

UNIVERSITY OF GHANA

COLLEGE OF BASIC AND APPLIED SCIENCES

HUMAN HEALTH RISK ASSESSMENT OF EXPOSURE TO HEAVY METALS

IN SELECTED MINING COMMUNITIES IN GHANA.

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DECLARATION

This thesis is the result of research work undertaken by Samuel Obiri in the Department of Nuclear Science and Applications, Graduate School of Nuclear and Allied Sciences, University of Ghana, under the supervision of Prof. Shiloh Osae, Prof. P. O. Yeboah and Dr. Sam Adu-kumi.



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ABSTRACT

Gold mining is an important economic activity in Ghana. However, processing and extraction of gold from its ores generate a lot of wastes. Studies conducted in different countries have established that long-term exposure to As, Cd, Pb and Hg in the wastes could cause cancer and other non-cancerous diseases such as cardiovascular diseases. It is within this context that, this thesis assessed cancer and non-cancer human health risk associated with exposure to As, Cd, Hg and Pb in water and sediment via oral and dermal contact by receptors such as adults workers in mining companies or artisanal small-scale mines (ASM), resident adults (i.e., who do not work in either mining companies or ASM) and resident children in selected mining communities in Ghana. The study further evaluated the health risk associated with ingestion of food crops such as cassava grown in soils the study areas as well as the determination of levels of As, Cd, Pb and Hg in human such as whole blood and serum among resident adults (non-mine workers i.e., workers of mining companies or ASM), and resident children in selected mining and non-mining communities in Ghana in line with USEPA risk assessment guidelines as there exist limited information on this subject in Ghana.

The study protocol was approved by the Ghana Health Service Ethical Review Committee. Random sampling techniques were adopted in selecting the sampling sites as well as recruitment of respondents who donated their blood samples as well as provided information about their personal health in relation to cardiovascular diseases. The collected water, sediment and cassava samples were stored under 4°C and conveyed to the CSIR-Water Research Institute laboratory for analysis.

The blood samples were collected by laboratory staff of Tarkwa Nsuaem Municipal Hospital, Cape Coast metropolitan Hospital, Health Centre at Bole and Nangodi respectively. The blood samples were separated into whole blood and serum by the laboratory staff and conveyed to Ghana Atomic Energy Commission for analysis of As, Cd and Hg using the NAA at Ghana Reactor 3 under -4°C ; while the Pb levels in the blood samples were determined using GFAAS at Heavy Metals Laboratory of CSIR-Water Research Institute.

The mean concentrations for As, Hg, Cd and Pb in whole blood of resident adults from mining communities in Tarkwa Nsuaem Municipality were; $38 \pm 0.20 \mu\text{g/L}^{-1}$, $63 \pm 0.23 \mu\text{g/L}^{-1}$, $303 \pm 117 \mu\text{g/L}^{-1}$ and $28 \pm 14 \mu\text{g/L}^{-1}$ respectively; while the levels of As, Hg, Cd and Pb measured in serum of resident adults from mining communities were; $65 \pm 14 \mu\text{g/L}^{-1}$, $358 \pm 22 \mu\text{g/L}^{-1}$, $134 \pm 12 \mu\text{g/L}$, $58 \pm 5.8 \mu\text{g/L}^{-1}$ respectively for As, Hg, Cd and Pb. These were found to have exceeded the permissible WHO guideline values. The mean concentrations of As, Cd, Hg and Pb in water samples ranged from $15 \mu\text{g/L}$ to $325 \mu\text{g/L}$ (As), $0.17 \mu\text{g/L}$ to $340 \mu\text{g/L}$ (Cd), $0.17 \mu\text{g/L}$ to $122 \mu\text{g/L}$ (Pb) and $132 \mu\text{g/L}$ to $866 \mu\text{g/L}$ (Hg) respectively. These measured concentrations of arsenic (As), mercury (Hg), cadmium (Cd) and lead (Pb) were used as input parameters to calculate the cancer and non-cancer health risks from exposure to these metals in surface water bodies and sediments based on an occupational exposure scenario using Central Tendency Exposure (CTE) and Reasonable Maximum Exposure (RME) parameters. The results of the non-cancer human health risk assessment for small-scale miners working around river Anikoko were expressed in terms of hazard quotients based on CTE parameters are as follows: 0.04 (Cd), 1.45 (Pb), 4.60 (Hg) and 1.98 (As); while cancer health risk faced by ASGM miners in Dumase exposed to As in

River Mansi via oral ingestion of water is 3.1×10^{-3} . The hazard quotient results obtained from this study in most cases were above the HQ guidance value of 1.0. Furthermore the cancer health risk results were found to be higher than the USEPA guidance value of 1×10^{-4} to 1×10^{-6} . The findings from case-control epidemiological studies established a strong relationship between exposure to As in drinking water and incidence of cardiovascular diseases among residents in the study areas. The results from this study calls for strong policy interventions to address the issue of health and safety of both mine workers and residents of mining communities in Ghana.

DEDICATION

This thesis is dedicated to the following:

- the Almighty God whose divine protection and guidance helped me to undertake this study;
- my children namely; Daniel Herbert Obiri, Awura Adwoa Oforiwaah Obiri and Nana Akua Gyiesiwah Obiri for their patience, prayers, support and words of encouragement in difficult times;
- my mother Mrs. Comfort Oforiwaah Obilie for supporting my education; and
- the memory of my late father Very Rev. Daniel K. Obilie of The Methodist Church, Ghana whose dream for me was to attain a PhD degree.

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To my mother, I say I big thank you for all that you done for me.

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LIST OF ACRONYMS

AF	Sediment-skin Adherence Factor
AAF _{so}	Chemical specific oral–sediment absorption factor
AAF _{sd}	Chemical specific dermal-water absorption factor
AAF _{wo}	Chemical specific oral-water absorption factor
AAF _{wd}	Chemical specific dermal-water absorption factor
AAS	Atomic Absorption Spectrophotometer
ADD	Average Daily Dose
ANOVA	Analysis of variance
AR	Analytical Grade
AWWA	American Water Works Association
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
ASGM	Artisanal small-scale gold mines
BBD	Bole-Bamboi District
BW	Body weight

CA	Cluster Analysis
CDC	Centre for Disease Control
CCMA	Cape Coast Metropolis
CSF	Cancer Slope Factors
CTE	Central Tendency Exposure
COC	Chemical of Concern
DA	Dermal Absorption Fraction
EC	Electrical Conductivity
ED	Exposure Duration
EF	Exposure Frequency
EPC	Exposure Point Concentration
ERP	Economic Recovery Programme
FDI	Foreign Direct Investment
FS	Fraction of Source
GDP	Gross Domestic Product
GHEITI	Ghana Extractive Industry Transparency Initiative

GSS	Ghana Statistical Service
HI	Hazard Index
HQ	Hazard Quotient
IARC	International Agency Research on Cancer
IDW	Inverse Distance Weighted Interpolation
IR	Ingestion Rate
Kp	Skin Permeability Coefficient
ND	Nabdam District
PCA	Principal Component Analysis
PHVD	Prestea Huni Valley District
TNMA	Tarkwa Nsuaem Municipality Assembly
UF	Uncertainty Factor
USEPA	United States Environmental Protection Agency
WHO	World Health Organization

TABLE OF CONTENTS

Declaration.....	<u>ii</u>
Abstract.....	<u>iii</u>
Dedication.....	<u>vi</u>
Acknowledgement	vii
List of Acronyms	ix
Table of Contents.....	xii
List of Tables	xvii
List of Figures.....	xx
Chapter One - Introduction	1
1.0 General Introduction.....	1
1.1 Importance of gold mining to Ghana’s socio-economic development	2
1.2 Gold deposit in Ghana	4
1.3 Problem statement.....	6
1.4 Research hypothesis.....	9
1.5 Research question	10
1.6 Objectives and scope of the study.....	10
Chapter Two - Literature Review	12
2.0 Gold mining, processng and extraction techniques in Ghana.....	12
2.1 Characteristics and environmental pollution associated with gold mine ailings	15
2.2 Status and trends of As, Cd, Hg and Pb contamination in ground water.....	18
2.3 Status and trends of As, Cd, Hg and Pb contamination in surface water	22

2.4 Status and trends of as, Cd, Hg and Pb contamination in sediments, nearby riverine soils and food crops grown in mining communities in Ghana.....	25
2.5 Status and trends of levels of As, Cd, Hg and Pb in human bio-samples from Ghana mining communities	28
2.6.0 Concepts of human health risk assessment	30
2.6.1 Hazard Identification for As, Cd, Hg and Pb	34
2.6.2 Dose-Response Assessment of As, Cd, Hg and Pb	37
2.6.3 Status and trends of human health risk assessment in mining communities in Ghana	40
2.7.0 Neutron Activation Analysis (NAA)	42
2.7.1 Basic Principles of NAA	43
2.7.2 Advantages of NAA	47
2.7.3 Disadvantages of NAA.....	48
2.8 Review of other analytical techniques used in the determination of heavy metals in environmental and bio-samples	49
Chapter Three - Methodology	51
3.0 General description and justification for selecting of the study sites	51
3.1.0 The Study Areas	52
3.1.1.0 Socio-demographic and climate of Tarkwa Nsuaem Municipal Assembly (TNMA)	52
3.1.1.1 Geology and hydrogeology of TNMA	54
3.1.1.2 Sample locations and Sampling Technique in TNMA	55
3.1.2.0 Socio-demographic and climate of Prestea Huni Valley District (PHVD)	57
3.1.2.1 The geology and hydrogeology of the PHVD	58
3.1.2.2 Sample locations and Sampling Technique in PHVD	60
3.1.3.0 Socio-demographic and climate of Cape Coast Metropolitan Assembly (CCMA)	61.

3.1.3.1 Geology and Hydrogeology of CCMA	62
3.1.3.2 Sample locations and Sampling Technique in CCMA	63
3.1.4.0 Socio-demographic and climate of Bole-Bamboi District Assembly (BBD)	63
3.1.4.1 Geology and Hydrogeology of BBD	64
3.1.4.2 Sample locations and Sampling Technique in BBD	65
3.1.5.0 Socio-demographic and climate of Nabdam District (ND)	66
3.1.5.1 Geology and Hydrogeology of ND	66
3.1.5.2 Sample locations and Sampling Technique in ND	67
3.2.0 Collection, preservation of water, sediment and cassava samples.....	69
3.2.1 Collection, preservation and storage of water samples.....	69
3.2.2 Collection, preservation and storage of sediment samples.....	69
3.2.3 Collection, preservation and storage of cassava samples.....	70
3.3.0 Reagents and chemicals used in the digestion/analysis of As, Cd, Pb and Hg in the samples.....	70
3.4.0 Digestion of environmental samples	73
3.4.1 Digestion of As, Cd, Pb and Hg in water samples.....	73
3.4.2 Digestion of As, Cd, Pb and Hg in sediment samples.....	74
3.4.3 Digestion of As, Cd, Pb and Hg cassava samples.....	75
3.5.0 Determination of pH, Turbidity, Conductivity and TDS	76
3.5.1 pH	76
3.5.2 Turbidity	77
3.5.3 Conductivity	77
3.5.4 Total Dissolved Solids (TDS)	77

3.6.0 Analysis of digested samples	78
3.6.1 Cold Vapour Technique for analysis of Hg in the digested samples	78
3.6.2 Analysis of Cd and Pb in the digested samples using Flame AAS.....	79
3.6.3 Analysis of As in the digested samples using the Arsine Hydride Generator coupled with Flame AAS	79
3.7 Quality Control	80
3.8.0 Sampling techniques, collection, preservation, storage and analysis of blood samples.....	81
3.8.1 Ethical Clearance for the sample collection	81
3.8.2 Exclusion and Inclusion Criteria	82
3.8.3 Sampling Techniques for the collection of the human bio-samples	82
3.8.4 Collection of whole blood and serum	84
3.8.5 Analysis of As, Cd and Hg in blood samples using NAA.....	85
3.8.5.1 Description of GHARR - 1	85
3.8.5.2 Determination of As, Cd and Hg in whole blood and serum using NAA techniques ...	86
3.8.5.3 Energy and Efficiency Calibration of HPGe	87
3.8.5.4 Quality Assurance for the analysis of heavy metals using NAA.....	88
3.8.6 Determination of Pb in whole blood and serum using Graphite Furnace AAS.....	89
3.8.7 Analysis of socio-economic collected data from donors of blood samples.....	89
3.8.8 Data treatment.....	89
3.9 Exposure Assessment and Risk Characterisation	91
3.9.1 Exposure Assessment	91
3.9.2 Risk Characterisation	95
3.9.2.1 Calculation of carcinogenic and non-carcinogenic health risk	95

Chapter Four – Results and Discussion	98
4.0 Concentration and seasonal variations of physico-chemical parameters water samples from the study areas.....	98
4.1 Concentration and seasonal trends of As, Cd, Pb and Hg in water samples	107
4.2 Concentration of As, Cd, Pb and Hg in sediment samples	115
4.3 Spatial variations of concentrations of As, Cd, Pb and Hg in water samples from the study areas	118
4.4 Concentration of As, Cd, Pb and Hg in food crops grown in the study areas	121
4.5 Concentrations and variations of heavy metals in human blood samples	123
4.6.0 Summary of carcinogenic health risk faced by all receptors in the study areas	135
4.6.1 Cancer health risk results faced by ASGM workers in the study areas	135
4.6.2 Cancer health risk results faced by resident adult non-miners in the study areas	140
4.6.3 Summary of non-carcinogenic health risk faced by all receptors in the study areas	146
4.7 Association between exposure to As, Hg and Pb in water, human blood and incidence of cardiovascular diseases among residents in the study areas.....	149
Chapter Five – Conclusion and Recommendation	155
5.0 Conclusion	155
5.1 Recommendation	156
Reference	158
Appendices.....	188

LIST OF TABLES

Table 2.0: Comparison of Analytical Instruments used in the determination of heavy metals in samples	50
Table 3.0: Sampling Locations in TNMA	56
Table 3.1: Sampling Locations in PHVD	60
Table 3.2: Sampling Locations in CCMA	63
Table 3.3: Sampling Locations in Tinga in BBD	65
Table 3.4: Sampling Locations in Nangodi in ND	67
Table 3.5: Instrument parameters for Flame and Cold Vapour AAS	80
Table 3.6: Percentage of As, Hg, Cd and Pb recovered from 10 µg/L solution during the reproducibility studies	81
Table 3.7: GHARR - 1 Characteristics	85
Table 3.8: Irradiation and counting schemes	86
Table 3.9: Receptor and intake parameters for receptors used in this study	97
Table 4.00: Descriptive statistics of physico-chemical TNMA for wet and dry season.....	98
Table 4.01: Descriptive statistics of physico-chemical PHVD for wet and dry season	100
Table 4.02: Pearson's product-moment correlation coefficient between water quality parameters from TNMA	101
Table 4.03: Pearson's product-moment correlation coefficient between water quality parameters from PHVD.....	101

Table 4.04: Total variance from TNMA.....	102
Table 4.05: Total variance from PHVD.....	103
Table 4.06: Descriptive statistics of physico-chemical CCMA for wet and dry season.....	104
Table 4.07: Pearson’s product-moment correlation coefficient of water samples from CCMA	104
Table 4.08: Descriptive statistics of physico-chemical Nangodi in ND for wet and dry season	105
Table 4.09: Descriptive statistics of physic-chemical Tinga in BBD for wet and dry season....	105
Table 4.10: Mean levels of As, Cd, Pb and Hg in water - TNMA for wet and dry seasons.....	107
Table 4.11: Pearson’s product-moment correlation coefficient heavy metals for dry season TNMA	108
Table 4.12: Mean levels of As, Cd, Pb and Hg in water - PHVD for wet and dry seasons.....	109
Table 4.13: Pearson’s product-moment correlation coefficient heavy metals wet season - PHVD	110
Table 4.14: Mean levels of As, Cd, Pb and Hg in samples from CCMA for wet and dry seasons	111
Table 4.15: Mean levels of As, Cd, Pb and Hg in samples from ND for wet and dry seasons ..	112
Table 4.16: Mean levels of As, Cd, Pb and Hg in samples from BBD for wet and dry seasons	113
Table 4.17: Levels of As, Cd, Pb and Hg in sediments from TNMA/PHVD.....	116
Table 4.18: Pearson’s product-moment correlation coefficient heavy metals in sediment samples from TNMA/PHVD	116
Table 4.19: Mean levels of As, Pb, Cd and Hg in cooked cassava samples in the study areas ..	122

Table 4.20: Demographic characteristics of the blood donors in this study	125
Table 4.21: Mean As, Cd, Pb and Hg levels in whole blood of resident adults (non-miners) from mining and non-mining communities in Ghana.....	127
Table 4.22: Mean As, Cd, Pb and Hg levels in serum of resident adults (non-miners) from mining and non-mining communities in Ghana	127
Table 4.23: Mean concentration of heavy metals in resident children of mining and non-mining areas serum (plasma).....	128
Table 4.24: Mean concentration of heavy metals in the whole blood of resident children of mining and non-mining areas.....	129
Table 4.25: Whole blood As, Cd, Pb and Hg concentration among mine workers (ASGM and mining companies) classified by their work section in the study areas.....	131
Table 4.26: Blood serum As, Cd, Pb and Hg concentration among mine workers (ASGM and mining companies) classified by their work section in the study areas.....	133
Table 4.27: Cancer health risk results from ingestion of arsenic in cooked cassava in the study areas	146
Table 4.28: Demographic characteristics of respondents with CVD cases as well as the controls	151
Table 4.29: Adjusted odds ratios for cardiovascular diseases, water and blood pressure	153

LIST OF FIGURES

Fig. 2.0: Two key elements in the definition of human health risk assessment	30
Fig. 2.1: The four basic steps involved in human risk assessment process	33
Fig. 2.2: Diseases associated with exposure to As in environmental media	36
Fig. 2.3: Mode of determination of concentration of elements in samples using NAA	44
Fig. 3.0: Map of Ghana showing major gold deposits	51
Fig. 3.1: Map showing sampling sites in TNMA, PHVD and CCMA	51
Fig. 3.2: Map showing sampling sites in ND and BBD	61
Fig. 3.3: Energy calibration of HPGe detector	68
Fig. 4.0: A comparison of pH, Turbidity, Colour, TDS, Conductivity and TSS in water samples from the study areas for dry season.	106
Fig. 4.1: Cluster tree of metals in water in various sampling locations.....	117
Fig. 4.2: Spatial distribution of As in the study areas	119
Fig. 4.3: Spatial distribution of Cd in the study areas	119.
Fig. 4.4: Spatial distribution of Pb in the study areas	120
Fig. 4.5: Spatial distribution of Hg in the study areas	121
Fig. 4.6: Cancer health risk faced by ASGM workers in PHVD exposed to As and Cd in sediment and water samples.	136
Fig. 4.7: Cancer health risk faced by ASGM workers in TNMA exposed to As and Cd in sediment and water samples.	137
Fig. 4.8: Cancer health risk faced by ASGM workers in Tinga in Bole – Bamboi District exposed to As and Cd in sediment and water samples.	138
Fig. 4.9: Cancer health risk faced by ASGM workers in Nabdam District exposed to As and Cd in sediment and water samples.....	139

Fig. 4.10: A graph comparing cancer health risk among ASGMworkers in the study areas.....	140
Fig. 4.11: Cancer health risk faced by resident adults non-miners in TNMA exposed to As and Cd in sediment and water samples	141
Fig. 4.12: Cancer health risk faced by resident adults in PHVD exposed to As and Cd in sediment and water samples.	142
Fig. 4.13: Cancer health risk faced by resident adults and children in CCMA exposed to As and Cd in sediment and water samples.	143
Fig. 4.14: Cancer health risk faced by resident children in Nabdam District exposed to As and Cd in sediment and water samples.	144
Fig. 4.15: Cancer health risk faced by resident children and adults in Tinga in the Bole - Bamboi District exposed to As and Cd in sediment and water samples.	145
Fig. 4.16: A graph comparing non-cancer human health risk across the study areas	147

CHAPTER ONE

INTRODUCTION

1.0 General Introduction

Mining and export of gold in Ghana began long before the advent of colonialism (Hilson, 2006), where the metal was mined using indigenous techniques. However, the use of modern technology in gold mining dates as far as AD 1880 in Tarkwa and was extended to Obuasi in 1898 (Hilson, 2002). Gold mining in Ghana has been a major pillar which the nation development relies on since independence. Currently, about 20% of the total land surface of Ghana is under mineral concession held by mining companies or licensed small – scale miners as well as those mining illegally, popularly called ‘galamsey’ (Aryee, 2001).

Metalloids such as arsenic (As) and heavy metals such as lead (Pb), mercury (Hg) and cadmium (Cd), naturally occurring pollutants associated with gold mining environmental levels have increased in some Ghanaian mining communities (WHO, 2007; Armah et al., 2012; Golow and Adzei, 2002). For instance, mercury pollution has been identified as a lingering problem in several of Ghana’s important artisanal gold mining communities. A burgeoning collection of studies (e.g., Golow and Adzei, 2002; Akbazaa et al., 2009) have demonstrated that contamination is now widespread in biota, with serious health implications for human populations residing within the country’s gold belts. Concerns from exposure to environmental pollutants have led to an increase in the measures taken to reduce the release of these chemicals into the environment and to protect miners and the general public.

Artisanal Small-scale Gold miners in most Ghanaian communities either work on 8 h shift basis (based on Central Tendency Exposure parameters) or 12 h a day (based Reasonable Maximum Exposure) so as to reduce their impact on the environment as well as human health. Their activities usually involve dredging of river beds for alluvial gold or washing of the gold ores blocked rivers and in the process incidentally ingest both the contaminated water and the sediment or through dermal exposure to the contaminants.

In spite of the above measures put in place by the miners to reduce their impact on the environment and human health, so as to consolidate their economic gains, there is growing concerns over the negative environmental destruction and health effects associated with the sector to the country as a whole, and to the communities directly impacted by gold mining activities in particular. These concerns have culminated in the call for undertaking human health risk assessment of the entire gold mining sector (Armah et al., 2012; Adimado and Amegbey, 2003).

1.1 Importance of gold mining to Ghana's socio - economic development

Gold mining has played a central role in the economic and political life of the country for the past 1000 years (Hilson, 2006). It generated huge foreign revenues to the country from 1991 to 2003. However, in 2004 revenues from gold mining was overtaken by the cocoa sector. Akabzaa *et al.*, (2009), the gold mining sector has attracted over US \$10 billion dollars of investments into the country from 1984 to 2009.

According to the Ghana Extractive Industry Transparency Initiative (GHEITI, 2014), the total contribution of mining to GDP increased from 9.5% in 2012 to 9.8% in accounting for a total foreign exchange earnings of US \$3,192,648.

The benefits of gold mining to Ghana's economy is not limited only to direct revenue that accrues to government, but gold mining in Ghana is seen as an employment creation industry. For example, large-scale mining companies together with other mine support services employed about 17,103 people in the year 2013 (Ghana Chamber of Mines, 2002) and the small-scale mining companies have employed approximately 260,662 people (GHETI, 2014; GSS, 2014).

Data from the Bank of Ghana shows that export of gold increased from 3.84 million ounces in 2016 to 4.61 million ounces in 2017 (Bank of Ghana, 2018; GRA, 2018). The 20 per cent growth in exports was driven mainly by a combination of modest increase in gold price and an upturn in the output of large-scale producers as well as the volume of gold exported by Licensed Gold Exporting Companies. As well, exports of manganese increased from 2 million tonnes in 2016 to 3 million tonnes in 2017 while shipments of bauxite also expanded from 1.14 million tonnes to 1.47 million tonnes over the same period (Ministry of Finance, 2018). The growth rate in shipments of manganese and bauxite are 50 per cent and 29 per cent respectively. On the other hand, exports of diamonds by PMMC shrank by 39 per cent, from 143,005 carats in 2016 to 87,068 carats in 2017 (Ministry of Finance, 2018; World Bank, 2018; World Gold Council, 2018).

However, in spite of the above impressive positive economic outlook from the gold mining sector in Ghana, several studies have highlighted the fact that, gold mining has resulted in the pollution of water bodies, food crops, air which provide essential services to host community members.

The overall net effect is that most of the residents in the mining communities are suffering from mining related diseases such as cancer and non-cancer disease due to exposures to toxic metals such as arsenic, mercury, cyanide used in the mining operations. These concerns expressed by different scientists suggest that, Ghana as a nation, should not only be interested in only the economic gains from the mining sector but rather pragmatic steps should be taken to address the negative environmental destruction and health effects associated with the sector to the country as a whole, and to the communities directly impacted by gold mining activities in particular. Such pragmatic decision and actions can only be taken by relevant stakeholders from data emanating from empirical studies that highlight human health associated with exposure to toxic chemicals from gold mining operations in Ghana. Hence, a study assessing human health risk assessment of the entire gold mining sector (Armah et al., 2012; Adimado and Amegbey, 2003) is very useful and timely.

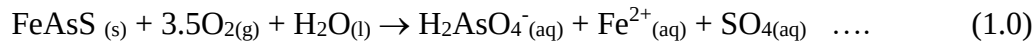
1.2 Gold deposit in Ghana

Gold deposits in Ghana are found in the Birimian and the Tarkwaian rock system (Carbo and Khalid, 2000; Kese, 1985). These gold deposits are exploited by both small and large-scale mining companies. The deposits are categorised into the following:

- The gold deposits found in the reef, vein or lode. It is usually found in the quartz veins,
- Sulphide ores in mineralized forms of the gold deposits. The tuffaceous phyllites contains the sulphide ores within the greenstone series.

- The ‘oxidised’ ores in gold bearing reefs. They are very close to the earth surface within an average depth of about 30 m to 80 m. They are highly lateritic, i.e., full of iron oxide, clays, weathered mica and quartz-pebbles.
- Lode gold are found in pegmatite dykes which are few metres or less in thickness. The gold may occur both in the free state or mixed with phyllites. It is also associated with cassiterite, wolfrinite, molybdenite and pyrite ores.

Scientists such as Kese (1985) and Rawlings et al., (2003) have found the following chemicals to be associated with gold ores. These include zinc sulphide, lead (II) sulphide, etc. The extraction of the gold from its ores releases hazardous chemicals into the environment. For example, the arsenopyrite gold ore reacts in the presence of oxygen according to the equation below:



The $\text{H}_2\text{AsO}_4^{-}$ in equation (1) above undergoes ionization to produce As^{3+} ions which are found in the mine tailings together with other toxic chemicals as shown in equations (1.1) and (1.2) below:



Improper disposal of the mine tailings containing arsenic and other toxic chemicals into the environment may contaminate water bodies, sediment and the biota.

Roasting of gold amalgam by small – scale miners in residential settings within the mining communities releases mercuric oxides into the air. When human beings are exposed to these toxic chemicals from processing of gold ores, it poses significant health hazard to them.

According to WHO (2007), exposure to very low concentration of toxic metals like, cadmium, mercury and lead cause multiple adverse health effects. This is due to the fact that the aforementioned metals can build up within the surroundings and individuals (WHO, 2007).

1.3 Problem Statement

Processing and extraction of gold from its ores involves the use of several toxic chemicals such as cyanide and mercury. It is evident that improper discharge of untreated mine process solution or spillage of mine tailings would result in catastrophic environmental effects and severe health hazards to human beings and animals. For instance, Asante *et al.*, (2007) found out that 58% of surface water samples ($n = 200$) from Tarkwa, a historic mining town, had arsenic levels in the range of $0.5 \mu\text{gL}^{-1}$ to $73 \mu\text{gL}^{-1}$ with a mean of $18 \mu\text{gL}^{-1}$, which exceeded the WHO drinking water permissible guideline value of $10 \mu\text{gL}^{-1}$; while $14 - 64 \mu\text{gL}^{-1}$ was measured by Smedley *et al.*, (1996) in drinking water from Obuasi. Tay and Momade (2006) measured high Cd concentration of $7.6 \mu\text{gL}^{-1}$ in water from bodies in the northern part of the Ashanti gold belt.

Studies quantifying the levels of arsenic, cadmium, lead, etc in environmental media from mining communities in southern Ghana have received much attention, than in mining communities in the three northern regions of Ghana. For example, Cobbina et al. (2011) measured cadmium concentration between 266.5 – 1,090 µg/L from borehole water at Kalin, Nabdam District, Upper East region.

From above, it means residents in mining communities may be continuously exposed to arsenic, cadmium, lead and mercury discharges into the environment. This would pose significant health risk to the people (Obiri et al., 2010).

Preliminary human health risk assessment studies conducted in mining communities in southern Ghana have revealed a strong correlation between exposure to arsenic, cadmium, mercury and lead by human beings and the attending health problems (Obiri et al., 2010). However, there is limited information on cancer and non-cancerous diseases faced by adults and children living in mining communities as well as workers of both large and small-scale gold mining companies in Ghana. For instance, as noted by the Cobbina et al., (2011), the non-cancer human health risk expressed in terms of hazard index for resident adults in Kalin exposed to arsenic was found to 2.4 as against the US Environmental Protection (USEPA) acceptable guideline value of 1.0. This result indicate that approximately, 2% of resident adults in the Kalin community in the Nabdam District are likely to suffer non-cancerous related diseases due to their exposure to arsenic from the environment.

According to Armah et al., (2012), the cancer health risk via oral ingestion of water from boreholes in Tarkwa a historic mining community in Ghana by resident adult is 2.4×10^{-3} by central tendency exposure parameters, which means approximately 2 out of every 1000 resident adult in the community are likely to suffer cancer related diseases due to exposure to carcinogenic chemicals in the environment.

In spite of the above scientific studies providing evidence of carcinogenic and non-carcinogenic diseases in some mining communities in Ghana due to exposure to heavy metals like arsenic, cadmium, mercury, etc in different environmental media and biota, a report by the Commission on Human Right and Administrative Justice (CHRAJ) issued in October 2008 entitled “The State of Gold Mining in Ghana”, revealed that residents of mining communities in Ghana, apart from high prevalence of malaria and other sexually transmitted diseases, they suffer cancerous diseases such as cancer of the skin, lung and bladder as well as other non-cancerous diseases such as hyper skin pigmentation, keratosis, black foot disease among several others which they attribute to the long exposure to heavy metals from mining operations in their communities. The CHRAJ report was unable to establish the veracity of the community claims but rather recommended that a study should be conducted to link the levels of toxic chemicals used in mining operations in Ghana in environmental media and human bio-samples to cancerous and non-cancerous diseases reported at health facilities in mining communities in the country (CHRAJ, 2008).

It is within this context that, this study proposes to quantify the risk of cancerous and non-cancerous diseases based on exposure to arsenic, cadmium, mercury and lead in the under listed mining communities such as Prestea, Dumasi, Twiagya, Bogoso all in Prestea Huni Valley District; and Teberebe, Effuantah, Bonsa, Wangarakrom just to mention a few in the Tarkwa Nsuaem Municipality of Western Region, as well as Tinga Bole-Bamboi district in Northern region and Nangodi in the Nabdam District of Upper East region of Ghana respectively. This will address knowledge gaps in human health risk assessment of the gold mining sector in the country.

1.4 Research Hypothesis

The general Ghanaian population residing in gold mining areas are exposed to different levels of the harmful arsenic, cadmium, lead and mercury via different pathways from the past and present mining activities. It is proposed that the current levels of the above named contaminants in different environmental samples and bio-samples from resident adults, children and workers of artisanal small-scale gold mine workers as well as that of large-scale mining companies in the following mining communities; Prestea, Dumasi, Twiagya, Bogoso all in Prestea Huni Valley District; and Teberebe, Effuantah, Bonsa, Wangarakrom just to mention a few in the Tarkwa Nsuaem Municipality of Western Region, as well as Tinga Bole-Bamboi district in Northern region and Nangodi in the Nabdam District of Upper East region of Ghana respectively would pose significant health challenge them as compared residents in non-mining communities such as Pedu, Effutu, Kwaprow, Kakumdo all in the Cape Coast Metropolis.

1.5 Research questions

The following questions would direct this study:

1. What are the concentration of arsenic, cadmium, lead and mercury in environmental samples and bio-samples of residents in the study areas; and how high are they compared to WHO/GS 175-1 guideline values?
2. How do residents of the study areas come into contact with the aforementioned toxic metals?
3. Are the measured arsenic, cadmium, mercury and lead concentrations potentially hazardous to residents of the mining communities in Ghana?
4. What are the cancerous and non-cancerous risks associated with the aforementioned metals quantified?
5. How do the results from the study compare with USEPA risk assessment guideline values?

1.6 Objectives of the study

The overall goal for the thesis is to quantify the carcinogenic and non-carcinogenic risks via ingestion and dermal contact to arsenic, lead, cadmium and mercury concentrations in water bodies, sediment and food crops by residents from mining communities in Ghana using the selected communities as case studies. The specific objectives are:

1. To measure levels of As, Cd, Hg, and Pb in environmental media, bio-samples of residents in the study areas;

2. To compare As, Cd, Hg and Pb concentrations in environmental media and bio-samples with Ghana Standards/World Health Organization (WHO) permissible limits;
3. To calculate the carcinogenic and non-carcinogenic health risks associated with the aforementioned toxic chemicals in Tarkwa Nsuaem Municipality, Prestea Huni Valley District, Bole-Bamboi District, Nabdam District and Cape Coast Metroplis for various exposure scenarios either acute or chronic;
4. To compare the cancer and non – cancer human health risks from (3) above with international acceptable guideline values.
5. To make policy recommendation to government and other agencies charged with the responsibility of protecting human lives and the environment.

CHAPTER TWO

LITERATURE REVIEW

2.0 Gold mining, processing and extraction techniques in Ghana

According to Kesse (1985), the gold bearing ores mined in Ghana are obtained from the Birimian and Tarkwaian rock systems. The gold ores basically consists of (Knight and Scott, 2001):

- Oxides mainly as iron oxides, i.e., haematite ($\alpha - \text{Fe}_2\text{O}_3$) and magnetite (Fe_3O_4),
- Sulphide mainly as pyrites (FeAuS) and the arsenopyrites (FeAuAsS),
- Transition mainly as partially oxidised sulphide forming a transition between the oxide and the sulphide ores,
- Alluvial ores mainly formed by gravity separation during sedimentary processes via movement of water, and
- Eluvial ores mainly formed from the disintegration of gold bearing rocks.

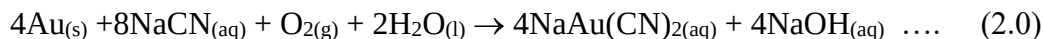
The aforementioned types of gold ores are mined by either large or small-scale gold mining companies operating in the country using two distinct processes such as:

Mining: is the process of extracting minerals i.e., gold from the earth crust. Mining methods used by both the small-scale or large-scale mining companies operating in Ghana are either surface mining popularly referred to as open-pit mining or deep shaft (i.e., underground mining).

However, depending on the location as well as the type of gold ore in question, both the small-scale or large-scale mining companies usually used a mixture of both open-pit or deep shaft gold mining techniques (Kesse, 1985).

Mineral Processing: Mineral processing is also known as mineral beneficiation and is defined as the process of physically separating and concentrating the gold ore from the waste using physical, chemical and sometimes microbiological techniques (Lottermoser, 2012; Cato, 1995; Allan, 1998; Lottermoser, 2010; Lottermoser et al., 2005; Lottermoser, 2011). Different mineral processing techniques are used by either small-scale or large-scale mining companies depending on the type of gold ore they are dealing with. Typical gold mineral processing technique used by small-scale mining companies include; digging and haulage of gold ores, crushing, washing and concentrating of the gold ore, mercury is finally added to the gold concentrate to form gold amalgam for the removal of the liberated gold from the tailings (mine waste). However, in some instances, small-scale gold mining companies have adopted whole ore amalgamation gold beneficiation techniques, which involves adding of mercury to the gold ore dug from the ground or from the beds of streams and rivers to form gold amalgam. The gold amalgam formed is then roasted in fire which lead to vaporization of the elemental mercury into a toxic plume leaving the gold behind. Mercury amalgamation is an age old technique used by both artisanal and small-scale gold mining (ASGM) and to a larger extent large-scale mining companies in time past, however, in recent times, the large-scale mining companies have stopped using mercury in the gold recovery process. Instead, they are now using other techniques such as cyanidation or bio-oxidation (Fashola et al., 2016).

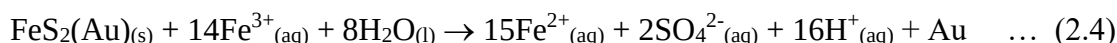
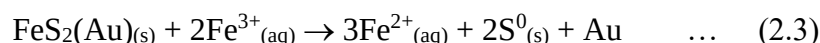
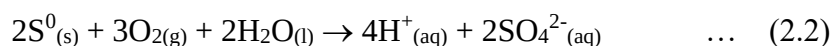
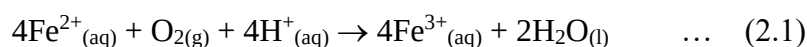
In the cyanidation process of gold recovery, the crushed and ground gold ore is first dissolved in cyanide solution (shown in equation 2.0).



The dissolved gold is recovered from the cyanide solution either by an adsorption onto activated carbon through an electrolytic process or by cementing with zinc. Depending on the quality of gold ore, the cyanidation process either takes place heap leach pads or leaching tanks. For ores with very high gold content, the tank leaching technique is employed. This involves leaching the crushed and ground ore in large tanks equipped with agitators to dissolve the gold which then adheres to pieces of the activated carbon. The activated carbon and the gold are then stripped of the solution and the barren solution together with the leached ore are discarded. In the case of very low grade gold ores, the heap leach is used and it involves extraction of the gold from crushed oxide gold ore piled onto plastic-lined pads with leaching solvents such as acids or cyanide to dissolve the gold which is collected at the bottom of the pad (Lottomoser, 2007). The use of cyanidation techniques have enabled mining companies to mine and process very low grade gold ores to meet the high demand for the commodity.

As noted by Rawlings et al., (2003), the sulphide gold ores which basically consist of either the pyrites or the arsenopyrites are not easily liberated directly via dissolution in cyanide solution. Hence, bio-oxidation techniques (which involves the use of biological organism such as bacteria) are used to assist in the oxidation of sulphide matrix in the gold ore to aid its fast dissolution in cyanide solution. The bacteria first either oxidise ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}) or reduce elemental sulphur (S^0) or other reduced sulphur compounds to sulphuric acid (H_2SO_4).

The Fe^{3+} and hydrogen ions (H^+) produced from the oxidation of ferrous iron to ferric iron then break down the sulphide matrix in either the pyrite or the arsenopyrite gold ore (Belzile et al., 2004). The under listed chemical equations summarises bio-oxidation of pyrite and arsenopyrite ores:



Mining and mineral processing in a typical gold mine result in the generation of large quantities of waste from the extracted ore as waste which is discharge into nearby environment (Adler and Racher, 2007).

2.1 Characteristics and environmental pollution associated with gold mine tailings

Tailings from a gold mine is defined as a mixture of finely ground rock that is left after the recovery of gold from the ore, water as well as other chemicals such as mercury or cyanide used in processing of the gold ore. In Ghana's large-scale mining industries, large volumes of mine tailings are dump in tailings containment ponds in compliance with the requirements in the Environmental Impact Assessment Regulations, LI 1652 of 1999.

However, as noted by Adimado and Amegbey (2003), due to structural defects in the constructions of most these tailings containment ponds, there have been 11 officially reported cyanide spillages from the collapse of such facilities resulting in the spillage unused cyanide solution and other toxic chemicals into the external environment between 1989 and 2003. Within the small-scale gold mining sector in Ghana, regulatory provisions in the LI 1652 are not strictly adhered to by the industry players, and has resulted in the pollution of water bodies, nearby soils and damage to the air quality (Rajee, et al., 2015).

The chemical and physical nature of gold mine tailings their properties are determined by the nature of the ore, geochemistry, the processing method used in extracting the gold from the ore (Frank et al., 2011; Davies and Rice, 2001). The physical properties of a typical gold mine tailings are poor aggregation and high hydraulic conductivity. It also have fine texture and limited cohesion ability. The lack of cohesion within the particles forming gold mine tailings is responsible for the varied moisture content and temperature which contribute immensely to the toxic nature of the waste (Frank et al., 2011; Davies et al., 2001). The aforementioned properties differentiate gold mine tailings from soil (Vega et al., 2004; Blight and Fourie, 2005).

Chemical properties of gold mine tailings depends on the type of gold ore, and the tailings is usually made up of approximately 6% pyrite, high salinity, and very low organic matter content (Vega et al., 2006). Hence, gold mine tailings cannot support plant growth. The high sulphides content of most of the gold mine waste result in high acidity and high metal concentrations leached into near groundwater or surface water within the vicinity of the tailings (Vega et al., 2004).

For example, Rafiei et al., (2010), reported a pH value of 7.35 in gold mine tailings in Iran, while Mitileni et al., (2011), reported pH values of 3.25 – 6.28 in gold mine tailings from South Africa; also Harish and David (2013) recorded very low pH value of 3.48–8.12 for gold mine tailings in India. As noted by Naicker et al., (2003) and Tutu et al., (2008), gold mine tailings with very low pH values have been observed in acid mine drainage arising from gold mining activity in studies from other gold producing countries. This characteristic feature of gold mine tailings (i.e., very low pH) contribute immensely to the elevated concentrations of toxic heavy metal ions such as arsenic (As), cadmium (Cd), nickel (Ni), lead (Pb), copper (Cu), zinc (Zn), cobalt (Co) and mercury (Hg) in nearby surface or groundwater (Da Silva et al., 2004).

Heavy metal pollution and acid mine drainage are very important environmental incidences that are of great concern environmentalists, communities that host the mines, regulators, etc. This is so because waste materials containing metal-rich sulphides from the gold mining activity stored in waste rock dumps or abandoned within the mine concession (Concas et al., 2006). Tailings and rock dumps undergoing weathering and physical or chemical changes impact negatively on surface and ground water quality. The underground impacts are caused by the influx of water into the underground workings and the subsequent dewatering of the aquifer.

Another source of environmental pollution from gold mines is the chemicals used in processing the gold. An estimated 1400 metric tons of mercury was used in 2011 by ASGM and an annual average of 1000 metric tons of inorganic Hg was discharged. One-third of this estimated value goes into the air and the rest is mixed up in heaps of tailings, soil and waterways (Rajaei et al., 2015).

Mercury can also be released into the environment as a result of present reprocessing of some old gold tailings dumps (Rajae et al., 2015).

2.2 Status and trend of arsenic, cadmium, mercury and lead contamination in groundwater

Over the years, several scientists have assessed different concentrations of contaminants such as Cd, As, Pb, Hg, etc in groundwater. For example, the lowest concentration of Cd ($<0.05 \mu\text{gL}^{-1}$) was reported by Obiri (2007) in groundwater from the Tarkwa gold mining area while the highest Cd concentration of $8.16 \mu\text{gL}^{-1}$ was reported by Cobbina *et al.*, (2013) for boreholes in small – scale gold mining communities in the Bole – Bamboi District Savannah Region of Ghana.

The lowest concentration of Cr ($0.03 \mu\text{gL}^{-1}$) was reported by Asante et al., (2007) for the Tarkwa gold mining area, while the highest Cr concentration of $45 \mu\text{gL}^{-1}$ was reported by Obiri (2007) for the Dumasi community in the Prestea Huni Valley District. Studies on the concentration of Pb in groundwater in mining communities have been measured by other Ghanaian scientists to be in the range of $0.03 \mu\text{g/L}$ and $45.0 \mu\text{g/L}$ (Armah et al., 2014).

The lowest concentration of Pb of $\leq 0.001 \mu\text{gL}^{-1}$ were measured in boreholes from Twiagya a mining community in the Prestea Huni Valley District by Obiri *et al.*, (2010) and in Dunkwa – on – Offin in Upper Denkyira Municipality by Ansa – Asare *et al.*, (2014) respectively; while Pb concentration of $1 \mu\text{gL}^{-1}$ has been measured in borehole water in Tinga a mining community in Bole-Bamboi District by Cobbina *et al.*, (2013).

Akabzaa *et al.* (2007) measured the highest Pb concentration of $96 \mu\text{gL}^{-1}$ in boreholes within the vicinity of the AngloGold Ashanti, Obuasi goldmine. Similarly, Obiri (2007) also measured Pb concentration of $120 \mu\text{gL}^{-1}$ in borehole water from Dumasi within the mining concession of Golden Star Resources, Prestea-Bogoso, Gold Mine in the Prestea Huni Valley District.

In the case of Hg levels in groundwater from mining communities in Ghana, different scientists have reported varying concentrations of Hg in groundwater. For example, the lowest Hg concentration $2.30 \mu\text{gL}^{-1}$ in groundwater from Tarkwa mining areas was measured by Asante *et al.*, (2007) while the highest concentration ($205 \mu\text{gL}^{-1}$) was reported by Tay and Momade (2006). Groundwater mercury concentrations were generally lower than surface water concentrations in most of the scientific studies conducted to assess the impact of gold mining operations in Ghana. The reason ascribed by Ghanaian scientists for the low mercury concentrations in groundwater from gold mining communities is due to the fact that, mineralogical the Birimian and Tarkwaian rock system where our gold ore deposits are found do not contain mercury minor element (Kesse, 1985). Hence, there is limited water-rock interaction for the immobilisation of mercury into the groundwater in boreholes drilled through the Birimian and Tarkwaian rock system (Kesse, 1985; Koartatsi, 2002). That is, mercury concentrations in groundwater in the gold mining communities can be attributed to seepage of leachate of unused mercury in mine tailings improperly disposed in the communities.

Also, mercury concentrations in groundwater varied spatially with higher mercury concentrations found in mining communities that have both artisanal small-scale gold mining (ASGM) and large-scale gold mining (LSGM) co-existing; while in communities with either artisanal small-scale gold mining (ASGM) or large-scale gold mining existing a lower to moderate high mercury concentrations were measured in the groundwater. According to Akabzaa et al. (2009), mercury levels in groundwater have been identified with water draining historic tailings dumps at Prestea and Tarkwa at levels higher than acceptable limits by WHO and EPA Ghana, because mercury was used in gold extraction by large-scale mines before the introduction of cyanide leaching technology. Thus the sources of mercury in water resources in the study area are due to both artisanal miners and remnants of mercury wastes formerly used by the large scale mining companies.

The concentration of arsenic in groundwater from mining communities in Ghana have been widely studied. A study by Asante *et al.*, (2007) recorded the lowest As concentration of $1.30 \mu\text{gL}^{-1}$ in borehole water from Tarkwa mining area, while Boadu *et al.*, (2000) measured the highest As concentration of $11,950 \mu\text{gL}^{-1}$ in borehole water from Konongo-Odumasi a historic mining town in the Ashanti region.

As noted by Smedley et al., (1996), arsenic concentrations in groundwater varies with the depth of the groundwater. For example, arsenic concentration of $64 \mu\text{gL}^{-1}$ in groundwater to the depth of between 40 -70 m as compared moderately low arsenic concentrations measured in groundwater of depth ranging between 0 – 20 m.

The reason for this variation of arsenic concentration with depth of the ground water is due to the fact that, the concentration of arsenic in the groundwater is believed to increase as a result of the longer residence times undergone by groundwater in the deeper parts of the aquifer than in shallow parts of the aquifer. That is, higher proportion of arsenic present as As^{3+} species is also higher in the deeper groundwater than shallow groundwater. However, deep mine exploration boreholes of depth ranging between 70–100 m, tend to have a much relatively low arsenic concentration between 5–17 $\mu\text{g l}^{-1}$ (Smedley et al., 1996). This is possibly due to the sorption of arsenic onto precipitating ferric-oxyhydroxides or the localization of low arsenic concentrations on the sulphide minerals.

