

University of Rajshahi

Rajshahi-6205

Bangladesh.

RUCL Institutional Repository

<http://rulrepository.ru.ac.bd>

Department of Chemistry

PhD Thesis

2005

Studies on Arsenic Accumulation in Agricultural Products

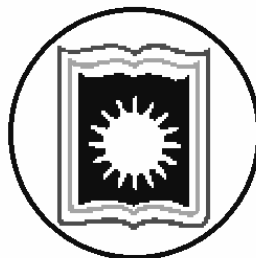
Islam, Md. Asadul

University of Rajshahi

<http://rulrepository.ru.ac.bd/handle/123456789/250>

Copyright to the University of Rajshahi. All rights reserved. Downloaded from RUCL Institutional Repository.

STUDIES ON ARSENIC ACCUMULATION IN AGRICULTURAL PRODUCTS



A THESIS

*SUBMITTED TO THE UNIVERSITY OF RAJSHAHI
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE OF DOCTOR OF PHILOSOPHY*

SUBMITTED BY

MD.ASADUL ISLAM

SESSION: JULY 2005

***MOTIHAR GREEN
JUNE, 2013***

***PHYSICAL RESEARCH LABORATORY
DEPARTMENT OF CHEMISTRY
UNIVERSITY OF RAJSHAHI,
BANGLADESH***

DECLARATION

I do hereby declare that the entire work as embodied in this dissertation towards the partial fulfillment of Ph. D. degree is the result of my own investigation except where due acknowledgement has been given. It has not concurrently been submitted elsewhere for any other degree.

(Md. Asadul Islam)

CERTIFICATE

This is to certify that the thesis entitled “Studies on Arsenic Accumulation in Agricultural Products” submitted by Md Asadul Islam is the outcome of his own research work carried out at the Department of Chemistry, University of Rajshahi, Bangladesh, under my supervision. Any part or whole of the dissertation has not been submitted elsewhere for any other degree.

(Dr. Entazul M. Huque)

Professor

Department of Chemistry

University of Rajshahi

Rajshahi, Bangladesh.

ABSTRACT

Many of the metals are considered as essential nutrients for maintaining sound human health throughout life. But intake of heavy metal contaminated vegetables, rice, meat, fish, drinking water and dust may pose a risk to human health. Arsenic in groundwater and its fate and transport in the environment have become matters of great concern in Bangladesh. Lead and cadmium pollutions are also considered to be global issues. Considering these, two separate studies were carried out to find out the potential ways for remedy of arsenic, cadmium and lead toxicity in vegetables as well as to assess human health risk due to arsenic, cadmium and lead exposures from dietary food products collected from two villages of Rajshahi City Corporation in Bangladesh.

In this investigation, six natural organic fertilizers that the Bangladeshi farmers use frequently are used to assess their potentials towards reduction of arsenic, cadmium and lead toxicity in vegetables plants. These are rotten cow-dung, rotten Dhunca, compost, drain mud, earthworm mud and water hyacinth. The vegetable plants are grown in open field and irrigated with the contaminated water. The concentrations of arsenic, cadmium and lead in the environmental samples were determined by Graphite Furnace - Atomic Absorption Spectroscopy (GF-AAS) technique. To assess human health risk due to consumption of dietary products, drinking water and dust, US Environmental Protection Agency's (USEPA) guidelines were applied.

The overall efficiency of the studied organic fertilizers towards simultaneous reduction of arsenic, cadmium and lead toxicity in vegetables was found as follows: Cow-dung > Drain mud > Rotten Dhuncha > Compost > Earthworm mud > Water hyacinth. Since cow-dung, drain mud, rotten Dhuncha and compost were able to reduce the concentration of arsenic, cadmium and lead in the vegetables simultaneously with respect to control, they could be recommended for remedy of arsenic, cadmium and lead

toxicity in vegetables plants. The observed concentration range of arsenic, cadmium and lead in the studied vegetables were (0.023-0.297), (0.001-0.050) and (0.004-0.783) mg kg⁻¹ on fresh weight basis. Based upon the accumulated concentrations of the concerned metals, the studied vegetables could be arranged in the follow the order: Bitter gourd < Water spinach < Okra < Indian spinach < Amaranth.

In the human health risk assessment investigation, arsenic, cadmium and lead concentration ranges in all the studied diet were found as (0.002-1.146), (0.0003-0.0170) and (0.001-0.056) µg g⁻¹ respectively (fw). Generally the concentrations of the concerned metals in the studied samples follow the order: Dust > Rice > fish > Vegetables > Drinking water. It was found that 99.29% of meal exposure was due to ingestion, while only 0.71% due to inhalation. Sample wise contribution towards average total Hazard Quotient (HQ) values was found as follows: rice> fish>drinking water> vegetables >dust for arsenic, drinking water> rice> fish> vegetables > dust for cadmium and rice> drinking water>fish> vegetables >dust for lead. For the same diet and atmosphere, As, Cd and Pb contribute to 60.79%, 20.89%, 18.32% respectively for children. But for average person, As, Cd and Pb contributes is 78.86%, 20.78% and 0.36% respectively. For senior person, the contribution is 70.15%, 17.50% and 12.38% respectively.

In case of children population sub-group average HI value was 2.215. It reflects that all children are at risk due to consumption of the studied diet. The HI values for Average Person and Senior population sub-group are 0.909 and 0.871 respectively. Hence both population sub-groups are in safe limit due to consumption of the same diet. Comparison among HI values suggests that the population sub-groups should be arranged in following order:

Children > Average Person > Senior.

ACKNOWLEDGEMENTS

All praises and thanks are due to the Almighty Allah for giving me strength, patience and ability to accomplish my research work. I am extremely pleased in expressing my deepest sense of gratitude, profound and sincere appreciation to my supervisor and chairman, Professor **Dr. Entazul M. Huque**, Department of Chemistry, University of Rajshahi for his skillful guidance and hearty assistance, constant encouragement and untiring help throughout the progress of this study, research work and also during the preparation of this thesis paper.

My thanks and appreciations are due to the former Chairman Prof. Dr. M. Samsul Islam, Department of Chemistry, University of Rajshahi for providing me with necessary laboratory facilities for carrying out the research work. I am very grateful to Associate Prof. Dr. M. Nazmul Islam for his generous assistance, guidance, encouragement and invaluable suggestions in carrying out the research work. My thanks and appreciations are also due to Dr. M. Nurul Islam and Dr. M. Monirul Islam for their invaluable suggestions, continuous encouragement and unbounded generosity during the research work.

I accord my hearty reverence to my teachers, Prof. Dr. Md. Azhar Ali, Prof. Dr. Basudeb Kumer Das and Dr. M. A. Mannan for their personal cooperation and sympathy they offered to me during the progress of the research work. My thanks are conveyed to all other honorable teachers Department of Chemistry, University of Rajshahi for their encouragement, brilliant criticism and helpful suggestions.

I gratefully acknowledge Authority of Rajshahi University, Grateful thanks are to Eng. Iftakher Nur and Eng. Emaz Uddin Ahmmed in Central Science Laboratory, University of Rajshahi for determination of metal concentration using GF-AAS.

I gratefully acknowledge Senior Scientific Officer, Soil Science Research Institute, Rajshahi and Mr. A. Jobber, Senior Chemist,

Department of Public Health and Engineering, Rajshahi for determination of metal concentration using GF-AAS and also for the determination of Fecal Coliform Bacteria by filter membrane method.

Many many thanks to my co-worker Md. Rustom Ali (Ph.D. fellow) for his helpful discussion, valuable suggestions, and scholastic guidance. I wish to express my admiration to Niamul, Rehena, Shahadat and also heartily thanks to Md. Nurunnabi Vai and Indrojit Dada who helped me during laboratory work. I am also thankful Md. Nasir uddin (Store keeper) and Md. Nazrul Islam (Deputy Registrar) Department of Chemistry, University of Rajshahi for his occasional help during my research work.

I gratefully acknowledge the authority of Explosives under the Ministry of Power and Mineral resources, Bangladesh for partial financial support and express my appreciation to Mr.Sankar Prosad Pal and all officers and staffs of Department of Explosives, Rajshahi. I am very much grateful to all of my family members for their encouragement, support and interest, in particular, my uncle Ad. Abdul Bari who always supported, encouraged and stimulated me in my whole learning process.

Finally, I would like to express my gratitude to my beloved parents. I owe a life-long debt to my wife Helena Akhter Pervin and child Labiba who continuously encouraged, helped actively and allowed me to continue this research.

June, 2013

The Author

ABBREVIATIONS AND SYMBOLS

A	Absorbance
ACS	American Chemical Society
D _{app}	Applied Dose
AAS	Atomic Absorption Spectroscopy
$\bar{C}$	Average Concentration of Trace Element
ADD	Average Daily Dose
AT	Average Time
BW	Body Weight
DEFRA	Department for the Environment, Food and Rural Affairs(UK)
DPHE	Department of Public Health Engineering (Bangladesh)
DDW	Double Distilled Water
EDL	Electrodeless Discharge Lamp
E	Exposure
ED	Exposure Duration
FL	Flame
FAO	Food and Agriculture Organization
GI	Gastro-intestinal
HI	Hazard Index
HQ	Hazard Quotient
HE's	Highly Exposed Individuals
HCL	Hollow Cathode Lamp
HG	Hydride Generation
IR(t)	Ingestion or Inhalation Rate
IRIS	Integrated Risk Information System
D _{int}	Internal dose
IARC	International Agency for Research on Cancer
LOAEL	Lowest Observed Adverse Effect Level
MADL	Maximum Allowable Daily Level

MADL	Maximum Allowable Limit
MHS	Mercury Hydride System
MDL	Method Detection Limit
MMA	Monomethylarsenic acid
NIST	National Institute of Standards and Technology (USA)
NOAEL	No Observed Adverse Effect Level
n	Number of Observation
ppm	Parts per Million
ppb	Parts per Billion
D _{pot}	Potential dose
QA	Quality Assurance
QC	Quality Control
QTA	Quartz Tube Atomizer
RfC	Reference Concentration
RfD	Reference Dose
SRM	Standard Reference Material
As _T	Total Arsenic Concentration
TSP	Triple Superphosphate
UV-VIS	Ultraviolet and Visible
US EPA	United State Environmental Protection Agency
$\lambda_{\max}$	Wavelength of maximum absorptivity
WHO	World Health Organization
CD	Cow-dung
CM	Compost
DM	Drain mud
EW	Earthworm mud
RD	Rotten Dhuncha
WH	Water hyacinth

CONTENTS

Abstract	i
Acknowledgements	iii
Abbreviations and Symbols	v
Table of Contents	vii
List of Figures	xi
List of Tables	xiv

CHAPTER 1: INTRODUCTION 1-74

1	Introduction	1
1.1	Review of Literature/Recent Studies:	1
1.2	Over view of Arsenic, Lead and Cadmium:	10
1.3	Chemical Properties of Arsenic, Lead and Cadmium:	11
1.4	Principal Compounds of Arsenic, Lead and Cadmium and Their Uses:	13
1.5	Solution Chemistry of Arsenic, Lead and Cadmium:	14
1.6	Sources of arsenic, lead and cadmium:	21
1.7	Worldwide Scenario of Groundwater Contamination:	23
1.8	Occurrence of Arsenic, Lead and Cadmium in Ground water of Bangladesh:	25
1.9	Scale and Magnitude of groundwater contamination in Bangladesh:	29
1.10	Arsenic, Lead and Cadmium in Soil:	30
1.11	Biotransformation:	33
1.12	Toxicity of Arsenic, Lead and Cadmium:	39
1.13	Clinical Manifestation and Treatment of arsenic, lead and cadmium:	42
1.14	Rationale of the study :	52
1.15	Objectives of the study:	54
1.16	Scope of the study:	54
1.17	References	56

CHAPTER 2: EXPERIMENTAL	75-108
2.1	Experiments 76
2.1.1	Materials 76
2.1.2	Standard Stock Solutions 76
2.1.3	Ternary Acid Mixture 78
2.1.4	Other Solutions 78
2.1.5	Other Materials 79
2.1.6	Standard Reference Materials 79
2.2	Instrumentation 79
2.2.1	Graphite Furnace – Atomic Absorption Spectroscopy (GF-AAS): 79
2.2.2	Atomic Absorption Spectrometry: 84
2.2.3	Principle of Atomic Absorption Spectroscopy: 86
2.2.4	UV-VIS Spectroscopy: 89
2.2.5	Theory of Arsenic, Lead and Cadmium determination in supplied samples following 92
2.3	Experimental Procedures 94
2.3.1	Materials 94
2.3.2	Standard Stock Solutions Preparation: 95
2.3.2.1	Stock standard: 95
2.3.2.2	Other solutions: 95
2.4	Methodology: 96
A	Soil Preparation and Irrigation: 96
B	Water Sampling and Analysis: 99
1	Sample collection and preservation: 99
2	Measurement of Physical Parameters: 99
3	Pretreatment of Water Samples 99
C	Plant Tissues Sampling and Analysis: 100
1	Sample Preparation: 100

2	Digestion of Biological Samplings:	100
3	Preparation Stock Solution:	101
4	Analysis of Samples for total arsenic, Lead and Cadmium:	101
D	Soil Sampling and Analysis:	101
1	Sample preparation:	101
2	Digestion of soil samples:	102
3	Preparation of stock solution of samples:	102
4	Analysis of samples for total Arsenic, Lead and Cadmium:	102
E	Fish Sampling, Digestion and Analysis:	102
1	Sample Preparation:	102
2	Digestion, Preparation of Stock Solution and Analysis:	103
F	Dust Sampling, Digestion and Analysis:	103
G	Estimation of Arsenic, Lead and Cadmium Concentrations in Air:	103
2.5	Quality Control	104
1	Cleaning of glassware:	104
2	Analysis of Reagent Blanks:	104
3	Crosschecking of concentration:	104
4	Duplicate and Triplicate Analysis	105
5	Recovery of Known Additions:	105
6	Analysis of Standard Reference Materials:	106
2.6	Statistical Analysis	106
2.6	References	107

CHAPTER 3: USE OF NATURAL ORGANIC FERTILIZERS TO REDUCE OF As, Pb AND Cd POISONING IN VEGETABLE PLANTS 109-148

3.1	Introduction	110
3.2	Experimental Procedures	116
3.2.1	Materials	116
3.2.2	Methods	117
3.3	Results And Discussion	117
3.3.1	Characterization of Cultivated Soil	117
3.3.2	Characterization of Irrigation Water	118
3.3.3	Characterization of Natural Organic Fertilizers	121
3.3.4	Plant Height and Yield	123
3.3.5	Concentration of Arsenic, Lead and Cadmium in	125
3.3.6	Statistical Analyses of Concentrations of Arsenic, Lead and Cadmium in The Vegetables	135
3.3.7	Efficiency of The Fertilizers	137
3.4	Conclusions	142
3.5	References	145

CHAPTER 4: RISK ASSESSMENT TO ARSENIC, 157-209
CADMIUM AND LEAD EXPOSURE.

4.	Introduction	157
4.1	Experimental	153
4.1.1	Materials	153
4.2.2	Methods	154
4.1.3	Results And Discussion	154
4.2.1	Concentrations of Arsenic, Lead and	168
	Cadmium in Environmental Samples:	
4.2.2	Statistical analyses of Arsenic, Lead and	173
	Cadmium in Environmental Samples:	
4.2.3	Individual Contribution from As, Cd and Pb	176
4.3	Risk assessment	198
4.4	Comparison	205
4.5	Conclusion	205
4.6	References	207

CHAPTER 5: RECOMMENDATIONS FOR FUTURE 203-205
WORK

5.1	Recommendations For Future Study	204
	Studies related to remedy of environmental	204
	Studies related to human health risk assessment	205

CHEPTER ONE: INTRODUCTION

Introduction:

1.1 Review of Literature/Recent Studies:

Arsenic is a highly toxic metalloid and its presence in food composites is a matter of concern to the wellbeing of both humans and animals. Some Recent studies have shown that a few of our foodstuffs such as vegetables, meat, fish, corn, cereals, poultry etc. are contaminated with arsenic [1, 2, 3]. It might be due to the presence of high concentrations of soluble arsenic in the soil where the foodstuffs are grown. The groundwater contamination by arsenic is already considered as a serious global environmental problem. It is one of the potential sources for intake of arsenic in the human body. Crops and vegetables grown on the arsenic-contaminated soils can also be a source of arsenic for human beings. Williams et al.[4] and Huq and Naidu [5] reported that Arum, Amaranth and Ipomoea a good arsenic accumulators. They found arsenic levels in Arum and Potato up to 153 and 2.4 mg kg⁻¹ (dry wt.) respectively. They also reported arsenic concentration in irrigation water in affected areas ranged from 0.136 to 0.555 mg L⁻¹. Williams *et al.* [4] from food market-basket survey showed that arsenic accumulation order as Arum stolon > Brinjal > Cucumber > Coriander > Potato > Long-yard Bean > Radish Leaf > Giant Taro (Mankochu) > Vegetable Papaya. Out of 20 countries in different parts of the world, where groundwater arsenic contamination and human sufferings have been reported so far, the magnitude is considered to be maximum in Bangladesh followed by West Bengal, India [6].

A study conducted by the British Geological Survey (BGS) showed that the groundwater from shallow tube-wells, a popular source of drinking water, in 61 out of 64 districts of Bangladesh was contaminated with arsenic [7]. An estimated 29 million people are directly exposed to this contamination and another 85 million are at risk [8]. Nearly 10,000 patients have been identified and a few deaths due to arsenic-related diseases have also been

reported [8-11]. Clearly, Bangladesh is facing probably the largest mass poisoning in history.

About 33 percent of total land of Bangladesh is under irrigation facilities [12] and the lands are irrigated with groundwater from semi-deep or shallow aquifers through tube-wells as well as low-lift pumps. Most of the groundwater used for irrigation in Bangladesh are contaminated with arsenic [13]. The widespread uses of groundwater for irrigation suggested that ingestion of irrigated crops and vegetables could be another major exposure route for arsenic [14-15].

Bhattacharya et al. [16] carried out a study to assess the level of severity of arsenic contamination of the people of highly arsenic affected Nadia district of West Bengal, India. They investigated the concentrations of arsenic in irrigation water, soil, rice, wheat, common vegetables and pulses. Results revealed that the arsenic-contaminated irrigation water ($0.318\text{--}0.643\text{ mg kg}^{-1}$) and soil ($5.70\text{--}9.71\text{ mg kg}^{-1}$) considerably influenced the accumulation of arsenic in rice, pulse and vegetables in the affected area. Arsenic concentrations of irrigation water samples were many fold higher than the WHO recommended permissible limit for drinking water (0.01 mg kgL^{-1}) and FAO permissible limit for irrigation water (0.10 mgL^{-1}). But, the levels of arsenic in soil were lower than the reported global average of 10.0 mg kg^{-1} and was much below the EU recommended maximum acceptable limit for agricultural soil (20.0 mg kg^{-1}) [17].

Arsenic causes serious health hazards to human beings through contamination of groundwater and drinking water. Among the inorganic species, As(III) is considered as the major As species present in natural water system and has higher toxicity than As(V), because it binds to particular chemical groups – sulfhydryl groups – found on proteins [18]. The epidemiological studies show that chronic As poisoning can cause

serious health hazards including cancer, hyperkeratosis, restrictive lung disease, peripheral vascular disease, gangrene, diabetes mellitus, hypertension and ischemic heart disease [19-20]. Arsenic seems to be a cancer promoter rather than a cancer initiator [21]. The World Health Organization (WHO) ranked this calamity as the largest poisoning of a population in history [22].

Uptake of arsenic by crop plants grown in soils contaminated with high concentrations of As and irrigated with As contaminated water has been reported by Abedin et al. [23]. Das et al. [24] reported that the concentrations of arsenic in some vegetables of Bangladesh like Kachusak (*Volocasia antiquorum*), potato (*Solanum tuberosum*), and kalmi sak (*Ipomea reptans*) exceed the food safety limits of 1.0 mg kg^{-1} . In the study of arsenic accumulation in the agriculture plants of China and Nepal, Huang et al. [25] and Dahal et al. [26] have found that arsenic accumulated in the range of 0.003-0.116 and < 0.01 -0.55 mg kg^{-1} (dry weight) respectively. In a similar study, significantly lower amount of uptake and translocation of five toxic metals (Cd, Cr, Pb, As and Hg) in the rice grain compared with the uptake in the rice straw and root parts have been reported by Liu et al. [27]. Higher amount of arsenic was reported to accumulate in the root of the rice plants as compared with other parts [28, 29, 16]. Studies [30-31] have shown that the contribution of As pollution to human health through food chain is many times greater than that of drinking water. Presence of As in vegetables and tubers crops and its translocation to the edible parts were observed to vary with crops [32] and even among the cultivars of the same crop [33]. Arsenic contamination in rainy season rice is not much of a problem due to its rain fed cultivation. But winter and summer crops are mainly cultivated through irrigation using ground water by shallow tube-well which in turn creates lot of problems with regard to As uptake. Lauren and Duxbury

[34] estimated of arsenic consumption from only rice and water indicating that arsenic from rice is an important source of exposure in the Bangladesh food system. They proposed that management strategies need to be developed to reduce arsenic uptake by rice. Despite their expectation that aerobic cultivation practices would reduce arsenic uptake by rice, results from experiments with permanent raised beds and System of Rice Intensification (SRI) have not shown differences in arsenic concentration to date (Fig-1.1).

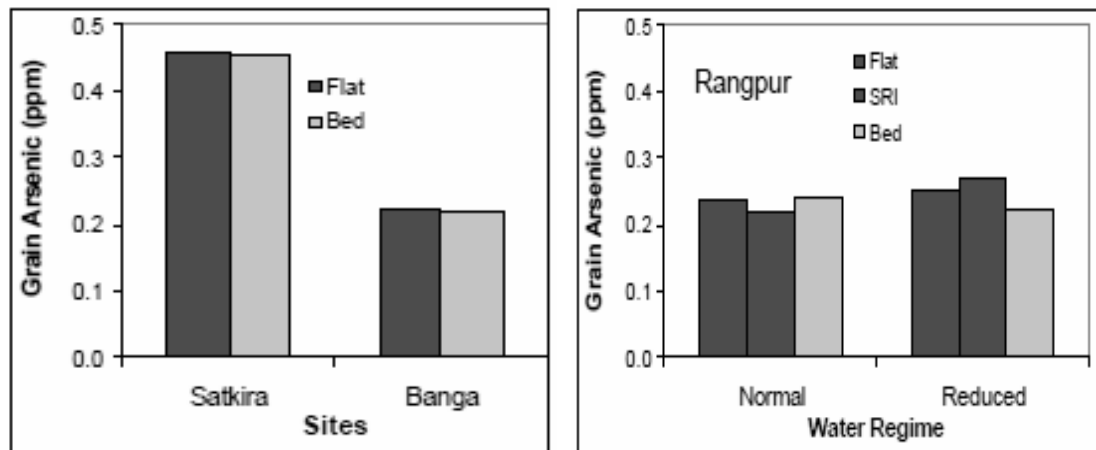


Fig.1.1 Reduce arsenic uptake by rice in aerobic cultivation practice.

Nevertheless, on a highly contaminated soil (68 mg/kg) Xie and Huang [35] observed a 22% increase in yield and a 24% decrease in rice grain arsenic after furrow drainage and maintenance of moist soil.

Rose et al. [36] furnished an avoidance approach. They identified in their study that 24% of total irrigated boro rice in Bangladesh is grown in areas where groundwater arsenic is greater than the $50 \mu\text{g L}^{-1}$, Bangladeshi standard. Many of the areas with high groundwater arsenic ($100\text{-}200 \mu\text{g L}^{-1}$; $>200 \mu\text{g L}^{-1}$) are found in the central part of the country near the Padma and Meghna rivers with ample surface water. Development of more irrigation schemes with these surface water sources could provide a clean substitute for contaminated groundwater and avoid further

degradation of soils in these areas. The recent work of Liu and colleagues [37] is an important contribution to the central part of Bangladesh. They demonstrated that differences in arsenic uptake between rice varieties are influenced by iron plaque formation and regulated by plant phosphorus status.

In most parts of rural Cambodia, groundwater is used for bathing, cooking, washing and drinking and is used also for crops irrigation. It is the main source of water among the rural population, after river [38]. The researches have shown that arsenic accumulation in plants depends on the plant species, soil composition, planting season, geographical location and growing method.

Davin et al. [39] investigated arsenic accumulation in crops (fruits and vegetables), particularly native species grown in arsenic-contaminated areas of Cambodia. The results of their study would be vital in deciding what crops can be grown in arsenic-contaminated areas, or what cultivation method is needed to reduce arsenic accumulation. During the last decades, the increasing demand of food safety has stimulated research regarding the risk associated with consumption of food stuffs contaminated by pesticides, heavy metals and/or toxins [40].

Heavy metals constitute a variety of groups that vary in their chemical properties and biological functions. Heavy metals are kept under environmental pollutant category due to their toxic effects in plants, human and food. Arsenic (As), Cadmium (Cd), Lead (Pb) and Mercury (Hg) is cumulative poisons. These heavy metals are persistence and not metabolized in other intermediate compounds and do not easily break down in environment. These metals are accumulating in food chain through uptake at primary producer level and then through consumption at consumer level. However, subsequent metal contamination discovered by testing water necessitated mitigation programs [41]. Like trace

elements, some heavy metals (e.g. copper, selenium, zinc) are essential to maintain the metabolism of the human body. However, at higher concentrations they can lead to poisoning [42]. Poisoning of heavy metal toxicity varies according to their oxidation state, solubility and many different inorganic and organic forms. The implication associated with heavy metal contamination is of great concern, particularly in agricultural production systems. These metals can pose a significant health risk to humans, particularly at elevated concentration above the very low body requirements [43]. Li and Tian [44] analyzed human health risk caused by heavy metals in a water source. They found the order of health risky gene toxic substances as $\text{Cr}^{6+} > \text{As} > \text{Cd}$ and that of body toxic substances as $\text{Pb} > \text{Cu} > \text{Hg}$.

Heavy metals are an important source of food contamination and health hazard. The main threats to human health are associated with exposure to arsenic, cadmium, lead, mercury and copper. Absorption of heavy metals through food has been shown to have serious consequences on health and thereby economic development associated with a decline in labor productivity as well as the direct cost of treating illnesses such as kidney disease, damage to the nervous system, diminished intellectual capacity, heart disease, gastrointestinal disease, bone fracture, cancer and death [45]. Trace elements are very harmful because of their no biodegradable nature, long biological half-lives, and their potential to accumulate in different body parts. High concentration of trace elements (Cu, Cd and Pb) in vegetables and fruits were related to prevalence of upper gastrointestinal cancer high [46]. The International Agency for Research on Cancer (IARC) classified lead as a possible human carcinogen in 1987 [47]. Lead (Pb) is a physiologic and neurological toxin that can affect almost every organ or system in the human body. It can reduce cognitive development and intellectual performance in children and damage

kidneys and the reproductive system [48]. Cadmium (Cd) accumulates in kidneys, where it damages filtering mechanisms. It also causes diarrhoea, stomach pains and severe vomiting, bone fracture, reproductive failure and possibly even infertility, damage to the central nervous system, damage to the immune system, psychological disorders and possibly DNA damage.

Singh *et al.* in their study [49] for risk assessment of heavy metal toxicity found that waste water irrigation led to the accumulation of heavy metals in soil and consequently into the vegetables. Heavy metal concentrations varied among the test vegetables which reflect the difference in their uptake capabilities and their further translocation to edible portion of the plants. Consequently, concentrations of Cd, Pb and Ni have crossed the safe limits for human consumption in all the vegetables. Percent contribution of fruit vegetables to daily human intake for Cu, Ni, Pb and Cr was higher than that of leafy vegetables. Target hazard quotient of heavy metals also suggests that the test vegetables had potential for human health risk due to consumption of plants grown in the area having long term use of treated and untreated waste water for irrigation. Consumption of these vegetables with elevated levels of heavy metals may lead to the high level of body accumulation leading to related health disorders.

Dietary exposure to heavy metals, namely Cadmium (Cd), Lead (pb), Zinc (Zn), Copper (Cu) has been identified as a risk to human health through the consumption of vegetable crops [50]. Heavy metals are given special attention throughout the globe due to their toxic and mutagenic effects even at very low concentration [51]. An exploratory study was carried out by Khan *et al.* [52] and they measured arsenic and cadmium in foods from Matlab, a rural area in Bangladesh, that is extensively affected by arsenic and the economy is agriculture-based. Results revealed that the

concentration of arsenic of cooking rice is reduced than that of raw rice. In contrast, concentration of cadmium did not decrease in cooked rice after discarding water. Compared to raw vegetables (e.g. arum), concentration of arsenic increased significantly ($p=0.024$) when cooked with arsenic-contaminated water. However, water generally not discarded when cooking vegetables to avoid loss of micronutrients consequently retains arsenic. They also showed in their study that arsenic and cadmium have entered the food-chain of Bangladesh, and the cooking practices influence the concentration of arsenic but not of cadmium in cooked food (Fig-1.2) [52].

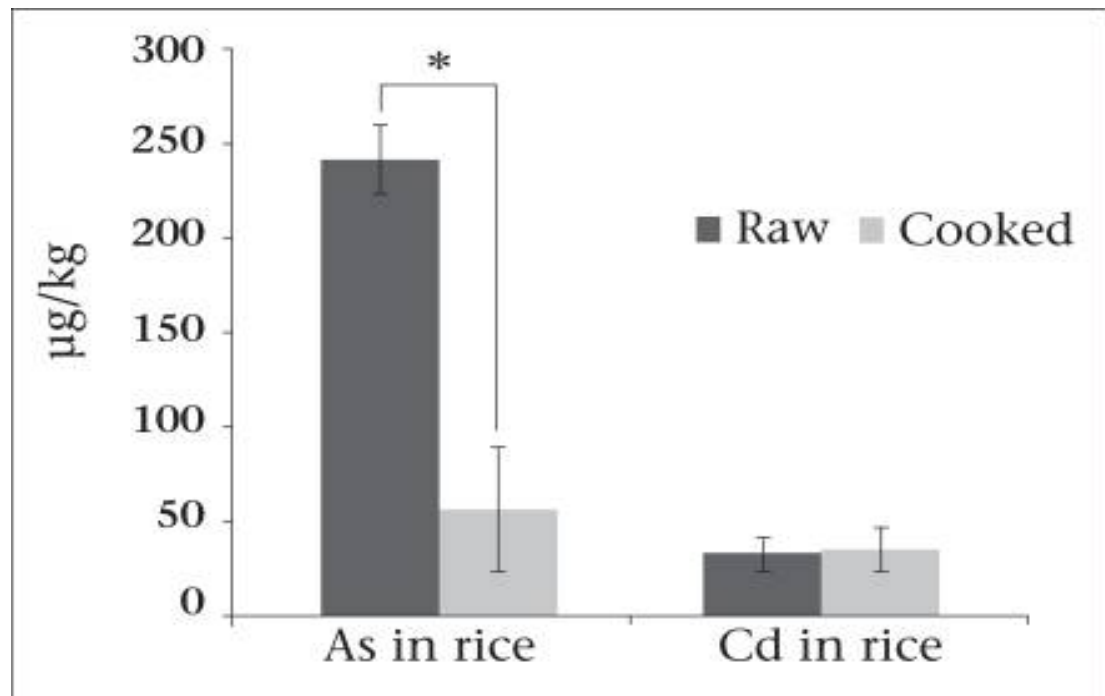


Fig.1.2 Concentrations of arsenic and cadmium in raw and cooked rice.

