boutron et al. 1995).

These factors determine the solubility of the zinc contained in soil, and

consequently, its bioavailability for uptake by organisms. Changes in soil pH, for example, dramatically alter the bioavailability of zinc in soil. Soils and sediments are more static compartments of the environment than air and surface waters.

In the vicinity of some old industrial sites, levels of zinc in the soil, usually in combination with other metals, can be elevated due to high emissions in the past *(historical contamination)*. Such sites need specific attention and appropriate risk management to limit exposure of the local ecosystem and prevent contamination from spreading to surrounding areas. Promising results have recently been obtained with metal immobilizing compounds that, when mixed with contaminated soils, fix zinc and other metals to the soil complex, rendering them less available for uptake by organisms (Van Gronsveld et al.1994).

Uses of Zinc

Modern life is inconceivable without zinc. Zinc provides the most cost-effective and environmentally efficient method of protecting steel from corrosion. In simple terms, zinc means that an average size home can now be built from six scrap automobiles instead of 1acre (0.4 hectares) of forest.

By protecting steel against corrosion, zinc helps save resources such as iron ore and energy1. By extending the life and durability of steel, zinc extends the life of capital investments, and in the case of public infrastructure - roads, bridges ports, power and water distribution, and telecommunications - helps save taxpayers' money too.Besides protecting steel against corrosion, zinc has many other uses: in brass and other alloys, in automotive equipment and household appliances, fittings, tools, toys.., in building and construction, in pharmaceuticals,

medical equipment and cosmetics, in tyres and all rubber goods, in fertilizers and animal feed.

Zinc is also an essential element which is indispensable for human health and for all living organisms. This essentiality makes the interaction between zinc and the environment complex.

The Value of Zinc to Man

Life on earth as we know it today has evolved in the presence of natural levels of zinc. Due to its general availability to organisms (bioavailability) and its characteristics, zinc has been used by nature to play a specific role in various biological reactions. As such, zinc is an essential element for all life, from man to the smallest micro-organism.

Organisms take up the essential elements they need from their environment that means directly from air, water, soil, and from food. When their cellular requirements for these elements are satisfied, growth and development are optimal. When uptake is too low, deficiency occurs and adverse effects can be observed. On the other side, uptake of too much of an essential element can lead to toxicity. Between these two extremes, each organism has a *concentration range* for each essential element within which its requirements are satisfied. Thus, an *Optimal Concentration Range* for zinc exists for each living organism, including man. Indeed, there is an *Optimal Concentration Range for Essential Elements (OCEE)* for each essential element and each living organism (Van Assche et al. 1996).

Zinc is an essential element in our diet, but too little or too much can be

harmful. Without enough dietary intake, people can experience a loss of appetite, decreased sense of taste and smell, decreased immune function, slow healing of wounds, and skin sores. Too little zinc can also result in poorly developed sex organs and retarded growth in young men (Walsh et al. 1995). If pregnant women do not have enough zinc, babies might have growth retardation, reproduction or cause birth defects. However, infertility, low birth weight, and skin irritation have been observed in laboratory animals such as rats, guinea pigs, mice, and rabbits given high doses of zinc.

The U.S. Environmental Protection Agency (EPA) has stated that adequate information to evaluate the carcinogenicity of zinc is not available. However, no studies exist that indicate zinc causes cancer in humans.

Zinc deficiency may increase the toxic effects of arsenic, copper, cadmium and lead; thus an adequate amount of zinc can be considered protective against the toxicity of these elements. However, too much zinc can increase the absorption of lead, which has been shown to have an additive effect on the hematological effects of zinc (OSPARCOM, 1996).

Zinc is essential for human health: Zinc plays an essential role in human metabolism. For example, zinc is vital for the proper functioning of more than 200 enzymes, for the stabilization of DNA and the expression of genes, and for the transfer of nervous signals (Van Assche et al. 1996). The human body contains 2-3 g of zinc (compared with 7g of iron) which is found everywhere in the body, with the highest levels in muscles, liver, kidneys, bones and prostate.The recommended daily zinc intake is 12 mg/day for adult women and 15

mg/day for adult men (Sandstead, 1982). Daily intake is not only dependent on food, but also on sex, age and general health status. Growing infants, children, adolescents, women in pregnancy and the elderly have a higher zinc requirement (Sandstead, 1982). Certain groups are known to have an increased demand for zinc and have a higher risk of not getting enough zinc.

Food is the primary source of zinc for man, with only a small part coming from drinking water. Food products differ in their zinc content. The major sources of zinc in the diet are red meat, poultry, fish, seafood, whole cereals and dairy products.

Plants and animals need zinc to grow: During the course of evolution, all living organisms have taken up the zinc available from their environment and used it for specific functions in their metabolism. Consequently, all organisms are conditioned to the bio-available zinc concentrations in their natural environment which are not constant but subject to seasonal variation.

To cope with these fluctuations, organisms have developed a mechanism (*homeostasis*) that allows them to regulate their zinc uptake within certain limits. When the limits of this regulation mechanism are exceeded, adverse effects can occur.

Deficiency is not widespread under natural conditions, due to background zinc levels in nature. Zinc deficiency commonly occurs under non-natural conditions, however, such as in modern agriculture, where zinc bioavailability.

Effects of Zinc on Human Health

Zinc is essential for human health but many adults and children may not be getting enough zinc in their diets. A comprehensive review of current knowledge about zinc and human health concluded that there is a potential for zinc deficiency on a worldwide scale (Walsh et al. 1995). In the United States, studies concluded that a substantial part of the general population is at risk from zinc deficiency. Mild chronic deficiency is even predicted in people consuming low meat diets rich in phytate and fiber. Symptoms of zinc deficiency include reduced sense of taste and smell, skin disorders, mental lethargy and reduced fertility. Zinc nutritional supplements can successfully balance insufficient dietary zinc intake but high doses can lead to gastro-intestinal disorders and are not recommended without medical advice.

There is considerable evidence that zinc deficiency in humans is a serious worldwide problem and outweighs the potential problem of accidental, self-imposed, or environmental exposure to zinc excess. Acute deficiency and chronic deficiency are well-known entities in human populations and are probably much more common than generally recognized. The importance of zinc for human health was first documented in 1963 (Prasad et al. 1963). During the past 25 years, deficiency of zinc in humans due to nutritional factors and several disease states has now been documented throughout the world. Prevalence of zinc deficiency is high in populations that consume large quantities of cereal proteins containing high amounts of phytate, an organic phosphate compound. Alcoholism, malabsorption, sickle cell anemia, chronic renal disease, and other

chronically debilitating diseases are now known to be predisposing factors for zinc deficiency in humans (Prasad, 1988).

Zinc deficiency is reflected in clinical syndromes which affect men and women of all ages and all socioeconomic and cultural classes in the world. It is neither prevalent in any specific area of the world nor associated with any specific or definitive biochemical marker, which can make its identification difficult and confusing. Its presence is manifested by a wide spectrum of symptoms, from acute, life threatening problems to mild sub-clinical or marginal disorders which may only vaguely disturb well being. The acute problems are often seen in profoundly ill patients treated in hospitals, whereas sub-clinical problems may be so vague that patients seek assistance outside traditional medical practice.

Based upon clinical data and using traditional, epidemiologic techniques, Henkin and Aamodt have reclassified zinc deficiency into three syndromes; these are a) acute, b) chronic, and c) subacute zinc deficiency (Henkin and Aamodt, 1983). Acute zinc deficiency is relatively uncommon and follows parenteral hyperalimentation or oral L-histidine administration. Chronic zinc deficiency is more common, usually resulting from chronic dietary lack of zinc. Sub- acute or latent zinc deficiency is the most common of these syndromes. It is estimated that there are 4 million people in the United States with this syndrome, the initial symptom being dysfunction of taste and olfaction; treatment with exogenous zinc restores taste and smell but this usually requires months before these functions are returned to normal (Henkin et al. 1976). Diagnosis of these disorders is most efficacious following oral administration of zinc tracers such as ^{65}Zn, ^{67}Zn, or

^{70}Zn with subsequent evaluation of the kinetics of transfer of the isotope into various body tissues, the formulation of the data by compartmental analysis, and the integration of the data by a systematic model of zinc metabolism. Obviously, these techniques are complex and technically difficult, not a routine means readily applicable to assessing zinc status in individuals. In fact, there are no simple means for assessing the status of zinc in the human population.

Clinical symptoms of human zinc-deficiency states exhibit a spectrum ranging from mild to severe and may even be fatal if unrecognized and not corrected (Prasad, 1988). The clinical manifestations of severely zinc deficient subjects include bullous pustular dermatitis, diarrhea, alopecia, mental disturbances, and intercurrent infections due to cell-mediated immune disorders. These severe signs are seen in patients with acrodermatitis enteropathica secondary to an inborn error of zinc absorption, patients receiving total parenteral nutrition without zinc, and patients receiving penicillamine therapy. Growth retardation, male hypogonadism, skin changes, poor appetite, mental lethargy, abnormal dark adaptation, and delayed wound healing are usual manifestations of moderate deficiency of zinc. Recent studies show that a mild or marginal deficiency of zinc in humans is characterized by neurosensory changes, oligospermia in males, decreased serum testosterone in males, hyperammonemia, decreased serum thymulin activity, decreased IL-2 production, decreased natural killer cell activity, alterations in T cell subpopulations (Prasad, 1988), impaired neuropsychological functions and decreased ethanol clearance (Milne et al. 1991). All the above manifestations are correctable by zinc supplementation.

Zinc is generally considered a relatively nontoxic metal (NAS/NRC, 1980). This classification is based on several characteristics: a) zinc is a metal essential to hundreds of biological processes and must be consumed in the diet for optimum health; b) zinc is relatively abundant in the natural environment; c) the recommended daily allowance (RDA) of zinc in the human population is 8 to 15 mg higher than many other essential metals; d) zinc does not appear to accumulate in the body with age; e) there are no known genetic abnormalities which result in excessive accumulation of zinc in the body, unlike metals such as copper (Wilson's disease) and iron (hemochromatosis); f) homeostatic mechanisms regulate the body burden of zinc such that increased intake is associated with decreased absorption and increased excretion; g) zinc may have antioxidant effects and does not participate in oxidation-reduction cycles like iron and other transition elements; h) administration of zinc for therapeutic purposes in man at doses above the RDA have not produced significant pathology; and i) administration of zinc to experimental animals in doses more that 100 times the RDA have not produced significant pathology.

Some zinc salts, such as zinc chloride, in sufficient concentration can injure epithelial tissue. Inhalation, exposure of the skin, or ingestion can produce local pathological effects. In addition, intake of excess zinc has been reported in human studies to affect levels of pancreatic enzymes (Faintuch et al. 1978) and lipoproteins in serum to alter the metabolism of copper (Simon et al. 1988) and iron (Solomons, 1988), and to alter immunological function (Chandra, 1984).