Cadmium occurs in gold bearing ore bodies as an isometric trace element in sphalerite and its concentration depends on the concentration of the sphalerite in the ore body. According to Kesse (1985), the sphalerite is also found in the Tarkwaian and Birimian rock system which host the gold deposits in Ghana.

As noted by Hilson (2010), most scientific studies on heavy metals contamination in Ghanaian gold mining communities have so far been focused in southwest Ghana, hence, the extent of pollution in mining communities in the northern part of the country, where mining activities are said to be expanding, is not well documented.

According to Cobbina et al., (2015), cadmium concentrations measured in ground water from boreholes in Nangodi ranged from 1.0 to 2,227 $\mu\text{g/L}$ with a mean of $5,340 \pm 0.088 \mu\text{g/L}$; while in Tinga, measured cadmium concentrations in boreholes ranged from 2 to 710 $\mu\text{g/L}$ with a mean concentration of $230 \pm 0.008 \mu\text{g/L}$.

In all these cases, the authors attributed the levels of the aforementioned heavy metals in the borehole water to the dissolution of the heavy metals from the Tarkwaian and Birimian rock systems from which they were drilled.

2.3 Status and trends of arsenic, cadmium, mercury and lead contamination in surface water

According to Younger (2001) mining has been identified as one of the causative factors for deteriorating water quality in mining communities. Hence, protection of water resources especially surface water from not being polluted from gold mining operations have been at the core of government of Ghana mining sector reform. As such, water conservation and protection policies from the negative impacts of gold mining are crucial to scientists in the academic communities, host mining communities, regulators as well as small-scale gold mining companies and large-scale gold mining companies.

It is in this regard that, several Ghanaian scientists have measured different levels of heavy metals such as arsenic, cadmium, mercury, lead, etc in surface waters from gold mining communities across the country. For instance, Armah et al., (2011), measured the lowest concentration of As ($4 \mu\text{gL}^{-1}$) in water samples from rivers Abonponi, Agonaben, Adisakrom, Bediabewu, Domebra, Nkwantakrom and Teberebe was reported by Armah *et al.*, (2011) in the Tarkwa mining area and the highest As concentration ($3,137 \mu\text{gL}^{-1}$) was reported by Akabzaa *et al.*, (2007). Similar findings have also been found by Boamponsem *et al.*, (2010) for Prestea in the Western region of Ghana.

The lowest concentration of Hg ($1 \mu\text{gL}^{-1}$) was reported by Akabzaa *et al.*, (2007) in three rivers in communities immediately downstream of Obuasi and its environs. Also, Kortatsi *et al.*, (2008) reported the highest mean concentration of Hg ($4,600 \mu\text{gL}^{-1}$) in water samples from the Tarkwa mining area.

A study by Armah *et al.*, (2010) measured the lowest concentration of Pb ($0.69 \mu\text{gL}^{-1}$) in water samples from the Tarkwa mining area, while the highest concentration of Pb ($515 \mu\text{gL}^{-1}$) was reported by Akabzaa *et al.*, (2007) in 4 mining communities within the operating areas of the then Ashanti Goldfields Company – Obuasi mine.

Only a few of the scientific studies that assessed the levels of heavy metals such as arsenic, cadmium, mercury and lead in surface water from gold mining communities in Ghana actually looked at the origin of such metals in the natural environment. Origins of contamination of surface water with heavy metals could either be from geogenic or anthropogenic source. The geogenic source of contamination of surface water occurs where ions of heavy metal or metalloids get dissolved from the rocks into the water as it moves through the mineral surfaces of the soil; while the anthropogenic source of heavy metal contamination of surface water occurs through series of manmade activities such as mining, construction enhances the release of ions of heavy metals or metalloids into the surface or groundwater (Kortatsi, 2006).

It must be noted that most of the scientific studies on the impact of gold mining on quality of surface water have focussed on the anthropogenic sources (Armah *et al.*, 2014; Tay and Momade, 2006; Kortatsi, 2006). According to the findings from such studies, indiscriminate dumping of mine waste rocks accelerate the weathering of reactive sulphides by creating the necessary conditions for weathering to take place.

That is, the mine waste rocks containing sulphides and other toxic materials are attacked by the air and water, which increase the surface area of the reactive component of the waste rocks, and create the opportunity for colonization by microorganisms that hastening the oxidation processes of the sulphides through acid mine drainage, which then act on other potentially toxic materials such as heavy metals to be released into the environment thereby contaminating surface water (Kusimi, 2012; Kortatsi, 2006; Fashola et al., 2016).

Another significant anthropogenic source of contamination of surface water in most mining communities in Ghana identified by scientist working on this subject is the issue of improper discharge of mine process solution or cyanide spillages. Hence, according to Adimado and Amegbey (2003), between 1989 and 2003, Ghana recorded officially 11 incidents of cyanide spillages.

The few studies that have focused on geogenic source of contamination of surface water in gold mining communities in Ghana, have relied on the use of multivariate statistical techniques as well as other techniques to characterised the geogenic factors. For example, Armah et al., (2012) applied principal component analysis and cluster analysis to discriminate between geogenic and anthropogenic sources of contamination of surface and groundwater in Tarkwa, a historic gold mining community in Ghana.

It must be emphasised that, despite large body of scientific literature on negative impact of gold mining on water quality in Ghana, there exist huge disparities in the distribution of such studies among gold mining communities in southern and northern part of the country (Armah et al., 2014; Rajaei et al., 2015).

Reviews of such few scientific studies have revealed that there are higher metal concentrations in water samples from such rivers or streams. For instance, Cobbina *et al.*, (2013) has reported that mercury concentration in surface water from Tinga in the Bole – Bamboi District of Northern Region of Ghana ranged from 10 – 172 μgL^{-1} with a mean of 50 μgL^{-1} as against the WHO guideline value of 6 μgL^{-1} ; while that of cadmium ranged 2 – 103 μgL^{-1} with a mean of 31 μgL^{-1} as against the WHO guideline value of 3 μgL^{-1} . Lower concentrations of arsenic and lead less than 1 μgL^{-1} were recorded by Cobbina *et al.*, (2013).

2.4 Status and trend of arsenic, cadmium, mercury and lead contamination in sediments, nearby riverine soils and food crops grown in mining communities in Ghana

Plants also absorb As, Cd, Hg and Pb posing serious health problems to man since they are source of food to man. The uptake of As, Cd, Hg and Pb from both the atmosphere through leaf surfaces and from the soil through the roots may account for the elevated levels of the metal in the food crops grown in mining areas (Alloway and Ayres 1994; Essumang *et al.*, 2007; Falandysz *et al.*, 2002).

Due to the serious health implications associated with ingestion of heavy metals from contaminated mine fields due to uptake by food crops grown in such fields, several scientific studies have been conducted to determine levels of heavy metals uptake by food crops grown in mining communities in Ghana (Essumang *et al.*, 2007; Hayford *et al.*, 2009; Golow *et al.*, 1996; Golow and Adzei, 2002; Amonoo – Neizer and Amekor, 1993; Akabzaa *et al.*, 2007; Amonoo – Neizer *et al.*, 1996; Adjorlolo – Gasokpoh *et al.*, 2012; Amasa, 1975).

These studies have focused mainly on the chemical content of toxic chemicals such as As, Hg, Cd, Zn, Pb, Cu, Ni, Mn, and Fe in food crops or other plant specimens. The most common food crop that has been used in most of the studies is cassava - *Manihot esculenta* (Adjorlolo – Gasokpoh *et al.*, 2012; Amonoo – Neizer *et al.*, 1996; Golow and Adzei, 2002). Other studies such as Essumang *et al.*, (2007) have measured the highest arsenic concentrations of 383 mg/kg in water cocoyam (*Colocasia esculenta*) grown in Tarkwa which exceeded the WHO permissible guideline value of 3 mg/kg. Akabzaa *et al.*, (2007) on the other hand concentrated on determination of heavy metals in fruits such as orange. Whilst Amonoo – Neizer and Amekor, 1993, Hayford *et al.*, 2009 and Amasa, 1975 have in addition to determining the levels of heavy metals in cassava grown in mining communities have also focused on other food crops such as cocoyam, plantain, palm tree, bananas, pepper, beans and pear.

A study by Amonoo – Neizer (1993) has categorised the food items analysed into cooked and uncooked, whereas Akabzaa *et al.*, (2007) did not distinguish between the cooked and uncooked forms. A study by Nyarko *et al.*, (2006) used lichens to determine the extent of heavy metals contamination in the Obuasi gold mining area; while Amonoo – Neizer (1993) measured the levels of heavy metals in star grass grown in mining communities. However, two studies (Amonoo – Neizer *et al.*, 1996 and Amasa, 1975) also used ferns; with Amonoo – Neizer (1993) and Amonoo – Neizer *et al.*, (1996) using the elephant grass as biologic specimen to determine the levels of heavy metal contamination in most mining communities.

Scientific studies to determine mercury concentrations in sediment from beds of rivers impacted by gold mining activities have revealed a higher levels which exceed the guidelines than sediments from non-gold mining communities.

For example, a study by Bonzongo et al., (2004) revealed that 59.3% of sediment samples collected from artisanal small-scale gold mining sites have mean mercury concentrations that exceeded the U.S. EPA guideline of 0.18 $\mu\text{g/g}$ Hg dry weight (d.w.). While mean mercury concentrations in sediments from non-mining communities varies from below detection limit to 0.494 $\mu\text{g/g}$ Hg (Donkor et al., 2006). Only few studies have explored seasonal variation of mean mercury concentrations in sediments from mining and non-mining communities. It was observed that, mean mercury sediment concentration was found to be higher in the dry season as compared to wet season (Donkor et al., 2006). Nartey et al., (2011), observed that the mean mercury concentrations in downstream sediments from rivers and streams in the Birim North District of Eastern were twice as high as the mean mercury concentrations recorded in upstream sediments of the same rivers and streams.

A similar study by Boamponsem et al. (2010) report of measuring the highest mercury concentration of 200mgkg^{-1} in bottom sediments from the Offin River basin. While Akabzaa et al., (2007) measured a mean lead concentration of 115mgkg^{-1} in sediments of stream and rivers in downstream communities within the concession of Anglogold Ashanti – Obuasi Mine.

A review of scientific investigation of heavy metals concentration of soils within gold mining communities in Ghana have revealed that, heavy metal concentrations in soil is higher than the same heavy metals reported in riverine sediments (Armah et al., 2014). However, a study by Hayford et al., (2009) reports of low arsenic concentration of 0.7mgkg^{-1} in soil; while Amasa (1975) measured the highest arsenic concentration of 2875mgkg^{-1} in soil within the concession of the then Ashanti Goldfields Company Limited.

The determination of mercury concentrations in soils within gold mining communities in Ghana have receive prominent attention by scientists in the country. Mixed results have been obtained from such studies. For instance, Oppong et al., (2010) recorded the lowest mercury concentration of 0.075 mgkg^{-1} in soil samples along the banks of the Pra River at Daboase in Western Region of Ghana; while Donkor et al., (2006) measured the highest mercury concentration of 2146 mgkg^{-1} in soil samples within the Offin River basin.

It can be seen that most of the scientific studies conducted within Ghana have focused on mining communities in the south (i.e., which are economically developed as well as having a history of long period of commercial gold mining) than mining communities in the northern part of Ghana. With active mining currently taking place in the northern part of the country, it is important for more research to be conducted in these areas so as to document the impacts of gold mining on the environment, human health of the residents.

2.5 Status and trends of levels of arsenic, cadmium, mercury and lead in human bio-samples from Ghanaian mining communities

Gold mine workers (i.e., from large or small-scale mining companies) as well as workers in other sectors of the economy and residents in gold mining communities are continuously being exposed to toxic chemicals such as arsenic, cadmium, mercury and lead. This is due to the fact that the aforementioned toxic chemicals which are (either used in processing the gold from its ores) or are found in mine tailings improperly discharged into the environment.

In the developed countries a lot of studies have been undertaken using different biological media (Lauwerys and Hoet, 1993; IPCS, 2000; IPCS, 2001).

For example, Batariova et al., (2006) found that human whole blood is proper for measuring recent exposures to Cd; while for long term exposure to Cd, urine is the preferred human sample. Since, it plays a useful role in quantifying the concentrations of toxic chemicals in internal organs of the body (Rodrigues et al., 2009).

However, in Ghana, most of the few scientific studies that have been conducted broadly looked at levels of heavy metals or metalloids concentrations in urine for gold mine workers and non-gold mine workers as well as licensed small – miners; with other studies using human hair, blood and nails as biological samples.

For instance, Amasa (1975) found out that the mean arsenic concentration of 3363.33 mg/kg in human hair of mine workers from the Pompora Treatment Plant of erstwhile Ashanti Goldfields Limited – Obuasi mines while in the case of non – miners the mean arsenic concentration was 38.7 mg/kg. From a study by Asante et al., (2007), it can be deduced that the non-mine worker/mine worker ratios for As, Hg, Mn, Cd, Zn, Cr, Cu, and Pb in were 1.2, 10.9, 1.3, 7.7, 0.3, 3.7, 1.4, and 1.2, respectively. This suggests that except for Zn, non mine workers generally had lower concentrations of heavy metals in their urine compared to their counterparts who were mine workers. As, Hg, Mn, Cd, Cr, Cu, Co and Pb concentrations in urine samples of small scale artisanal gold miners in the Upper East region are as follows; 114.52 μgL^{-1} , 2.01 μgL^{-1} , 0.45 μgL^{-1} , 601.27 μgL^{-1} , 26.58 μgL^{-1} , 40.85 μgL^{-1} , 2.32 μgL^{-1} and 1.34 μgL^{-1} , respectively.

This suggests that except for Zn, non-mine workers generally had lower concentrations of heavy metals in their urine compared to their counterparts who were mine workers. Adimado and Baah (2002) reported mercury levels of 30.2 μgL^{-1} and 218 μgL^{-1} in the Western and Central regions of Ghana, respectively.

2.6.0 Concepts of human health risk assessment

Human health risk assessment is defined as an attempt to identify in qualitative sense, and then to quantify scientifically, the health risk associated with exposure to a named toxic chemical. It is also defined as the process of estimating health effects that might result from exposure to a named pollutant (USEPA, 2008; WHO, 2010). From the above definition of human health risk assessment, two key concept emerges, i.e., risk is a function of exposure and the toxicity of the named pollutant. These two key concepts are shown in Fig. 2.0; and mathematically, it can be expressed as:

$$\text{Risk} = \text{Exposure} \times \text{Toxicity} \quad \dots \quad (2.5)$$

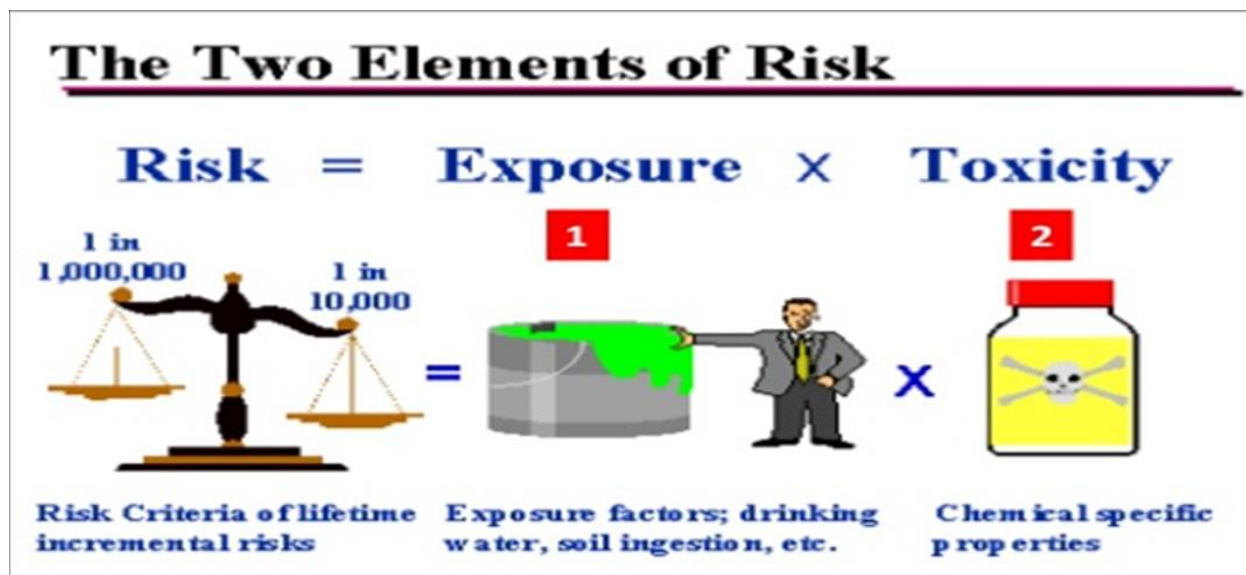


Fig. 2.0: Two key elements in the definition of human health risk assessment. (Source: WHO Risk Assessment Toolkit)

A toxic chemical according to USEPA (2008) is defined as any chemical that poses unreasonable risk to the health and safety of a human being. It does so through the alteration of the normal function or the destruction of life of an organism. The extent of damage to an organ or tissue in the human body is an indication of the chemical's toxicity at a specified concentration.

Hence, toxicity of a chemical is defined as the likelihood of a chemical to cause harm to human beings when ingested, inhaled or absorbed through the skin layers (USEPA, 2008). It is also defined as the basic property of the chemical that reflects its inherent ability to produce to organs or tissues within the human body.

The toxicity of a chemical is categorised on the basis of its exposure duration, i.e., acute, chronic and sub-chronic. According to USEPA (2008), acute toxicity is caused by relatively exposure to large dose of the toxic chemical over a short time interval. The duration of time from the exposure to the toxic chemical to the onset of the clinical signs or symptoms associated with that toxic chemical as well as the intensity of the effects is often higher and very lethal. While, for chronic toxicity is caused by repeated exposure to relatively small doses of lower concentration of the toxicants over a long period of time. The development of symptoms for chronic toxicity may be months or even years and its intensity is gradual and less severe than the acute toxicity. Whereas, sub-chronic toxicity often takes places over a shorter period of time, usually weeks or months (USEPA, 2008).

As noted by USEPA (2008), the toxicity of a given chemical depends on the mode through which human beings are exposed. That is, factors or conditions under which human beings get into contact with that toxic chemicals.

For most chemicals, the factors through which human beings get into contact with is either through water, sediment, soil, air or eating of contaminated food crops/animals.

Hence, human health risk associated with exposure to a named toxic chemical depends on its mode of absorption (i.e., exposure), distribution, metabolism and excretion (Alloway and Ayers, 1993). The absorption refers to the movement of the toxic chemical from the site of initial contact with the biologic system across a biologic barrier into either the blood stream or the lymphatic system. Once, the toxic chemical gets into the blood stream, it is distributed to various tissues or organs of the human body. Similarly, the toxic chemical get metabolised or excreted from the human body (USEPA, 2008).

The three principal exposure routes for absorption of toxic chemicals into human body are:

- **Ingestion:** this occurs when the toxic chemical is found in food, animal products or water as well as in soil or sediment where it is incidentally taken into the human through the gastrointestinal tract;
- **Inhalation:** this is second major exposure pathway for gaseous toxicants or liquid or particulate aerosols as well as dust containing toxic chemicals through the lungs into the blood stream; and
- **Skin:** this is the third major exposure pathway for most toxic chemicals through either the sweat glands or the hair follicles; as the outermost keratinized layer of thick-walled epidermal cells such as the stratum and corneum are generally impermeable to most toxicants (USEPA, 2008).

In accordance with USEPA, risk assessment guidelines, the four main key steps that needs to be followed when assessing the human risk associated with exposure to a given toxic chemical in an environmental media (USEPA, 1997; USEPA, 2008) is shown in Fig. 2.1.

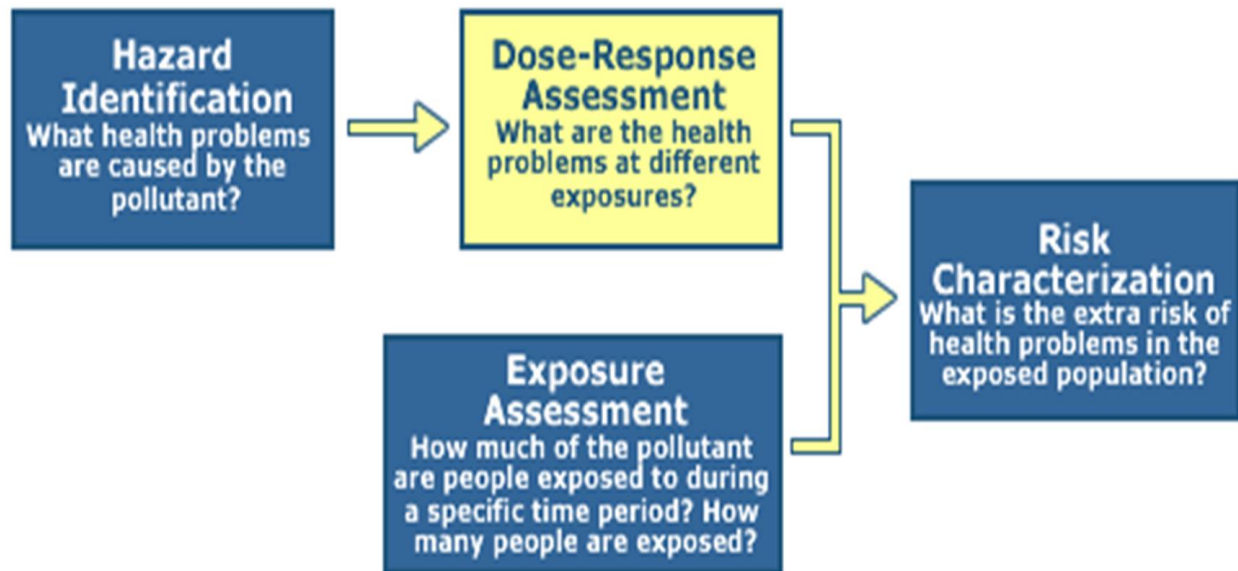


Fig. 2.1: The four basic steps involved in human health risk assessment process (Source: WHO Risk Assessment Toolkit)

From Fig. 2.1, the four key steps are as follows:

- **Hazard identification:** This is the first step in the human health risk assessment process. It addresses the issue of negative human health problems that are caused by that pollutant.

- **Dose-response assessment:** This step of the human health risk assessment process assess the health problems associated with exposure to that named pollutant under different exposure scenarios.
- **Exposure assessment:** This segment of the human health risk process deals with estimating the concentration of the pollutant that people are exposed to and the number of people in a given population that are exposed.
- **Risk characterisation:** This is the final step in the risk assessment process and it deals with estimating the risk associated with that pollutant in the given population, i.e., the extra risk of health problems faced by people exposed to that pollutant.

The above four important steps in the human health risk assessment would be used in this study and further discuss in subsequent sections of the thesis.

2.6.1 Hazard Identification of arsenic, cadmium, mercury and lead

According to Kesses (1985), cadmium is found in the in gold bearing ore bodies as sphalerite. Improper discharge of mine tailings as well as cyanide spillages contribute to presence of elevated levels of cad^{2+} ions in the environment. According to International Agency for Research on Cancer (IARC, 2004) and USEPA (2008), cadmium is a class B human carcinogen, i.e., there is ample evidence to link exposure to cadmium and incidence of cancer among human beings. In addition to its carcinogenic nature, it is known to cause the following non-carcinogenic diseases among human beings who are exposed:

- Hypertension,
- Upper respiratory tract infections,

- Metallic taste in the mouth,
- Mutagenic and teratogenic effects, and
- Kidney disease and damage to the skeletal system (USEPA, 2008; Satarug et al., 2011)

According to Abudul-Wahab and Marikar (2012), Lead is a toxic non-essential heavy metal found in water, soil, sediment, food crops and air in gold mining communities as part of the gold bearing ores in the form of galena (PbS). Lead is a neurotoxin, i.e., it affects the central nervous system of human beings exposed. According to both IARC (2004) and USEPA (2008), lead is not a human carcinogen. That is, there is no evidence of it to cause cancer among human beings. However, when children are exposed to, it leads to lowering of their intelligent quotient (IQs). Other symptoms associated with exposure to lead in an environmental media include but not limited to; stomach aches, dizziness, renal diseases, etc (Gonzalez-Estecha et al., 2010; Shahid et al., 2012).

As noted by Kesse (1985), elevated levels of arsenic in different environmental media is linked to the mining and processing of the arsenopyrite gold ores. Exposure to arsenic in different environmental media is known to cause the following non-carcinogenic diseases such as; peripheral vascular disease, severe disturbances of the cardiovascular and central nervous systems which may lead to death, bone marrow depression, haemolysis, hepatomegaly, melanosis, polyneuropathy and encephalopathy may also be observed (ATSDR, 2007; Tchounwou et al., 2012; Nigra et al., 2017). Other disease associated with exposure to arsenic has been summarized in Fig. 2.2.

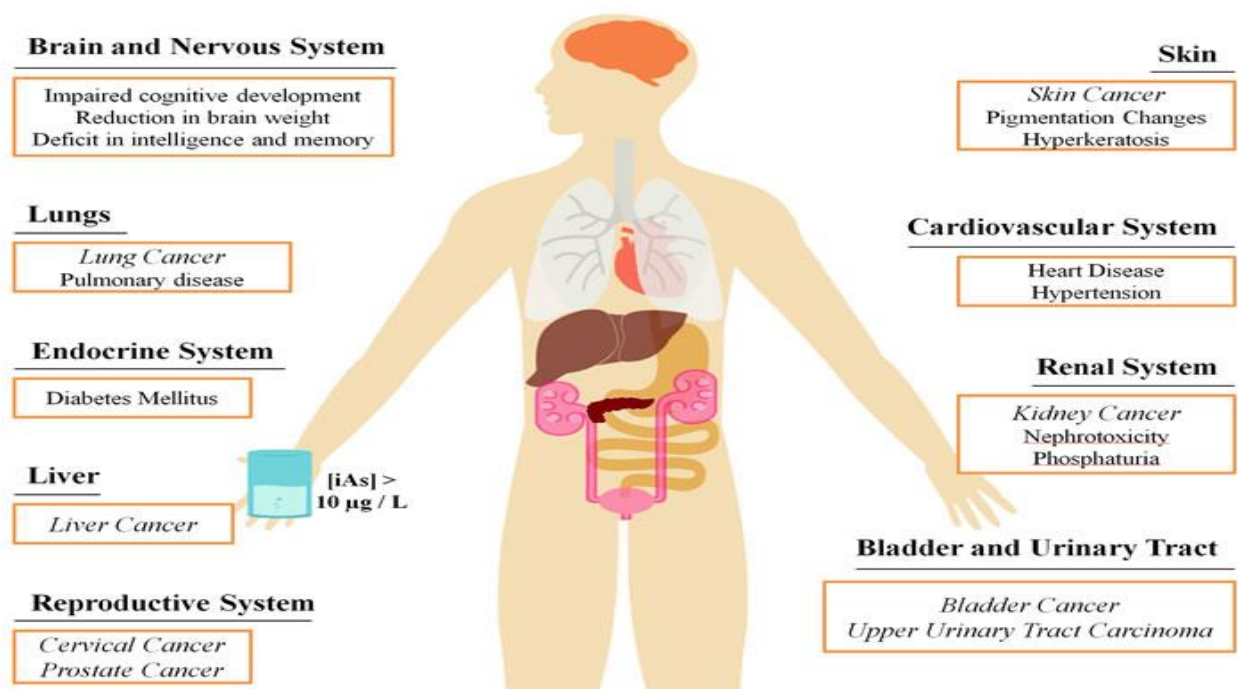


Fig. 2.2: Diseases associated with exposure to arsenic in environmental media (Source: Agency for Toxic Substances and Disease Registry, ATSDR, 2007)

In gold mining communities in Ghana, mercury is used to extract the gold from the ores and as such, improper disposal of the mine tailings as well as roasting of the gold amalgam by artisanal small-scale gold miners contribute to elevated levels of mercury in the environment (Essumang et al., 2007; Armah et al., 2010). Exposure to mercury is known to cause kidney damage, tremor, restlessness, anxiety, depression, sleep disorder, paresthesia and numbness in the hands and feet while high doses may lead to death. Total brain damage can occur in early exposure while late exposure results in localized damage to the cerebellum, motor cortex and the visual cortex (Weiss et al., 2002).

2.6.2 Dose-response assessment of arsenic, cadmium, mercury and lead

This section of the thesis reviews dose-response data used in assessing different health outcomes associated with exposure to arsenic, cadmium, mercury and lead in the environmental media. It reviews the dose-response relationship for carcinogenicity (i.e., oral and dermal cancer slope factors) for arsenic and cadmium as well as non-carcinogenicity dose-response relationship (i.e., oral and dermal reference doses) for arsenic, cadmium, mercury and lead in the different environmental media.

According to USEPA (2008), the term cancer risk is defined as the incremental probability that an individual would develop cancer during his/her lifetime after exposure to a carcinogen. The term incremental means that, the cancer risk is above a background risk by individuals in the course of life. The background cancer risk is known as the cancer slope factor (CSF).

The cancer slope factor (CSF) is an upper-bound estimate for the carcinogenicity potential of the toxic chemical and is used to calculate the cancer health risk by relating the average intake of the carcinogenic chemical to the incremental risk of an individual developing a cancer over his/her lifetime (USEPA, 2008). Cancer slope factors are classified based on the route of exposure pathway. Hence, in this study, only the oral and dermal exposure routes were considered. Therefore the cancer slope factors used are the oral and dermal cancer slope factors respectively.

Similarly, the term non-cancer risk is defined as the probability that an individual within a sensitive population exposed to a toxic chemical may experience a negligible adverse health effects over his/her lifetime (USEPA, 2008). This probability is estimated by comparing it with the reference dose.

In line with USEPA risk assessment guidelines (USEPA, 2008), the reference dose is defined as the highest dose does not cause observable adverse health effects (i.e., NOAEL – No Observed Adverse Effect). If no NOAEL value exist for that particular chemical, then the Lowest Observed Adverse Effect Level (LOAEL) is used by dividing it with an uncertainty factor of 100 to account for interspecies variations (USEPA, 2008). The reference dose (RfD) is also classified just like the cancer slope factors in relation to the exposure pathway. The reference dose considered in this thesis is oral and dermal reference dose for arsenic, cadmium, mercury and lead in the different environmental media.

The USEPA has concluded that arsenic is a class ‘A’ human carcinogen based on evidence from different studies that have established that exposure to arsenic in different environmental media causes cancers of the skin, liver, lung, blood and the bladder (USEPA, 2008, Tseng, 2005). It has therefore recommended an oral cancer slope factor value of 1.5 mg/kg/day. Since, absorption of arsenic from the gastrointestinal walls is almost 100%, there is no need to adjust the oral cancer slope factor value in order to get the dermal cancer slope factor.

Similarly, the USEPA recommended oral RfD value of 3×10^{-4} mg/kg/day is used to quantify the non-cancer health associated with exposure to arsenic in the different environmental media. Since, the absorption of arsenic via oral route is approximately 100%, there is no need to adjust the oral reference dose value in order to calculate the dermal reference value for arsenic (USEPA, 2008).

In the case of cadmium, the USEPA has concluded that it is a class ‘B1’ human carcinogen based on evidence from studies that linked exposure to cadmium in environmental media to various cases of cancer in human beings.

As such, an oral cancer slope factor value of 6.1×10^2 mg/kg/day was used to quantify the cancer health via exposure to cadmium in the environment media. Since, oral absorption of cadmium in drinking water is not approximately 100%, hence, in line with USEPA risk assessment guidelines, the oral cancer slope factor (USEPA, 2008) value was adjusted.

The recommended oral reference dose value for cadmium in drinking water by the USEPA is 5×10^{-4} mg/kg/day, and this value was used in the calculation of non-cancer health via oral ingestion of cadmium in drinking water (USEPA, 2008). This value is based on the highest level of cadmium recorded in human renal cortex after oral ingestion of cadmium in drinking water. Again, because oral absorption of cadmium in drinking water is less than 5%, the oral reference dose for cadmium would be adjusted in order to calculate for the dermal exposure route. This will be done so as to be consistent with the USEPA guidelines.

According to USEPA (2008), mercury is not a classifiable human carcinogen. It has a carcinogenicity potential of class 'D'. This classification is based on absence of evidence from scientific studies to suggest that it is a human carcinogen. The oral reference dose for mercury in drinking water is 3×10^{-4} mg/kg/day. Since, oral absorption of mercury in drinking water is less than 7%, there is the need to adjust the oral reference dose, so as to be able to calculate for the dermal reference dose for mercury in drinking water and the other environmental media (USEPA, 2008).

Similarly, studies conducted by the USEPA have revealed that lead is not a human carcinogen. It has a carcinogenicity potential of class 'D', which is based on the fact no scientific studies have been able to link exposure to lead in drinking water or other environmental media to cancer cases among human beings.

However, the oral reference dose for calculation of non-carcinogenic risk via drinking of contaminated water is 3.6×10^{-2} mg/kg/day. Also, in line with USEPA risk assessment guidelines, the oral reference dose for lead in drinking water has to be adjusted so as to calculate the dermal reference dose, since its oral absorption is less than 5% (USEPA, 2008).

2.6.3 Status and trends of human health risk assessment in mining communities in Ghana.

The concept and practice of human health risk assessment of toxic chemicals is a new concept that is fairly gaining grounds and usage in Ghana (Obiri *et al.*, 2010). It has been applied by few scientists in Ghana to quantify the negative health risks due to exposure toxicants. For instance, Adu-kumi (2011), applied concepts of risk assessment to estimate the health effects associated with exposure to Lindane in Ghana and found out that, the estimated daily intake (EDI) of lindane by an average Ghanaian adult of body weight 60 Kg was 0.000038 mg/(kg-bw)/day, which lower than the acceptable daily intake (ADI) value of 0.005 mg/(kg-bw)/day set by the Joint Meeting on Pesticide Residues of the World Health Organisation (WHO, 2010); while Atiemo *et al.*, (2011) also calculated the health risks associated with exposure to toxic chemicals in vehicular fallout along the Tema motorway.

As noted by Armah *et al.*, (2014), several scientific studies conducted in Ghana have revealed that residents of mining communities are constantly exposed to toxic chemicals such as As, Cd, Pb, Mn, Hg, etc in rivers/streams, groundwater, sediment, soil and food crops. Other studies have also found high levels of As, Cd, Hg and Pb in human human samples such as blood, fingernails, hair and urine due to their continuous exposure in environmental media and food crops,

Only few of such studies used the results of the levels of the aforementioned chemicals to calculate the health risk associated with these chemicals (Armah *et al.*, 2012, Obiri *et al.*, 2006a; Cobbina *et al.*, 2011). For example, a study by Obiri *et al.*, (2006), focussed on quantifying the cancer health risks associated with arsenic by workers of a mining company. Other studies by Obiri *et al.*, 2006b; Obiri *et al.*, 2010a; Armah *et al.*, 2012; Ofosu-Aseidu *et al.*, 2013 and Cobbina *et al.*, 2013 rather focussed on assessing the cancer and non – cancer health risk faced by resident adults and children from either oral or dermal contact with As, Cd, Mn, Pb, Hg, etc in either water, soil, sediment or food crop.

Similarly, Ansa-Asare *et al.*, (2014), found out that the cancer health risk associated with ingestion of arsenic in river Tano by resident adult via central tendency exposure parameters (CTE) 1.4×10^{-2} and 1.8×10^{-2} by reasonable maximum exposure parameters. This means that, approximately 1 out of every 100 adult, who ingest arsenic in river Tano is likely to suffer from cancer related disease by CTE parameters as against approximately 2 of every 100 adult in the same area likely to suffer from cancer related diseases by RME parameters respectively. The results obtained by Ansa-Asare *et al.*, was found to be higher than the USEPA acceptable cancer health risk value of 1×10^{-4} to 1×10^{-6} , i.e., approximately 1 case of cancer in every 10,000 people to 1 case of cancer per every 1,000,000 people.

Other Ghanaian scientists have also applied the concept of risk assessment to estimate the cancer and non-cancer health risk associated with exposure to heavy metals. For example, a study by Darko *et al.*, (2019), assessed the human health risk and bioaccessibility of heavy metals in topsoils in the Gbani mining community in the Upper East Region of Ghana.

The authors found out that the non-cancer human health risk expressed in terms of hazard index was less than 1 and is within the USEPA acceptable value of 1.

Also, Tay et al., (2019), applied non-carcinogenic risk assessment techniques to assess the health faced by residents in the Amansie and Adansi Districts in Ashanti Region of Ghana. They found out that, the health risks associated with heavy metals such as iron, cadmium, lead and mercury were below the acceptable guideline values. Furthermore, Bempah and Ewusi (2016), recorded high hazard index in heavy metal contamination and human health risk assessment around Obuasi gold mine.

2.7 Neutron Activation Analysis (NAA) as an analytical technique for determination of concentration of heavy metals in environmental and bio-samples

Neutron Activation Analysis (NAA) is an analytical technique used for both qualitative and quantitative measurement of chemical elements and their total masses present in representative portions of samples. As an analytical technique, it is used for identification and quantification of the elements, above all of the trace elements in the sample.

NAA has applications in chemistry but also in other research fields, such as geology, archaeology, medicine, environmental monitoring and even in the forensic science. The technique is based on the conversion of a stable atomic nuclei into radioactive ones by bombarding with neutrons and measuring the radiation emitted as result of the decay of the radioactive nuclei.

NAA techniques can be put into two main categories. These categories are (1) prompt gamma-ray neutron activation analysis (PGNAA), where measurements take place during irradiation, or (2) delayed gamma-ray neutron activation analysis (DGNAA), where the measurements follow radioactive decay. The delayed gamma –ray neutron activation analysis more often comes to mind when one mentions NAA it is generally assumed that measurement of the delayed gamma rays is intended.

2.7.1 Basic Principles of Neutron Activation Analysis

An incident neutron is absorbed by a stable nucleus (target nucleus) forming a compound nucleus in an excited state. The compound nucleus de-excites by emitting an elementary particle (neutron, proton or electron), an alpha particle or a photon (gamma ray). The product nucleus is either stable or radioactive. For a radioactive product nucleus, it decays by emitting beta particle(s) and delayed gamma ray(s). Measurement of the delayed gamma ray emitted by the radioactive product nucleus is termed as “normal” neutron activation analysis and is generally represented by equation (2.6) (Landsberger, 1994).

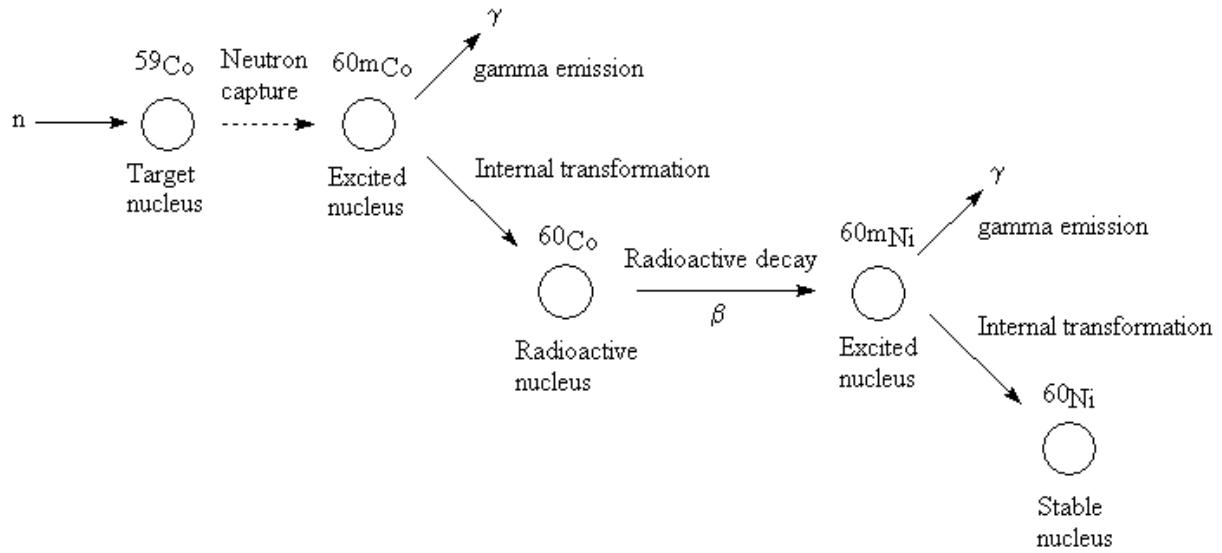
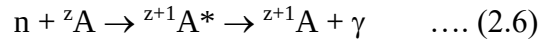


Fig. 2.3: Mode of analysis of concentration of elements in samples using NAA techniques



Where;

n = high flux neutron used in irradiating the target nucleus of the compound

${}^z\text{A}$ = the target nucleus

${}^{z+1}\text{A}^*$ = the target nucleus of the compound in an excited state which de-excites to emit gamma rays

${}^{z+1}\text{A}$ = the product obtained after irradiating the target nucleus. ${}^{z+1}\text{A}$ is radioactive.

According to Landsberger, (1994), the radioactive substance produced in equation (2.6) above undergoes decaying and the number of activity produce is governed by the usual decay equation as shown in equation (2.7);

$$R = N \int_{\text{whole energy range}} \sigma(E) \phi(E) dE \quad \dots \quad (2.7)$$

Where;

R = the reaction rate,

ϕdE = the flux of neutrons with kinetic energy between E and E + dE in n.cm⁻²s⁻¹,

$\sigma(E)$ = the neutron capture cross-section in cm². The neutron capture is defined as the probability of a relative capture reaction occurring between the neutron and a nucleus given in terms of the incident neutron,

N = the number of atoms of the element in the target nucleus of the sample.

The radioactivity in the target nucleus of the sample according to Landsberger (1994) is given by equation (2.8) below;

$$A = \sigma \phi \left(\frac{m}{M} \right) N_A S D C \theta P_\gamma \eta \quad \dots \quad (2.8)$$

Where;

A = the measured activity in Bq from the product of an expected reaction;

σ = the activation cross-section of the reaction in cm²;

ϕ = the activating neutron flux in n.cm⁻²s⁻¹;

m = the mass of the element in g;

M = the atomic weight of the element to be determine in g/mol;

N_A = the Avogadro's constant, which is 6.022 x 10²³ atoms/mol;

S = the saturation factor which is given as $S = [1 - \exp(-\lambda t_1)]$, λ is the constant of the Reactive product and t_1 is the time for irradiation;

- D = the decay factor, which expressed as $D = \exp(-\lambda t_d)$, and t_d is the time for decay;
- C = the correction factor for nuclide decay during the counting time expressed as $C = [1 - \exp(-\lambda t_c)]$, and t_c is the time for counting;
- θ = the relative natural isotopic abundance of the activated isotope;
- P_γ = the probability of emission of photon with energy E; and
- η = the detector efficiency for the measured gamma radiation energy

By using the comparator method based on equal weights for sample and standard, same irradiation, same decay, same counting times and using the same geometry, equation (2.8) can be simplified as follows;

$$C_{sam} = C_{std} \left(\frac{A_{sam}}{A_{std}} \right) \quad \dots \quad (2.9)$$

Where;

- C_{sam} = the unknown concentration of the element in the sample,
- C_{std} = the known concentration of the element in the standard,
- A_{sam} = the activity of the sample, and
- A_{std} = the activity of the standard.

Using the terms D and C from equation (2.8) in equation (2.9) and normalising the weights between the standards, equation can re-written in ppm as;

$$C_{sam} = C_{std} \left(\frac{A_{sam}}{A_{std}} \right) \left(\frac{D_{std}}{D_{sam}} \right) \left(\frac{C_{std}}{C_{sam}} \right) \left(\frac{W_{std}}{W_{sam}} \right) \quad \dots \quad (3.0)$$

Where;

W_{sam} = the weight of the sample,

W_{std} = the weight of the standard, and the other symbols have their meanings as defined in equations (2.8) and (2.9).

From equation (3.0), the concentration of element in the sample can be determined if the element of interest in the sample is radioactive and capable of emitting at least one gamma-ray photon. The emitted gamma ray photon is detected on a gamma ray detector using HPGE. However, if the activation product after irradiation is stable, and as such cannot emit gamma ray photon, then it cannot be detected.

According to Faanu et al., (2011), in the NAA, the duration of irradiation of the sample depends on the characteristics of the sample. The duration of the irradiation depends on the neutron flux density, mass of the sample and the efficiency of the gamma detector.

The samples to be irradiated are specially sealed in capsules and transferred to the reactor core and irradiated with high flux neutrons.

2.7.2 Advantages of Neutron Activation Analysis (NAA)

According to Alfassi (1994), NAA has comparative advantage over the conventional methods for analysing chemicals substances such as atomic absorption spectrometry (AAS), X-Ray Fluorescence (XRF), X-Ray Diffraction (XRD) or the induced coupled plasma – mass spectrometry (ICP-MS). Notable among them are as follows:

1. The NAA has high sensitivity that allows the detection of elements in the order of 10^{-6} to $10^{-12}\mu\text{g}$.
2. NAA is basically a multi-elemental technique. This is due to the fact that most elements in the sample become radioactive during the irradiation of the sample; and if each of these radioactive elements are “isolated” instrumentally, and their concentrations or relative abundances in the earth crust determined simultaneously.
3. NAA is non-destructive analytical technique, i.e., the sample is not destroyed during the analysis process as other conventional analytical procedure would do.
4. NAA is an accurate ($\sim 1\%$ accuracy) and precise ($\sim 5\%$ precision) method of measuring elemental concentrations.

2.7.3 Disadvantage of Neutron Activation Analysis (NAA)

1. The high cost of NAA equipment and irradiation facilities makes it an expensive analytical technique.
2. The inability of NAA to determine the chemical state of the element of interest in the sample.
3. The need to work with significant levels of radioactivity, with their attendant radiation safety and legal problems.
4. The long times needed to complete some analyses, and complex analysis sometimes needed to unscramble the γ -ray spectra in a given experiment.

2.8 Review of other analytical techniques used in the determination of heavy metals in environmental and bio-samples

According to Armah et al., (2014), mining, processing of gold from its ores generates a lot of toxic wastes. Improper discharge of mining wastes have been found in many studies to cause heavy metal pollution, which lead to serious environmental, social and human health problems. Regulatory agencies, policy makers as well as the general public who are exposed to toxic chemicals in the environment require accurate and timely information on concentrations of the different heavy metals in various environmental and bio-samples to make important decision with the view of minimizing the negative health impacts associated with exposure to such toxic substances.

It is within this context that different analytical techniques together with its instrumentation have been developed to measure the concentration of heavy metals such as arsenic, cadmium, mercury and lead just to mention a few in various environmental and bio-samples. The most predominant techniques are atomic absorption spectrometry (AAS); atomic emission/ fluorescence spectrometry (AES/AFS); inductively coupled plasma mass spectrometry (ICP-MS); inductively coupled plasma optical emission spectrometry (ICP-OES); neutron activation analysis (NAA), X-ray fluorescence (XRF); and anodic stripping voltammetry (AVS).

According to Helaluddin et al., (2016), the type of sample and concentration range of heavy metal to measured usually influence the selection of the analytical technique to be used. In a systematic review of analytical techniques used in the determination of heavy metals in environmental and bio-samples by Helaluddin et al., (2016), the authors found out that the atomic

absorption/emission spectroscopy techniques does not only provide analysis for a wide range of heavy metals but also ensure immense reliability by exhibiting good precision and accuracy at trace level at a moderately lower cost of analysis (as shown in Table 2.0).