Most of the vegetables contain more than 80 percent water. Dietary plants also contain hundreds of antioxidants. Heavy metal contamination of the food items is one of the most important aspects of food quality assurance [53-55]. Saha and Zaman worked on concentration of selected toxic

metals in groundwater and some cereals grown in Shibganj area of Chapai Nawabganj, Rajshahi, Bangladesh [56]. This study is an attempt to assess the extent of toxic metals, including Pb, Cd, Cr, Mn and As in groundwater and some cereals collected from Shibganj area of Chapai Nawabganj, Rajshahi, Bangladesh. The chemical results of groundwater show that the mean concentrations of Pb, Cd and Mn exceed the values of WHO, USEPA as well as Bangladesh Drinking Water Standards. However, As content is within the permissible limits in terms of Bangladesh Drinking Water Standards, but exceeds the WHO and USEPA values. Cr is in negligible quantity in the examined groundwater samples. Results revealed that wide variation in toxic metal content among the three cereal samples. In general, the mean toxic metal concentrations in cereals (rice, wheat and arid bean) are lower than the respective established safe limits for these elements, except for lead concentration in all samples.

Emissions of heavy metals from the industries and vehicles may be deposited on the vegetable surfaces during their production, transport and marketing. Jassir et al. [57] have reported elevated levels of heavy metals in vegetables sold in the markets at Riyadh city in Saudi Arabia due to atmospheric deposition.

Heavy metals are non biodegradable and persistent environmental contaminants, which may be deposited on surface soil and then absorbed into the tissues of vegetables [57]. Plants may adsorb heavy metals from the deposits on the parts of the plants exposed to the polluted air and contaminated soils [50, 57, 58, 59, 60]. Heavy metal contamination of vegetables may also occur due to irrigation with contaminated water [61-62] and emission of heavy metals from the industries and vehicles during

transport and marketing, the addition of fertilizers and metal based pesticides, the harvesting process, storage and/or at the point of sale [63]. To our knowledge most of the researches were done by collecting samples from sources like households, market-basket survey etc. Hence we have chosen land field in order to carry out research work by using model water, contaminated ground water and natural organic fertilizers.

With this background information, the present study was undertaken to evaluate the arsenic, lead and cadmium contamination in edible parts of plants and relative pattern of arsenic, lead and cadmium accumulated by selected vegetables (Bitter gourd, Indian spinach, Water spinach, Red amaranth and Okra) grown in preferred field in Puthia, Rajshahi, Bangladesh. At the same time, to investigate the impacts of natural organic fertilizers towards reduction of As, Pb and Cd toxicity in vegetable plants and also to find out the potential ways for remedy of As, Pb and Cd toxicity in human food chain and to assess health risks due to As, Pb and Cd exposures from dietary food products.

1.2 Over view of Arsenic, Lead and Cadmium:

Arsenic (As) is an element of group 15 and period 4 of the periodic table. Arsenic naturally occurs in many minerals, usually in conjunction with sulfur and metals, and also as a pure elemental crystal. Arsenic has some properties of metal such as it is electropositive, has high density and relatively low specific electrical resistivity, forms compounds with O, Cl and S, and also unstable hydride (AsH_3). Arsenic also resembles with non-metals in some properties like it is brittle, its oxides such as As_2O_3 and As_2O_5 are acidic, etc. Hence, it is classified chemically as a metalloid. Elemental arsenic is an α -crystalline metallic form. In compounds, arsenic typically exists in one of three oxidation states -3, +3 and +5 [64].

Lead (Pb) is the heaviest member of the carbon family which remains in Group 14 and period 6 of the periodic table. But it behaves very differently from carbon. Lead is one of only a few elements known to ancient peoples, e.g. a small statue found in Egypt. Freshly cast lead is silvery in colour, but quickly turns dull grey to bluish grey, as its surface oxidizes in the presence of air. In compounds, lead typically exists in one of two oxidation states +2 and +4 [65].

Cadmium (Cd) in its purest form, is a soft lustrous, silver-white, ductile, malleable metal. It is situated in groups 12 and period 5 in the periodic table. Cadmium and its congeners are not always considered transition metals, in that they do not have partly filled d or f electron shells in the elemental or common oxidation states. Cadmium is much less abundant in the earth's crust than zinc. For its ductile and malleable properties, it is easily cut with a knife. It resembles with zinc in many of its physical properties. In compounds, cadmium typically exists in one of two oxidation states +1 and +2 [66].

Table 1.1. Some notable properties of arsenic, lead and cadmium:

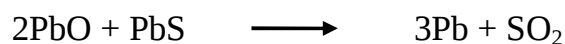
Property	Unit	Arsenic	Cadmium	Lead	Remark
Atomic number		33	48	82	
Atomic mass	g mol ⁻¹	74.922	112.411	207.200	
Electronic configuration		[Ar]3d ¹⁰ 4s ² 4p ³	[Kr]4d ¹⁰ 5s ²	[Xe]4f ¹⁴ 5d ¹⁰ 6s ² 6p ²	Ground state
Oxidation state (stable)		-3,0, (+3),+5	0,+1,(+2)	0,(+2),+4	() represents Stable O.S.
Metallic radius	pm	139	151	154	
Ionic radius	pm	69 (+3)	109 (+2)	132(+2)	
Melting point	⁰ C	816*	321	327.46	*(α form)
Boiling point	⁰ C	615*	767	749	*(α form)
Latent heat of fusion	kJK ⁻¹ mol ⁻¹	27.74	6.192	4.77	

Latent heat of sublimation	$\text{kJK}^{-1}\text{mol}^{-1}$	31.97		179.5	
Specific heat	$\text{Jg}^{-1}\text{k}^{-1}$	0.33	26.02		
Cristal shape		Rhombohedral	hcp	Cubic	
Density	gcm^{-1}	2.03	8.70	11.34 gcm^{-3}	At 20°C
Ionization enthalpy	Kjmol^{-1}	947	866	715	1^{st}
Hardness		3.5	2.0	-	Mohs' scale

1.3 Chemical Properties of Arsenic, Lead and Cadmium:

When heated in air, arsenic combines with oxygen to form arsenic trioxide

$[4\text{As(s)} + 3\text{O}_2(\text{g}) \longrightarrow 2\text{As}_2\text{O}_3(\text{s})]$. Its oxides such as As_2O_3 and As_2O_5 are acidic. Arsenic does not dissolve in water or in most of cold acids. But It reacts with some hot acids to form arsenous acid (H_3AsO_3) or arsenic acid (H_3AsO_4) [67]. Like metal chloride, AsCl_3 undergoes hydrolysis reaction partially. Lead (Pb) is a moderately active metal in water and in most cold acids. It reacts more rapidly with hot acids. It does not react with oxygen in the air readily and does not burn. Various oxidized forms of lead are easily reduced to the metal, e.g.



Lead is not attacked by sulfuric or hydrochloric acids. But it dissolves in nitric acid as follows:



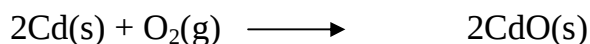
When Pb is heated with nitrates of alkali metals, metallic lead oxidizes to form PbO (also known as litharge), leaving the corresponding alkali nitrite. It is soluble in nitric and acetic acids, from which solutions it is possible to precipitate halide, sulfate, chromate, carbonate (PbCO_3), and basic carbonate salts of lead $[\text{Pb}_3(\text{OH})_2(\text{CO}_3)_2]$. Its sulfide can also be precipitated from acetate solutions. These salts are all poorly soluble in water [67].

Lead dioxide (PbO_2), a powerful oxidizing agent, dissolves in alkali hydroxide solutions to form the corresponding plumbates [68].

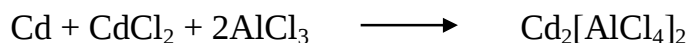


Lead also has an oxide with mixed +2 and +4 oxidation states, red lead (Pb_3O_4), also known as minium. Lead readily forms an equi-molar Pb(OH)_2 with sodium metal that reacts with alkyl halides to form organo-metallic compounds of lead such as tetraethyl lead.

Cadmium (Cd) reacts slowly with oxygen in moist air at room temperatures, forming cadmium oxide [69]. Although cadmium usually has an oxidation state of +2, it also exists in the +1 state. Cadmium is a moderately active metal in water and in most cold acids. It dissolves slowly. It reacts more rapidly with hot acids. Cadmium burns in air to form brown amorphous cadmium (II) oxide (CdO); the crystalline form of this compound is a dark red which changes color when heated, similar to zinc oxide.



Hydrochloric acid, sulfuric acid and nitric acid dissolve cadmium by forming cadmium chloride (CdCl_2), cadmium sulfate (CdSO_4), or cadmium nitrate [$\text{Cd(NO}_3)_2$] respectively. The oxidation state +1 can be attained by dissolving cadmium in a mixture of cadmium chloride and aluminium chloride, forming the Cd_2^{2+} cation, which is similar to the Hg_2^{2+} cation in mercury (I) chloride [69].



It does not react with water. It also reacts with halides to form CdX_2 .

1.4 Principal Compounds of Arsenic, Lead and Cadmium and Their Uses:

Arsenic has a fascinating history as a healer and killer. Early physicians, recommended arsenic for the treatment of some diseases, e.g., arsephenamine was used to treat syphilis. Arsenic in its inorganic form is

used for the production of pressure-treated wood. Arsenic is also used in the leather tanning industry as a preservative. It is widely used as a weed killer and also used as an insecticide. Some binary compounds of arsenic are halides, oxides, hydrides, sulfides, telluride's and selenides. As_2O_3 is widely used laboratory reagent. All these arsenic compounds have been used in different sectors.

A relatively small amount of lead is used to make lead compounds. Although the amount of lead is small, the variety of uses for these compounds is large. For examples, some important lead compounds such as lead acetate ($\text{Pb}(\text{C}_2\text{H}_3\text{O}_2)_2$) and Lead naphthenate ($\text{Pb}(\text{C}_7\text{H}_{12}\text{O}_2)$) are used in insecticides, waterproofing, varnishes, dyeing of cloth, production of gold, hair dye, wood preservative, paint and varnish drier. Lead antimonite ($\text{Pb}_3(\text{SbO}_4)_2$) is used as staining of glass, porcelain and other ceramics. Lead azide ($\text{Pb}(\text{N}_3)_2$) is used as a “primer” for high explosives. Lead chromate (“chrome yellow”, PbCrO_4) as industrial paints (use restricted by law). Lead fluoride (PbF_2) is used to make lasers, specialized optical glasses. Lead iodide (PbI_2) is used in photography and cloud seeding to produce rain. Lead naphthenate ($\text{Pb}(\text{C}_7\text{H}_{12}\text{O}_2)$) is utilized for wood preservative, insecticide, additive for lubricating oil, paint and varnish drier. Lead phosphite ($2\text{PbO}.\text{PbHPO}_3$) is used to screen out ultraviolet radiation in plastics and paints. Lead stearate $\text{Pb}(\text{C}_{18}\text{H}_{35}\text{O}_2)_2$ is commonly used to make soaps, greases, waxes, paints, lubricant, drier for paints and varnishes. Lead telluride (PbTe) is used to make semiconductors, photoconductors, and other electronic equipment [70].



Realgar (As)
anglesite

A small ball of cadmium metal with a coin

Lead ore- galena and

Fig-1.3: Picture of arsenic, cadmium and lead ores mineral.

A popular use of cadmium compounds is as coloring agents. The two compounds most commonly used are cadmium sulfide (CdS) and cadmium selenide (CdSe). The sulfide is yellow, orange, or brown, while the selenide is red. These compounds are used to color paints and plastics. There is concern about possible environmental effects of using cadmium. However, no satisfactory substitutes have been found [71]. Cadmium salts of organic acids, generally cadmium laurate or cadmium stearate in combination with barium sulfate were widely used in the past as heat and light stabilizers for flexible polyvinyl.

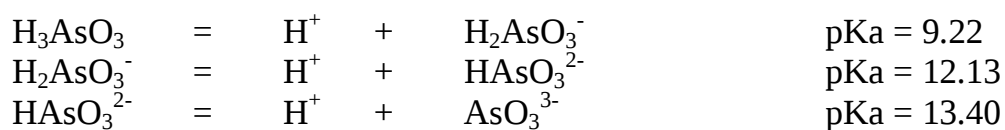
1.5 Solution Chemistry of Arsenic, Lead and Cadmium:

Most of the chemical reactions take place in aqueous solution. The reacting substance should be dispersed condition in a suitable medium before appreciable interaction taking place. Arsenic chemistry in aqueous systems is quite complex because it can exist in four oxidation states. The valence and nature of its species depends on the oxidation-reduction conditions and pH of the water. The chemistry of arsenic with reference to acid-base, oxidation-reduction, adsorption-desorption and precipitation-dissolution are as follows:

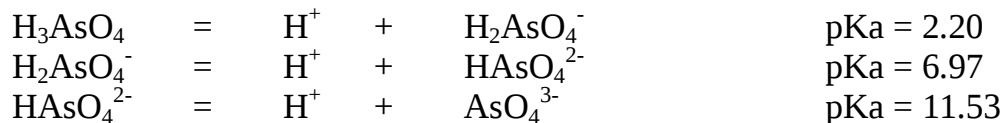
Acid-Base Chemistry: Arsenic is usually found as an anion with acid characteristics in the trivalent (+3) and pentavalent (+5) forms. The pentavalent (+5) arsenate species are H_3AsO_4 , H_2AsO_4^- , HAsO_4^{2-} and AsO_4^{3-} . The trivalent (+3) arsenite species include H_3AsO_3 , H_2AsO_3^- , HAsO_3^{2-} and AsO_3^{3-} . The pentavalent arsenic species are predominant and thermodynamically stable in the oxygen-rich aerobic environments such as surface water, whereas trivalent arsenic species are predominant in moderately reducing anaerobic environments such as groundwater [72].

The oxides of both arsenic (III) and arsenic (V) are soluble in water. The dissolution implies direct reaction with water, hydration, where the oxides behave like non-metals and exhibit acidic character. Arsenic (III) forms arsenious acid (also called arsonic acid), while arsenic (V) forms arsenic acid (also called arsinic acid). The two acids dissociate to form respective arsenite and arsenate ions as shown in the following reactions [73]:

Dissociation of arsenious acid:



Dissociation of arsenic acid:



The predominance diagram of arsenic species as a function of pH is shown in Figure 1.4 [73].

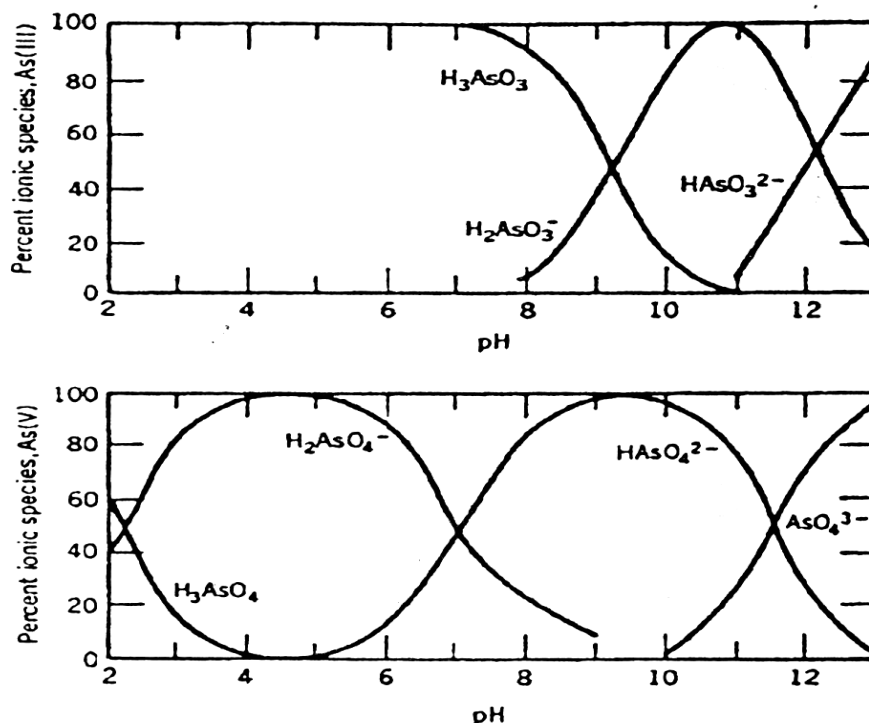


Figure 1.4. Predominance diagram of As(III) and As(V) as a function of pH. [73]

From the above figure it is evident that arsenic acid is stronger than arsenious acid. Within the pH range of natural water (particularly groundwater) (pH 6-9), the trivalent inorganic arsenic is found as non-dissociated arsenious acid (H_3AsO_3); while pentavalent arsenic is primarily found as the ionized dihydrogen arsenate ($H_2AsO_4^-$) and monohydrogen arsenate ($HAsO_4^{2-}$). The stability and the predominance of the arsenic species in the aquatic environment at different pH range [74] is shown in the following Table 1.2.

Table 1.2. Stability and predominance of arsenic species at different pH range.

pH range	0 – 9	10 – 12	13	14
As (III)	H_3AsO_3	$H_2AsO_3^-$	$HAsO_3^{2-}$	AsO_3^{3-}
pH range	0 – 2	1– 6	7 – 11	12 – 14
As (V)	H_3AsO_4	$H_2AsO_4^-$	$HAsO_4^{2-}$	AsO_4^{3-}

Redox Chemistry: Arsenic is a redox sensitive element. The mobility of arsenic is controlled, in large part, by oxidation-

reduction (redox) transformations. The valence in which arsenic exists is related to both pH and redox potential, E_h . The hypothetical electron activity at equilibrium, pE , is used interchangeably with E_h . These parameters are related by $pE = (F/2.3RT) E_h$, where T , F and R are temperature, the Farady and gas constant respectively. Thus at 25°C , $2.3RT/F = 0.059 \text{ V}$ and $pE = E_h / 0.059$. The equation linking arsenic speciation to pH and pE are readily available, but E_h versus pH diagrams (Fig. 1.4), which indicate the predominant soluble species and relevant solids, are the most convenient way of presenting the information.

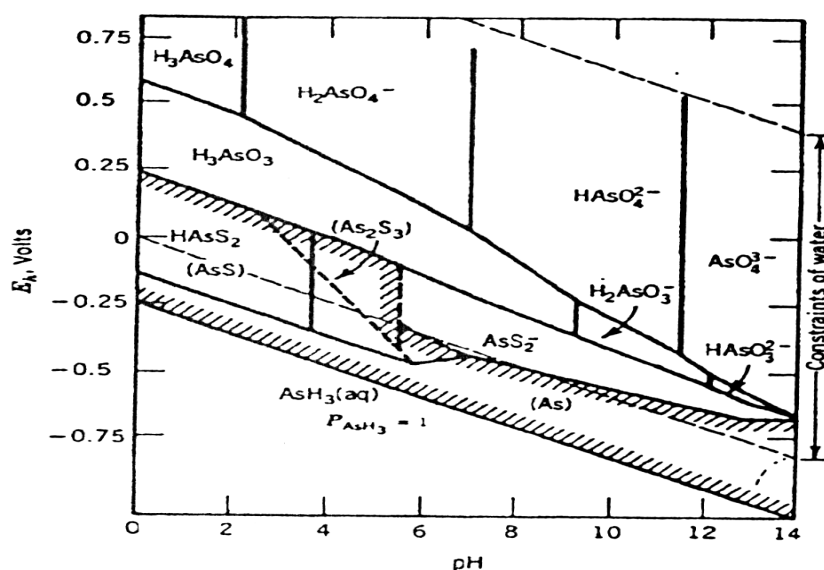


Figure 1.5. The E_h -pH diagram for As at 25°C and 1 atm with total arsenic 10^{-5} M and total sulfur 10^{-3} M . Solid species are enclosed in parenthesis in cross-hatched area, which indicates solubility less than $10^{-5.3} \text{ M}$ [73].

The E_h -pH diagram for arsenic (total conc. 10^{-5} M) in a system containing oxygen, H_2O and sulfur (total conc. 10^{-3} M) is shown in Figure 1.3 [10]. The diagram represents equilibrium conditions of arsenic under various redox potentials. Well-aerated surface waters would tend to induce high E_h values and, therefore, any arsenic present should be in the arsenate [As(V)] form. Mildly reducing conditions, which can be found in groundwater, should produce

arsenite [As(III)]. By determining the pH and E_h of water, it is possible to determine which species of arsenic will be prevalent.

Arsenate is dominant in oxygenated water while arsenite is dominant in non-oxygenated water. Although thermodynamics can provide prediction of possible changes in a given non-equilibrium conditions, they give no insight to the rate at which those changes will occur. While As(III) and As(V) acid-base reactions can be assumed to occur instantaneously, changes between oxidation states require indeterminate time in natural waters. For instance, the conversion of As(III) to As(V) in oxygenated water is thermodynamically favored, yet the transformation takes days, weeks or months depending on the specific conditions. The reduction of As(V) to As(III) is similarly kinetically constrained. This is the reason why As(V) can be found in some anoxic waters [75]. This process is however known to be facilitated through catalysis and require bacterial mediation. In strongly acidic or alkaline solutions, the presence of copper salts, carbon, certain catalysts and higher temperatures can increase the arsenic oxidation rate [76]. Catalytic oxidation of arsenic can be achieved by powdered active carbon and dissolved oxygen in stirred reactors. The rate of oxidation can be described by a first-order equation.

Adsorption – Desorption Reactions: Adsorption-desorption reactions are due to solid-solution interactions and are very important in determining the mobility of arsenic. Both arsenate and arsenite adsorb on surfaces of a wide range of solids including iron, aluminum and manganese oxides, and clay materials. The extent of adsorption is strongly dependent on the arsenic speciation, redox status, arsenic concentration, pH and the concentration of competing anions such as PO_4^{3-} .

Adsorption-desorption of arsenic onto iron oxide surfaces are important controlling reactions in the subsurface because iron oxides

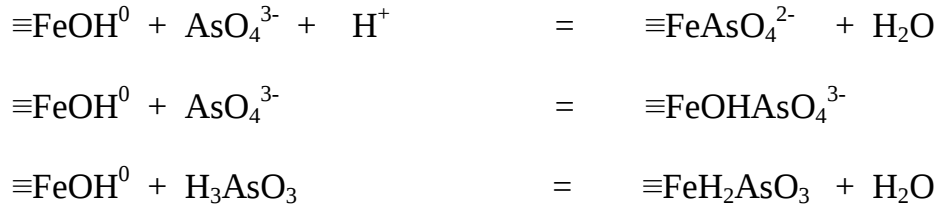
are widespread in the hydro-geologic environment as coating on other solids. As(III) sorption is practically independent of pH over the range of pH of interest in groundwater (pH 6-9) whereas As(V) sorption declines rapidly above pH 8.5 or so. Therefore, while As(V) is much more strongly bound at low pH, at about pH 8-9 or above, As(III) binding can be greater than As(V) binding under similar conditions. This rapid decline in As(V) adsorption therefore occurs in a pH range that is found in groundwater and is one factor that can lead to high As concentrations in groundwater. The pH dependence of As(V) adsorption-desorption appears to be related to the change in net charge on iron-oxide surface with pH.

The situation changes dramatically in the presence of phosphate. Phosphate competes very effectively with As(V) for sorption sites and reduces As(V) sorption greatly [77]. At low As(V) concentrations, the high As/P molar ratios mean that P outcompetes As very effectively and is calculated to reduce the sorption by one to three or more orders of magnitude. The isotherm is now linear at low As concentrations and is close to being a Langmuir isotherm overall. This is because the surface potential is now governed by the phosphate sorption and is essentially independent of the As(V) concentration over much of the isotherm.

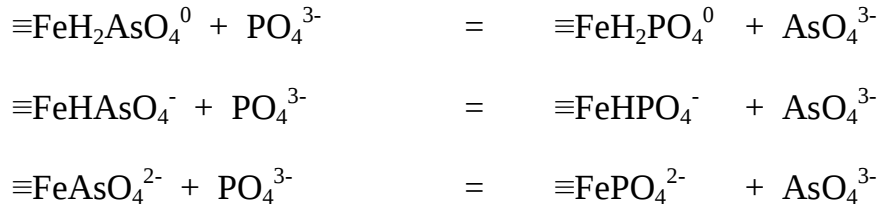
Adsorption-desorption reactions on solid surfaces are usually traced using surface complexation model (SCM). Adsorption-desorption reactions of arsenate and arsenite on hydrous ferric oxide using the generalized two-layer model [78] are shown below:

Adsorption reactions:

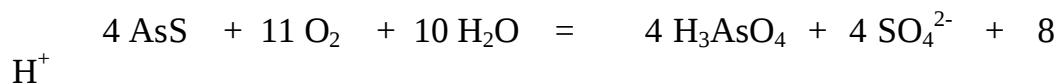
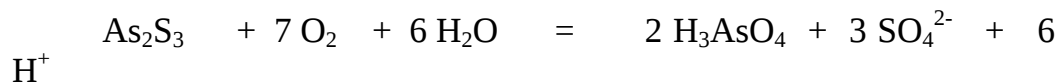
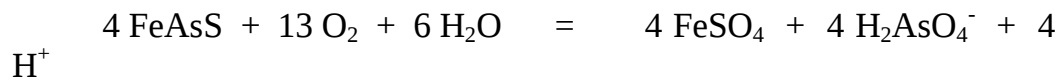
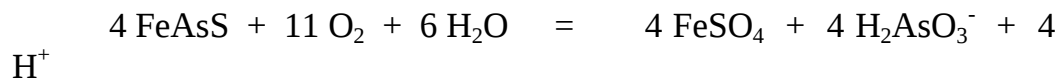




Desorption reactions:

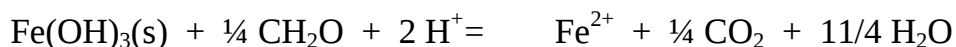


Precipitation – Dissolution Reactions: Precipitation-dissolution reactions are important controlling mobility of arsenic in the subsurface. In Bangladesh, reductive dissolution of iron oxyhydroxides and consequent release of adsorbed arsenic could be important mechanism of arsenic mobilization in the subsurface. Oxidative dissolution reactions of FeAsS, As₂S₃ and AsS are also reported [79]:



High concentrations of arsenic often are associated with redox sensitive iron oxides and sulfide minerals. Iron oxides are frequently dissolved under reducing conditions (e.g., in the presence of organic matter) [80], but often precipitate under oxidizing conditions. Sulfide minerals generally are unstable under oxidizing

conditions, but may precipitate under reducing conditions (e.g., precipitation of As_2S_3).



Lead has three oxidation states that should be considered when looking at aqueous reactions, IV, II and 0. $\text{Pb}(\text{IV})$ is so insoluble that the species it forms do not need to be considered in the aqueous phase. $\text{Pb}(\text{II})$ dissolves in the aqueous phase to form six compounds including Pb^{2+} , $\text{Pb}(\text{OH})_2$, PbCO_3 , $\text{Pb}_3(\text{CO}_3)_2(\text{OH})_2$ and $\text{Pb}(\text{OH})_3^-$. Finally, $\text{Pb}(0)$ as with $\text{Pb}(\text{IV})$ has a negligibly small dissolved concentration [81].

Solution chemistry of lead depends on its oxidation state, pH of the solution and the equilibrium solubility of the solution.

The dissolution rates of both lead carbonate and lead phosphate increase with decreasing pH. Dissolution rates of lead carbonates and phosphates are comparable to those of calcium carbonates and phosphates. Dissolution rates are lower at lower temperatures.

In solution cadmium exists either dissolved or as part of insoluble complexes. Soluble cadmium is quite mobile in water and soil solution. Solubility is promoted by acidic condition (low pH value). Transformation processes for cadmium in soil solution are mediated by sorption form and desorption to water and include precipitation, dissolution, complexation and ion exchange. Low pH of soil solution tends to increase the availability of cadmium. Partitioning into the sediment is enhanced by precipitation and sorption to mineral surfaces and organic materials as well as by action of sediment bacteria. The

levels in the sediment tend to be at least an order of magnitude higher than in the overlying water column. High levels of organic material in the water promote formation of organic complexes with cadmium which are poorly soluble. Also in reducing environment, cadmium may precipitate out as cadmium sulfide.

1.6 Sources of arsenic, lead and cadmium:

The natural sources of arsenic, lead and cadmium include minerals, rocks, sediments, soils and the atmosphere. A number of sources have been observed as a major constituent in minerals, including elemental arsenic, lead, cadmium and their sulphides and oxides. A list of some of the most common arsenic, lead and cadmium minerals [77] are given in Table 1.3. Most are ore minerals

Table 1.3. Major arsenic, lead and cadmium minerals occurring in nature.

Mineral	Composition	Occurance
Native arsenic	As	Hydrothermal vein
Nicolite	NiAs	Vein deposits norites
Realgar	AsS	Vein deposits, often associated with orpiment, clays and limestones, also deposits from hot springs
Arsenolite	As ₂ O ₃	Secondary mineral formed by oxidation of arsenopyrite,
Orpiment	As ₂ S ₃	Native arsenic and other As As minerals
Arsenopyrite	FeAsS	Hydrothermal veins, hot springs, volcanic sublimation product
Scorodite	FeAsO ₄ .2H ₂ O	The abundant As mineral, dominantly,

Native lead	Pb	Secondary mineral, smelter wastes ;
Galena	PbS	Native born, brass, Galilean;
Anglesite	PbSO ₄	Mineral galena; Mineral anglesite, <u>Quartz</u> , <u>Djurleite</u> , <u>Covellite</u> , including a bladed microscopic lead sulfate
Cerussite	PbCO ₃	silicate;
Minim	Pb ₃ O ₄	General mineral, silica and quartz;
Chrome yellow	PbCrO ₄	
Mimetite	PbCl ₂ Pb ₃ (AsO ₄) ₂	
Native	Cd	
Cadmium	CdS	
Cadmium	ZnS with Cd	
sulphide		
Sphalerite		

1.7 Worldwide Scenario of Groundwater Contamination:

More than 70 countries are globally affected directly or indirectly with arsenic contamination in drinking water and 38 countries are affected more severely at present. The major affected countries of arsenic poisoning include Argentina, Chile, Mexico, Taiwan, and some parts of the United States.. At the last quarter of 20th century three Asian countries (Bangladesh, China, and India) come to lime light due to their suffering from groundwater arsenic contamination. The major source of arsenic contamination was contaminated hand tube-wells. As of 2010 September, 13 Asian countries are arsenic affected and the level of arsenic contamination in Asian countries is more severe than the rest of the world. In India, flood plains of all the states in Ganga and Brahmaputra rivers are arsenic affected. Access to safe water supply is one of the most important factors of health and socio-economic development [82]. More than 150 million people are affected worldwide by arsenic contamination in 70 countries, out of which 30 million people in India are at risk [80]. Arsenic contaminations in groundwater depend on the type of environments and sources under which they

are developed. Arsenic is toxic in nature and the excess quantity of its use in drinking water leads to several health hazards

Drinking water can also have dangerously high levels of lead. As many as one of five Americans are exposed to dangerously high levels of lead in water. Lead rarely found in water as its source. The water becomes contaminated as it moves through the water distribution system. The lead can come from lead pipes or connectors, lead solder used to connect pipes and fumes, brass fixtures, and lead lined tanks in water coolers. The most serious problems come when the water is acidic. The acidic water will greatly increase the amount of lead that will leach from lead plumbing. In 1986, the Federal Government has made it illegal to use lead solder (greater than 2% lead in solder) or nine (9%) in waterlines [83]. Newer pipes may pose more of a hazard than older pipes. In older pipes, a mineral scale develops on the interior of the pipe, preventing the lead from leaching into the water. Newer pipes do not have this scale.