The industrial use of zinc affects the environmental distribution of this

metal; zinc is frequently found in industrial waste sites. Currently, regulatory agencies in the United States are concerned with the possible significance of increased exposure to zinc through environmental sources such as increases in drinking water, particularly through leaching of zinc into ground water surrounding waste sites. Excess exposure to zinc is also potentially a hazard for industrial workers, especially through inhalation of welding fumes and exposure in smelting operations. Oral intake at levels above the RDA is also of concern in individuals who self medicate with dietary supplements of zinc and those who are treated with these preparations for therapeutic purposes.

CHAPTER THREE

EXPERIMENTAL

Chemicals

Concentrated tetraoxosulphate (Vl) acid (H_2SO_4), Concentrated Hydrochloric acid (HCl), Concentrated Trioxonitrate (V) acid (HNO_3), Potassium Permanganate ($KMnO_4$), Stannous Chloride ($SnCl_2.2H_2O$), Hydroxylamine Hydrochloride ($NH_2OH.HCl$), Potassium Persulphate ($K_2S_2O_8$), Nitrogen gas, Acetylene, Mercury Standards, Cadmium Standard, Zinc Standards.

Equipment

Atomic Absorption Spectrophotometer (AAS) (Unicam 929). Specification for cadmium analysis: wavelength = 228.8nm, lamp current = 8.0mA, slit width = 0.5nm, burner angle = 0.0, fuel gas flow rate =1.8, support gas is air, spray time = 3.0sec, burner height = 7.0nm, integrated time = 5.0sec. Specification for zinc analysis- wavelength = 213.9nm, lamp current = 8.0mA, slit width = 0.5nm, burner angle = 0.0, fuel gas flow rate = 2.0, support gas is air, spray time = 3.0sec, burner height = 7.0nm, integrated time = 5.0sec.

Automatic Mercury Analyzer (HG5000) Specification for mercury analysis- wavelength = 253.7nm, reductant = 10% w/v $SnCl_2.2H_2O$.

Preparation of Reagents

5% w/v $KMnO_4$

Exactly 50g of AnalaR $KMnO_4$ was weighed into a 1000mL graduated flask and dissolved with double distilled water. The solution was made to the mark.

5% w/v $K_2S_2O_8$

Exactly 50g of AnalaR $K_2S_2O_8$ was weighed into a 1000mL graduated flask and dissolved with double distilled water. The solution was made to the mark.

6% w/v $NH_2OH.HCl$

Exactly 60g of AnalaR $NH_2OH.HCl$ was weighed into a 1000mL graduated flask and dissolved with double distilled water. The solution was made to the mark with double distilled water.

10% w/v $SnCl_2.2H_2O$ Solution

Exactly 100g of AnalaR $SnCl_2.2H_2O$ was dissolved in 200mL of 36% AnalaR HCl and diluted to 1000mL with double distilled water in a 1000mL graduated flask.

0.5% v/v H_2SO_4

Exactly 5mL of 98% AnalaR H_2SO_4 was diluted with double distilled to 1000mL in a 1000mL graduated flask.

Aqua regia

Three volumes of 36% AnalaR HCl was added to one volume of 69.0-70.5% AnalaR HNO_3. This reagent was prepared immediately before use.

Preparation of Standard Solutions

Mercury standard solutions: standard solutions were prepared from the 1000ppb Merck standard stock solution using the dilution formula $C_1V_1 = C_2V_2$ where C_1 is the concentration of the stock solution, V_1 is the volume of the stock solution needed, C_2 is the concentration and V_2 the volume of the standard solution being prepared. 0.05, 0.1, 0.2, 0.3, 0.4, and 0.5mL of the stock solution was measured with a pipette into a 100mL graduated flask and diluted with double distilled water to the mark to prepare the following concentrations of mercury standard solutions; 0.5, 1.0, 2.0, 3.0, 4.0 and 5.0ppb.

Cadmium standard solutions: standard solutions were prepared from the 1000ppm Merck standard stock solution using the same dilution formula as above, 0.05, 0.1, 0.2, 0.3, 0.4 and 0.5mL of the stock solution was measured with a micro pipette into a 100mL graduated flask and diluted with double distilled water to the mark to prepare the following concentrations of cadmium standard solution 0.5, 1.0, 2.0, 3.0, 4.0 and 5.0ppm.

Zinc standard solutions: standard solutions were prepared from the 1000ppm Merck standard stock solution using the same procedure above, 0.05, 0.1, 0.2, 0.3, 0.4 and 0.5mL of the stock solution was measured with a micro pipette into a 100mL graduated flask and diluted with double distilled water to the mark to prepare the following concentrations of zinc standard solution 0.5, 1.0, 2.0, 3.0, 4.0 and 5.0ppm.

Sampling

Colocasia esculenta (Cocoyam) leaves, *Manihot esculenta* Crantz. (Cassava) leaves were sampled from farms in the study areas using gloved hands into clean polyethylene bags and tied. *Colocasia esculanta* (Taro) corm was uprooted and a stainless steel cutlass was used to cut the corm into clean polyethylene bags and tied. In a particular farm, six different samples were selected at random. The samples were transported to the laboratory.

Sample Preparation

In the laboratory the leaves were cleaned to remove all dirt particles and air dried. The taro corm was peeled using a stainless steel knife. The samples were kept in clean polyethylene bags sealed at both ends and stored in the fridge.

Digestion of sample for Zinc and Cadmium Analysis (USEPA 1991)

The air dried leaves were chopped into smaller pieces and mixed well using a spatula. Exactly 1.0g of the chopped leaf sample was weighed and 20mL of the aqua regia was added in a 100mL digestion flask. The mixture was stirred with a glass stirring rod intermittently until the green colour of the chopped leaves disappeared. The mixture was then put on a hot plate at 100°C in a fume chamber, small portions of the aqua regia, about 2-5mL were added to the mixture on the hot plate to ensure that the solution did not dry out, until the digestion was complete (that is when the digest was light in colour and the solution was clear and has stopped producing fumes). The volume of the digest was reduced to 5mL.

The mixture was cooled, filtered and diluted to the 100mL mark with double distilled water in a 100mL volumetric flask and stored at room temperature for the AAS analysis.

The air dried taro corms were chopped into smaller pieces and mixed well using a spatula. Exactly 1.0g of the chopped corm sample was weighed and 20mL of the aqua regia was added in a 100mL digestion flask. The mixture was stirred with a glass stirring rod intermittently until the colour of the chopped corms turned whitish. The mixture was then put on a hot plate at 100°C in a fume chamber, small portions of the aqua regia, about 2-5mL were added to the mixture on the hot plate to ensure that the solution did not dry out, until the digestion was complete (that is when the digest was light in colour and the solution was clear and has stopped producing fumes). The volume of the digest was reduced to 5mL. The mixture was cooled, filtered and diluted to 100mL with double distilled water in a 100mL volumetric flask and stored at room temperature for the AAS analysis.

Digestion of blank for Zinc and Cadmium Analysis

Exactly 1.0mL of double distilled water was measured into a 100mL digestion flask and 20mL of the aqua regia was added and mixed thoroughly. The mixture was put on a hot plate at 100°C in a fume chamber. Small portions of the aqua regia, about 2-5mL were added to the mixture on the hot plate to ensure that the solution did not dry out, until the digestion was complete (that is when the digest was light in colour and the solution was clear and has stopped producing fumes). The volume of the digest was reduced to 5mL. The mixture was cooled,

filtered and diluted to 100mL with double distilled water in a 100mL volumetric flask and stored at room temperature for the AAS analysis.

Digestion of sample for Mercury Analysis (USEPA 1991)

The air dried leaves were chopped into smaller pieces and mixed well using a spatula. Exactly 1.0g of the chopped leaf sample was weighed and 20mL of the aqua regia was added in a 100mL digestion flask. The mixture was covered tightly and left overnight to digest. The digest was placed in a hot water bath at 80°C for 30 minutes in a fume chamber. Small portions of the aqua regia, about 2-5mL were added to the mixture to ensure that the solution did not dry out, until the digestion was completed (that is when the digest was light in colour and the solution was clear and has stopped producing fumes). The volume of the digest was reduced to 5mL. The digest was cooled to 4°C in an ice bath. Exactly 15mL of 5% $KMnO_4$ solution was added to the digest, followed by 8mL of 5% $K_2S_2O_4$ solution to oxidize all mercury present in the solution to the +2 state. The digest was returned to the water bath for additional 30 minutes heating at 90°C. The sample was removed from the water bath, allowed to cool to room temperature and 6mL of 12% Hydroxylamine Hydrochloride solution was added to reduce the excess $KMnO_4$ in the solution. The resulting solution was filtered into a 100mL graduated flask and diluted to the 100mL mark with double distilled water and stored in the fridge for the AAS analysis.

The air dried taro corms were chopped into smaller pieces and mixed well using a spatula. Exactly 1.0g of the chopped corm sample was weighed and

20mL of the aqua regia was added in a 100mL digestion flask. The mixture was covered tightly and left overnight to digest. The mixture was placed in a water bath at 80°C for 30 minutes in a fume chamber, small portions of the aqua regia, about 2-5mL were added to the mixture to ensure that the solution did not dry out, until the digestion was judged to be complete (that is when the digest was light in colour and the solution was clear and has stopped producing fumes). The volume of the digest was reduced to 5mL. The digest was cooled to 4°C in an ice bath. Exactly 15mL of 5% $KMnO_4$ solution was added to the digest, followed by 8mL of 5% $K_2S_2O_4$ solution to oxidize all alkyl mercury present in the solution to the +2 state. The digest was returned to the water bath for additional 30 minutes of heating at 90°C. The sample was removed from the water bath, allowed to cool to room temperature and 6mL of 12% Hydroxylamine Hydrochloride solution was added to reduce the excess $KMnO_4$ in the solution. The resulting solution was filtered into a 100mL graduated flask and diluted to the mark with double distilled water and stored in the fridge for the AAS analysis.

Digestion of Blank for Mercury Analysis

Exactly 1.0mL double distilled water was measured into a 100mL digestion flask and 20mL of the aqua regia was added. The mixture was covered tightly and left overnight to digest. The mixture was placed in a water bath at 80°C for 30 minutes in a fume chamber, small portions of the aqua regia, about 2-5ml were added to the mixture to ensure that the solution did not dry out, until the digestion was complete (that is when the digest was light in colour and the

solution was clear and has stopped producing fumes). The volume of the digest was reduced to about 5mL. The digest was cooled to 4°C in an ice bath. 15mL of 5% $KMnO_4$ solution was added to the digest, followed by 8mL of 5% $K_2S_2O_4$ solution to oxidize all mercury present in the solution to the +2 state. The digest was returned to the water bath for additional 30 minutes heating at 90°C. The digest was removed from the water bath, allowed to cool to room temperature and 6mL of 12% Hydroxylamine Hydrochloride solution was added to reduce the excess $KMnO_4$ in the solution. The resulting solution was filtered into a 100mL graduated flask and diluted to the mark with double distilled water and stored in the fridge for the AAS analysis.