Table 2.0: Comparison of analytical instruments used in the determination of heavy metals in environmental and bio-samples

Parameter	Flame AAS	Graphite Furnace AAS	Hydride Generation AAS & Cold Vapour AAS	ICP – OES	ICP – MS
Detection Limit	Very good for some elements ppm	Excellent for some elements ppb	Excellent for hydride forming metals ppb	Very good for most elements ppb	Excellent for most elements ppb
Sample throughput (i.e., time required for analysis)	10-15 secs per analyte	5-6 mins per analyte	30-50 secs per analyte	60 elements in < 1 minute	All elements in < 1minute
Interferences Spectral	Few	Few	Very Few	Many	Few
Chemical (matrix)	Many	Very Few	Very Few	Few	Some
Sample volume required	Large	Very Small	Large	Medium	Very small to medium
Cost per analysis	Moderate	Moderate to high	Moderate to high	High	Very High

Source: Helaluddin et al., (2016)

CHAPTER THREE

METHODOLOGY

3.0 General description and justification for selecting the study sites

Gold deposits in Ghana stretch from the coast to the north (Fig. 3.0). However, mining of gold in the northern part of Ghana had not received much attention in the past until recently as compared to the south where the mining activities even pre-dates Ghana's independence. The two (Nabdam and Bole-Bamboi Districts) mining sites in the north were selected because they are important sites where small-scale mining operations take place. This is expected to impact negatively on the environment and the health of residents.

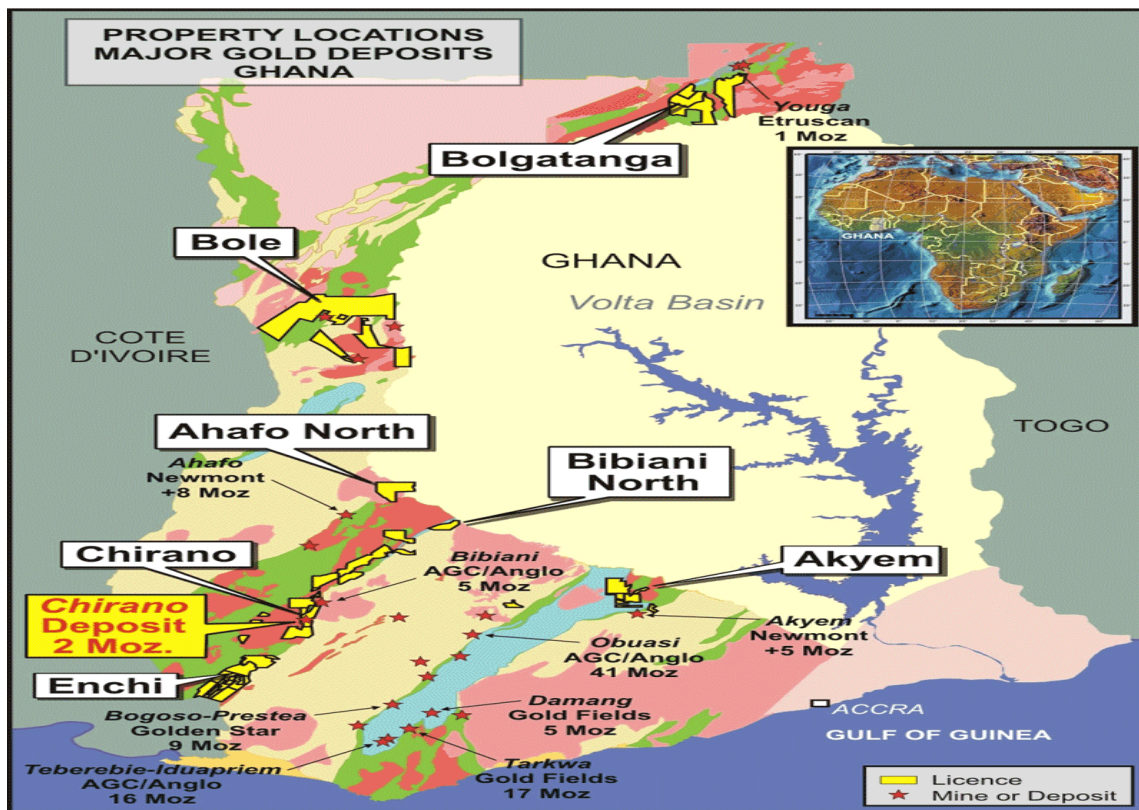


Fig. 3.0: Map of Ghana showing major gold deposits. Source: Minerals Commission

Similarly, the two mining sites selected in southern Ghana were Tarkwa Nsuaem Municipal Assembly (TNMA) and Prestea Huni Valley District (PHVD). These mining sites were chosen because of the long history of mining, it is expected to generate huge environmental impacts that could have significant negative impact on the health of residents. According to Kusimi and Kusimi (2012), out of 16 multinational large-scale mining companies in Ghana, eight of these mining companies are located in the Prestea Huni Valley District and the Tarkwa Nsuaem Municipality, in addition to the large number of artisanal small-scale mining companies.

3.1.0 The study areas

3.1.1.0 Socio-demographic and climate of Tarkwa Nsuaem Municipality (TNMA)

The Tarkwa Nsuaem Municipality is one of the twenty-two districts in the then Western Region. The following large-scale mining companies; Gold Fields Ghana Ltd (GGL) – Tarkwa Mine, AngloGold Ashanti – Iduapriem Mine (AGA) and Ghana Manganese Company Ltd – Nsuta (GMC) are found in Tarkwa, the administrative capital of the Tarkwa Nsuaem Municipality. Other large-scale mining companies and artisanal small-scale gold mining companies are located in the surrounding towns in the municipality.

The municipality has a total land surface area of 2,354 km² and lies between latitude 5°14' N and 5°25'N; and on longitude 1°53'W and 2°42'W (Fig.3.1). The municipality lies within the tropical evergreen rain forest agro-climatic zone. It has bimodal rainfall pattern. The first and largest peak occurs in June, whilst the second and smaller peak occurs in October. Around 53% of all rain in the region falls between March and July.

The mean annual rainfall is approximately 1874mm with min and max values of 1449 mm and 2608 mm, respectively. The area is very humid and warm with temperatures between 26-30°C (Dickson and Benneh, 1980).

According to the 2010 Population and Housing Census Analytical for the municipality, it has a total population of 90,477, with males accounting for 51.6% and females 48.4%. The municipality has a youthful population (57.5%), children aged below 15 years (38.1%) and an elderly population of 4.4% (i.e., people aged 60 years and above). This means that, the municipality has a higher dependency ratio of 69.6%, with the females (72.6%) being more dependent than the males (67.1%). For most of the resident who live in the rural areas where access to potable water such as pipe-borne water and education are virtually non-existent (GSS, 2010), they depend on streams, rivers and boreholes for their source of drinking water. The people main occupation for most residents in the metropolis is farming as well as scouting through the nearby forest reserves for non-timber forest products such as snails, oil palm, herbs and so on as their source of income. Only a small fraction of the population are employed by either the mining companies or the legal artisanal small-scale mining companies. The high dependency ratio in the municipality can be attributed to the fact that most of the residents especially those who live in the rural areas and lost their farmlands to mining have no alternative livelihood support (GSS, 2010; CHRAJ, 2008).

3.1.1.1 Geology and hydrogeology of Tarkwa Nsuaem Municipality

Geologically the municipality lies on the Birimian and the Tarkwaian geological formations. The Birimian and Tarkwaian rock systems are important economic formations as a result of the fact that they are rich with minerals like gold, bauxite and iron ore just to mention a few (Kesse, 1985). For instance, the gold deposits are found in the following auriferous formations:

- Reef vein or lode-type gold deposits. This is defined as gold deposits found in the quartz-veins. They are also referred to as gold bearing lode or dyke. This type of gold deposits are usually found in the Birimian rock systems; and
- Auriferous quartz-pebble conglomerates that occur as intrusive veins in the phyllites and greenstones within the Tarkwaian and Birimian rock system. The auriferous can be found in two places, these are; (1) near or at the contact of the lower and upper Birimian rocks, and (2) the reefs within the Birimian rocks.

As noted by Kuma and Younger (2001), the Tarkwaian and Birimian rocks are made up of coarse, poorly sorted immature sediments with low roundness typical of braided stream environment (Tay and Momade, 2006). Also, the Tarkwaian rock system has sediments that have been subjected to low grade metamorphism into middle greenschist and amphibolites facies (Kesse, 1985).

The hydrogeological characteristics of both surface and groundwater in the Tarkwa Nsuaem Municipality depends on the water-rock interactions. This water-rock interaction aids in the dissolution of mineral ions such as that of heavy metals into both ground water and surface water resources. Hence, the concentrations of the mineralized ions in both surface and groundwater from the municipality depends on the extent of exposure of these minerals in the Tarkwaian and Birimian rock system (Kortatsi, 2006).

The dissolution of the mineral content in the Tarkwaian and Birimian rock system is usually governed by redox reaction. For instance, exposure of arsenic-sulphides rocks such as arsenopyrite (FeAsS), and pyrite, anargite (Cu_3AsS_4) present in the Birimian formation of the study area, tennantite [$(\text{Cu}, \text{Fe})_{12}\text{As}_4\text{S}_{13}$], realgar (As_2S_2), orpiment (As_2S_3) and arsenides of silver and lead to the atmosphere, result in them be oxidized by O_2 , Fe^{3+} and NO_3^- , which eventually contribute to the release of ions of arsenic and other heavy metals into both surface and groundwater. Similarly, the reduction process occurs when certain microorganism in the environment in the presence of organic matter and FeOOH acting as a source of oxygen mediate to reduction process which reduces As^{+5} to As^{3+} ions as well as that of other heavy metals (Sambu and Wilson, 2008; Harter, 2003).

3.1.1.2 Sample locations and sampling techniques in Tarkwa Nsuaem Municipality (TNMA)

Random sampling techniques were adopted in selecting the sampling sites where environmental samples such as water and sediment were taken for this study in the Tarkwa metropolis. A list of all the communities in the metropolis were written down on pieces of papers. The papers were then put in a bowl. The bowl was well shaken to ensure that the papers were well mixed, and the names of ten communities were drawn randomly without replacement from the bowl. The aforementioned random sampling procedures were followed in the selection of environmental samples such as water, sediment and the cassava samples for this study in each of the selected communities.

Tables 3.0 discusses names of communities where samples (i.e., water, sediment and cassava) were taken, number of samples taken at each site in the Tarkwa Nsuaem Municipality.

Table 3.0: Sampling locations in the Tarkwa Nsuaem Municipality

Sampling Station	Community	Type of samples collected/Species	Number of samples collected per each station
River Asuman	Abekoase	Surface water and sediment	6
River Achofe	Mile 8	Surface water and sediment	6
Borehole at Abekoase	Abekoase	Groundwater	6
River Ateberebe	Teberebe	Surface water and sediment	6
River Huni	Huniso	Surface water and sediment	6
River Subri	Subriso	Surface water and sediment	6
River Betinyere	Teberebe	Surface water and sediment	6
River Ahumabruen	Adisakrom	Surface water and sediment	6
River Agonaben	Wangarakrom	Surface water and sediment	6
River Adeyie	Aboponiso	Surface water and sediment	6
Cassava Farm	Tamso	CSIR-CRI Buroni Bankye	6
	Tarkwa Bansa	CSIR-CRI Buroni Bankye	6
	Effuantah	CSIR-CRI Buroni Bankye	6
	Nsuaem	CSIR-CRI Buroni Bankye	6

Source: Field Data (2013)

In the case of the cassava samples, judgemental sampling techniques were adopted. The reason for this sampling technique was that, the cassava specie used in this study was the ‘CSIR-CRI Buroni Bankye’ which grows very well in the forest agro-ecological zone in the metropolis as well as the Guinea Savannah agro-ecological zone for Bole-Bamboi and Nabdam Districts respectively. The CSIR-CRI Buroni bankye matures in six months. Hence, communities where this type of cassava specie is grown in the Tarkwa Nsuaem Municipality are presented in Table 3.0 above.

3.1.2.0 Socio-demographic characteristics and climate of Prestea Huni Valley District (PHVD)

Bogoso is the administrative capital for the Prestea Huni Valley District Assembly. The district was carved from the then Wassa West District in 2012. The district host the following multinational large-scale mining companies; Golden Star Resources, Prestea-Bogoso Gold Mine. Wexford Gold Limited, Sankofa Goldfields Limited, Goldfields Ghana Limited – Damang Mine, etc. There are sizeable number of licensed small – scale miners as well as illegal miners popularly called galamsey operators also work within the area. The district is located between latitude 5°40 and longitude 2°50 in a tropical evergreen rain forest. As such the soil is very rich for cultivation of food and cash crops. Prior to active mining operations of small-sale mines, large-scale mines or illegall mines (popularly called ‘galamsey operators’), the main occupation of the people in the study area, they were primarily subsistence farmers who are engaged in rubber, oil palm and cocoa plantations. Food crops such as maize, cassava, plantain, etec are also cultivated by them (Avortri *et al.*, 2002)

The population of Prestea Huni Valley district and Tarkwa Nsuaem Municipality is approximately 198,112 and is mainly composed of the indigenous Wassa tribe as well as other tribal entities in Ghana (GSS, 2010).. The district lies within the south western equatorial climatic region while the northern part is within the wet semi equatorial climate. Generally, the rainfall pattern follows the northward advance and the southward retreat of the inter-tropical convergence zone that separates dry air from Sahara and the moisture-monsoon air from the Atlantic Ocean.

The north air mass, locally called the Harmattan, brings in hot and dry weather during December to February (Dickson and Benneh, 1980). The area is characterized by double rainfall maxima. The first and largest peak occurs in June, whilst the second and smaller peak occurs in October. Around 53% of all rain in the region falls between March and July. The mean annual rainfall is approximately 1874mm with min and max values of 1449 mm and 2608 mm, respectively. The area is very humid and warm with temperatures between 26-30°C (Dickson and Benneh, 1980).

3.1.2.1 The geology and hydrogeology of the Prestea Huni Valley District

The gold deposits are found in the Birimian volcanic gold belt. Geologically the Birimian volcanic gold belts generally extend to a length of about 100 km and a width of about 40 km, which is separated by a basin of isoclinally folded metasedimentary rocks. It also consist of metamorphosed tholeiitic to calc-alkaline basalts (Abouchami et al., 1990).

According to Kesse (1985), the gold deposits in the Prestea Huni Valley District is found in the phyllites. It range from soft and friable black carbonaceous and graphitic types to hard and compact greyish siliceous types. It is subdivided as follows:

- Grey siliceous phyllites
- Carbonaceous and graphitic phyllites
- Spotted or “Carbonated” phyllites and
- Tuffaceous phyllites

According to Kortatsi (2004), groundwater flow in the Prestea Huni Valley District is localised due to numerous low hills that act as groundwater divides. The Birimian and the Tarkwaian rocks that underlying the district is not highly porous, hence groundwater flow is highly restricted to flow zones along the fissures and joints, quartz veins, and other intrusives (Kortatsi, 2006).

The clay, silts, sandy clays, and clayey sands through which surface water flows through were formed mostly from the weathering of the Birimian and Tarkwaian rocks. Hence, in the district, there two main types of aquifers. These are:

- The aquifer in weathered regolith occurs above the transition zone between fresh and weathered rock. Due to the soils content of clay and silt, these aquifers have relatively high porosity and storage, but low permeability.
- The aquifer in the fractured/fissured zone occurs below the transition zone. They have relatively high transmissivity, but low storage. Yield of shallow wells varies from 0.4 to 18 m³ h⁻¹ with an average of 2.4 m³ h⁻¹. The depth of wells varies between 18 m and 75 m with an average of 35.4 m but has little or no effect on borehole yields

The recharge of groundwater in the area occurs mainly by direct infiltration. In some places groundwater is in hydraulic contact with rivers and recharge from them can also take place (Kortatsi, 2006).

3.1.2.2 Sample locations and sampling techniques in Prestea Huni Valley District (PHVD)

Random sampling techniques as described in section 3.1.1.2 above in this thesis were used in selecting the sampling sites (i.e., surface and ground water as well as sediments) for this study. The names of communities where either surface water or groundwater as well as sediment were collected is summarised in Table 3.1. It also, indicate the number of samples collected from each sampling station.

Table 3.1: Sampling locations in the Prestea Huni Valley District (PHVD)

Sampling Station	Community	Type of samples collected/Species	Number of samples collected per each station
River Aprepre	Dumase	Surface water and sediment	6
River Anikoko	Anikoko	Surface water and sediment	6
River Abodwesh	Himan	Surface water and sediment	6
River Ankobra	Prestea	Surface water and sediment	6
River Amenkuman	Bondaye	Surface water and sediment	6
River Bodwire	Brakwaline	Surface water and sediment	6
River Twiagya	Twiagya	Surface water and sediment	6
River Mansi	Kwameanimpah	Surface water and sediment	6
Borehole	Dumase	Groundwater	6
River Asasre	Prestea	Surface water and sediment	6
Cassava samples	Prestea	CSIR-CRI Buroi Bankye	6
	Bogoso	CSIR-CRI Buroi Bankye	6

Source: Field Data (2013)

Similarly, CSIR-CRI Buroi Bankye were selected from cassava farms located in Prestea and Bogoso. The rationale for selecting this type of cassava species have been explained into details in section 3.1.1.2 above.

3.1.3.0 Socio-demographic and climate of Cape Coast Metropolitan Assembly (CCMA)

The Cape Coast metropolis is bounded on the south by the Gulf of Guinea, on the west by the Komenda/Edina/Eguafo/Abriem District, east by the Abura/Asebu/Kwamankese District and North by the Twifo Heamng Lower Denkyira District. It occupies an area of 122 Km². Cape Coast is the administrative headquarters of the metropolis and the region. The soil is generally lateritic which are obtained from the weathering of granitic rocks. It has double maximum rainfall totalling between 750mm and 1000mm. Most of the residents engage in fish farming and cultivation of food crops. The rest are government workers. The metropolis also lies on the gold bearing Tarkwaian and Biriman rocks systems. As per the 2000 population and housing census, the population of Cape Coast Metropolis was 115,300 (Fig. 3.1).

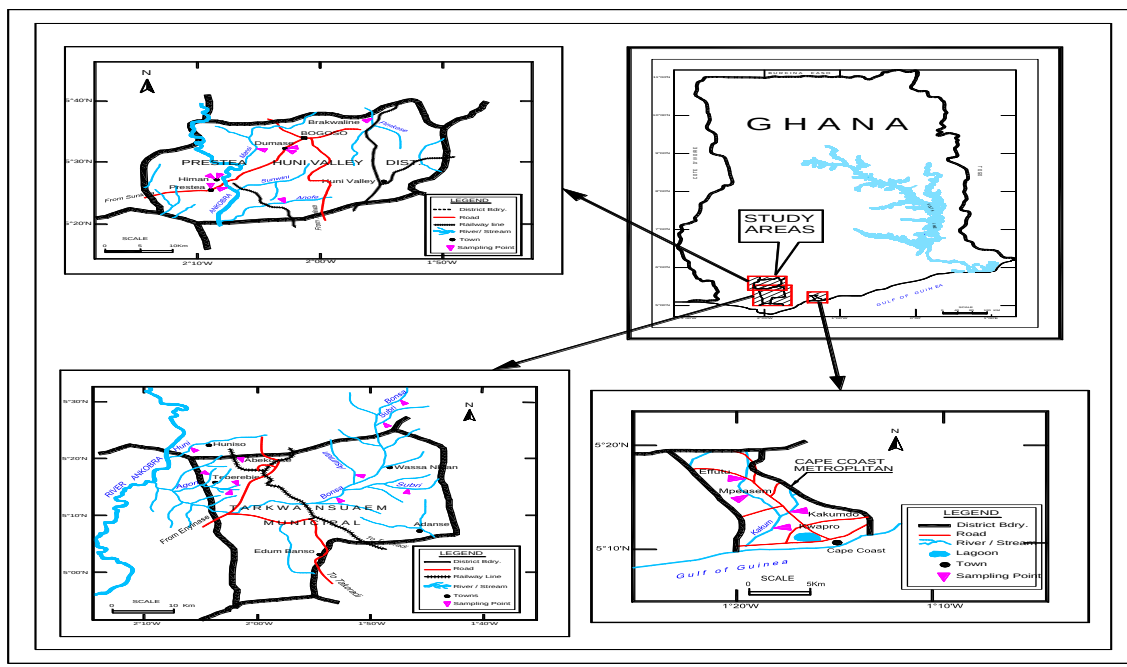


Fig. 3.1 Map showing sampling sites in Tarkwa Nsuaem, Prestea Huni Valley and Cape Coast metropolis. Source: Map produced by the Cartographic section of CSIR - Water Research Institute.

3.1.3.1 Geology and hydrogeology of Cape Coast Metropolis

According to Kesse (1985), the Cape Coast Metropolis falls within the Birimian basin extending from the Western Region and the Kibi belt of the Birimian rock formations. Also, Yidana (2010) has observed that, the geology of the metropolis is underlain by the Precambrian Birimian which is intruded by the Cape Coast –type, granitoid and pegmatites. According to Yidana (2010), groundwater occurs within the fractures of these rocks.

The aforementioned rocks are crisscrossed by mafic dykes (dolerite) which was inferred from aeromagnetic data. Generally, the undulating hills in the metropolis are overlain by sandy and clayey silts whilst the valleys are overlain by clayey gravel with lateritic soils (Yidana et al., 2010; Yidana et al., 2012; Yidana et al., 2011; Kesse, 1985)).

Groundwater availability in the metropolis depends on the fractures, veins and faults within the rocks. For examples, when the quartz veins and pegmatites are fractured and to some extent brecciated as a result of brittle deformation as a consequence of Proterozoic Eburnean orogenic activities, water aquifers within these rocks can be abstracted. The water-bearing and yielding capacities of these rocks depend on the extent of these secondary permeabilities (Banoeng-Yakubo et al., 2009; Banoeng-Yakubo et al., 2011).

3.1.3.2 Sample locations and sampling techniques in Cape Coast Metropolitan Assembly (CCMA)

Random sampling techniques were adopted in the selection of the sampling station as well as the type of environmental samples collected from each station is the same as described in section 3.1.1.2 above and have been summarised in Table 3.2 below. The sampling stations have also been shown in Fig. 3.1 above.

Table 3.2: Sampling locations in the Cape Coast Metropolis

Sampling Station	Community	Type of samples collected/Species	Number of samples collected per each station
River Kakum	Kwaprow	Surface water and sediment	6
River Kakum	Kakumdo	Surface water and sediment	6
Asuboni stream	Effutu	Surface water and sediment	6
Asuboni stream	Mpeasem	Surface water and sediment	6
Tap water	Brimsu headworks	Surface water	6
Cassava Farm	UCC Research Farm	CSIR-CRI Buroni Bankye	6

Source: Field Data (2013)

In the case of the cassava samples, judgemental sampling techniques were adopted. The rationale for this sampling technique has already been discussed in section 3.1.1.2 above.

3.1.4.0 Socio-demographic and climate of Bole-Bamboi District Assembly (BBD)

The Bole-Bamboi District is one of the seven metropolitan, municipal or district assembly (MMDAs) in the Savannah Region of Ghana. The district was established by Legislative Instrument, LI 1786 of 2004. The district lies between latitudes 8°10.5' and 09° and longitude 1.50E' and 2.45 W. According to the district analytical report of the 2010 Population and Housing Census, the district has a population of 61,593 comprising 51.4 percent of males and 49.6 percent of females.

It is one of the sparsely populated district in the Savannah region with a population density of about 10 persons per kilometer square

The district is inhabited by the following ethnic groups: Gonja, Vagla, Safalba and Mo. Residents in the district are predominantly farmers with very few engage in small-scale artisanal gold mining. The district also lies within the Guinea Savannah agro-ecological zone.

3.1.4.1 Geology and hydrogeology of Bole-Bamboi District

According to Kesse (1985), the geology of the district is underlain by the Precambrian basement consists mainly of rocks from the Birimian Supergroup and associated granitoid intrusions, although sediments and metamorphosed sediments of the Tarkwaian Group are also present to a smaller extent as a result of the uplift and erosion of Birimian rocks. Also, a small portion of the Buem Structural Unit is found within the south-eastern part of the district (Osae et al., 2006).

The Birimian Supergroup features a series of narrow, sub-parallel northeast and north trending greenstone belts of mafic to intermediate volcanics, volcanoclastics, and interbedded sediments intruded by intermediate belt granitoids showing extensive granitization. Gold deposits are found within these rocks.

Groundwater occurrence and flow in this context are therefore mainly controlled by secondary porosity as a result of chemical weathering, faulting and fracturing (from tectonic activity and isostatic uplift).

3.1.4.2 Sample locations and sampling techniques in Bole-Bamboi District (BBD)

Gold mining activities are predominantly held in the Tinga community and was randomly selected. Random sampling techniques were adopted in the selection of boreholes drilled in the community to provide alternative source of drinking water (Fig. 3.2). In addition, the only stream in the community which is also used for washing of gold ores by small-scale mining operations including the illegal ones was also selected. This stream also serves as the source of water for domestic activities of residents. The number of environmental samples collected from each station has been summarised in Table 3.3 below. The sampling stations have also been shown in Fig. 3.2 above.

Table 3.3: Sampling locations in Tinga community within the Bole-Bamboi District

Sampling Station	Community	Type of samples collected/Species	Number of samples collected per each station
Borehole 1	Tinga	Groundwater	6
Borehole 2	Tinga	Groundwater	6
Borehole 3	Tinga	Groundwater	6
Borehole 4	Tinga	Groundwater	6
Stream at Tinga	Tinga before the stream is used to wash the gold ores	Surface water and sediment	6
Stream at Tinga	Gold washing portion	Surface water and sediment	6
Stream at Tinga	Downstream of gold washing portion	Surface water and sediment	6
Cassava Farm	Tinga	CSIR-CRI Buroni Bankye	6

Source: Field Data (2013)

In the case of the cassava samples, judgemental sampling techniques were adopted. The rationale for this sampling technique has already been discussed in section 3.1.1.2 above.

3.1.5.0 Socio-demographic and climate of Nabdam District Assembly (ND)

The Nabdam district was carved out from the Talensi-Nabdam district in 2012 by an LI 2105. It has a total land size of 249.9 km². The district has bimodal climatic season, i.e., the rainy season which is usually erratic begins from May and end in October each year, while the dry season, which is characterized by dry, warm and hazy weather condition start from October and end in April each year. It has an annual rainfall of 950mm.

The socio-economic livelihood of residents in the district depends of agriculture. The inhabitants of the District are mainly Nabit and Guruni speaking people. They both belong to the Nabdam ethnic group (GSS, 2010). The District lies on latitudes 10° 47" and 10° 57" North and Longitudes 0° 31" and 1° 15" west.

3.1.5.1 Geology and hydrogeology of Nabdam District (ND)

According to Kesse (1985), the geology of the Nabdam district is made up of the Birimian metavolcanic and metasedimentary rocks. These rocks also consist of andesitic and basaltic lavas, tuff, schist, amphibolite, phyllite, quartzite and metaconglomerate. They are intruded by granite.

Groundwater storage and flow in the District are controlled by the thickness of the overburden, the degree of decomposition, and the type and intensity of fracturing (Adugbire et al., 2010) of the aforementioned rocks. The groundwater is also found in fractures and faults mostly located within schist, phyllites, quartz veins and decomposed granites to depths of about 18 and 50 m. Water quality is fairly good, although there are reported cases of faecal coliforms in some wells.

3.1.5.2 Sample locations and sampling techniques in Nabdam District (ND)

Gold mining activities in the Nabdam District are held in the Nangodi community and was randomly selected (Fig. 3.2). Random sampling techniques were adopted in the selection of boreholes drilled in the community to provide alternative source of drinking water. In addition, the hand dug-out wells as well as community dug-out wells which are also used for both washing of gold ores by small-scale mining operations including the illegal ones and by residents for domestic chores were selected for this study. The number of environmental samples collected from each station has been summarised in Table 3.4 below. The sampling stations have also been shown in Fig. 3.2 above.

Table 3.4: Sampling locations in Nangodi community within the Nabdam District

Sampling Station	Community	Type of samples collected/Species	Number of samples collected per each station
Borehole 1	Nangodi	Groundwater	6
Hand dug-out well A	Nangodi	Groundwater	6
Hand dug-out well B	Nangodi	Groundwater	6
Dug-out well A	Nangodi	Surface water	6
Dug-out well B	Nangodi	Surface watert	6
Cassava Farm	Nangodi	CSIR-CRI Buroni Bankye	6

Source: Field Data (2013)

In the case of the cassava samples, judgemental sampling techniques were adopted. The rationale for this sampling technique has already been discussed in section 3.1.1.2 above.

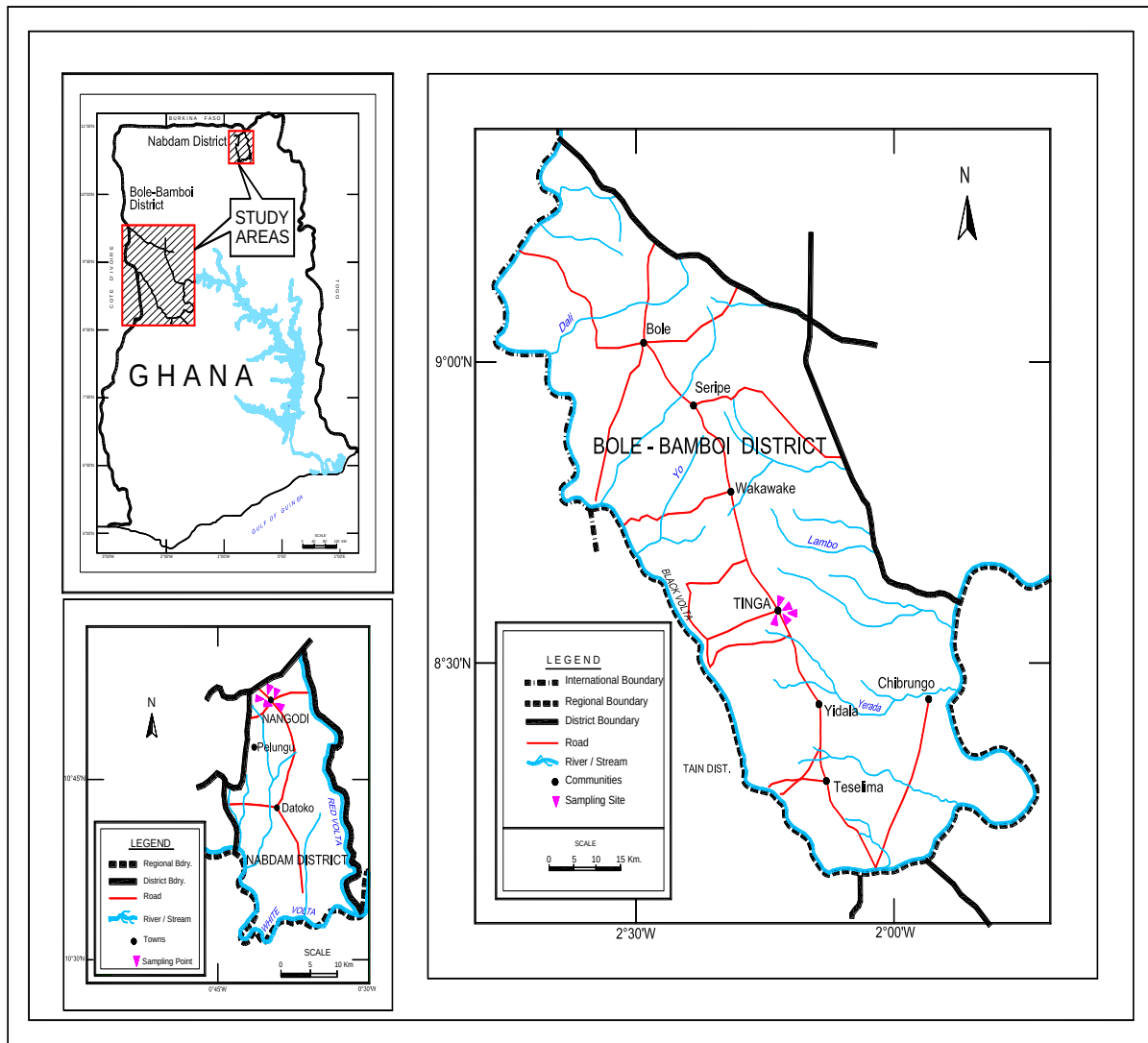


Fig. 3.2: Map showing sampling sites in Nangodi (Nabdam District) and Tinga (Bole-Bamboi District) study areas. Source: Map produced by the Cartographic section of CSIR – Water Research Institute.

3.2 Collection, preservation and storage of environmental samples

3.2.1 Collection, preservation and storage of water samples

Water samples were obtained from rivers, streams, boreholes, hand dug/dug-out wells and tap water in the study areas as described in Tables 3.0 to 3.4 above. A volume of 1.5L of the water samples were collected into plastic containers that has been rinsed with 1:1 concentration of HNO₃ acid and double distilled water and fixed with identification labels for the determination of heavy metals and metalloids. The samples were stored in an ice – chest containing ice-blocks at a temperature of 4°C and later conveyed to the CSIR- Water Research Institute laboratory for analysis. In the laboratory, the acidified samples were filtered using Whatman’s No. 0.45µm filter paper. The 0.45µm membrane filter paper was used because the analyte of interest in this work is the total dissolved metals. pH, turbidity and conductivity of the samples were determined using hand potable pH, turbidity and conductivity meters respectively on the field (AWWA, 1998).

Additional 1.5 L water samples were collected from the same sampling point for the determination of physico–chemical parameters such as pH, turbidity, conductivity and TDS. These samples were not acidified with nitric acid but were stored in an ice – chest at 4°C and conveyed to the laboratory for analysis.

3.2.2 Collection, preservation and storage of sediment samples

10g of sediments from the beds of rivers and streams in the study areas were collected at the depth of 30 cm from the bed of each stream/river at each sampling point as described in Tables 3.0 to 3.4 above into plastic containers with identification labels fixed using Teflon – coated auger.

The samples were stored in an ice – chest at a temperature of 4°C containing ice-blocks and later conveyed to CSIR-Water Research Institute for laboratory analysis. The Teflon – coated auger was washed after each sampling so as to avoid cross contamination (AWWA, 1998).

3.2.3 Collection, preservation and storage of cassava samples

CSIR-CRI buroni bankye cassava species samples were collected each of the sampling stations as discussed in Tables 3.0 to 3.4 above. 20 g of the cassava samples were uprooted using stainless cutlass and transferred into labelled black polythene bags. They were then placed in an ice – chest containing ice-blocks and then conveyed to the CSIR-Water Research Institute laboratory under 4°C for further analysis.

3.3 Reagents and chemicals used in the digestion/analysis of As, Cd, Hg and Pb in water, sediment and cassava samples

1. 12% w/w Hydroxylamine Hydrochloride solution ($\text{NH}_2\text{OH}.\text{HCl}$): 12 g of previously dried AR grade $\text{NH}_2\text{OH}.\text{HCl}$ was dissolved in 100 mL volumetric flask with 100 mL double distilled water. This solution was prepared weekly.
2. 5% w/w Potassium Permanganate solution (KMnO_4): 5 g of previously dried AR KMnO_4 was dissolved in 100 mL volumetric flask with 100 mL double distilled water. This solution was prepared weekly.

3. 3% v/v Hydrochloric acid solution (HCl): 30 mL of AR 36% m/v concentrated HCl was diluted to 1 L in a 1000 mL volumetric flask with double distilled water.
4. 1.1% Stannous Chloride solution ($\text{SnCl}_2 \cdot 2\text{H}_2\text{O}$): 1.1 g of previous dried AR $\text{SnCl}_2 \cdot 2\text{H}_2\text{O}$ was dissolved in 50 mL of 3% v/v HCl solution and later made up to 100 mL with double distilled water in 100 mL volumetric flask. This solution was prepared on weekly basis.
5. 5 % w/w Potassium persulphate solution ($\text{K}_2\text{S}_2\text{O}_8$): 5 g of previously dried $\text{K}_2\text{S}_2\text{O}_8$ was dissolved in 50 mL double distilled water in 100 mL volumetric flask. It was later made up to 100 mL using double distilled water. The solution was prepared every week.
6. Aqua Regia: To one volume of 69.0 – 70.5% v/v AR conc. HNO_3 , three volumes of AR conc. HCl was added to it. This solution was prepared fresh before use.
7. Analytical grades of HClO_4 , HCl, H_2SO_4 , etc were used in their concentrated forms.
8. 30% v/v Hydrogen peroxide solution (H_2O_2): This was used in its concentrated form.
9. Mercury standard stock solution (Hg): 1.0 g of previously dried AR HgO was dissolved in 10 mL conc. HCl. It was later diluted to 1 L with 1% v/v HCl solution. The concentrations of the four standard Hg concentrations; 0.5, 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respective solutions were prepared from the Hg standard stock solution. For example, 0.5 $\mu\text{g/L}$ Hg standard solution was prepared by diluting 0.5 mL of Hg standard stock solution prepared above to 1 L with double distilled water in 1000 mL volumetric flask. This procedure was repeated for the other Hg concentrations i.e., 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respectively. These solutions were prepared daily.

10. Arsenic standard stock solution (As): 1.320 g of previously dried AR arsenous oxide (As_2O_3) was dissolved in 25 mL of 20% KOH. Neutralized with 20% v/v H_2SO_4 solution to phenolphthalein end point and diluted to 1 L with 1% H_2SO_4 . The concentrations of the four standards As concentrations; 0.5, 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respective solutions were prepared from the As standard stock solution.

For example, 0.5 $\mu\text{g/L}$ As standard solution was prepared by diluting 0.5 mL of As standard stock solution prepared above to 1 L with double distilled water in 1000 mL volumetric flask. This procedure was repeated for the other As concentrations i.e., 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respectively. These solutions were prepared daily.

11. Cadmium standard stock solution (Cd): Cadmium standard stock solution was prepared by dissolving 1.0 g of AR cadmium metal in 10 mL conc. HCl. It was diluted to 1 L with 1% v/v HCl to give a standard cadmium stock solution. The concentrations of the four standard Cd concentrations; 0.5, 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respective solutions were prepared from the Cd standard stock solution. For example, 0.5 $\mu\text{g/L}$ Cd standard solution was prepared by diluting 0.5 mL of Cd standard stock solution prepared above to 1 L with double distilled water in 1000 mL volumetric flask. This procedure was repeated for the other Cd concentrations i.e., 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respectively. These solutions were prepared daily.

12. Lead standard stock solution (Pb): Lead standard stock solution was prepared by dissolving 1.0 g of AR Pb metal in 10 mL conc. HNO_3 . It was diluted to 1 L with 1% v/v HNO_3 to give a standard Pb stock solution. The concentrations of the four standard Pb concentrations; 0.5, 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respective solutions were prepared from the Pb standard stock solution. For example, 0.5 $\mu\text{g/L}$ Pb standard solutions were prepared by diluting 0.5 mL of Pb standard stock solution prepared above to 1 L with double distilled water in 1000 mL volumetric flask. This procedure was repeated for the other Pb concentrations i.e., 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respectively. These solutions were prepared daily.

3.4 Digestion of environmental samples

3.4.1 Digestion of As, Cd, Hg and Pb in water samples

In the determination of As, Cd and Pb concentrations of the water samples, to every 100 mL of the sample I measured, 5 mL of concentrated. HNO_3 and 5 mL conc. HClO_4 solutions were added. The mixture was heated until the volume was reduced to 20 mL. It was allowed to cool at room temperature and filtered through 0.45 μm filter paper into 100 mL volumetric flask, the final volume was adjusted to 100 mL by adding double distilled water and stored for analysis of As, Cd and Pb (AWWA, 1998).

Similarly, for the determination of Hg concentrations in the water samples, to every 100 mL of the sample I measured, 5 mL of concentrated H_2SO_4 and 2.5 mL of concentrated HNO_3 were added followed by the addition of 15 mL of KMnO_4 . The mixture was heated in a water bath for 2 hours at 95°C .

It was removed and allowed to cool at room temperature, 6mL of hydroxylamine hydrogen chloride was added to reduce the excess permanganate and filtered through 0.45 μm filter paper into 50 mL volumetric flask and stored for analysis (AWWA, 1998).

3.4.2 Digestion of sediment samples for As, Cd, Hg and Pb analysis

In the laboratory, I removed pieces of roots, pebbles and other foreign objects from the sediment samples. The sediment samples were air – dried to a constant weight, homogenized in a porcelain mortar, sieved through 90 μm and made into a composite sample.

For the determination of As, Cd and Hg, to every 1.0 g of the sediment sample I weighed into a flask bottom, 10 mL of 1:1 concentration of HNO_3 and HClO_4 acid were added and refluxed for 15 mins without boiling, and later cooled. This process was repeated for 1 Hr. The solution was then evaporated to 5 ml without boiling and cooled. Two mL of double distilled water and 3 ml of 30%v/v H_2O_2 were added, and warmed until effervescence was minimal, it was followed by the addition of 5 ml concentration of HCl and 10 ml double distilled water, refluxed for another 15 mins, cooled, filtered through Whatman's No. 0.45 μm filter and stored for the analysis of As, Cd, Pb and Hg (AWWA, 1998; USEPA Method 3050B, 2001).

In the case of Hg, to every 1.0 g of the sediment was weighed into a beaker and 5 mL of double distilled water and 5 ml aqua regia were added respectively, heated in a water bath at 95°C for 2 mins, removed from the water bath and cooled at room temperature. An additional 50 mL of double distilled water and 15 ml of KMnO_4 were added, heated in a water bath for 30 mins at 95°C for complete oxidation of mercury in the sediment samples respectively.

The samples were removed from the water bath, cooled at room temperature and 6 mL of hydroxylamine hydrogen chloride solution was added to reduce the excess KMnO_4 before it was filtered through Whatman's 0.45 μm filter paper into a 50 ml volumetric flask. It later came up to the, 50 ml mark with the addition of double distilled water, stored for analysis (USEPA Method 7470A, 1983; AWWA, 1998).

3.4.3 Digestion of cassava samples for As, Cd, Hg and Pb analysis

In the laboratory, the samples were washed thoroughly with double distilled water. The samples were then chopped into pieces, cooked in 400 ml beaker for 15 mins, and mashed in bits using mortar and pestle. The mashed samples were stored in a refrigerator for further analysis.

For the digestion of cadmium, cobalt, manganese, lead, copper, zinc, platinum and arsenic, exactly 2 g of the stored mashed *M. esculenta* (cassava) were weighed into a 500 mL beaker; 40 ml of concentrated HNO_3 was added and left overnight to allow for complete digestion process. After the initial digestion, 10 ml of 30% v/v hydrogen peroxide (H_2O_2) was added to complete the digestion and oxidation of the trace metals in the samples into their ionic forms. The samples were then filtered through Whatman No. 154 filter paper into a 100 ml volumetric flask. The contents of the 100 ml flask were later adjusted to the 100 ml mark with double distilled water (AWWA, 1998).

Similarly, for the digestion of mercury in the samples, 2.0 g of the stored mashed *M. esculenta* (cassava) were weighed into a 100 ml beaker and 4 ml of concentrated HNO_3 followed by the addition of 4 ml concentrated H_2SO_4 were added to the weighed samples and then placed in a water bath at 80°C for 30 mins.

The samples were then cooled to 4°C in an ice bath and 15 ml of KMnO_4 solution were added to the samples followed by 8 ml of $\text{K}_2\text{S}_2\text{O}_3$ and then placed in water bath at 95°C for 30 mins for complete oxidation of the mercury in the samples. The samples were removed, allowed to cool to room temperature; 6 mL of $\text{NH}_2\text{OH} \cdot \text{HCl}$ was added to reduce the excess permanganate colour in the mixture. The samples were then filtered through Whatman No. 154 filter paper into a 100 ml volumetric flask. The contents of the 100 ml flask were later adjusted to the 100 ml mark with double distilled water (AWWA, 1998).

3.5 Determination of pH, Turbidity, Conductivity and Total Dissolved Solids (TDS) in the water samples

3.5.1 pH

The pH of the water samples were measured using a HANNA, pH meter model HI 98130. Prior to the measurement of pH of the water samples, the pH meter was calibrated, with three standard solutions (pH 4.0, 7.0, and 10.0). This was done by submerging the pH probe into the calibrated solutions and holding it for 2 mins. In the determination of the pH of the water sample, the pH probe was submerge into the water samples and the submerged probe was held for 2 mins to ensure a stabilised reading. After the measurement of each sample, the probe was rinsed with deionized water to avoid cross contamination among different samples (AWWA, 1998).

3.5.2 Turbidity

The turbidity of the water samples were determined using HACH Turbidimeter model 2100P. In the determination of the turbidity of the water samples, 10 mL of the sample was poured into the sample holder and placed inside the meter for 2 mins until the reading stabilized. The stable reading was then recorded. To avoid cross contamination, the sample holder was rinsed several times with double distilled water (AWWA, 1998).

3.5.3 Conductivity

In the determination of the conductivity of the water samples, 10 mL of the sample was poured into 100 mL beaker and the probe of Jenway 4510 Conductivity/TDS meter was dipped into the sample. The probe was kept in the sample contained in the beaker for 10 secs until the reading stabilized. The conductivity meter was calibrated using standard solution. After each measurement, the probe was rinsed repeatedly with double distilled water to avoid cross contamination (AWWA, 1998).

3.5.4 Total Dissolved Solids (TDS)

In the determination of the total dissolved solids in the water samples, 10 mL of the sample was poured into 100 mL beaker and the probe of Jenway 4510 Conductivity/TDS meter was dipped into the sample. The probe was kept in the sample contained in the beaker for 10 secs until the reading stabilized. The Jenway 4510 Conductivity/TDS meter was calibrated using standard solution. After each measurement, the probe was rinsed repeatedly with double distilled water to avoid cross contamination (AWWA, 1998).

3.6 Analysis of the digested samples

3.6.1 Cold vapour technique for the analysis of mercury in the digested samples

The cold vapour technique was used in the analysis of mercury concentrations in the digested samples. The samples (i.e., water, sediment and cassava) were analysed using Shimadzu Atomic Absorption Spectrophotometer model 6300 AA coupled with mercury hollow cathode lamp. The instrument was interfaced with a computer. The mercury lamp has a wavelength of 253.7 nm.

The concentration of mercury in the blank was determined as follows, double distilled water was used to zero the instrument. This was followed by using four mercury standard solutions of concentrations; 0.5, 1.0, 1.5 and 2.0 $\mu\text{g/L}$ respectively to calibrate the instrument. The blank solutions as well as the digested sample solutions together with carrier solution 3% v/v HCl and a reducing agent 1.1% w/v SnCl_2 in 3%v/v HCl were automatically sucked into a mixing chamber. This was done by bubbling nitrogen gas from a pump, resulted in the generation of the free mercury from the digest in the presence of reducing agent in the mercury vapour generator. The mercury vapour generated then swept into the absorption cell mounted on the Shimadzu AAS model 6300AA where measurements were made automatically. The cell was aligned in the path of mercury hollow cathode lamp operating at 4 mA and a mercury lamp of wavelength 253.7 nm. The mercury vapour generated was carried into the hollow cathode by an argon gas, where light rays from the mercury lamp was absorbed by mercury atoms in the vapour. The concentration of mercury in the digested samples is proportional to the amount of light rays absorbed by the mercury atoms in the hollow cathode. The instrument parameters have been described in Table 3.5 below (AWWA, 1998).

3.6.2 Analysis of cadmium and lead in the digested samples using Flame Atomic Absorption Spectrophotometer

The Flame AAS (Atomic Absorption Spectrophotometer) Shimadzu model 6300 AA was used to determine the concentrations of cadmium and lead in the digested samples. Double distilled water was first used to zero the instrument. The blank solutions as well as standard solutions of cadmium and lead of concentrations 0.5, 1.0, 1.5 and 2.0 µg/L respectively were aspirated in turns to plot the calibration curve. The calibration curve was stored on the computer for further analysis.