Components causing lead contamination include lead pipes, lead-based copper piping solders and brass fixtures. Lead pipes for conveying drinking

Water has been used for centuries because of its flexibility, durability and long life. Lead pipes are used in the United States however, lead concentrations exceeding the regulation levels owing to lead pipe were reported in a number of cities. Levels of lead contamination found in natural raw waters, treatment plants or in distribution mains are typically low and rarely exceed drinking water regulations. However, high lead levels are usually found at consumers taps. Contamination of lead in groundwater origin from the dissolution of lead from soil and earth crust. Lead particulate from the combustion of leaded gasoline, fossil and ore smelting can contaminate local surface water by surface runoff. Lead itself is a minor component in the earth crust. A wide distribution of lead in sedimentary rock and soils are reported. An average lead content of 10 ppm is usually found in upper ground soil and 7 to 12.5 ppm in sedimentary rock [84]. This means that lead generally present in a form of carbonates

and hydroxide complex in soil. The solubility of lead controls the lead dissolution into surrounding water. Strongly absorption by soil and complexation by humus can further limit the lead concentrations in surface waters and groundwater. Children are more affected by the same amount of lead from these and other sources than adults. Foods such as baby formula may pose a significant lead poisoning hazard if lead contaminated water is used in its preparation. As many as 5% of children have high enough lead intake through water and foods to cause health risks [85].

Waters sources near cadmium emitting industries, both with historic and current operations have shown a marked elevation of cadmium in water sediments and aquatic organisms [86, 87]. In surface water and ground water, cadmium can exist as the hydrated ion or as ionic complexes with other inorganic or organic substances.

Cadmium in soluble complex forms may migrate in water. On the other hand, cadmium is relatively nonmobile in insoluble complexes or adsorbed to sediments. Cadmium is taken up and retained by aquatic and terrestrial plants and is concentrated in the liver and kidney of animals that eat the plants [88]. Cadmium exposure through drinking water supply is a minor concern for the U.S. population. In supply water the cadmium concentration limit is to $<5\mu\text{gL}^{-1}$ [89]. Cadmium may be released to water by natural weathering process, by discharge from industrial facilities or sewage treatment plants, atmospheric deposition, by leaching from landfills or soils or phosphate fertilizers [90, 91]. Cadmium may also leach into drinking water supplies from pipes in the distribution system [89]. The average level of cadmium in ocean water has been reported between $<5\text{mg/L}$ and 10mg/L higher level of cadmium is reported in water near coastal areas and in marine phosphates and phosphorities [91].

1.8 Occurrence of Arsenic, Lead and Cadmium in Groundwater of Bangladesh:

Groundwater arsenic problem in Bangladesh is explained by a number of hypotheses. The most widely accepted hypothesis is “Oxy-hydroxide Reduction” and it was supported by extensive modeling and in consistence with the findings of many workers, e.g., BGS/DPHE [77].

A nationwide survey [77] jointly conducted in Bangladesh in 1999 by the British Geological Survey (BGS) Team, UK and Department of Public Health Engineering (DPHE), Bangladesh suggests that the groundwater arsenic problem in Bangladesh arises on account of an unfortunate combination of three factors. These are geological source of arsenic (As is present in the aquifer sediments), mobilization of arsenic (As) is released from the sediments to the groundwater and transport of arsenic (As) is flashed away in the natural groundwater circulation.

Geological source of arsenic:

Previously a number of anthropogenic explanations had been given for the occurrence of arsenic in groundwater. While it is possible that some may explain isolated cases of arsenic contamination, none of the anthropogenic explanations can account for the regional extent of groundwater contamination in Bangladesh and West Bengal. There is no doubt that the source of arsenic is of geological. There have been insufficient analyses of the alluvial sediments to provide a regional picture but current data suggest that arsenic is usually in the range 2-20 mg/kg; only slightly greater than typical sediments (2-6 mg/kg). However, it appears that an unusually large proportion of the arsenic is present in a potentially soluble form. The high groundwater arsenic concentrations are associated with the grey sands rather than the brown sands. There is a good correlation between extractable iron and arsenic in the sediments and a relatively large

proportion (often half or more) of the arsenic can be dissolved by acid ammonium oxalate, an extract that selectively dissolves hydrous ferric oxide and other poorly ordered oxides. It therefore appears likely that a high proportion of the arsenic in the sediments is present as adsorbed arsenic.

The greatest arsenic concentrations are mainly found in the fine-grained sediments especially the grey clays. A large number of other elements are also enriched in the clays including iron, phosphorus and sulphur. In Nawabganj, the clays near the surface are not enriched with arsenic to any greater extent than the clays below 150 m. It is likely that the original sources of arsenic existed as both sulphide and oxide minerals. Oxidation of pyrite in the source areas and during sediment transport would have released soluble arsenic and sulphate. The sulphate would have been lost to the sea but the arsenic, as As(V), would subsequently have been sorbed by the secondary iron oxides formed. These oxides are present as colloidal-sized particles and tend to accumulate in the lower parts of the delta. The finer-grained sediments tend to be concentrated in the lower energy parts of the delta. This is likely to be responsible for the greater contamination in the south and east of Bangladesh.

The map of arsenic contaminated groundwater shows that highly contaminated areas are found in the catchments of the Ganges, Brahmaputra and Meghna rivers strongly suggesting that there were multiple source areas for the arsenic. The types of sediment deposited in the delta region have been strongly influenced by global changes in sea level during the Pleistocene glaciations. For example, sea level was more than 100 m lower at the peak of the last Ice Age around 18,000 years ago. At that time the major rivers incised deep valleys into the soft sediments of the delta. All of the highly contaminated groundwaters occur in sediments deposited since that time, while those sediments predating the low sea level stand contain little or no arsenic-contaminated groundwater.

Mobilisation of the arsenic – redox processes:

Microbial oxidation of the organic carbon has depleted the dissolved oxygen in the groundwater. This is reflected by the high bicarbonate concentrations found in groundwater in recent sediments. There is a relationship between the degree of reduction of the groundwaters and the arsenic concentration – the more reducing, the greater the arsenic concentration.

The highly reducing nature of the groundwaters has led to the reduction of some of the arsenic to As(III) and possible desorption of arsenic since As(III) is normally less strongly sorbed by the iron oxides than As(V) under the near neutral pH groundwater conditions observed. Further reduction will lead to the partial dissolution of the poorly crystallized ferric oxide with consequent release of iron and additional arsenic. Other strongly sorbed ions, especially phosphate, will also be released by iron oxide dissolution. The relatively high phosphate concentrations present in the ground waters will compete with arsenic for sorption sites and is another factor that favours high groundwater arsenic concentrations. It may also make arsenic treatment more difficult. The ‘pyrite oxidation’ hypothesis proposed by scientists from West Bengal is therefore unlikely to be a major process, and The ‘oxyhydroxide reduction’ hypothesis is probably the main cause of arsenic mobilization in groundwater. It is difficult to account for the low sulphate concentrations if arsenic had been released by oxidation of pyrite. Moreover, mineralogical examination suggests that the small amounts of pyrite present in the sediments have been precipitated since burial.

Transport of arsenic within the aquifers :

Present groundwater movement is very slow because of the extremely low hydraulic gradients found in the delta region. Except where modified by

pumping, groundwater circulation is largely confined to the shallow layers affected by local topographic features and the presence of rivers. Close to rivers, the enhanced groundwater flow may lead to a greater dispersion of arsenic along river banks. Annual fluctuations of the water table, typically about 5 m, will affect groundwater and arsenic movement in the shallow layers. There may have been some flushing of arsenic from the shallowest layers.

At greater depths, groundwater moves slowly in response to the low regional gradients. This is consistent with the old age of the waters. The lateral and vertical spread of contaminants is slow even without considering the retardation due to sorption. Modeling suggests that even in the most permeable layers, arsenic movement is likely to be limited to a few meters a year.

The permeability of the silty clay layers is low and in the case of a narrow horizon of silty clay, water will preferentially move through the adjacent more permeable sandy layers. This effectively protects the silty clay layers from strong leaching and possibly preserves arsenic-rich zones. This relative lack of water and arsenic movement and the strong stratification of the aquifer therefore both preserve the high concentrations of arsenic from leaching and lead to the great spatial variability observed. The conclusion from this is that in the absence of man's intervention significant short-term (less than a few decades) variations in arsenic concentrations are unlikely to occur at depth.

1.9 Scale and Magnitude of groundwater contamination in Bangladesh:

Bangladesh is the worst affected country of the world, as its 61 out of 64 districts have arsenic contaminated groundwater above World Health Organization(WHO) level of 10 mgL^{-1} . Arsenic contamination in

groundwater from the Padma-Meghna plain was first reported in 1995 [92, 93]. During 1998, 41 of the 64 districts in Bangladesh were identified as having concentrations of arsenic in groundwater exceeding $50 \mu\text{g L}^{-1}$ [94]. In a preliminary study, workers analyzed 52,000 tube well-water samples from Bangladesh and found that 2,000 and 2,450 villages, had arsenic levels of $>50 \mu\text{g L}^{-1}$ and $10 \mu\text{g L}^{-1}$ respectively [95]. The survey revealed that 43.0% of the water samples exceeded $10 \mu\text{g L}^{-1}$, and 31.0% exceeded $50 \mu\text{g L}^{-1}$. They identified arsenic in hand-tube well water samples $>50 \mu\text{g L}^{-1}$ (permissible limit of arsenic in drinking-water in India and Bangladesh) in 50 of the 64 districts. The populations directly and indirectly at risk were 52 and 32 million respectively. Almost 19.8% ($n=19,000$) of patients screened showed skin lesions, whereas 6.1% of children ($n=5,000$) had skin lesions. In their biomarker study in Bangladesh, 83% of hair samples contained toxic levels of arsenic, and 93% of nail samples and 95% of urine samples showed arsenic above normal levels. It is to be noted that the normal level of arsenic in hair, nails and urine ranged from 80 to $250 \mu\text{g kg}^{-1}$ (levels $\geq 1,000 \mu\text{g kg}^{-1}$ indicate toxicity), 430 to $1,030 \mu\text{g kg}^{-1}$ and 5 to $40 \mu\text{g per 1.5 litres per day}$ respectively [96-97]. The area and population of 50 arsenic-affected districts in Bangladesh are 118,849 sq km and 105 million respectively compared to the total area and population of the country—148,393 sq km and 120 million respectively [97]. This does not mean that all the affected people are drinking arsenic-contaminated water and suffering from arsenic-related diseases, but no doubt they are at risk. The highest conc. of arsenic in groundwater of Bangladesh was found as $3,200 \mu\text{g L}^{-1}$ [98-99]. The groundwater of Bangladesh is moderately contaminated with lead (Pb) and least contaminated with cadmium (Cd) [94].

1.10 Arsenic, Lead and Cadmium in Soil:

Several field studies conducted in Bangladesh showed that irrigation with As-rich groundwater resulted in elevated topsoil As concentrations of up to

~80 mg kg⁻¹ in the upper paddy soil layers, whereas background soil As contents were typically <10 mg kg⁻¹ [94, 96-98]. While increasing irrigation, As concentrations in topsoil of paddy fields were reported to decrease significantly during the monsoon season in regions with pronounced monsoon flooding [94]. In regions with less intense flooding during the monsoon season nearly all As added through irrigation was retained in the soil [98]. Several field studies reported relations between As in soil/irrigation water and rice plants [95, 97-101]. Pot experiments with natural or spiked soil/irrigation water As concentrations [95, 99, 101, 102] revealed similar trends and yield reduction was observed to occur at high As concentrations [102,103]. Panaullah et al. [98] reported approximately 80% grain yield decrease from ~9 to ~2 t ha⁻¹ (i.e. by a factor of 4.5) across a soil As gradient of ~80 to ~10 mg kg⁻¹. Khan et al. [104] supported these findings in a field trial and reported yield reductions of *boro* and *aman* rice of up to 80% . A global “normal” range of 0.08-0.20 mg kg⁻¹ for As in rice grain was recently derived by Zavala and Duxbury [105]. Approximately half of the samples from Bangladesh considered in their study contained more than the upper limit of this range. Meharg and Rahman reported grain As values up to 1.84 mg kg⁻¹ from southwestern Bangladeshi districts, showing the highest soil As concentrations within the country. Recently it was found that in addition to causing elevated grain As levels, increasing soil As concentrations may also affect human nutrition by decreasing the content of important nutrients such as Se, Ni and Zn in rice grain grown in the field. Williams et al. [95] showed that rice plants physiologically control As transfer from straw into grain, resulting in exponentially decreasing transfer factors with increasing straw As content. Adomako et al. [100] recently confirmed this trend for data from Bangladesh. They further showed that straw and grain As are correlated with soil As and that As was more efficiently transferred to grain in the Bangladeshi plants compared to the European and US grown plants. According to the authors,

this difference in the grain to straw transfer is probably due to differing climatic conditions, i.e., temperature versus subtropical or inherent cultivar differences [100].

The concentrations of lead in soil are typically in the range of 5-100 mgkg⁻¹ [106], whereas lead from anthropogenic sources may result in concentrations exceeding 10,000 mg kg⁻¹. In particular, soil in or adjacent to lead smelters, lead mines, houses painted with lead paint, orchards treated with lead arsenate, and urban areas where there has been heavy automobile traffic is likely to contain high concentrations of lead. Ingestion of soil may therefore result in exposure to lead. This may occur by direct ingestion, deposition of soil on the hands, contamination of food, or by inhalation. The relationship between soil and lead varies greatly by soil type [106], perhaps due to behavioral differences among individuals and differences in the rates of absorption of lead from different sources.

The average natural abundance of cadmium in the earth's crust has most often been reported from 0.1 to 0.5 ppm, but much higher and much lower values have also been cited depending on a large number of factors. Igneous and metamorphic rocks tend to show lower values, from 0.02 to 0.2 ppm whereas sedimentary rocks have much higher values, from 0.1 to 25 ppm. Naturally, zinc, lead and copper ores, which are mainly sulphides and oxides, contain even higher levels, 200 to 14,000 ppm for zinc ores and around 500 ppm for typical lead and copper ores. The raw materials for iron and steel production contain approximately 0.1 to 5.0 ppm, while those for cement production contain about 2 ppm. Fossil fuels contain 0.5 to 1.5 ppm cadmium, but phosphate fertilizers contain from 10 to 200 ppm cadmium [107].

Cadmium in soils must be distinctly classified in three separate areas with regard to their relative effects on human health and the environment These

three areas are agricultural soils, non-agricultural soils, and controlled landfills. Cadmium in controlled landfills is virtually immobile, and is unlikely to have any effect on human health or the environment simply because it is so well contained [108]. Cadmium in non-agricultural soil will generally not affect human health because it does not enter the food chain readily. Cadmium in agricultural soils is likewise relatively immobile under normal conditions, but could become more mobile under certain conditions such as increased soil acidity and usage of phosphate fertilizers, manure or sewage sludge.

Since cadmium is a naturally occurring component of all soils, all food stuffs will contain some cadmium and therefore all humans are exposed to natural levels of cadmium. Although much attention has been focused upon the cadmium content of agricultural soils, it is important to recognize that the cadmium content of food items varies more as a function of the nature of the crop grown and the agricultural practices followed. Except in cases of extreme contamination, the concentration of cadmium in soils is not the primary determinant of cadmium in the human diet. For example, leafy vegetables and potato tubers naturally accumulate higher levels of cadmium than do fruits and cereals [103]. Moreover, tillage and crop rotation practices similarly have a greater impact upon the cadmium content of food than does the concentration of cadmium in soils [109]. Cadmium absorption may also depend on other factors.

1.11 Biotransformation:

In general, the removal of electrons from or addition of electrons to the atom influences the chemical activity and therefore the ability of metallic elements to interact with tissue targets (ligands). Examples of charge relevance in crossing lipid barriers are represented by $\text{Fe}^{2+}/\text{Fe}^{3+}$ and $\text{Hg}^{+}/\text{Hg}^0$ passages. Among the other metabolic transformations, the most

important is bioalkylation, where mercury, tin, and lead undergo in microorganisms, whereas arsenic and selenium are additionally bioalkylated as part of their metabolic pathways in higher organisms. Alkylation reactions produce more hydrophobic species, leading to an increased bioavailability, penetration to cells and through the blood–brain barrier, as well as accumulation in fatty tissues. Biotransformation of arsenic involves methylation, leading to the formation and excretion of monomethylated and dimethylated compounds (Fig. 1.6). In most mammals, only trivalent arsenic species are methylated i.e. in the metabolism, reduction and methylation alternate. Possibly, As(III) is bound to a dithiol, a carrier protein, before the methyl groups are attached. S-Adenosyl methionine is the main methyl donor in arsenic methylation.

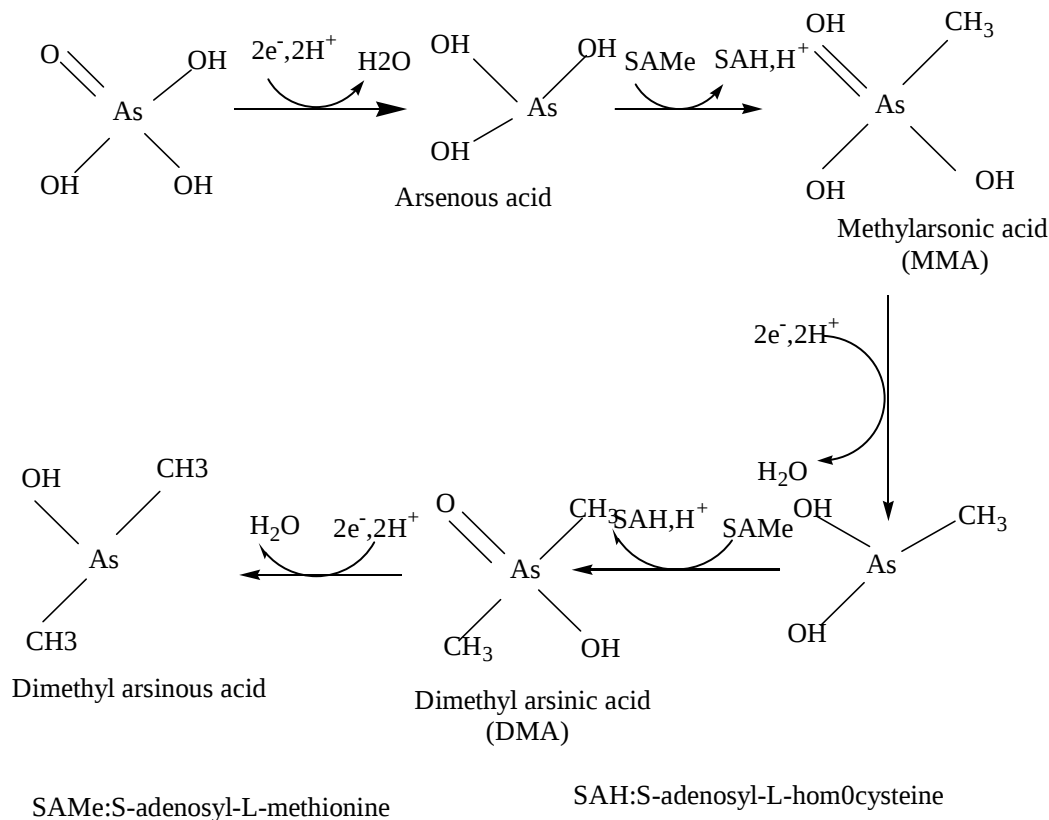


Fig 1.6 Arsenic biotransformation.

The liver is an important site of arsenic methylation, especially following ingestion, when the absorbed arsenic initially passes the liver. This is

supported by studies showing a marked improvement in the methylation of arsenic in patients with end-stage liver disease following liver transplantation. However, arsenic may also be methylated in other tissues, as methylating activity has been detected in several different tissues of male mice. The highest activity was detected in the testes, followed by kidney, liver, and lung due to two different enzymatic activities. Moreover, observations in humans repeatedly ingesting low inorganic arsenic doses or acutely intoxicated by As_2O_3 also suggest a different rate for two methylation steps and an inhibitory effect of the trivalent inorganic form for the second methylation step leading to DMA. According to the suggested mechanism of arsenic methylation, the methyl groups react with arsenic in its trivalent form. Experimental studies have shown that a major part of absorbed As(V) as arsenate is rapidly reduced to As(III) as arsenite, probably mainly in the blood. As arsenite is more toxic than arsenate, this initial step in the biotransformation of arsenate may be regarded as a bioactivation. However, much of the formed arsenite is distributed to the tissues, where it is methylated to MMA and DMA. It has been shown that arsenite is taken up in hepatocytes much more readily than arsenate. At physiological pH, arsenites are present mainly in undissociated form, which facilitates passage through the cellular membrane, whereas arsenate is in an ionized form. Glutathione and probably other thiols serve as reducing agents for arsenate and MMA. Depletion of hepatic glutathione in rats and hamster by buthionine sulfoximine was shown to decrease the methylation of inorganic arsenic. Reduplicate activity of arsenate has been detected in human liver. The population variation in arsenic metabolite production indicates a genetic polymorphism in the regulation of enzymes responsible for arsenic methylation. The role of speciation in arsenic metabolism in a case of arsine intoxication was assessed by examining the urinary arsenic species of the patient for 1 month. As excreted species, with quite different excretion patterns among species: arsenite excretion followed an

exponential curve; an important elimination of MMA was observed early 1 or 2, while DMA elimination increased progressively and culminated on day 5, when MMA excretion tended to decrease. Less than 5% of the total amount was excreted as arsenate, and it disappeared after day 10. The conversion of arsenate to arsenite seemed to be influenced by the amount of arsenite and by synthesis of other metabolites. The fact that DMA excretion culminated after only a few days, while MMA excretion was still elevated, seems to confirm the existence of two different methylating enzymatic systems. Arsenobetaine seemed to be excreted independently of other species, being probably linked to uptake of arsenic from meals. Irrespective of the type and extent of exposure, the average relative distribution of arsenic metabolites in the urine of various population groups seems to be fairly constant (e.g. 10–30% inorganic arsenic, 10–20% MMA, and 60–70% DMA). However, there are certain exceptions, e.g., people living in certain areas of Taiwan, China, seem to have an unusually high percentage of MMA in urine, 20–30% on average. [110]

The rate of absorption of lead after ingestion can range from 3 to 80%. It is heavily influenced by food intake, much higher rates of absorption occurring after fasting than when lead is ingested with a meal. This effect may be due mainly to competition from other ions, particularly iron and calcium, for intestinal transport pathways. Absorption is also affected by age, the typical absorption rates in adults and infants being 10% and 50%, respectively [111, 112].

Several pharmacokinetic models have been developed in an attempt to relate exposure to lead concentrations in blood and other tissues. A model developed by the United States Environmental Protection Agency (1994) of lead in air, diet, dust, paint, soil, and drinking-water allows modeling of distribution in a 10-compartment model that includes plasma, erythrocytes, trabecular bone, and cortical bone. The parameters in the model were chosen to represent children, and the model presumes that exposure and

pharmaco-kinetics are in a steady state. Population distributions are derived by using the model output to estimate the population geometric mean and a fixed geometric standard deviation. The concentration of lead in blood is the most widely used biomarker of exposure, largely because it is the measure that is the easiest to obtain. Blood lead concentration is typically reported in micrograms per deciliter. Because blood concentrations are strongly influenced by recent exposure, they may not reflect long-term exposure, particularly to intermittent or infrequent, high levels. A marker that was widely used before techniques for measuring blood lead became widely available is plasma delta-aminolaevulinic acid dehydratase activity, which correlates strongly with blood lead concentration [112]. Related markers that have sometimes been used are the urinary and blood concentrations of delta-amino-laevulinic acid and plasma d-aminolaevulinic acid synthesise. Both of these markers correlate positively with exposure to lead.

O'Flaherty [113] developed another pharmacokinetic model which represents the temporal relationships in the transfer of lead between blood, bone, and other tissues. It is thus more useful for predicting physiological lead concentrations when exposure is inconstant. It also accounts for changes that occur as a function of age. In particular, the model accounts for differences in pharmacokinetics attributable to changes in body size and in age-related changes in absorption, bone growth, or metabolism that occur during childhood, pregnancy, menopause, and old age. The model does not attempt to account for population variation, but when it is used in a simulation designed to represent population variation, the exposure inputs and some of the biological parameters (such as absorption rates) can be varied to reflect population differences.

Lead that is deposited in physiologically inactive cortical bone may persist for decades without substantially influencing the concentrations of lead in

blood and other tissues [113], lead that is deposited in physiologically active trabecular bone is in equilibrium with blood. The accumulation of high concentrations in blood when exposure is reduced may be due to the ability of bone to store and release lead. Dietary lead that is not absorbed in the gastrointestinal tract is excreted in the faeces. Lead that is not distributed to other tissues is excreted through the kidney and to a lesser extent by biliary clearance [112]. The best-documented biochemical mechanism is inhibition of delta-aminolaevulinic acid dehydratase, resulting in binding of lead to sulfhydryl groups. The concentrations of coproporphyrin in urine and of 5-aminolaevulinate in blood and urine are increased in individuals exposed to high concentrations of lead as a result of inhibition of this enzyme.

In human body, Cd is basically taken up by the liver. In the liver, Cd can bind with glutathione (GSH) and be excreted into bile. Moreover, Cd can bind to metallothionein (MT) and be stored. Some Cd bound to MT leaks into the plasma and then is taken up by the kidney. The balance between CdMT and no bound Cd in renal tissue may be responsible for the degree of Cd nephrotoxicity [114,115]. Therefore, susceptibility to Cd toxicity among individual may probably be related to genetic variations of Cd-metabolizing enzymes. Glutathione S-transferase (GST) gene is one of the potential candidate susceptibility genes-because it plays a significant role in Cd biotransformation and detoxification [116]. The principal function of GST enzymes is conjugations of hydrophobic and electrophilic compounds with reduced glutathione. The intracellular binding reaction with GSH is catalyzed by the GSTs and leads to stable GSH-metal conjugates being transported out of the cell and excreted via feces and urine [116]. Given that metabolism and detoxification of cadmium (as an electrophile) is mediated by glutathione and GSTs are involved in the complexation of electrophilic substances with glutathione, it is reasonable to hypothesize that genetic polymorphisms in GST genes could result in differences in

sensitivity to cadmium.

In recent years, the possibilities of applying molecular techniques to toxicogenetics are considerably focused, especially for medical investigations and determinations of factors influencing chemical poisonings. These include biochemical and genetic determinants related to acute and chronic effects. To our knowledge, there are limited data on genetic predisposition influence on cadmium detoxification in the human body. Therefore, this study investigated the association of glutathione S-transferases genes (GSTT1, GSTM1, and GSTP1) polymorphisms and blood cadmium concentration in a non occupationally exposed population.

1.12 Toxicity of Arsenic, Lead and Cadmium:

The relative toxicity of different As species to plants depends on a range of factors including experimental conditions and plant species and examined plant parameters [117]. Therefore, one should be cautious about using a generalized classification of As species according to toxicity. Taking that into account, inorganic As is generally regarded as being more toxic than organic As, with As (III) being the more toxic form [118]. Most toxicity experiments have been carried out with plants grown in water only (hydroponics). Such a design can be useful to study, for example, uptake mechanisms, internal transport, metabolism, and toxic effects. The design is however not suitable to generate toxicity data to evaluate concentrations in the environment because all interactions with the soil matrix influencing bioavailability are neglected. Exposure to arsenic is mainly via intake of food and drinking water, food being the most important source in most populations. Long-term exposure to arsenic in drinking-water is mainly related to increased risks of skin cancer, but also some other cancers, as well as other skin lesions such as hyperkeratosis and pigmentation changes. Occupational exposure to arsenic, primarily by inhalation, is causally associated with lung cancer. Clear exposure-response relationships and

high risks have been observed.

Toxicity experiments are also carried out with plants grown in soil to which a certain amount of As is added (spiked soil) shortly before the experiment. This setup has various limitations as well. Adding As to reach a certain soil concentration suggests that the results are representative of the field. However, in the field As is added over a number of years. The prolonged contact time between As and the soil in the field can result in a lower solubility of As and therefore lower uptake by plants in the field. Therefore, experiments with spiked soils often result in an overestimation of the adverse effects compared to the actual field situation [119].

In other studies, As has been added via irrigation water to the soil during the experiments. This is more in agreement with the field situation in Bangladesh compared to hydroponics and spiked soils. However, this experimental setup neglects that As levels in irrigation water in the field are relatively constant and that As is slowly added to the soils over a period of many years. Ideally, experiments should be performed with naturally contaminated soils and constant As concentrations in irrigation water.

To date, toxic effects have only been related to the irrigation water concentration and the total soil concentration. The As concentration in soil water (pore water) will surely deviate from the irrigation water because of interactions with the soil matrix. Total As in the soil is also unlikely to be a good predictor of As uptake and toxicity for different soil types as only part of the As in the soil is likely to be potentially available to the plants. Dose–response relationships based on irrigation water concentrations or total soil concentrations are only valid for the experiment from which they were derived and cannot be extrapolated to any other system. With all these limitations in mind, various studies of the above mentioned types will be summarized and discussed.

Soil micro-organisms may also be affected by As toxicity [70]. Effects of

As and on the soil microbial community can be expected with As(III) being more toxic than As(V). Microbes can adapt to As contamination, but this can be accompanied by a change in density and structure of the community. Ghosh et al. [120] reported that microbial biomass and activity were negatively correlated with total and bioavailable As in soil samples from West Bengal. However, the description of the used soils was limited, making it difficult to assess if there were any other reasons like different soil types and land use that could explain the results.

As human activities increases, especially with the application of modern technologies, pollution, and contamination of the human food chain has become inevitable. Heavy metals uptake by plants grown in polluted soils has been studied to a considerable extent [121]. Heavy metal contamination in vegetables cannot be underestimated as these foodstuffs are important components of human diet. Heavy metal contamination of the food items is one of the most important aspects of food quality assurance [122,123].

The symptom picture of arsenic toxicity is characterized by dermal lesions, anemia, and an increased risk for cardiovascular disease, diabetes, and liver damage.

Cadmium has a significant effect on renal function, and as a result alters bone metabolism, leading to osteoporosis and osteomalacia. Cadmium-induced genotoxicity also increases risk for several cancers. The mechanisms of arsenic- and cadmium-induced damage include the production of free radicals that alter mitochondrial activity and genetic information. The metabolism and excretion of these heavy metals depend on the presence of antioxidants and thiols that aid arsenic methylation and both arsenic and cadmium metallothionein-binding [124].

Both arsenic and cadmium are easily available in the environment, and the exposure via food, water, and other occupational sources can contribute to a

spectrum of diseases. Cd exposure may pose adverse health effects, including kidney dysfunction, and skeletal disorders, and may also affect bones and result in fractures [118]. Long-term exposure to Pb may cause neurological disorders such as memory deterioration, prolonged reaction times, and reduced cognitive ability [125].