Sample Analysis

Mercury concentrations in the samples were determined using the Automatic Mercury Analyzer (HG5000), equipped with a mercury hollow cathode lamp which employed the cold vapour technique for the determination of mercury. The Flame Atomic Absorption Spectrophotometer (AAS) (Unicam 929) was also used for the determination of Cadmium and Zinc in the samples.

Operation of the Automatic Mercury Analyzer:

1. The instrument was switched on and left for 20 minutes to warm up
2. The start button was pressed to purge the system for 3 minutes
3. The reset button was pressed to stop purging
4. A 5mL aliquot of the sample was introduced into the reaction vessel using a micropipette

5. Then 0.5mL of the $SnCl_2.2H_2O$ was added from a dispenser
6. The start button was pressed immediately the stannous chloride was dispensed. After 30 seconds the 4 way valve rotated to allow the mercury vapour generated in the reaction vessel to flow into the absorption cell so as to generate a peak. Immediately the peak fell, a beep sounded
7. Immediately after the beep, the tap was opened to expel the waste
8. The reset button was pressed to stop purging after which the next sample was introduced
9. The tip of the micropipette was replaced with a new one
10. The process was repeated from step 4 for other samples, standard solutions and the blank.

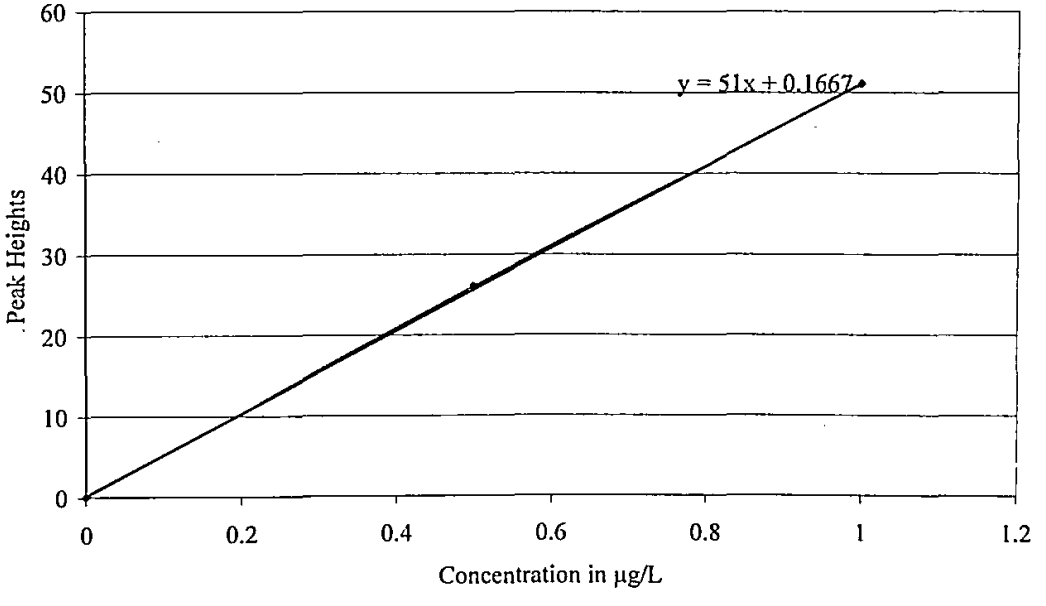

Fig 1a: A calibration curve showing peak heights observed for known concentrations of mercury.

The following peak height values were obtained; 0.0, 25 and 51 for 0.0, 0.5 and 1.0µg/L standard mercury solutions and the calibration curve shown in figure 1a above was plotted using the values obtained. The equation of the curve was used to calculate the concentration of mercury in the samples since the instrument measured the concentration in peak heights. From the equation y is the peak height therefore the concentration x in µg/L = (y-0.1667)/51.

The concentration in µg/L was converted to µg/g using the equation from USEPA (1992) as shown below:

$$\text{Mercury concentration in } \mu g/g = \frac{A \times B}{\text{Sample weight in g}}$$

Where A = concentration of mercury in the digested solution in µg/L

B = final volume of the digested solution in L.

Cadmium and Zinc Analysis

A blank solution was first used to zero the instrument after which the standard solutions of known concentrations of the metal of interest were aspirated in turns to obtain a calibration curve for the instrument after which the metal's concentrations in the samples were determined.

A blank solution of the samples was first aspirated into the Air-Acetylene flame of the instrument followed by the sample solutions containing the metal of interest. The aspirated solution was atomized in the Air-acetalene flame. The beam from the cathode lamp absorbed by the atomized metals is proportional to the concentration of the atoms in the path and was recorded by the instrument.

The concentration in mg/L was converted to µg/g using the equation from USEPA (1992) as shown below:

Metal concentration in μg/g = $\frac{A \times B}{\text{Sample weight in g}}$

Where A = concentration of metal in the digested solution in μg/L

B = final volume of the digested solution in L

CHAPTER FOUR

RESULTS AND DISCUSSION

In this chapter the levels of the mercury, cadmium and zinc recorded in cassava leaves, cocoyam leaves and taro are presented in tables and figures. The results obtained for the levels of mercury in the above mentioned crops from UCC, Agona junction, Tarkwa, Bankyim and Prestea are presented in table 5-9 (Appendix I) and figures 2, 3 and 4. The results for the levels of zinc in the same crops from the same sampling sites are presented in table 10-14 (Appendix I) as well as figures 5, 6 and 7. Similarly, the results obtained for the levels of cadmium are presented in table 15-19 (Appendix I) and figures 8, 9 and 10. The discussion of the results is in three parts: In the first part the result of the sample obtained for the samples from farms in the mining communities are compared with those from non-mining communities.

Secondly the results obtained for the samples from farms in mining communities and from farms in non-mining communities are compared with accepted standards from WHO and EPA to see if there is any variation in the levels of mercury, cadmium and zinc reported in the samples from the study area.

And finally a statistical tool SPSS is used to find the correlations between concentrations in samples of different vegetables from the same sample site. A T-Test is also use to find the difference in the means of the concentrations in the samples from the mining communities and non-mining communities and to see if

the difference in the means of concentrations recorded from the mining communities and from the non-mining communities are significant.

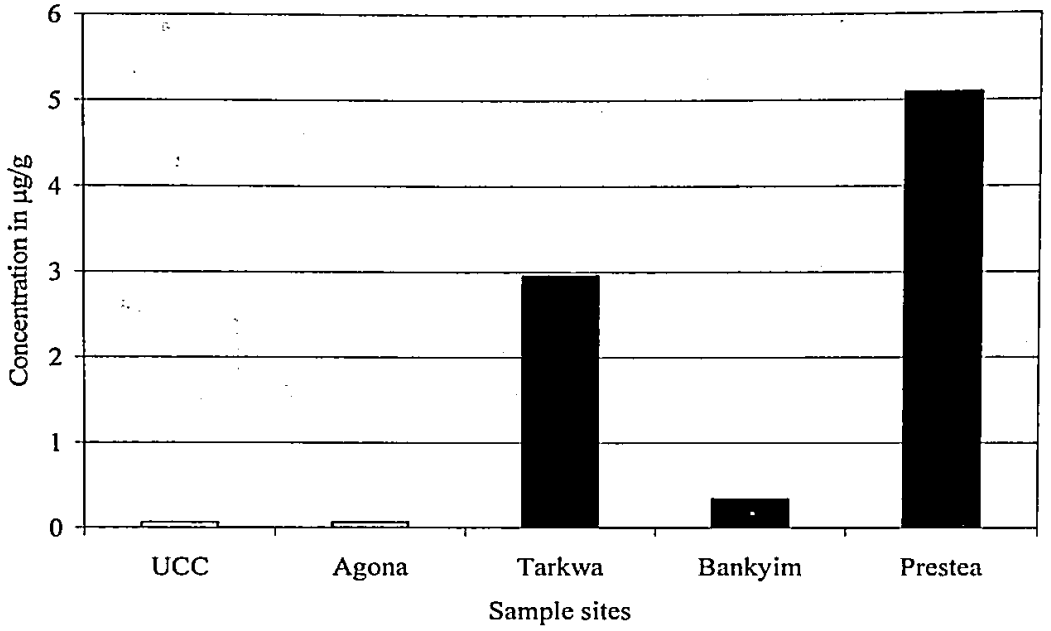

Fig 2: Variation of mercury in cassava leaves along the places sampled

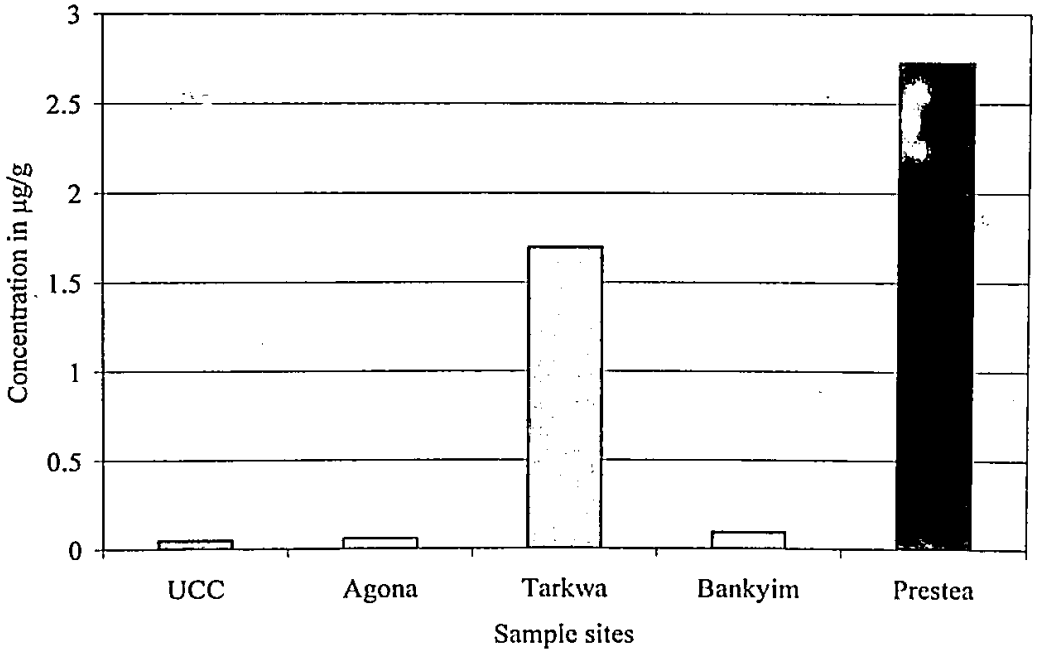

Fig 3: Variation of mercury in cocoyam leaves along the places sampled.