In the determination of cadmium and lead concentrations in the digested samples, 5 mL of the samples were aspirated and burned in the air-acetylene flame to generate lead and cadmium atoms. The cadmium and lead atoms so generated absorb characteristic light rays from cadmium and lead lamps respectively. The amount of light rays absorbed is proportional to the concentration of lead and cadmium atoms in the digested samples (AWWA, 1998).

3.6.3 Analysis of arsenic in the digested samples using the hydride generator for flame AAS

Arsenic concentrations in the digested samples (i.e., water, sediment and cassava) were determined using Shimadzu Flame AAS (Atomic Absorption Spectrophotometer) model 6300AA coupled with arsine hydride generator (Table 3.5). A double distilled water was first used to zero the instrument. After which the arsenic concentrations in the blank were determined by adding 5 mL of 0.4% NaBH₄ and 5 mL of 0.5 M HCl to produce an arsine gas (AsH₃) in the arsine hydride generator.

The generated arsine gas is aspirated and burn in air-acetylene flame to generate arsenic atoms. The arsenic atoms generated then absorb light rays from an arsenic lamp. The amount of light rays absorbed by the arsenic atoms is proportional to the concentration of arsenic ions in the digested samples (AWWA, 1998).

Table 3.5: Instrument parameters for Flame and Cold Vapour AAS for the analysis of arsenic, mercury, cadmium and lead in digested samples

Parameter	Arsenic	Cadmium	Mercury	Lead
Wavelength/nm	193.7	228.8	263.7	217
Slit Width/nm	0.5	0.5	0.5	0.5
Lamp	Arsenic	Cadmium	Mercury	Lead
Current/mA	8	8	8	8
Burner Height/nm	7	7	7	7
Burner Angle/degree	0	0	0	0
Gas Flow Rate, L/min	2	2	2	2
Fuel	Acetylene	Acetylene	-	Acetylene
Supporting Gas	Air	Air	-	Air
Carrier Gas	-	-	Argon	-
Reducing Agent	0.5% NaBH ₄	-	1.1% SnCl ₂	-

Source: Shimadzu AAS, model 6300AA, Operating Manual

3.7 Quality Control

Certified reference material (CRM) such as NIST – 1646a for estuarine sediment, and Norwegian Water Research Institute (NIVA 1042I) for water samples as well as NIST 8432 for cassava samples with concentration of 10µg/L each of As, Hg, Cd and Pb were respectively were used to spike the digested samples solution for arsenic, cadmium, mercury and lead in water, sediment and cassava samples. They were determined in the same way as the blank and samples using the cold vapour technique for the mercury, flame AAS technique for the determination of cadmium and lead as well as arsenic concentrations by the flame AAS coupled with the hydride generator.

The results obtained from such analysis were used in the reproducibility studies presented in Table 3.6.

In the reproducibility studies, the percentage of As, Hg, Cd and Pb recovered by Shimadzu AAS model 6300AA for both the cold vapour technique and flame technique as well as the flame technique coupled with arsine gas generator ranged from 96.3% to 99.7% with a standard error $\pm$ 0.11 to 0.14. The standard error was less than 1, suggesting that the method used in analysing the samples were reproducible (see Table 3.6 below).

Table 3.6: Percentage of As, Hg, Cd and Pb recovered from 10 μ g/L solution during the reproducibility studies

Metal of Interest	Amount recovered from 10 μ g/L					Standard Deviation	Percentage Recovered
	1	2	3	4	Mean		
As	9.76	9.34	9.59	9.81	9.63	0.21	96.3
Hg	9.92	9.96	9.94	9.51	9.83	0.22	98.3
Cd	10.2	9.94	9.91	9.83	9.97	0.26	99.7
Pb	9.60	9.81	9.63	10.2	9.81	0.27	98.1

3.8 Sampling techniques, collection, preparation, storage and analysis of blood samples

3.8.1 Ethical Clearance for the sample collection

An ethical clearance was obtained from the Ghana Health Service, Ethics Committee. Informed consent was sought from participants. In the case of children below the age of 12, the consent was sought from their parents or guardians; while for older children (i.e., $\geq$ 12 years), their consent was sought from their parents and the children themselves.

3.8.2 Exclusion and inclusion criteria

Only participants who met the under listed eligibility criteria were recruited to be part of this study:

- Have been residing in the study area continuously for at least 7 years;
- Give their consent to be part of the study;
- Agree to have their bio-samples taken at a health facility in the study areas;
- Must not be smoking before or currently (Gonzalez-Estecha *et al.*, 2010) the tobacco content in cigarette contains appreciable levels of cadmium, arsenic, etc);
- Has not had any form of dental filling (have had dental filling of mercury amalgam were excluded); and
- Agree to answer a set of questionnaire.

Participants who did not meet the above criterion were excluded.

3.8.3 Sampling techniques for the collection of the human bio-samples

Random sampling technique was used for this section of the thesis. Using Kish-Leslie formula and with an assumption that 50% of households in the study area will report symptoms related to air pollution (a percentage chosen to maximize sample size because we did not find any prior published studies in a similar setting), a precision of 10%, an alpha of 5% and a design effect of 2 to account for clustering of symptoms around households we obtain the required sample size as follows:

$$sample\ size, n = g \times \frac{z^2 pq}{d^2} \quad \dots \quad (3.0)$$

Where:

g = design effect = 2,

p = proportion of respondents showing symptoms of diseases associated with arsenic, cadmium, mercury and lead exposure in the environmental samples,

$q = 1 - 0.5$, hence, $q = 0.5$,

d = precision = 0.1

Assuming a non-response rate of 10%, we estimate a minimum sample size of 210 households within the study area.

The underlying assumption for this formula is as follows:

- 50% of households in the study area will report symptoms related to exposure to arsenic, cadmium, mercury and lead in environmental samples;
- a precision of 10%;
- an alpha of 5% (95% CI)
- a design effect of 2 to account for clustering of symptoms around households.

Based on this calculation, a total of 850 participants who met the exclusion criteria discussed in section 3.8.1 above, were recruited for this study. The breakdown is summarised as follows: 200 participants from Tarkwa Nsuaem Municipality (10 large-scale mine workers, 40 artisanal small-scale gold mine workers, 70 resident adult non-mine workers and 30 resident children); 150 participants from Prestea Huni Valley District (10 large-scale mine workers, 40 artisanal small-scale gold mine workers, 70 resident adult non-mine workers and 30 resident children); 110 participants from Nabdram District (i.e., 40 artisanal small-scale gold miners, 45 resident adult non-

mine workers and 25 resident children); 115 participants from Bole-Bamboi District (i.e., 35 artisanal small-scale gold miners, 50 resident adult non-mine workers and 30 resident children); and 100 participants from Cape Coast Metropolis (i.e., 80 resident adult non-mine workers and 20 resident children).

These recruited study participants voluntarily consented and satisfied the inclusion criterion approved by the Ghana Health Service, Ethical Committee. The participants from the mining communities were both miners and non-miners. Their bio-samples (i.e., whole blood and serum) were taken by the laboratory staff of Ghana Health Service facilities in the study areas.

3.8.4 Collection of whole blood and serum samples

10 mL of venous blood samples were collected from the arm of the respondents into 2 well-labelled containers. The first container has ethylenediaminetetraacetic acid (EDTA); while the second container does not have an anticoagulant using a sterilised needle. The samples were stored at 4°C, and later conveyed to the Ghana Atomic Energy Commission, Ghana Reactor 1 laboratory for analysis. The blood samples were collected very early in the morning before participants eat their breakfast. At the laboratory, the samples were freeze dried, after which the freeze dried samples were weighed into a thin plane polyethylene film using an electronic balance, wrapped and heat sealed into a pellet using a hot rod. The pellets are then packed into a polyethylene capsule (vial) and again heat sealed.

3.8.5 Analysis of As, Cd and Hg in whole and serum blood samples using NAA

3.8.5.1 Description of GHARR-1

The GHARR-1 at the Ghana Atomic Energy Commission is a Miniature Neutron Source Reactor (MNSR). It is a SLOWPOKE type of a reactor, developed and constructed by China Institute of Atomic Energy (CIAE). It is a simple, small, reliable and safe reactor. GHARR-1 adopts the pool-tank structure, and employs highly enriched uranium as fuel, light water as moderator and coolant, and metal beryllium as reflectors. The reactor is cooled by natural convection. The parameters of the instrument is summarised in Table 3.7.

Table 3.7: GHARR-1 Characteristics

S/No.	Parameter	Description
1	Reactor Type	Pool – Tank
2	Rated Thermal Power	30 kW
3	Fuel	UAl ₄ in Al base material
4	U-235 Enrichment	90.2%
5	Core Shape	Cylindrical
6	Core Diameter	23 cm
7	Active Core Height	23 cm
8	Fuel Element Shape	Thin rod/cylindrical
9	Fuel Element in Core	344
10	Total U-235 Loading in core	<1.0 Kg
11	Reactor continuous operating time (at rated power)	>2.5 Hours
12	Refuel period	More than 10 years
13	Burn up	1%
14	Temperature Coefficient	0.01 Milli Kelvin Per Celsius
15	Control Rod (Cd)	One at centre of the core
16	Total number of inner irradiation sites	10
17	Number of inner irradiation sites	5
18	Thermal neutron flux (at rated power) in inner sites	5.0×10^{12} n/cm ² .s
19	Number of outer irradiation sites	4 (2 larger, 3 smaller)
20	Thermal neutron flux (at rated power) in outer sites	5×10^{11} n/cm ² .s
21	Reactor cooling	Natural Convection

Source: NAA operating manual

3.8.5.2 Determination of As, Cd and Hg in whole blood and serum using NAA

500 mg each of whole blood and serum sample and reference material (Trace Elements in Freeze Dried Animal Blood IAEA-A-13) were weighed into small polyethylene vials and put into bigger polyethylene vials of diameter 1.2 cm and height 2.35 cm containing the samples were put into bigger polyethylene capsule of diameter 1.6 cm and height 5.5 cm (Rabbit capsule), thus giving it double encapsulation. The samples together with the certified reference material were irradiated in the Ghana Research Reactor (GHARR-1) at the Ghana Atomic Energy Commission, operating at 15 KW at a thermal flux of $5 \times 10^{11} \text{ n.cm}^{-2}.\text{s}^{-1}$.

They were transferred into irradiation sites via pneumatic transfer system at a pressure of 0.60 Mpa. The irradiation was categorized according to the half-life of the element of interest. The counting time, the time for irradiation, as well as the delayed time is presented in Table 3.8 below.

Table 3.8: Irradiation and counting schemes

Element	Isotope	Nuclide	Half Life / Hours	Gamma-ray Energy (Kev)	Irradiation Time /s	Delay Time /Hours	Counting Time/s
As	⁷⁶ As	⁷⁵ As	26.2	559.1	60	24	120
Cd	¹¹⁴ Cd	¹¹⁵ Cd	53.3	336.2	60	24	120
Hg	¹⁹⁷ Hg	¹⁹⁶ Hg	64.1	77.6	60	24	120

After the irradiation, radioactivity measurement of induced radionuclide was performed by a computer based γ -ray spectrometry set-up. It consists of an n-type HPGe detector coupled to a computer based multi-channel analyzer (MCA). The relative efficiency of the detector is 25% with energy resolution of 1.8 keV at γ -ray energy of 1332keV of ⁶⁰Co.

Through appropriate choice of cooling-time, detector's dead time was controlled to be less than 10%. Identification of γ -ray of product radionuclide was identified through the energies and quantitative analysis of the concentration was achieved using the γ -ray spectrum analysis software, ORTEC MEASTRO-32 (Lavi and Alfassi, 1990; Nixon et al., 1996).

3.8.5.3 Energy and Efficiency calibration of HPGe

The energy emitted associated with the emission of the gamma rays of each particular nuclide in the sample. This emitted energy is directed to the multichannel analyser (MCA) of the gamma spectroscopy system. The MCA sorts the absorbed gamma rays into various unique channels. This is done by using gamma sources with known energies. The gamma rays emitted by the sources are measured (^{137}Cs : 661.657 keV, ^{60}Co : 1173 keV and 1332 keV are suitable for this task) by the detector. A relation between channel number and energy is obtained by plotting energy against channel numbers. Based on this relation, the energies at the various channels of the MCA is then determined through HPGe detector using the SpectrLine GP software.

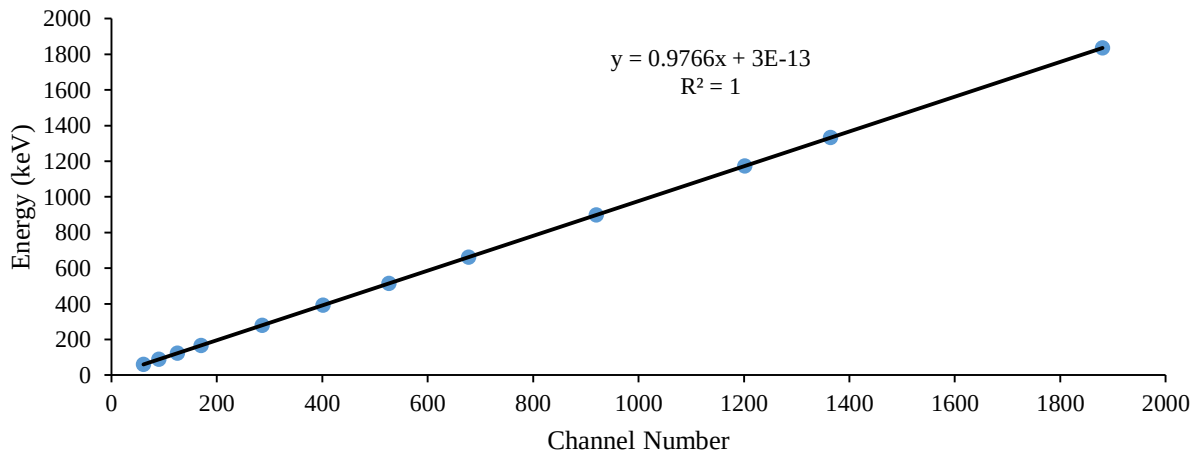


Fig.3.3: Energy calibration of HPGe detector

The energy calibration equation is shown in Fig. 3.3. From the graph, the relationship between the channel numbers of the multichannel analyzer and energy of the gamma rays of As, Cd and Hg were sensitive (since the coefficient of variation, $r = 1$).

Efficiency calibration is associated with the quantitative aspect of NAA. It determines the detector's response to gamma rays at various energies (relative efficiency) and how many ray hitting the HPGe are actually accounted for in photo peaks making up the spectrum. Just as with energy calibration, gamma emitting sources are used for efficiency calibration. The source should emit gamma rays covering a broad spectrum of energies (from low to high energy ranges). Single (^{152}Eu) or a combination (^{241}Am , ^{109}Cd , ^{57}Co , ^{113}Sn , ^{137}Cs , ^{65}Zn , ^{88}Y , ^{60}Co) of gamma sources can be employed for this task. An efficiency curve (energy against efficiency) is plotted from which an efficiency equation is derived.

3.8.5.4 Quality Assurance for Analysis of As, Cd and Hg in blood samples using NAA

A certified reference material (CRM) with matrix same or similar to the samples to be analyzed is prepared and irradiated together with the samples under the same conditions. Counting of the CRM is also done using the same counting geometry as the sample. Analysis of the CRM is then carried out and results compared to the z- values on the CRM's certificate, which was found to be within the acceptable value of ± 2 .

3.8.6 Determination of Pb in whole blood and serum using Graphite Furnace AAS

In the determination of Pb levels in whole blood and serum, 0.1 mL each of whole blood or serum was added to 0.4 mL ‘diluent’ (contains 0.25% Triton X -100, 200 ppm ammonium phosphate, and 750 magnesium nitrate) so as to help in the hemolysis, sequester the Pb and increase atomization efficiency. 20 μ L of the diluted sample was analysed using Buck 210 VGP Atomic Absorption Spectrophotometer with the Model 220 Graphite Furnance (GFAAS).

3.8.7 Analysis of socio-demographic data collected from donors of the blood samples

The data obtained from the administration of the questionnaires were analyzed using SPSS version 21. General descriptive statistics was employed to analyze the data. A non – parametric t – test was used to verify whether or not there were significant differences between mean concentrations of As, Pb, Cd and Hg in whole blood and serum according to gender dichotomy.

The mean differences in the concentrations of As, Pb, Cd and Hg among the various ages categories, type of drinking water and type of housing were analyzed using analysis of variance (ANOVA) techniques. Spearman correlation was used to verify the inter-metal or inter-metalloid correlations in both the whole blood and serum.

3.8.8 Data Treatment

The laboratory data obtained from this study were subjected to the following statistical tests; standardized skewness, standard deviation and variance. In addition, the test of normality was undertaken to determine whether the sample results are normally distributed. Also, multivariate statistical techniques were employed to explain the hidden structures within the sets.

3.9 Exposure Assessment and Risk Characterisation

3.9.1 Exposure Assessment

This involves the determination of the amount (concentration), duration, and pattern of exposure to heavy metals or metalloids in environmental media and biota;

Exposure Route: In this segment of the study, ingestion and dermal contact to toxicants in rivers/streams and sediment as well as ingestion of cooked food stuffs grown in the study areas are considered important exposure route through which resident children, adults and mine workers (artisanal small-scale mining companies) are exposed to arsenic, cadmium, mercury and lead.

Similarly, the focus of this study was to assess the probability of resident adults' age ≥ 18 years and resident children between the ages of 1 – 17 years who are not engaged in artisanal small-scale mining or large-scale gold mining stand the risk of suffering from cancerous and non-cancerous diseases. However, as noted in a report issued by Human Right Watch (HRW, 2015), that children below the ages of 18 years working at ASGM sites in Ghana were excluded in this study. They were excluded because the study did not want to legitimise an illegal operation because children of that age were by law (Act 560) are not expected to engage in hazardous work such as ASGM.

The operational definition for non-cancer health effect for this study is disease conditions such as damage to central nervous system, diabetes mellitus, cardiovascular and reproductive systems. In the case of the carcinogenic health risk, it means development of cancer or tumours when human beings are exposed to carcinogenic chemicals.

Exposure Scenario: The value of the carcinogenic risk depends on the exposure scenario being evaluated. In this thesis, the Central Tendency Exposure (CTE) is defined as when resident adults, resident children and adult mine workers (both large-scale and artisanal small-scale gold miner) are exposed to average (50%) concentrations of arsenic, cadmium, mercury and lead in water, sediment and cassava samples.

Similarly, the Reasonable Maximum Exposure (RME) as used in this thesis, means that resident adults, children, and adult mine workers are exposed to 95% of the concentration of arsenic, cadmium, mercury and lead in water, sediment and cassava samples (Obiri et al., 2006a; USEPA, 2001; Asante-Duah, 2002).

Ingestion of sediment: Resident adults and ASGM miners working outdoors as well as resident children who usually play in the contaminated water bodies may ingest sediment through incidental contact of the mouth with hands and clothing.

According to the USEPA General Exposure Factors Handbook (USEPA, 1997; USEPA, 2008), the formula for calculating the average daily dose (ADD) for each of the above receptors, i.e., the dose of arsenic, cadmium, mercury and lead that resident adult non-mine workers, children and adult mine workers are likely to be exposed to via ingestion of sediment is:

$$ADD = \frac{EPC \times IR \times AAF_{so} \times ED \times EF \times 10^{-6}}{BW \times AT} \quad \dots \quad (3.1)$$

Where:

ADD = Average daily dose, mg/kg/day;

EPC = Exposure Point Concentration, i.e., concentration of As, Cd, Pb and Hg in sediment (mg/kg) to be impacted upon by resident adults, children and ASGM workers;

IR = Sediment ingestion rate (mg/day);

AAF_{so} = Chemical-specific oral - sediment absorption adjustment factor (mg/mg);

ED = Exposure Duration (yrs), the value for Ed varies from resident adults, children and mine workers (i.e., large-scale and artisanal small-scale gold workers). For resident adults, the study assumed that they spend 4 hours every day on outdoor activities, while children may spend approximately 3 hours, and mine workers may spend on the average 8 and 12 hours a day according to the Labour Act, Act 590 respectively for both CTE and RME parameters;

EF = Exposure Frequency (events/year);

BW = Body weight (kg);

AT = Averaging Time, which is equal to the life expectancy of a resident Ghanaian.

With the exception of EPC, ED and BW, the rest were default values in the Risk Integrated Software for clean-up of hazardous waste sites (RISC 4.02) developed by BP for the Superfund sites (Spence, 2001). Bodyweight of 58.6 kg was used for resident adults, in line with data from the Ghana Statistical Service (GSS, 2010).

The average life expectancy for resident adult in Ghana is 65.8 years (i.e., 63.4 years for men and 68.2 years for women), however in this study, a default value of 70 years was used as life expectancy for resident adults, children and adult ASGM workers in the study areas, as the study wanted to look at how the situation would be like should the concentrations of As, Cd, Pb and Hg in the sediment remains the same and resident adults, children and adult ASGM workers continuously ingest them for 70 years.

Dermal contact with sediment: Using equation (3.2), the amount of arsenic, cadmium, mercury and lead from the sediment through the skin into the bloodstream on daily basis by all the receptors is calculated as follows:

$$ADD = \frac{EPC \times SA \times AAF_{sd} \times FS \times ED \times EF \times 10^{-6}}{BW \times AT} \quad \dots \quad (3.2)$$

Where:

SA = Total skin surface area exposed to the sediment (cm²);

AAF_{sd} = Chemical-specific dermal - sediment absorption adjustment factor (mg/mg);

AF = Sediment – to – skin adherence factor (mg/cm²/event).

FS = Fraction of skin area exposed to the sediment (unitless)

The other variables used in this equation have been previously defined in equation (3.1) above.

Ingestion of water and cassava: The formula for calculating daily intake of arsenic, cadmium, mercury and lead via ingestion of water and cassava by each of the receptors is:

$$ADD = \frac{EPC \times IR \times AAF_{wo} \times ED \times EF \times 10^{-6}}{BW \times AT} \quad \dots \quad (3.3)$$

Where:

EPC = exposure point concentration of a metal in the drinking water (µg/L),

IR = water ingestion rate per unit time (L/day);

AAF_{wo} = Chemical-specific oral – water adjustment factor (µg/L)

Other variables have been defined in equations (3.1) and (3.2) above.

Dermal contact with water: According to the USEPA (1999), equation (3.4) is used in calculating the uptake of the toxicants in water bodies via dermal exposure pathway in this study.

$$ADD = \frac{EPC \times K_p \times SA \times AAF_{wd} \times ED \times EF \times 10^{-6}}{BW \times AT} \quad \dots \quad (3.4)$$

Where all the symbols have the same meaning as in equation (3.2) above, with the exception K_p and AAF_{wd}.

K_p = Chemical-specific skin permeability coefficient (cm/hr);

AAF_{wd} = Chemical-specific dermal – water absorption adjustment factor (µg/L)

The skin permeability coefficient (K_p) estimates the permeability of the aforementioned toxicants through the skin layers into the blood stream of resident adults, default values from the RISC 4.02 software were used for each of the toxicants in this study.

Default values from the Risk Integrated Software for clean-up of hazardous waste sites (RISC 4.02) were used for the following parameters in equations (3.2), (3.3) and (3.4) above; IR, AAF_{wo} , AAF_{wd} , AAF_{so} , AAF_{sd} , SA, EF, ED, K_p for each toxicant.

3.9.2 Risk Characterisation

3.9.2.1 Calculation of non – carcinogenic health risk

According to the USEPA risk assessment guidelines, non-cancer health risk is quantified as hazard index (HI). It is expressed mathematically as:

$$HI = \sum HQ \quad \dots \quad (3.5)$$

Where HQ = is the hazard quotient for individual chemical and individual exposure route.

Mathematically, HQ is calculated as:

$$HQ = \frac{ADD}{RfD} \quad \dots \quad (3.6)$$

Where:

ADD = the average daily dose resident adult, children and adult ASGM worker is exposed to As, Pb, Cd and Hg in water and sediment via oral and dermal exposure routes respectively (mg/kg/day);

RfD = the reference dose for each metal or metalloid for each exposure route.

This is defined as the daily concentration of arsenic, cadmium, mercury and lead that receptors are exposed via oral or dermal exposure pathway which is likely not to cause any appreciable health risk during their lifetime.

Default values from RISC 4.02 software were used for oral and dermal reference dose is presented in Table 3.9.

3.9.2.2 Calculation of carcinogenic health risk

The average daily dose and cancer slope factors are combined to calculate excess cancer risk according to equation (3.7) below:

$$\text{Cancer Health Risk} = \text{ADD} \times \text{CSF} \quad \dots \quad (3.7)$$

Where:

ADD = the average daily dose of As or Cd in water and sediment via oral and dermal exposure routes in the study area by resident adults, children and adult ASGM workers;

CSF = Cancer slope for each metal or metalloid and for each exposure route.

Table 3.9 Receptor and intake parameters for resident adults, children and adult ASGM workers

Parameter	Resident Adults		Resident children		Adult ASGM workers	
	CTE	RME	CTE	RME	CTE	RME
Life time (years)	70	70	70	70	70	70
Body weight (kg)	58.6	58.6	15	15	58.6	58.6
Exposure frequency for sediment (events/year)	40	350	130	350	40	350
Exposure duration for sediment (year)	9	30	6	6	9	30
Ingestion rate for sediment (mg/day)	40	100	90	200	40	100
Total skin surface area (cm ²) – SA	18,400	23,000	6,800	7,280	18,400	23,000
Fraction of skin exposed to sediment (-)	0.11	0.25	0.13	0.55	0.11	0.25
Sediment/skin adherence factor (mg/cm ²)	0.2	0.2	0.2	0.2	0.2	0.2
Exposure frequency for water (events/year)	40	350	130	350	40	350
Exposure duration for water (year)	9	30	6	6	9	30
Ingestion rate for water (mg/L)	2	6			2	6

Source: USEPA (2008)

CHAPTER FOUR

RESULTS AND DISCUSSION

4.0 Concentration and seasonal variations of physico-chemical parameters of water samples from the study areas.

Tables 4.0 and 4.1 summarises the descriptive statistics for water parameters from Tarkwa Nsuaem Municipality (TNMA) and Prestea Huni Valley District (PHVD).

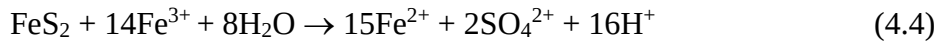
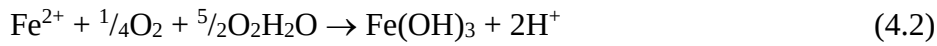
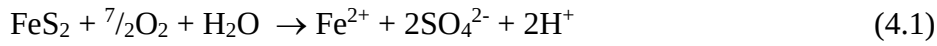
Table 4.00: Descriptive statistics of physico-chemicals of water samples from TNMA for dry and wet seasons.

Parameter	Dry season			Wet season			GS 175/1- WHO Guideline value
	Range	Mean	Standard Deviation	Range	Mean	Standard Deviation	
pH	4.23 – 8.12	-	-	6.28 – 8.78	-	-	6.5 – 8.5
Turbidity (NTU)	4.60 – 950	120.7	13.1	5.12 – 1,150	68.9	165.5	5
TSS (mg/L)	1.0 – 2,940	205.5	816.7	25.4 – 7,856	1,234	456.7	-
TDS (mg/L)	10 – 3,625	1,347	528	25 – 2,897	623.5	601	1,000
Conductivity	22 – 3,170	689.8	1,042	45 – 5,986	998.8	321	-

From Table 4.00 above, the pH value for water samples from TNMA during the wet season ranged from 6.28 to 8.78 as compared the recommended WHO Guideline Value of 6.5 to 8.5. This means that pH of water samples from TNMA exceeded the WHO guideline value. It also found out that pH of water samples collected from the TNMA in the dry season that, 31 out of a total of 60 water samples collected had pH values ≤ 4.23 . This indicates that most (51.6%) of the water samples from TNMA were moderately acidic. This observation is in conformity with an assertion made by Balistrieri *et al.*, 1999. Also, in the wet season, 28 out of 60 water samples collected had pH values below 6.28, i.e., during the rainy most of the surface water in the study is diluted with the runoff which result in the pH of the surface water been weakly acidic.

This finding is consistent with the assertions made by Zobrist *et al.*, (2009) that improper discharge of mine effluents containing acidic water into surface water bodies within the concession of the mines or mine pit dewatering can result in water bodies from mining communities to have very low or moderately low pH values. They further asserted that oxidation of arsenopyrite or pyrite waste rocks can produce acidic waters (through a process called acid mine drainage) as noted by Zobrist *et al.*, (2009).

According to Nordstorm and Southam (1997), the oxidation of pyrites waste rocks generate acid via the following reactions which takes place usually in abiotic and microbial conditions;



The amount of acid produce from the oxidation of the pyrite or arsenopyrite oxidation waste rocks depend on the acid-neutralising capacity of the waste rocks (presence of calcite or carbonates). Waste rocks with low acid-neutralising capacity tend to produce strong acidic water over several years depending on other environmental factors such as the mineralogy and hydrology in the deposit and the local climatic conditions. The converse is true for waste rocks with high acid-neutralising capacity.

The summary of descriptive statistics for the physico-chemical parameters for water samples from the Prestea Huni Valley District (PHVD) for both wet and dry season is presented in Table 4.01 below.

Table 4.01: Descriptive statistics of physico-chemicals of water samples from PHVD for dry and wet seasons.

Parameter	Dry season			Wet season			GS 175/1-WHO Guideline value
	Range	Mean	Standard Deviation	Range	Mean	Standard Deviation	
pH	5.46 – 8.59	-	-	5.56 - 7.34	-	-	6.5 – 8.5
Turbidity (NTU)	21.0 – 1,261	258.5	2.23	2.00 – 896	342.2	16.5	5
TSS (mg/L)	41.0 – 2,890	576.9	12.8	35.4 – 3,856	634.8	43.2	-
TDS (mg/L)	7.5 – 1,325	534.5	18.9	46.7 – 2,987	1,656	24.8	1,000
Conductivity	15.0 – 2,257	819.8	17.9	39.8 – 2,170	998.6	32.1	-

From Table 4.01 above, it can be seen that, the pH of water samples from PHVD for dry season ranged from 5.46 to 8.59, which is below the GS 175/1-WHO guideline value. That is, pH of 34 (56.6%) out of 60 water samples from the PHVD had moderately low pH values; while the remaining 43.3% of the water samples from PHVD had pH values that ranged from neutral to moderately basic.

The mean turbidity values for both the dry and wet season were found to have exceeded the GS 175/1-WHO guideline value of 5 NTU. The mean turbidity values for 50% of water samples from the different sampling points in the PHVD exceeded the guideline value of 5 NTU while the other 50% of the sampling points were found to be below the acceptable guideline value. Also, the TDS values for both dry and wet season exceeded the GS 175/1-WHO acceptable guideline value of 1,000 mg/L.

An analysis of the results obtained from Pearson correlation revealed that at $P < 0.05$ two – tailed, showed a positive correlation between pH – TDS, pH – Electrical Conductivity.

The positive correlation between pH – TDS and pH – Electrical Conductivity indicates the strong influence of pH on concentration or mobilities of these ions in solution. As noted by Elzahabi and Yong (2001), pH and TDS are of major concern as they affect the dissolution of ions in solution (Tables 4.02).

Table 4.02: Pearson product-moment correlation coefficient between water quality parameters from TNMA

	pH	Turb	Cond	TDS	TSS
pH	1				
Turb	-0.866*	1			
Cond	0.793*	-0.185*	1		
TDS	0.893**	-0.171*	0.995**	1	
TSS	-0.787*	0.962**	-0.132	-0.116	1

*Correlation is significant at the 0.05 level (2 – tailed)

**Correlation is significant at the 0.01 level (2 – tailed)

In the dry season, the Pearson product moment correlation coefficient between the water quality parameters from the PHVD for the dry season is presented in Table 4.03.

Table 4.03: Pearson product-moment correlation coefficient between water quality parameters from PHVD

	pH	Turb	Cond	TDS	TSS
pH	1				
Turb	-0.397*	1			
Cond	0.909**	-0.583*	1		
TDS	0.797**	-0.175*	0.698**	1	
TSS	0.385*	0.537*	0.904*	0.937*	1

*Correlation is significant at the 0.05 level (2 – tailed)

**Correlation is significant at the 0.01 level (2 – tailed)

Negative relationships were found to exist between pH – Turbidity; Cond – turbidity; and TDS – Turbidity respectively at two tailed, $P < 0.05$. Also, significant strong positive correlations ($r > 0.5$) were found between pH – TDS, TDS–Conductivity and TSS - TDS respectively.

The result of the PCA analysis is presented in Table 4.04 below.

Table 4.04: Total Variance Explained

Component	Initial Eigen values ^a			Extraction Sums of Squared Loadings				Rotation Sums of Squared Loadings	
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	1.756 x 10 ⁶	54.3	54.3	1.756 x 10 ⁶	54.3	54.3	1.090 x 10 ⁶	33.7	33.7
2	1.098 x 10 ⁶	33.9	88.2	1.098 x 10 ⁶	33.9	88.2	1.739 x 10 ⁶	53.7	87.4
3	1.094 x 10 ⁵	3.38	91.6	1.094 x 10 ⁵	3.38	91.57	1.350 x 10 ⁵	4.17	91.6

From the results of the principal component analysis (PCA) in Table 4.04, three components were extracted. These components can be used to explain more than 91.6% of the total variance (Table 4.04). Component 1 accounted for about 54.3% of the variance and extracted the following parameters, i.e., conductivity, turbidity and TDS. This suggests strong rock-water interactions for both wet and dry seasons for water samples. The loadings contributing to component 2 is pH, conductivity and TDS. Component 1 and component 2 explains the natural processes such as water-rock interactions taking place in the Birimian and Takwaian rock systems which have high sulphur and iron content in the pyrite or arsenopyrite ores.

These ores have high acid generating potential, which through acid mine drainage process contribute to the elevated pH levels in the water samples. Component 3, also showed high loading for TDS and TSS, which accounted for 3.38% of the variations in the hydrochemistry of the TNMA.

Four principal components (Eigen values >1) were extracted from the water data sets (Table 4.05). These cumulative explain about 70% of the variance in the data. Group 1 (or factor 1, expressing about 23.2% of the total variance) includes mainly hydrochemical properties of water (pH, conductivity and TDS); group 2 (explaining about 21.0% of the total variance) includes turbidity and TSS. Group 3, accounts for 13.0% of the total variance, and includes TDS and conductivity. Group 4 explains 11.4% of the total variance shows loading of pH which originated from the oxidation of the arsenopyrite and the pyrite generating high acidity of the water samples due to acid mine drainage.

Table 4.05: Variance in water quality data from PHVD and TNMA explained by the four principal components Analysis

Component	Initial Eigenvalues			Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	2.784	23.203	23.203	2.784	23.203	23.203	2.656	22.135	22.135
2	2.525	21.044	44.247	2.525	21.044	44.247	2.499	20.824	42.959
3	1.564	13.030	57.277	1.564	13.030	57.277	1.610	13.418	56.377
4	1.377	11.476	68.753	1.377	11.476	68.753	1.485	12.376	68.753

Extraction Method: Principal Component Analysis.

From Table 4.06, pH values of water samples in CCMA for both wet and dry season are within the acceptable GS 175/1-WHO guideline. This suggest that, the pH of the water samples in the CCMA are neither acidic nor basic.

Table 4.06: Descriptive statistics for physico – chemical parameter for water samples from CCMA for dry and wet seasons

Parameter	Dry season			Wet season			GS 175/1-WHO Guideline value
	Range	Mean	Standard Deviation	Range	Mean	Standard Deviation	
pH	6.45 – 7.45	-	-	6.72 – 7.45	-	-	6.5 – 8.5
Turbidity (NTU)	2.20 – 11.00	5.62	2.97	2.500 – 12.00	5.900	2.85	5
TSS (mg/L)	17.20 – 506	113.5	11.9	17.2 – 219.00	79.23	43.5	-
TDS (mg/L)	6.80 – 62.50	23.5	14.9	1.80 – 191.0	34.76	33.5	1,000
Conductivity	4.40 – 64.10	23.1	14.7	0.80 – 144.0	47.11	35.7	-

A strong positive correlation was found to exist between the following physico-chemical parameters in water samples from CCMA; Turb – TSS, Turb – TDS, pH – Cond and pH – TDS which is significant at $P \leq 0.05$ two tailed (Table 4.07).

Table 4.07: Pearson product-moment correlation coefficient between water quality parameters from CCMA

	pH	Turb	Cond	TDS	TSS
pH	1				
Turb	0.234	1			
Cond	0.789*	-0.405**	1		
TDS	0.893*	0.419	0.892*	1	
TSS	0.345	0.810**	0.456**	0.639*	1

*Correlation is significant at the 0.05 level (2 – tailed)

**Correlation is significant at the 0.01 level (2 – tailed)

Table 4:08 below provides a summary of descriptive statistics for concentration results of physico-chemicals of water samples from the Nangodi mining community in the Nabdam District for both wet and dry season.

Table 4.08: Descriptive statistics for hydrochemical parameters for samples from Nangodi in Nabdam district for the dry and wet seasons.

Parameter	Dry season			Wet season			GS 175/1- WHO Guideline value
	Range	Mean	Standard Deviation	Range	Mean	Standard Deviation	
pH	6.90 – 9.11	-	-	5.1 – 8.4	-	-	6.5 – 8.5
Colour	2.8 -747	99.1	162.2	0.6 – 1324	48.5	16.7	15.0
Turbidity (NTU)	9 – 172	48.5	36.0	2.5 – 1500	66.6	23.9	5
TSS (mg/L)	1.38 – 19.8	3.61	2.88	15 – 2625	537	572	-
TDS (mg/L)	4.4 – 313	19.5	48.3	1.8 – 544	111	131.7	1,000
Conductivity	1.5 – 13.6	4.34	2.59	0.5 – 112	29.1	25.5	-

Nangodi mining community in the Nabdam District ranged from 6.90 to 9.11 as compared to pH of water samples from TNMA which ranged of 4.23 to 8.12 pH; and that recorded in water samples from CCMA ranged from 6.72 to 7.45. The difference in the pH of the water samples may be due to the water – rock interaction.

Table 4.09: Descriptive statistics for physico-chemical parameters in water samples from Tinga in Bole - Bamboi district for the dry and wet seasons.

Parameter	Dry season			Wet season			GS 175/1- WHO Guideline value
	Range	Mean	Standard Deviation	Range	Mean	Standard Deviation	
pH (pH units)	6.85 – 8.93	-	-	5.23 – 7.20	-	-	6.5 – 8.5
Colour	29.5 – 516.7	221.2	185.4	0.00 – 1324	95.9	227.7	15.0
Turbidity (NTU)	54.2 – 841.7	358	305.6	0.00 – 1770	274.6	489.0	5
TSS (mg/L)	41.9 – 73.9	61.2	27.1	30 – 633	179.9	158.5	-
TDS (mg/L)	9.24 – 18.4	14.6	3.09	3.78 – 197	19.1	34.2	1,000
Conductivity	2.83 – 5.45	3.89	0.98	1.13 – 48.5	8.15	10.1	-

Similar results were obtained for the physico–chemical parameters in water samples from Tinga mining community in the Bole – Bamboi District (Table 4.09 above). pH of water samples from Tinga mining community in the Bole – Bamboi district ranged from 6.85 to 8.93 as against the pH of water samples from Nangodi in the Nabdam which ranged between district and 6.to 9.11, while that for Cape Coast metropolis ranged from 6.45 to 7.45, and that of TNMA had pH values in the range of 4.23 to 8.12 as well as 5.46 to 8.59 as pH values for water samples in the PHVD. During the wet season, the mean pH of water samples from Tinga mining community ranged from 5.23 to 7.20. The comparison of physical parameters such as pH, electrical conductivity, TDS, turbidity and TSS in the water samples from dry seasons for all study areas are presented in fig. 4.0

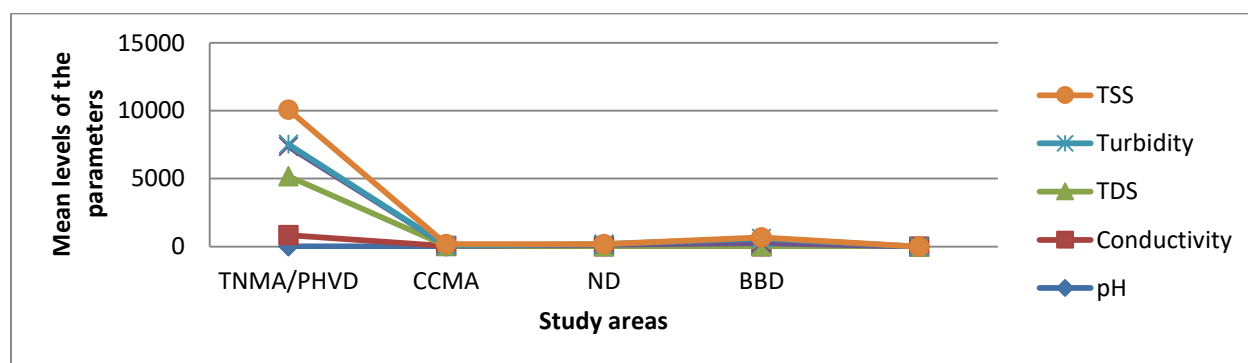


Fig. 4.0: Comparison of pH, Turbidity, TDS, Conductivity and TSS in water samples from the study areas for dry season.

No noticeable variations in pH and conductivity of water samples from the study areas in the dry season. However, there were slight variations in mean TSS, turbidity, conductivity and TDS values for water samples from ND, CCMA and BBD as compared to TNMA and PHVD. This variation may be attributed to water – rock interaction as well as other anthropogenic activities such as mining.

4.1 Concentration and seasonal variations of As, Cd, Pb and Hg in water samples from the study areas

The mean concentrations of heavy metals such as As, Cd, Pb and Hg measured in water samples from TNMA/PHVD for both wet and dry seasons have been presented in table 4.10 below

Table 4.10: Mean concentrations of As, Cd, Pb and Hg in water samples from TNMA for both dry and wet seasons

Sampling Point	Concentration in µg/L							
	Wet Season				Dry Season			
	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD
River Asuman	141 ± 12.6	14 ± 0.06	278 ± 3.26	118 ± 1.98	890 ± 23.5	28 ± 2.08	589 ± 4.59	979 ± 9.51
River Achofe	184 ± 9.72	30 ± 1.05	222 ± 7.12	168 ± 3.42	703 ± 22.6	32 ± 2.43	493 ± 8.17	185 ± 7.81
Borehole at Abekoase	24 ± 0.86	26 ± 1.32	141 ± 3.42	66 ± 0.58	24 ± 0.95	48 ± 4.65	596 ± 54.8	25 ± 0.35
River Ateberebe	69 ± 1.78	19 ± 0.24	113 ± 7.52	88 ± 3.35	632 ± 21.4	26 ± 1.98	351 ± 4.82	821 ± 46.5
River Huni	99 ± 4.76	28 ± 1.12	136 ± 7.62	196 ± 2.76	293 ± 0.88	35 ± 3.67	589 ± 54.9	979 ± 19.5
River Subri	106 ± 12.3	35 ± 0.02	51 ± 1.11	102 ± 1.09	204 ± 24.8	34 ± 5.23	596 ± 6.13	215 ± 11.2
River Betinyere	59 ± 0.43	32 ± 2.21	259 ± 12.3	298 ± 6.23	553 ± 6.58	39 ± 3.65	493 ± 8.71	185 ± 7.81
River Ahumabruen	66 ± 2.34	39 ± 1.13	145 ± 4.91	184 ± 7.62	251 ± 4.43	13 ± 0.53	381 ± 23.1	144 ± 2.83
River Agonaben	52 ± 0.34	23 ± 0.23	30.6 ± 1.13	90.3 ± 3.60	49.5 ± 2.21	21 ± 0.24	31.3 ± 0.31	59 ± 0.45
River Adeyie	62 ± 0.18	19.1 ± 0.34	35.7 ± 0.54	5.68 ± 0.04	33.6 ± 0.23	20.3 ± 1.12	85 ± 0.74	18 ± 1.08
GS 175/1 - WHO Guideline Value	10	15	50	300	10	15	50	300

***bold figures exceed GS 175/1 - WHO Guideline values. **SD = Standard Deviation**

Arsenic concentrations in water samples from TNMA varies from 24 µg/L in the borehole water from Abekoase to 184 µg/L in river Achofe during the wet season, while in the dry season concentration of arsenic in the samples also varied from 24 µg/L to 890 µg/L. The presence of arsenic in the water samples can be attributed to water-rock interactions of the arsenic content in the arsenopyrite ores. It can also be observe in Table 4.11 that, concentrations, of arsenic and cadmium were above the permissible guideline values.

The results of Pearson's product moment correlation is shown in Table 4.12 below.

Table 4.11: Pearson's product moment correlation coefficients for pH, Turbidity, TDS, conductivity and heavy metals concentrations in water samples from TNMA

	pH	Turbidity	TDS	Cond	As	Cd	Pb	Hg
pH	1							
Turbidity	0.237**	1						
TDS	0.400**	-0.169	1					
Cond	0.415**	-0.182	0.993**	1				
As	0.567*	0.146	-0.516**	0.567**	1			
Cd	0.534*	0.037	-0.537**	0.576**	0.664*	1		
Pb	0.091	-0.061	-0.057	-0.033	0.829**	0.603*	1	
Hg	0.035	-0.014	-0.015	-0.128	0.940**	-0.038	0.063	1

*Correlation is significant at the 0.05 level (2 – tailed)

**Correlation is significant at the 0.01 level (2 – tailed)

A strong positive metal - metal correlation were found to exist between As – Cd (At $P < 0.01$, two tailed the $r = 0.664$), As – Pb (significant with $r = 0.829$; $P < 0.01$ 2-tailed) and As – Hg ($r = 0.940$; $P < 0.01$, two tailed).

Similar inter – metal correlation were found to exist between levels of heavy metals in the water samples from TNMA for the wet season. For example, the Pb-As correlation was recognized as the strongest with correlation coefficients $r= 0.802$. Also, significant strong correlations ($r>0.5$) were found between As – Cd, Pb – Cd, Pb – Hg, Pb – As and Hg – As. This observation is in conformity with has been reported in literature (Munksgaard and Parry, 2001).

The concentrations of heavy metals in water samples from the Prestea Huni Valley District (PHVD) have been summarised in Table 4.12.