The general population is exposed to lead from air and food in roughly equal proportions. During the last century, lead emissions to ambient air have caused considerable pollution, mainly due to lead emissions from petrol. Children are particularly susceptible to lead exposure due to high gastrointestinal uptake and the permeable blood-brain barrier. Blood levels in children should be reduced below the levels so far considered acceptable, recent data indicating that there may be neurotoxic effects of lead at lower levels of exposure than previously anticipated. Although lead in petrol has dramatically decreased over the last decades, thereby reducing environmental exposure, phasing out any remaining uses of lead additives in motor fuels should be encouraged. The use of lead-based paints should be abandoned, and lead should not be used in food containers. In particular, the public should be aware of glazed food containers, which may leach lead into food.

1.13 Clinical Manifestation and Treatment of arsenic, lead and cadmium:

Arsenic may be absorbed by ingestion, inhalation, and permeation of skin or mucous membranes and may cause adverse health effects.

(a) Non-Cancer:

By ingestion, as little as 20 milligrams of arsenic may be life-threatening to man. Acute airborne exposure to high concentrations of arsenic (III) trioxide in occupational settings causes irritation of the eyes, nasal mucosa, and bronchi. Non-carcinogenic effects associated with chronic occupational exposure to high concentrations of airborne arsenic include nasal septum

ulceration and perforation, respiratory tract irritation and peripheral neuropathy. The most sensitive non carcinogenic endpoints are vascular disorders (hemolysis), neurological disturbances, and adverse reproductive effects. Other reported effects from acute inhalation exposure include gastrointestinal effects (nausea, diarrhea, and abdominal pain). Chronic inhalation exposure to inorganic arsenic in humans is associated with irritation of the skin and mucous membranes, while chronic oral exposure has resulted in gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, and liver or kidney damage [126]

An acute non-cancer reference exposure level (REL) of $1.3 \times 10^{-2} \mu\text{g}/\text{m}^3$ is listed for arsine in the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program, Revised 1992 Risk Assessment Guidelines. The toxicological endpoint considered was blood. For arsenic, California Air Pollution Control Officers Association lists a chronic non-cancer REL of $0.5 \mu\text{g}/\text{m}^3$ with toxicological endpoints of the respiratory and central and peripheral nervous systems, and skin (CAPCOA, 1993). The United States Environmental Protection Agency has not established a Reference Concentration (RfC) for inorganic arsenic. The RfC for arsine is $5 \times 10^{-2} \mu\text{g}/\text{m}^3$ based on increased hemolysis, abnormal red blood cell morphology, and increased spleen weight in rats, mice and hamsters. The United States Environmental Protection Agency estimates that inhalation of this concentration or less over a lifetime would not likely result in the occurrence of chronic non-cancer effects. The oral Reference Dose (RfD) for inorganic arsenic is 0.3 micrograms per kilogram per day based on hyper pigmentation, keratosis and possible vascular complications in humans.



Fig1.7.Clinical manifestation of chronic arsenic toxicity.

The United States Environmental Protection Agency has not established an RfD for arsine [123]. The most prominent chronic manifestations involve the skin, lungs, liver and blood systems. This was first diagnosed in West Bengal and Bangladesh patient of Khulna in December, 1984 [127,128] and by Prof. K. C. Saha in July 1982 at School of Tropical Medicine, Calcutta, India [129].

(b) Short-Term Effects:

Ingestion of large doses of arsenic usually results in symptoms within 30 to 60 minutes, but may be delayed when taken with food. Acute arsenic poisoning usually starts with a metallic or garlic-like taste, burning lips and dysphasia. These gastrointestinal symptoms are the result of intestinal injury caused by dilatation of splanchnic vessels leading to mucosal vesiculation. These vesicles rupture causing bleeding, diarrhea and protein wasting. Gastrointestinal symptoms often result in dehydration and electrolyte imbalance, and may lead to the development of hypotension and hypoxia [130]. Fatal arsenic poisonings have been reported after oral exposure to estimated single doses of 2 g [131], 8 g [132] and 21 g [133]. Non-fatal outcomes (usually following treatment) have been documented after oral single doses of 1-4 g [134]. Cullen et al. [135] found a serious

acute effects in children after exposure to as little as 0.7 mg of As_2O_3 . Incidents in which continuous or repeated exposure to high levels of arsenic over a short period of time have also been described. Following consumption of water containing 108 mg As/L for one week, two of nine persons died, four developed encephalopathy and eight showed gastrointestinal symptoms.

(c) Chronic Arsenic Exposure:

Chronic exposure to lower levels of arsenic has long since been linked to adverse health effects in human populations. The earliest reports date back to the latter part of the 19th century when the onset of skin (including pigmentation changes, hyperkeratosis and skin cancers) were linked to the consumption of arsenic in medicines and drinking water [136]. In the early 1900s, numerous reports of skin disorders in Argentina, Chile, Mexico and Taiwan, were attributed to arsenic exposure via drinking water, were published [137]. In the 1940s the discovery of a case of lung cancer, believed to be the result of exposure to arsenical dust in a British factory, sparked a series of more detailed investigations into the matter. These in turn revealed unexpectedly high lung cancer rates in a number of different occupational exposure situations. Of the earliest reported cases of chronic arsenic poisoning, that of Blackfoot disease (BFD) or Wu Chiao Ping as it is locally known, is perhaps the most notorious. This peripheral vascular disease, which leads to progressive gangrenes of the legs, has been recognized in parts of Taiwan since the 1920s. During the 1950s its prevalence increased markedly, and since the late 1950s it has been the subject of intensive study.

(d) Diabetes Mellitus:

Diabetes mellitus has also been linked with drinking water arsenic exposure. Lai et al. [138] assessed the relationship between ingested inorganic arsenic and prevalence of diabetes mellitus in 891 adults residing

in southern Taiwan. Their study found that residents in the BFD-endemic areas had a two-fold increase in the prevalence of diabetes mellitus (after adjustment for age and sex) when compared to residents in Taipei and the entire Taiwan population. The authors also described a dose-response relationship between the level of arsenic in water and the prevalence of diabetes after adjusting for age, sex, body mass index and activity level. Positive associations were also demonstrated in two further studies from Taiwan. An excess mortality from diabetes among the arsenic exposed population in four townships, relative to local and national rates, has been reported by Tsai et al. [139]. The incidence of diabetes mellitus in a cohort of inhabitants of the Taiwan BFD area was related to the cumulative exposure to arsenic in drinking water [140]. A positive association with diabetes has also been found in Bangladesh [141] used the presence of keratosis as an indicator of arsenic exposure and showed elevated risks for diabetes in those exposed to arsenic in their drinking water (prevalence ratio= 5.9). On the other hand, in the Utah mortality study, Lewis et al.[142] failed to find a significant excess in the number of deaths from diabetes in males and females exposed to elevated levels of arsenic in drinking water. However, in the US diabetes is a condition with a low-case fatality rate, so an association with diabetes mellitus may not be observed. More work is needed prior to deciding whether there is an association in the US between diabetes and drinking water arsenic. Two occupational studies have found an association between arsenic exposure and diabetes mellitus. In both cases, however, the results were of borderline significance. Based on a case-referent analysis involving glass workers found a slightly elevated risk for diabetes among those glasswork employees considered to be exposed to inhaled forms of arsenic. In a smaller (12 exposed cases) case- referent study in the Rönnskär cohort [143], a slightly elevated risk of diabetes mellitus associated with arsenic exposure was also observed.

(e) Neurological Effects:

It is generally accepted that acute arsenic poisoning causes neurological effects in humans, especially in the peripheral nervous system. To date, however, little work has been done on the possibility that lower-level, long-term exposure to arsenic may also lead to neurological effects. Of the limited number of available studies on this topic, several have described the onset of various neurological symptoms in subjects exposed to arsenic [144]. For example, reported a positive association between electromyography (EMG) abnormalities and arsenic levels in drinking water and hair samples in residents of Waverley, Nova Scotia, Canada. Among those using water with more than 1 mg/L arsenic, the frequency of EMG abnormalities was 50%. Workers at a copper smelting plant exposed to arsenic trioxide were examined for peripheral neuropathy [145]. Although a handful of studies have suggested that changes in nerve function may occur following exposure to arsenic, such studies are typically limited by their small sample populations, differing end-points, and methods of measurement and probable co-exposure to other known neurotoxins [128].

Exposure to inorganic arsenic can cause various health effects, such as irritation of the stomach and intestines, decreased production of red and white blood cells, skin changes and lung irritation. It is suggested that the uptake of significant amounts of inorganic arsenic can intensify the chances of cancer development, especially the chances of development of skin cancer, lung cancer, liver cancer, and lymphatic cancer. A very high exposure to inorganic arsenic can cause infertility and miscarriages with women, and it can cause skin disturbances, declined resistance to infections, heart disruptions, and brain damage with men and women. Finally, inorganic arsenic can damage DNA. A lethal dose of arsenic oxide is generally regarded as 100 mg organic arsenic can cause neither cancer,

nor DNA damage. But exposure to high doses may cause certain effects to human health, such as nerve injury and stomach aches [146].

Lead is one out of four metals that have the most damaging effects on human health. It can enter the human body through uptake of food, water and air. The health effects of lead have become much better understood since the middle of the 20th century. At one time, the metal was regarded as quite safe to use for most applications. Now lead is known to cause both immediate and long-term health problems, especially with children. It is toxic when swallowed, eaten, or inhaled. Exposure to lead is typically greater in children, who are also more susceptible to the effects of lead than adults. Children are particularly at risk for the subtle adverse effects of chronic low-dose lead exposure, as are pregnant women/fetuses.

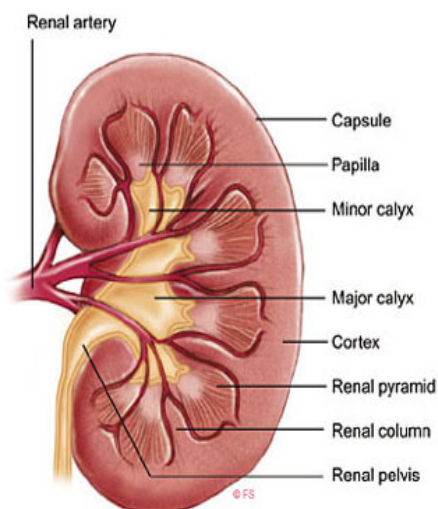
Lead can enter a fetus through the placenta of the mother. Because of this it can cause serious damage to the nervous system and the brains of unborn children [147]. Children absorb about 50% of ingested lead [148]. They have an abnormal desire to eat materials like dirt, paper, and chalk. Some symptoms of lead poisoning include nausea, vomiting, extreme tiredness, high blood pressure, and convulsions (spasms). Over a long period of time, these children often suffer brain damage. They lose the ability to carry out normal mental functions. Other forms of lead poisoning can also occur. For example, Lead poisoning through inhalation can lead to nerve damage and problems with the gastrointestinal system (stomach and intestines). Today, there is an effort to reduce the use of lead in consumer products. For instance, older homes are often tested for lead paint before they are resold. Lead paint has also been removed from older school buildings [149]. Lead exerts adverse effects on numerous organs and systems including the central nervous system (CNS), the kidneys and on the hematopoietic (blood forming) system. The identification of causal relationships between

exposure and effects is complicated by the delay between the time of lead exposure and the onset of effects [150].

Dermal absorption of inorganic lead has been demonstrated in animals and human subjects, however, few studies have provided quantitative estimates of dermal absorption and the contribution of the dermal intake to lead body burden [151]. Absorption of lead through the skin has only been measured accurately for organic lead compounds. Exposure to lead from cosmetics previously occurred from use of progressive hair dye preparations containing lead acetate [152], however, lead acetate is now prohibited for such use in Canada. The Drinking Water Guidelines in Canada limit the lead content to a Maximum Acceptable Concentration (MAC) of 0.010 milligrams per liter (0.010 ppm) of water [153]. Acceptable oral intake of lead impurities includes 0.1 ppm (US FDA for candy) to 10 ppm (USP for nutritional supplements). The World Health Organization (WHO) has established 25 micrograms of lead per kilogram of body weight per week as a provisional tolerable weekly intake (PTWI) for children. Health Canada's Natural Health Products Directorate (NHPD) limits lead in products applied to the skin to 10 ppm (NHPD Compendium of Monographs).



Fig 1.8. Brain damage due to Lead exposure



Kidney damage due to Lead exposure

In general, the amount of cadmium that will cause health problems will vary depending on: (1) the type of exposure (eating or breathing), (2) the duration of the exposure (short-or long-term), and (3) the form of cadmium (pure cadmium or some combination). Studies show that humans can experience lung irritation after breathing as little as 1.0 milligrams per cubic meter of air (mg/m^3) of cadmium-contaminated air for a short period of time (less than or equal to 14 days). Breathing $0.01 \text{ mg}/\text{m}^3$ of cadmium-contaminated air over the long-term (greater than 14 days) has resulted in chronic lung disease and kidney disease in humans. Human uptake of cadmium takes place mainly through food and water. Foodstuffs that are rich in cadmium can greatly increase the cadmium concentration in human bodies. Examples are liver, mushrooms, shellfish, mussels, cocoa powder, and dried seaweed. The major sources of cadmium in humans are cigarette smoking, certain foods (such as shellfish, liver, and kidney meats), coal burning, and contaminated water. Those most at risk for high intake of cadmium are people who work directly with the metal. Manufacturing plants where batteries are made use cadmium as a fine powder where it can easily be inhaled. Workers must be careful in handling cadmium. There is growing concern about the dangers of cadmium in the environment. Rechargeable batteries are made with cadmium and nickel. Cadmium can escape from landfills (where trash is buried) and get into the ground and ground water. From there, it can become part of the food and water those humans and animals ingest. Low levels of cadmium cause nausea, vomiting, and diarrhea. Inhaled, cadmium dust causes dryness of the throat, choking, headache, and pneumonia-like symptoms [154].

An exposure to significantly higher cadmium levels occurs when people smoke. Tobacco smoke transports cadmium into the lungs. Blood will transport it through the rest of the body where it can increase effects by

potentiating cadmium that is already present from cadmium-rich food. Other high exposures can occur with people who live near hazardous waste sites or factories that release cadmium into the air and people that work in the metal refinery industry. When people breathe in cadmium it can severely damage the lungs.

This may even cause death. Cadmium is first transported to the liver through the blood. There, it is bond to proteins to form complexes that are transported to the kidneys. Cadmium accumulates in kidneys, where it damages filtering mechanisms. This causes the excretion of essential proteins and sugars from the body and further kidney damage. It takes a very long time before cadmium that has accumulated in kidneys is excreted from a human body [155].

Other health effects that can be caused by cadmium are [155]:

- Diarrhoea, stomach pains, and severe vomiting
- Bone fracture
- Reproductive failure and possibly even infertility
- Damage to the central nervous system
- Damage to the immune system
- Psychological disorders
- Possibly DNA damage or cancer development

The effects of extensive cadmium exposure is not known, but are thought to include heart and kidney disease, high blood pressure, and cancer. A cadmium poisoning disease called *itai-itai*, Japanese for “ouch-ouch,” causes aches and pains in the bones and joints. In the 1970s, a number of cases of itai-itai were reported in Japan when waste from a zinc refinery got into the public water supply. Those wastes contained cadmium compounds [154].

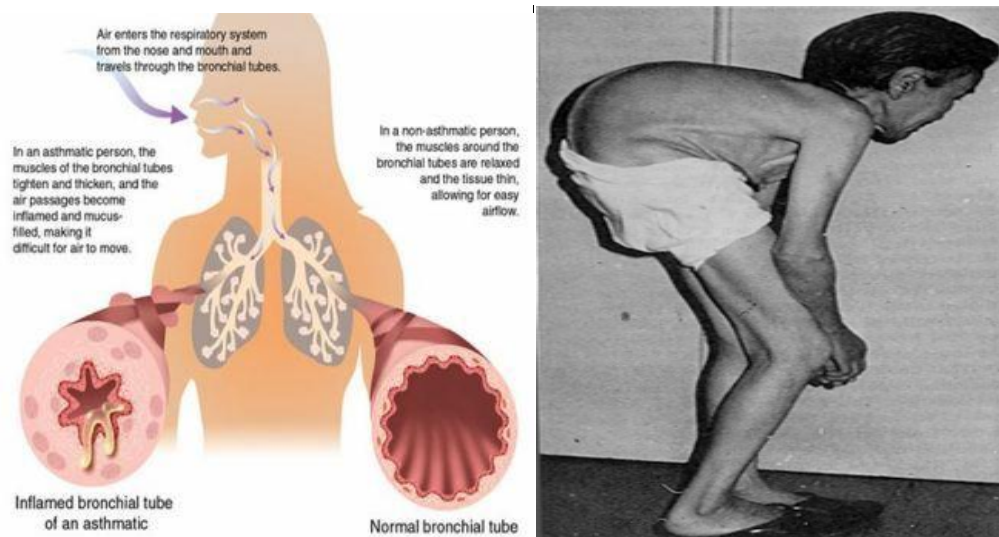


Fig 1.9. heart due to cadmium exposure

Bone fracture due to cadmium exposure

1.14 Rationale of the study :

Arsenic, lead and cadmium are the most abundant elements in the earth's crust and also are a toxic element that occur naturally in all environment media and may pollute air, soil, sediments and water causing serious health hazards to both human and animal. Recently arsenic in groundwater and its fate and transport in the environment have become matters of great concern in Bangladesh. BGS/DPHE nationwide survey [156,157] shows that in 61 out of 64 districts of Bangladesh, the groundwater is contaminated with arsenic. An estimated 29 million people are directly exposed to the arsenic problem [158] and 85 million are at the risk of arsenic contamination [159]. It has been reported [160] that about 10,000 patients are suffering from arsenicosis and a few death occurred due to arsenic poisoning. In this point of view, Bangladesh is facing probably the largest mass poisoning in history.

In car engines lead is burned, so that lead salts (chlorines, bromines, oxides) will originate. These lead salts enter the environment through the exhausts of cars and may pollute water, soil and air. Lead can end up in

water through corrosion of leaded pipelines in a water transporting systems. Lead accumulates in the bodies of water organisms. Lead is a particularly dangerous chemical as it can accumulate in individual organisms, but also in entire food chains. It can enter the human body through uptakes of food, water, and air. These will experience health effects from lead poisoning. Hence, lead pollution is considered to be a worldwide issue.

Cadmium is emitted to the environment as a result of both natural and anthropogenic activities. Cadmium is released to the aquatic environment from a range of anthropogenic sources, including non-ferrous metal mining and smelting (from mine drainage water, waste water, tailing pond overflow, rainwater runoff from mine areas), plating operations, phosphate fertilizers, sewage-treatment plants, landfills and hazardous waste sites [161]. Cadmium ends up in surface waters when waste from fertilizer production is dumped by production companies. Cadmium can be transported over great distances when it is absorbed by sludge. This cadmium-rich sludge can pollute surface water as well as soils. Cadmium accumulated in both human and animal body causes serious diseases like lung cancer, kidney damage, diarrhea, bone fracture, damage to the central nervous system and damage to the immune system.

Soil is an important natural resource for mankind but it also serves as a medium for the accumulation, transformation and migration of toxicants like arsenic, lead and cadmium. For example, arsenic, lead and cadmium may accumulate in soil through the use of arsenical pesticides, lead pesticides, cadmium-rich sludge, irrigation and disposal of industrial and animal wastes.

The widespread use of groundwater and pesticides of arsenic, lead and cadmium for irrigation and cultivation suggests that ingestion of the irrigated crops could be another major exposure route of arsenic, lead and cadmium.

Since the arsenic, lead and cadmium problem in Bangladesh is terrible at present, it will be invaluable to understand the entrance of arsenic, lead and cadmium into human food chain from sources other than contaminated drinking water. Arsenic, lead and cadmium concentration in edible parts of plants due to irrigation by arsenic, lead and cadmium contaminated water – its extend and solution is still remains unfocused. No significant research is carried out yet for remedy of As, Pb & Cd poisoning in plants. This serious issue that needs to be addressed for ensuring safety of human health, leads us to conduct this investigation.

1.15 Objectives of the study:

General Objective :

To find out the potential ways for remedy of As, Pb and Cd toxicity in human food chain and to assess health risks due to As, Pb and Cd exposures from dietary food products.

Specific Objectives

- To investigate the pattern of As, Pb and Cd accumulation in vegetables after irrigation with contaminated water.
- To investigate the impacts of natural organic fertilizers towards reduction of As, Pb and Cd toxicity in vegetable plants.
- To compare the heavy metal status in vegetables, fish, rice, dust and drinking water consumed by the inhabitants of Rajshahi City Corporation (RCC) area.
- To assess overall potential risk due to As, Pb and Cd exposure from dietary consumption for different population sub-groups of RCC area.

1.16 Scope of the study:

Arsenic, Lead and Cadmium are dietary toxic constituents and are present in many foods such as meat, fish, poultry, vegetables, grains, cereals, seafood, soft drinks, mushrooms, shellfish, cocoa powder and dried seaweed. Arsenic, lead and cadmium content in the agricultural foodstuffs

might increase to many folds, when successive irrigation are made with arsenic, lead and cadmium contaminated water. This investigation has been conducted to the commonly grown vegetables, cereals and grains.

The precise dimension of the arsenic, lead and cadmium problems in the field of agriculture is not yet known in Bangladesh. The reasons include fluctuation of concentration of arsenic, lead and cadmium load in soil of up taking of heavy metal pollutants from soil of specific plants. Considering these facts, we carried out our studies in field with fresh soil, except for successive harvesting, irrigated with both natural underground and model water, this not only removes the possibility of introducing previous contamination but also facilitates to find out the actual several relationship like arsenic, lead and cadmium concentration of water, soil and plant tissues, etc. There was scope to find out the pattern of arsenic, lead and cadmium accumulation in some commonly grown vegetables, cereals and grains with respect to time due to irrigation. This allows to estimate the maximum allowable daily level (MADL) in different vegetables and grains. Attempt could be made to determine the threshold concentration of arsenic, lead, cadmium, in irrigation water, beyond which on application might cause toxicity of arsenic, lead and cadmium in edible parts of the plants. This might help in formulating policy or guideline value for irrigation after proper calibration. The cumulative effects of arsenic, lead and cadmium deposition due to successive harvesting for the same sets of vegetable plants could be determined to estimate arsenic, lead and cadmium load per harvesting.

There was opportunity to trail arsenic, lead and cadmium distribution pattern within the plant system quantatively. Based upon statistical regression analysis of the available data, the concentration of arsenic, lead and cadmium in edible plant tissues could be correlated with those of irrigation water and soil, and establishment of several mathematical models. Also the potential risk of arsenic, lead and cadmium exposure from

consumption of commonly grown vegetables and grains could be assessed precisely. Attention could be paid to find out the factors that influence the accumulation of arsenic lead and cadmium in vegetables. The effects of inorganic and organic fertilizers, arsenic, lead and cadmium species in different oxidation states and pH of irrigation water were also studied in this regard. The findings should throw the light on sorting out the potential ways for remedy of arsenic, lead and cadmium toxicity in vegetables and grains.

1.17 References:

- [1] Huq SMI, Naidu R. Arsenic in groundwater of Bangladesh: contamination in the food chain. In: Arsenic contamination: Bangladesh perspective. Ahmed MF (ed). Dhaka, ITN- Bangladesh, (2003), pp 203-26.
- [2] Meharg AA, Rahman MM. Arsenic contamination of Bangladesh paddy field soils: Implications for rice contribution to arsenic consumption. *Environ Sci. Technol.* (2003); 37: 229-34.
- [3] Das HK, Mitra AK, Sengupta PK, Hossain A, Islam F, Rabbani GH. Arsenic concentrations in rice, vegetables, and fish in Bangladesh: A preliminary study. *Environ Int.* (2004); 30: 383-87.
- [4] Williams P. N., Islam M. R., Adomako E. E., Raab A., Hossain S. A., Zhu Y. G., Feldmann J., and Meharg A. A., *Environ. Sci. Technol.* 40, 4903 (2006). <http://dx.doi.org/10.1021/es060222i>.
- [5] Huq S.M.I. and Naidu R., Arsenic in Groundwater and Contamination of the F Chain: Bangladesh Scenario, Bundschuh et al. (ed) (Taylor & Francis Group, London, 2005).
- [6] Sanyal, S.K. (2005). Arsenic contamination in agriculture: A threat to water-soil-crop-animal-human continuum. Presidential Address, Section of Agriculture and Forestry Sciences, 92th Session of the Indian Science Congress Association. Ahmedabad, Gujarat, India.

- [7] BGS, Technical Report WC/00/19, British Geological Survey, UK, 2: 77(2001).
- [8] Engconsult Limited, Toronto, Canada, (2002). <http://www.engconsult.com/arsenic/arsstat.html> [accessed 22 June 2009].
- [9] SOES-DCH, School of Environmental Studies, Jadavpur University, Calcutta and Dhaka Community Hospital, Dhaka, 2000.
- [10] Fazal M. A., Kawachi T., and Ichion E., Water International 26, 370 (2001). <http://dx.doi.org/10.1080/02508060108686929>.
- [11] New Age, Op-Ed page, Dhaka, Bangladesh, September 14, (2005). <http://www.newagebd.com/2005/sep/14/oped.html>
- [12] Statistical year book of Bangladesh, Bangladesh Bureau of Statistics, Statistics Division, Ministry of Planning, Peoples Republic of Bangladesh. P. 10.(1996).
- [13] Khan A. W., Ahmad S. A., Sayed M.H.S.U., Hadi. S.A., Khan M. H., Jalil M. A., Ahmed R., and Faruque M. H., (1998), Arsenic contamination in ground water and its effect on human health with particular reference to Bangladesh, Presented in International Conference on Arsenic Pollution of Ground water in Bangladesh. (8-12 Feb. 1998), p. 109.
- [14] WHO. Arsenic and arsenic compounds. Environmental Health Criteria 22. 2nd ed, Geneva, World Health Organization, (2001).
- [15] Duxbury JM., Mayer AB., Laure JG., Hassan N. Food chain aspects of arsenic contamination in Bangladesh: Effects on quality and productivity of rice. J Environ Sci Health A Tox Hazard subst Environ Eng. 2003; 38: 61-69.
- [16] Bhattacharya P., Samal A.C., Majumdar J., Santra S.C.(2009): Accumulation of arsenic and its distribution in rice plant (*Oryza sativa* L.) in Gangetic West Bengal, India. Paddy and Water Environment doi:10.1007/s10333-009-0180-z

- [17] FAO (1985) Water quality guidelines for maximum crop production. Food and Agricultural Organization/UN.www. Fao.org /docrep/T0551E.2006/9/13
- [18] Baig J.A., Kazi T.G., Shah A.Q., Kandhro G.A., Afridi H.I., Arain M.B., Jamali M.K. and Jalbani N. (2010): Speciation and evaluation of Arsenic in surface and ground water samples: a multivariate case study. *Ecotoxicol. Environ. Safety*, 73: 914-923.
- [19] Guha-Mazumder D.N., Haque R., Ghose N., De B.K., Santra A. and Chakraborty D. (2000): Arsenic in drinking water and the prevalence of respiratory effects in West Bengal, India. *Int. J. Epidemiology*, 29: 1047-1052.
- [20] Morales K.H., Ryan L., Kuo T.L., Wu M.M. and Che, C.J.(2000): Risk of internal cancers from arsenic in drinking water. *Environmental Health Perspectives*, 108: 655-661.
- [21] Feldstein L.A. (1986).Cumulative exposure to arsenic and its relationship to respiratory cancer among copper smelter employees. *Journal of Occupational Medicine*, 28, 296 – 302.
- [22] Smith A.H., Lingas E.O., Rahman M. Contamination of drinking-water by arsenic in Bangladesh: a public health emergency. *Bull world Health Organ.* (2000); 78: 1093-103.
- [23] Abedin M.J., Cresser M.S., Mcharg A.A., Feldmann J. and Howells J. C. (2002). Arsenic accumulation and metabolism in rice (*Oryza sativa* L.). *Environmental Sci. Technol.*, 36: 962-968.
- [24] Das H. K, Mitra A. K, Sengupta P. K, Hossain A, Islam F, Rabbani G.H Arsenic concentrations in rice, vegetables, and fish in Bangladesh ; a preliminary study.
- [25] Huang R. Q., Gao S. F., Wang W. L., Staunton S., & Wang G. (2006). Soil arsenic availability and the transfer of soil

arsenic to crops in suburban areas in Fujian Province, southeast China. *Science of the Total Environment*, 368, 531– 541.

- [26] Dahal B. M., Fuerhacker M., Mentler A., Karki K. B., Shrestha R. R., & Blum W. E. H. (2008). Arsenic contamination of soils and agricultural plants through irrigation water in Nepal. *Environmental Pollution*, 155, 15
- [27] Liu W. X., Shen L. F., Liu J. W., Wang Y. W., & Li S. R. (2007). Uptake of toxic heavy metals by rice (*Oryza sativa* L.) cultivated in the agricultural soil near Zhengzhou city, People's Republic of China. *Bulletin of Environmental Contamination and Toxicology*, 79, 209– 213.
- [28] Norra S., Berner Z.A., Agarwala P., Wagner F., Chandrasekharam D. & Stüben D. (2005). Impact of irrigation with arsenic rich groundwater on soil and crops: A geochemical case study in West Bengal delta plain, India. *Applied Geochemistry*, 20, 1890 – 1906.
- [29] Rahman M. A., Hasegawa H., Rahman M. M., Rahman M.A., & Miah M. A. M. (2007). Accumulation of arsenic in tissues of rice plant (*Oryza sativa* L.) and its distribution in fractions of rice grain. *Chemosphere*, 69, 942– 948.
- [30] Roychowdhury T., Tokunaga H. and Ando, M. 2003. Survey of arsenic and other heavy metals in food composites and drinking water and estimation of dietary intake by the villagers from an arsenic-affected area of West Bengal, India. *Sci. Total Environment*, 308: 15-35.
- [31] Díaz O.P., Leyton I., Muñoz O., Núñez N., Devesa, V., Súnier M.A., Vélez D. and Montoro R. 2004. Contribution of water, bread

and vegetables (raw and cooked) to dietary intake of inorganic arsenic in a rural village of Northern Chile. *J. Agric. Food Chem.*, 52: 1773-1779.

- [32] Alam M.G.M., Snow E.T. and Tanaka A. 2003. Arsenic and heavy metal contamination of vegetables grown in Samta village, Bangladesh. *Sci. Total Environment*, 308 (1/3):83-96.
- [33] Kundu R. and Pal S. 2010. Arsenic accumulation in potato cultivars under arsenic affected belt of West Bengal. *Proc. Nat. Symp. Biotechnol. Environment*, NIT, Durgapur, West Bengal, pp.364-367.
- [34] Lauren J.G. and Duxbury J.M. (2010). Management Strategies to reduce Arsenic uptake by Rice.
- [35] Xie Z.M. and Huang C.Y. (1998): Control of arsenic toxicity in rice plants grown on an arsenic-polluted paddy soil. *Commun. Soil Sci. Plant Anal.* 4:2471-2477.
- [36] Rose. Z., Duxbury J.M., Paul D.N.R. and Gloria De.S.D. (2005): Potential for arsenic contamination of rice in Bangladesh: Spatial analysis and mapping of high risk areas. *Int. J. Risk assessment and management*. In press.
- [37] Liu.W-J.,Zhu Y-G, Smith .F.A. and Sith S.E.(2004): Do phosphorus nutrition and iron plaque alter arsenate (As) uptake by rice seedling in hydroponic culture? *New Phytologist* 162: 481-488. 2005. Do iron plaque and genotypes affect arsenate uptake and translocation by rice seedling (*Oriza sativa* L.) grown in solution culture? *J. Exp. Botany*. 55(403): 1707-1713.
- [38] UNICEF WES Section, Headquarters (2007): Water, Sanitation and Hygiene Annual Report. New York: UNICEF. (available from the WES Section, NYHQ upon request).