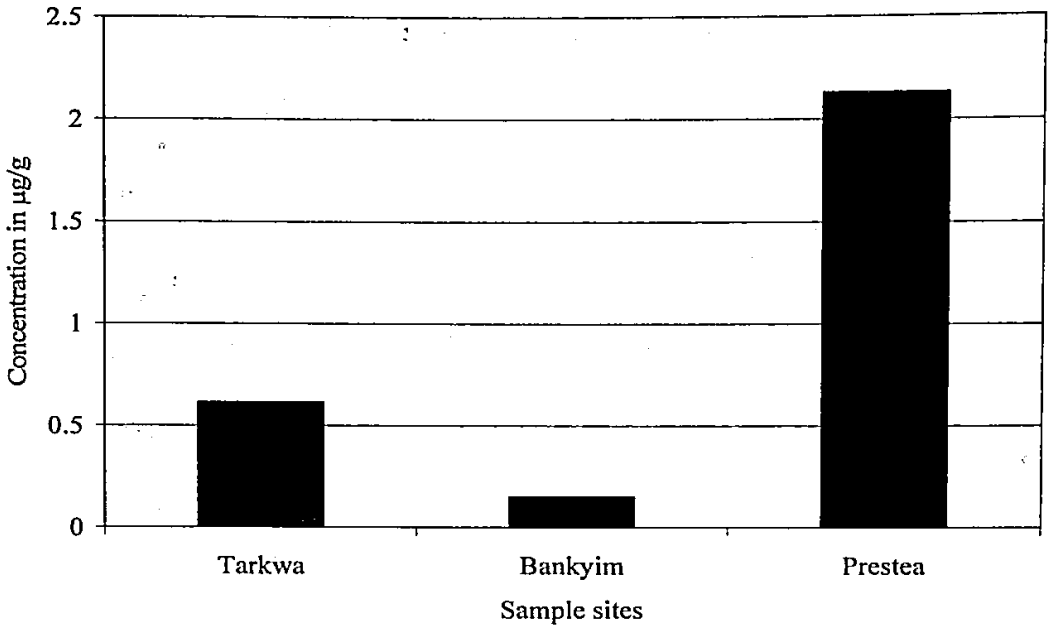

Fig 4: Variation of mercury in taro along the places sampled

Comparison of levels of Mercury in samples from mining communities with samples from non mining communities

The results of the analyses of mercury in the samples collected from Bankyim, Tarkwa and Prestea are depicted in figures 2 to 4 and tables 5 to 9 (appendix 1). From the figures it is clearly shown that the levels of mercury in all the samples were higher at Bankyim, Tarkwa and Prestea when compared with samples taken from UCC farm and Agona junction. The mean concentration in µg/g ranged from 0.0552 to 0.0722µg/g and 0.0448 to 0.0561µg/g as show in table 5 (Appendix I) for cassava leaf and cocoyam leaf samples from UCC.

Agona had concentrations from 0.0543 to 0.0986µg/g and 0.0464 to 0.0845µg/g as show in table 6 (Appendix I) for cassava leaf and cocoyam leaf samples respectively. For the same vegetable samples from Bankyim, Tarkwa and

Prestea the mean levels are as follows: Bankyim: 0.1248 to 0.07052µg/g and 0.0744 to 0.1169µg/g, table 7 (Appendix I) and the value for taro is from 0.0505 to 0.1063µg/g (table 7); Tarkwa: 1.5422 to 7.0578µg/g and 1.1028 to 2.9365µg/g (table 8, Appendix I) and taro is 0.3774 to 1.0281µg/g (table 8, Appendix I); Prestea: 2.7557 to 11.5525µg/g and 1.8858 to 5.0513µg/g (table 9, Appendix I) and taro is 1.8195 to 3.1342µg/g (table 9, Appendix I).

It is clear from the figures that the levels of mercury at Prestea were always the highest for the samples, an indication of increase disposal or deposit of mercury in the environment. Since most of the mercury comes from galamsey activities, the high levels of mercury in these samples compared to those from UCC and Agona Junction where the control samples were taken from can be linked to the mining activities taking place in these areas especially by the 'galamsey' operators. The diagrams proved the finding to be true that galamsey activities are predominant at Prestea. This is followed closely by Tarkwa an old mining town with galamsey activities but not as in Prestea. The levels at Agona and UCC are the natural levels since no galamsey activities are found there.

During the period of sampling it was realized that Prestea had the highest number of 'galamsey' operators followed by Tarkwa and a handful of about 3-5 were spotted at Bankyim. There was however no 'galamsey' operator spotted at Agona even though it has a history of mining operations during 1878-1920 by European companies (T.E. Anin 1989).

It was also found that Tarkwa had more mining companies than Prestea but the levels of mercury found in samples from Prestea were generally higher

than those from Tarkwa. It is therefore clear that most of the mercury in the environment comes from these 'galamsey' operations.

From the results it was also realized that cassava leaves had the highest concentration of mercury followed by cocoyam leaves. This could be explained by the fact that mercury is volatile, so apart from the mercury transported from the roots through the stem to the leaves there was also aerial adsorption of mercury by the leaves from the atmosphere. Even though the surface area of cocoyam leaf is larger than the surface area of cassava leaves, hence it is expected that if aerial adsorption of mercury contributed to the mercury in the samples then the values for cocoyam leaf samples should be higher than those of cassava leaves it is not so because the water content of cocoyam leaf is higher than that of cassava leaves. Another reason may be that cocoyam plants (the leaf of cocoyam is kotombre) are generally shorter than cassava plants therefore mercury vapour will settle on the cassava leaves first before getting to the cocoyam leaves.

The very high concentrations recorded in early April could be due to the fact that it was in the dry season so the temperature was high and therefore evaporation of mercury to the atmosphere was high therefore the plants adsorbed more. Since fresh samples were used the biomass of the sample increased because water was lost to the environment by the plant more in the dry season than in the raining season.

Comparison of levels of Mercury recorded in the samples with Standards

The normal concentration of mercury found in plant leaves (Alloway 1990), based on Bowen (1976) is between 0.005-0.17μg/g and the phytotoxic concentration is between 1-3μg/g. From the results presented in tables 5-9 (Appendix I) the mercury concentrations are within the ranges for normal and phytotoxic levels.

Cassava leaves, cocoyam leaves and taro are edible plants and because mercury can bioaccumulate as it move up the food chain, there is cause for concern regarding the level of mercury in these crops. This is because exposures to high levels of metallic, inorganic, or organic mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning result in: irritability, shyness, tremors, changes in vision or hearing, and memory problems.

Biochemical effects of excessive concentration of Hg in vegetable plants will lead to inhibition of protein synthesis; bonding to sulphydral groups and competition for sites with essential metabolites; changes in the permeability of cell membranes (Kabata-Pendias and Pendias 1984). In plant materials, organic and inorganic mercury compounds inactivate the spindle-fiber mechanism at cell division causing aneuploidy and/or polyploidy (WHO 1976).

Statistical Analysis of mercury results (T-Test)

From table 2 the results of the T-Test shows that the concentration of mercury in samples from mining communities are generally higher than in samples from non-mining communities and the difference is significant. There is

therefore cause for concern regarding contamination of vegetable crops grown in mining communities.

Table 2: T-Test results for concentrations of mercury in the samples

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair	[Hg] in Cas from Nm	.063519	36	.0195918	.0032653
1	- [Hg] in Cas from M	.707869	36	.6840828	.1140138

Paired Samples Correlations:		N	Correlation	Sig.
Pair	[Hg] in Cas from Nm			
1	and [Hg] in Cas from M	36	.256	.131

Paired Samples Test: Paired Differences			Std. Error	95%ConfidenceInterval	
		Mean	Stdv.	Mean	Lower	Upper
Pair	[Hg] in Cas					
1	from Nm					
	- [Hg] in Cas from M	-.6443500	.6793218	.1132203	-.8741994	-.4145006

Paired Samples Test:			t	df	Sig. (2-tailed)
Pair	[Hg] in Cas	- [Hg] in Cas			
1	from Nm	from M	-5.691	35	.000

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair	[Hg] in Kot from Nm	.055903	36	.0151644	.025274
1	[Hg] in Kot from M	1.213831	36	1.0819631	.1803272

Table 2 continued

Paired Samples Correlations:		N	Correlation	Sig.
Pair 1	[Hg] in Kot from Nm and [Hg] in Kot from M	36	-.644	.000

Paired Samples Test: Paired Differences			Std. Error Mean	95% Confidence Interval	
		Mean	Stdv.		Lower	Upper
Pair 1	[Hg] in Kot from Nm - [Hg] in Kot from M	-1.159583	1.0722638	.01787106	-1.5207602	-.7951565

Paired Samples Test:			t	df	Sig. (2-tailed)
Pair 1	[Hg] in Kot from M	- [Hg] in Kot from Nm	-6.480	35	.000

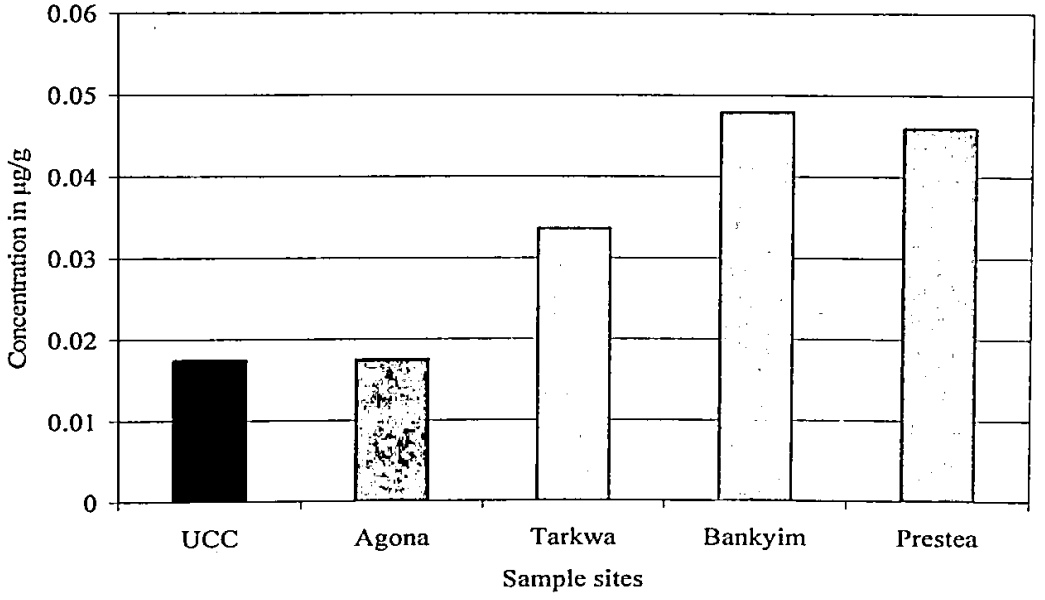

Fig 5: Variation of zinc in cassava leaves along the places sampled.