Table 4.12: Mean concentrations of As, Cd, Pb and Hg in water samples from PHVD for both dry and wet seasons

Sampling Point	Concentration in $\mu\text{g/L}$							
	Wet Season				Dry Season			
	As $\pm$ SD	Pb $\pm$ SD	Cd $\pm$ SD	Hg $\pm$ SD	As $\pm$ SD	Pb $\pm$ SD	Cd $\pm$ SD	Hg $\pm$ SD
River Aprepre	271 $\pm$ 3.80	287 $\pm$ 12,6	186 $\pm$ 2.45	188 $\pm$ 0.08	348 $\pm$ 7.68	931 $\pm$ 8.76	805 $\pm$ 14.6	187 $\pm$ 2.43
River Asasre	198 $\pm$ 3.61	272 $\pm$ 8.32	642 $\pm$ 21.3	183 $\pm$ 1.01	800 $\pm$ 2.36	26 $\pm$ 0.05	142 $\pm$ 0.20	283 $\pm$ 5.85
River Abodwesh	89 $\pm$ 3.78	39 $\pm$ 1.31	233 $\pm$ 7.41	142 $\pm$ 5.24	371 $\pm$ 6.78	39 $\pm$ 3.12	414 $\pm$ 1.95	522 $\pm$ 2.63
River Ankobra	54 $\pm$ 6.34	242 $\pm$ 1.03	316 $\pm$ 11.3	903 $\pm$ 13.6	495 $\pm$ 22.5	18 $\pm$ 0.28	313 $\pm$ 0.34	59 $\pm$ 0.12
River Anikoko	51 $\pm$ 2.34	28 $\pm$ 1.04	306 $\pm$ 1.24	289 $\pm$ 2.78	841 $\pm$ 1.58	165 $\pm$ 4.72	580 $\pm$ 10.6	287 $\pm$ 4.61
River Amenkuman	62 $\pm$ 1.98	29 $\pm$ 0.05	284 $\pm$ 9.21	259 $\pm$ 7.65	366 $\pm$ 17.8	38 $\pm$ 3.78	353 $\pm$ 1.95	188 $\pm$ 1.96
River Bodwire	68 $\pm$ 1.82	191 $\pm$ 1.34	335 $\pm$ 10.3	18.0 $\pm$ 0.02	336 $\pm$ 3.46	119 $\pm$ 1.12	85 $\pm$ 8.74	8.0 $\pm$ 0.08
River Twiagya	86.9 $\pm$ 1.72	22 $\pm$ 4.56	258 $\pm$ 3.87	938 $\pm$ 6.52	369 $\pm$ 1.18	20.4 $\pm$ 1.13	161 $\pm$ 0.98	939 $\pm$ 20.6
River Mansi	281 $\pm$ 0.63	19 $\pm$ 2.56	318 $\pm$ 0.98	224 $\pm$ 4.23	997 $\pm$ 1.32	45 $\pm$ 2,34	903 $\pm$ 10.8	673 $\pm$ 1.41
Borehole at Dumasi	48 $\pm$ 1.55	21 $\pm$ 2.12	329 $\pm$ 1.21	292 $\pm$ 2.31	88 $\pm$ 1.79	56 $\pm$ 2.45	703 $\pm$ 1.76	792 $\pm$ 1.48
GS 175/1 - WHO Guideline Value	10	15	50	300	10	15	50	300

***bold figures exceed GS 175/1 - WHO Guideline values. **SD = Standard Deviation**

The concentration of arsenic in water samples in the dry season ranged from 18 µg/L to 931 µg/L (Table 4.12). In the wet season, arsenic concentration varied from 48 µg/L in the borehole at Dumasi to 271 µg/L in River Aprepre. Similar, variations of concentrations of cadmium, mercury and lead in water samples in both the dry and wet seasons. In most of the sampling stations, the concentrations of arsenic, cadmium, mercury and lead exceeded the GS 175/1-WHO guideline values.

The correlation matrix in Table 4.13 below shows significant inter-metal relationships at $P \leq 0.05$ or $P \leq 0.01$ two tailed. Strong correlations ($r \geq 0.5$) were found between As – Cd, As – Pb and As – Hg.

Table 4.13: Pearson's Product – Moment Correlation Coefficients for pH, Turbidity, TDS, Conductivity and heavy metals from PHVD

	pH	Turbidity	TDS	Cond	As	Cd	Pb	Hg
pH	1							
Turbidity	-0.282**	1						
TDS	0.416**	-0.161	1					
Cond	0.430**	-0.176	0.997**	1				
As	0.818**	-0.070	0.611**	0.712**	1			
Cd	0.901**	-0.085	-0.217**	0.691*	0.512*	1		
Pb	0.029	-0.291*	0.605	-0.121	0.802*	0.620	1	
Hg	0.034	-0.113	-0.008	0.710	0.510*	0.042	0.520*	1

*Correlation is significant at the 0.05 level (2 – tailed)

** Correlation is significant at the 0.01 level (2 – tailed)

The strong As – Cd, As – Pb, As – Hg, Cd – Pb, Cd – Hg and Pb – Hg correlations suggest water bodies in the PHVD are highly polluted with effluents from the mining operations. Several factors might have caused this increase, notably among them are increasing number artisanal

small-scale gold mining activities by both the licensed operators and those engaging in the illegal operations popularly called “galamsey”. Other factors may include, the leachates from the improper disposal of mine tailings as well as cyanide spillages from tailing containment ponds of the large-scale mining companies (Obiri et al., 2016; Zobrist et al., 2009).

Tables 4:14 below present the mean levels of heavy metals from water samples from CCMA in dry season. Arsenic concentration ranged from 0.001 µg/L in the treated water from the Brimsu head works to 0.004 µg/L in Asuboni stream at Effutu near Cape Coast.

Comparing the mean concentrations of As, Pb, Cd and Hg in water samples from CCMA for the dry season with GS 175-1/WHO permissible guideline values revealed that they were all below the permissible guideline values.

Table 4.14: Mean levels of heavy metals in water samples from CCMA for dry and wet season

Sampling Point	Concentration in µg/L							
	Wet Season				Dry Season			
	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD
River Kakum (Kwaprow)	0.004 ± 0.01	0.006 ± 0.03	0.004 ± 0.01	0.003 ± 0.01	0.003 ± 0.02	0.001 ± 0.02	0.004 ± 0.01	0.004 ± 0.01
River Kakum (Kakumdo)	0.002 ± 0.01	0.004 ± 0.05	0.003 ± 0.01	0.003 ± 0.01	0.003 ± 0.01	0.008 ± 0.05	0.033 ± 0.01	0.004 ± 0.01
Asuboni Stream (Effutu)	0.001 ± 0.01	0.006 ± 0.01	0.003 ± 0.01	0.004 ± 0.01	0.002 ± 0.01	0.010 ± 0.05	0.004 ± 0.01	0.004 ± 0.01
Tap water from Brimsu	0.001 ± 0.01	0.003 ± 0.01	0.002 ± 0.01	0.006 ± 0.02	0.001 ± 0.02	0.001 ± 0.02	0.033 ± 0.06	0.004 ± 0.01
Asuboni Stream at Mpeasem	0.001 ± 0.01	0.005 ± 0.01	0.003 ± 0.01	0.002 ± 0.01	0.001 ± 0.01	0.013 ± 0.03	0.004 ± 0.01	0.004 ± 0.01
GS 175-1/WHO Permissible Guideline Value	10	15	50	300	10	15	50	300

*SD = Standard Deviation

The concentrations of mercury (Hg), arsenic (As), lead (Pb) and cadmium (Cd) obtained from surface and ground water bodies used for drinking and other domestic chores in Nangodi is presented in Table 4.15. Mercury concentrations in water samples from Nangodi in the dry season ranged from 25 to 91 µg/L, whilst the mercury concentrations in water samples from Nangodi in the wet season ranged from 5 to 72 µg/L (Table 4.15). Hg concentration in the water bodies for both wet and dry season from the Nangodi study area was found to be below GS 175 -1/WHO (GS 175 – 1/WHO, 2008) permissible limit of 300 µg/L for drinking water (Table 4.16). The result presented in Table 4.16 is consistent with findings made by Cobbina et al., (2012).

Table 4.15: Mean levels of As, Cd, Pb and Hg in water samples from Nabdam District for dry/wet season

Sampling Point	Concentration in µg/L							
	Wet Season				Dry Season			
	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD
BHN	44** ± 0.01	440 ± 24.0	33.5 ± 0.02	5 ± 0.001	54 ± 1.20	482 ± 34.5	51 ± 2.34	25 ± 0.01
HDWA	10 ± 0.01	80 ± 0.01	223 ± 0.31	63 ± 0.01	68 ± 2.11	98 ± 0.78	208 ± 1.24	56 ± 0.24
HDWB	14 ± 0.00	251 ± 0.05	444 ± 0.97	32 ± 0.01	17 ± 1.05	405 ± 0.98	475 ± 0.01	35 ± 0.05
DA	65 ± 0.00	222 ± 0.09	533 ± 0.02	72 ± 0.01	84 ± 1.04	266 ± 0.27	574 ± 0.98	83 ± 0.05
DB	23 ± 0.04	223 ± 0.21	1,090 ± 32.1	20 ± 0.06	43 ± 1.14	268 ± 0.40	199 ± 2.24	91 ± 1.14
GS 175-1/WHO Permissible Guideline Value	10	15	50	300	10	15	50	300

*SD = Standard Deviation; **Bold figures exceeded GS 175-1/WHO Permissible Guideline Value

The concentrations of arsenic, cadmium, mercury and lead in water samples from the Tinga community in the Bole-Bamboi District is presented in Table 4.16 below.

Table 4.16: Mean levels of As, Cd, Pb and Hg in water samples from Bole-Bamboi District for dry/wet season

Sampling Point	Concentration in µg/L							
	Wet Season				Dry Season			
	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD	As ± SD	Pb ± SD	Cd ± SD	Hg ± SD
BHT1	2 ± 0.00	8 ± 0.00	40 ± 0.00	127 ± 0.09	7 ± 0.01	17 ± 0.54	41 ± 2.12	153 ± 4.34
BHT2	2 ± 0.00	10 ± 0.00	40 ± 0.00	70 ± 0.01	9 ± 0.00	14 ± 2.15	44 ± 0.02	99 ± 1.12
BHT3	10 ± 0.00	8 ± 0.00	27 ± 0.01	127 ± 0.02	13 ± 0.01	23 ± 1.67	42 ± 0.97	143 ± 5.05
BHT4	2 ± 0.00	10 ± 0.00	34 ± 0.02	40 ± 0.01	5 ± 0.02	19 ± 0.05	39 ± 0.05	45 ± 0.06
EGWPS	1 ± 0.00	35 ± 0.01	11 ± 0.01	27 ± 0.02	6 ± 1.12	56 ± 4.32	27 ± 0.13	33 ± 0.67
GWPS	1 ± 0.00	70 ± 0.03	4 ± 0.00	27 ± 0.25	5 ± 0.00	88 ± 3.12	16 ± 0.76	33 ± 0.04
AWPS	3 ± 0.00	70 ± 0.00	4 ± 0.00	27 ± 0.02	5 ± 0.01	88 ± 5.42	15 ± 1.23	33 ± 0.01
GS 175-1/WHO Permissible Guideline Value	10	15	50	300	10	15	50	300

*SD = Standard Deviation

High levels of Hg recorded in Tinga were from borehole BHT1 and BHT3 (borehole in Tinga labelled 1, 3), located close to where mined ore is washed, whilst the lowest level was recorded from BHT4 (borehole in Tinga labelled 4), which was far from the mining premises as compared to other sampling sites (Table 4.16).

All sampling sites at Tinga recorded Hg concentration exceeding the GS 175 – 1/WHO permissible guideline value. While the concentration of arsenic in water samples in the Tinga community were found to be within the GS 175 – 1/WHO permissible guideline value but in Nangodi community in the Nabdam District were found to have exceeded the permissible guideline value.

A comparison of cadmium concentrations in water samples from Nangodi were found to be higher than the GS 175/1-WHO guideline values than what was recorded in the Tinga community. Also, cadmium concentrations in water samples from Nangodi and Tinga, important artisanal small-scale gold mining (ASGM) communities in Nabdam District and Bole-Bamboi District respectively were found to be same as cadmium concentrations in water samples in ASGM sites along the Birim River in the East Akim Municipality.

To better understand the data sets of arsenic, cadmium, mercury and lead contaminations in water samples from Bole-Bamobi and Nabdam District respectively, the water quality data in Tables 4.15 and 4.16 were subjected to principal component analysis. Using the eigen values ≥ 1 , three components were extracted that explains 73.7% of variance in the data sets. Component 1, accounted for 22.0% of the variance in water quality data sets. Water quality parameters for this variance are cadmium, lead and mercury. Component 2, showed loading for arsenic, mercury and cadmium; while component 3, accounted for pH, Hg and arsenic concentrations in the water samples.

This means that, the two districts are located on the Bole-Bamboi-Nangodi geological belt of the Birimian and Tarkwaian rock systems (Kesse, 1985). That is, mining operations in the two districts may have contributed to cadmium, mercury, lead and arsenic concentration in the water samples.

4.2 Concentration of As, Cd, Hg and Pb in sediment samples from study areas

Blasting, crushing of arsenopyrite-bearing rocks as well as spillages of mine effluents into the natural environment in mining communities introduce a lot heavy metals into nearby soils or sediments. Sediments are known to be the ultimate sinks for heavy metals discharged into the environment (Yu et al., 2001).

The mean concentrations of heavy metals such as As, Cd, Pb and Hg in sediments from TNMA and PHVD have been presented in Table 4.18 below. The measured metal concentrations varied greatly as follows: As, 0.469 – 2.451 mg kg⁻¹ ; Hg, 0.083 – 0.448 mg kg⁻¹; Pb, 0.005 – 5.88 mg kg⁻¹ and Cd, 0.0044 – 5.27 mg kg⁻¹. For instance, the mean concentrations of As, Hg, Pb and Cd in sediment from in river Aprepre from TNMA/PHVD were found to be 12,360, 44.0, 14,700 and 4,150 times greater, respectively for As, Hg, Pb and Cd than the WHO background values (Table 4.17).

Table 4.17: Levels of heavy metals in sediments from mining area (TNMA and PHVD)

Sampling Point	Concentration in mg/kg			
	As $\pm$ SD**	Pb $\pm$ SD	Cd $\pm$ SD	Hg $\pm$ SD
Abodweseh	1.246 $\pm$ 0.04	2.22 $\pm$ 0.89	2.125 $\pm$ 1.24	0.432 $\pm$ 0.02
Achofe	1.023 $\pm$ 0.12	5.88 $\pm$ 1.19	1.563 $\pm$ 2.13	0.186 $\pm$ 0.57
Betinyere	1.765 $\pm$ 0.09	2.581 $\pm$ 4.58	1.236 $\pm$ 1.09	0.102 $\pm$ 0.07
Ahomabruen	1.365 $\pm$ 1.23	0.001 $\pm$ 0.02	1.451 $\pm$ 3.12	0.239 $\pm$ 0.24
Ateberebe	2.451 $\pm$ 2.14	0.226 $\pm$ 0.01	1.126 $\pm$ 4.32	0.083 $\pm$ 0.12
Asuman	0.987 $\pm$ 2.10	0.011 $\pm$ 0.01	1.235 $\pm$ 1.08	0.389 $\pm$ 0.08
Huni	0.605 $\pm$ 0.06	0.669 $\pm$ 1.13	1.572 $\pm$ 2.04	0.448 $\pm$ 0.08
Subri	0.857 $\pm$ 0.18	0.045 $\pm$ 1.09	1.234 $\pm$ 2.89	0.124 $\pm$ 1.12
Aprepre	0.469 $\pm$ 0.01	0.512 $\pm$ 0.08	1.621 $\pm$ 0.89	0.300 $\pm$ 0.09
Bodwire	0.789 $\pm$ 0.08	1.124 $\pm$ 0.98	0.673 $\pm$ 1.14	0.321 $\pm$ 0.10
Asasre	0.985 $\pm$ 3.41	0.416 $\pm$ 0.23	0.916 $\pm$ 1.32	0.134 $\pm$ 0.89
Achesua	1.652 $\pm$ 1.87	0.005 $\pm$ 0.07	0.713 $\pm$ 2.12	0.157 $\pm$ 0.25
Nsuabena	1.984 $\pm$ 0.65	1.250 $\pm$ 2.31	0.044 $\pm$ 0.78	0.188 $\pm$ 0.78
Donyame	1.365 $\pm$ 2.76	0.530 $\pm$ 2.14	0.613 $\pm$ 0.98	0.110 $\pm$ 0.65
Kwakronkron	1.345 $\pm$ 1.76	0.034 $\pm$ 0.01	5.272 $\pm$ 1.13	0.433 $\pm$ 1.07
GS 175-2/WHO Guideline value	0.01	0.015	0.05	0.3

***Bold figures exceed permissible guideline value; **SD = Standard Deviation**

The result of the Pearson's correlation analysis is presented in Table 4.18 below.

Table 4.18: Pearson's Product-Moment Correlation matrix for metal concentrations in sediment samples from TNMA/PHVD

	As	Pb	Cd	Hg
As	1			
Pb	0.820*	1		
Cd	0.882*	0.520**	1	
Hg	0.704*	-0.093	0.522**	1

* Correlation is significant at the 0.01 level (2 – tailed)

** Correlation is significant at the 0.05 level (2 – tailed)

At $p \leq 0.01$, strong significant correlation was observed between As – Hg, As – Cd, As – Pb; while

Pb – Cd correlation was found to be significant at $p \leq 0.05$ two tailed.

Arsenic (As) is known to be originated from the arsenopyrite-bearing rocks, as such mining of this gold bearing rocks or natural mineral weathering processes might have contributed to the release of As into the environment. Hg is also widely used by illegal gold miners to extract the gold from its ores.

Hierarchal cluster analysis (HCA) categorised the data into 2 main groups based on sampling locations (Figure 4.1).

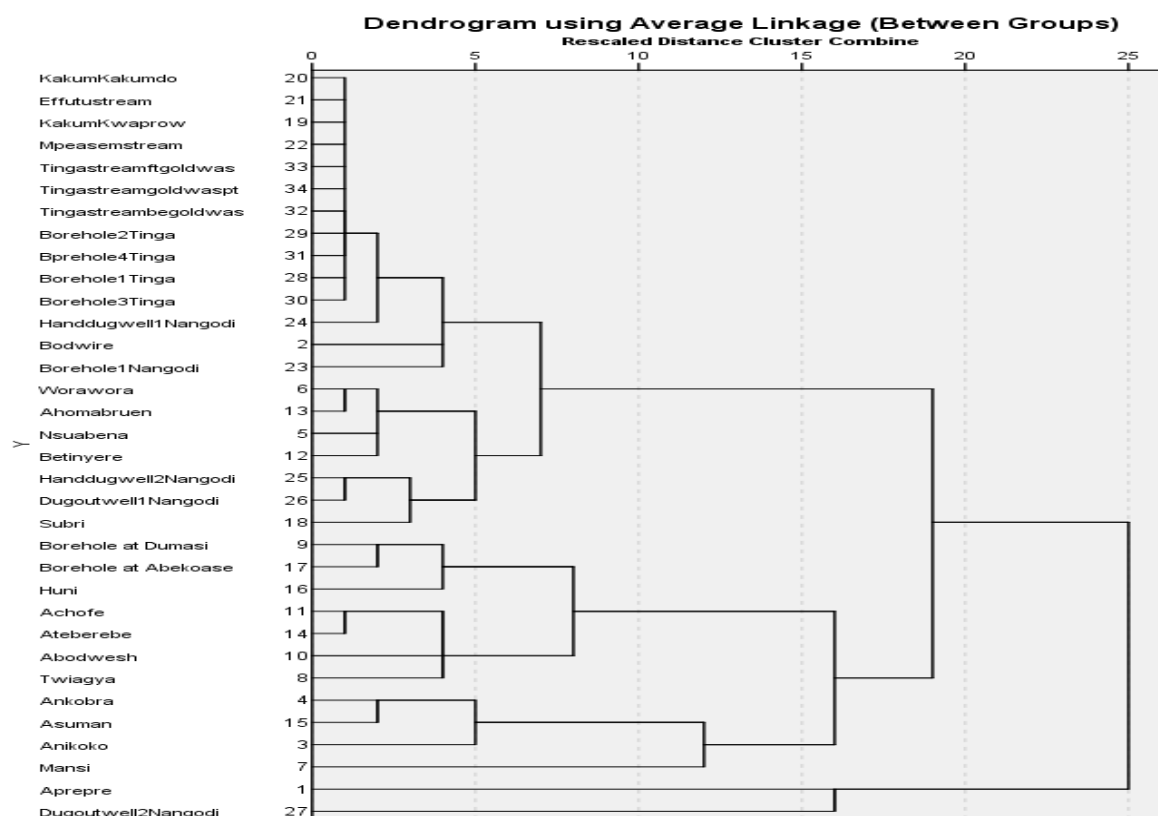


Fig. 4.1: Cluster tree of metals in water from various sampling locations

Cluster one comprises sampling locations at TNMA/PHVD whereas cluster two is made up of sampling locations at Tinga and Nangodi. Cluster one consists of the physicochemical properties (conductivity, TDS, turbidity, etc); cluster two consists of geochemical parameters as well as metals associated with mining (pH, As, Cd, Hg and Pb).

The results of the cluster analysis confirms the observation made in the principal component analysis that, water-rock interactions within the Birimian and Tarkwaian rock system as well as the indiscriminate discharge of mine tailings is responsible for the elevated levels of arsenic, cadmium, mercury and lead in the water samples.

4.3 Spatial variation of As, Cd, Hg and Pb in the study areas

Since, point source sampling protocol was used in this study, it means that the determined concentrations of water quality parameters may not provide the much needed information about the extent of pollution in the entire study areas. It is within this context that the spatial variation analysis of how each of the parameters is distributed in the study areas were carried out.

The results presented in figures 4.2 to 4.5 shows that, the underlying geological materials as well as the intensity of mining operations have contributed to the immobilisation of arsenic, cadmium, mercury and lead into the environment are the main reason accounting for variability in contaminants concentrations in the study areas.

Metal concentrations within the GS 175-1/WHO water guideline value is considered as desirable (Figures 4.2 – 4.5).

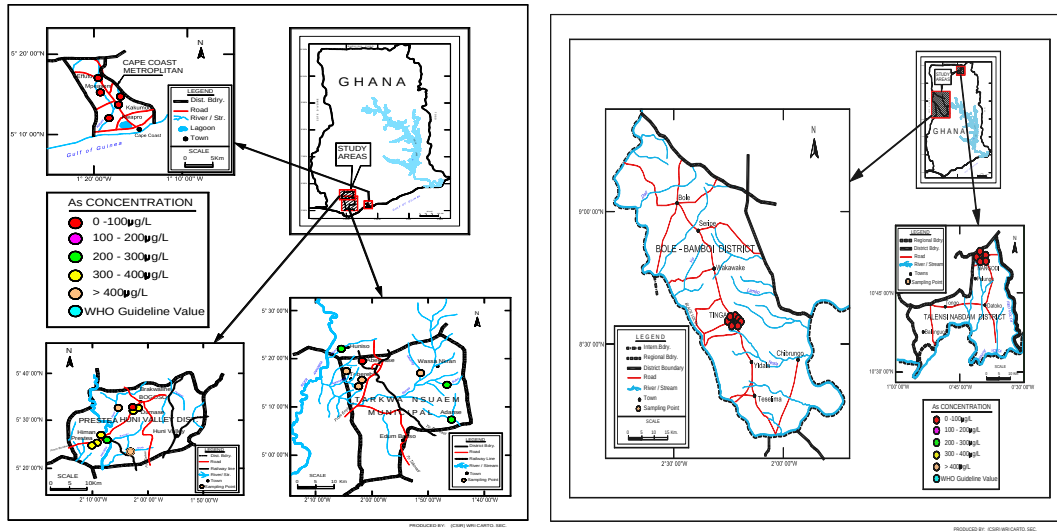


Figure 4.2: Spatial distribution of As concentrations in all the sampling locations in the study areas

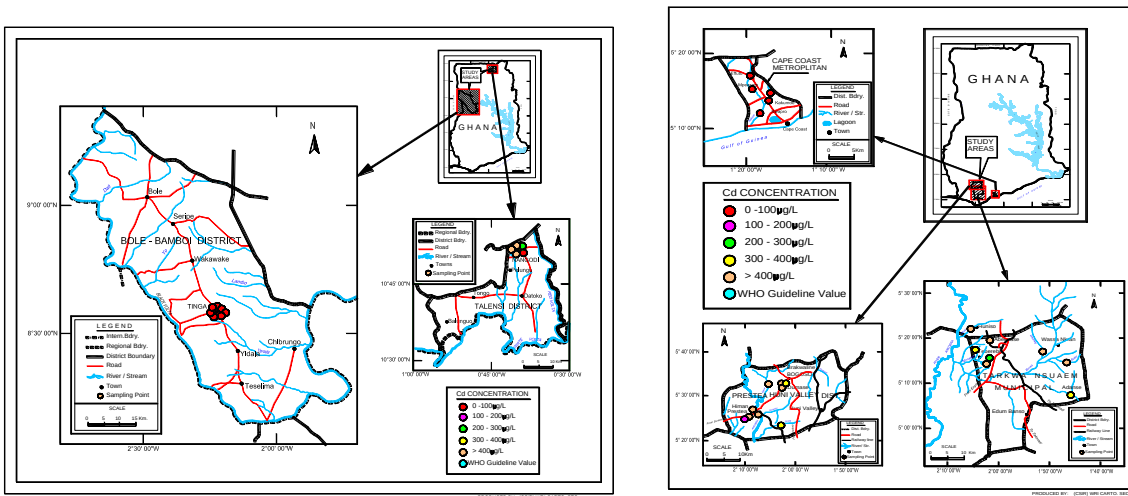


Figure 4.3: Spatial distribution of Cd concentrations for all the sampling locations in the study areas.

The order of magnitude of IDW-estimated concentrations of Cd was as follows: Tarkwa > Prestea > Tinga > Nangodi > Cape Coast. Generally, Cd was evenly distributed across water sampling locations.

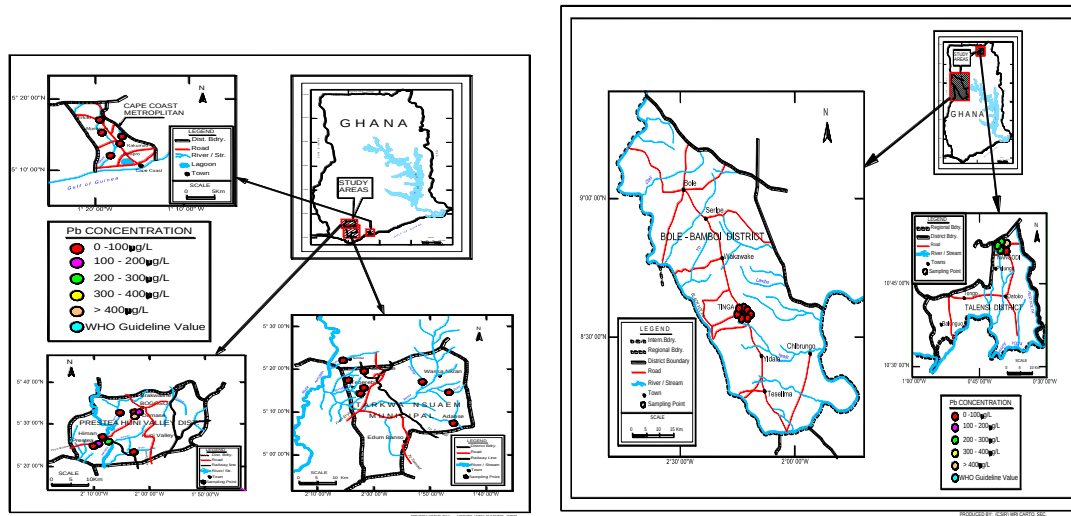


Figure 4.4: Spatial distribution of Pb concentrations for all sampling locations in the study areas.

The order of magnitude of IDW-estimated concentrations of Pb as follows: Tarkwa > Prestea > Tinga > Nangodi > Cape Coast. Generally, there is a Pb pollution gradient from south (predominantly mining areas) to relatively new mining sites in the 3 northern regions.

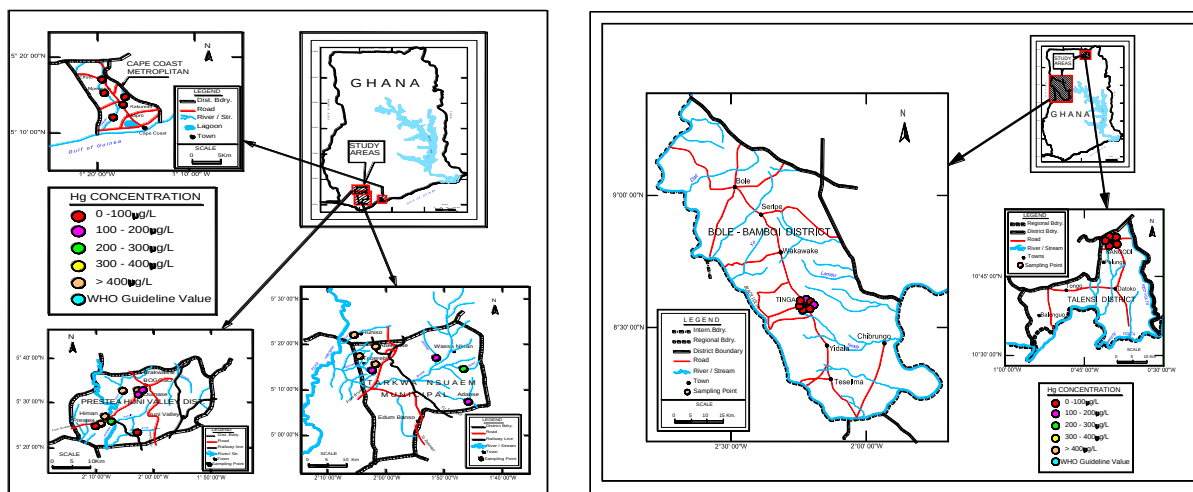


Figure 4.5: Spatial distribution of Hg concentrations for all the sampling locations in the study areas.

The decreasing order of magnitude of IDW-estimated concentrations of Hg as follows: Tarkwa > Prestea > Tinga > Nangodi > Cape Coast.

4.4 Concentrations of heavy metals in food crops grown in mining and non – mining communities

Tuber crops such as cassava grown on contaminated soils absorb these toxic chemicals from the soil via symplastic pathways. Levels of toxic chemicals absorbed by these tuber crops are used as indicators to measure the extent of contamination of the soil. Also, human beings or animals who consume these contaminated food crops are usually at risk.

Table 4.19 summaries the mean arsenic, cadmium, mercury and lead concentrations in cooked CSIR-CRI bankye buroni from the study areas.

From Table 4.19 below, it could be observed that the concentration of cadmium absorbed by CSIR-CRI bankye buroni grown in Tarkwa in the TNMA is 8.80 mg/kg, significantly higher than the WHO permissible levels of daily oral intake for cadmium in cassava, as well as the same cassava sample from the University of Cape Coast (UCC) Research Farm.

Table 4.19 Mean levels of As, Pb, Cd and Hg in Cooked Cassava samples in the study areas

Sampling Point	Concentration in mg/kg			
	As $\pm$ SD*	Pb $\pm$ SD*	Cd $\pm$ SD*	Hg $\pm$ SD*
Tamso	22.9 $\pm$ 5.45	1.014 $\pm$ 0.06	10.8 $\pm$ 1.19	15.5 $\pm$ 1.34
Tarkwa	21.5 $\pm$ 3.19	0.567 $\pm$ 0.02	12.2 $\pm$ 2.54	19.8 $\pm$ 1.14
Prestea	30.7 $\pm$ 9.89	8.843 $\pm$ 1.01	7.64 $\pm$ 1.45	16.9 $\pm$ 4.13
Bogoso	19.8 $\pm$ 7.86	0.890 $\pm$ 0.09	11.3 $\pm$ 1.34	45.6 $\pm$ 5.67
UCC Research Farm	0.0001 $\pm$ 0.01	0.004 $\pm$ 0.01	0.876 $\pm$ 0.08	2.65 $\pm$ 0.92
Nsuaem	15.5 $\pm$ 5.68	1.050 $\pm$ 0.08	9.25 $\pm$ 1.09	20.2 $\pm$ 3.09
Effuantah	3.66 $\pm$ 3.12	0.273 $\pm$ 0.12	1.431 $\pm$ 1.34	3.21 $\pm$ 0.09
Tinga	32.1 $\pm$ 12.8	5.35 $\pm$ 2.43	12.4 $\pm$ 1.43	98.6 $\pm$ 13.7
Nangodi	20.6 $\pm$ 7.89	6.47 $\pm$ 2.13	19.2 $\pm$ 2.19	113.5 $\pm$ 15.6
GS 175 -2/WHO guideline value	0.0003	-	0.00054	0.0003

* SD = Standard Deviation; ** Bold figures exceeds GS – 175 – 2/WHO Guideline Values

From Table 4.19, the decreasing order of arsenic concentration in the CSIR-CRI bankye buroni grown in the study areas are as follows: Prestea > Tamso > Tarkwa > Bogoso > Nsuaem > Effuantah > Tinga > Tinga > UCC Research Farm respectively. The consumption of cooked cassava contaminated with arsenic, cadmium, mercury and lead by human beings may pose serious health risk to them. This finding is consistent with an earlier works by other scientists (Amasa, 1975; Amono – Neizer, et al., 1994; Essumang et al., 2007; Obiri et al., 2006b).

4.5 Concentrations and variations of heavy metals in human blood samples from the study areas

This section of the thesis sought to evaluate the levels of heavy metals such as arsenic, cadmium, lead and mercury in blood samples of resident adults and children as well as workers of mining companies including ASGM operators in the study areas.

The socio-demographic data of participants who donated their blood samples have been presented in Table 4.20. It can be seen from Table 4.20 that, out of 400 participants that were randomly selected from TNMA/PHVD (i.e., 300 resident adults which is made up of both miners and non – miners, and 100 children), only 350 of them met the exclusion criteria (comprising 260 adults and 90 children). From this number, only 300 (comprising of 240 adults and 60 children) of them gave their consent and agreed to be transported to the Tarkwa Municipal Hospital for their blood sample to be taken, giving an overall response rate of 85.7%. (Table 4.20).

Similarly, in the Cape Coast Metropolis (CCMA), a non – mining community, a total of 150 participants including 100 resident adults and 50 children were randomly selected to be part of this study, out of which 50 (30 adults and 20 children) of them did not meet the exclusion criteria. The remaining participants comprising of 80 adults and 20 children participated in the study, resulting in an overall response rate of 66.7%.

For Nangodi mining community - Nabdam District, a total of 150 participants (100 adults – miner and non – miners as well as 50 children) were randomly recruited to be part of this study, out of which 40 (15 adults and 25 children) of them did not meet the exclusion criteria.

The remaining 110 participants which is made up of 85 adults (miners and non – miners) and 25 children actually participated in the study giving an overall response rate of 73.3%.

In the case of the Tinga mining community, a total of 150 participants were randomly recruited which is made up of 100 adults (including miners and non – miners) and 50 children to be part of this study, out of which 35 (15 adults and 20 children) of them did not meet the exclusion criteria. The remaining 115 participants which is made up of 85 adults (miners and non – miners) and 30 children actually participated in this study giving an overall response rate of 76.7%.

It can be seen from Table 4.20 below that more men are actively involved in mining operations in the study areas such as TNMA, PHVD, BBD and ND. That is, out of the 20 large-scale mine workers who participated in this study, 15 of them are men.

In the ASGM sector, there are 110 men as against 45 women who enrolled in this study. The higher number of male subjects than the females working with either large – scale mining companies or the artisanal small – scale mines in the participating mining communities is due to the fact that mining is seen as a masculine job.

How, for resident adult non-mine workers category, more adult women participated from the study areas than men. For example, 80 women as against 60 men from Tarkwa Nsuaem Municipality/Prestea huni Valley District, and 25 women as against 20 men from Nangodi in Nabdam District. This observation is due to the fact that usually most of the men who are not directly employed by either the mining company or ASGM operators often leave the women behind and travel to other towns in search of greener pastures.

Table 4.20: Demographic characteristics of the study population

Demographic factors	Gold Mining Areas						Non – gold mining area		Overall Total	Overall %
	TNMA/ PHVD	%	Nangodi	%	Tinga	%	CCMA	%		
Occupation:										
<i>Adults Miners :</i>										
Miners: 1. *Companies	20	6.6	-	-	-	-	-	-	20	3.2
2. **ASGM	80	26.7	40	36.4	35	30.4	-	-	155	24.8
Adult Non – Miners	140	46.7	45	40.9	50	43.5	80	80	315	50.4
Children	60	20.0	25	22.7	30	26.1	20	20	135	21.6
Sex:										
<i>Adult Miners</i>										
1. Companies -Male (M)	15	5.0	-	-	-	-	-	-	15	2.4
Female (F)	5	1.7	-	-	-	-	-	-	5	0.8
2. ASGM - Male (M)	60	20.0	30	27.3	20	17.4	-	-	110	17.6
Female (F)	20	6.7	10	9.1	15	13.0	-	-	45	7.2
<i>Non - Miners</i>										
Adult: Male (M)	60	20.0	20	18.2	35	30.4	45	45	160	25.6
Female (F)	80	26.6	25	22.7	15	13.0	35	35	155	24.8
Children: M	30	10.0	15	13.6	20	17.4	10	10	75	12
F	30	10.0	10	9.1	10	8.8	10	10	60	9.6
Age (years):										
<i>Adults</i>										
20 – 29	55	18.3	23	20.9	32	27.8	21	21.0	131	21.0
30 – 39	102	34.0	21	19.1	15	13.0	19	19.0	157	25.2
40 – 49	60	20.0	27	24.5	20	17.4	26	26.0	133	21.3
≥ 50	23	7.7	14	12.7	18	15.7	14	14.0	69	11.0
<i>Children</i>										
5 – 10	27	9.0	12	10.9	14	12.2	8	8.0	61	9.8
11 – 16	33	11.0	13	11.9	16	13.9	12	12.0	74	11.8
Number of years of staying in the study areas:										
5 – 20 years										
21 – 36 years	180	60.0	55	50.0	60	52.2	46	46.0	341	54.6
>36 years	90	30.0	30	27.3	35	30.4	24	24.0	179	28.6
	30	10.0	25	22.7	20	17.4	30	30.0	105	16.8
Use of earthenware cooking materials:										
Yes	190	79.2	55	64.7	65	76.5	20	25.0	330	67.3
No	50	20.8	30	35.3	20	23.5	60	75.0	160	32.7
Type of drinking water:										
Streams/rivers/dug-out	177	59.0	25	22.7	54	47.0	7	7.0	256	42.1
Boreholes	83	27.7	85	77.3	61	53.0	6	6.0	241	39.5
Tap water	40	13.3	-	-	-	-	88	88.0	128	20.4
Housing conditions:										
Mud house	124	41.3	43	39.1	55	47.8	-	-	222	35.5
Mud house with concrete floor	121	40.3	47	42.7	35	30.5	15	15.0	218	34.9
Concrete house	55	18.4	20	18.2	25	21.7	85	85.0	185	29.6

*Companies refers to workers of large – scale mining companies. ** ASGM refers to workers in legal small – scale gold mines

From Table 4.20, 67.3%) of participants used earthenware to cook their food. However, within the various study areas, there exist variations in the number of participants who use earthenware to cook. For example, 79.2% of participants in TNMA/PHVD, 64.7 % of participants in Nangodi, 76.5% of participants in Tinga), and 25% of participants in CCMA respectively used the earthenware to cook. The earthenware are made of clay; as no chemical analyses are carried on the clay used to make the earthenware which is usually obtained from the mining communities, it could be a potential source of exposure to arsenic, cadmium, mercury or lead.

In this study, 256 out of the total 525 participants for this study (48.8%) from the selected mining communities for this study depend on streams/rivers or hand dug – out wells as the source of drinking water; 229 (43.6%) reported that they depend on boreholes whilst 40 (7.61%) of them mostly in urban centres in the Tarkwa Nsuaem Municipality and Prestea Huni Valley District are served with tap water; whereas in the non – mining communities in Cape Coast Metropolis, we found out that most of the participants 88 (88%) are served with tap water, 7% and 6% of the participants respectively depend on stream/rivers and boreholes for their source of drinking water.

The overall mean whole blood concentrations of arsenic, cadmium, mercury and lead for resident adults in the study areas have been summarised in Table 4.21.

From Table 4.21, it can be seen that, resident adult non-miners in TNMA, PHVD, BBD and ND have elevated levels of arsenic, cadmium and mercury in their whole blood than their counterparts in Cape Coast, a non-mining area. The mean concentration of arsenic, cadmium and mercury in whole blood of resident adult non-miners exceeded the WHO permissible guideline value.

Table 4.21 Mean arsenic, cadmium, mercury and lead levels in whole blood of resident adults non – miners) from mining and non – mining areas in Ghana.

Toxic Chemicals	Gold Mining Areas						Non – gold mining area		WHO Guideline Value (µg/L)
	TNMA/PHVD (n = 140)		Nangodi (n = 45)		Tinga (n = 40)		CCMA (n = 80)		
	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	
Arsenic	38	9.71	25	0.62	15.4	0.89	0.032	1.12	2.0
Cadmium	303	17.1	114	5.23	201	0.35	0.050	0.01	0.3
Mercury	63	0.23	72	1.02	72.1	0.86	1.10	1.02	2.0
Lead	28	13.6	15	0.05	22.2	0.61	2.40	1.08	50

***bold figures exceed WHO guideline value**

Similarly, as shown in Table 4.21, the concentration of lead in whole blood of resident adults in the study areas, i.e., TNMA/PHVD, ND, BBD and CCMA were found to be within the WHO permissible guideline. This observation is consistent with the assertion made by Kesse (1985) that, the Birimian and Tarkwaian rocks does have elevated concentrations of lead in them.

The mean concentrations of arsenic, cadmium, mercury and lead in blood serum of resident adult non-miners have been summarised in Table 4.22.

Table 4.22: Mean arsenic, cadmium, mercury and lead levels in serum of resident adults non – miners) from mining and non – mining areas in Ghana.

Toxic Chemicals	Gold Mining Areas						Non – gold mining area		WHO Guideline Value (µg/L)
	TNMA/PHVD (n = 140)		Nangodi (n = 45)		Tinga (n = 40)		CCMA (n = 80)		
	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	
Arsenic	28.5	3.14	7.82	0.43	13.1	0.25	0.18	0.02	1.0
Cadmium	9.52	0.58	5.14	0.25	3.13	0.76	0.03	0.01	0.1
Mercury	14.9	0.76	8.57	1.28	15.6	1.80	0.24	0.09	1.0
Lead	5.89	0.66	3.89	0.49	9.87	0.12	0.09	0.01	0.1

***bold figures exceed WHO guideline value**

In Table 4.22 above, mean concentration of As and Cd in blood serum of resident adults decreases according to the following order; TNMA/PHVD > Tinga > Nangodi > Cape Coast; while in the case of Hg and Pb the order is Tinga > TNMA/PHVD > Nangodi > Cape Coast respectively. It also shows that, resident adults from the mining areas had high levels of As, Cd, Hg and Pb in their blood serum than their counterparts from non-mining areas. This observation is consistent with similar findings made by other scientists (Obiri et al., 2016; Sanders et al., 2014).

Tables 4.23 and 4.24 below gives mean concentrations of As, Cd, Hg and Pb in whole blood and serum of resident children in the study areas.

Table 4.23 Mean concentration of heavy metals in serum of resident children in the study areas

Toxic Chemicals	Gold Mining Areas						Non – gold mining area		WHO Guideline Value (µg/L)
	TNMA/PHV D (n = 60)		Nangodi (n = 25)		Tinga (n = 30)		CCMA (n = 20)		
	Conc. µg/L	SD	Conc . µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	
Arsenic	52	3.20	2.57	0.12	6.15	0.92	0.39	0.48	1.0
Cadmium	10	3.12	3.18	0.39	1.25	0.19	0.015	0.04	0.1
Mercury	163	0.52	4.78	0.03	9.06	1.36	0.63	0.01	1.0
Lead	10	2.11	1.31	0.01	1.49	0.22	0.091	0.01	0.1

***Bold figures have exceeded the WHO guideline values**

It can be seen from Table 4.23, that arsenic, cadmium, mercury and lead concentrations in blood serum of resident children in the mining areas were higher than those in the non-mining areas. These concentrations also exceeded the WHO guideline value.

Table 4.24: Mean concentration of heavy metals in the whole blood of resident children in the study areas.

Toxic Chemicals	Gold Mining Areas						Non – gold mining area		WHO Guideline Value (µg/L)
	TNMA/PHV D (n = 60)		Nangodi (n = 25)		Tinga (n = 30)		CCMA (n = 20)		
	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	Conc. µg/L	SD	
Arsenic	10.57	1.58	7.76	1.16	3.48	0.52	0.18	0.03	2.0
Cadmium	6.55	0.98	5.91	0.89	4.67	0.70	0.012	0.01	0.3
Mercury	12.61	1.91	10.2	0.81	9.76	1.46	0.39	0.26	2.0
Lead	3.80	0.57	5.08	0.81	4.53	0.68	0.020	0.01	50

***bold figures exceed WHO guideline values**

The results of the t – test shows that, at $\alpha = 0.05$ (95% CI). there was statistical significant association between mean concentrations of arsenic, cadmium, mercury and lead levels in whole blood and serum of men and women from the mining communities, ;

The t-test also revealed a statistically significant association between participants who cooked in earthen wares and the concentration of arsenic, mercury, cadmium and lead in their whole blood or serum as against those who do not cook in earthen ware.

Arsenic, cadmium, mercury and lead concentrations in both whole and blood serum were statistically significant with the participant's length of stay in mining communities; although the such correlation was lower in men, $r = 0.275$ ($\alpha = 0.01$) than in women, $r = 0.561$. This findings is consistent with observations made by the following scientists Seifert et al., (2000), Batariova et al. (2006) and Jakubowski et al. (1996).

Tables 4.25 and 4.26 gives demographic characteristics of mine workers as well as the mean concentration of toxic chemicals measured in whole and blood serum samples collected from a total of 190 mine workers, out of which 20 of them are workers of mining companies operating in Tarkwa Nsuaem Municipality/Prestea Huni Valley District, with the remaining 170 of them working with the artisanal small – scale mines from Nangodi in Nabdam District, Tinga in the Bole – Bamboi District as well as in TNMA/PHVD.