- [39] Dr. Davin and Sokchea Mr. Hak, Institute of Technology of Cambodia.(2012): Accumulation of Arsenic in Fruits and vegetables Grown in Arsenic-contaminated Areas in Cambodia.
- [40] J.P.F.D' Mello Food Safety: Contaminants and Toxins. CABI publishing, Wallingford, Oxon, UK, Cambridge, MA. 480(2003).
- [41] Mukesh K. Raikwar, Punnet Kumar, Manoj Singh and Anand Singh, Toxic effect of heavy metals in livestock health, Indian Veterinary World, January 2008, Vol.1(1):28-30, pp-28.
- [42] Available at: <http://www.tip2000.com/health/waterpollution.asp>
[accessed September 20, 2011].
- [43] U.C. Gupta, S.C. Gupta. Trace elements toxicity relationship to crop production and livestock and human health: Implication for management. Commun Soil Sci Plant Anal, 29,1491-1522 (1998).
- [44] Li P.Y., Qian H., Human health risk assessment for chemical pollutants in drinking water source in shizuishan city, J. Environ. Health. Sci. Eng.,Vol.8, No.1, 2011,pp. 41-48. No.1, 2011,pp.41-48.
- [45] Jarup L, Hazards of heavy metal contamination. Medical British Journal, 2003, 68: 167-182.
- [46] Jasim Uddin Ahmed and Md. Abdul Goni, Heavy metal contamination in water soil, and vegetables of the industrial areas in Dhaka, Bangladesh, Earth and Environmental Science Environmental Monitoring and Assessment, Volume 166, Numbers 1-4, pp. 347-357.
- [47] Donadinil G., Spalla S. and Beone G.M., Arsenic, Cadmium and Lead in Beers from the Italian Market, J. Inst. Brew. (2008), 114(4), 283-288.
- [48] Qin F., Chen W. Lead and Copper levels in tea samples marketed in Beijing, China. Bull Environ Contam Toxicol..78, 128-131 (2007).

- [49] Sing A., Sharma R.K., Agrawal M. and Marshall F.M., (2010): Risk assessment of heavy metal toxicity through contaminated vegetables from waste water irrigated area of Varanasi, India. *Trop. Ecol.*, 51: 375-387.
- [50] Kachenko A.Y.G., Singh B. Heavy metals contamination in vegetables grown in urban and metal smelter contaminated sites in Austilia, *Water, air and Soil pollution*, 169, 101-123 (2006).
- [51] Das A.K.. Metal ion induced toxicity and detoxification by chelation therapy. In *A text book on medical aspects of bio-inorganic chemistry*, 1st ed, CBS, Delhi. 17-58 (1990).
- [52] Khan S.I., Ahmad A.K.M., Yunus M., Rahman M., Hore S.K., Vahter M., Wahed M.A., Arsenic and cadmium in food chain in Bangladesh-an exploratory study.
- [53] M.A. Radwan, A.k. Salama. Market Basket 216 Survey for some heavy metals in Egyptian fruits and vegetables. *Food & Chemical Toxicology*. 44(8), 1273-1278(2006).
- [54] X.wang,T. Sato, B.Xing, S. Tao. Health risk of heavy metals to the general public in Tianjan, China via consumption of vegetables and fish. *Sci Tot Environ* 350 (1-3), 28-37 (2005).
- [55] S.Khan, Q. Cao, Y.M. Zheng, Y.Z. Huang, Y.G. Zhu. Health risk of heavy metals in contaminated soils and food crops irrigated with waste water in Beijing, China. *Environ pollut* 152(3), 686-692(2008).
- [56] Narottam Saha and M.R. Zaman, Concentration of selected toxic metals in groundwater and some cereals grown in Shibganj area of Chapai Nawabganj, *Current science*, vol. 101, no. 3, 10 august 2011, pp-427.
- [57] M.S. Jassir, A. Dhaker, M.A. Khaliq. Deposition of heavy metals on green leafy vegetables sold on road side of Riyadh city, Saudi

Arabia. Bull. Environ. Contam Toxicol 75, 1020-1027(2005).

- [58] Khairiah T. Zalifae M.K Yin Y.H. Aminath A. The uptake of heavy metals by fruit type vegetables grown in selected agricultural areas. Pak J Biol Sc 7(2), 1438-1442(2004).
- [59] Singh S., Kumar M. Heavy metal load of soil, water and vegetables in periurban Delhi. Environ. Monitor Assess 120,71-79 (2006).
- [60] Sharma R.K., Agarwal M., Marshal F.M. Atmospheric deposition of heavy metals (Cd, Pb, Zn & Cu) in various city. India. Environ Monit Assess 142(1-3),269-278(2008).
- [61] Singh K.P., Mohan D., Sinha S., Dalwan R. Impact assessment of treated /untreated wastewater toxicants discharged by sewage treatment plants on health, agricultural and environmental quality in wastewater disposal area. Chmosphere 55,227-255(2004).
- [62] Sharma R.K., Agarwal M., F.M. Marshal. Heavy metal Contamination of soil & vegetables in suburban areas of Varanasi, India. Ecotoxicology and Environmental Safety. 66, 258-266(2007).
- [63] A. Maleki, M.A Zarasvand. Heavy metals in selected edible vegetables and estimation of their daily intake in Sanandaj, Iran. South East Asian J Trop Med public health 39,335-340(2008).
- [64] Albert Cotton F. and Geoffey Wilkson, Advanecd Inorganic Chemistry 5th Edition and Lee J.D.; Concise Inorganic Chemistry 5th Edition.
- [65] Cotton F.A.(1999): "Survey of Transition-Metal Chemistry". Advanced Inorganic Chemistry (6 ed.). John Wiley and Sons. Pp. 633. ISBN 0471199575.
- [66] Available:<http://www.chemistryexplained.com/elements/AC/Arsenic.html#ixzz1e1cKz0F1> [accessed November 24, 2011]
- [67] Colin Baird, Environmental chemistry.4th Edition.

- [68] Linus, Pauling (1947). General Chemistry. Freeman W.H. ISBN 0486656225.
- [69] Windholz, Martha (1976). Merck Index of Chemicals and Drugs, 9th ed., monograph 8393. Merck. ISBN [Special: Book Sources 091191263| 091191263]
- [70] Available at: <http://www.chemistryexplained.com/elements/A-C/Cadmium.html> [accessed November 30, 2011]
- [71] Ghosh M. M. and Yuan J. R., Adsorption of arsenic and organoarsenicals on hydrous oxides. *Environmental Progress*, 6(3) (1987) 150-157
- [72] Montgomery J. M., Water treatment principles and design. James M. Montgomery Consulting Engineers, Inc., Wiley Interscience Publication, John Wiley & Sons, 1985.
- [73] Gupta S. K. and Chen K. Y., Arsenic removal by adsorption. *Journal Water Pollution Control Federation*, 50(3) (1978) 493-506.
- [74] Dahi E., Environmental chemistry of arsenic. Paper presented at ITN-Bangladesh training course on Water Supply and Waste Management. ITN Center, BUET, Dhaka, Aug. 9-12, 1997
- [75] Ferguson and Davis J., A review of arsenic cycle in natural waters. *J. Water Res.*, 6 (1972) 1259-1274.
- [76] BGS/DPHE, Arsenic contamination of groundwater in Bangladesh. *BGS Technical Report WC/00/19*, Vol 2: Final report. British Geological Survey (UK) and Department of Public Health Engineering (Bangladesh), 2001, pp. 77-207.
- [77] Dzombak D. A. and Morel F. M. M., Surface complexation modeling – hydrous ferric oxide. John Wiley & Sons, New York, 1990.
- [78] Bhumbla D. K. and Keefer R. F., Arsenic mobilization and bioavailability in soils. In: J. O. Nriagu (Ed.), *Arsenic in the Environment*, Part 1, Chapter 3. John Wiley & Sons, London, 1994, pp. 51-81.

- [79] Cvjetanovic B (1986): Health effects and Impacts of Water Supply and Sanitation, World Health Statistics Quarterly, 39(1)pp, 105-117.
- [80] Ravenscroft P., Burgess W.G., Ahmed K.M., Burren M. and J. Perrin (2008): Arsenic in Groundwater of the Bengal Basin Bangladesh: Distribution, field relations and hydrological setting, Hydrology Journal 13pp. 727-51.
- [81] Pankow, James. Aquatic Chemistry Concept. Department of Environmental Science and Engineering. Lewis Publishers. (1991).
- [82] National Association of Physicians for Environment (NAPE). Conference Summanitationry. <http://www.intr.net/napenet/airsum>. Html Thron, (1993).
- [83] Bhumbala D. K. and Keefer R. F. Arsenic mobilization and bioavailability in soils. In: J. O. Nriagu (Ed.), Arsenic in the Environment, Part 1, Chapter 3. John Wiley & Sons, London, (1994), pp. 51-81.
- [84] Arnold H.L, Odam R.B, James W.D. *In: Andrew's Disease of the skin: clinical dermatology*. 8th ed. Philadelphia, PA: Saunders, (1990):121-2.
- [85] Ioanid N, Bors G, Popa I. [Contribution to the knowledge on background arsenic values in fingernails and the values encountered in cases of arsenic polyneuritis]. Zeitschrift der Gesamten Gerichtlichen Medizin (1961);52:90-4 (German).
- [86] Angelo R.T, Cringan M.S, Chamberlain D.L, et al. Residual effects of lead and zinc mining on freshwater mussels in the Spring River Basin (Kansas, Missouri, and Oklahoma, USA). Sci Total Environ. (2007); 384:467–496. [PubMed]

- [87] Brumbaugh W.G, Schmitt C.J., May T.W. Concentrations of cadmium, lead, and zinc in fish from mining-influenced waters of Northeastern Oklahoma: Sampling of blood, carcass, and liver for aquatic biomonitoring. *Arch Environ Contam Toxicol.* (2005); 49:76–88. [PubMed]
- [88] Elinder C.G, Edling C., Lindberg E. et al. Assessment of renal function in workers previously exposed to cadmium. *Br J Ind Med.* (1985a); 42:754–760. [PMC free article] [PubMed]
- [89] EPA. Consumer factsheet on: Cadmium U.S. Environmental Protection Agency. (2006a): http://www.epa.gov/OGWDW/contaminants/dw_contamfs/cadmium.html.
- [90] EPA. Cadmium contamination of the environment: An assessment of nationwide risk. Washington, DC: U. S. Environmental Protection Agency, Office of Water Regulations and Standards; (1985a). EPA 440485023.
- [91] Morrow H. Kirk-Othmer encyclopedia of chemical technology. John Wiley & Sons, Inc.; 2001. Cadmium and cadmium alloys. Pp.471–507. [http://www.mrw.intersciencewile.com/emrw/9780471238966/kirk/article/cadmcar.a01/current/pdf?hd=All%2Ccadmium.\(April%2029%2C2008\).](http://www.mrw.intersciencewile.com/emrw/9780471238966/kirk/article/cadmcar.a01/current/pdf?hd=All%2Ccadmium.(April%2029%2C2008).)
- [92] Chakraborti D, Sengupta M. K, Rahman M. M, Ahamed S, Chowdhury U. K, Hossain M. A *et al.* Groundwater arsenic contamination and its health effects in the Ganga-Meghna-Brahmaputra plain. *J Environ Monit* (2004);6:74-83.
- [93] British Geological Survey. Groundwater studies for arsenic contamination in Bangladesh. Phase I: Rapid investigation phase. Final report. Newcastle upon Tyne: Mott MacDonald Ltd., (1999),S1-S12.

- [94] Saha G. C., Ali, M. A. Dynamics of arsenic in agricultural soils irrigated with arsenic contaminated groundwater in Bangladesh. *Sci. Total Environ.* (2007), 379, 180-189.
- [95] Williams P. N., Villada A., Deacon C., Raab A., Figuerola J., Green A. J., Feldmann J., Meharg A. A. Greatly enhanced arsenic shoot assimilation in rice leads to elevated grain levels compared to wheat and barley. *Environ. Sci. Technol.* (2007), 41, 6854-6859.
- [96] van Geen A., Zheng Y., Cheng Z., He Y., Dhar R. K., Garnier J. M., Rose J., Seddique A., Hoque M. A., Ahmed K. M. Impact of irrigating rice paddies with groundwater containing arsenic in Bangladesh. *Sci. Total Environ.* (2006), 367, 769-777.
- [97] Hossain M. B., Jahiruddin M., Panaullah G. M., Loeppert R. H., Islam M. R., Duxbury J. M. Spatial variability of arsenic concentration in soils and plants, and its relationship with iron, manganese and phosphorus. *Environ. Pollute.* (2008), 156, 739-744.
- [98] Panaullah G. M., Alam T., Hossain M. B., Loeppert R. H., Lauren J. G., Meisner C. A., Ahmed Z. U., Duxbury J. M. Arsenic toxicity to rice (*Oryza sativa L.*) in Bangladesh. *Plant Soil* 2009, 317, 31-39.
- [99] Rahman M. A., Hasegawa, H., Rahman M. M., Miah M. A. M., Tasmin A. Arsenic accumulation in rice (*Oryza sativa L.*): Human exposure through food chain. *Ecotox. Environ. Safe.* 2008, 69, 317-324.
- [100] Adomako E. E., Solaiman A. R. M., Williams P. N., Deacon C., Rahman G., Meharg A. A. Enhanced transfer of arsenic to grain for Bangladesh grown rice compared to US and EU. *Environ. Int.* (2009), 35, 476-479.

- [101] Duxbury J. M., Panaullah G. M. Remediation of arsenic for agriculture sustainability, food security and health in Bangladesh. Water.
- [102] Khan M.A., Islam M.R., Panaullah G.M., Duxbury J.M., Jahiruddin M., Loeppert R. H. Fate of irrigation-water arsenic in rice soils of Bangladesh. *Plant Soil* 2009, 322, 263-277.
- [103] Li R. Y., Stroud J. L., Ma J. F., McGrath S. P., Zhao F. J. Mitigation of arsenic accumulation in rice with water management and silicon fertilization. *Environ. Sci. Technol.* (2009), 43, 3778-
- [104] Khan M. A., Islam M. R., Panaullah G. M., Duxbury J. M., Jahiruddin M., Loeppert R. H. Accumulation of arsenic in soil and rice under wetland condition in Bangladesh. *Plant Soil* (2010), DOI: 10.1007/s11104-010-0340-3.
- [105] Zavala Y. J., Duxbury J. M. Arsenic in rice: I. Estimating normal levels of total arsenic in rice grain. *Environ. Sci. Technol.* (2008), 42, 3856-3860.
- [106] WHO (1995) Environmental Health Criteria 165: Inorganic Lead, Geneva: International Programme on Chemical Safety.
- [107] Cook M. E., and Morrow H., (1995) "Anthropogenic Sources of Cadmium in Canada," National Workshop on Cadmium Transport Into Plants, Canadian Network of Toxicology Centres, Ottawa, Ontario, Canada, June 20-21, 1995.
- [108] Eggenberger U. and Waber H. N., (1998). "Cadmium in Seepage Waters of Landfills: A Statistical and Geochemical Evaluation, "Report of November 20, 1997 for the OECD Advisory Group on Risk Management Meeting, February 9-10, 1998, Pads.
- [109] Mench M. J., (1998). "Cadmium availability to plants in relation to major long-term changes in agronomy systems." *Agricult. Ecosyst. Environ.* 67: 175-187.

- [110] Narottam Saha and M.R. Zaman Concentration of selected toxic metals in ground water and some cereals grown in Shibganj area of Chapai Nawabganj, *Current science*, vol.101, no.3,10 august (2001), pp-427.
- [111] O' Flaherty E.J. (1995) Physiologically based models for bone-seeking elements. V. Lead absorption and disposition in childhood. *Toxicol.Appl. Pharmacol.*, 131, 297-308. O' Flaherty, E.J. (1991) Physiologically based models for bone-seeking elements. III. Lead absorption and disposition in childhood. *Toxicol. Appl.*
- [112] Agency for Toxic Substances and Disease Registry (1997) *Toxicological Profile for Lead*, Atlanta G.A: Department of Health and Human Services.
- [113] Rabinowitz M.R., Wetherill G.W. & Kopple J.D. (1976) Kinetic analysis of lead metabolism in healthy humans. *J. Clin. Invest.*, 58,260-170.
- [114] Klaassen C. D., Liu J. and Choudhuri S. "Metallothionein: an intracellular protein to protect against cadmium toxicity," *Annual Review of Pharmacology and Toxicology*, vol. 39, pp. 267–294, (1999), [View at Publisher](#) · [View at Google Scholar](#) · [View at PubMed](#) · [View at Scopus](#).
- [115] Jin T., Lu J. and Nordberg M. "Toxicokinetics and biochemistry of cadmium with special emphasis on the role of metallothionein," *NeuroToxicology*, vol. 19, no. 4-5, pp. 529–536, (1998). [View at Scopus](#).
- [116] Rossini A., Rapozo D. C. M., Amorim et al. L. M. F. "Frequencies of GSTM1, GSTT1, and GSTP1 polymorphisms in a Brazilian population," *Genetics and Molecular Research*, vol.1, no.3, pp. 233–240, (2002). [View at Scopus](#).

- [117] N. Ballatori, "Transport of toxic metals by molecular mimicry," *Environmental Health Perspectives*, vol.110, no.5, pp.689–694, (2002). View at Scopus.
- [118] Civantos D.P., Lopez Rodriguez A, Aguado-Borruey J.M. & Narvaez J.A., Fulminant malignant arrhythmia and multi organ failure in acute arsenic poisoning. *Chest*, 108(6):1774-1775)
- [119] Ociel Muñoz, Vicenta Devesa, María Angeles Suñer, Dinoraz Vélez, Rosa Montoro, Inés Urieta, Mari Luz Macho, and Mercedes Jalón; Total and Inorganic Arsenic in Fresh and Processed Fish Products; *J. Agric. Food Chem.*, 48 (9), pp 4369–4376, (2000).
- [120] Ghosh A.K., Bhattacharyya P., Pal R. Effect of arsenic contamination on microbial biomass and its activities in arsenic contaminated soils of Gangetic West Bengal, India.Vol.30.June (2004).Pages 491-499.
- [121] Yusuf A.A., Arowolo T.A. and Bamgbose O. (2003). Cadmium, copper and nickel levels in vegetables from industrial and residential areas of Lagos City, Nigeria. *Food Chem. Toxicol.*, 41:
- [122] Marshall, (2004), Enhancing food chain integrity: Quality assurance mechanism for air pollution impacts on fruits and vegetables systems. Crop Post Harvest Program, Final Technical Report (R7530)]
- [123] Khan S., Q. Cao Zheng Y.M., Huang Y.Z. and Zhu Y.G. 2008. Health risk of heavy metals in contaminated soils and food crops irrigated with waste water in Beijing, China. *Environ. Pollut.*, 152: 686-692.
- [124] Patrick L. Toxic metals and antioxidants: Part II. The role of antioxidants in arsenic and cadmium toxicity. *Altern Med Rev.* 2003;8:106–28.

- [125] Jarup L., 2003. Hazards of heavy metal contamination. Br. Med. Bull., 68: 167-182
- [126] Yao W., Millero F.J. Adsorption of phosphate on manganese dioxide in seawater. Environ. Sci. Technol. 30 (1996) 536-541.
- [127] Ruixia L., Jinlong G., Hongxiao T. Adsorption of fluoride, phosphate and arsenate on a new type of ion exchange fiber. J. Colloid Interface Sc (2002) 268-274.
- [128] U.S. EPA.1994a. U.S. Environmental Protection Agency. Integrated Risk Information System (IRIS) (Online). 1, 2, 4-Trichlorobenzene. Office of Health and Environmental Assessment, USEPA, Cincinnati, OH.
- [129] Saha, K. C. Chronic arsenical dermatoses from tube-well water in West Bengal during 1983-87. Indian J. Dermatology. 40(1), pp 1-12, 1995.
- [130] WHO (1981) Arsenic. Environmental Health Criteria 18 Geneva, World Health Organization. Pp-174.
- [131] WHO (2001) Arsenic and arsenic compound. Environmental Health Criteria 22 (Second edition). Geneva, World Health Organization
- [132] Brayer AF, Callahan CM, & Wax PM (1997) acute arsenic poisoning from ingestion of "snakes". Pediatric Emergency Care, 13(6): 394-396).
- [133] Levin-Scherz JK, Patrick JD, Weber FH, et al. 1987. Acute arsenic ingestion. Ann Emerg Med 16(6): (702-704).
- [134] Benramdane L, Accominotti M, Fanton L, 1999a. Arsenic speciation in human organs following fatal arsenic trioxide poisoning a case report. Clin. Chem. 45(2):301-306).
- [135] Cullen N.M., Wolf L.R. & Clair St. D. (1995): Pediatric arsenic ingestion. Am J Emerg Med, 13:432-435.

- [136] NRC (1999) United States Nation Research Council. Arsenic in drinking water. National Academy Press, Washington DC.310.
- [137] Armstrong BK, McNulty JC, Levitt LJ, Williams KA, & Hobbs MST (1979) Mortality in gold and coal miners in Western Australia with special reference to lung cancer. *Br J Ind Med*, 36:199-205).
- [138] Lai M.S., Hsueh Y.M., Chen C.J., Shyu M.P, Chen S.Y., Kuo T.L., Wu M.M. & Tai T.Y. (1994) Ingested inorganic arsenic and prevalence of diabetes mellitus. *Amer J Epidemiol*, 139:484-492.
- [139] Tsai S.M., Wang T.N., Ko Y.C.(1999).Mortality for certain diseases in areas with high levels of arsenic in drinking water. *Arch Environ Health* 54(3):186-193.
- [140] Zaldivar R. (1974). Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning. *Beitr Path Bd*, 151: 384-400.
- [141] Tseng C.H., Tseng C.P., Chiou Y.M., Chong C.K., Chen C.J., Epidemiologic evidence of diabetogenic of arsenic. *Toxicology Letters* 133 (2002).
- [142] Lewis D.R, Southwick J.W, Ouellet-Hellstrom R, Rench J, Calderon R.L. Drinking water arsenic in Utah: A cohort mortality study. *Environ Health Perspect.* 1999 May; 107(5):359-65.
- [143] Tseng C.H, Tai T.Y, Chong C.K, et al. 2000. Long-term arsenic exposure and incidence of non-insulin dependent diabetes mellitus: A cohort study in arseniasis-hyper endemic villages in Taiwan. *Environ Health Perspect.* 108(9):847-851.
- [144] Rahman M & Axelson O (1995), Diabetes mellitus and arsenic exposure: a second look at case-control data from a Swedish copper smelter. *Occupy Environ Med*, 52: 773-774.
- [145] Axelson O, Dahlgren E, Jansson CD, & Rehnlund SO (1978) Arsenic exposure and mortality: a case-referent study from a

Swedish copper smelter. Br J Ind Med, 35: 8-15.

- [146] Hindmarsh J, McLetchie OR, Heffernan LPM, Hayne OA, Ellenberger HA, McCurdy RF, & Thiebaut HJ (1977) Electromyographic abnormalities in chronic environmental arsenicalism. Clin Chem and Chem Toxicol of Metals, 1: 287-293.
- [147] Feldman RG, Niles CA, Kelly-Hayes M, Sax DS, Dixon WJ, Thompson DJ, & Landau E (1979) Peripheral neuropathy in arsenic smelter workers. Neurology, 29: 939-944.
- [148] Available at: <http://www.chemistryexplained.com/elements/L-P/Lead.html> [accessed November 28, 2011]
- [149] U.S. Department of Human and Health Services. Public Health Services Agency for Toxic Substances. Toxicological Profile for Lead. (August, 2007).
- [150] Guy R., Hostynek J.J.; Hinz R.S. and Lorence C.R.. Metals and the Skin: Topical Effects and Systemic Absorption. New York, New York. 1999., Hostynek, J.J. Lead, Manganese, and Mercury: Metals in Personal-Care Products. Cosmetics and Toiletries Magazine. Volume 116, no. 8. August 2001.
- [151] Health Canada, Cosmetics Division. Lead Acetate Risk Assessment. 2006.
- [152] Hostynek J.J. Lead Manganese and Mercury: Metals in Personal-Care Products. Cosmetics and Toiletries Magazine; Volume 116, pp no. 8. August 2001.
- [153] Health Canada. It's Your Health: Effects of Lead on Human Health. November 2008.
- [154] Available at : [Http://www.chemistryexplained.com/elements/A-C/Cadmium.htm1#ixzz1elksNxzm](http://www.chemistryexplained.com/elements/A-C/Cadmium.htm1#ixzz1elksNxzm) [accessed December 5, 2011]

- [155] Available at: <http://www.chemistryexplained.com/elements/L-P/Lead.html#b> [accessed November 29, 2011]
- [156] BGS/DPHE, Arsenic contamination of groundwater in Bangladesh. BGS Technical Report WC/00/19, Vol 2: Final report. British Geological Survey (UK) and Department of public Health Engineering (Bangladesh), 2001, pp. 77-207.
- [157] Geological Survey (UK) and Department BGS/DPHE, 2001. Arsenic contamination of groundwater in Bangladesh. BGS Technical Report WC/00/19, Vol 2: Summary. British of public Health Engineering (Bangladesh).
- [158] Ahmed M.F. Arsenic contamination in Bangladesh: severity of the problem and possible consequences. In: Proc. Arsenic in the Asia-Pacific region workshop, Adelaide, Australia, Nov. 20-23-2001.
- [159] Quamruzzaman Q., Rahman M., Das R., Uddin K. J., Mohiuddin G., and Chakraborti S., Sustainable management of Bangladesh arsenic problem. In: Proc. Int. conf. on As contamination of ground water in Bangladesh: cause, effect and remedy. Khaka, Bangladesh, Jan. 12-13 2002.
- [160] Eng Consult Limited, Arsenic calamity of Bangladesh. In: Statistics of arsenic calamity. Toronto, Canada, 2003. (Cited the sources of BBS, Dhaka Community Hospital, NIPSOM and DPHE). Available: <http://www.eng-consult.com/arsenic/arsstat.html> (accessed 10 February)
- [161] ATSDR (2008). *Draft Toxicological Profile for Cadmium*. Atlanta, Georgia: US Department of Health and Human Services.

CHAPTER TWO :EXPERIMENTAL

2.1. EXPERIMENTS:

Experiment comprises the following features sequentially:

- ◆ The materials and reagents used in this investigation
- ◆ Instrumentation that comprises system description and operating conditions of the principal instruments involved
- ◆ Theories related to the measurement of the parameters involved
- ◆ The methodologies of water, plant tissue, fish, dust and soil sampling and analysis
- ◆ The adapted techniques for analysis of samples for physical, chemical and biological parameters
- ◆ Quality control (QC) or quality assurance steps taken
- ◆ The statistical analyses of the data obtained

2.1.1. MATERIALS:

All chemicals and reagents used in this investigation were of high purity analytical grade. Distilled deionized water (DDW) was used throughout. Several standard solutions, reducing solutions, buffer solutions, ternary acid mixture and other solutions were prepared according to the standard procedures [1, 2, 3, 4, 5]. All chemicals or reagents were used in this investigation without further purification.

2.1.2. Standard Stock Solutions:

i) Arsenic (As^{3+}) [1000 ppm]: 1.320 g of As_2O_3 (Fluka, Switzerland) was dissolved in 20 mL of DDW containing 4 g of NaOH (Merck, Germany). After dissolution, it was diluted to 200 mL followed by adjustment of pH to 5.5 with 2M HCl (Merck, Germany) and finally diluted with DDW to 1000 mL.

ii) Lead (Pb) [1000 ppm]: 1.599 g of $\text{Pb}(\text{NO}_3)_2$ was dissolved in about 150 mL of distilled de-ionized water. 10 mL of concentrated HNO_3 was added to it and diluted up to 1000 mL with DDW.

iii) Cadmium (Cd) [1000 ppm]: 2.036 g of CdCl_2 was dissolved in 250 mL of distilled de-ionized water. After dissolution, it was diluted up to 1000 mL.

iv) Iron (Fe^{3+}) [200 mg L^{-1}]: 20 mL of 98% H_2SO_4 (Aldrich, USA) was added slowly to 50 mL of DDW and 1.404 g of ferrous ammonium sulfate [$\text{Fe}(\text{NH}_4)_2(\text{SO}_4)_2 \cdot 6\text{H}_2\text{O}$] (BDH, England) was dissolved in it. Then 0.1 N KMnO_4 (Merck, Germany) solution was added drop wise until a faint pink color persisted. This was diluted with DDW to 1000 mL. Freshly prepared standard iron solution was used for calibration purpose.

v) Calcium (Ca^{2+}) [500 mg L^{-1}]: To 1.249 g of CaCO_3 (Merck, Germany), previously dried at 180°C for 1 h, 50 mL of DDW was added. The suspension was dissolved by adding 10.0 mL of concentrated HCl drop wise and diluted to 1000 mL with the water.

vi) Magnesium (Mg^{2+}) [1000 mg L^{-1}]: 1.658 g of MgO (Aldrich, USA) was dissolved in a minimum amount of 1:1 HNO_3 . 10.0 L of concentrated HNO_3 was added to it and diluted to 1000 mL with DDW.

vii) Manganese (Mn^{2+}) [1112.4 mg L^{-1}]: In a 1 L beaker, 3.20 g of KMnO_4 (Merck, Germany) was taken. It was dissolved with 1 L of distilled water and heated the solution at $90\text{--}95^\circ\text{C}$ for several hours. After cooling and filtering to 1 L measuring flask, it was then diluted up to mark with DDW.

viii) Sodium (Na^+) [1000 mg L^{-1}]: 2.542 g of NaCl (Fluka, Switzerland), previously dried at 140°C for 4 h, was dissolved in DDW and diluted to 1000 mL.

ix) Potassium (K^+) [1000 mg L^{-1}]: 1.907 g of KCl (Merck, Germany), previously dried at 110°C for 4 h, was dissolved in DDW and diluted to 1000 mL.

x) Chloride (Cl^-) [500 mg L^{-1}]: In a 500 mL measuring flask, 0.412 g of NaCl (Fluka, Switzerland) that was dried previously at 140°C was taken. It was then dissolved with DDW and diluted up to the mark.

xi) Fluoride (F^-) [100 mg L^{-1}]: In a 500 mL measuring flask, 0.221 g of anhydrous NaF (Merck, Germany) was taken. It was then dissolved with DDW and diluted up to the mark.

xii) Sulfate (SO_4^{2-}) [100 mg L^{-1}]: In a 1 L measuring flask, 0.1479 g of anhydrous Na_2SO_4 (BDH, England) was taken. It was then dissolved with DDW and diluted up to the mark.

The stock solutions were further diluted with appropriate amount of distilled deionized water (DDW) to prepare standard solutions of the desired concentrations.

2.1.3. Ternary Acid Mixture:

In order to prepare ‘ternary acid mixture’ three concentrated acids, namely, 69% HNO_3 , 98% H_2SO_4 and 60% HClO_4 were mixed together in the volume ratio of 10:1:4.