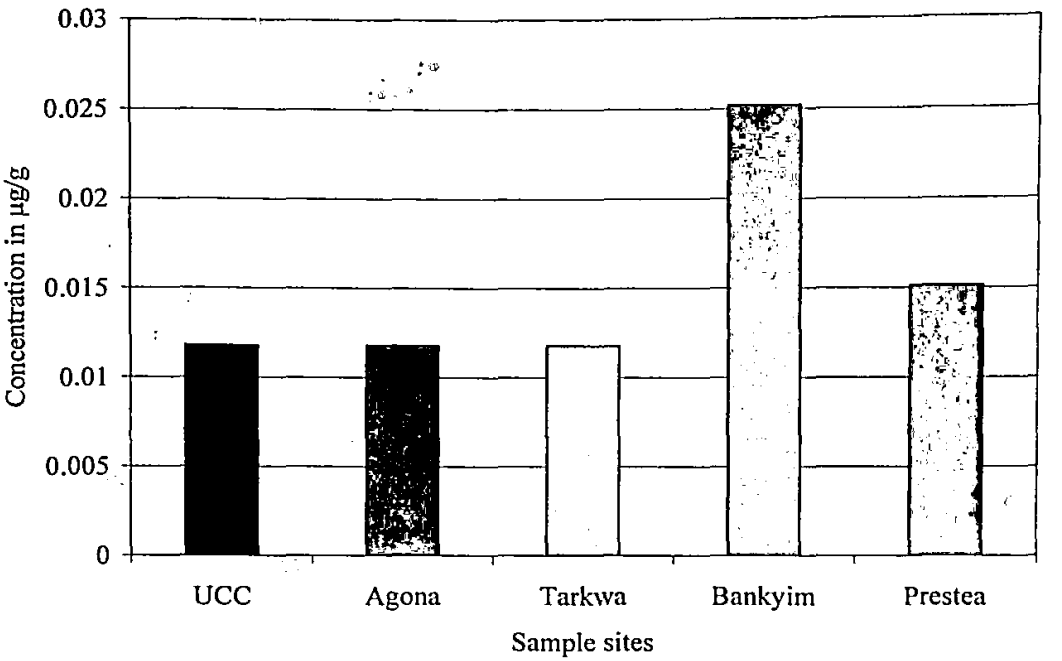

Fig 6: Variation of zinc in cocoyam leaves along the places sampled.

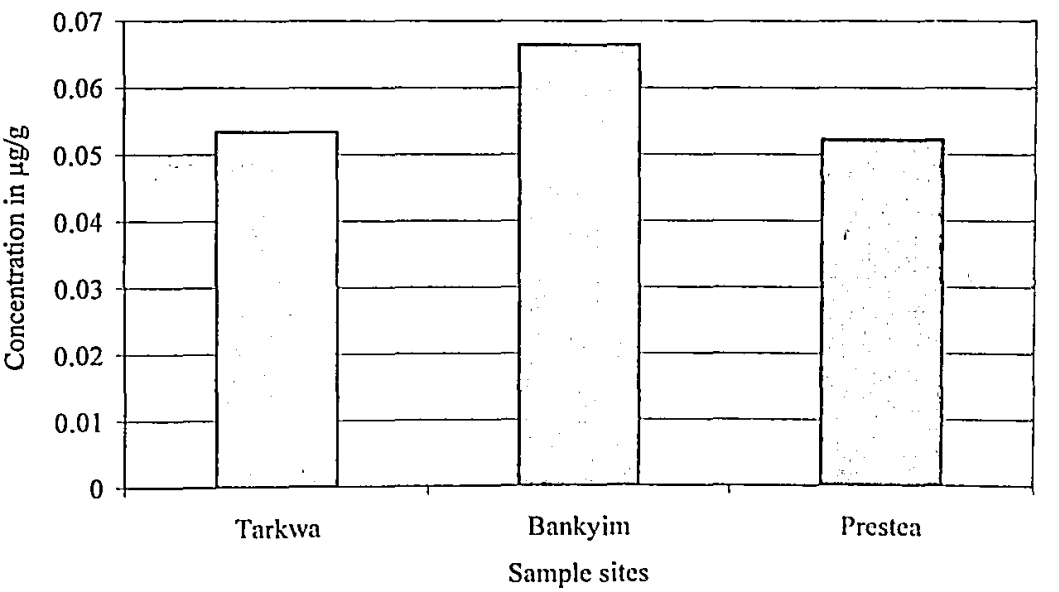

Fig 7: Variation of zinc in taro along the places sampled.

Comparison of levels of Zinc in samples from mining communities with samples from non mining communities

As shown in figures 5, 6, and 7 above the levels of zinc in the samples from the study area are generally higher as compared with the control samples from UCC and Agona. The mean concentration in µg/g for samples from UCC is 0.0175µg/g for cassava leaves and 0.0118µg/g for cocoyam leaves (as shown in Appendix I); Agona recorded mean concentrations ranging from 0.0128 to 0.0272µg/g for cassava leaves and 0.0108 to 0.0210µg/g for cocoyam leaves. For the same vegetable samples from Tarkwa, Bankyim and Prestea the following concentration ranges were recorded; Tarkwa: 0.0334 to 0.0434µg/g and 0.0195 to 0.0220µg/g, the level for taro is 0.0489 to 0.0555µg/g; Bankyim: 0.0301 to 0.0546µg/g and 0.0252µg/g, the range for taro is from 0.0588 to 0.0707µg/g; Prestea: 0.0454-0.0470µg/g and 0.0180-0.0265µg/g, the range for taro is from 0.4930 to 0.0555µg/g. The high levels of zinc in samples from the study area as compared to samples from UCC could be attributed to the release of zinc into the environment from large scale mining activities. Most of the mines in the study area use zinc dust in the recovery of gold. The contribution from mining activities to the environment is about 100% at the minimum and about 350% at the maximum of the levels of zinc found in samples from the control sample sites.

From the results it was realized that unlike in the case of mercury which recorded higher values in the leafy samples, the levels of zinc are higher in the taro samples. This showed that the major route for the uptake of zinc by the plant

is through the root. Rain removes zinc dust particles from the air. Zinc compounds can move into the soil, groundwater, into lakes, streams, and rivers.

Comparison of levels of Zinc recorded in the samples with Standards

The normal zinc concentration generally found in plant leaves is 1-400µg/g (Alloway, 1990 based largely on Bowen 1979) and the phytotoxic concentration is 100-400µg/g. From the results presented in figures 5, 6 and 7 above, the concentration of zinc in all the cassava and cocoyam leaf samples are within the normal concentration limits for plant leaves. Most of the zinc in soil stays bound to soil particles. It builds up in fish and other organisms, but it doesn't build up in plants. There is no cause for concern regarding the levels of zinc in the selected vegetable crops. This is because zinc is also an essential nutrient that is extremely important to long term health. Zinc is necessary for the functioning of various enzymes and plays an essential role in DNA, RNA and protein synthesis. The major symptoms of zinc deficiency are delayed growth and slow maturation (WHO 1996).

However in 1996, the WHO Expert Consultation Committee on Trace Elements recommended that the adult population mean intake of zinc should not exceed 45 mg/day in order to avoid zinc-related interactions (WHO 1996). Zinc is an essential element in our diet. Too little zinc can cause health problems, but too much zinc is also harmful. Recommended Dietary Allowance (RDA) is 15 milligrams a day for men (15 mg/day); 12 mg/day for women; 10 mg/day for children; and 5 mg/day for infants. EPA drinking water limit is 5 ppm. EPA also requires that releases of more than 1,000 (or in some cases 5,000) pounds of zinc

or its compounds into the environment be reported.

Harmful health effects generally begin at levels from 10-15 times the RDA (in the 100 to 250 mg/day range Rats that were fed large amounts of zinc became infertile or had smaller babies. In contrast, tolerable limits are usually set at higher levels than the RDIs and are set at a level below which toxic effects should not occur (i.e. Tolerable Limits are the upper health standard).

Statistical Analysis of zinc results (T-Test)

Table 3: T-Test results for concentrations of zinc in the samples.

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair 1	[Zn] in Cas from Nm	.017527	30	.0063535	.0011600
	[Zn] in Cas from M	.045077	30	.0154463	.0028201

Paired Samples Correlations:		N	Correlation	Sig.
Pair 1	[Zn] in Cas from Nm and [Zn] in Cas from M	30	-.399	.029

Paired Samples Test: Paired Differences			Std. Error	95% Confidence Interval	
		Mean	Stdv. Mean	Lower	Upper
Pair 1	[Zn] in Cas from Nm - [Zn] in Cas from M	-.0275500	.0189029 .0034512	-.0346085	-.0204915

Paired Samples Test:			t	df	Sig. (2-tailed)
Pair 1	[Zn] in Cas from Nm	[Zn] in Cas from M	-70983	29	.000

Table 3 continued

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair 1	[Zn] in Kot from Nm	.013267	30	.0043573	.0007955
	[Zn] in Kot from M	.020930	30	.0064905	.0011850

Paired Samples Correlations:		N	Correlation	Sig.
Pair 1	[Zn] in Kot from Nm and [Zn] in Kot from M	30	-.174	.357

Paired Samples Test: Paired Differences		Mean	Stdv.	Std. Error Mean	95% Confidence Interval Lower	Upper
Pair 1	[Zn] in Kot from Nm - [Zn] in Kot from M	-.0076633	.0084249	.0015382	-.0108092	-.0045174

Paired Samples Test:		t	df	Sig. (2-tailed)
Pair 1	[Zn] in Kot from Nm - [Zn] in Kot from M	-4.982	29	.000

From table 3 the results of the T-Test shows that the concentration of zinc in samples from mining communities are generally higher than in samples from non-mining communities and the difference is significant.

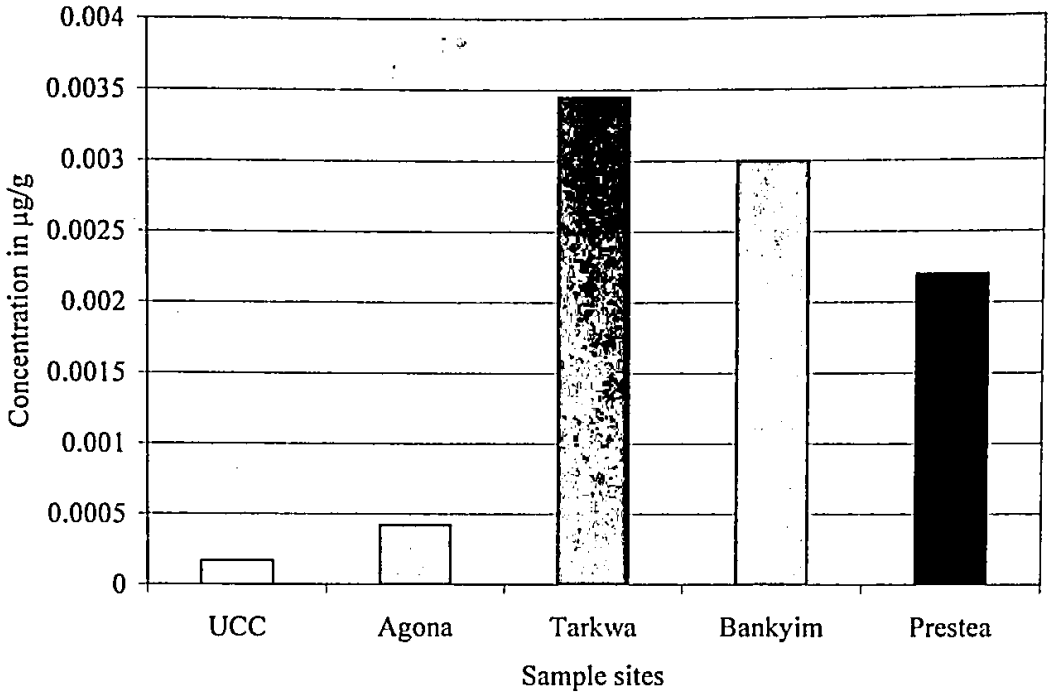

Fig 8: Variation of cadmium in cassava leaves along the places sampled.