Table 4.25: Whole blood arsenic, cadmium, lead and mercury concentration among mine workers (ASGM and mining companies) classified by work section in the study areas

Work section	TNMA/PHVD				Nangodi				Tinga			
	Conc. µg/L (Mean ± SD)				Conc. µg/L (Mean ± SD)				Conc. µg/L (Mean ± SD)			
	As	Cd	Hg	Pb	As	Cd	Hg	Pb	As	Cd	Hg	Pb
<u>Mining companies</u>												
Crushing/screening	3.1±0.1	1.10±1.1	0.09 ± 0.1	0.5±0.2								
Drilling/blasting	2.1±0.1	1.9±0.1	0.01	1.3±0.1								
Grinding	1.8±0.2	0.98±0.1	0.01	1.4±0.2								
Loading/dumping	-	-	-	-								
Maintenance workshop	-	-	-	-								
Mineral characterization lab	1.51±1.1	1.45±0.2	0.01	2.1±0.1								
Office work	-	-	-	-								
Security	-	-	-	-								
Tailings dam	2.23±1	1.4±3.6	0.02	2.07±0.2								
Overall mine workers	10.7±4	6.86±2.2	0.14	7.37±3.1								
<u>Artisanal Small – scale Gold Mines</u>												
Digging/blasting/drilling	23±0.9	13.1±2.6	3.72±0.3	1.23±0.3	2.75±0.1	3.33±0.1	4.27±0.1	2.01±0.1	4.14±0.2	5.34±0.1	2.06±0.3	1.35±0.2
<u>Grinding/sieving of ores</u>	14±0.1	3.46±0.3	2.73±0.5	2.15±0.1	2.57±0.4	1.37±0.2	5.18±0.7	3.18±0.4	4.78±0.7	2.06±0.3	8.76±1.3	3.16±0.5
<u>Washing of ores</u>	20±0.5	7.68±0.7	2.21±0.5	1.22±0.3	9.06±1.3	5.64±0.6	7.23±1.1	6.15±0.4	4.99±0.8	6.65±1.1	3.81±0.6	4.37±0.6
<u>Amalgamation</u>	10±0.1	5.31±0.2	15.2±1.2	1.18±0.3	6.15±0.9	4.82±0.7	9.78±1.1	3.32±0.5	6.76±0.3	2.00±1.2	11.2±0.6	4.99±2.1
Roasting gold amalgam	20±0.3	4.04±0.3	28.3±3.1	2.03±0.2	3.81±0.5	3.92±0.8	10.2±2.6	3.27±0.1	2.39±0.4	2.53±0.8	15.9±0.4	3.49±0.5

*Overall workers	ASGM	107±5.3	33.6±6.0	52.2±0.2	7.81±2.3	24.34±0.1	19.08±2.3	16.68±5.6	17.93±4.5	23.06±4.2	18.58±3.3	41.72±6.2	17.36±2.6
WHO Value	Guideline	2	0.3	2.0	50	2	0.3	2	50	2	0.3	2	50

Table 4.26: Serum arsenic, cadmium, lead and mercury concentration among mine workers (ASGM and mining companies) classified by work section in the study areas

Work section (n)	TNMA/PHVD				Nangodi				Tinga			
	Conc. µg/L (Mean ± SD)				Conc. µg/L (Mean ± SD)				Conc. µg/L (Mean ± SD)			
	As	Cd	Hg	Pb	As	Cd	Hg	Pb	As	Cd	Hg	Pb
<u>Mining companies</u> Crushing/scree ning	3.21±0.5	4.05±0.6	-	1.85±0.3	-	-	-	-	-	-	-	-
Drilling/blasting	2.34±0.4	3.12±0.5	-	3.12±0.5	-	-	-	-	-	-	-	-
Grinding	2.26±0.3	3.81±0.6	-	3.25±0.5	-	-	-	-	-	-	-	-
Loading/dumping	1.65±0.2	0.33±0.05	-	1.61±0.2	-	-	-	-	-	-	-	-
Maintenance workshop	0.28±0.04	1.85±0.3	-	0.38±0.06	-	-	-	-	-	-	-	-
Mineral characterization laboratory	3.29±0.2	2.26±0.3	-	1.86±0.3	-	-	-	-	-	-	-	-
Office work	-	-	-	-	-	-	-	-	-	-	-	-
Security	-	-	-	-	-	-	-	-	-	-	-	-
Tailings dam	3.48±0.8	1.99±0.3	-	1.61±0.2	-	-	-	-	-	-	-	-
Overall mine workers	16.51±4.2	13.6±3.2	-	13.68±1.5								
<u>Artisanal Small – scale Gold Mines</u> Digging/blasting/Drilling	7.09±1.1	5.08±0.8	3.64±0.6	1.29±0.2	3.78±0.4	6.03±0.9	5.53±0.8	4.20±0.6	8.25±0.5	4.21±0.2	3.39±0.6	2.67±0.4
Grinding/sieving	4.82±0.7	3.79±0.6	6.79±1.1	7.23±1.2	4.89±0.7	5.18±0.8	6.65±1.0	6.32±0.9	9.06±1.4	5.29±0.2	15.5±0.7	9.49±0.8
Washing of ores	8.31±0.6	2.30±0.4	2.88±0.4	5.43±0.7	7.69±1.5	2.33±0.4	4.67±0.7	2.88±0.4	6.32±0.5	10.2±0.5	14.8±0.4	5.53±0.8
Amalgamation	9.81±2.1	7.33±2.3	18.1±0.9	8.62±0.4	6.97±1.1	7.29±1.8	13.8±0.5	4.72±0.2	5.64±0.6	8.51±0.3	9.87±0.5	8.53±0.6

Roasting gold amalgam	9.01±0.2	7.42±0.4	8.82±0.6	8.05±3.2	6.13±0.5	6.39±0.4	8.64±0.3	9.10±0.6	6.54±2.1	7.14±.01	15.4±0.8	3.13±0.4
Overall ASGM workers	39.04±2.1	25.92±5.6	40.23±9.2	30.62±4.2	29.46±5.2	27.22±0.5	39.29±5.6	27.22±0.9	35.81±5.6	35.35±8.2	59.96	29.35±4.2
WHO Guideline Value	1.0	0.1	1.0	0.1	1.0	0.1	1.0	0.1	1.0	0.1	1.0	0.1

It can be seen from Tables 4.25 and 4.26 that, the levels of arsenic, cadmium, mercury and lead in whole blood or serum of participants engaged in mining operations in the study areas varies according to the section that the participant is working. For example, workers at the milling section of the large-scale mining companies have higher arsenic concentrations than their counterparts at the maintenance workshop.

Also, a comparison of workers at the milling section where the gold ores are mined in the large-scale mining companies have lower arsenic blood concentrations than the colleagues working in the same unit in the artisanal small-scale gold mining companies. The reason is that, in the large-scale mining companies, there is strict enforcement of wearing of protective equipment as well as observance of health and safety protocols.

4.6.0 Summary of carcinogenic and non-carcinogenic health risk faced by all the receptors in the study areas.

This section presents the cancer health risk results via oral and dermal contact to arsenic and cadmium in water, sediment as well as ingestion of cooked cassava by the following receptors, i.e., resident adult non-mine workers, resident children and ASGM workers.

4.6.1 Cancer health risk results faced by ASGM workers in Prestea Huni Valley District, Bole – Bamboi District, Nabdram District and Tarkwa Nsuaem Municipality

The cancer health risk faced by resident adult ASGM workers in the PHVD have been summarised in Fig. 4.6. The other cancer result obtained via oral and dermal contact with water or sediment from all the sampling stations in the PHVD have been presented in appendix 5.

The results indicate that, the cancer health risk in most cases exceeded the USEPA acceptable cancer health risk value of 1×10^{-4} to 1×10^{-6} ; i.e., 1 case of cancer in every 10,000 people to 1 case of cancer in every 1,000,000 people.

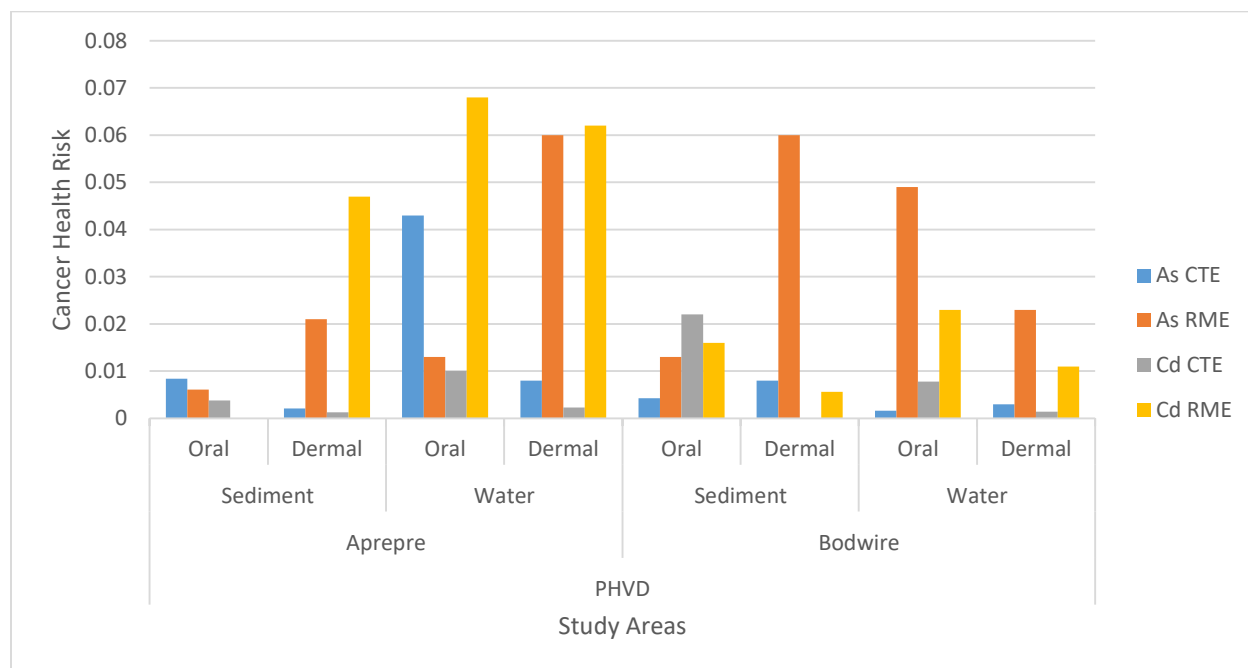


Fig. 4.6: Cancer health risk faced by ASGM workers in PHVD exposed to As and Cd in water and sediment samples

The results in Fig. 4.6 shows that, the estimated lifetime cancer risk for artisanal miners working along River Aprepre in Dumase in the Prestea Huni Valley District (PHVD). The cancer health risk result obtained is 4.5×10^{-2} and 2.3×10^{-2} via CTE parameters for oral and dermal contact of arsenic in the contaminated water from River Aprepre. This means that, approximately 5 artisanal mine (ASGM) workers out of 100 workers are likely to suffer from arsenic-related cancer diseases over a 70 year period as a result of ingestion of arsenic contaminated water from

River Aprepre via CTE parameters whilst approximately 2 ASGM workers out of 100 would develop arsenic-related cancer via dermal contact with water samples from River Aprepre based on RME parameters. Similarly, 9 and 8 out of every 100 artisanal mine workers working along River Aprepre are likely to suffer from cancer related diseases due to exposure to cadmium in water samples from the River Aprepre.

Similar cancer health risk result faced by ASGM workers in TNMA is presented in Fig. 4.7 below.

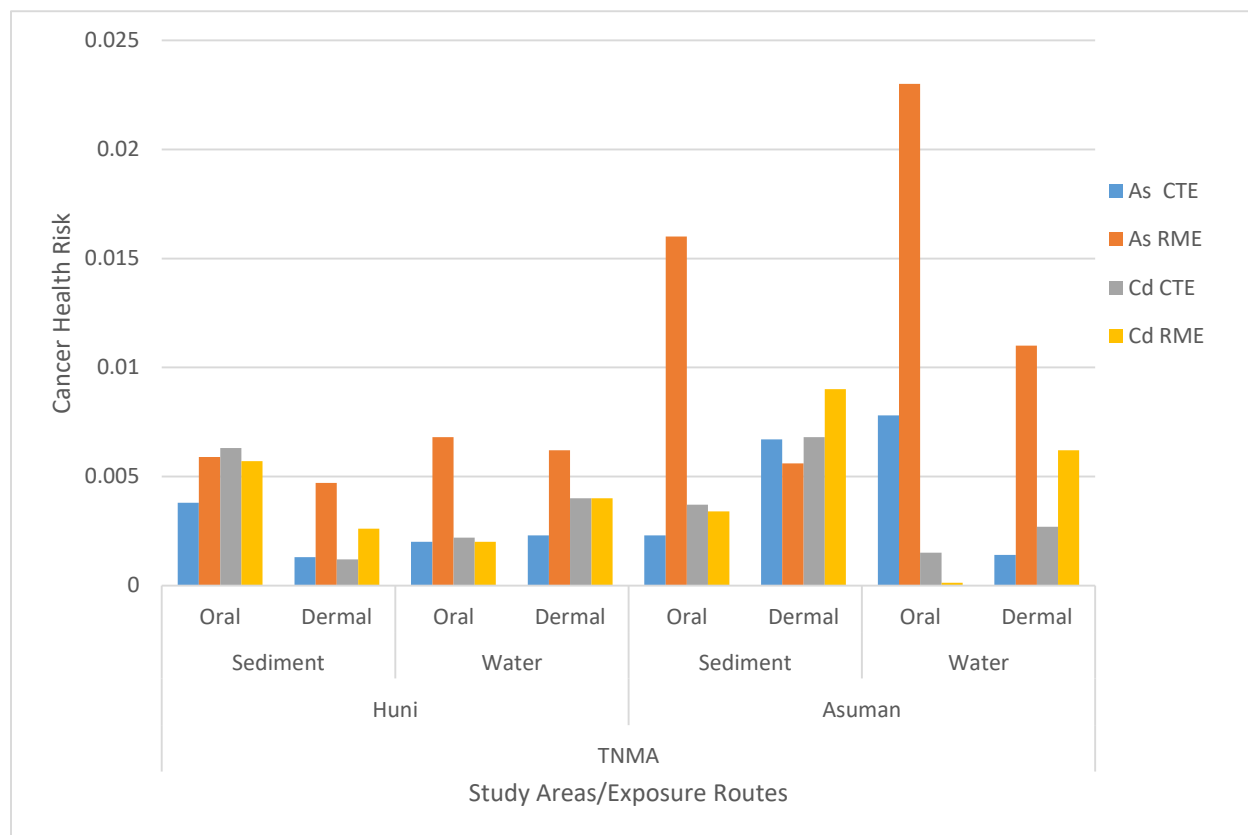


Fig. 4.7: Cancer health risk faced by ASGM workers in TNMA exposed to As and Cd in sediment and water samples.

For example, in Fig. 4.7 above, the estimated cancer health risk faced by ASGM workers in TNMA from exposure to As in river Huni via oral ingestion of sediment 3.8×10^{-3} (CTE) and 5.9×10^{-3} (RME), which means that approximately 4 out of 1,000 workers are likely to suffer from cancer related cases by CTE parameters while approximately 6 out of 1,000 ASGM worker would suffer from cancer related diseases by RME parameters respectively.

The cancer health risk faced by ASGM workers in Nangodi and Tinga mining communities are shown in Fig. 4.8 and Fig. 4.9 below respectively.

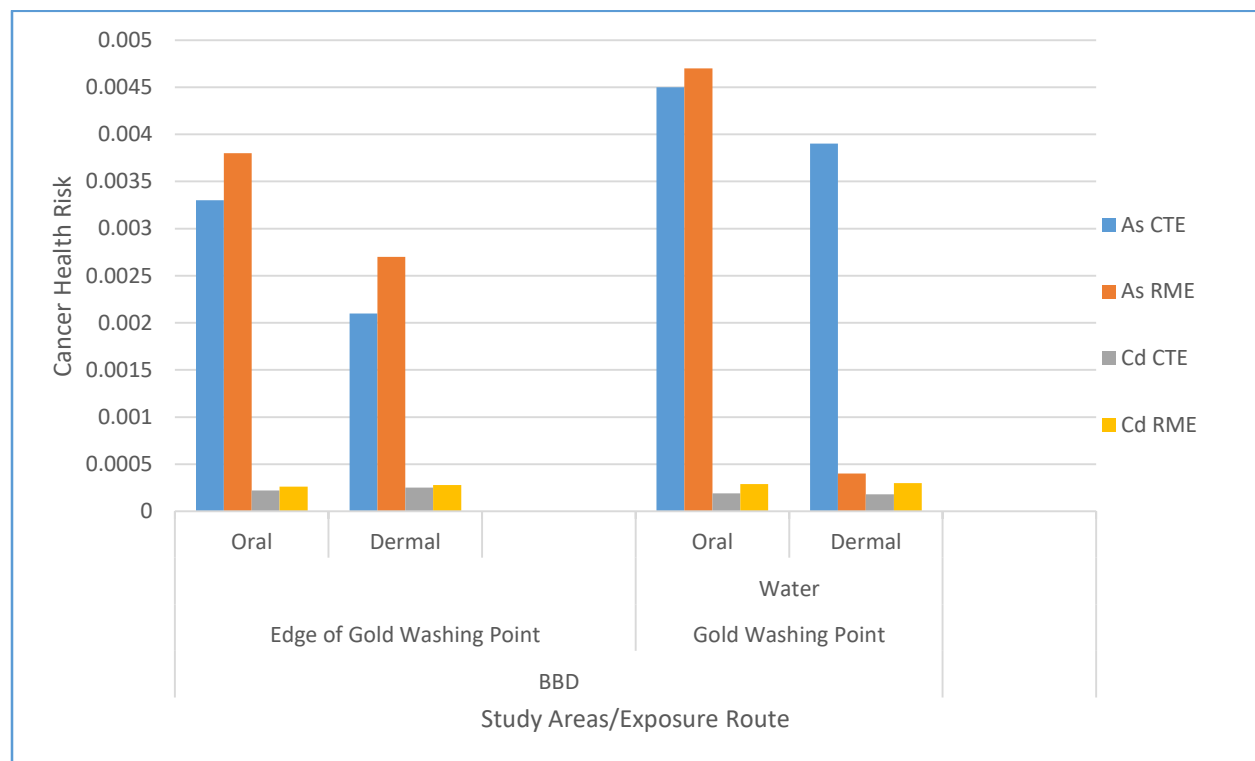


Fig. 4.8: Cancer health risk faced by ASGM workers in Tinga in Bole – Bamboi District exposed to As and Cd in water samples.

The cancer health risk faced by ASGM workers via ingestion of As in water at the sampling site GWPS (i.e., gold washing part of the stream) was 4.2×10^{-3} (which means 3 out of 1000 ASGM workers are likely to suffer from cancer related diseases from ingestion of As in the water from the stream), while for dermal contact to As in the sediment we have approximately 3 out of 1000 ASGM likely to show cancer related symptoms by CTE parameters respectively. The cancer health risk results faced by ASGM workers in Tinga from exposure to As and Cd in water samples from the boreholes were in some cases found to have exceeded the USEPA acceptable cancer risk.

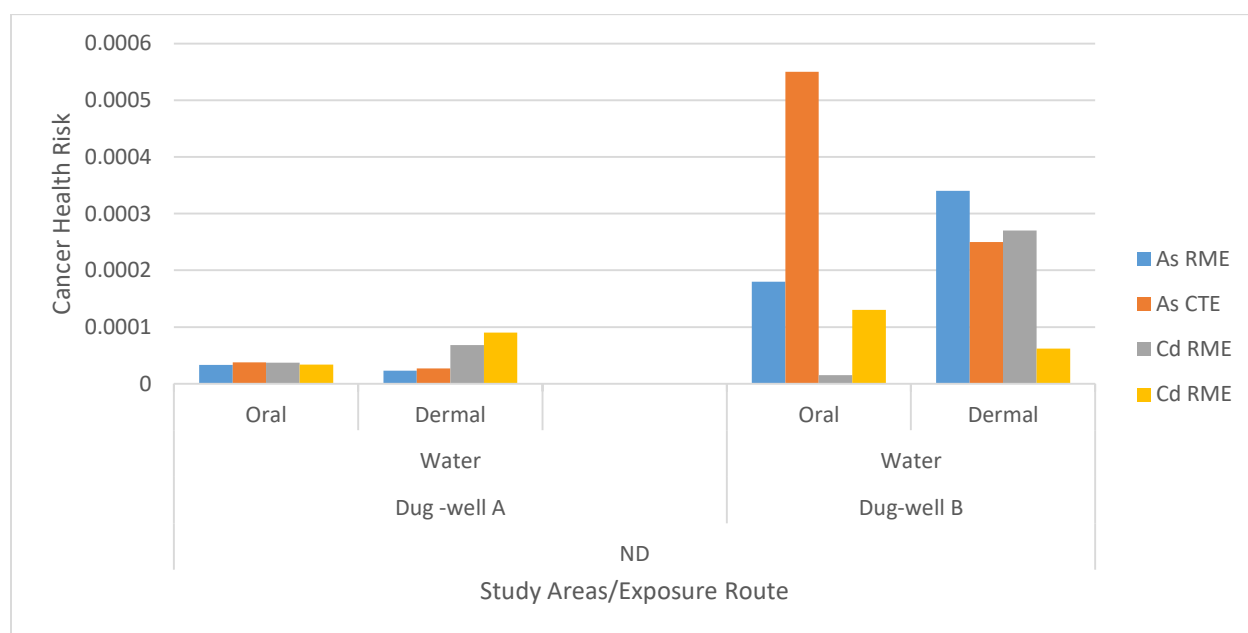


Fig. 4.9: Cancer health risk faced by ASGM workers in Nabdam District exposed to As and Cd in sediment and water samples.

From Fig. 4.9, the cancer health risk faced by ASGM workers in Nangodi community via oral and dermal contact with As and Cd in water samples from the dug – out wells A and B were found to be within the USEPA acceptable cancer range of 1×10^{-4} to 1×10^{-6} for both CTE and RME parameters.

That is, 1 case of cancer in every 10,000 people to 1 case of cancer in every 1,000,000 people.

A comparison of the cancer health risk faced by ASGM workers in Nabdam district is less than what was obtained for ASGM workers in TNMA, PHVD and Bole-Bamboi district respectively. This is due to the fact that activities of artisanal gold mining are relatively new in communities within the Nabdam district. This notwithstanding, the findings of this study holds several policy implications for ASGM workers in the Nabdam district (Fig. 4.10).

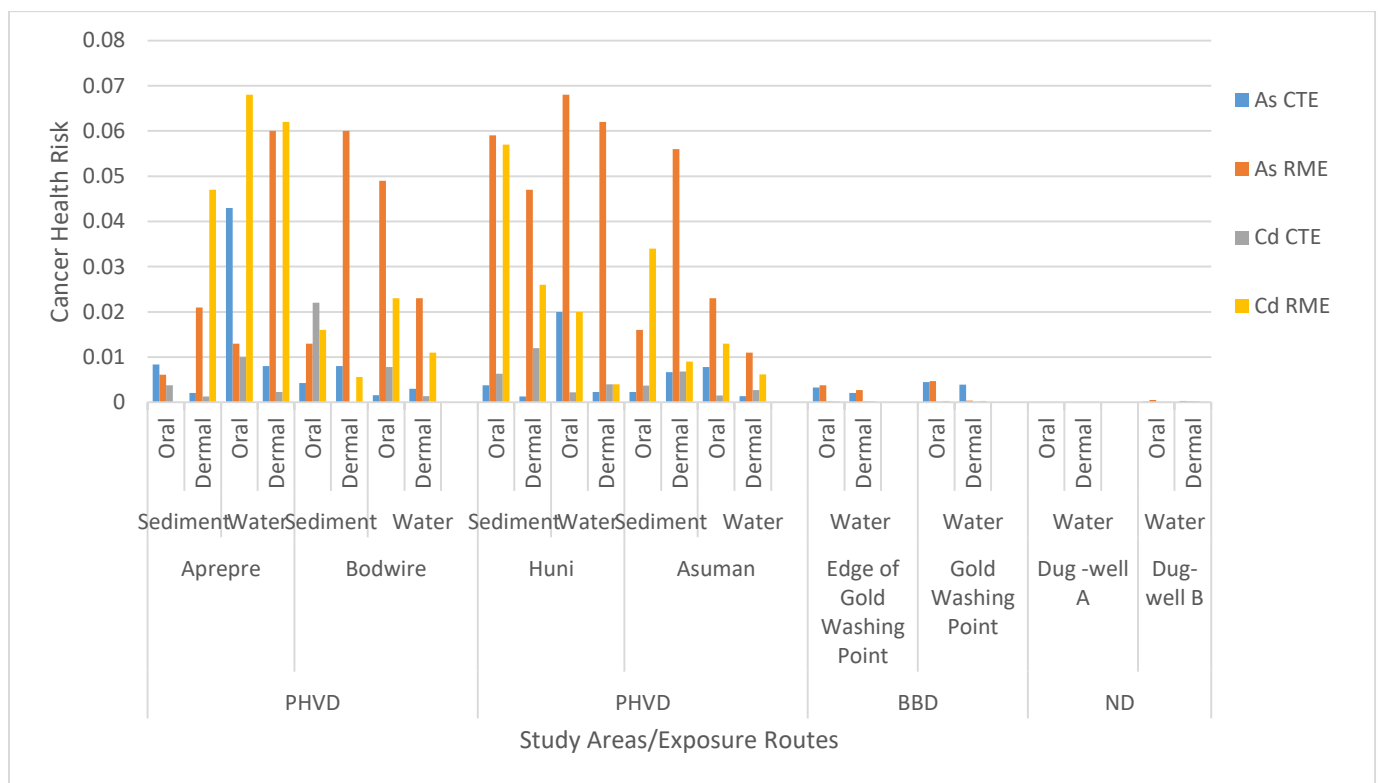


Fig. 4.10: A graph comparing cancer health cases among ASGM workers in the study areas

4.6.2 Cancer health risk results faced by resident adults in the study areas

The cancer health risk faced by resident adults via oral ingestion of arsenic in water from river Huni in Huniso in TNMA is 5.6×10^{-3} and 5.9×10^{-3} (Fig. 4.11) by both CTE and RME parameters respectively. This means that approximately, 6 out of 1000 resident adults in Huniso who drink water from river Huni are likely to suffer cancer related disease.

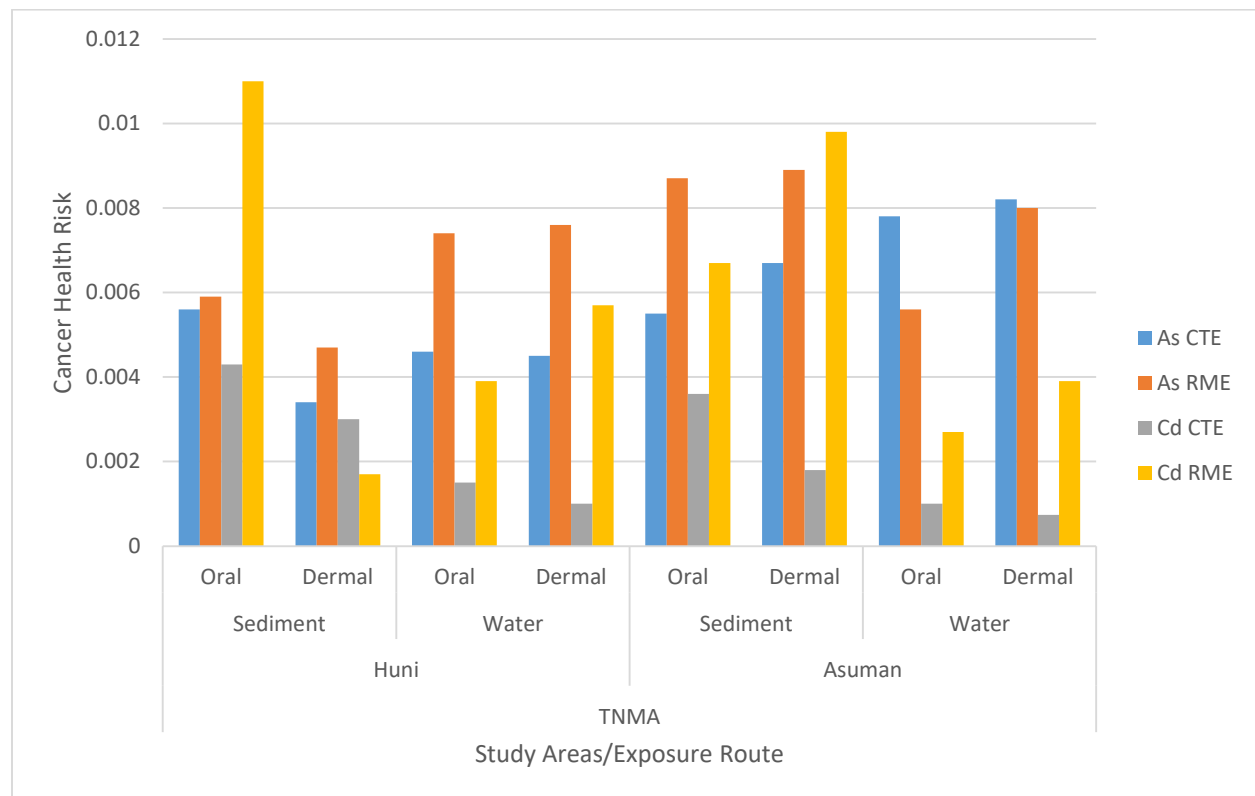


Fig. 4.11: Cancer health risk faced by resident adults in TNMA exposed to As and Cd in sediment and water samples.

Similarly, the cancer health risk faced by resident adult non-mine workers in the PHVD is presented in Fig. 4.12.

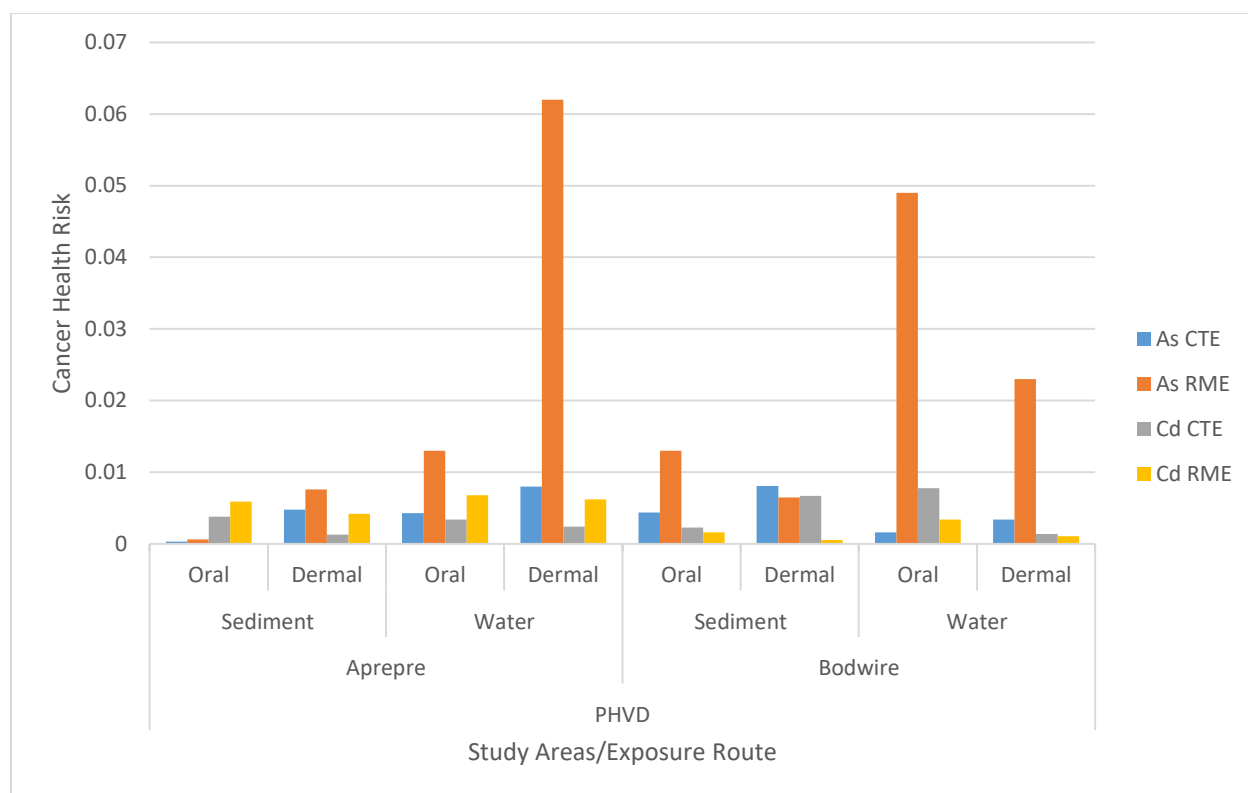


Fig. 4.12: Cancer health risk faced by resident adults in PHVD

It can be seen in Fig. 4.12, that the cancer health faced by resident adults in Dumase via oral ingestion of As in River Aprepre is 4.4×10^{-3} by CTE parameters, while that of dermal contact is 8.1×10^{-3} (CTE). This means that, approximately, 4 and 8 out of every 1,000 are likely to suffer from cancer related diseases via oral and dermal contact to arsenic in River Aprepre.

The results of cancer health risk from exposure to arsenic by resident adults in Kawprow (Fig. 4.13) near Cape Coast are as follows; for resident adults, it is 2.3×10^{-6} and 3.5×10^{-6} (by CTE parameters) via oral and dermal contact, which means that approximately, 2 and 4 resident out of every 1,000,000 people are likely to suffer from cancer related diseases respectively by CTE and RME parameters.

The above estimated lifetime cancer health calculated in Cape Coast Metropolis were found to be in line with USEPA acceptable cancer health risk, 1.0×10^{-6} to 1.0×10^{-4} which means one case of cancer in every 1,000,000 people to one case of cancer in every 10,000 people.

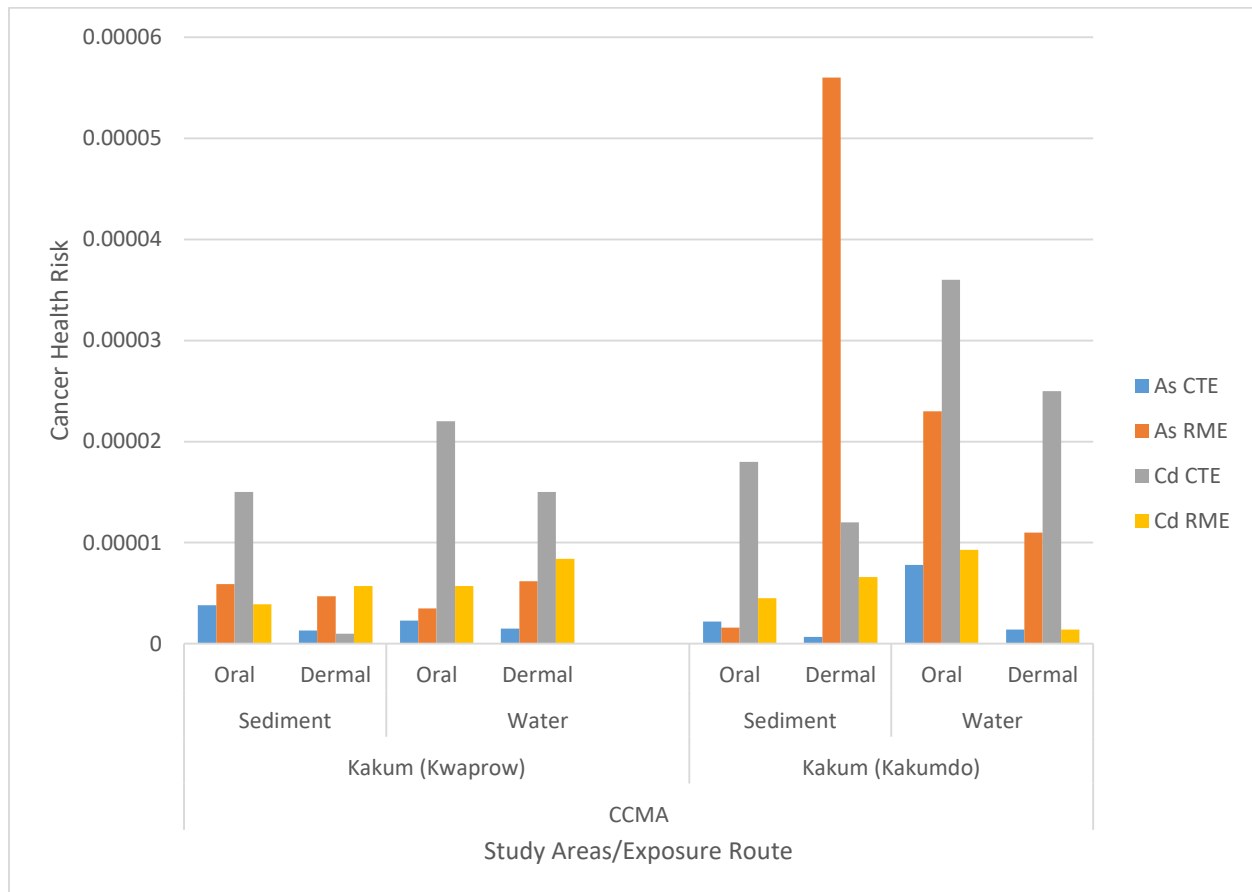


Fig. 4.13: Cancer health risk faced by resident adults and children in CCMA exposed to As and Cd in sediment and water samples.

The cancer health risk for resident adults in Nangodi a mining community in Nabdam District in the Upper East region (Fig. 4.14)

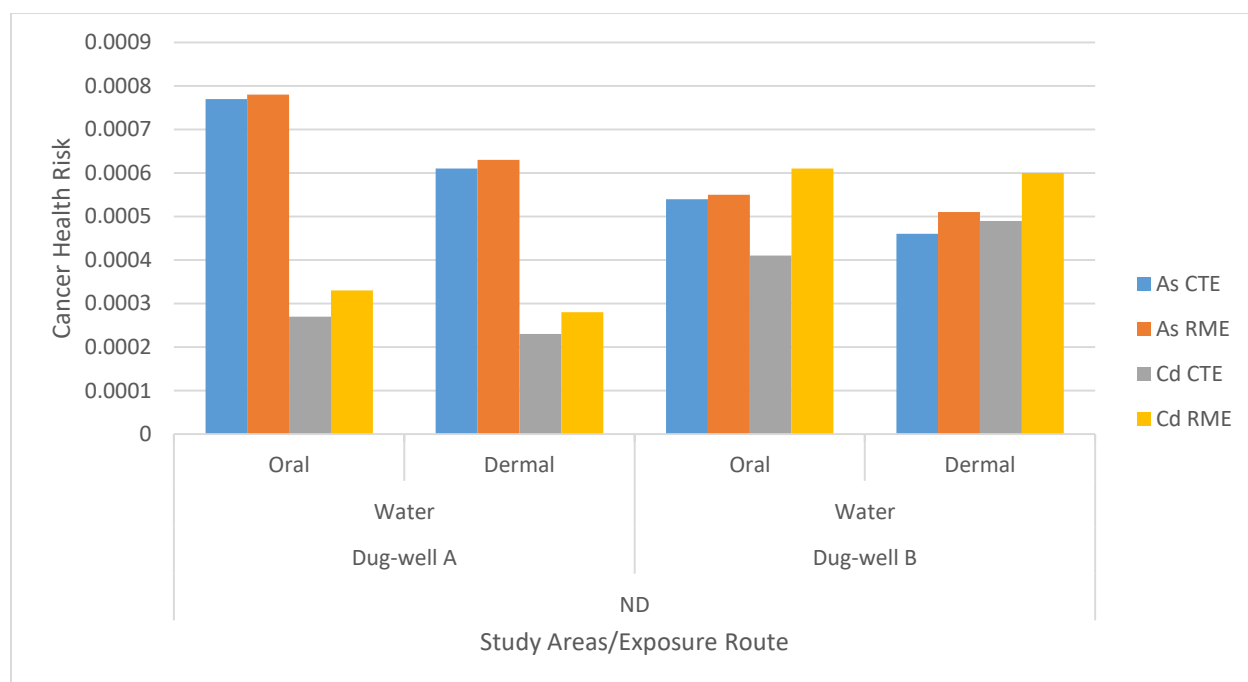


Fig. 4.14: Cancer health risk faced by resident adults in Nabdam District exposed to As and Cd in sediment and water samples.

The results obtained from the cancer health risk assessment for resident adults in the Nangodi community exposed to As and Cd in their drinking water were within the USEPA acceptable cancer risk value of 1×10^{-4} to 1×10^{-6} , i.e., 1 case of cancer per every 10,000 to 1,000,000 people. For instance, resident who drink water from Dug-out Well A, the cancer health risk via exposure to arsenic is 7.7×10^{-4} (CTE) and 7.8×10^{-4} (RME). This means that, approximately 8 adults out of 10,000 people are likely to contract cancer related diseases associated with exposure to arsenic.

However, in Tinga mining community in the Bole – Bamboi District (Fig. 4.15 below), the cancer health risk faced by resident adults were found to be higher than what resident adults in the Nangodi community face.

This may be due to the fact, the only stream where the gold mining activities takes place is far from town. For example, the cancer risk faced by resident adult at the edge of the stream before washing of gold ore via oral contact with As is 7.9×10^{-3} (RME parameters) and 7.8×10^{-3} (CTE parameters); which means approximately 8 out of 1000 resident adults in Tinga are likely to suffer from cancer related diseases.

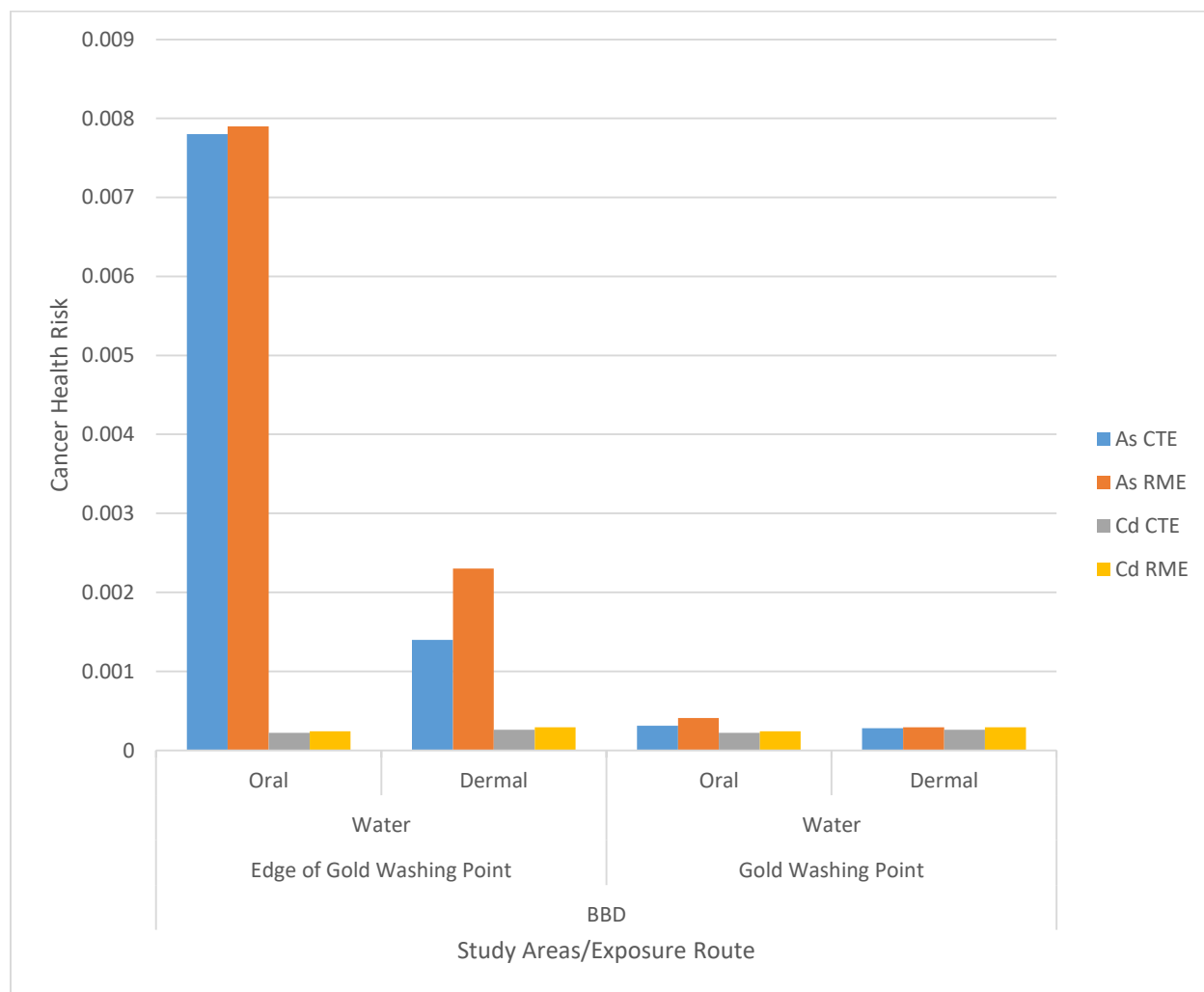


Fig. 4.15: Cancer health risk faced by resident adults in Tinga in the Bole - Bamboi District exposed to As and Cd in sediment and water samples.

Similarly, the cancer health risk via eating cooked cassava in mining communities in the study areas are summarised in Table 4.27 below.

Table 4.27: Cancer health risk results from ingestion of arsenic in cooked cassava grown in the study areas

Sampling point	Cancer health risk							
	Arsenic				Cadmium			
	Adults		Children		Adults		Children	
	CTE	RME	CTE	RME	CTE	RME	CTE	RME
Tamso	0.083	0.98	0.063	0.092	2.3×10^{-5}	6.2×10^{-4}	3.9×10^{-6}	2.8×10^{-4}
Tarkwa	0.0012	0.0099	0.076	0.0019	7.6×10^{-5}	3.1×10^{-4}	3.3×10^{-4}	3.9×10^{-4}
Prestea	0.12	0.010	0.080	0.00020	6.8×10^{-4}	5.4×10^{-4}	8.1×10^{-4}	8.8×10^{-4}
Bogoso	0.0012	0.45	0.23	0.063	5.6×10^{-4}	4.6×10^{-4}	5.8×10^{-4}	6.8×10^{-4}
UCC Research Farm	5.4×10^{-6}	6.5×10^{-6}	3.4×10^{-6}	7.8×10^{-6}	2.3×10^{-4}	2.8×10^{-4}	1.8×10^{-4}	2.5×10^{-4}
Tinga	3.8×10^{-4}	4.2×10^{-4}	0.00025	0.00029	2.7×10^{-4}	1.7×10^{-4}	3.3×10^{-4}	1.8×10^{-4}
Nangodi	0.00045	0.00062	0.00032	0.00036	3.8×10^{-4}	3.4×10^{-4}	1.5×10^{-4}	2.6×10^{-4}

From Table 4.27 above, the estimated lifetime cancer health risk for resident adults in Tamso who eat cassava grown in that area by both CTE and RME parameters are 8.3×10^{-2} and 9.8×10^{-2} respectively. This means that, 8 and 10 out of every 100 resident adults in that area are likely to suffer from cancer related cases associated with ingestion of arsenic.

4.6.3 Summary of non-carcinogenic health risk faced by all the receptors in the study areas.

In this study, a non – cancer health risk from exposure to arsenic, cadmium, mercury and lead via either oral contact and dermal contact to the aforementioned chemicals in water

bodies/sediments or oral ingestion of above named chemicals uptake by cassava grown in the study areas are expressed in terms of hazard indices. A hazard index indicates whether the estimated exposures for individual present a potentially significant non – cancer health risk based on comparison to a USEPA recommended reference dose (RfD).

For instance, the hazard index greater than 1.0, then there is high probability that the adverse health effects associated with exposure to such chemical may occur (USEPA, 1989a, 1997, 2001a). For example, the hazard index for resident adults in Dumasi exposed to arsenic in River Aprepre is 9.8 (CTE) and 7.8 (RME). The results show that the probability of resident adult in Dumasi experiencing non-cancerous diseases associated with arsenic intoxication such as cardiovascular diseases is very high (Fig. 4.16).

It can be seen that in most cases where the hazard indices are greater than 1, resident adults, galamsey operators and children in the areas are prone to headache, dizziness, gastrointestinal, skin hyper pigmentation and kidney failures. These are diseases associated with exposure to arsenic, cadmium, lead and mercury.

It can be inferred from appendix 32 that the hazard indices recorded in the Cape Coast Metropolis by all the receptors were lower than those recorded in the Tarkwa Nsuaem Municipality and Prestea Huni Valley District. Additionally, the hazard indices for Cape Coast were in line with the acceptable USEPA hazard index value of 1.0.