2.1.4. Other Solutions:

i) HCl solution: 2M HCl solution was made by taking 82.5 mL of 37 % HCl in a 500 mL measuring flask and filling up to the mark with DDW. 5M HCl solution was also prepared similarly. 1.5% (v/v) HCl solution was also prepared by adding 15 mL of conc. HCl to DDW and made up to 1000 mL.

ii) NaOH solution: 1%(w/v) NaOH solution was prepared by dissolving 10 g of NaOH pellets in DDW and made up to 1000 mL.

iii) Model Water: Shallow tube-well water supplemented with appropriate amounts of the standard arsenic, lead and cadmium solutions was used as model water for irrigation purposes.

Besides these, a few special solutions, prepared according to standard procedure, were utilized.

2.1.5. Other Materials:

The seeds of the vegetables were bought from regional office of BARC (Bangladesh Agricultural Research Council). The natural organic fertilizers were collected from the local farmers.

The commercial gases (99.99% pure nitrogen and acetylene) used were collected from BOC, Bangladesh and 99.999% pure argon gas from Singapore. Cultural media (OXOID, UK) was used for counting Fecal Coliform bacteria. Some reagents of HACH, USA were also used.

2.1.6. Standard Reference Materials:

Two standard reference materials (SRM) were used to check the accuracy of the methods. Tomato Leaves (SRM 1573a) were from the U.S. National Institute of Standards and Technology (Gaithersburg, MD) and Pond Sediment (NIES 2) from the National Institute for Environmental Studies (Japan).

2.2. INSTRUMENTATION:

2.2.1 Graphite Furnace – Atomic Absorption Spectroscopy (GF-AAS):

a. System Description: Total arsenic, lead and cadmium concentrations of water as well as plant tissues and soil digestates were analyzed by GF-AAS, using a Shimadzu AA-6800 (Kyoto, Japan) atomic absorption spectrometer. It is a double beam AA spectrometer and capable of performing automated single element analysis. The system is equipped with a computer with Microsoft® Windows CE® compatible “WinLab32™” software and a Canon BJC-2100SP printer. It is also coupled with an auto-sampler, ASC-6100 (Shimadzu, Japan).

The basic features of Shimadzu AA-6800 spectrometer system include: i) Light sources, ii) Graphite Furnace Atomizer, iii)

Monochromator, iv) Detector and v) Display system. One type of line radiation source (lamp) was used in the instrument – Lumina™ Hollow Cathode Lamp (HCL). The furnace atomizer or burner system is of laminar or premix type and comprises the following components: i) Air-cooled premix type Burner, ii) Burner head, iii) Nebulizer, iv) Chamber (Pyrolytic graphite tube), v) a detector (generally a photomultiplier tube) and vi) Gas control system (incorporated in the spectrometer).

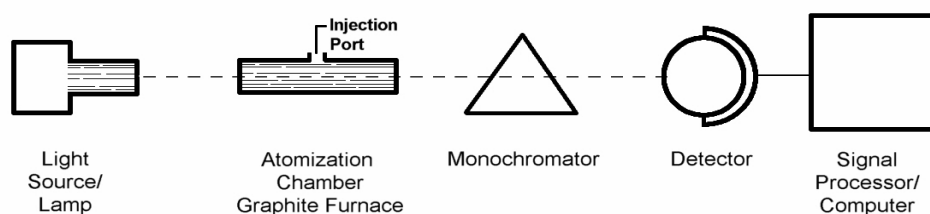


Fig 2.1: Diagram of the basic components of a Graphite Furnace Atomic Absorption Spectrometer.

Graphite furnace atomic absorption spectrometry (GF-AAS) has become a popular analytical technique in recent years, mainly because of its enhanced sensitivity that allows measurement in the picogram (10^{-12} g) range.

GF-AAS operates in the wavelength range from 190 to 900 nm and includes a Littrow configuration grating monochromator. The monochromator offers a choice of six spectral slit widths (0.1, 0.2, 0.5, 1.0, 2.0 and 5.0 nm). It provides excellent detection limits for concentrations of metals in aqueous and solid samples. As a direct result of the high sensitivity, another practical problem that must be faced is the control of external contamination. Contamination can be introduced to standards and samples alike and come from a variety of sources, including reagents (such as matrix modifier), solvents (used in dissolution procedures), the laboratory atmosphere and contaminated glassware. Taken together these problems can give the impression that GF-AAS is a

technique that many complex and poorly understood steps to be made before an analytical method can be successfully developed and proved.

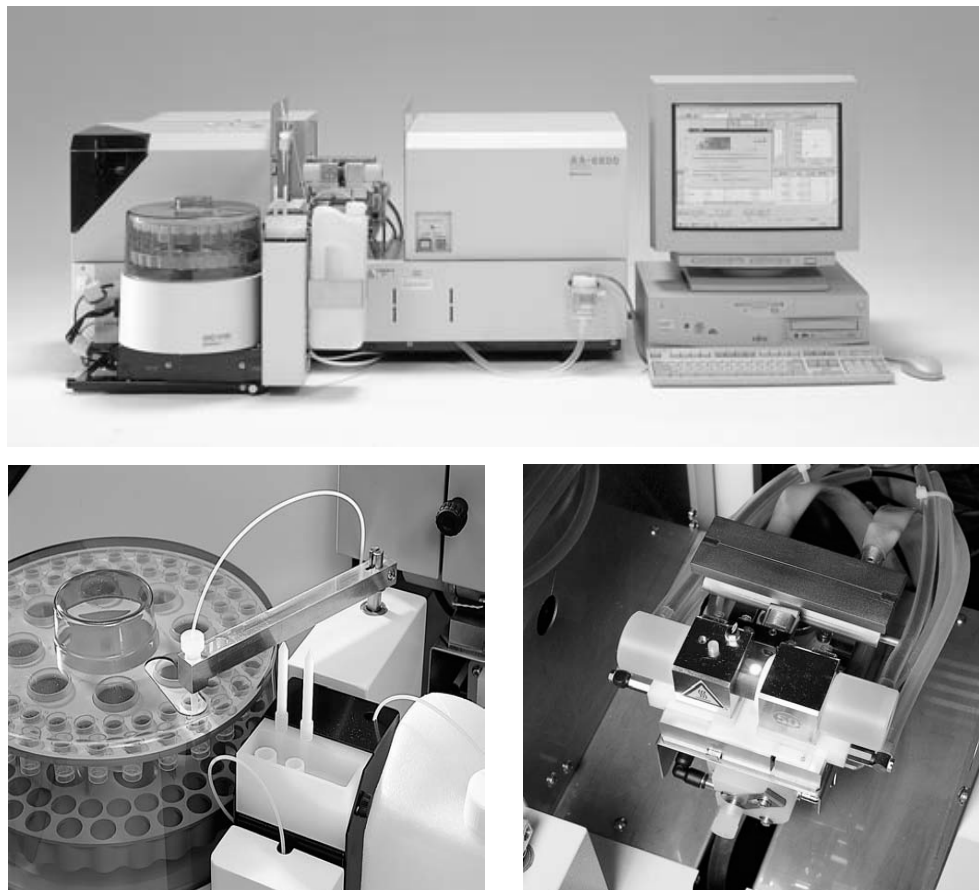


Figure: 2.2 Graphite Furnace - Atomic Absorption Spectroscopy (GF-AAS)

The system contains high speed Deuterium Arc Background corrector to compensate spectral interferences caused by molecular absorption and particulate caused light-scattering [6].

b. Optical System: The GF-AAS features a true electric optical double-beam system that compensates for changes that may occur in lamp intensity during an analysis. The light source (HCL) emits a sharp atomic line (spectrum) specific to the element of which it is made and focused through sample cell into the monochromator. Source modulation can be accomplished with a rotating chopper in order to accomplish selective amplification of the lamp emission signal. In the monochromator, the wavelength of light are dispersed by high-dispersion dual-blazed grating and the analytical line of interest is isolated through exit slit and focused

onto the detector. The detector PMT generates an electrical current depending on the light intensity. The instrument electronics further measures the amount of light attenuation in the sample cell based on signal to noise ratios and convert those reading to the actual sample concentration.

The spectrometer uses a deuterium (D_2) arc lamp that is installed internally to correct for background absorbance caused by non-atomic species in the atom cloud. When the deuterium arc background corrector is used, two alternating sets of measurements are made: one with the primary source and a second with the background corrector source. The primary source measures a combination of background and atomic absorption. The continuum source measures only the background. The difference between the two measurements yields the atomic concentration.

c. The instrumental conditions for the determination of Arsenic (As), Cadmium (Cd) and Lead (Pb) concentrations are summarized in the following table.

Table 2.2.1. Operational conditions of AAS.

Element	As	Cd	Pb
Wavelength (nm)	193.7	228.8	283.3
Slit width (nm)	0.2	0.2	1.0
Lamp mode	BGC-D2	BGC-D2	BGC-D2
Lamp	HCL	HCL	HCL
Lamp current (mA)	12	8	10
Measurement mode	Peak height	Peak height	Peak height
Standard ($\mu\text{g L}^{-1}$)	0, 4, 12, 20	0, 0.1, 0.3, 0.5	0, 4, 12, 20
Calibration curve	Linear	Linear	Linear
Correlation coefficient	0.999	0.999	0.999
Replicate	3	3	3
Mixture modifier	10 ppm $\text{Pd}(\text{NO}_3)_2$	10 ppm $\text{Pd}(\text{NO}_3)_2$	10 ppm $\text{Pd}(\text{NO}_3)_2$
Sample volume (μL)	20	20	20

Table 2.2.2. Temperature program for GF-AAS.

Element	Step	Temperature (°C)	Time (sec)	Heating mode	Flow rate (L min ⁻¹)
As	1	150	20	RAMP	0.10
	2	250	10	RAMP	0.10
	3	600	10	RAMP	1.00
	4	600	10	STEP	1.00
	5	600	3	STEP	0.00
	6	2200	2	STEP	0.00
	7	2500	2	STEP	1.00
Cd	1	150	20	RAMP	0.10
	2	250	10	RAMP	0.10
	3	500	10	RAMP	1.00
	4	500	10	STEP	1.00
	5	500	3	STEP	0.00
	6	2200	2	STEP	0.00
	7	2400	2	STEP	1.00
Pb	1	150	20	RAMP	0.10
	2	250	10	RAMP	0.10
	3	800	10	RAMP	1.00
	4	800	10	STEP	1.00
	5	800	3	STEP	0.00
	6	2400	2	STEP	0.00
	7	2500	2	STEP	1.00

2.2.2. Atomic Absorption Spectrometry:

The atomic absorption spectrometry uses absorption of light of intrinsic wavelengths by atoms. All atoms are classified into those having low energies and those having high energies. The state having low energies is called the ground state and the state having high energies is called the excited state. The atom in the ground state absorbs external energies and is put in the excited state. For example, sodium is mainly in two excited states, having higher energies by 2.2eV and 3.6eV respectively than in the ground state, as shown in Fig. 2.1 When 2.2eV energy is given to the sodium atom in the ground state, it moves up to the excited state (I) and when 3.6eV energy is given, it moves up to the excited state (II). Energy is given as light, and 2.2eV and 3.6eV respectively correspond to energy

of light at 589.9nm and 330.3nm wavelength. In the case of sodium in the ground state, only light of these wavelengths are absorbed and no other wavelength light is absorbed at all [7]

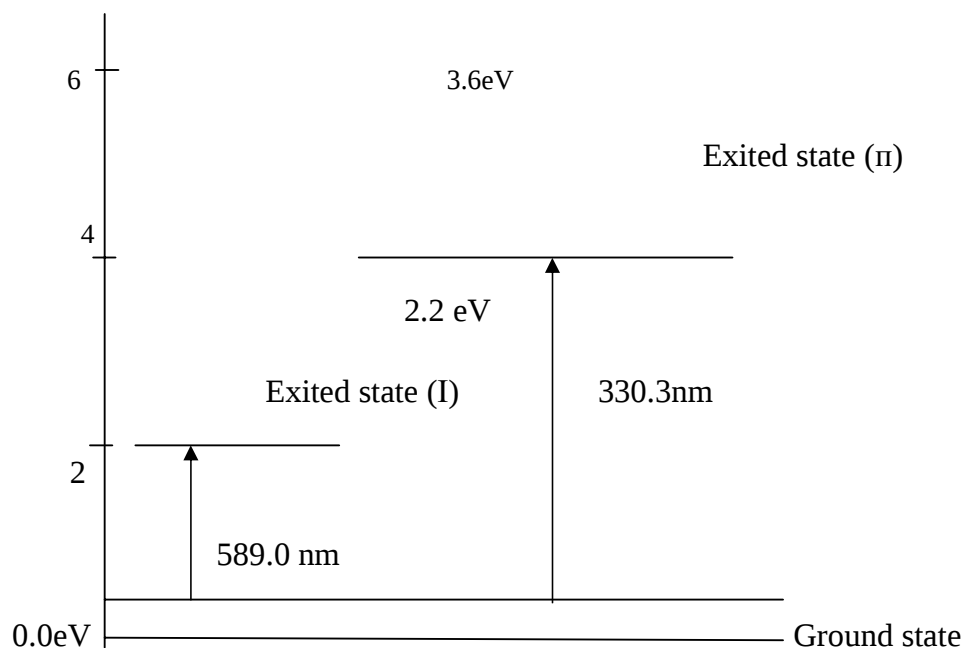


Fig. 2.3 Sodium energy states

The difference between energies in the ground state, and in the excited state is fixed by the element and wavelength of light to be absorbed. Atomic absorption spectrometry uses the hollow cathode lamp (HCL). The HCL gives off light characteristic to the elemental wavelength being measured. Thus, the light absorbed measures the atomic density [7].

Graphite furnace AAS has been used for the off-line measurement of elements in the elution fractions of LC, although insufficient detection limits proved to be a serious drawback in the case of many clinical applications, where the concentrations of the elemental species in the biological fluids and tissues are very low. When species can be converted to hydrides, such as is routinely done for mercury, selenium, arsenic, and antimony, then hydride generation AAS is a very interesting and cheap detection technique. An on-line method was developed for the speciation

of arsenic in human serum, including MMA, DMA, arsenobetaine and arsenocholine. It has been applied for the speciation of arsenic in persons with abnormally high arsenic concentrations in serum, such as dialysis patients. The method is based on cation exchange LC separation, UV photo-oxidation for sample digestion, and continuous hydride generation AAS for the measurement of arsenic in the LC eluent. By developing the technique of argon segmented flow in the post-column eluent, a substantial improvement in chromatographic resolution for the separation of these four arsenic species was obtained. The LC separation, photo oxidation, hydride generation, and AAS measurement could be completed on-line within 10 min. The response is different for the different species. The detection limits (as arsenic) were 1.0, 1.3, 1.4, and 1.5 $\mu\text{g/l}$ for MMA, DMA, arsenobetaine, and arsenocholine, respectively, in serum. The concentration of the four species was determined in serum samples of six patients with chronic renal insufficiency. Only arsenobetaine and DMA were significantly detected by this method. The main part of arsenic in human serum is arsenobetaine. No MMA, arsenocholine, or inorganic arsenic were detected in these six samples. AAS with a quartz tube atomizer is a very sensitive, specific, rugged, and comparatively inexpensive detector for GC. GC coupled with AAS has been described as a sensitive instrumentation for mercury speciation (Emteborg et al., 1996). On-line solid-phase extraction coupled to graphite furnace AAS has also been explored. Cold vapors AAS is the most widely used technique for measuring mercury. Direct coupling of solid-phase micro extraction and quartz tube AAS has been used for selective and sensitive determination of methyl mercury in seafood [8].

2.2.3. Principle of Atomic Absorption Spectroscopy:

Atomic Absorption deals with the excitement of atoms. An atom absorbs light at discrete wavelengths. In order to measure this narrow light

absorption with maximum sensitivity it is necessary to use a line source which emits the specific wavelengths that can be absorbed by the atom. The two most common line sources are the “hollow cathode lamp (HCL)” and the “electrode less discharge lamp (EDL)”. In HCL, when an electrical potential is applied between the anode and cathode, some of the fill gas atoms become positively charge and accelerated through the electrical field to collide with the negatively charge cathode and dislodge individual metal atoms in a process called “sputtering” (Figure 2.1). Sputtered metal atoms are then excited to an emission state through a kinetic energy transfer by impact with fill gas ions [9]. The excited metal atom emits radiation at a wavelength characteristic for the sputtered metal. An EDL consists of a quartz bulb containing the analyte element, or a salt of the analyte element, in an inert gas atmosphere. When a radio frequency (RF) field is applied to the bulb, the inert gas will be ionized and the coupled energy will vaporize and excite the element specific atoms inside the bulb, causing them to emit their characteristic spectrum. This emission spectra are typically much more intense than those from HCLs.

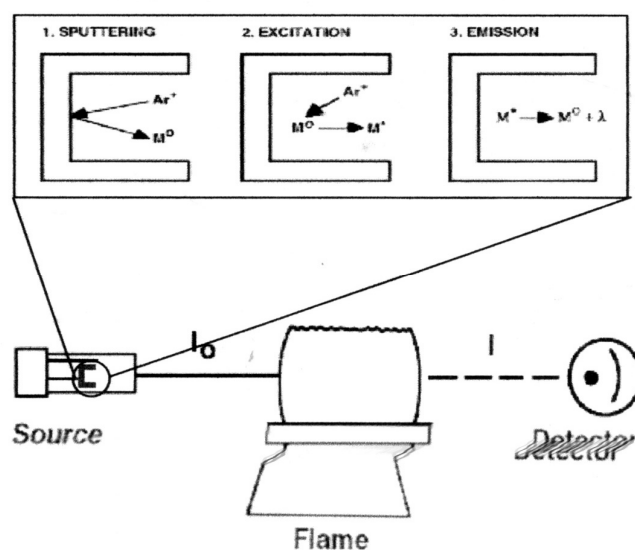


Figure 2.4 Atomic absorption process.

In order to get the atomic absorption process to occur, individual atoms from the sample (Which starts out as a solution of ions) must be produced. First, by the process of nebulization, the sample is aspirate into the burner chamber by the Ventura action caused by the flow of oxidant gas around the capillary tip, where it mixes as a fine aerosol with the fuel and oxidant gases. At this point, the metals are still in solution in the fine aerosol droplets. As these tiny droplets pass into the heat of the flame, the process of evaporation desolation removes the solvent and leaves tiny solid particles of sample material. As more heat is applied, liquefaction will take place, and additional heat will vaporize the sample (Figure 2.4). At this point the metal of interest, called analyte, is still bound up with some anion to form a molecule which does not exhibit the atomic absorption phenomenon. By applying still more heat energy, this molecule is dissociated into the individual atoms which make it up. Since the thermal energy from the flame is responsible for producing the absorbing species, flame temperature is an important parameter governing the flame process. Several energy levels are possible, but usually only the first excited level (E_1) is of concern, since a very few electrons are excited beyond E_1 . The number of ground state metal atoms formed in step 5 of the flame process (Figure 2.5) will determine the amount of light absorbed.

	$M^+ + A^-$	(Solution)
1)Nebulization	$M^+ + A^-$	(Aerosol)
2) Desolation	MA	(Solid)
3) Liquefaction	MA	(Liquid)
4) Vaporization	MA	(Gas)
5) Atomization	$M^0 + A^0$	(Gas)
6) Excitation	M^*	(Gas)

7) Ionization $M^+ + e^-$ (Gas)

Figure 2.5 The flame process. “ M^+ ” is metal cation and “ A^- ” is the associated anion.

The radiation beam intensity is attenuated by an amount that is proportional to the concentration of the element under consideration in the atomizer. Hence, Beer’s law is employed for quantitative determination of the analyte:

$$A = \epsilon bc$$

Where, A is Absorbance, ϵ molar extinction coefficient and b the thickness of the absorbing medium (i. e., the length of the light path intercepted by the absorption species in absorption cell) and c is the concentration of absorbing species.

When a molecule absorbs ultraviolet or visible light, its electrons get normally promoted to the excited state. In most of the cases, several electronic transitions like $\sigma \longrightarrow \sigma^*$, $n \longrightarrow \sigma^*$, $\pi \longrightarrow \pi^*$, $n \longrightarrow \pi^*$, occur resulting in the formation of several bands [10, 11].

For determination of the concentration of the analyte, wavelength of maximum absorptivity or minimum transmittance ($\lambda_{\max}$) of the analyte is evaluated by scanning the solution containing the analyte within the wavelength ranges. The wavelength corresponding to the maximum absorbance in the curve of absorbance versus wavelength gives the value. Then a series absorbencies are recorded by a UV-VIS spectrophotometer for a series of prepared standard solutions of the compound concerned at the $\lambda_{\max}$. A calibration curve is obtained by plotting absorbencies versus concentrations. The calibration curve should be straight line passing through the origin if Beer’s law ($A = \epsilon bc$, where A is the absorbencies, c is concentration of the analyte, b path length and ϵ molar extinction coefficient) is obeyed [12]. Measuring the absorbance of the solution

containing the analyte at the $\lambda_{\max}$, the corresponding concentration is determined from the calibration curve.

Absorbance of analyte was determined from UV-VIS spectrophotometer and slope from calibration curve (absorbance vs. conc. in $\mu\text{g/L}$). Then total arsenic concentration was estimated as follows:

$$\text{Conc. of As (mg/L)} = \frac{\frac{\text{Absorbance}}{\text{Slope}} \times \mu\text{g As}}{\text{Total vol. of sample in generator flask (=70mL)}} \times \text{Dilution factor}$$

2.2.4 UV-VIS Spectroscopy:

a. System Description: The UV-VIS spectrophotometer used in our colorimetric analysis was DR/4000 U (HACH Company, Colorado, USA). The DR/4000 U is a microprocessor controlled, single-beam spectrophotometer. The followings are the essential components of the system: i) Light sources, ii) Monochromator, iii) Detector and iv) Display system.

Two types of continuous light sources were used in DR/4000 U spectrophotometer: tungsten/halogen lamp and deuterium lamp [13]. The tungsten/halogen lamp is useful for the wavelength region between 240 and 2500 nm and deuterium lamp between 160 and 380 nm. In our investigation, the tungsten/halogen lamp was used.

The optical system is composed of a split-beam diffraction grating Seya-Namoika monochromator. The monochromator employs entrance and exit slits, rotating concave holographic grating, order sorting filter, fused silica beam splitter and convex fused silica lens (Figure 2.6). It provides a full 190 to 1100 nm wavelength range with a nominal bandwidth of 4 nm, wavelength accuracy ± 1 nm and resolution ± 0.1 nm [13].

The detector system comprises a photomultiplier tube (PMT) and an electrical signal processor. The DR/4000 U is a spectrophotometer with backlit, graphic LCD. With a measurement range of 190 to 1100

nm, it provides results in operator-selectable readout modes of Absorbance (ABS), Percent Transmittance (%T) or Concentration. Its dynamic range is -3.0 to 3.0 A with photometric linearity of ± 0.002 A [13].

A read-only memory (ROM) is provided with preprogrammed calibration data for over 130 test procedures to provide direct readout in concentration mode. DR/4000 U features a Standard Addition Option to improve the accuracy and precision of analyses and reports the percent of standard recovery. The instrument also provides for storage of up to 200 user-generated calibrations. Data can be transferred from DR/4000 U to computer using HachLink™ software as Excel spreadsheet. It also accommodates various sample cells of different sizes, but stray light is <1%.

b. Optical System: The optical system of DR/4000 U single-beam spectrophotometer is composed of a split-beam diffraction grating Seya-Namoika monochromator, tungsten/halogen and deuterium light sources and a PMT detector (Figure 2.6).

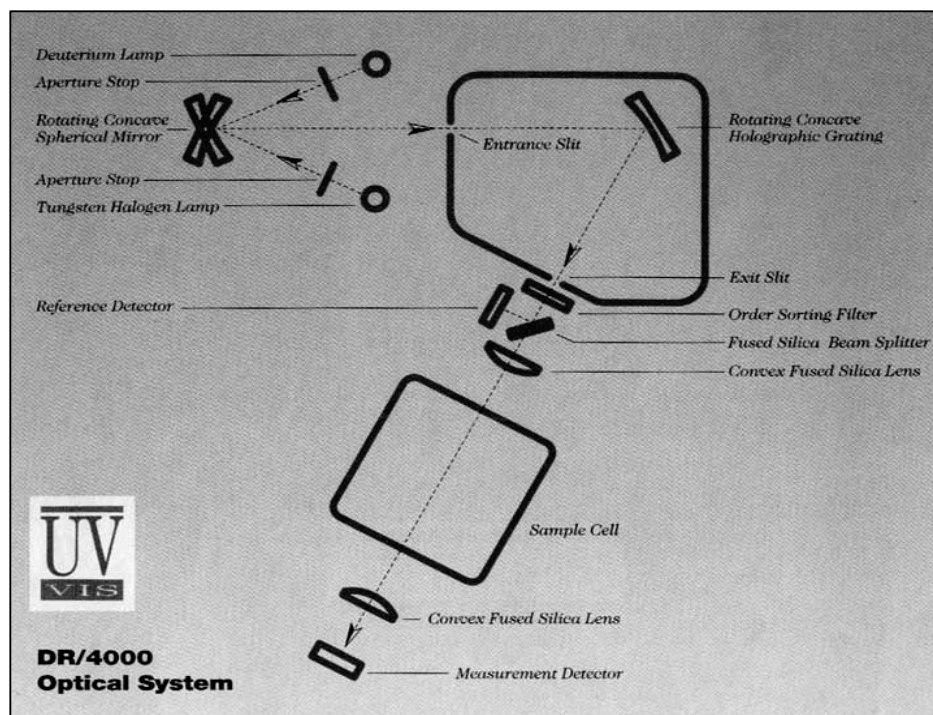


Figure 2.6. Optical system of DR/4000 U UV-VIS spectrophotometer [13].

The primary source of light (tungsten/halogen or deuterium lamp) is dispersed into component wavelengths with the help of a rotating

concave dispersion grating. The monochromatic light of desired wavelength, obtained through exit slit adjustment, is passed through the reference solvent and the sample solution and is focused on the detector. When the sample absorbs light, its intensity is lowered. Thus the detector receives an intense beam from reference cell and a weak beam from sample cell. This results in the generation of pulsating or alternating currents which flow from photomultiplier tube to signal processor. The signal for the absorption of the contents of the reference cell is automatically subtracted from those of the sample cell by signal processor, giving a net signal corresponding to the absorption for the analyte. The microprocessor controlled DR/4000 U featuring a reference detector automatically correct for the “dark current” of the photocell, i.e., the small current which passes even when the cell is not exposed to radiation.

2.2.5. Theory of Arsenic, Lead and Cadmium determination in supplied samples following spectrophotometry:

The basis of the spectrophotometric analysis is the color of a solution varies with the concentration of components. The color of a solution is the indication of complex formation between the reagent added and the components. The spectrophotometry deals with the monochromatic light source. The chief advantage of spectrophotometric method is that it provides a simple means for determining minute quantities of substances. The technique is based on the theory given by Lambert and Beer. For valid application of Beer- Lambert law, the following conditions should be fulfilled.

- a) The sample solution should not be highly concentrated.
- b) The used light should be monochromatic.
- c) The incident radiation should not be reflect or scattered before absorption.
- d) Molecules or ions in solution should not remain associated, dissociated, hydrated or it's forming complex.

- e) The solute should not remain in equilibrium with other solutes in solution.
- f) The pH value of the solution should not be changed in any way.
- g) The environmental parameters e.g. temperature & pressure should not be fluctuated largely.

In order to apply the Beer-Lambert's law, the solution must be very dilute, because- Firstly, the absorptivity of a solution depends on the refractive index. In case of highly concentrated solution, association among the solutes may occur. Thus refractive index varies with concentration but it is approximately constant in case of dilute solution.

Secondly, in case of highly concentrated solution, the concentration of the solution slightly changes due to the association through hydrogen bonding in solutes, ion pair formation, solvation or any other chemical process. But in dilute solution all these process are negligible. So, in order to determine the concentration of solution, the concentration of experimental solution must be a definite limit. But the different solution has different limits. So before determining the concentration of the unknown solution, its limit must be determined.

If the law is valid, a plot between absorbance and concentration is a straight line passing through origin. But when the solution is highly concentrated, the curve deviates from the straight line. These deviations may be positive or negative but in most cases the deviation becomes negative which is shown by the dotted lines in figure 2.7.

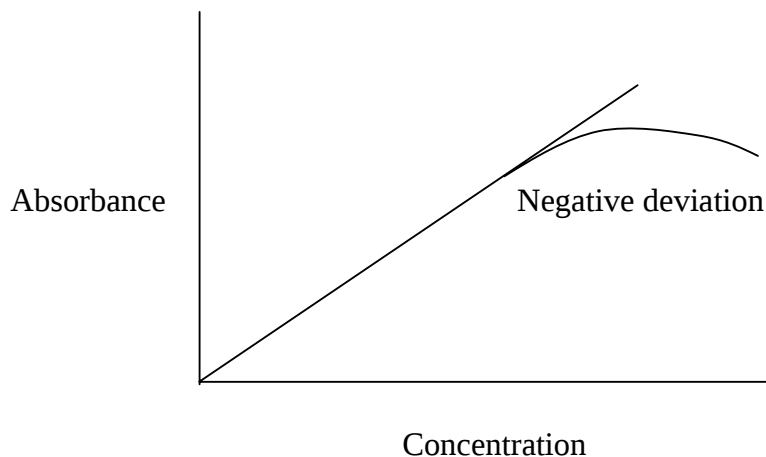


Fig. 2.7 Calibration line of absorbance versus concentration of solution.

Solid environmental samples such as soils, biological samples, etc contain a large amount of organic arsenic, both in +3 and +5 oxidation state. In liquid environmental samples like geothermal water, inorganic arsenic predominates. The environmental samples, when subjected to pretreatment with a mixed acid such as nitric-sulfuric-perchloric, gives mainly inorganic arsenic (v) along with a small quantity of inorganic arsenic (III) [14].

Total arsenic concentrations (As_T) of the decomposed sample aliquots can be determined either by GF-AAS technique. In GF-AAS [2, 4, 14, 15], the sample is first treated chemically so that the analyte is present in ionic form in acidic solution. For hydride generation arsenic must be present in +3 oxidation state. To attain the condition, potassium iodide (KI) solution, which is dispensed into the acidic sample solution, converts inorganic arsenic (III) to its hydride, arsine (AsH_3). The volatile metal hydride (AsH_3) is driven from solution by a stream of carrier gas Ar and transported to the heated QTA. The hydride decomposes in the QTA, the analyte is atomized and its atomic (As) absorption is measured,

2.3. EXPERIMENTAL PROCEDURES :

2.3.1. Materials

All analytical reagents and chemicals used in this experiment were of high purity analytical grade. Deionized double distilled water was used throughout the experimental work. Laboratory glassware was kept overnight in 10% (v/v) nitric acid. Before use, the glassware were rinsed with deionized water and dried in a dust free environment. All the apparatus like conical flask, burette, pipette and screw capped polyethylene bottle used in this work were washed with detergent solution and then rinsed with tap water and finally with distilled water.

Several standard solutions, ternary acid mixture and other solutions were prepared according to the following procedure [1, 2, 3, 4, 5].