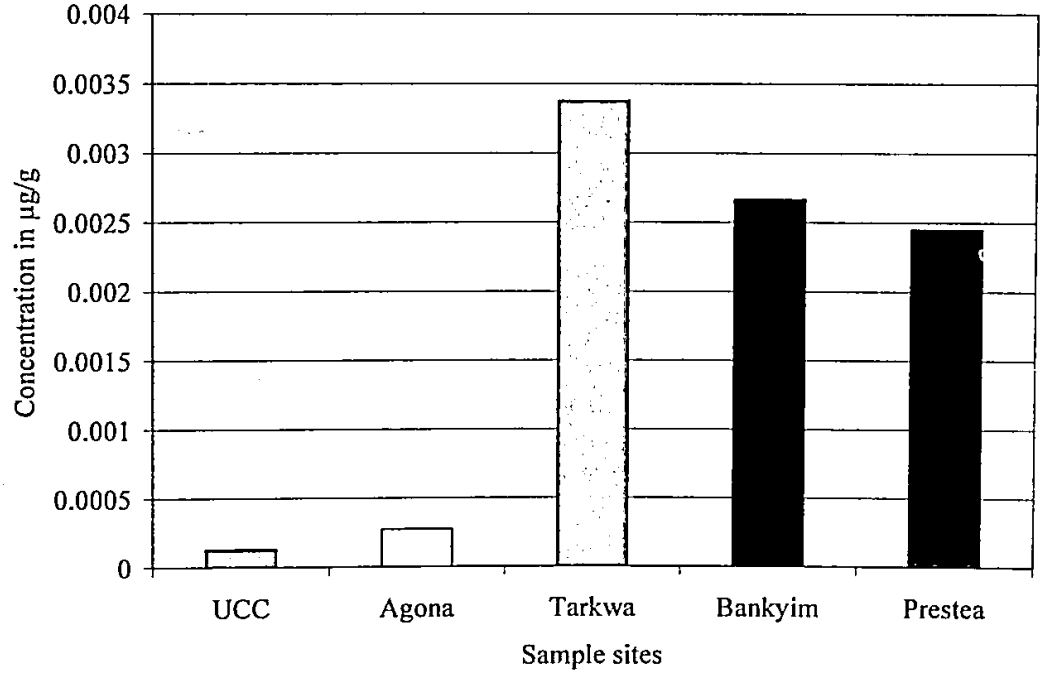

Fig 9: Variation of cadmium in cocoyam leaves along the places sampled.

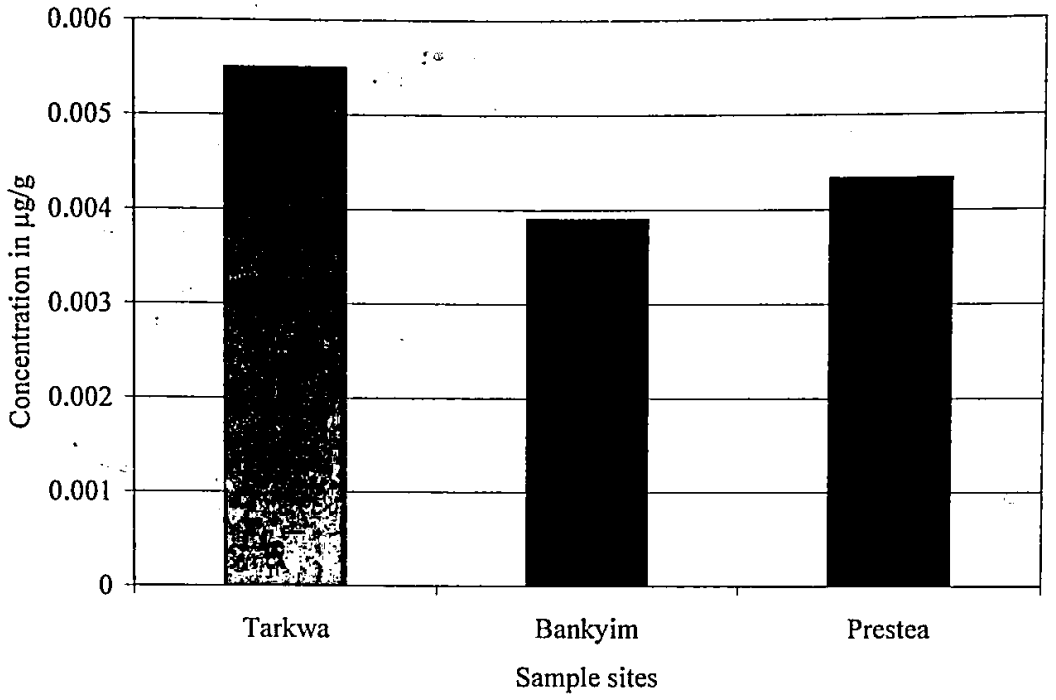

Fig 10: Variation of cadmium in taro along the places of sampled.

Comparison of levels of Cadmium in samples from mining communities with samples from non mining communities

As reported in the case of mercury and zinc, the levels of cadmium in the the samples from the mining communities as shown in tables 17-19 (Appendix I) are higher than samples from UCC and Agona (table 15 and 16, Appendix I). The variations can be clearly seen in figure 8, 9 and 10 for the various vegetable samples. Unlike mercury and zinc which are employed in the processing of gold cadmium is not. However cadmium is always an impurity in zinc hence its consideration in this work.

The mean concentrations in µg/g recorded for samples from UCC are 0.00017µg/g for cassava leaf samples and 0.00013µg/g for cocoyam leaf samples. At Agona Junction the mean concentration ranged from 0.0003 to 0.0005µg/g for

cassava leaf samples and 0.00023 to 0.00032 for cocoyam leaf samples. Samples from Tarkwa recorded mean concentrations from 0.00029 to 0.0044μg/g for cassava leaves, 0.0025 to 0.0043μg/g for cocoyam leaves and 0.0053-0.0059μg/g for taro corms. For the same samples from Bankyim the mean concentration ranges are from 0.0024 to 0.0035μg/g, 0.0028 to 0.0030μg/g and 0.0031 to 0.0048μg/g respectively and samples from Prestea recorded the following mean concentration ranges, 0.0019 to 0.0025μg/g, 0.0022 to 0.0029μg/g and 0.0041 to 0.0045μg/g.

Comparison of levels of Cadmium recorded in the samples with Standards.

The normal cadmium concentration generally found in plant leaves is 0.1-2.4μg/g (Alloway, 1990 based largely on Bowen 1979) and the phytotoxic concentration is 5-30μg/g. From the results presented in tables 15-19 (Appendix I) above, the concentration of cadmium in all the cassava and cocoyam leaf samples are within the normal concentration limits for plant leaves. It is widely accepted that approximately 2% to 6% of the cadmium ingested is actually taken up into the body (WHO 1992, ATSDR). Much of the cadmium which enters the body by ingestion comes from terrestrial foods. Some have estimated that 98% of ingested cadmium comes from terrestrial foods, while 1% comes from aquatic foods such as fish and shellfish, and 1% arises from cadmium in drinking water (Van Assche 1998). Because cadmium can bioaccumulate in the body, there is cause for concern regarding the levels in the selected vegetable crops.

The World Health Organization (WHO) has established a provisional tolerable weekly intake (PTWI) for cadmium at 7 μg/kg of body weight. At an

absorption rate of 5% from ingestion, the average person is believed to retain about 0.5 to 1.0 µg of cadmium per day from food.The tolerable limit for cadmium, which was set at the 33rd meeting, was maintained at the 55th meeting of the FAO/WHO Joint Expert Committee on Food Additives at 7 Âµg/kg bw/week (WHO 2001b).

Food and Drug Administration (FDA) limit in food colours: 15 ppm. Occupational Safety and Health Administration OSHA is planning to limit all cadmium compounds to 1 or 5mg/m^3 Breathing contaminated workplace air.

4.3.3 Statistical Analysis of cadmium results (T-Test)

Table 4: T-Test results for concentrations of cadmium in the samples

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair 1	[Cd] in Cas from Nm	.0000370	30	.0001896	.0000346
	[Cd] in Cas from M	.003020	30	.0008491	.0001550

Paired Samples Correlations:		N	Correlation	Sig.
Pair 1	[Cd] in Cas from Nm and [Cd] in Cas from M	30	-.224	.233

Paired Samples Test: Paired Differences			Std. Error	95% Confidence Interval	
		Mean	Stdv.	Mean	Lower	Upper
Pair 1	[Cd] in Cas from Nm - [Cd] in Cas from M	-.0026500	.0008274	.0001511	-.0029590	-.0023410

Table 4 continued

Paired Samples Test:		t	df	Sig. (2-tailed)
Pair 1	[Cd] in Cas from Nm - [Cd] in Cas from M	-17.542	29	.000

Paired Samples Statistics:		Mean	N	Stdv	Std. Error Mean
Pair 1	[Cd] in Kot from Nm	.000247	30	.0001074	.0000196
	[Cd] in Kot from M	.002987	30	.0009310	.0001700

Paired Samples Correlations:		N	Correlation	Sig.
Pair 1	[Cd] in Kot from Nm and [Cd] in Kot from M	30	.093	.626

Paired Samples Test: Paired Differences			Std. Error	95% Confidence Interval	
		Mean	Stdv.	Mean	Lower	Upper
Pair 1	[Cd] in Kot from Nm - [Cd] in Kot from M	-.0027400	.0009272	.0001693	-.0030862	-.0023938

Paired Samples Test:		t	df	Sig. (2-tailed)
Pair 1	[Cd] in Kot from Nm - [Cd] in Kot from M	-16.186	29	.000

From table 4 the results of the T-Test shows that the concentration of cadmium in samples from mining communities are generally higher than in samples from non-mining communities and the difference is significant.