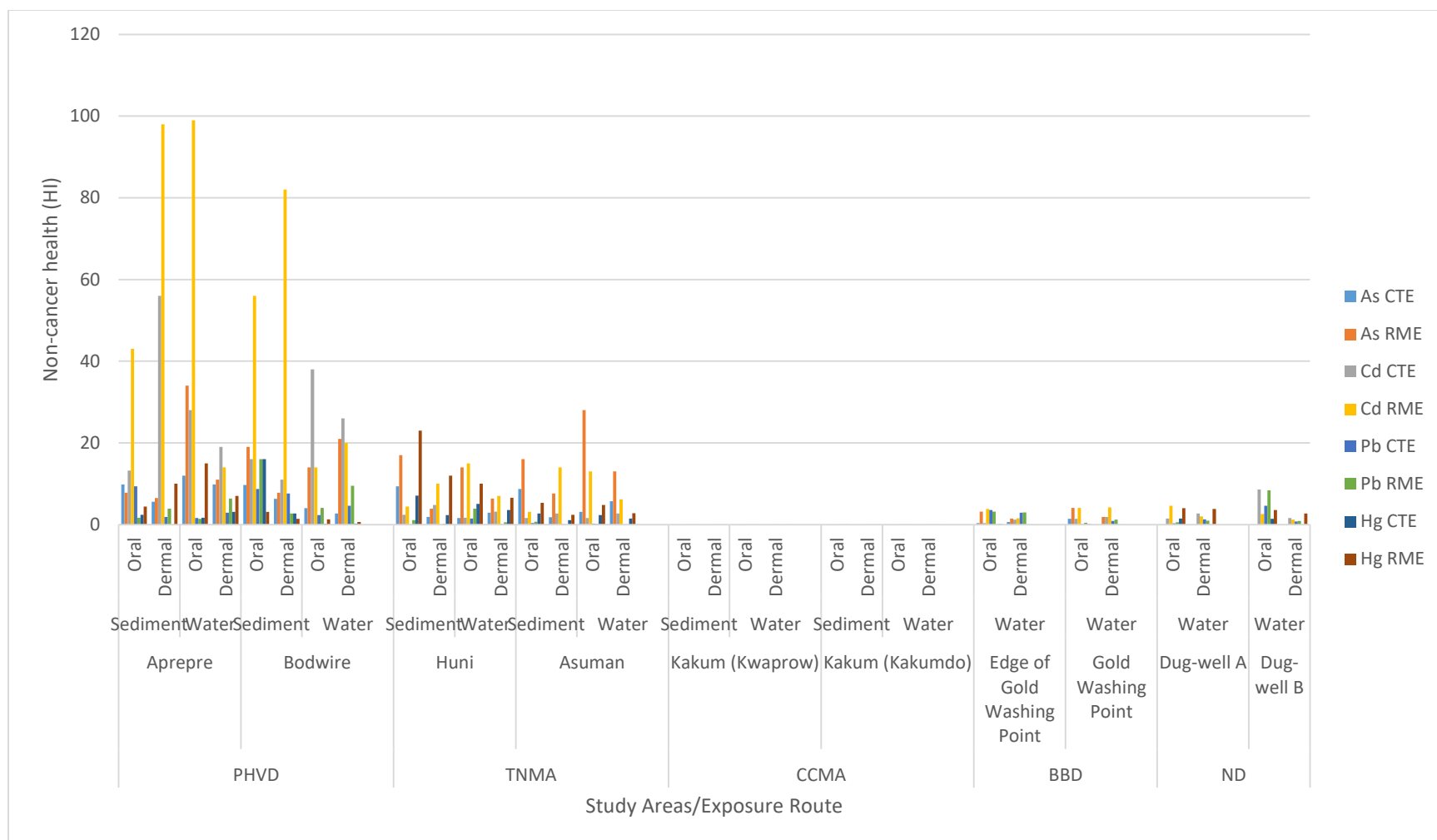


Fig. 4.16: A graph comparing non-cancer human health risk across the study areas.

4.7 Association between exposure to arsenic, mercury and lead in water, human blood and incidence of cardiovascular diseases among residents of residents in the study areas

According to Jennrich (2013), cardiovascular disease (CVD) is listed among the leading cause of death of millions of people all over the world. Cardiovascular disease refers to pathological changes within the heart, blood vessels, and blood flow. It occurs when the endothelium cells (which is a boundary layer between the blood stream and vascular wall; and has important functions in vasodilatation, modulation of inflammatory processes, and haemostatis) of the heart is damaged. The Framingham Heart Study identified several major risk factors for cardiovascular diseases. The identified risk factors are as follows; high serum cholesterol, cigarette smoking, hypertension, diabetes and low HDL levels (D'Agostino et al., 2008).

Other studies conducted in different countries have been able to link cardiovascular diseases (CVD) with exposure to environmental contaminants such as arsenic, mercury and lead in water samples as well as human human samples such as blood and urine (Weinhold, 2004; CERCLA, 2012; ATSDR, 2012; Balakumar and Kaur, 2009; Mumford et al., 2007; Navas-Acien et al., 2005; Chen et al., 2011; Wade et al., 2015). However, in Ghana no studies of this nature have been conducted to link cardiovascular diseases with exposure environmental contaminants.

From the summarised results of the hazard index via exposure to arsenic, mercury and lead in drinking water by resident adults in TNMA and PHVD (Fig. 4.20) as well as the elevated levels of arsenic, mercury and lead in whole blood and serum (Tables 4.23 and 4.24) ; also judging from the fact that exposure to arsenic, lead and mercury are likely to cause non-cancer diseases such as

CVDs, this section of the study investigated the association between exposure to arsenic, lead and mercury in drinking water and the presence of cardiovascular diseases among residents of mining communities in Ghana.

The data was collected from participants (who voluntarily gave their blood) through a personal questionnaire, which included questions on demographic characteristics such as age, gender, income, education level, and occupation. Other data collected include body mass index, smoking habit, family history of cardiovascular disease, and personal medical history, particularly whether they have been diagnose of any cardiovascular disease. In this study, participants who are either smoking current or are former smokers were excluded from taking part in this study.

The socio-demographic data obtained prior to the collection of blood samples were linked to the concentrations of arsenic, mercury and lead in their whole blood or serum were linked to the levels of arsenic, mercury and lead in drinking water as well as their medical records. This was done to help in the assessment of the association between exposure to arsenic, mercury and lead incidence of cardiovascular diseases. In addition to above, data obtained via the address of participants and the duration of residence in the study area, source of drinking water and signs of arsenicosis (a disease due to arsenic exposure) helped in the assessment of association between exposure to arsenic and incidence of cardiovascular diseases.

Socio-demographic characteristics of respondents (including participants who have been diagnosed with CVDs referred to as cases; and also shows symptoms of either arsenicosis or any other disease associated with Hg or Pb that can be easily diagnosed) and controls (refers to participants who have not been diagnosed with CVDs) are shown in Table 4.28.

Table 4.28: Demographic characteristics of respondents with CVD cases as well as the controls

Characteristics	Cases (N=240); N (%)	Control (N =170); N (%)
<i>Gender</i>		
Male	170 (70.8)	90 (52.9)
Female	70 (29.2)	80 (47.1)
<i>Age (years)</i>		
20 – 29	62 (25.8)	48 (28.2)
30 – 39	70 (29.2)	68 (40.0)
40 – 49	70 (29.2)	37 (21.8)
≥ 50	33 (13.8)	22 (10.1)
<i>Body mass index (kg/m²)</i>	49.2 (20.5)	19.9 (11.7)
<i>Mean blood levels:</i>		
As	21.3 (16.7)	8.94 (18.1)
Hg	41.0 (16.7)	7.65 (24.2)
Pb	14.1 (16.6)	3.35 (21.2)
<i>Mean water levels:</i>		
1. Arsenic (As)		
≤10 µg/L	50 (20.8)	35 (20.6)
10 – 300 µg/L	160 (66.7)	123 (72.4)
≥301 µg/L	30 (12.5)	12 (7.0)
2. Mercury (Hg)		
≤10 µg/L	20 (8.3)	19 (11.2)
10 – 300 µg/L	189 (78.8)	131 (77.0)
≥301 µg/L	31 (12.9)	20 (11.8)
3. Lead (Pb)		
≤10 µg/L	40 (16.7)	36 (21.2)
10 – 300 µg/L	170 (70.8)	104 (61.2)
≥301 µg/L	30 (12.5)	30 (17.6)
<i>Arsenicosis or any other disease:</i>		
Yes	178 (74.2)	157 (92.4)
No	62 (25.8)	13 (7.6)
<i>History of cardiovascular disease in your family</i>		
Yes	28 (11.7)	132 (77.7)
No	212 (88.3)	38 (22.3)
<i>Education</i>		
No formal education	56 (23.3)	34 (20.0)
Primary School	94 (39.2)	78 (45.9)
Secondary	80 (33.3)	45 (26.5)
Tertiary	10 (4.2)	13 (7.6)
<i>Income (USD per day):</i>		
≤ \$1	76 (31.7)	86 (50.6)
≥\$1	164 (68.3)	84 (49.4)
<i>Mean years of staying in the study areas</i>		
5 – 20 years	24 (10)	18 (10.6)
21 – 36 years	186 (77.5)	132 (77.6)
≥ 36 years	30 (12.5)	20 (11.8)

From Table 4.28 above, a total of 410 respondents were recruited to be part of this study. Out of this number, 240 of them were classified as cases for cardiovascular diseases while 170 did not meet the operating definition for cardiovascular diseases and were considered as controls. The operating case definition for cardiovascular diseases adopted in this study is as follows;

- Acute Myocardial Infarction (MI) which was defined as having 2 of 3 findings: (1) Recent chest pain consistent with angina for approximately 30 minutes or more; (2) ECG changes consistent with MI: ST segment elevation greater than or equal to 1.0 mV in 2 contiguous leads (2 leads following each other); (3) Positive enzymes: Elevation of CK/MB or troponin consistent with MI.
- Cardiomyopathy. Defined ejection fraction less than or equal to 35% without valvular heart disease.
- Chest pain suggestive of angina with any one of the following: changes on ECG consistent with previous MI (Q-waves); documented enzyme changes consistent with previous MI; changes in serial ECG tracings (for example T-wave changes); ECG changes on treadmill stress (exercise) testing consistent with injury or ischemia.

It was observed that out of the 240 respondents with cardiovascular cases, 178 of them showed signs of arsenicosis, a disease associated with exposure to arsenic; while 157 of the control respondents did show signs of arsenicosis disease but did not meet the case definition for cardiovascular disease and were rather showing upper respiratory symptoms (colds, flu, and other allergies), injuries, gastrointestinal symptoms, and regular examinations.

It is interesting to note that, 77.7% of respondents within the control group reported having a family history of diabetes, hypertension or heart disease (Table 4.28) as against 11.7% of respondents with cardiovascular cases. Similarly, 66.7% of the respondents had arsenic levels in their drinking water ranging between 10 µg/L and 300 µg/L; while the mean arsenic blood of 21.3 µg/L was recorded among respondents with cardiovascular cases as against 8.94 µg/L arsenic levels in the control group. A strong positive correlation was found among subjects with water arsenic measures above 10 µg/L ($n = 325$, $r = 0.65$, $p < 0.05$ two tailed). Another significant positive correlation was found between the levels of arsenic in their blood samples and arsenic in their drinking water.

The results of logistic regression model with drinking water arsenic concentration as a continuous and categorical exposures is presented in Table 4.29.

Table 4.29: Adjusted odds ratios for cardiovascular disease, water and concentration of heavy metals in blood of respondents

	Adjusted OR	95% CI	p-value
<i>Continuous exposure</i>			
Water arsenic (>10µg/L)	2.1	1.0 – 4.3	0.021
Blood arsenic (2 µg/L)	1.6	0.78 – 2.4	0.09
<i>Categorical exposures</i>			
Water arsenic <10 µg/L	1.2	1.0 – 4.3	0.38
10 – 300	3.5	1.9 – 3.3	0.36
>300	2.5	1.1 – 6.33	0.21

The adjustment in the odds ratio were made to account for confounding factors such as BMI, age, sex, history of arsenicous disease and family history of cardiovascular diseases among the respondents.

With regard to continuous water arsenic exposure of greater than 10 µg/L, the adjusted odds ratio (AORs) (i.e., AOR = 2.1; 1.0 – 4.3 95% CI) for water arsenic exposures. In the categorical analysis, water arsenic concentration between 10 µg/L and 300 µg/L had an AOR of 3.5, 95% CI 1.9 – 3.3.

This study measured relatively high levels of arsenic exposures in water samples and further observed a strong association between exposure to elevated levels of water arsenic and cardiovascular disease. The odds of having cardiovascular disease among those with drinking water arsenic concentration ≥ 300 µg/L was approximately 3 times higher compared to those with drinking water with ≤ 10 µg/L arsenic.

The major limitation of this is the lack of sufficient data to evaluate departures from non-linearity and threshold effects ≤ 10 µg/L, under the assumption of a linear trend, a 10 µg/L increase in water arsenic was associated with a 12% increase in the odds of cardiovascular disease. The other limitation is the lack of information on previous arsenic water exposures by the respondents. It is generally accepted that, this limitation may introduce exposure misclassification among the respondents; the overall impact of this bias was mitigated through the sampling technique adopted in this section of the study. The criterion for selecting respondents for this study was that, they must be permanent resident in the study area for the past five years prior to the commencement of this study.

In spite of the above shortcomings, the results obtained are consistent with studies conducted in other countries which provide increasing evidence of associations between arsenic and cardiovascular disease at low to high levels of chronic arsenic exposure (Mumford et al., 2007; James et al., 2015; Moon et al., 2013; Mordukhovich et al., 2009; Chen et al., 2007; Chen et al., 2013).

CHAPTER FIVE

CONCLUSION AND RECOMMENDATION

5.0 Conclusion

This study comprehensively assessed the human health risk associated with oral and dermal contact to As, Cd, Pb and Hg in water, sediment and food crops grown in selected mining communities. The receptors used in this study are resident adults, children, adult ASGM workers as well as adult workers of mining companies in the study areas. The receptors are assumed to be exposed to As, Cd, Pb and Hg in environmental media (such as water and sediment) and food crops such as cassava grown in the study areas.

From the results of the study, the following conclusion can be drawn:

1. The concentration of As, Cd, Hg and Pb in water samples from Tarkwa Nsuaem Municipal Assembly/Prestea Huni Valley District (TNMA/PHVD) with long history of mining in Ghana were found to be higher than those obtained in emerging mining areas such as Tinga in Bole-Bamboi District as well as Nangodi in Nabdam District.
2. From the principal component analysis (PCA) and factor analysis (FA), it was realised that the source of As, Cd, Pb and Hg in the water samples could be attributed to mining of the arsenopyrite ores.
3. Elevated levels of As, Cd, Hg and Pb were found in found in food crops such as cassava grown in mining communities than those obtained from non-mining communities.

4. Exposure to As, Cd, Hg and Pb in the various environmental media such as water, sediment and food crops grown in the study areas are responsible for the elevated levels of the aforementioned toxic chemicals in whole blood or serum of ASGM mine workers, workers of large-scale mining companies and resident adults/children living in mining communities than those living in non-mining communities.
5. The results of the study revealed that resident adults, children as well as artisanal mine workers in the study area are at risk of developing cancerous and other non-cancerous diseases due to exposure to the aforementioned metals and metalloids. This is due to the fact that the cancer health risk result in most cases exceeded the acceptable figure of 1×10^{-6} to 1×10^{-4} , i.e., 1 case of cancer in every 1,000,000 people to 1 case of cancer per every 10,000 people.
6. The cancer and non-cancer health risk results for the various study areas are as follows; TNMA/PHVD > Nangodi > Tinga > CCMA.
7. The results of case-control epidemiological study provide evidence of strong association between exposure to moderately high arsenic levels in drinking water and incidence of cardiovascular diseases among residents of selected mining communities in Ghana.

5.1 Recommendation

From the findings of this study the following recommendations are made:

1. Air quality assessment should be taken seriously to ascertain the risk pose by inhalation of As, Cd, Hg and Pb particulate matter in the atmosphere of the study areas.

2. A more detailed research programme should be undertaken to explore which of the following methods for removal of heavy metals and metalloids would be cost-effective, i.e., bioremediation, phytoremediation or the use of nanoparticles.
3. Since, there is lack of consistent toxicological data such as ingestion rate for water, chemical-specific absorption fraction, etc hinders effective assessment of cancer and non-cancer health risk studies in Ghana. It is recommended that, the Ghana Standards Authority, Food and Drugs Authority in collaboration with Ghana Health Service and Statistical Service be resourced to undertake a study of this nature to provide such data needed for risk assessment.

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Appendices

Appendix 1 (Attached Publication 1)

Human health risk assessment of artisanal miners exposed to toxic chemicals in water and sediments in the Prestea Huni Valley District of Ghana.

Samuel Obiri^{1,2,*}, Philip O. Yeboah^{1,†}, Shiloh Osae^{1,†}, Sam Adu-kumi^{3,†}, Samuel J. Cobbina^{4,†}, Frederick A. Armah^{5,†}, Benjamin Ason^{6,†}, Edward Antwi^{7,†} and Reginald Quansah^{8,†}

Appendix 2 (Attached Publication)

Assessing the environmental and socio-economic impacts of gold artisanal mining on the livelihoods of communities in the Tarkwa Nsuaem Municipality in Ghana.

Samuel Obiri ^{1,2,*}, Precious A. D. Mattah ^{3,†}, Memuna M. Mattah ^{4,†}, Frederick A. Armah ^{5,†}, Shiloh Osae ^{1,†}, Sam Adu-kumi ^{6,†} and Philip O. Yeboah ^{1,†}

Appendix 3 (Attached Publication)

Levels of arsenic, mercury, cadmium, copper, lead, zinc and manganese in Serum and Whole Blood of Resident Adults from Mining and Non - mining Communities in Ghana

Samuel Obiri^{1,2*}, Philip O. Yeboah¹, Shiloh Osae¹ and Sam Adu – Kumi³.

Appendix 4

(List of related publications)

1. Armah, F. A., Luginaah, I. and **Obiri, S.** (2012): Assessing environmental exposure and health impacts of gold mining in Ghana. *Toxicol. Environ. Chem.* dx.doi.org/10.108
2. **Obiri, S.**, Cobbina, S. J., Armah, F. A. and Luginaah, I. (2013): Assessment of cancer and non-cancer health risk from exposure to PAHs in street dust in the Tamale Metropolis, Ghana. *J. Environ. Sci. Health Part A.* 48: 408 – 416.
3. Ofosu-Asiedu, L., Cobbina, S. J. and **Obiri, S.** (2013): Non-cancer human health risk assessment from exposure to cadmium, copper, lead and mercury in surface water and ground water in Konongo-Oduamse Municipality, Ghana. *J. Environ. Ecotox.* 5(4):106 – 112.
4. Cobbina, S. J., Nkuah, D., Tom-Dery, D. and **Obiri, S.** (2013): Non-cancer risk assessment from exposure to mercury (Hg), cadmium (Cd), arsenic (As), copper (Cu) and lead (Pb) in boreholes and surface water in Tinga in the Bole-Bamboi District, Ghana. *J. Toxicol. Environ. Health Sci.* 5 (2): 22 – 36.
5. Quansah, R., Armah, F. A., Essumang, D. K., Luginaah, I., Clarke, E., Marfoh, K., Cobbina, S. J., Nketiah-Amponsah, E., Namuujju, P. B., **Obiri, S.** and Dzodzomenyo, M. (2015): Association of arsenic with adverse pregnancy outcomes/infant mortality: A systemic review and meta-analysis. *Environ. Health Persp.* 123: 412 – 421.
6. Rajae, M., **Obiri, S.**, Green, A., Long, R., Cobbina, S. J., Nartey, V., Buck, D., Antwi, E. and Basu, N. (2015): Integrated Assessment of artisanal and small-scale gold mining in Ghana – Part 2: Natural Sciences Review. *Int. J. Environ. Res. Public Health.* 12: 8971 – 9011.

Appendix 5

Cancer health risk results from exposure to arsenic by resident adults, mine workers, galamsey operators and children in the Prestea Huni Valley District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Galamsey operators/Mine workers		Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	3.2×10^{-5}	6.2×10^{-4}	8.4×10^{-6}	6.1×10^{-4}	2.3×10^{-5}	3.7×10^{-4}
		Dermal	1.9×10^{-6}	7.6×10^{-4}	2.1×10^{-6}	2.1×10^{-4}	7.1×10^{-6}	2.9×10^{-4}
	Water	Oral	4.3×10^{-3}	1.3×10^{-2}	2.4×10^{-3}	7.1×10^{-2}	2.7×10^{-3}	1.8×10^{-2}
Bodwire	Sediment	Oral	4.3×10^{-4}	1.3×10^{-2}	7.6×10^{-6}	5.2×10^{-4}	2.0×10^{-5}	3.1×10^{-4}
		Dermal	8.0×10^{-4}	6.0×10^{-3}	2.1×10^{-6}	1.8×10^{-4}	6.0×10^{-6}	2.4×10^{-4}
	Water	Oral	1.6×10^{-3}	4.9×10^{-2}	2.2×10^{-3}	6.6×10^{-2}	1.1×10^{-3}	1.7×10^{-2}
Ankobra	Sediment	Oral	1.3×10^{-5}	9.3×10^{-4}	2.8×10^{-6}	6.8×10^{-2}	2.7×10^{-3}	1.8×10^{-2}
		Dermal	3.9×10^{-6}	3.3×10^{-4}	8.4×10^{-7}	3.1×10^{-2}	3.3×10^{-3}	1.6×10^{-2}
	Water	Oral	4.7×10^{-4}	6.7×10^{-4}	7.7×10^{-4}	6.5×10^{-3}	7.2×10^{-3}	2.6×10^{-3}
Twilagya	Sediment	Oral	7.6×10^{-4}	2.7×10^{-4}	9.6×10^{-3}	9.8×10^{-3}	2.1×10^{-5}	7.6×10^{-5}
		Dermal	5.2×10^{-4}	4.0×10^{-4}	7.6×10^{-5}	7.6×10^{-4}	1.4×10^{-5}	1.1×10^{-5}
	Water	Ingestion	1.4×10^{-4}	2.3×10^{-3}	1.9×10^{-3}	1.3×10^{-2}	6.1×10^{-3}	3.8×10^{-3}
Anfoe	Sediment	Oral	3.6×10^{-4}	2.4×10^{-4}	1.8×10^{-3}	1.8×10^{-2}	8.9×10^{-3}	3.2×10^{-2}
		Dermal	1.0×10^{-4}	8.4×10^{-3}	1.6×10^{-3}	1.5×10^{-2}	6.0×10^{-3}	4.7×10^{-2}
	Water	Oral	6.0×10^{-3}	5.3×10^{-3}	8.4×10^{-2}	3.3×10^{-2}	1.3×10^{-2}	4.8×10^{-2}
Worawora	Sediment	Oral	3.2×10^{-6}	2.5×10^{-4}	9.0×10^{-1}	7.8×10^{-2}	5.4×10^{-3}	4.1×10^{-3}
		Dermal	9.8×10^{-4}	8.1×10^{-4}	7.2×10^{-2}	5.6×10^{-2}	2.5×10^{-3}	1.9×10^{-3}
	Water	Oral	1.0×10^{-2}	3.1×10^{-2}	7.8×10^{-1}	6.5×10^{-1}	9.3×10^{-2}	2.8×10^{-1}
Mansi	Sediment	Oral	4.3×10^{-6}	3.1×10^{-4}	2.8×10^{-2}	7.5×10^{-2}	2.7×10^{-6}	8.1×10^{-5}
		Dermal	2.3×10^{-3}	1.1×10^{-4}	5.4×10^{-3}	8.4×10^{-3}	5.0×10^{-6}	3.9×10^{-5}
	Water	Oral	2.3×10^{-3}	6.9×10^{-2}	4.5×10^{-2}	8.3×10^{-2}	1.4×10^{-3}	4.1×10^{-3}
Borehole at Dumase	Water	Dermal	4.2×10^{-3}	3.2×10^{-2}	6.2×10^{-2}	7.3×10^{-2}	2.5×10^{-3}	1.9×10^{-3}
		Oral	7.1×10^{-2}	4.3×10^{-2}	4.5×10^{-2}	7.4×10^{-2}	1.1×10^{-4}	3.4×10^{-4}
		Dermal	1.3×10^{-3}	9.9×10^{-2}	5.6×10^{-3}	5.9×10^{-3}	2.1×10^{-4}	1.6×10^{-4}

Appendix 6

Cancer health risk results from exposure to cadmium by resident adults, mine workers, galamsey operators and children in the Prestea Huni Valley District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Galamsey operators/Mine workers		Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	3.8×10^{-3}	5.9×10^{-2}	2.3×10^{-5}	3.7×10^{-4}	6.2×10^{-4}	9.3×10^{-4}
		Dermal	1.3×10^{-3}	4.7×10^{-2}	7.1×10^{-6}	2.9×10^{-4}	7.6×10^{-4}	3.3×10^{-4}
	Water	Oral	1.0×10^{-2}	6.8×10^{-2}	2.7×10^{-3}	1.8×10^{-2}	1.3×10^{-2}	6.7×10^{-2}
Bodwire	Sediment	Oral	2.2×10^{-3}	1.6×10^{-2}	2.0×10^{-5}	3.1×10^{-4}	1.3×10^{-2}	2.7×10^{-4}
		Dermal	6.7×10^{-3}	5.6×10^{-5}	6.0×10^{-6}	2.4×10^{-4}	6.0×10^{-3}	4.0×10^{-4}
	Water	Oral	7.8×10^{-3}	2.3×10^{-2}	1.1×10^{-3}	1.7×10^{-2}	4.9×10^{-2}	2.3×10^{-3}
Ankobra	Sediment	Oral	7.6×10^{-5}	5.3×10^{-4}	2.7×10^{-3}	1.8×10^{-2}	9.3×10^{-4}	2.4×10^{-4}
		Dermal	5.3×10^{-4}	1.8×10^{-4}	3.3×10^{-3}	1.6×10^{-2}	3.3×10^{-4}	8.4×10^{-3}
	Water	Oral	2.6×10^{-3}	7.7×10^{-2}	7.2×10^{-3}	2.6×10^{-3}	6.7×10^{-4}	5.3×10^{-3}
Twiahya	Sediment	Oral	3.2×10^{-6}	2.5×10^{-2}	2.1×10^{-5}	7.6×10^{-5}	2.7×10^{-4}	2.4×10^{-4}
		Dermal	9.8×10^{-7}	8.1×10^{-3}	1.4×10^{-5}	1.1×10^{-5}	4.0×10^{-4}	3.2×10^{-5}
	Water	Ingestion	1.0×10^{-1}	3.1×10^{-2}	6.1×10^{-3}	3.8×10^{-3}	2.3×10^{-3}	7.7×10^{-2}
Anfoe	Sediment	Oral	2.2×10^{-6}	1.6×10^{-4}	8.9×10^{-3}	3.2×10^{-2}	2.4×10^{-4}	2.5×10^{-4}
		Dermal	6.7×10^{-5}	5.6×10^{-5}	6.0×10^{-3}	4.7×10^{-2}	8.4×10^{-3}	8.1×10^{-4}
	Water	Oral	1.6×10^{-3}	3.1×10^{-2}	1.3×10^{-2}	4.8×10^{-2}	5.3×10^{-3}	3.1×10^{-2}
Worawora	Sediment	Oral	3.8×10^{-3}	5.9×10^{-2}	5.4×10^{-3}	4.1×10^{-3}	2.5×10^{-4}	4.3×10^{-2}
		Dermal	1.2×10^{-3}	4.7×10^{-2}	2.5×10^{-3}	1.9×10^{-3}	8.1×10^{-4}	9.9×10^{-2}
	Water	Oral	6.2×10^{-4}	9.6×10^{-3}	9.3×10^{-2}	2.8×10^{-1}	3.1×10^{-2}	4.3×10^{-4}
Mansi	Sediment	Oral	9.1×10^{-2}	6.1×10^{-3}	2.7×10^{-6}	8.1×10^{-5}	3.1×10^{-4}	6.7×10^{-5}
		Dermal	1.3×10^{-3}	5.6×10^{-3}	5.0×10^{-6}	3.9×10^{-5}	1.1×10^{-4}	4.6×10^{-5}
	Water	Oral	8.4×10^{-2}	5.6×10^{-3}	1.4×10^{-3}	4.1×10^{-3}	6.9×10^{-2}	7.1×10^{-2}
Borehole at Dumase	Sediment	Oral	8.5×10^{-3}	5.7×10^{-3}	2.5×10^{-3}	1.9×10^{-3}	3.2×10^{-2}	3.7×10^{-3}
		Dermal	9.6×10^{-2}	6.6×10^{-2}	1.1×10^{-4}	3.4×10^{-4}	4.3×10^{-2}	7.2×10^{-3}
	Water	Oral	4.5×10^{-1}	8.3×10^{-2}	2.1×10^{-4}	1.6×10^{-4}	9.9×10^{-2}	4.9×10^{-3}

Appendix 7

Cancer health risk results from exposure to arsenic by resident adults, mine workers, galamsey operators and children in the Tarkwa Nsuaem Municipality

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult workers/ASGM operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	3.8×10^{-3}	5.9×10^{-2}	2.3×10^{-5}	3.7×10^{-4}	6.2×10^{-4}	9.3×10^{-4}
		Dermal	1.3×10^{-3}	4.7×10^{-2}	7.1×10^{-6}	2.9×10^{-4}	7.6×10^{-4}	3.3×10^{-4}
	Water	Oral	2.0×10^{-2}	6.8×10^{-2}	2.7×10^{-3}	1.8×10^{-2}	1.3×10^{-2}	6.7×10^{-2}
		Dermal	2.3×10^{-3}	6.2×10^{-2}	3.4×10^{-3}	1.7×10^{-2}	6.0×10^{-3}	5.1×10^{-2}
Asuman	Sediment	Oral	2.2×10^{-3}	1.6×10^{-2}	2.0×10^{-5}	3.1×10^{-4}	1.3×10^{-2}	2.7×10^{-4}
		Dermal	6.7×10^{-3}	5.6×10^{-5}	6.0×10^{-6}	2.4×10^{-4}	6.0×10^{-3}	4.0×10^{-4}
	Water	Oral	7.8×10^{-3}	2.3×10^{-2}	1.1×10^{-3}	1.7×10^{-2}	4.9×10^{-2}	2.3×10^{-3}
		Dermal	1.4×10^{-3}	1.1×10^{-2}	1.3×10^{-3}	1.6×10^{-2}	2.3×10^{-2}	1.4×10^{-3}
Abodwesh	Sediment	Oral	7.6×10^{-5}	5.3×10^{-4}	2.7×10^{-3}	1.8×10^{-2}	9.3×10^{-4}	2.4×10^{-4}
		Dermal	5.3×10^{-4}	1.8×10^{-4}	3.3×10^{-3}	1.6×10^{-2}	3.3×10^{-4}	8.4×10^{-3}
	Water	Oral	2.6×10^{-3}	7.7×10^{-2}	7.2×10^{-3}	2.6×10^{-3}	6.7×10^{-4}	5.3×10^{-3}
		Dermal	4.7×10^{-3}	3.5×10^{-2}	4.9×10^{-3}	3.8×10^{-3}	5.1×10^{-4}	1.8×10^{-3}
Ateberegbe	Sediment	Oral	3.2×10^{-6}	2.5×10^{-2}	2.1×10^{-5}	7.6×10^{-5}	2.7×10^{-4}	2.4×10^{-4}
		Dermal	9.8×10^{-7}	8.1×10^{-3}	1.4×10^{-5}	1.1×10^{-5}	4.0×10^{-4}	3.2×10^{-5}
	Water	Oral	2.0×10^{-1}	3.1×10^{-2}	6.1×10^{-3}	3.8×10^{-3}	2.3×10^{-3}	7.7×10^{-2}
		Dermal	1.9×10^{-1}	1.4×10^{-2}	7.2×10^{-3}	3.8×10^{-3}	1.4×10^{-3}	3.5×10^{-2}
Achofe	Sediment	Oral	2.2×10^{-6}	1.6×10^{-4}	8.9×10^{-3}	3.2×10^{-2}	2.4×10^{-4}	2.5×10^{-4}
		Dermal	6.7×10^{-5}	5.6×10^{-5}	6.0×10^{-3}	4.7×10^{-2}	8.4×10^{-3}	8.1×10^{-4}
	Water	Oral	1.6×10^{-3}	3.1×10^{-2}	1.3×10^{-2}	4.8×10^{-2}	5.3×10^{-3}	3.1×10^{-2}
		Dermal	3.0×10^{-3}	1.1×10^{-2}	3.0×10^{-2}	6.9×10^{-2}	1.8×10^{-3}	1.4×10^{-2}
Subri	Sediment	Oral	3.0×10^{-6}	6.9×10^{-2}	6.6×10^{-4}	5.5×10^{-6}	2.4×10^{-4}	3.1×10^{-4}
		Dermal	2.3×10^{-3}	3.2×10^{-2}	2.9×10^{-5}	2.0×10^{-5}	3.2×10^{-5}	1.1×10^{-4}
	Water	Oral	6.7×10^{-5}	2.4×10^{-4}	8.6×10^{-4}	2.5×10^{-3}	7.7×10^{-2}	6.9×10^{-2}
		Dermal	4.6×10^{-5}	3.5×10^{-5}	1.6×10^{-4}	1.2×10^{-4}	3.5×10^{-2}	3.2×10^{-2}
Betinyere	Sediment	Oral	3.8×10^{-3}	5.9×10^{-2}	5.4×10^{-3}	4.1×10^{-3}	2.5×10^{-4}	4.3×10^{-2}
		Dermal	1.2×10^{-3}	4.7×10^{-2}	2.5×10^{-3}	1.9×10^{-3}	8.1×10^{-4}	9.9×10^{-2}
	Water	Oral	6.2×10^{-4}	9.6×10^{-3}	9.3×10^{-2}	2.8×10^{-1}	3.1×10^{-2}	4.3×10^{-4}
		Dermal	1.9×10^{-3}	7.6×10^{-3}	1.7×10^{-3}	1.3×10^{-2}	1.4×10^{-2}	3.1×10^{-5}
Ahomabruen	Sediment	Oral	9.1×10^{-2}	6.1×10^{-3}	2.7×10^{-6}	8.1×10^{-5}	3.1×10^{-4}	6.7×10^{-5}
		Dermal	1.3×10^{-3}	5.6×10^{-3}	5.0×10^{-6}	3.9×10^{-5}	1.1×10^{-4}	4.6×10^{-5}
	Water	Oral	8.4×10^{-2}	5.6×10^{-3}	1.4×10^{-3}	4.1×10^{-3}	6.9×10^{-2}	7.1×10^{-2}
		Dermal	8.5×10^{-3}	5.7×10^{-3}	2.5×10^{-3}	1.9×10^{-3}	3.2×10^{-2}	3.7×10^{-3}
Borehole – Abekoase	Water	Oral	9.6×10^{-2}	6.6×10^{-2}	1.1×10^{-4}	3.4×10^{-4}	4.3×10^{-2}	7.2×10^{-3}
		Dermal	4.5×10^{-1}	8.3×10^{-2}	2.1×10^{-4}	1.6×10^{-4}	9.9×10^{-2}	4.9×10^{-3}

Appendix 8

Cancer health risk results from exposure to cadmium by resident adults, mine workers, galamsey operators and children in the Tarkwa Nsuaem Municipality

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult Mine workers/ASGM operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	0.0043	0.11	0.0063	0.057	0.0017	0.0044
		Dermal	0.003	0.017	0.012	0.026	0.0012	0.0064
	Water	Oral	0.0015	0.039	0.0022	0.020	0.00059	0.015
		Dermal	0.001	0.0057	0.0040	0.0040	0.00004	0.0022
Asuman	Sediment	Oral	0.0036	0.067	0.0037	0.034	0.001	0.20
		Dermal	0.0018	0.0098	0.0068	0.0090	0.000069	0.38
	Water	Oral	0.0010	0.027	0.0015	0.013	4.0	1.0
		Dermal	0.00074	0.0039	0.0027	0.0062	2.7	1.5
Abodwesh	Sediment	Oral	0.0013	0.034	0.0019	0.019	5.8	1.3
		Dermal	0.00071	0.0050	0.0035	0.035	2.7	1.9
	Water	Oral	0.0018	0.045	0.0025	0.023	0.0068	0.0017
		Dermal	0.0012	0.066	0.0046	0.010	0.0046	0.0025
Ateberebe	Sediment	Oral	0.0015	0.039	0.0022	0.020	0.000059	0.015
		Dermal	0.0010	0.0057	0.0040	0.0040	0.000004	0.0022
	Water	Oral	0.0022	0.057	0.0032	0.029	0.0087	0.0022
		Dermal	0.0015	0.0084	0.0059	0.013	0.0059	0.033
Achofe	Sediment	Oral	0.0018	0.045	0.0019	0.019	0.0068	0.0017
		Dermal	0.0012	0.066	0.0035	0.035	0.0046	0.0025
	Water	Oral	0.0036	0.093	0.0052	0.046	0.014	0.036
		Dermal	0.0025	0.014	0.0095	0.021	0.0095	0.052
Subri	Sediment	Oral	0.0021	0.055	0.0031	0.027	0.0082	0.0021
		Dermal	0.0015	0.0080	0.0056	0.013	0.0056	0.0031
	Water	Oral	0.0016	0.040	0.0022	0.020	0.00060	0.00016
		Dermal	0.0011	0.0059	0.0041	0.0093	0.00041	0.000023
Betinyere	Sediment	Oral	0.0019	0.048	0.0026	0.024	0.0071	0.0018
		Dermal	0.0013	0.0069	0.0049	0.011	0.0049	0.0027
	Water	Oral	0.0017	0.043	0.0024	0.021	0.00064	0.00016
		Dermal	0.0011	0.0062	0.0044	0.0098	0.00044	0.00024
Ahomabru en	Sediment	Oral	0.0035	0.090	0.0050	0.045	0.014	0.0035
		Dermal	0.0024	0.013	0.0092	0.021	0.0092	0.0051
	Water	Oral	0.0033	0.084	0.0047	0.042	0.00013	0.0032
		Dermal	0.0022	0.012	0.0086	0.019	0.00086	0.0047
Borehole – Abekoase	Water	Oral	0.0054	0.14	0.0077	0.070	0.021	0.054
		Dermal	0.0037	0.020	0.014	0.032	0.014	0.0078

Appendix 9

Cancer health risk results from exposure to arsenic by resident adults, mine worker, galamsey operators and children in the Cape Coast Metropolis

Sampling point	Exposure media	Exposure route				
			Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow) Cape Coast	Sediment	Oral	3.8×10^{-6}	5.9×10^{-6}	6.2×10^{-4}	9.3×10^{-4}
		Dermal	1.3×10^{-6}	4.7×10^{-6}	7.6×10^{-4}	3.3×10^{-4}
	Water	Oral	3.0×10^{-6}	6.8×10^{-6}	1.3×10^{-4}	6.7×10^{-4}
		Dermal	2.3×10^{-6}	6.2×10^{-6}	6.0×10^{-4}	5.1×10^{-4}
Kakum (Kakumdo)	Sediment	Oral	2.2×10^{-6}	1.6×10^{-6}	1.3×10^{-4}	2.7×10^{-4}
		Dermal	6.7×10^{-7}	5.6×10^{-5}	6.0×10^{-4}	4.0×10^{-4}
	Water	Oral	7.8×10^{-6}	2.3×10^{-5}	4.9×10^{-5}	2.3×10^{-3}
		Dermal	1.4×10^{-6}	1.1×10^{-5}	2.3×10^{-5}	1.4×10^{-3}
Asuboni stream (Effutu)	Sediment	Oral	7.6×10^{-5}	5.3×10^{-4}	9.3×10^{-4}	2.4×10^{-4}
		Dermal	5.3×10^{-4}	1.8×10^{-4}	3.3×10^{-4}	8.4×10^{-5}
	Water	Oral	2.6×10^{-5}	7.7×10^{-6}	6.7×10^{-4}	5.3×10^{-5}
		Dermal	4.7×10^{-5}	3.5×10^{-6}	5.1×10^{-4}	1.8×10^{-5}
Tap water from Brimsu	Water	Oral	3.0×10^{-6}	3.1×10^{-6}	2.3×10^{-5}	7.7×10^{-5}
		Dermal	1.9×10^{-6}	1.4×10^{-6}	1.4×10^{-5}	3.5×10^{-5}
Mpeasem	Sediment	Oral	2.2×10^{-6}	1.6×10^{-4}	2.4×10^{-4}	2.5×10^{-4}
		Dermal	6.7×10^{-5}	5.6×10^{-5}	8.4×10^{-5}	8.1×10^{-4}
	Water	Oral	1.6×10^{-6}	3.1×10^{-6}	5.3×10^{-5}	3.1×10^{-4}
		Dermal	3.0×10^{-6}	1.1×10^{-6}	1.8×10^{-5}	1.4×10^{-4}

Appendix 10

Cancer health risk results from exposure to cadmium by resident adults, mine worker, galamsey operators and children in the Cape Coast Metropolis

Sample Point	Exposure Media	Exposure Route	Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow)	Soil	Ingestion	0.00015	0.0039	0.00022	0.00020
		Dermal	0.00010	0.00057	0.000040	0.0000040
	Water	Ingestion	0.00022	0.0057	0.00032	0.0029
		Dermal	0.000015	0.000084	0.000059	0.00013
Kakum (Kakumdo)	Soil	Ingestion	0.00018	0.0045	0.0019	0.0019
		Dermal	0.000012	0.00066	0.000035	0.00035
	Water	Ingestion	0.00036	0.0093	0.00052	0.00046
		Dermal	0.000025	0.00014	0.000095	0.000021
Asuboni stream (Effutu)	Soil	Ingestion	0.00021	0.0055	0.00031	0.0027
		Dermal	0.000015	0.000080	0.000056	0.00013
	Water	Ingestion	0.0016	0.040	0.0022	0.00020
		Dermal	0.0011	0.0059	0.0041	0.000093
Tap water from Brimsu	Water	Ingestion	0.0019	0.0048	0.00026	0.0024
		Dermal	0.000013	0.000069	0.000049	0.000011
Mpeasem	Soil	Ingestion	0.00017	0.0043	0.0024	0.0021
		Dermal	0.000011	0.000062	0.000044	0.000098
	Water	Ingestion	0.00035	0.0090	0.0050	0.0045
		Dermal	0.000024	0.00013	0.000092	0.000021

Appendix 11

Cancer health risk results from exposure to arsenic by resident adults, mine worker, galamsey operators and children in Bole – Bamboi District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult operators ASGM		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	0.00043	0.00017	0.00015	0.0039	0.00022	0.00024
		Dermal	0.00016	0.00089	0.00010	0.00057	0.00016	0.00019
BHT2	Water	Oral	0.00020	0.00050	0.00018	0.0045	0.00015	0.00026
		Dermal	0.00012	0.00065	0.000012	0.00066	0.00017	0.00019
BHT3	Water	Oral	0.00014	0.00036	0.00036	0.0093	0.00035	0.00043
		Dermal	0.00003	0.00015	0.00025	0.00053	0.00056	0.00057
BHT4	Water	Oral	0.00038	0.00042	0.00013	0.00025	0.00047	0.00052
		Dermal	0.00023	0.00025	0.00012	0.00023	0.00082	0.00099
EGWPS	Sediment	Oral	0.00022	0.00026	0.0027	0.0032	0.0006	0.00067
		Dermal	0.000067	0.00062	0.0022	0.0029	0.0002	0.00013
	Water	Oral	0.0078	0.0079	0.0033	0.0038	0.0037	0.0048
		Dermal	0.0014	0.0023	0.0021	0.0027	0.0029	0.0037
GWPS	Sediment	Oral	0.00054	0.00055	0.0042	0.0046	0.00044	0.0049
		Dermal	0.00037	0.00042	0.0031	0.0033	0.0003	0.0036
	Water	Oral	0.0036	0.0038	0.0045	0.0047	0.0046	0.0052
		Dermal	0.0024	0.0034	0.0039	0.0004	0.0043	0.0044
AWPS	Sediment	Oral	0.0048	0.0056	0.0059	0.0040	0.0042	0.0045
		Dermal	0.0034	0.0042	0.0047	0.0051	0.0034	0.0039
	Water	Oral	0.0067	0.0068	0.0076	0.0076	0.0077	0.0079
		Dermal	0.0045	0.0045	0.0061	0.0062	0.0065	0.0069

Appendix 12

Cancer health risk results from exposure to cadmium by resident adults, mine worker, galamsey operators and children in Bole – Bamboi District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult operators ASGM		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	0.00023	0.00035	0.00032	0.00055	0.00034	0.00037
		Dermal	0.00017	0.00021	0.00028	0.00048	0.00026	0.00028
BHT2	Water	Oral	0.00032	0.00027	0.00050	0.00054	0.00051	0.00061
		Dermal	0.00011	0.00029	0.00049	0.00052	0.00053	0.00055
BHT3	Water	Oral	0.00036	0.00094	0.00024	0.00072	0.00024	0.00044
		Dermal	0.00015	0.00074	0.00021	0.00061	0.00018	0.00030
BHT4	Water	Oral	0.00028	0.00033	0.00038	0.00046	0.00055	0.00059
		Dermal	0.00025	0.00029	0.00027	0.00041	0.00035	0.00049
EGWPS	Sediment	Oral	0.0014	0.00021	0.00018	0.00024	0.00031	0.00044
		Dermal	0.00023	0.00025	0.00017	0.00020	0.00035	0.00036
	Water	Oral	0.00022	0.00024	0.00022	0.00026	0.00056	0.00074
		Dermal	0.00026	0.00029	0.00025	0.00028	0.00046	0.00062
GWPS	Sediment	Oral	0.00033	0.00035	0.00023	0.00025	0.00029	0.00038
		Dermal	0.00032	0.00034	0.00021	0.00028	0.00028	0.00042
	Water	Oral	0.00031	0.00041	0.00019	0.00029	0.00033	0.00047
		Dermal	0.00028	0.00029	0.00018	0.00030	0.00039	0.00041
AWPS	Sediment	Oral	0.00063	0.00067	0.00044	0.00048	0.00036	0.00062
		Dermal	0.00042	0.00047	0.00043	0.00045	0.00068	0.00071
	Water	Oral	0.00064	0.00073	0.00052	0.00064	0.00073	0.00076
		Dermal	0.00058	0.00061	0.00055	0.00060	0.00075	0.00080

Appendix 13

Cancer health risk results from exposure to arsenic by resident adults, mine worker, galamsey operators and children in Nabdam District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult operators ASGM		Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	0.00063	0.00068	0.0012	0.0015	0.00017	0.00041
		Dermal	0.00012	0.00025	0.0020	0.0022	0.00011	0.00036
HDWA	Sediment	Oral	0.00043	0.00045	0.0027	0.0046	0.00059	0.0015
		Dermal	0.00027	0.00033	0.0015	0.0020	0.00040	0.0022
	Water	Oral	0.00077	0.00078	0.0033	0.0038	0.0052	0.0055
		Dermal	0.00061	0.00063	0.0023	0.0027	0.0034	0.0038
HDWB	Sediment	Oral	0.00034	0.00044	0.00018	0.00055	0.0068	0.0017
		Dermal	0.00031	0.00039	0.00034	0.00025	0.0046	0.0025
	Water	Oral	0.00054	0.00055	0.0025	0.0041	0.0072	0.0075
		Dermal	0.00046	0.00051	0.0029	0.0039	0.0071	0.0072
DA	Sediment	Oral	0.00037	0.00065	0.00031	0.00039	0.00025	0.00033
		Dermal	0.0006	0.00044	0.00042	0.00028	0.00021	0.00029
	Water	Oral	0.00072	0.00086	0.0035	0.0037	0.0015	0.0039
		Dermal	0.00052	0.00058	0.0032	0.0036	0.0010	0.0027
DB	Sediment	Oral	0.00043	0.00055	0.0022	0.0038	0.00018	0.00045
		Dermal	0.00035	0.00014	0.0021	0.0029	0.00012	0.00066
	Water	Oral	0.00065	0.00057	0.0056	0.0058	0.00020	0.00059
		Dermal	0.00066	0.00054	0.0047	0.0059	0.00001	0.00040