2.3.2 Standard Stock Solutions Preparation:

2.3.2.1 Stock standard:

The standard samples used for atomic absorption metals or salts dissolved in acid. When it is stored for a long period it is precipitated, or absorbed by the container wall due to hydroxide and carbonate produced, and its concentration changes. The standard solutions available on the market are supplied in accordance with the standard solution examination system. It is based on the national standard, and is acid or alkaline. The guarantee period of one to two years is shown and it must be used within this period. The stock solution prepared by the standard solution method is a highly concentrated solution that is acidic or alkaline with a metal concentration of 1mg/ml. However, one year or longer use is not recommended. In storing any standard solution, avoid direct sunlight and store it in a cool place.

2.3.2.2. Other solutions:

i) HC1 solution: 2M HC1 solution was made by taking 82.5 mL of 37% HC1 in a 500 mL measuring flask and filling up to the mark with DDW. 5M HC1 solution was also prepared similarly. 1.5% (v/v) HC1 solution was also prepared by adding 15 mL of conc. HC1 to DDW and made up to 1L.

ii) NaOH solution: 1% (w/v) NaOH solution was prepared by dissolving 10g of NaOH pellets in DDW and made up to 1000 mL.

Tap water supplemented with appropriate amounts of the standard arsenic (III) solution was used as model water and natural arsenic contaminated groundwater of tube-wells as natural water for irrigation purposes.

The commercial gases (99.99% pure nitrogen and acetylene) used were collected from BOC, Bangladesh and argon gas from Singapore. Some reagents of HACH, were also used. Standard Reference Materials: Two external standard reference materials (SRM) were used to check the accuracy of the methods. Tomato Leaves (SRM1573a) were obtained from the U.S. National Institute of standards and Technology (Gaithersburg, MF) and Pond Sediment (NIES 2) from the National Institute for Environmental Studies (Japan). Internal reference material of water was prepared very cautiously from ACS- certified Arsenic Reference standard solution of HACH, USA (Cat.No. 14571-42) through successive dilution with distilled deionized water (DDW).

2.4. Methodology:

A. Soil Preparation and Irrigation:

A land was divided into 6×4 beds, each of area $1.5 \text{ ft} \times 1.5 \text{ ft}$. 2 additional beds were prepared for control and 1 for blank purposes. With fresh soil, NPK fertilizers and insecticide were mixed well (except blank). Among 6 natural organic fertilizers (Cow-dung, Water hyacinth, Compost, Drain mud, Rotten Dhuncha and Earthworm fertilizer), each was added as 10, 50, 100 and 200 g / kg of soil. After 120 hours, seeds of Okra, Bindweed, Basil, Korola and Amaranth were sown.

Control-1		Blank		Control-2	
CD-10	WH-10	CM-10	DM-10	RD-10	EW-10
CD-50	WH-50	CM-50	DM-50	RD-50	EW-50
CD-100	WH-100	CM-100	DM-100	RD-100	EW-100
CD-200	WH-200	CM-200	DM-200	RD-200	EW-200

Fig 2.8 Schematic diagram of prepared bed.

After seeding, tube-well water amended with As, Pb and Cd solutions (each having conc. of 5 mg L^{-1}) was used for irrigation purpose. A total of 8

times of water were applied within the duration of 70-80 days as 3.0 L / bed / irrigation. The average interval of irrigation was 7 days.



Cow-dung



Compost



Earthworm



Drain mud



Water hyacinth



Rotten Dhuncha

Fig-2.9. Natural organic fertilizers.



Indian Spinach



Red Amaranth



Bitter Gourd



Water Spinach



Okra

Fig- 2.10. Cultivated vegetables.

B. Water Sampling and Analysis:

1. Sample collection and preservation: Before to analysis, the tube wells were flushed with 2-3 tube-well volumes of groundwater, (e.g. 70L for tube well having depth of 20 m and i. d. of 4 cm) [16, 17]. the crystal clear water samples were collected in 500 mL acid washed polyethylene bottles, acidified with 10.0 mL of conc. HNO_3 to keep the pH below 2 and stored in refrigerator at 4°C [5].

2. Measurement of Physical Parameters: p^{H} and dissolved oxygen (DO) of aquifer water were measured on-site with a microprocessor-based $\text{p}^{\text{H}}/\text{mV}/^\circ\text{C}$ meter p^{H} 211 (HANNA, Italy) and a DO-meter sensionTM 6 (HACH, USA). Before analysis, the pH-meter was with calibrated with buffer solutions (at pH 7.01 and 10.01) and DO-meter with electrolyte filling solution. Redox potential (E_{h}) was also measured on-site with the same $\text{pH}/\text{mV}/^\circ\text{C}$ meter. Electrical conductivity (EC) was measured on-site with a digital conductivity meter EC 214 (HANNA, Italy) after calibrating with conductivity solution (HI 7030). All the instruments have manual temperature compensation mode.

3. Pretreatment of Water Samples: Before to analysis of water samples for total iron and manganese, the water samples were subjected to mild digestion with HNO_3 (method 3030 E) [5]. 100 mL of well-mixed, acid- preserved water sample was taken in an Erlenmeyer flask (125 mL). 5mL of 68% HNO_3 was added to it and the flask was placed on a hot plate to evaporate (without boiling) to 15-20 mL. Additional conc. HNO_3 was added and heating was continued until digestion was complete as shown by a light-colored, clear solution. It was transferred to a 100 mL volumetric flask with two rinsing. The pH of the solution was adjusted to 4.0 with 1.0 M

NaOH solutions followed by dilution with distilled, deionized water up to the mark.

For analyzing total arsenic (As_T) Lead and Cadmium and Chromium, the water samples were subjected to vigorous digestion with nitric acid- sulfuric acid method (method 3030 G) [5].

C. Plant Tissues Sampling and Analysis:

1. Sample Preparation: Fresh plant tissue (vegetables) samples were rinsed several times with distilled water, cut into pieces and dried in oven at $80^{\circ}C$ for 48 hours. Drying of the plant materials was performed as quickly as possible to avoid the growth of molds and microorganism or contained respiration and to minimize loss of weight by enzymatic action in the tissues [2, 3]. The dried plant materials were subjected to grinding with ceramic mortar and pestle followed by sieving with 0.5 mm screen.

2. Digestion of Biological Samplings: Oxidation of organic matter of plant tissue and release of the elements such as arsenic was carried out through Wet Oxidation by means of oxidizing acids such as the HNO_3 - H_2SO_4 - $HClO_4$ ternary mixture [2, 3, 4, 18,]. All biotic samples (dried and powdered) were accurately weighed ($1.0 \pm 0.005g$) and taken in a 500 mL Erlenmeyer flask. After adding 10 mL of 69% HNO_3 , the flask was swirled to moisten the entire mass of the tissue and was placed on a steam plate for 30 minutes. It was then placed on the electric plate at $180^{\circ}C$. The suspension was boiled nearly to dryness. This pre-digestion with HNO_3 required about 45 minutes.

The digestion flask and contents were cooled slightly. Then 10 mL of the ternary acid mixture (prepared by mixing 69% HNO_3 , 98% H_2SO_4 and 60% $HClO_4$ in the volume ratio of 10:1:4) was added. The digestion was carried out on the electric hot plate at $220^{\circ}C$ until dense white fumes of H_2SO_4 and $HClO_4$ were vigorously evolved. The digestion was stopped

when the residues in the flask were clear and white and only slightly moist with H_2SO_4 . The HClO_4 , at that point, had been largely removed. This digestion with ternary acid mixture require about 2 hours.

3. Preparation Stock Solution: The digestion flask containing the residue from the wet oxidation of plant tissue was cooled and 10 mL of 82% HCl was added. The flask was swirled and polished and then the solution was poured into a 100 mL measuring flask. Two additional rinsing of the digestion flask were given with small portion of 6N HCl . The digestion flask was washed twice with distilled deionized water and dilute to make a final volume of 100 mL.

4. Analysis of Samples for total arsenic, Lead and Cadmium: After filtration and subsequent dilution, the aliquot was subjected to hydride generation reactions to determine the concentration of total As, Pb and Cd by both GF-AAS and UV-VIS Spectroscopy. The procedure was the same as that for measuring total As, Pb and Cd in water. Blanks were made under similar experimental and reagent conditions. Other parameters were also measured using standard procedures.

D. Soil Sampling and Analysis:

1. Sample preparation: The composition of soil near the roots of the plants may differ from other areas due to absorption by plants. Hence the soils were collected from 3-6 cm beneath the surface layer and most distant from the root and were taken in labeled polyethylene bags. The soils were then dried in an electric oven at 105°C for about 24 hours. The dried mass was cooled and ground with ceramic mortar and pestle to make finer particles followed by sieving with 0.5 mm screen.

2. Digestion of soil samples: Digestion of soil samples were carried out by *modified method of Small and Mc Cants* with the oxidizing acids mixture such as H_2SO_4 - HClO_4 acid mixture [4, 19]. Accurately 0.5

± 0.005 g of the soil sample was heated in a 250-mL Erlenmeyer flask until fuming with 2 mL of 98% H_2SO_4 . When the organic matter had been destroyed and the soil assumed a gray color, the flask was cooled and 3 mL of 70% HClO_4 were added with a few boiling chips. The mixture was boiled on an electric hot plate at 220°C for about 2 hours. Heating was discontinued when the soil was near to dryness.

3. Preparation of stock solution of samples:

The digestion flask and the contents cooled and 10 mL of 82% HCl was added. The flask was swirled and polished and then the solution was poured into a 100mL measuring flask. Two additional rinsing of the digestion flask were given with small portions of 6N HCl . The digestion flask was also washed twice with distilled, deionized water and diluted to make a final volume of 100 mL.

4. Analysis of samples for total Arsenic, Lead and Cadmium.

After filtration and subsequent dilution, the aliquot was subjected to hydride generation reactions to determine the concentration of total As, Pb and Cd by GF-AAS and UV-VIS Spectroscopy. The procedure was the same as that for measuring total As, Pb and Cd in water. Blanks were made under similar experimental and reagent conditions. Other parameters were also measured using standard procedures.

E. Fish Sampling, Digestion and Analysis:

1. Sample Preparation: The fresh fish purchased by the household owner was immediately kept in a refrigerator at -20°C without any processing. When frozen it was preserved in an ice-box for transport to the laboratory. The skin of the sample was then removed, washed with distilled deionized water and cut as small “fillets”, comprised of muscle tissue [20, 21]. It was then dried at 80°C in an oven for 48 hours. All the processes were performed as soon as possible to avoid the growth molds and

microorganism and to minimize loss of weight by enzymatic action in the tissues [2, 3]. The dried fish materials were grinded with ceramic mortar and pestle followed by sieving with a screen.

2. Digestion, Preparation of Stock Solution and Analysis: The dried and ground fish samples were digested with ternary acid mixture following the same procedure as described details in case of plants tissues. Preparation of stock solution and analysis for total arsenic, cadmium and lead of the digested fish samples were also the same. This is referred to section 2.4 C.

F. Dust Sampling, Digestion and Analysis: In our investigation, the dust samples were collected from inside of the studied households. The samples were dried in an oven at 105°C, sieved with a 0.5 mm screen and digested with H₂SO₄-HClO₄ acid mixture [3]. Preparation of stock solution and analysis for total arsenic, cadmium and lead of the digested dust samples were also the same as those of soil. This is discussed in section 2.4 D in detail.

G. Estimation of Arsenic, Lead and Cadmium Concentrations

in Air: The concentration of inhaled arsenic in indoor air was estimated from existing indoor dust using the following equation [22, 23]:

$$[\text{As}]_{\text{air}} = D \times M_{\text{PM}} \times F_{\text{PM}} \times F_{\text{count}}$$

Where, D is the concentration of dust in the air (assumed 70.0 $\mu\text{g}/\text{m}^3$ which is the annual mean dust concentration for many countries like the United States). M_{PM} is the arsenic concentration on airborne particulate matter (assumed equal to $[\text{As}]_{\text{dust}}$ where dust is derived from the soil), F_{PM} is the proportion of particulate matter that is respirable [assumed $\text{PM}_{10} \sim 73\%$ as 27% of airborne particles are generally found to be $> 10 \mu\text{m}$ in equivalent diameter] and F_{cont} is the fraction of dust that is derived from

the contaminated source. For time spent outdoors, F_{cont} was assumed to be equal to $[M_{soil}]$; when indoors, $F_{cont} = 0.445[M_{soil}]$ because 44.5% of indoor dust is considered to be derived from outdoor soil. Therefore,

$$\begin{aligned}[As]_{air} &= (70.0 \mu\text{g}/\text{m}^3) \times [As]_{dust} \times 0.27 \times 0.445 \\ &= 0.070 \times [As]_{dust} \times 0.27 \times 0.445 \quad \mu\text{g}/\text{m}^3\end{aligned}$$

Similar calculations were made for estimating concentration of inhaled lead and cadmium in indoor air.

2.5 Quality Control

To ensure quality of the measurements, analysis of reagent blanks, and analysis of duplicates, recovery of known additions and analysis of standard reference materials (SRM) were made.

1) Cleaning of glassware: All glassware were sunk into dilute HNO_3 solution about 24 h and then washed through double distilled water. After cleaning, put them in a electric oven under 100°C till complete dry. Beside the clean glassware, other equipment were handled with clean, accuracy and sincerity.

2) Analysis of Reagent Blanks: Replicate analyses of reagents blanks were common for each set of experiment. These were performed for estimation of As, Cd and Pb content in rice, vegetables, fish, water and dust.

3) Crosschecking of concentration: For assurance of getting result we have taken some pre-ready sample solution to Borendro laboratory and DPHE laboratory, Rajshahi. Borendro lab used kids' method and DPHE used HG-AAS system to analyze sample. DPHE is a renowned government institution which is performing large research and survey type work. Borendro lab has performed specially arsenic contamination based research. Results have matched to previous result without little deviation. After justifying, all calculations were performed and were finalized data.

4) Duplicate and Triplicate Analysis: At least 5% of total samples were analyzed in duplicates and when the agreement is not good enough those were measured in triplicates to assess precision.

Each duplicates result was analyzed for Relative Percent Difference (%RPD) by the following equation:

$$\% \text{ RPD} = [(DW_1 - DW_2) / [(DW_1 + DW_2)/2] \times 100$$

The acceptable %RPD for duplicates of digestion method is not specified by United States Environmental Protection Agency SW846 or by other standard methods. The Contract Laboratory Program (CLP) inorganic statement of work (Method 200 series CLP-M) lists a %RPD control limit of 20% [24, 25]. If any result of a specific sample is not comparable to the other two results, the deviated result was discarded while calculating the average.

5. Recovery of Known Additions: This method is also known as “Method of Standard Additions” which allows to verify the presence or absence of matrix interference. By taking known amounts of the samples (table 2.10), nothing was added to the first sample, a measured amount of standard that approximates analyte concentration was added to the second and twice the first volume was added to the third. The samples were digested following the same procedure and were diluted to the same volume so that the final concentrations of the original sample constituents are the same in case. Only the amount of added analyte differs by a known amount. Four replicate experiments were performed separately. Recovery for the environmental samples (edible plant tissues, soils and groundwater) were then analyzed.

6. Analysis of Standard Reference Materials: Two external standard reference materials (SRM) were used to check the accuracy of the accuracy of the methods. Tomato Leaves (SRM 1573a) were obtained from the U. S. National Institute of Standards and Technology (Gaithersburg, MD) and Pond Sediment (NIES 2) from the National

Institute for Environmental Studies (Japan). Internal reference material of water was prepared very cautiously from ACS-certified Arsenic Reference Standard Solutions of HACH, USA (Cat.No.14571-42) through successive dilution with distilled deionized water (DDW). Recoveries in the samples were in the range of 91-140% for As_T , Pb_T and Cd_T .

2.6. Statistical Analysis:

Statistical analyses have conducted on our getting data. It is a very good process to justify of data accuracy. Over all accuracy of data were performed through statistical analysis. To do this, have used two reliable software. The name of the softwares are excel and SPSS (version 11.5) [26]. Using this software have been calculated range of concentration, arithmetic mean, standard deviation, geometric mean, median and variance.

2.7 Reference:

- [1] A.I. Vogel's Textbook of quantitative inorganic analysis: theory and practice. 2nd Edition, RICHARD CLAY AND COMPANY LTD., Bungay, Suffolk, Great Britain, 1957.
- [2] Loon J. C. V., Selected methods of trace metal analysis: biological and environmental samples. In: Chapter 3 and 5, John Wiley & Sons, New York, 1985, pp. 53-112.
- [3] Jackson M. L., Soil chemical analysis. In: Plant Tissue Analysis- Mineral Constituents, Chapter 12, Prentice – Hall, Inc., Englewood Cliffs, N.J., 1958, pp. 326-338.
- [4] Perkin-Elmer, Analytical methods for atomic absorption spectroscopy. Perkin-Elmer, Inc., Shelton, Connecticut, U.S.A., 1996.
- [5] APHA, AWWA and WEF, Standard methods for the examination of water and wastewater. American Public Health Association, American Water Works Association and Water Environment Federation. Eaton A. D., Clesceri L. S. and Greenberg A. E. (Eds.), 19th edition, United Book Press, Inc., Baltimore, Maryland, 1995.
- [6] Graphite-Furnace Atomic Absorption Spectroscopy, Shimadzu AA-6800, Japan. 2013.
- [7] Atomic absorption spectrophotometry cookbook, Section 1. Basic Conditions of Analysis of Atomic Absorption Spectrophotometry, P-1-4.
- [8] Elemental Speciation in Human Health Risk Assessment, pp- 42
- [9] Beatty R. D. and Kerber J. D., Concepts, instrumentation and techniques in atomic absorption spectrophotometry. 2nd edition, The Perkin-Elmer Corporation, Norwalk, CT, U.S.A., 1993.

- [10] Banwell C. N., Fundamentals of molecular spectroscopy. 2nd edition, NcGraw-Hill Book Company (UK) Limited, Maidenhead, Berkshire, England, 1972.
- [11] Sharma Y. R., Elementary organic spectroscopy: principles and chemical applications. In: Ultra-violet and visible spectroscopy, Chapter 2. 2nd edition (reprint), S. Chand & Company Ltd., New Delhi, India, 1994.
- [12] Skoog D. A., D. M. West and F. J. Holler, Fundamentals of analytical chemistry. In: Chapter 21 and 24. 6th edition, Saunders College Publishing, Orlando, Florida, pp. 535-559 and 614-630.
- [13] HACH, Instrument and procedures manual. HACH Company, Loveland, Colorado, U.S.A., 2000.
- [14] H. Sakamoto, Y. Susa, H. Ishiyama, T. Tomiyasu and K. Anazawa, Determination of trace amounts of total arsenic in environmental samples by hydride generation flow injection-AAS using a mixed acid as a pretreatment agent. Analytical Sciences, 17 (2001) 1067-1071.
- [15] Perkin-Elmer, MII 15 mercury hydride system user's guide. Perkin-Elmer, Inc., Shelton, Connecticut, U. S. A., 2000.
- [16] Berg M., Tran H.C., Nguyen T.C., Pham H.C. Schertenleib R. and Giger W., Arsenic contamination of groundwater and drinking water in Vietnam: A human health threat. Environmental Science & Technology, 35(13) (2001) 2621-2626.
- [17] BAMWSP, Guidelines of arsenic Screening in Groundwater. Bangladesh Arsenic Mitigation Water Supply Project, Ministry of Local Government and Rural Development. Govt. of Bangladesh, Dhaka, 1999.
- [18] Milton A. and Johnson M., Arsenic in the food chains of a revegetated metalliferous mine trailing pond. Chemosphere, 39(5) (1990) 121-143.

- [19] Hesse P. R., A textbook of soil chemical analysis. In: Total (elemental) analysis and some trace elements, Chapter16, CBS Publishers & Distributor, Delhi, India, 1994, pp. 371-435.
- [20] U.S. EPA. 2000. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories; Volume I: Fish Sampling and Analysis. Third edition. Office of Water. U.S. Environmental Protection Agency. Washington, D.C.
- [21] Cal EPA. 2005. General Protocol for Sport Fish Sampling and Analysis. Office of Environmental Health Hazard Assessment (OEHHA), California Environmental Protection Agency, California.
- [22] U.S. EPA, Guidelines for exposure assessment. Federal Registrar 57(104):22888-22938, Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC, 1992.
- [23] Thistle Publishing, Inc., Risk Assistant, version 1.1 for Windows. Thistle Publishing, Inc., Alexandria, VA, USA, 1995.
- [24] Murnock, J.M., Vertical and seasonal distributions and relationships of arsenic in Presque Isle sediments. M. S. Thesis, Gannon University, Erie, PA, 2002.
- [25] Northeast Analytical Inc., Guide to environmental analytical methods. 4th Edition, Genium Publishing corporation, Chenectady, NY, 1998.
- [26] SPSS, SPSS for Windows. Release 11.5.0 (standard version), SPSS Inc., U.S.A., 2002

CHAPTER THREE

Use of Natural Organic Fertilizers to Reduce of As, Pb and Cd Poisoning in Vegetable Plants.

Introduction:

Vegetables act as a very good accumulators of As, Pb, Cd etc. The extent of arsenic, cadmium and lead deposition differ greatly from vegetables to vegetables as well as organs to organs. Zhang et al [1] conducted a pot experiment to investigate the growth and arsenic (As) uptake of pakchoi cabbage and tomatoes grown in a grey Fluvo-aquic soil. They observed that arsenic (As) accumulation in pakchoi cabbage were significantly greater than those of shoots of tomato. They also found the biomass, As content and As accumulation in both vegetables showed significant difference with species. Arsenic accumulation is apparent in pakchoi cabbage. The accumulation of arsenic, cadmium and lead by plants is influenced by a number of factors such as the As, Cd and Pb concentration in soil, As, Cd & Pb availability and redox potential of As, Cd and Pb. According to A.J.M. Baker plants can be divided into three groups depending on the response to the concentration of an element in soil. These are accumulators, indicators and excluders. The accumulators concentrate the element in the aboveground parts of the plants. The indicators reflects the external concentrations of the elements and the excluders prevents element uptake until the soil concentration is too high [2].

De Temmerman et al. [3] showed that the accumulation of As in terrestrial and emergent plants generally occurs via the roots and shoots in areas with atmospheric depositions of arsenic. Azizur Rahman et al.[4] studied and showed that aquatic macrophytes accumulate As from the water column as well as from the sediment. For uptake via roots, plants are able to modify the rhizosphere by the exudation of organic acids to render elements available. Marschner [5] showed that the organic acid has a major effect on the mobilization of elements bound to ion exchange sites in the rhizosphere. Moreno –Jimenez et al. [6] found that the action of organic acids originating from plant roots may also increase As

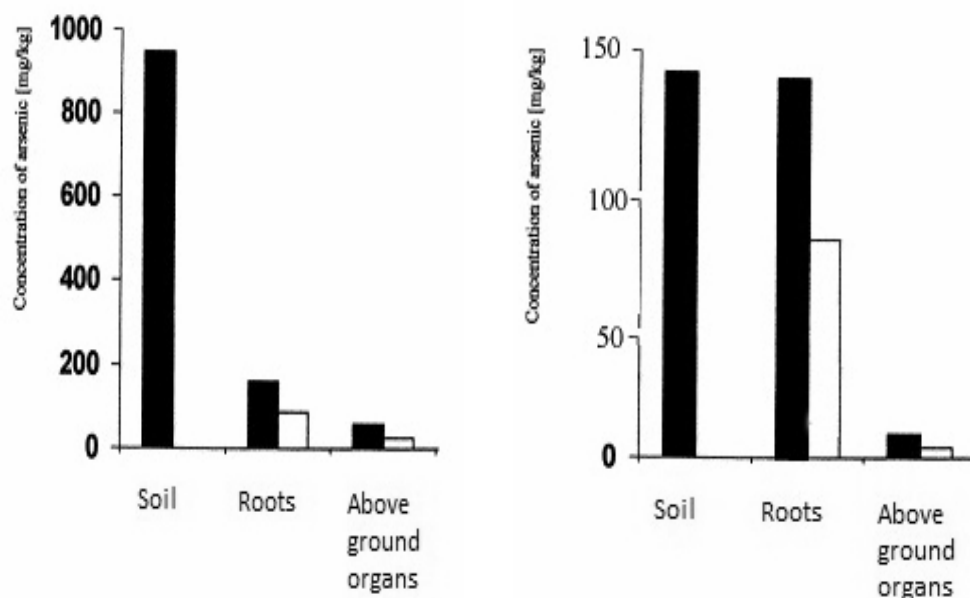
availability in soil leading to an increased plant As uptake. Silva Gonzaga et al [7] showed a correlation between the As concentration in the plant and the As concentration in the soil for the action of organic acids on As availability in soil. Ghosh et al. [8] showed that the bacteria in the rhizosphere may also help to increase the As availability, probably by the soluble arsenates from insoluble FeAsO_4 and AlAsO_4 in the soil, resulting in a higher plant As uptake.

Di Lonardo et al.[9] and Afrous et al.[10] showed that the terrestrial and emergent plants such as *Populus alba* and *Typha latifolia* primarily and most commonly accumulate As in the roots and low concentration in the shoot. Ma et al. [11] and Smith et al. [12] showed that the distribution of arsenic may be higher to the shoots compared with the roots in some plant species like hyper accumulating fern *Pteris Vittata*. (*Raphanus sativus*) and in some non-hyper accumulating plants like radish. Bergqvist and Greger [13] found that the As concentration in the shoots of submerged plants may be higher than the As concentration in the root due to the As accumulation by the shoots from the surrounding water column. Zhao et al. [14] found that arsenic accumulation occurs through silicon aquaglyceroporins and Azizur Rahman et al. [15] showed that it also facilitates the transport of MMA and DMA. Moreno – Jimenez et al. [16] showed that the only known paths for arsenate accumulation in plants are through phosphate transporters.

Anastasia and Kender [17] found that most of the arsenic was concentrated in the root tissues of blueberry, from where the accumulated arsenic was transferred to stem and leaves. Lue et al [18] found the descending order of arsenic concentration in plants as root to stem, stem to leaves and to edible plant tissues.

Pitten et al [19] studied uptake of arsenic by terrestrial plants and concluded that the above-ground organs contained less arsenic than the roots. Their findings were represented in the following Histograms

showing the concentration of arsenic in soil adhering to the roots, the roots and the above-ground organs of *Holcus lanatus*.



(a) For highly contaminated sites. (b) For average contaminated sites.

Figure 3.1. Concentration of arsenic in soil adhering to the roots, the roots and above-ground organs of *H. Lanatus*[19]. Black bars indicate samples that were analyzed immediately after the harvest, clear bars identify samples that were washed prior to the arsenic analysis to clean the plant of adhering soil and dust particles.

Some authors observed that high levels of arsenic in soil were not reflected in plants, while the other authors observed the reverse cases. For instances, Pitten et al [19]. found that in spite of the considerable high arsenic contamination of the soil (mean value 923 mg kg^{-1}), the terrestrial plant arsenic contamination remained comparatively low (mean value 4.1 mg kg^{-1} , dry wt.) They established the phenomenon that plants took up the organoarsenic compounds triphenylarsine and dimethylphenylarsine, not diphenylchloroarsine diphenylcyanoarsine or phenyldichloroarsine from soil. Milton and Johnson [20] found high levels of arsenic in metalliferous mine (UK) tailings ($630 \pm 34 \text{ mg kg}^{-1}$) that were not

reflected in live vegetation ($0.5 - 1.1 \text{ mg kg}^{-1}$) due to physic-chemical nature of the tailings which mitigate against plant uptake. Williams and Whetstone [21] found 14 ppm of arsenic in alpha alpha grass against soil arsenic content of 60 ppm. The transfer of arsenic from soil to vegetation would be evident from soil/plant ratios of arsenic concentration. It is practically notable that there is a large variation in the soil/vegetation ratios for arsenic elsewhere. It illustrates the important interactions between arsenic speciation and environmental impact, and the difficulty of assessing environmental risk from substrate concentrations of total arsenic alone.

Huq and Naidu [22] found that the relationship between soil arsenic and the corresponding arsenic in plant was negative, while the relationship between water arsenic and plant arsenic was positively correlated with $R^2=0.55$ with a multiple R-value of 0.7416.

Liu and Gao [23] established that the soil arsenic and sweet potato arsenic were positively related. Ali et al. [24] studied on rice plants in Bangladesh and observed no strong correlation ($R^2=0.21$) between the presence of arsenic in soil and rice plant root. The same authors [24] had shown the correlations between arsenic in grains and arsenic in other parts of rice plants (BR-29 variety) collected from Srinagar and Sonargaon of Bangladesh.

CAL Limei et al.[25] studied the spatial distribution, sources, accumulation characteristics and potential risk of heavy metals in the agricultural soils and vegetables. The results show that there are higher accumulation of heavy metals in agricultural soils such as Cu, Zn, Ni, Pb, Cd and Hg. They found the ratios of vegetables samples in which Ni, Pb and As concentrations higher than the Maximum Levels of Contaminants in Foods and the bio-concentration factors (BCF) of heavy metals in vegetables is in the order of $\text{Cd} > \text{Zn} > \text{Cu} > \text{As} > \text{Ni} > \text{Hg} > \text{Cr} > \text{Pb}$. They focused on potential risk of heavy metals for food safety and human's health from agricultural soils and vegetables in Dongguan city, Guandong Province, China.

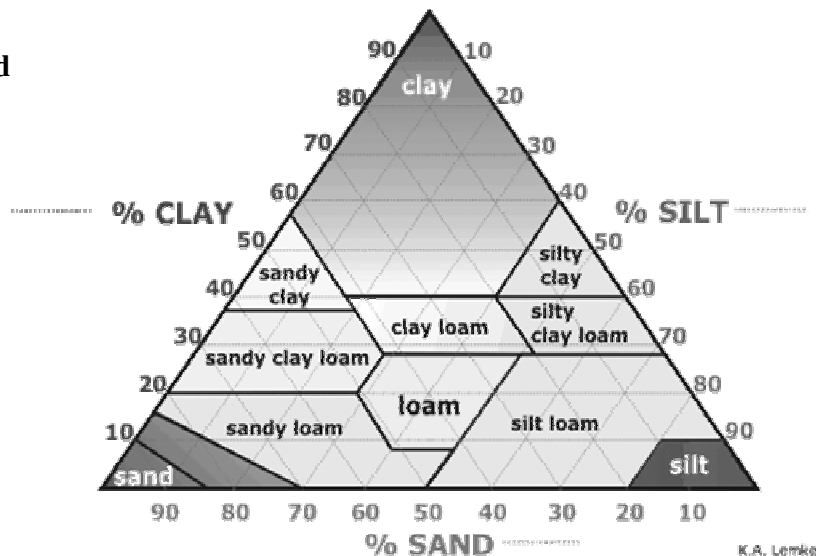
Soil is a natural body consisting of layers that are primarily composed of four constituents viz- minerals, organic matter, water and air. Soil is the end product of the influence of the climate, relief, biotic activities and parent materials acting over a period of time [26]. The uptake of arsenic, cadmium and lead by plants are dependent upon soil properties and plant physiological factors. Soils have physical and chemical characteristics. The physical characteristics of soil are texture, structure, bulk density, porosity, permeability, shear strength, soil profile and color. The chemical characteristics of soil are acidity, nutrient content and cation exchange capacity. These properties will determine the success or failure of any crop or pasture grown in the soil. The soil texture is simply determined by the relative proportions of sand, silt and clay in the soil. Soil texture is a function of sand, silt and clay particle sizes is shown in Fig 3.2.