CHAPTER FIVE

SUMMARY, CONCLUSON AND RECOMMENDATION

Summary of Mercury Results

The mean concentrations in all the cassava leaves from the mining communities were higher than the mean concentration of 0.0722 µg/g from UCC and Agona which served as a control for this analysis (mean 0.0552 and 0.0722 µg/g, range 0.0544 µg/g to 0.0979 µg/g) (Table 5, Appendix I). The concentrations of mercury in cassava leaves is greater than concentrations in cocoyam leaves from the same sample site; the mean concentration for cocoyam leaves from UCC was 0.0561µg/g (mean 0.0448 and 0.0561µg/g, range 0.0389 to 0.0649µg/g) (Table 5, Appendix I) and these are less than all the cocoyam leaf samples analyzed from the mining communities. The highest mercury concentration was recorded in cassava leaf samples from Prestea which was15.0040µg/g (Table 9, Appendix I). Even though taro was not found on the UCC farm at the time of sampling, samples of taro corms from Bankyim recorded the least concentration of mercury with a mean value of 0.0505, 0.0588, 0.1063, 0.0644; range 0.0389 to 0.1259µg/g (Table 7, Appendix I), again Prestea recorded the highest concentration of 2.1342µg/g (Tableb9, Appendix I). Statistical analysis of the result showed that the concentrations of mercury in the samples from the mining communities are significantly greater than concentrations found in the control samples.

Summary of Zinc Results

Zinc was detected in all the samples. The concentrations of zinc in the samples from the mining communities were all higher than samples from UCC and Agona. UCC samples recorded a mean of 0.0175 and 0.0118µg/g for cassava leaves and cocoyam leaves respectively (Table 10, Appendix I). Taro corm samples from Bamkyim recorded the highest concentrations with the highest being 0.0970µg/g (Table 12, Appendix I). In the survey, samples from Bankyim recorded the highest concentrations of zinc followed by Tarkwa, Prestea, UCC and Agona. The concentrations range from 0.0098-0.0338µg/g for cocoyam leaves; 0.0103-0.0715µg/g for cassava leaves and 0.0334-0.0970µg/g for taro corm samples from the mining communities. Some samples show relatively high concentrations. Statistical analysis of the result showed that the concentrations of zinc in the samples from the mining communities are significantly greater than that of the control samples. Concentrations of zinc in the leafy vegetables were much lower than those in the taro corm. Differences between the leafy samples and the corm samples show that the uptake of zinc by the plant is mainly through the root.

Summary of Cadmium Results

Cadmium was detected in all the samples. The concentrations of cadmium in the samples from the mining communities were all higher than samples from UCC and Agona. UCC samples recorded a mean of 0.00017 and 0.00013µg/g for cassava leaves and cocoyam leaves respectively (Table 15, Appendix I). The concentration range for samples from mining communities is from 0.0019-

0.0059µg/g. Statistical analysis of the result showed that the concentrations of cadmium in the samples from the mining communities are significantly greater than that of the control samples.

Conclusion

Apart from mercury, the levels of the elements cadmium, and zinc for cocoyam leaves, cassava leaves and taro do not exceed the appropriate tolerable level. Although level of mercury has exceeded the tolerable level, this value applies to total mercury, part of which is organic mercury and the other inorganic mercury. All forms of mercury are harmful therefore; it is considered that the concentrations of mercury in the samples are a cause for concern. There is no cause for alarm on the level of zinc and cadmium in the samples studied.

It is concluded that the concentrations of the elements cadmium, and zinc in cocoyam leaves, cassava leaves and taro do not provide any cause for concern for individuals eating these foods. However due to the high levels of mercury in these samples it is not safe for consumers to eat these vegetables. The aims of this survey were to produce data on concentrations for mercury cadmium and zinc in cocoyam leaves, cassava leaves and taro corm, to allow a comparison between samples from mining areas and samples from non mining communities; to compare the results with WHO, EPA and other standards. These aims were all achieved and the survey was successful.

Recommendation

The Tarkwa mining district (Tarkwa-Prestea) in the Wassa West district of

the Western Region was the most vulnerable and most impacted by mining activities because it has the highest concentration of mines in a single location in Africa. It is recommended that given the above unique situation in the Tarkwa region, studies should be conducted to ascertain the health effects associated with the consumption of these vegetables in the mining communities of Tarkwa.

Concentration and prolonged mining activities by both large and small-scale mining companies in the area have given rise to various environmental problems. They include serious mining related health problems, and social problems (such as prostitution, drug menace, high cost of living, inadequate shelter, etc). Physical environmental impacts in the area include polluted community water source and vegetable crops, noise pollution, virtual intrusion, active de-watering effects on groundwater sources, land, and forest and vegetation degradation. It is recommended that geoscientific benchmarks on various potential toxicants in mine wastewater, soils and stream sediments and from community water sources should be developed for the area, considering the cumulative impacts resulting in the activities of so many mines in the area.

Having seen the escalating result of mercury from Prestea and Tarkwa due to the activities of galamsey operators, it is recommended that galamsey operators should be taught how to use retorting technique to minimize the amount of mercury that get to the environment. To ensure that the galamsey operators use these retort mechanism, a law should be passed to enforce the use of retort machines.

There is a need for critical review of the mining environmental guidelines

to ensure environmental compliance by mining companies and galamsey operators.

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APPENDIX I

Table 5: Levels of Hg in µg/g in cassava leaves and kotombre from UCC farm.

1st sample		2nd sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0502	0.0979	0.0428	0.0565
0.0530	0.0933	0.0448	0.0546
0.0449	0.0463	0.0389	0.0548
0.0649	0.0702	0.0448	0.0546
0.0607	0.0712	0.0487	0.0565
0.0631	0.0544	0.0487	0.0546
m=0.0561	m=0.0722	m=0.0448	m=0.0552
s=0.0080	s=0.0205	s=0.0037	s=0.0010

Table 6: Levels of Hg inµg/g in cassava leaves and kotombre from Agona junction.

1st sample		2nd sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0369	0.0531	0.0577	0.0556
0.0467	0.0527	0.0492	0.0525
0.0665	0.0534	0.0448	0.0550
0.0456	0.0650	0.0497	0.0581
0.0507	0.0655	0.0367	0.0417
0.0426	0.0677	0.0403	0.0629
m=0.0478	m=0.0596	m=0.0464	m=0.0543
s=0.0103	s=0.0072	s=0.0075	s=0.0071

Table 6 continued			
3rd sample		4th sample	
kotombre	cassava leaves	kotombre	cassava
1.0993	0.0845	0.0546	0.0605
0.0852	0.0901	0.0546	0.0564
0.0821	0.0902	0.0565	0.0585
0.0824	0.0829	0.0546	0.0585
0.0753	0.1286	0.0546	0.0605
0.0826	0.0114	0.0546	0.0565
m=0.0845	m=0.0986	m=0.0549	m=0.0585
s=0.0845	s=0.0185	s=0.0008	s=0.0010

Table 7: Levels of Hg in µg/g in cassava leaves kotombre and taro from Bankyim.

1st sample			2nd sample	
kotombre	cassava leaves	taro corms	cassava leaves	taro corms
0.0621	0.2336	0.0598	0.1241	0.3021
0.0634	0.2150	0.0685	0.1193	0.3210
0.0812	0.1590	0.0561	0.2083	0.3100
0.0928	0.1332	0.0626	0.1204	0.4003
0.0962	0.0887	0.0612	0.1423	0.3349
0.0507	0.1548	0.0445	0.1350	0.5660
m=0.0744	m=0.1640	m=0.0588	m=0.1416	m=0.3774
s=0.0184	s=0.0533	s=0.0081	s=0.0339	s=0.0126
3rd sample			4th sample	
kotombre	cassava leaves	taro corms	cassava leaves	taro corms
0.1363	0.6060	0.0735	0.1565	0.0605

Table 7 continued				
0.1048	0.8130	0.1259	0.0977	0.0624
0.0963	0.7280	0.1073	0.0977	0.0644
0.0999	0.9490	0.1096	0.0722	0.0663
0.1538	0.6042	0.1122	0.0703	0.0683
0.1102	0.5310	0.1091	m=0.1248	m=0.0644
m=0.1169	m=0.7052	m=0.1063	s=0.0708	s=0.0028
s=0.0229	s=0.1562	s=0.0174		

Table 8: Levels of Hg in μg/g in cassava leaves, kotombre and taro from Tarkwa.

1st sample			2nd sample		
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
1.2530	1.6160	0.5276	0.9154	1.4980	0.3021
1.3370	1.8560	0.4723	0.9385	1.6920	0.3210
1.0120	1.7440	0.4104	1.1120	1.6730	0.3100
0.8950	1.5121	0.6043	1.0320	1.5760	0.4003
1.1220	1.5430	0.6310	1.0210	1.4320	0.3349
0.9980	1.5130	0.3981	1.2540	1.7550	05660
m=1.1028	m=1.6307	m=0.5073	m=1.0548	m=1.6043	m=0.3774
s=0.1674	s=0.1410	s=0.0976	s=0.1241	s=0.1240	s=0.0988

3rd sample			4th sample	
kotombre	cassava leaves	taro corms	cassava leaves	taro corms
2.2370	7.3860	1.2139	1.6075	0.9605
2.7820	7.3310	0.9824	1.6663	0.3918
4.4280	5.9330	0.7694	1.5095	0.3330

Table 8 continued				
2.6750	7.6640	1.0286	0.9997	0.8232
2.9450	6.2480	1.0400	1.9212	0.4310
2.5520	7.7850	1.1340	1.5487	0.3526
m=2.9365	m=7.0578	m=1.0281	m=1.5422	m=0.5487
s=0.7686	s=0.7745	s=0.1519	s=0.3029	s=0.2714

Table 9: Levels of Hg in μg/g in cassava leaves, kotombre and taro from Prestea.

1st sample			2nd sample		
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
2.0620	2.0870	1.6210	2.0620	1.8980	1.9680
2.1470	4.0270	2.1500	2.1470	3.5340	1.7930
2.4680	4.0270	1.5410	2.4680	2.2060	1.7650
1.3050	3.1990	2.0550	1.3050	2.8310	2.0230
1.8970	2.0580	1.8090	1.8970	2.0574	1.7160
2.2150	3.9110	1.9570	2.2150	4.0080	1.6520
m=2.0157	m=3.2182	m=1.8555	m=1.8858	m=2.7557	m=1.8195
s=0.3957	s=0.9396	s=0.2430	s=0.4306	s=0.8608	s=0.1455
3rd sample			4th sample		
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
5.4470	11.033	3.6870	1.9016	4.0585	1.7252
3.7160	15.004	3.2830	2.0389	2.7839	1.7840
3.8480	7.5980	2.8260	2.1369	2.5095	1.6663
5.8970	14.380	3.2180	1.9016	2.6075	1.8820
6.1820	9.0350	2.6390	1.9801	4.4114	1.8624
5.5180	12.2650	3.1520	1.7840	2.7448	1.3918

Table 9 continued					
m=5.0513	m=11.552	m=3.1342	m=1.9572	m=2.8526	m=1.7186
s=1.0180	s=2.9209	s=0.3675	s=0.1230	s=0.6071	s=0.1796

Table 10: Levels of Zn in µg/g in cassava leaves and kotombre from UCC farm.

kotombre	cassava leaves
0.0122	0.0090
0.0111	0.0179
0.0120	0.0190
0.0113	0.0210
0.0112	0.0180
0.0130	0.0198
m=0.0118	m=0.0175
s=0.0007	s=0.0039

Table 11: Levels of Zn in µg/g in cassava leaves and kotombre from Agona Junction.