Appendix 14

Cancer health risk results from exposure to cadmium by resident adults, mine worker, galamsey operators and children in Nabdam District

Sampling point	Exposure media	Exposure route	Cancer health risk					
			Adults		Adult ASGM operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	0.00022	0.00057	0.0063	0.057	0.0017	0.0044
		Dermal	0.00012	0.00084	0.012	0.026	0.0012	0.0064
HDWA	Sediment	Oral	0.00016	0.00017	0.0022	0.020	0.0005	0.015
		Dermal	0.00015	0.00016	0.0040	0.0040	9 0.0000 4	0.0022
	Water	Oral	0.00027	0.00033	0.0037	0.034	0.001	0.20
		Dermal	0.00023	0.00028	0.0068	0.0090	0.0000 69	0.38
HDWB	Sediment	Oral	0.00028	0.00054	0.0015	0.013	0.0068	0.0017
		Dermal	0.00039	0.00042	0.0027	0.0062	0.0046	0.0025
	Water	Oral	0.00041	0.00061	0.0018	0.045	0.0025	0.023
DA	Sediment	Dermal	0.00038	0.00082	0.0010	0.0057	0.0040	0.0040
		Oral	0.00022	0.00052	0.0015	0.039	0.0022	0.020
	Water	Oral	0.00087	0.00088	0.0022	0.057	0.0032	0.029
DB	Sediment	Dermal	0.00014	0.00020	0.0012	0.066	0.0035	0.035
		Oral	0.00015	0.00019	0.0018	0.045	0.0019	0.019
	Water	Oral	0.00034	0.00036	0.0036	0.093	0.0052	0.046
		Dermal	0.00029	0.00035	0.0025	0.014	0.0095	0.021

Appendix 15

Results of non – cancer health effects from exposure to arsenic in the Prestea Huni Valley District

Sampling Point	Exposure Media	Exposure Route	Resident Adults		Galamsey Operators		Resident Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	0.098	0.78	0.022	2.2	4.3	5.7
		Dermal	0.056	0.065	0.045	1.4	4.6	4.5
	Water	Oral	1.2	34	7.1	2.3	5.1	10
		Dermal	0.98	11	0.34	1.1	0.23	0.015
Bodwire	Sediment	Oral	0.097	1.9	0.05	0.035	0.39	0.89
		Dermal	0.063	0.78	0.025	0.67	0.023	0.19
	Water	Oral	0.040	0.14	5.1	10	5.1	10
		Dermal	0.027	0.21	0.83	0.015	0.23	0.015
Ankobra	Sediment	Oral	0.02	0.71	2.7	5.3	0.2	0.071
		Dermal	0.013	0.01	0.43	0.0078	0.13	0.010
	Water	Oral	0.17	6.3	230	480	0.71	6.3
		Dermal	0.12	0.91	0.38	0.79	0.12	0.91
Twiagya	Sediment	Oral	2.7	99	3,700	7,400	2.7	99
		Dermal	1.9	140	6.0	11	1.9	14
	Water	Ingestion	1.9	67	3,700	730	2,500	5,000
		Dermal	1.3	9.8	6.0	1.2	4.1	7.3
Anfoe	Sediment	Oral	1.3	2.0	7.4	1.50	7.4	1.50
		Dermal	0.055	0.29	0.12	0.21	0.12	0.21
	Water	Oral	0.27	9.8	3.70	7.40	3.70	1.1
		Dermal	0.19	1.4	0.60	1.1	0.60	0.062
Worawora	Sediment	Oral	0.23	8.3	1.70	3.20	3.10	6.20
		Dermal	0.16	1.2	0.35	0.72	0.51	0.90
	Water	Oral	0.34	12	4.60	9.20	4.60	9.20
		Dermal	0.23	1.8	0.75	1.3	0.75	1.3
Mansi	Sediment	Oral	2.3	3.0	2.0	4.0	20	40
		Dermal	0.45	1.0	0.33	0.58	0.10	0.58
	Water	Oral	0.0004	0.014	2.40	4.90	0.18	6.5
		Dermal	0.00027	0.0021	0.40	0.71	0.12	0.95
Borehole at Dumase Aprepre	Water	Oral	0.54	1.9	730	1,500	0.54	19
		Dermal	0.37	2.8	1.2	2.1	0.37	2.8

Appendix 16

Results of non – cancer health risk from exposure to cadmium in the Prestea Huni Valley District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	3.2	4.3	2.7	5.3	2.7	3.3
		Dermal	0.56	0.098	0.0043	0.0077	0.0043	0.0077
	Water	Oral	0.028	0.0099	0.21	0.42	0.0016	0.056
		Dermal	0.019	0.0014	0.0034	0.0061	0.0011	0.082
Bodwire	Sediment	Oral	0.0016	0.0056	0.037	0.074	0.028	0.099
		Dermal	0.0011	0.0082	0.061	0.011	0.019	0.014
	Water	Oral	0.0038	0.14	5.1	10	0.0038	0.020
		Dermal	0.0026	0.020	0.0084	0.015	0.0026	0.14
Ankobra	Sediment	Oral	0.044	1.6	59	120	0.038	0.34
		Dermal	0.030	0.23	0.097	0.017	0.070	0.16
	Water	Oral	0.053	1.9	59	120	0.038	0.34
		Dermal	0.036	0.28	0.097	0.013	0.070	0.16
Twiagya	Sediment	Oral	0.015	0.56	21	42	21	42
		Dermal	0.011	0.081	0.034	0.061	0.034	0.061
	Water	Ingestion	0.014	0.50	19	37	19	37
		Dermal	0.0094	0.073	0.030	0.054	0.030	0.054
Anfoe	Sediment	Oral	0.003	0.11	8.8	16	4.0	8.0
		Dermal	0.002	0.016	0.018	0.037	0.0065	0.012
	Water	Oral	0.003	0.036	1.3	2.7	1.3	2.7
		Dermal	0.0068	0.0053	0.0023	0.0039	0.022	0.0039
Worawora	Sediment	Oral	0.51	18	0.690	100	6.90	140
		Dermal	0.35	2.7	1.1	2.0	1.1	2.0
	Water	Oral	0.23	8.1	300	610	300	610
		Dermal	0.15	1.2	0.50	0.89	0.50	0.89
Mansi	Sediment	Oral	0.23	8.2	140	0.026	310	620
		Dermal	0.16	1.2	0.29	0.60	0.50	0.90
	Water	Oral	0.42	15	560	1,100	560	1,100
		Dermal	0.28	2.2	0.92	1.6	0.92	1.6
Borehole at Dumase Aprepre	Water	Oral	0.36	13	480	970	0.36	1.9
		Dermal	0.24	1.9	7.9	1.4	0.24	13

Appendix 17

Results of non – cancer health risk from exposure to mercury in the Prestea Huni Valley District

Sample Point	Exposure Media	Exposure Route	Adults		Mine workers/Galamsey Operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	2.4	4.4	1.8	5.6	5.1	10
		Dermal	0.048	0.01	0.98	1.1	0.083	0.015
	Water	Oral	1.7	1.5	1.3	2.3	1.7	0.15
		Dermal	3.1	7.0	0.25	0.53	0.31	0.7
Bodwire	Sediment	Oral	16	3.1	0.25	0.46	0.4	0.1
		Dermal	2.7	1.4	0.15	0.1	0.6	0.14
	Water	Oral	0.16	1.3	110	200	0.15	1.3
		Dermal	0.27	0.62	0.22	0.46	0.27	0.62
Ankobra	Sediment	Oral	2.3	2.1	1,700	3,200	2.7	99
		Dermal	4.3	9.7	3.5	7.3	1.9	14
	Water	Oral	1.6	14	170	35	120	220
		Dermal	3.0	6.6	3.5	7.3	2.4	5.0
Twiagya	Sediment	Oral	0.047	0.42	3.5	6.3	35	0.15
		Dermal	0.086	0.19	0.70	0.15	0.070	0.032
	Water	Ingestion	0.23	2.7	37	74	17	32
		Dermal	0.43	0.97	0.60	1.1	320	0.72
Anfoe	Sediment	Oral	0.20	1.8	17	32	15	27
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
	Water	Oral	0.29	2.6	220	390	220	390
		Dermal	0.54	1.2	0.44	0.91	0.44	0.91
Worawora	Sediment	Oral	1.5	0.56	21	42	21	42
		Dermal	1.1	0.081	0.34	0.061	0.034	0.061
	Water	Oral	1.4	0.50	19	37	19	37
		Dermal	0.94	0.073	0.030	0.054	0.030	0.054
Mansi	Sediment	Oral	0.36	0.11	8.8	16	4.0	8.0
		Dermal	0.29	0.16	0.18	0.37	0.65	0.12
	Water	Oral	0.38	0.036	1.3	2.7	1.3	2.7
		Dermal	0.68	0.53	0.23	0.039	0.022	0.39
Borehole at Dumase Aprepre	Water	Oral	0.38	0.14	5.1	10	0.0038	0.020
		Dermal	0.26	0.020	0.0084	0.015	0.0026	0.14

Appendix 18

Results of non – cancer human health risk from exposure to lead by resident adults, mine workers/galamsey operators and children in the PHVD

Sampling Point	Exposure Media	Exposure Route	Resident Adults		Galamsey Operators		Resident Children	
			CTE	RME	CTE	RME	CTE	RME
Aprepre	Sediment	Oral	94	170	9.4	17	200	400
		Dermal	0.19	0.39	1.9	3.9	0.33	0.58
	Water	Oral	0.16	1.4	110	210	0.16	1.4
		Dermal	0.29	0.64	0.23	0.48	0.29	0.64
Bodwire	Sediment	Oral	8.7	16	1.4	2.5	0.0018	0.016
		Dermal	1.8	7.6	0.0027	0.0057	0.0034	0.0076
	Water	Oral	0.31	2.8	230	410	0.31	2.8
		Dermal	0.57	1.3	0.46	0.95	0.57	1.3
Ankobra	Sediment	Oral	0.46	4.2	340	620	0.54	19
		Dermal	0.85	1.9	0.69	1.4	0.37	2.8
	Water	Oral	10	93	330	615	760	1,400
		Dermal	1.9	4.4	0.75	2.4	1.5	3.2
Twiagya	Sediment	Oral	0.44	3.9	320	590	320	590
		Dermal	0.086	1.8	0.65	1.4	0.65	1.4
	Water	Ingestion	0.19	1.7	300	610	140	260
		Dermal	0.36	0.80	0.50	0.89	0.29	0.60
Anfoe	Sediment	Oral	0.20	1.8	140	260	150	260
		Dermal	0.36	0.81	0.29	0.60	0.29	0.61
	Water	Oral	0.36	3.2	260	480	260	480
		Dermal	0.66	1.5	0.53	1.1	0.53	1.1
Worawora	Sediment	Oral	0.23	2.7	370	740	170	320
		Dermal	0.43	0.97	0.60	1.1	320	0.72
	Water	Oral	0.20	1.8	170	320	150	270
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
Mansi	Sediment	Oral	0.29	2.6	220	390	220	390
		Dermal	0.54	1.2	0.44	0.91	0.44	0.91
	Water	Oral	0.00037	0.0011	0.0043	0.11	0.0063	0.057
		Dermal	0.00067	0.00050	0.003	0.017	0.012	0.026
Borehole at Dumase Aprepre	Water	Oral	0.0021	0.055	0.0031	0.027	0.0031	0.027
		Dermal	0.0015	0.0080	0.0056	0.013	0.0056	0.013

Appendix 19

Results of non – cancer human health risk from exposure to arsenic by resident adults, mine/galamsey operators and children in the Bole-Bamboi District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	0.16	1.4	11	21	0.16	1.4
		Dermal	0.29	0.64	0.23	0.48	0.29	0.64
BHT2	Water	Oral	0.31	2.8	23	41	0.31	2.8
		Dermal	0.57	1.3	0.46	0.95	0.57	1.3
BHT3	Water	Oral	10	93	330	615	760	140
		Dermal	1.9	44	0.75	2.4	1.5	3.2
BHT4	Water	Oral	0.19	1.7	30	61	14	26
		Dermal	0.36	0.80	0.50	0.89	0.29	0.60
WGWPS	Sediment	Oral	0.20	1.8	14	26	15	26
		Dermal	0.36	0.81	0.29	0.60	0.29	0.61
	Water	Oral	0.36	3.2	26	48	26	48
		Dermal	0.66	1.5	0.53	1.1	0.53	1.1
GWPS	Sediment	Oral	2.7	8.1	2.1	7.6	2.7	3.3
		Dermal	0.058	0.39	1.4	1.1	0.0043	0.0077
	Water	Oral	1.4	4.1	6.1	38	0.0016	0.056
		Dermal	0.025	1.9	7.2	3.8	0.0011	0.082
AWPS	Sediment	Oral	1.1	3.4	8.9	3.2	0.028	0.099
		Dermal	2.1	1.6	6.0	4.7	0.019	0.014
	Water	Oral	3.5	5.1	1.3	4.8	0.0038	0.020
		Dermal	1.7	2.3	3.0	6.9	0.0026	0.14

Appendix 20

Results of non – cancer human health risk from exposure to cadmium by resident adults, mine/galamsey operators and children in Bole - Bamboi District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	0.16	1.4	11	21	0.16	1.4
		Dermal	7.9	1.8	10	18	6.01	8.9
BHT2	Water	Oral	8.7	16	1.4	2.5	0.18	0.16
		Dermal	0.31	2.8	23	41	0.31	2.8
BHT3	Water	Oral	0.46	4.2	34	62	0.54	19
		Dermal	10	93	33	61	76	14
BHT4	Water	Oral	4.4	3.9	3.2	5.9	3.2	1.5
		Dermal	1.9	1.7	30	61	14	26
EGWPS	Sediment	Oral	0.20	1.8	14	26	15	26
		Dermal	3.6	4.5	16	28	22	32
	Water	Oral	0.36	3.2	26	48	26	48
		Dermal	1.23	1.52	28	32	28	34
GWPS	Sediment	Oral	2.7	8.1	2.1	7.6	2.7	3.3
		Dermal	2.9	8.8	2.6	7.5	3.0	3.1
	Water	Oral	1.4	4.1	6.1	38	0.16	0.056
		Dermal	1.9	4.2	4.8	16	2.64	5.4
AWPS	Sediment	Oral	0.032	0.79	0.083	0.20	0.002	0.047
		Dermal	3.5	0.98	1.01	1.20	1 0.002 5	0.058
	Water	Oral	110	250	59	63	15	17
		Dermal	100	240	60	62	16	19

Appendix 21

Results of non – cancer human health risk from exposure to lead by resident adults, mine/galamsey operators and children in Bole-Bamboi District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	2.94	3.17	9.4	17	2.0	4.0
		Dermal	1.16	4.4	11	21	0.16	1.4
BHT2	Water	Oral	8.7	16	1.4	2.5	1.18	0.16
		Dermal	7.3	2.8	23	41	2.31	2.8
BHT3	Water	Oral	1.4	4.2	34	62	54	19
		Dermal	10	93	33	61	76	14
BHT4	Water	Oral	4.4	3.9	3.2	5.9	3.2	1.5
		Dermal	1.9	1.7	30	61	14	26
EGWPS	Sediment	Oral	1.20	1.8	14	26	15	26
		Dermal	2.5	2.4	13	25	12	23
	Water	Oral	36	32	26	48	24	42
		Dermal	29	30	20	32	23	36
GWPS	Sediment	Oral	2.7	8.1	2.1	7.6	2.7	3..3
		Dermal	1.8	6.1	1.9	5.3	2.2	3.1
	Water	Oral	1.4	4.1	6.1	3.8	1.6	6.2
		Dermal	0.9	1.2	3.2	4.1	2.3	4.5
AWPS	Sediment	Oral	7.9	8.0	9.0	9.8	6.0	8.9
		Ingestion	3.2	7.9	8.3	8.5	2.1	4.7
	Water	Oral	4.1	5.0	5.9	6.3	15	17
		Dermal	3.2	3.6	4.8	5.6	22	24

Appendix 22

Results of non – cancer human health risk from exposure to mercury by resident adults, mine/galamsey operators and children in Bole-Bamboi District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHT1	Water	Oral	4.3	12	18	23	4.5	3.4
		Dermal	2.1	9.0	12	9.0	1.2	2.9
BHT2	Water	Oral	12	19	5.8	12	0.39	0.78
		Dermal	25	55	2.9	9.8	0.078	0.098
BHT3	Water	Oral	3.2	30	2.1	4.3	22	44
		Dermal	6.0	4	0.78	1.3	12	23
BHT4	Water	Oral	0.046	0.41	2.1	4.3	120	240
		Dermal	0.084	0.019	1.9	2.2	45	98
EGWPS	Sediment	Oral	0.013	0.12	2.1	4.3	20	40
		Dermal	0.024	0.055	1.9	2.2	9.5	24
	Water	Oral	0.012	0.11	5.8	12	9.3	19
		Dermal	0.022	0.049	2.3	7.6	5.6	4.7
GWPS	Sediment	Oral	0.025	0.023	100	210	0.52	1.0
		Dermal	0.043	0.010	56	120	0.45	0.98
	Water	Oral	0.086	0.077	2.2	4.4	1.1	2.2
		Dermal	0.016	0.036	1.3	2.4	0.056	1.89
AWPS	Sediment	Oral	9.8	18	2.7	5.3	0.044	1.6
		Dermal	0.02	0.041	0.0043	0.0077	0.030	0.23
	Water	Oral	19	37	0.0013	0.012	34	62
		Dermal	0.030	0.054	0.0025	0.0055	0.068	0.14

Appendix 23

Results of non – cancer human health risk from exposure to arsenic by resident adults, mine/galamsey operators and children in the Nabdam District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	0.0015	0.039	0.0022	0.020	0.00059	0.015
		Dermal	0.001	0.0057	0.0040	0.0040	0.00004	0.0022
HDWA	Sediment	Oral	0.0036	0.067	0.0037	0.034	0.001	0.20
		Dermal	0.0018	0.0098	0.0068	0.0090	0.000069	0.38
	Water	Oral	0.0010	0.027	0.0015	0.013	4.0	1.0
		Dermal	0.00074	0.0039	0.0027	0.0062	2.7	1.5
HDWB	Sediment	Oral	0.0013	0.034	0.0019	0.019	5.8	1.3
		Dermal	0.00071	0.0050	0.0035	0.035	2.7	1.9
	Water	Oral	0.0018	0.045	0.0025	0.023	0.0068	0.0017
		Dermal	0.0012	0.066	0.0046	0.010	0.0046	0.0025
DA	Sediment	Oral	0.0015	0.039	0.0022	0.020	0.000059	0.015
		Dermal	0.0010	0.0057	0.0040	0.0040	0.000004	0.0022
	Water	Oral	0.0022	0.057	0.0032	0.029	0.0087	0.0022
		Dermal	0.0015	0.0084	0.0059	0.013	0.0059	0.033
DB	Sediment	Oral	0.0018	0.045	0.0019	0.019	0.0068	0.0017
		Dermal	0.0012	0.066	0.0035	0.035	0.0046	0.0025
	Water	Oral	0.0036	0.093	0.0052	0.046	0.014	0.036
		Dermal	0.0025	0.014	0.0095	0.021	0.0095	0.052

Appendix 24

Results of non – cancer human health risk from exposure to cadmium by resident adults, mine/galamsey operators and children in the Nabdam District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	1.3	3.8	5.9	1.5	2.2	2.0
		Dermal	2.3	1.7	4.0	2.2	4.0	3.0
HDWA	Sediment	Oral	2.2	6.5	1.0	2.0	3.7	3.4
		Dermal	4.0	3.0	6.9	3.8	6.8	9.0
	Water	Oral	8.6	2.6	4.0	1.0	1.5	1.3
		Dermal	1.6	1.2	2.7	1.5	27	6.2
HDWA	Sediment	Oral	1.2	1.1	5.8	1.3	1.9	1.9
		Dermal	2.0	2.0	2.7	1.9	3.5	3.5
	Water	Oral	1.5	4.6	6.8	1.7	2.5	2.3
		Dermal	2.7	2.0	4.6	2.5	4.6	10
DA	Sediment	Oral	1.3	3.8	5.9	1.5	22	20
		Dermal	2.3	1.7	4.0	2.2	40	40
	Water	Oral	1.8	5.5	8.7	2.2	32	29
		Dermal	3.4	2.5	5.9	3.3	59	13
DB	Soil	Oral	1.2	1.1	6.8	7.0	1.9	19
		Dermal	2.0	2.0	4.6	25	0.35	35
	Water	Oral	3.0	8.9	1.4	3.6	52	46
		Dermal	5.5	4.1	9.5	5.2	95	21

Appendix 25

Results of non – cancer health effects from exposure to mercury in Nabdam District

Sampling Point	Exposure Media	Exposure Route	Resident Adults		Galamsey Operators		Resident Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	2.7	7.0	0.39	0.38	3.8	0.15
		Dermal	0.031	4.78	0.23	0.14	1.7	0.1
HDWA	Sediment	Oral	9.1	2.3	13	21	6.5	0.36
		Dermal	1.5	1.8	12	9.8	3.0	0.18
	Water	Oral	1.5	4.0	22	20	2.6	0.10
		Dermal	0.014	3.8	2.3	5.6	1.2	0.74
HDWB	Sediment	Oral	1.1	2.7	15	23	1.1	0.13
		Dermal	0.075	0.83	6.6	7.8	2.0	0.71
	Water	Oral	1.4	3.6	20	18	4.6	0.18
		Dermal	0.001	2.7	12	1.4	2.0	0.12
DA	Sediment	Oral	1.3	3.5	1.9	1.7	3.8	0.15
		Dermal	0.98	2.2	0.98	0.56	1.7	0.10
	Water	Oral	0.17	4.3	2.4	2.1	5.5	0.22
		Dermal	0.0023	0.0022	0.78	1.3	2.5	0.15
DB	Sediment	Oral	2.2	5.6	0.0003	0.0028	1.1	0.18
		Dermal	0.043	3.8	1		2.0 ⁷	0.12
	Water	Oral	0.21	0.55	0.31	0.27	8.9	0.36
		Dermal	0.045	0.12	0.0012	0.023	4.1	0.25

Appendix 26

Results of non – cancer health risk from exposure to lead in the Nabdam District

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
BHN	Water	Oral	12	25	0.98	1.1	0.39	0.78
		Dermal	10	14	0.34	0.13	0.45	0.34
HDWA	Sediment	Oral	0.15	0.24	0.27	0.21	0.19	0.37
		Dermal	0.78	0.17	0.43	0.58	0.49	0.29
	Water	Oral	0.3	0.6	0.17	6.3	22	44
		Dermal	1.3	0.98	0.11	4.3	12	24
HDWB	Sediment	Oral	3.8	7.0	0.12	0.91	130	270
		Dermal	1.2	6.6	0.023	0.076	75	127
	Water	Oral	4.6	8.4	2.7	9.9	1.20	2.40
		Dermal	0.78	0.91	1.4	6.7	5.6	1.23
DA	Sediment	Oral	13	24	1.9	1.4	20	40
		Dermal	9.3	13	0.98	0.13	13	15
	Water	Oral	12	22	1.9	6.7	9.3	19
		Dermal	2.9	7.2	1.8	3.8	6.4	9.4
DB	Sediment	Oral	0.25	0.43	1.3	9.8	0.52	1.0
		Dermal	0.3	0.23	0.98	4.5	0.36	0.89
	Water	Oral	0.86	0.16	12	35	1.1	2.2
		Dermal	0.23	0.34	3.5	24	0.45	0.78

Appendix 27

Results of non – cancer health risk from exposure to cadmium in the Tarkwa Nsuaem Municipality

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey Operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	241	44	18	56	51	10
		Dermal	48	10	0.098	1.1	0.83	0.15
	Water	Oral	17	15	13	23	17	15
		Dermal	3.2	7.0	0.25	0.53	0.31	0.7
Asuman	Sediment	Oral	16	3.1	0.25	0.46	0.4	0.1
		Dermal	2.7	1.4	0.15	0.1	0.6	0.14
	Water	Oral	0.16	1.3	110	200	0.15	1.3
		Dermal	0.27	0.62	0.22	0.46	0.27	0.62
Abodwesh	Sediment	Oral	23	21	170	320	27	99
		Dermal	4.3	9.7	3.5	7.3	1.9	14
	Water	Oral	16	14	17	35	12	22
		Dermal	3.0	6.6	3.5	7.3	2.4	5.0
Ateberebe	Sediment	Oral	0.47	0.42	3.5	6.3	35	0.15
		Dermal	0.86	0.19	0.70	0.15	0.70	0.32
	Water	Oral	0.23	2.7	37	74	17	32
		Dermal	0.43	0.97	0.60	1.1	320	0.72
Achofe	Sediment	Oral	0.20	1.8	17	32	15	27
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
	Water	Oral	2.9	2.6	22	39	22	39
		Dermal	0.54	1.2	0.44	0.91	0.44	0.91
Subri	Sediment	Oral	4.4	1.6	59	120	0.38	0.34
		Dermal	0.030	0.23	0.97	0.017	0.70	0.16
	Water	Oral	0.53	1.9	59	120	0.38	0.34
		Dermal	0.36	0.28	0.97	0.13	0.70	0.16
Betinyere	Sediment	Oral	1.5	0.56	21	42	21	42
		Dermal	1.1	0.81	0.34	0.061	0.034	0.061
	Water	Oral	1.4	0.50	19	37	19	37
		Dermal	0.94	0.73	0.30	0.054	0.30	0.54
Ahomabruen	Sediment	Oral	0.36	0.11	8.8	16	4.0	8.0
		Dermal	0.29	0.16	0.18	0.37	0.65	0.12
	Water	Oral	0.38	0.36	1.3	2.7	1.3	2.7
		Dermal	0.68	0.53	0.23	0.039	0.022	0.39
Borehole – Abekoase	Water	Oral	0.38	0.14	5.1	10	0.38	0.20
		Dermal	0.26	0.20	0.84	0.15	0.26	0.14

Appendix 28

Results of non – cancer human health risk from exposure to arsenic by resident adults, mine workers/galamsey operators and children in Tarkwa Municipality

Sampling Point	Exposure Media	Exposure Route	Resident Adults		Galamsey Operators		Resident Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	9.4	1.70	9.4	17	20.0	40.0
		Dermal	0.19	0.39	1.9	3.9	0.33	0.58
	Water	Oral	0.16	1.4	1.10	21.0	0.16	1.4
		Dermal	0.29	0.64	0.23	0.48	0.29	0.64
Asuman	Sediment	Oral	8.7	16	1.4	2.5	0.18	0.16
		dermal	1.8	7.6	0.27	0.57	0.34	0.76
	Water	Oral	0.31	2.8	23.0	41.0	0.31	2.8
		Dermal	0.57	1.3	0.46	0.95	0.57	1.3
Abodwesh	Sediment	Oral	0.46	4.2	34.0	62.0	0.54	19
		Dermal	0.85	1.9	0.69	1.4	0.37	2.8
	Water	Oral	10	93	33.0	61.5	76.0	14.0
		Dermal	1.9	4.4	0.75	2.4	1.5	3.2
Ateberebe	Sediment	Oral	0.44	3.9	32.0	59.0	32.0	59.0
		Dermal	0.086	1.8	0.65	1.4	0.65	1.4
	Water	Oral	0.19	1.7	30.0	61.0	14.0	26.0
		Dermal	0.36	0.80	0.50	0.89	0.29	0.60
Achofe	Sediment	Oral	0.20	1.8	14.0	26.0	1.50	2.60
		Dermal	0.36	0.81	0.29	0.60	0.29	0.61
	Water	Oral	0.36	3.2	2.60	4.80	2.60	4.80
		Dermal	0.66	1.5	0.53	1.1	0.53	1.1
Subri	Sediment	Oral	1.6	14	17	32	12	22
		Dermal	3.0	6.6	3.5	7.3	2.4	5.0
	Water	Oral	0.047	0.42	35	63	35	0.15
		Dermal	0.086	0.19	0.070	0.15	0.070	0.032
Betinyere	Sediment	Oral	0.23	2.7	37	74	17	3.20
		Dermal	0.43	0.97	0.60	1.1	3.20	0.72
	Water	Oral	0.20	1.8	1.70	3.20	1.50	2.70
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
Ahomabruen	Sediment	Oral	0.29	2.6	220	3.90	2.20	3.90
		dermal	0.54	1.2	0.44	0.91	0.44	0.91
	Water	Oral	0.37	11	4.3	11	6.3	5.7
		Dermal	0.67	5.0	0.3	0.17	1.2	2.6
Borehole – Abekoase	Water	Oral	0.21	0.55	0.31	0.027	0.31	0.27
		Dermal	0.15	0.80	0.56	0.013	0.56	0.13

Appendix 29

Results of non – cancer human health risk from exposure to mercury by resident adults, mine/galamsey operators and children in the Tarkwa Nsuaem Municipality

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	7.1	23	2.1	4.3	1.1	2.1
		Dermal	2.3	12	1.4	2.9	0.9	1.2
	Water	Oral	5.1	10	5.8	12	0.39	0.78
		Dermal	3.6	6.6	4.3	6.5	0.029	0.56
Asuman	Sediment	Oral	2.7	5.3	7.4	15	0.19	0.37
		Dermal	1.1	2.4	2.8	6.0	0.11	0.29
	Water	Oral	2.30	4.80	2.1	4.3	22	44
		Dermal	1.5	2.78	1.2	2.3	12	35
Abodwesh	Sediment	Oral	3.70	7.4	2.1	4.3	1.30	2.70
		Dermal	1.6	3.9	1.1	3.0	1.0	1.98
	Water	Oral	3.7	7.30	2.1	4.3	1.20	2.40
		Dermal	3.2	5.9	2.0	3.1	1.1	2.2
Ateberebe	Sediment	Oral	7.4	15	2.1	4.3	20	40
		Dermal	3.4	6.8	0.25	2.4	6.4	19
	Water	Oral	3.70	7.40	5.8	12	9.3	19
		Dermal	2.0	5.8	2.5	6.2	7.9	12
Achofe	Sediment	Oral	1.70	3.20	10.0	21.0	0.52	1.0
		Dermal	0.037	0.73	7.34	9.3	0.025	0.98
	Water	Oral	4.60	9.20	2.2	4.4	1.1	2.2
		Dermal	4.4	6.8	2.0	3.2	0.18	1.8
Subri	Sediment	Oral	0.23	2.7	37	74	17	3.20
		Dermal	0.43	0.97	0.60	1.1	3.20	0.72
	Water	Oral	0.20	1.8	1.70	3.20	1.50	2.70
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
Betinyere	Sediment	Oral	0.29	2.6	220	3.90	2.20	3.90
		Dermal	0.54	1.2	0.44	0.91	0.44	0.91
	Water	Oral	0.37	11	4.3	11	6.3	5.7
		Dermal	0.67	5.0	0.3	0.17	1.2	2.6
Ahomabruen	Sediment	Oral	0.21	0.55	0.31	0.027	0.31	0.27
		Dermal	0.15	0.80	0.56	0.013	0.56	0.13
	Water	Oral	0.38	0.36	1.3	2.7	1.3	2.7
		Dermal	0.68	0.53	0.23	0.039	0.022	0.39
Borehole – Abekoase	Water	Oral	0.38	0.14	5.1	10	0.38	0.20
		Dermal	0.26	0.20	0.84	0.15	0.26	0.14

Appendix 30

Results of non – cancer human health risk from exposure to lead by resident adults, mine/galamsey operators and children in the Tarkwa Nsuaem Municipality

Sample Point	Exposure Media	Exposure Route	Adults		Galamsey operators		Children	
			CTE	RME	CTE	RME	CTE	RME
Huni	Sediment	Oral	0.043	1.1	0.63	5.7	1.7	4.4
		Dermal	0.03	0.17	0.12	2.6	1.2	0.64
	Water	Oral	1.5	3.9	2.2	2.0	5.9	15
		Dermal	0.1	0.57	0.40	0.040	4.0	2.2
Asuman	Sediment	Oral	0.36	0.67	0.37	0.34	0.10	0.20
		Dermal	0.18	0.098	0.068	0.090	0.069	0.38
	Water	Oral	0.10	0.27	0.15	0.013	4.0	1.0
		Dermal	0.074	0.039	0.027	0.0062	2.7	1.5
Abodwesh	Sediment	Oral	0.13	0.34	0.19	0.019	5.8	1.3
		Dermal	0.071	0.050	0.035	0.035	2.7	1.9
	Water	Oral	0.18	0.45	0.25	0.23	0.68	0.17
		Dermal	0.012	0.066	0.046	0.010	0.046	0.025
Ateberebe	Sediment	Oral	0.015	0.039	0.22	0.020	0.59	0.15
		Dermal	0.0010	0.0057	0.040	0.0040	0.004	0.022
	Water	Oral	0.22	0.57	0.32	0.29	0.87	0.22
		Dermal	0.015	0.084	0.059	0.013	0.0059	0.033
Achofe	Sediment	Oral	0.18	0.045	0.19	0.19	0.068	0.17
		Dermal	0.012	0.066	0.035	0.035	0.0046	0.025
	Water	Oral	0.0036	0.093	0.0052	0.046	0.014	0.036
		Dermal	0.0025	0.014	0.0095	0.021	0.0095	0.052
Subri	Sediment	Oral	0.21	0.055	0.031	0.027	0.082	0.021
		Dermal	0.015	0.080	0.0056	0.013	0.0056	0.0031
	Water	Oral	0.16	0.040	0.22	0.020	0.60	0.16
		Dermal	0.011	0.0059	0.041	0.0093	0.041	0.023
Betinyere	Sediment	Oral	0.19	0.048	0.0026	0.024	0.0071	0.0018
		Dermal	0.013	0.0069	0.0049	0.011	0.0049	0.0027
	Water	Oral	0.17	0.043	0.24	0.021	0.064	0.16
		Dermal	0.011	0.0062	0.044	0.0098	0.0044	0.024
Ahomabruen	Sediment	Oral	0.35	0.90	0.50	0.45	0.14	0.35
		Dermal	0.024	0.013	0.092	0.021	0.092	0.051
	Water	Oral	0.33	0.84	0.47	0.42	0.13	0.32
		Dermal	0.022	0.012	0.086	0.019	0.086	0.047
Borehole – Abekoase	water	Oral	0.54	0.14	0.77	0.70	0.21	0.54
		Dermal	0.0037	0.020	0.014	0.032	0.014	0.0078

Appendix 31

Results of non – cancer human health risk from exposure to arsenic by resident adults and children in the Cape Coast Metropolis

Sample Point	Exposure Media	Exposure Route	Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow)	Sediment	Oral Dermal	0.00037 0.000067	0.00011 0.000050	0.0063 0.00012	0.57 0.00026
	Water	Oral Dermal	0.013 0.00023	0.038 0.00017	0.0022 0.00040	0.20 0.0030
Kakum (Kakumdo)	Sediment	Oral Dermal	0.22 0.040	0.65 0.030	0.037 0.0068	0.0034 0.00090
	Water	Oral Dermal	0.086 0.0016	0.026 0.0012	0.15 0.0027	0.13 0.0062
Asuboni stream (Effutu)	Sediment	Oral Dermal	0.0012 0.20	0.11 0.0020	0.19 0.0035	0.19 0.035
	Water	Oral Dermal	0.15 0.027	0.46 0.20	0.25 0.0046	0.23 0.0010
Tap water from Brimsu	Water	Oral Dermal	0.18 0.034	0.55 0.025	0.32 0.059	0.29 0.13
Mpeasem	Sediment	Oral Dermal	0.12 0.0020	0.11 0.020	0.0019 0.00035	0.019 0.0035
	Water	Oral Dermal	0.30 0.055	0.89 0.41	0.052 0.0095	0.46 0.21

Appendix 32

Results of non – cancer human health risk from exposure to cadmium by resident adults and children in the Cape Coast Metropolis

Sample Point	Exposure Media	Exposure Route	Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow)	Sediment	Oral	1.2×10^{-7}	1.1×10^{-7}	1.4×10^{-7}	4.1×10^{-6}
		Dermal	2.0×10^{-7}	2.0×10^{-7}	2.5×10^{-7}	1.9×10^{-6}
	Water	Oral	1.5×10^{-7}	4.6×10^{-6}	2.9×10^{-7}	8.7×10^{-6}
		Dermal	2.7×10^{-7}	2.0×10^{-6}	5.3×10^{-7}	4.0×10^{-6}
Kakum (Kakumdo)	Sediment	Oral	1.3×10^{-7}	3.8×10^{-6}	2.7×10^{-7}	8.1×10^{-6}
		Dermal	2.3×10^{-7}	1.7×10^{-6}	5.0×10^{-7}	3.7×10^{-6}
	Water	Oral	1.8×10^{-7}	5.5×10^{-6}	4.5×10^{-7}	1.3×10^{-5}
		Dermal	3.4×10^{-7}	2.5×10^{-6}	8.2×10^{-7}	6.2×10^{-6}
Asuboni stream (Effutu)	Sediment	Oral	1.2×10^{-7}	1.1×10^{-7}	1.2×10^{-8}	7.7×10^{-6}
		Dermal	2.0×10^{-7}	2.0×10^{-7}	3.2×10^{-8}	8.5×10^{-6}
	Water	Oral	3.0×10^{-7}	8.9×10^{-6}	6.0×10^{-6}	2.8×10^{-5}
		Dermal	5.5×10^{-7}	4.1×10^{-6}	4.4×10^{-7}	3.5×10^{-5}
Tap water from Brimsu	Water	Oral	1.8×10^{-7}	5.3×10^{-6}	7.2×10^{-7}	2.0×10^{-6}
		Dermal	3.2×10^{-7}	2.4×10^{-6}	4.1×10^{-6}	1.3×10^{-6}
Mpeasem	Sediment	Oral	1.3×10^{-7}	3.9×10^{-6}	3.2×10^{-7}	1.8×10^{-7}
		Dermal	2.4×10^{-7}	1.8×10^{-6}	2.3×10^{-8}	1.2×10^{-8}
	Water	Oral	1.5×10^{-7}	4.6×10^{-6}	9.9×10^{-7}	4.7×10^{-6}
		Dermal	2.8×10^{-7}	2.1×10^{-6}	5.6×10^{-6}	2.1×10^{-6}

Appendix 33

Results of non – cancer human health risk from exposure to mercury by resident adults and children in the Cape Coast Metropolis

Sample Point	Exposure Media	Exposure Route	Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow)	Sediment	Oral	0.00015	0.0039	0.00022	0.00020
		Dermal	0.00010	0.00057	0.000040	0.0000040
	Water	Oral	0.00022	0.0057	0.00032	0.0029
		Dermal	0.000015	0.000084	0.000059	0.00013
Kakum (Kakumdo)	Sediment	Oral	0.00018	0.0045	0.0019	0.0019
		Dermal	0.000012	0.00066	0.000035	0.00035
	Water	Oral	0.00036	0.0093	0.00052	0.00046
		Dermal	0.000025	0.00014	0.000095	0.000021
Asuboni stream (Effutu)	Sediment	Oral	0.00021	0.0055	0.00031	0.0027
		Dermal	0.000015	0.000080	0.000056	0.00013
	Water	Oral	0.0016	0.040	0.0022	0.00020
		Dermal	0.0011	0.0059	0.0041	0.000093
Tap water from Brimsu	Water	Oral	0.0019	0.0048	0.00026	0.0024
		Dermal	0.000013	0.000069	0.000049	0.000011
Mpeasem	Sediment	Oral	0.00017	0.0043	0.0024	0.0021
		Dermal	0.000011	0.000062	0.000044	0.000098
	Water	Oral	0.00035	0.0090	0.0050	0.0045
		Dermal	0.000024	0.00013	0.000092	0.000021

Appendix 34

Results of non – cancer human health risk from exposure to lead by resident adults, mine/galamsey operators and children in the Cape Coast Metropolis

Sample Point	Exposure Media	Exposure Route	Adults		Children	
			CTE	RME	CTE	RME
Kakum (Kwaprow)	Soil	Oral	0.000034	0.00017	0.042	0.00069
		Dermal	0.000068	0.000085	0.014	0.00018
	Water	Oral	0.025	0.0034	0.000086	0.00043
		Dermal	0.0017	0.00012	0.0000096	0.000011
Kakum (Kakumdo)	Soil	Oral	0.0056	0.000037	0.0063	0.0086
		Dermal	0.00028	0.000028	0.00012	0.00076
	Water	Oral	0.068	0.00044	0.00023	0.000058
		Dermal	0.00043	0.000076	0.0000045	0.000023
Asuboni stream (Effutu)	Soil	Oral	0.000025	0.0077	0.00052	0.013
		Dermal	0.0000067	0.00083	0.00018	0.0034
	Water	Oral	0.0031	0.0026	0.077	0.0065
		Dermal	0.000024	0.000056	0.0014	0.00053
Tap water from Brimsu	Water	Oral	0.0071	0.00022	0.00056	0.00073
		Dermal	0.00058	0.000067	0.000098	0.000035
Mpeasem	Soil	Oral	0.0068	0.0000067	0.023	0.98
		Dermal	0.000016	0.00000045	0.0087	0.067
	Water	Oral	0.00021	0.00067	0.62	0.78
		Dermal	0.000019	0.000078	0.15	0.078

Appendix 35

Questionnaire for the collection of blood samples

QUESTIONNAIRE ID**TOWN/VILLAGE.....**
LOCALITY..... **ENUMERATION AREA**
ENUMERATION AREA CODE..... **HOUSE No.....**
GPS COORDINATES.....

POPULATION STUDY OF SELF-REPORTED SYMPTOMS/HEALTH CONDITION

I would like to ask you about symptoms that you have had recently and in the past one year. Kindly answer them as accurately as you can. Thank you for your time!

KEY : 0 = Never 1=Once in the year 2= twice in the year 3= every quarter 4= throughout the year

RESPIRATORY SYSTEM, ALLERGY/ENT

In the past 1 year, have you ever had ...

1.	An asthmatic attack?	0 [] 1 [] 2 [] 3 [] 4 []
2.	Allergic reaction?	0 [] 1 [] 2 [] 3 [] 4 []
3.	Cough and /or sputum production on most days for more than 3 months?	0 [] 1 [] 2 [] 3 [] 4 []
4.	Wheezing or whistling sound in the chest when breathing?	0 [] 1 [] 2 [] 3 [] 4 []
5.	Shortness of breath?	0 [] 1 [] 2 [] 3 [] 4 []
6.	Chest tightness lasting for ≥ 30 min	0 [] 1 [] 2 [] 3 [] 4 []
7.	Cough/sore throat not caused by a cold lasting for ≥ 14 days	0 [] 1 [] 2 [] 3 [] 4 []
8.	itchy or irritated eyes	0 [] 1 [] 2 [] 3 [] 4 []

9.	nose bleeds	0 [] 1 [] 2 [] 3 [] 4 []
10.	Stuffy nose/rhinitis /catarrh	0 [] 1 [] 2 [] 3 [] 4 []
11.	Hearing loss or dysfunction	0 [] 1 [] 2 [] 3 [] 4 []
12.	Tinnitus/ringing in the ears	0 [] 1 [] 2 [] 3 [] 4 []

CARDIOVASCULAR

In the past 1 year, have you ever had ...

13.	High blood pressure	0 [] 1 [] 2 [] 3 [] 4 []
14.	Chest pain	0 [] 1 [] 2 [] 3 [] 4 []
15.	Heart palpitations	0 [] 1 [] 2 [] 3 [] 4 []
16.	Ankle swelling	0 [] 1 [] 2 [] 3 [] 4 []

DERMAL

In the past 1 year, have you ever had ...

I would like to ask you about symptoms that you have had recently and in the past one year. Kindly answer them as accurately as you can. Thank you for your time!

KEY : 0 = Never 1=Once in the year 2= twice in the year 3= every quarter 4= throughout the year

17	Rashes or skin problems	0 [] 1 [] 2 [] 3 [] 4 []
18	Dermatitis	0 [] 1 [] 2 [] 3 [] 4 []
19	Skin irritation (over areas of the body that stayed for 3 days or more and does not include measles, chicken pox, bites, or allergic reaction)	0 [] 1 [] 2 [] 3 [] 4 []
20	Burning skin	0 [] 1 [] 2 [] 3 [] 4 []

21	Itching	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
22	Hair loss	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
MUSCULOSKELETAL		
In the past 1 year, have you ever had ...		
23	Muscle weakness	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
24	Joint pain, aching, or stiffness?	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
NEUROLOGIC/PSYCHIATRIC		
In the past 1 year, have you ever had ...		
25	Severe headache or migraine	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
26	Dizziness	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
27	Difficulty remembering	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
28	Difficulty sleeping	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
29	Anxiety/Nervousness	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
30	Numbness	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
31	Substance use (other than alcohol and tobacco)	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
32	Excessive alcohol problems	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
33	Seizures/blackouts	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
GASTROINTESTINAL		
In the past 1 year, have you ever had ...		
34	Stomach problems/ulcers	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐

35	Bleeding	0 [] 1 [] 2 [] 3 [] 4 []
36	Any kind of liver disease	0 [] 1 [] 2 [] 3 [] 4 []

I would like to ask you about symptoms that you have had recently and in the past one year. Kindly answer them as accurately as you can. Thank you for your time!

KEY 0 = Never 1=Once in the year 2= twice in the year 3= every quarter 4= throughout the year

37	Nausea	0 [] 1 [] 2 [] 3 [] 4 []
38	Vomiting	0 [] 1 [] 2 [] 3 [] 4 []
39	Abdominal pain	0 [] 1 [] 2 [] 3 [] 4 []
40	Diarrhoea	0 [] 1 [] 2 [] 3 [] 4 []
41	Yellowing of the eyes	0 [] 1 [] 2 [] 3 [] 4 []

GENITOURINARY

In the past 1 year, have you :

42	Experienced frequent urination especially during the day?	0 [] 1 [] 2 [] 3 [] 4 []
43	Passed excessive amounts of urine?	0 [] 1 [] 2 [] 3 [] 4 []
44	Experienced painful urination?	0 [] 1 [] 2 [] 3 [] 4 []
45	Been urinating excessively at night?	0 [] 1 [] 2 [] 3 [] 4 []
46	Passed blood stained urine?	0 [] 1 [] 2 [] 3 [] 4 []
47	Been told you have a kidney disease/problem	0 [] 1 [] 2 [] 3 [] 4 []

ENDOCRINE

In the past 1 year, have you :

48	Gained weight excessively	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
49	Lost weight excessively	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
50	Noticed any neck swelling (goitre)	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
HEMATOLOGIC		
In the past 1 year, have you :		
51	Experienced any abnormal bleeding (from the gums, mouth, nose, rectum)	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
52	Been told by your doctor that you are anaemic?	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
53	Experienced lack of energy or fatigue ≥ 3 days?	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
OTHER		
54	Do you smoke?	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
55	Do you take alcohol	0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐
BIOMETRIC AND PERSONAL INFORMATION		
56.	What is your current marital status? (<i>please kindly tick</i>)	Married ☐ Separated ☐ Divorced ☐ Widowed ☐ Never married ☐ Living Partner ☐

57.	What is the highest level of education attained? (please kindly tick)	No Schooling [] Primary [] JHS [] SHS [] Vocational [] Tertiary [] Other (specify)
58.	What is your current occupation?	Nil [] Pensioner [] Farming [] Fishing [] Trader [] Government Worker [] Other (Specify).....
59.	What was your former occupation (if any)?	Nil [] Pensioner [] Farming [] Fishing [] Trader [] Government Worker []

		Other (Specify).....
60	Age	
61	How long have you lived in this community?	
62	Sex	Male [] Female []
63	Height (in meters)	
64	Weight (in Kg)	