1st sample		2nd sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0104	0.0116	0.0096	0.0278
0.0112	0.0145	0.0101	0.0108
0.0126	0.0107	0.0142	0.0147
0.0104	0.0116	0.0112	0.0167
0.0100	0.0145	0.0102	0.0175
0.0102	0.0170	0.0101	0.0138

Table 11 continued			
m=0.0108	m=0.0133	m=0.0109	m=0.0169
s=0.0010	s=0.0024	s=0.0017	s=0.0058
3rd sample		4th sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0148	0.0122	0.0260	0.0256
0.0109	0.0178	0.0241	0.0254
0.0118	0.0103	0.0205	0.0293
0.0118	0.0103	0.0164	0.0303
0.0104	0.0114	0.0205	0.0251
0.0113	0.0145	0.0185	0.0277
m=0.0118	m=0.0128	m=0.0210	m=0.0272
s=0.0015	s=0.0029	s=0.0035	s=0.0022

Table 12: Levels of Zn µg/g in kg in cassava leaves, kotombre and taro from Tarkwa.

1st sample		2nd sample		
kotombre	cassava leaves	kotombre	cassava leaves	taro corms
0.0122	0.0451	0.0246	0.0264	0.0402
0.0133	0.0204	0.0222	0.0242	0.0569
0.0304	0.0234	0.0116	0.0419	0.0663
0.0121	0.0245	0.0246	0.0422	0.0398
0.0325	0.0414	0.0171	0.0362	0.0407
0.0144	0.0458	0.0319	0.0532	0.0493
m=0.0195	m=0.0334	m=0.0220	m=0.0373	m=0.0489
s=0.0096	s=0.0119	s=0.0070	s=0.0109	s=0.0096

Table 12 continued				
3rd sample			4th sample	
kotombre	cassava leaves	taro corms	cassava leaves	taro corms
0.0234	0.0478	0.0578	0.0470	0.0471
0.0191	0.0321	0.0658	0.0570	0.0452
0.0181	0.0224	0.0598	0.0344	0.0624
0.0239	0.0457	0.0334	0.0348	0.0500
0.0160	0.0336	0.0681	0.0498	0.0607
0.0214	0.0377	0.0511	0.0376	0.0674
m=0.0204	m=0.0204	m=0.0560	m=0.0434	m=0.0555
s=0.0033	s=0.0033	s=0.0070	s=0.0092	s=0.0135

Table 13: Levels of Zn in µg/g in cassava leaves, kotombre and taro from Bankyim.

1st sample	2nd sample		
cassava leaves	kotombre	cassava leaves	taro corms
0.0321	0.0188	0.0344	0.0742
0.0291	0.0181	0.0582	0.0540
0.0275	0.0273	0.0686	0.0529
0.0305	0.0311	0.0511	0.0510
0.0352	0.0243	0.0715	0.0584
0.0264	0.0317	0.0434	0.0625
m=0.0301	m=0.0252	m=0.0545	m=0.0588
s=0.0032	s=0.0059	s=0.0144	s=0.0086

3rd sample		4th sample	
cassava leaves	taro corms	cassava leaves	taro corms
0.0615	0.0705	0.0292	0.0528
0.0367	0.0596	0.0615	0.0453

Table 13 continued			
0.0585	0.0772	0.0534	0.0489
0.0584	0.0606	0.0675	0.0575
0.0498	0.0595	0.0691	0.0392
0.0515	0.0970	0.0466	0.0619
m=0.0527	m=0.0707	m=0.0546	m=0.0699
s=0.0091	s=0.0147	s=0.0151	s=0.0135

Table 14: Levels of Zn in µg/g in cassava leaves, kotombre and taro from Prestea.

1st sample			2nd sample		
kotombre			kotombre	cassava leaves	taro corms
0.0185			0.0275	0.0417	0.0515
0.0171			0.0216	0.0448	0.0430
0.0196			0.0162	0.0282	0.0456
0.0104			0.0192	0.0557	0.0522
0.0176			0.0193	0.0516	0.0542
0.0246			0.0219	0.0512	0.0493
m=0.0180			m=0.0210	m=0.0454	m=0.0493
s=0.0046			s=0.0038	s=0.0098	s=0.0043
3rd sample			4th sample		
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
0.0191	0.0520	0.0534	0.0220	0.0305	0.0528
0.0164	0.0498	0.0434	0.0268	0.0432	0.0453
0.0169	0.0577	0.0611	0.0338	0.0374	0.0489
0.0230	0.0412	0.0536	0.0201	0.0679	0.0575

Table 14 continued					
0.0213	0.0350	0.0594	0.0268	0.0482	0.0392
0.0206	0.0465	0.0618	0.0297	0.0464	0.0619
m=0.0196	m=0.0470	m=0.0555	m=0.0265	m=0.0456	m=0.0520
s=0.0026	s=0.0081	s=0.0068	s=0.0050	s=0.0127	s=0.0082

Table 15: Levels of Cd in µg/g in cassava leaves and kotombre from UCC farm.

kotombre	cassava leaves
0.0001	0.0002
0.0002	0.0001
0.0001	0.0003
0.0002	0.0001
0.0001	0.0002
0.0001	0.0001
m=0.00013	m=0.00017
s=0.00005	s=0.00008

Table 16: Levels of Cd in µg/g in cassava leaves and kotombre from Agona Junction.

1st sample		2nd sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0003	0.0002	0.0002	0.0005
0.0002	0.0004	0.0004	0.0002
0.0003	0.0002	0.0003	0.0002
0.0002	0.0002	0.0001	0.0007

Table 16 continued			
0.0003	0.0003	0.0003	0.0002
0.0001	0.0005	0.0001	0.0004
m=0.00023	m=0.0003	m=0.00027	m=0.0004
s=0.00008	s=0.0001	s=0.00010	s=0.0002
3rd sample		4th sample	
kotombre	cassava leaves	kotombre	cassava leaves
0.0003	0.0004	0.0002	0.0005
0.0004	0.0005	0.0003	0.0004
0.0002	0.0005	0.0002	0.0003
0.0004	0.0003	0.0004	0.0006
0.0002	0.0007	0.0003	0.0007
0.0002	0.0006	0.0005	0.0006
m=0.00028	m=0.0005	m=0.00032	m=0.0005
s=0.00009	s=0.0001	s=0.00012	s=0.0001

Table 17: Levels of Cd in μg/g in cassava leaves, kotombre and taro from Tarkwa.

1st sample		2nd sample		
kotombre	cassava leaves	kotombre	cassava leaves	taro corms
0.0018	0.0019	0.0041	0.0062	0.0047
0.0011	0.0013	0.0029	0.0033	0.0057
0.0043	0.0047	0.0026	0.0027	0.0037
0.0033	0.0023	0.0032	0.0032	0.0065
0.0021	0.0044	0.0021	0.0009	0.0042
0.0022	0.0042	0.0033	0.0012	0.0038

Table 17 continued					
m=0.0025	m=0.0031		m=0.0030	m=0.0029	m=0.0053
s=0.0011	s=0.0015		s=0.0007	s=0.0019	s=0.0008
	3rd sample			4th sample	
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
0.0026	0.0039	0.0047	0.0039	0.0034	0.0068
0.0049	0.0046	0.0057	0.0026	0.0049	0.0056
0.0047	0.0027	0.0037	0.0027	0.0026	0.0048
0.0022	0.0020	0.0065	0.0072	0.0046	0.0063
0.0044	0.0042	0.0042	0.0057	0.0054	0.0054
0.0035	0.0027	0.0043	0.0034	0.0057	0.0066
m=0.0037	m=0.0034	m=0.0053	m=0.0043	m=0.0044	m=0.0059
s=0.0011	s=0.0010	s=0.0010	s=0.0018	s=0.0012	s=0.0008

Table 18: Levels of Cd in µg/g in cassava leaves, kotombre and taro from Bankyim.

1st sample	2nd sample		
cassava leaves	kotombre	cassava leaves	taro corms
0.0028	0.0032	0.0032	0.0034
0.0015	0.0015	0.0026	0.0024
0.0024	0.0036	0.0023	0.0045
0.0028	0.0024	0.0028	0.0031
0.0025	0.0031	0.0032	0.0012
0.0026	0.0027	0.0035	0.0036
m=0.0024	m=0.0028	m=0.0029	m=0.0031
s=0.0005	s=0.0007	s=0.0004	s=0.0010

Table 18 continued				
3rd sample		4th sample		
cassava leaves	taro corms	kotombre	cassava leaves	taro corms
0.0021	0.0052	0.0026	0.0039	0.0062
0.0035	0.0053	0.0021	0.0024	0.0048
0.0040	0.0041	0.0033	0.0033	0.0036
0.0033	0.0021	0.0028	0.0044	0.0046
0.0027	0.0062	0.0039	0.0036	0.0041
0.0033	0.0038	0.0031	0.0031	0.0054
m=0.0032	m=0.0045	m=0.0030	m=0.0035	m=0.0048
s=0.0007	s=0.0014	s=0.0006	s=0.0007	s=0.0009

Table 19: Levels of Cd in µg/g in cassava leaves, kotombre and taro from Prestea.

1st sample			2nd sample		
kotombre			kotombre	cassava leaves	taro corms
0.0016			0.0022	0.0021	0.0046
0.0021			0.0025	0.0024	0.0038
0.0022			0.0019	0.0019	0.0041
0.0032			0.0026	0.0011	0.0039
0.0018			0.0014	0.0022	0.0043
0.0022			0.0032	0.0017	0.0037
m=0.0022			m=0.0023	m=0.0019	m=0.0041
s=0.0006			s=0.0006	s=0.0005	s=0.0003
3rd sample			4th sample		
kotombre	cassava leaves	taro corms	kotombre	cassava leaves	taro corms
0.0021	0.0023	0.0054	0.0029	0.0031	0.0053
0.0027	0.0027	0.0035	0.0036	0.0021	0.0049

Table 19 continued					
0.0028	0.0018	0.0037	0.0022	0.0026	0.0047
0.0025	0.0021	0.0042	0.0031	0.0023	0.0052
0.0023	0.0019	0.0048	0.0025	0.0020	0.0035
0.0019	0.0021	0.0051	0.0032	0.0029	0.0045
m=0.0024	m=0.0022	m=0.0044	m=0.0029	m=0.0025	m=0.0044
s=0.0003	s=0.0003	s=0.0008	s=0.0005	s=0.0004	s=0.0008