***Black: high organic content**

***Yellow/red/orange: iron**

***Light gray-white: calcium or silica**

Percent clay, silt and sand



- clay <0.002 mm
- silt 0.002-0.05 mm
- sand 0.05-2.0 mm

Fig 3.2.Texture triangle of soil.

Of all the factors influencing the evolution of soil, water is the most powerful factor due to its involvement in the solution, erosion, transportation, and deposition of the materials of soil. The amount of

water available for plant uptake is determined by a number of factors including soil water potentials (matrix and osmotic), soil depth and soil layering [27]. The mixture of water and dissolved and suspended materials is called the soil solution. Water is central to the solution, precipitation and leaching of minerals from the soil profile. Finally, water affects the type of vegetation that grows in a soil, which in turn affects the development of the soil profile.

The most influential factor in stabilizing soil fertility are the soil colloidal particles, clay and humus, which behave as repositories of nutrients and moisture and so act to buffer the variations of soil solution ions and moisture. Their contributions to soil nutrition are out of proportion to their part of the soil. Colloids act to store nutrients that might otherwise be leached from the soil or to release those ions in response to changes of soil pH [28].

Cation exchange capacity (CEC) of a soil is the measure of the capacity of the soil to hold positively charged ions (cations). With a decrease in CEC the elements' availability in the soil is known to increase due to the presence of fewer binding sites. In the heavy metal-contaminated soil the CEC change marginally.

The pH indicates the acidity or alkalinity (basic) of the soil. Different plants have differing optimum soil pH requirements. The majority of plants prefer a pH of around 6 to 7, which is very slightly acid. The soil pH is important in determining the availability of soil minerals. At extremes pH some minerals are "locked up" and cannot be used by the plants, whilst other minerals may become toxic. Aluminium, iron, zinc, copper, manganese and boron become more soluble under acidic conditions and can reach toxic levels.

The greatest influence on plant nutrition is soil pH, which is a measure of the hydrogen ion (acid-forming) soil reactivity, and is in turn a function of the soil materials, precipitation level, and plant root behavior. Soil pH

strongly affects the availability of nutrients. Plants require a number of essential nutrients for growth and development. Both the soil and the atmosphere can provide these nutrients. Some of these minerals are needed in large amounts (major elements or macronutrients) and others are needed in smaller amounts (trace elements or micronutrients).

The organic material of the soil has a powerful effect on its development, fertility, and available moisture. Following water and soil colloids, organic material is next in importance to soil's formation and fertility. The importance of soil organic matter in supplying nutrients, contributing to cation exchange capacity and improving soil structure is well recognized [29]. With a decrease in soil organic matter there is an increase in the availability of heavy metals to plants due to fewer binding sites being present in the soil.

3.2. EXPERIMENTAL PROCEDURES:

3.2.1 Materials

The materials employed for the investigation is described in section 2.1. The only additional material is LaurylSulfate broth, a cultural media for Fecal Coli form bacteria counting. Sterilized distilled deionized water (DDW) was used for the process involved in counting Fecal Coli form.

LaurylSulfate broth: A quantity of 38.10 g of the powdered cultural media (OXOID, UK) (comprised 20.0, 5.0, 2.75, 2.75, 5.0 and 0.1 g of Tryptose, Lactose, K_2HPO_4 , KH_2PO_4 , NaCl and Sodium lauryl sulfate, respectively) was taken in a beaker and 500 mL of sterilized DDW was added to it and heated gently (not evaporated) until completely dissolved. The deep red colored broth was then sterilized in an autoclave by maintaining a temperature and pressure of $121^{\circ}C$ and 0.106 MPa respectively for about 10 minutes [18]. After attaining at room temp., it

was stored in refrigerator. Caution was exercised for having no bubble in the broth.

3.2.2. Methods:

The methods of fertilizers and soil preparation, and irrigation [20] were the same as mentioned in chapter 2. Water, vegetables and soil sampling and analysis procedures [21-24] are discussed elaborately in the previous chapter, and hence not mentioned here. Only the method of microbiological examination is described here.

Microbiological examination

Membrane Filter (MF) method was used to count total and Fecal Coli form bacteria. A volume of 100 ml water was filtered through gridded nontoxic Nitrocellulose membrane filter (Millipore, USA), 47 mm in diameter having pore size of 0.45 μm , under manual vacuum condition. It was placed in a glass Petri dish, with support of absorbing pad, containing 4 ml of the specific cultural media and incubated in a B-28 incubator (Binder, Germany) for 24 h at 35°C for total coli form and 44.5°C for Fecal Coli form. Finally the produced colonies were counted with aid of a microscope.

3.3 RESULTS AND DISCUSSION:

3.3.1 Characterization of Cultivated Soil

The soils that were cultivated for the study were characterized by measuring physical, chemical and biological parameters. The physical parameters include pH, redox potential (E_h), moisture content, organic matter (OM), sand and clay/silt content. The chemical parameters include total concentrations of arsenic, lead, cadmium, nitrogen and sulfur. Besides these, water soluble arsenic, exchangeable phosphorus and reactive calcium and iron were measured. The only microbiological parameter was counting of Fecal Coli form bacteria. These are represented in Table 3.3.1. All the parameters were measured using standard procedures and properly calibrated instruments.

Table 3.3.1. Selected physical, chemical and microbiological parameters of the tested soil.

Parameter	Unit	Cultivated soil		
		Blank [†]	Control	Fertilizer amended [‡]
<i>Physical:</i>				
pH	unit less	6.92	6.41	6.15
Redox Potential (E _h)	mV	35	44	64
Moisture Content	%	8.82	11.97	13.85
Organic Matter (OM)	%	0.67	0.79	1.16
Sand	%	20.99	21.81	21.13
Clay	%	16.98	18.90	18.88
Silt	%	62.03	59.29	59.99
<i>Chemical:</i>				
Exchangeable P	mg L ⁻¹	155	106	111.4
Total N	mg L ⁻¹	0.03	0.07	0.06
Total S	mg L ⁻¹	20.4	29.0	31.7
Reactive Ca	mg L ⁻¹	0.413	0.101	0.149
Reactive Fe	mg L ⁻¹	0.400	0.115	0.121
Total As	mg L ⁻¹	1.565	4.027	2.518
Water soluble As	mg L ⁻¹	0.7931	0.6941	1.117
Total Pb	mg L ⁻¹	0.046	0.192	0.174
Total Cd	mg L ⁻¹	0.011	0.038	0.042
<i>Microbiological:</i>				
<i>Fecal Coli form</i>	CFU/100 g	1.2 × 10 ⁴	5.7 × 10 ⁵	2.4 × 10 ⁸

[‡] averaged values are considered.

[†] in blank soil no water was applied.

Table 3.3.1 reflects that the studied soil is slightly acidic in nature and the acidity is increased slightly after being amended with the fertilizer. It is to be noted that the majority of vegetable plants prefers a soil pH of around 6 to 7 (characteristic soil of moist climate) [30], and our studied soil was within the range.

The soil contains very low fraction of organic matter (OM), although it is improved after mixing the fertilizer. Consequently, moisture content is increased from control to fertilizer amended soil. Analysis of sand, clay and silt shows that the soil is of silt loam type. Calculation of hydraulic

properties [31] reflects that the soil has the saturation of $0.474 \text{ m}^3 \text{ water} / \text{m}^3 \text{ soil}$ and the available water is $0.175 \text{ m}^3 \text{ water} / \text{m}^3 \text{ soil}$. Microbiological examination shows that amongst the bacteria, only *Fecal Coli form* is increased to about 10^4 fold when fertilizers are added. The organic matter of the soil in Bangladesh is from $< 1.0\%$ to 3.5% and the pH range from 2.0 to 5.5 [32]. In this point of view, the soil of our study field is similar to barind tracts of Rajshahi in Bangladesh.

3.3.2 Characterization of Irrigation Water:

The tube-well water of shallow aquifer of depth 78 ft was used as control irrigation water. For preparation of model water calculated amounts of As_2O_3 , $\text{Pb}(\text{NO}_3)_2$ and CdCl_2 were mixed with the water to achieve the desired concentration of 5 ppm each. The waters were characterized by measuring physical, chemical and biological parameters. The physical parameters include pH, redox potential (E_h) and electrical conductivity (EC). The chemical parameters include total concentrations of As, Pb, Cd and Fe as well as available concentrations of Ca, Mg, Mn, Cl and F. The only microbiological parameter was counting of Fecal Coli form bacteria. These are represented in Table 3.3.2. All the parameters were measured using standard procedures and properly calibrated instruments.

Table 3.3.2. Selected physical, chemical and microbiological parameters of the irrigation water.

Parameter	Unit	Irrigation water	
		Tube-well	Solution amended (Model water)
Physical:			
Temperature	°C	29.4	29.6
pH	unit less	6.83	7.01
Redox Potential (E _h)	mV	30	33
Electrical Conductivity (EC)	µS cm ⁻¹	564	518
Chemical:			
Total As	mg L ⁻¹		
Total Pb	mg L ⁻¹		
Total Cd	mg L ⁻¹		
Total Fe	mg L ⁻¹	0.42	0.30
Ca	mg L ⁻¹	352.7	168.3
Mg	mg L ⁻¹	111.8	72.9
Mn	mg L ⁻¹	0.68	0.32
Cl ⁻	mg L ⁻¹	188.9	395.6
F ⁻	mg L ⁻¹	1.97	0.48
Microbiological:			
Fecal Coli form	CFU/100 g	0	0

From the above table it is clear that the tube-well water is nearly neutral in nature. The higher value of electrical conductivity indicates the presence of more ions in water. The concentrations of calcium and magnesium indicate the tube-well water is hard. The water also contains considerable amounts of chloride and fluoride. It is also free from Fecal Coli form bacteria.

It is interesting to note that the metal ion concentrations except added As, Pb and Cd in model water are less than those in tube-well water. Hence the hardness of the model water is less than the tube-well water. This is

probably due to formation of complexes that reduces the free ions concerned.

3.3.3. Characterization of Natural Organic Fertilizers:

The studied six natural organic fertilizers are rotten cow-dung, rotten dhuncha tree (*Eschynomene Cannabina*), compost, drain mud, dried water hyacinth and earthworm fertilizers. These six fertilizers are frequently used by Bangladeshi farmers traditionally.

These fertilizers were characterized by measuring physical, chemical and biological parameters. The physical parameters include moisture content, pH and redox potential (E_h). The chemical parameters include total concentrations of N, P and S as well as water soluble concentration of As, Pb and Cd. The only microbiological parameter was counting of Fecal Coli form bacteria. These are shown in Table 3.3.2. All the parameters were measured using standard procedures and properly calibrated instruments.

It is reflected from Table 3.3.2 that moisture content is the maximum in water hyacinth (11.45%) and minimum in drain mud (2.45%) with the average of 6.71%. All the fertilizers except water hyacinth and rotten Dhuncha are alkaline in nature and ranges from 7.27 to 7.73. The fertilizers are in reduced condition. The N, P and S contents in the fertilizers are characteristics of Bangladeshi soil. They also contain a significant fraction of As, Pb and Cd which is water soluble. The fertilizers also contain a huge amount of bacteria, e.g., only Fecal Coli form bacteria of order of 10^7 - 10^8 per 100 g of fertilizer.

Table 3.3.3. Selected physical, chemical and microbiological parameters of the studied natural organic fertilizers.

Fertilizer	Moisture Content (%)	pH	E _h (mV)	Organic Matter (%)	Organic C (%)	Total N (%)	Total P (as P ₂ O ₅) (%)	Total S (as SO ₄) (%)	Water Soluble As (mg kg ⁻¹)	Water Soluble Cd (mg kg ⁻¹)	Water Soluble Pb (mg kg ⁻¹)	Fecal Coliform (CFU/100g)
Cow-dung	8.15	7.72	- 89	50.72	30.32	1.13	0.62	0.47	0.187	0.133	3.783	1.3×10^7
Drain Mud	2.45	7.41	- 47	35.33	30.09	0.27	0.33	0.07	0.169	0.119	2.834	1.5×10^7
Compost	4.65	7.27	- 63	65.17	15.32	0.29	0.83	0.12	0.204	0.135	4.044	1.6×10^8
Earthworm Mud	3.19	7.62	- 84	30.72	17.74	0.18	0.51	0.19	0.191	0.128	3.362	8.8×10^6
Water Hyacinth	11.45	6.60	23	32.94	19.10	0.52	0.86	0.12	0.174	0.130	3.711	3.7×10^6
Rotten Dhuncha	10.36	6.35	- 8	18.12	10.51	1.68	1.02	1.25	0.143	0.123	3.448	1.4×10^7
Average	6.71	7.16	- 45	38.83	20.51	0.68	0.70	0.37	0.178	0.128	3.530	3.6×10^7

3.3.4. Plant Height and Yield:

It was generally observed that as the amount of applied natural organic fertilizers was increased, the height of the vegetable plants was also increased. The effect of studied six natural organic fertilizers for the height of studied five vegetable plants is represented in Fig. 3.3.

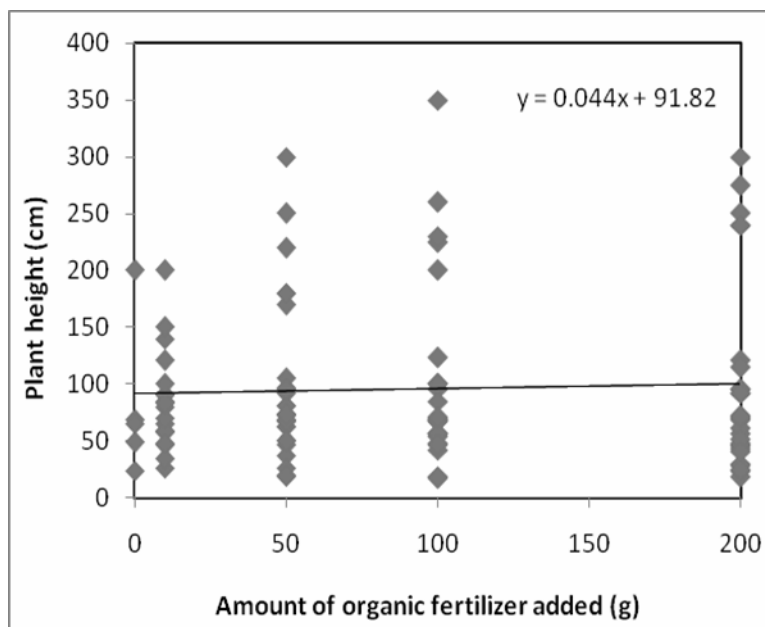


Figure-3.3. Plant height as a function of organic fertilizer added.

The above trend is valid for each of the fertilizers involved. One, namely, the fertilizer cow-dung, is shown below as an example.

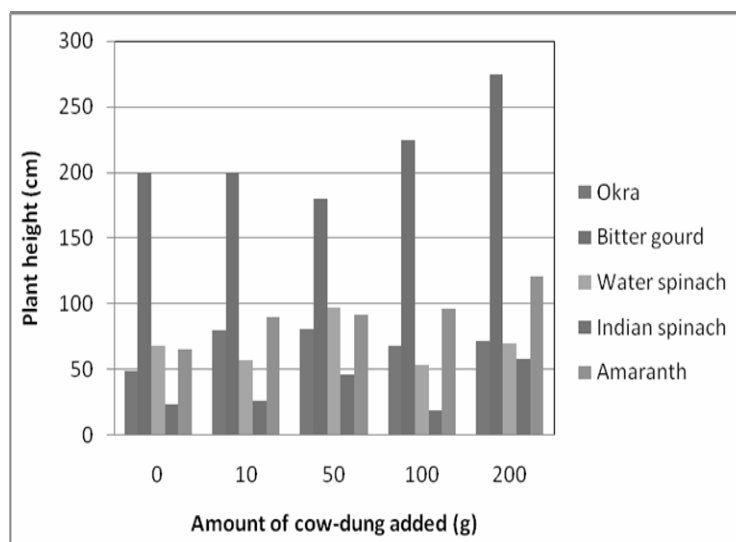


Figure-3.4. Plant height as a function of cow-dung fertilizer added.

It was generally noticed that as the amount of the natural organic fertilizers were increased, the yield, that is, the amount of vegetables obtained was increased. This is depicted in the following figures, where fresh weight of vegetables is considered. The increment roughly follows a linear pattern (Fig 3.5 (a) and (b)).

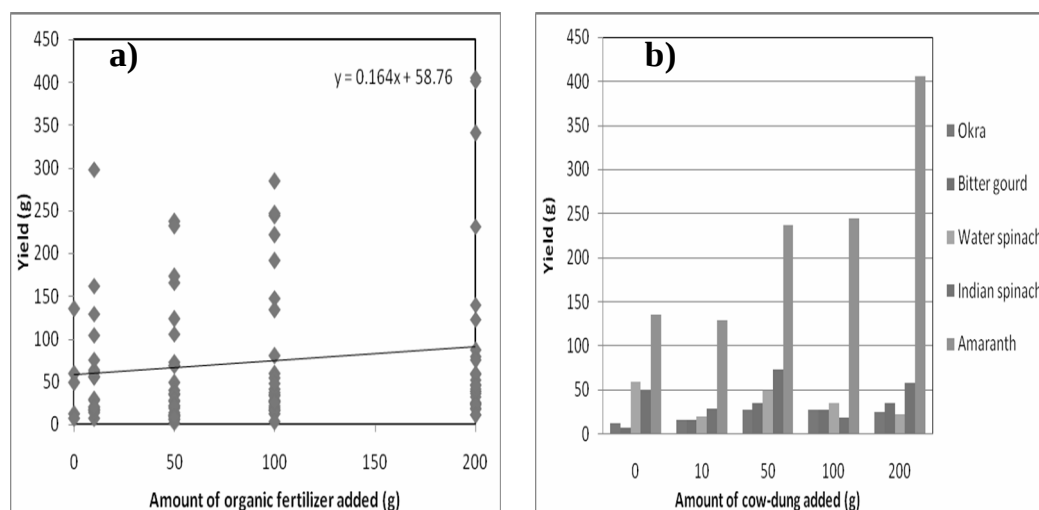


Figure-3.5.Vegetable yields as a function of cow-dung fertilizer added [a) for all fertilizers, b) for cow-dung alone].

From the above discussion it obvious that as the amount of natural organic fertilizers was increased in soil, both plant height and vegetable yield were increased. This means that plant health was improved when more organic fertilizers was added to soil. This is probably due to the following two reasons. Firstly, when natural organic fertilizer was added, the soil becomes enriched in organic matter (OM), which is supported in our data (Table 3.3.31st). The OM, being most part of cellulose, has a tendency to adsorb the toxic ions of As, Pb and Cd. Thus the bioavailability of As, Pb and Cd for the vegetable plants is decreased. Secondly, the natural organic fertilizers being enriched in micro-organism such as bacteria (Table 3.3.33rd), the microbial activities are increased when they are added to soil. As a result of these, reduction, biomethylation, etc. occur that may generate some volatile gases that reduce

As, Pb and Cd content in soil. Such process for arsenic is shown in the Figure 3.6. Both factors contribute to detoxification of As, Pb and Cd poisoning in plants.

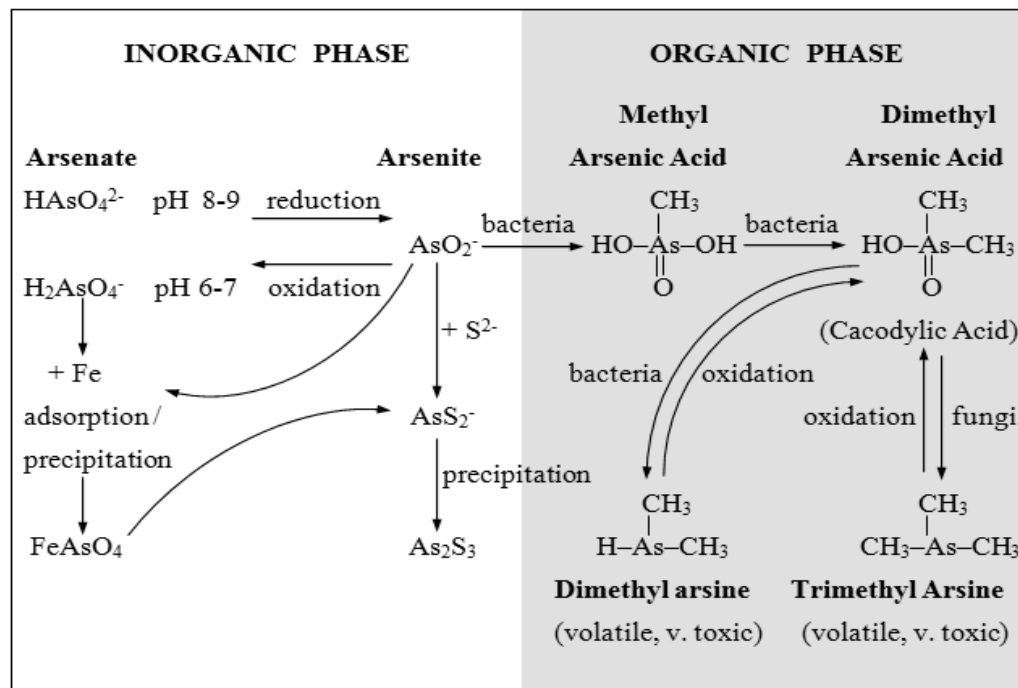


Figure-3.6. Chemical forms of arsenic and their transformations in soil [28].

3.3.5. Probable Mechanism for Reduction of Arsenic, Cadmium and Lead Load in Vegetables Due to Application of The Fertilizers:

Several mechanisms might be employed for reduction of arsenic, cadmium and lead load in vegetables due to application of natural organic fertilizers. Among these two mechanisms are suggested by our investigations. These include – 1) adsorption of heavy metals and metalloids on soil organic matter contents and 2) bio-methylation and other reactions of heavy metals and metalloids induced by soil microorganisms to generate volatile gases. These are also supported with our data (Table-3.3.4).

Table-3.3.4.Organic matter and Fecal Coli form status of the fertilizer amended soils.

Type of bed	Organic Matter (%)	Fecal Coliform (CFU/100g soil)
Control-1	0.76	1.20×10^4
Control-2	1.03	1.12×10^5
CD-10	0.96	2.10×10^6
CD-50	1.34	7.40×10^5
CD-100	1.43	2.50×10^7
CD-200	1.43	3.00×10^6
CM-10	0.83	4.08×10^6
CM-50	1.43	5.30×10^7
CM-100	0.90	1.96×10^8
CM-200	1.43	7.36×10^8
WH-10	0.93	4.00×10^6
WH-50	1.43	5.40×10^7
WH-100	1.58	2.10×10^8
WH-200	1.93	7.80×10^8
DM-10	0.93	3.80×10^6
DM-50	1.03	5.20×10^7
DM-100	1.00	2.00×10^8
DM-200	0.97	7.30×10^8
RD-10	1.00	5.30×10^6
RD-50	1.29	6.80×10^7
RD-100	1.43	2.30×10^8
RD-200	1.29	8.60×10^8
EW-10	1.09	1.40×10^6
EW-50	0.93	4.80×10^7
EW-100	1.03	3.20×10^8
EW-200	1.26	8.80×10^8

The findings are represented in Figure 3.7 [where organic matter (OM) is expressed in % and Fecal Coliform (FC) bacteria in CFU/100 g soil]. Obviously, as the studied natural organic fertilizers are applied, both of

soil organic matter and Fecal Coli form bacteria increases. The increment is less for OM and more for FC.

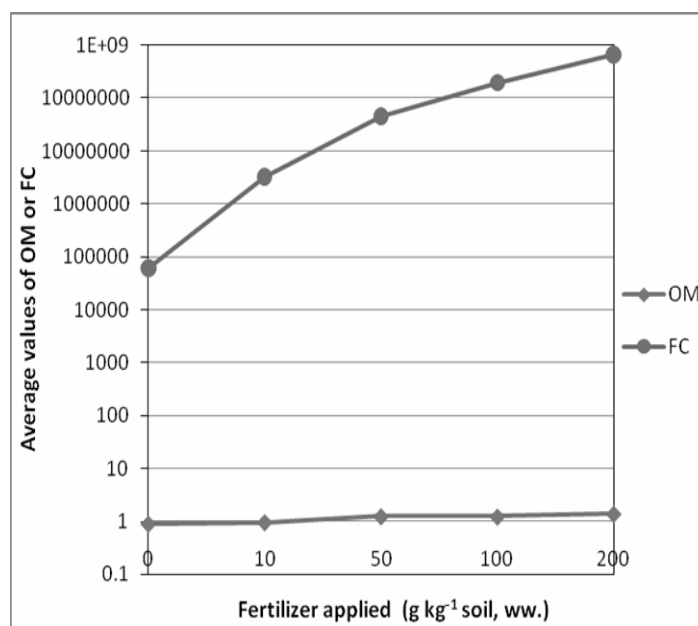


Figure 3.7. Average values of organic matter and Fecal Coliform of the fertilizer amended soils [the zero value stands for control].

Soil organic matter (SOM) plays an important role in the maintenance and improvement of soil properties. It is a dynamic material and is one of the major sources of nutrient elements for plants. SOM is derived to a large extent from plant residues together with the small quantities of animal remains, excreta and microbial tissues. SOM is made up of a large number of substances of widely different chemical composition and the amount of each substance varies with the type, nature and age of plants. All the organic compounds constituting SOM can be summarized in the following Table-3.3.5.

Table-3.3.5.Organic substances of undecomposed soil organic matter [33,34].

Category	Type	Examples
Nitrogenous	Water soluble	Nitrates, ammonical compounds, amides, amino

		acids, etc.
	Water insoluble	Proteins, nucleoproteins, peptides, alkaloids purines, pyridines, chitin, etc.
Non-nitrogenous	Carbohydrates	Sugars, starch, hemicellulose, gums, mucilage, pectins, etc.
	Miscellaneous	Lignin, tannins, organic acids, etc.
	Ether soluble	Fats, oils, wax, etc.

Since pH of our studied soil was slightly acidic (average value of 6.15), the trivalent inorganic arsenic should be found as non-dissociated arsenious acid (H_3AsO_3); while pentavalent arsenic as dihydrogen arsenate ion (H_2AsO_4^-) and monohydrogen arsenate (HAsO_4^{2-}). Since As(III) sorption is practically independent at pH of 6-9, but As(V) is much more strongly bound at low pH [35], it is reasonable that the adsorbing material for arsenic is H_2AsO_4^- and HAsO_4^{2-} .

The oxide surfaces, notably iron, manganese, and aluminum oxides, carbonate surfaces, and insoluble organic matter can generate a significant number of positive charges as the pH decreases. The edges of clay minerals also carry pH dependent charge. These edge sites may be important sites of retention of anions at pH below the point of zero charge (PZC) [36].

Cellulose used as an adsorbent. In some studies carried out previously, chemical modification of cellulosic materials significantly enhanced their ion-binding properties, providing greater flexibility in their application to a wide range of heavy metals adsorption [37, 38, 39]. The entire modified agricultural wastes contain cellulose that possesses some ion-exchange properties due the presence of small number of carboxyl, hydroxyl, sulphur, cyano and amino group in their structure. The chemical modification is expected to improve metal ion binding capacity of

materials. Bead cellulose loaded with iron oxyhydroxide (BCF) [40] and Starch and Carboxymethyl cellulose (CMC) stabilized with synthetic nanoscale zerovalent iron (NZVI) [41] were applied for the adsorption and removal of arsenate and arsenite from aqueous systems. Cellulose beads are considered to be a promising adsorbent due to their special properties like hydrophilic, porous, high surface area. Studies on applying cellulose beads as carriers for arsenic removal are still scarce. Cellulose beads and cellulose derivative beads are widely used as ion exchangers, packing materials for chromatography, adsorbents for heavy metal ions and metalloids, proteins, cosmetic additives and carriers for immobilization of biocatalysts [42,43,44]. Results revealed that starch stabilized particles (S-nZVI) presented an outstanding ability to remove both arsenate and arsenite. In all cases, the adsorption capacity for arsenite and arsenate was pH dependent. The maximum removal efficiency of both arsenic species was obtained at $\text{pH} = 5 \pm 0.1$ and starched nanoparticles was effective in slightly acidic and neutral pH values. In general, the removal rate of arsenate decreased with increasing pH [41].

It has been observed that Pb forms strong complexes with SOM, and that it can compete with most other metals for adsorption sites on SOM [45, 46,47; 48]. Lead has a strong affinity for organic ligands. The Pb sorption capacity of the SOM is estimated to be at least 810 mmol kg^{-1} [49]. Manceau et al. [50] used x-ray absorption fine structure (XAFS) spectroscopy to predict that the speciation of Pb in a contaminated soil was 60% Pb-salicylate complexes and 40% Pb-catechol complexes. These complexes involve the formation of a five-membered chelate complex between the phenol functional groups of the catechol, and a six membered chelate complex formed between the phenol and carboxyl functional groups of the salicylate. Our findings reveal that as SOM is

increased, lead bioavailability is decreased causing a decrease in lead load in vegetables. This is in accordance with the findings of the others.

Organic matter is able to bind heavy metals and metalloids. The soil content of high organic matter could reduce the content of bioavailable metal species as a result of complexation of heavy metal ions. This process could be used for remediation to protect plants against metal pollution.

A brown coal preparation was used as an amendment of soils artificially contaminated with Cd, Pb and Zn. The content of bioavailable zinc, lead and cadmium in soil decreased along with an increase in the amendment dosage of a brown coal preparation [51]. Elevated levels of heavy metals in soils depend on soil pH value, organic matter and clay contents and the fertilization [52, Fytianos et al]. The fluctuation of mentioned parameters can significantly affect their bioavailability [53, 54,]. In recent studies, organic matter has been implicated for alleviating bioavailability of heavy metals in soils. Some researches showed that amendment of contaminated soils with organic matter reduced bioavailability of heavy metals [55].

Cadmium may be absorbed by clay minerals, carbonates or hydrous oxides of iron and manganese or may be precipitated as cadmium carbonate, hydroxide, and phosphate. Evidence suggests that adsorption mechanisms may be the primary source of Cd removal from soils [56,57]. The chemistry of Cd in the soil environment is controlled by pH. Under acidic conditions Cd solubility increases and very little adsorption of Cd by soil colloids, hydrous oxides, and organic matter takes place. At pH values greater than 6, cadmium is absorbed by the soil solid phase or may be precipitated, and the solution concentrations of cadmium are greatly reduced. Cadmium forms soluble complexes with inorganic and organic ligands. The formation of these complexes decreased the bioavailability of cadmium as well as cadmium load in vegetables.

Soil organisms decompose plant and animal residues yielding carbon dioxide and water or synthesized into compounds that comprise humus. It is estimated that 60-80% of the total soil metabolism is due to the microflora. The relative number of soil flora and fauna commonly found in surface soil [34] is presented in Table-6.

Table-3.3.6. Relative number of soil flora and fauna found in surface soil^a.

Organism	Number	
	per square meter	per gram
Microflora		
Bacteria	$10^{13} - 10^{14}$	$10^8 - 10^9$
Actinomycetes	$10^{12} - 10^{13}$	$10^7 - 10^8$
Fungi	$10^{10} - 10^{11}$	$10^5 - 10^6$
Algae	$10^9 - 10^{10}$	$10^4 - 10^5$
Microfauna		
Protozoa	$10^9 - 10^{10}$	$10^4 - 10^5$
Nematoda	$10^6 - 10^7$	$10 - 10^2$
Other fauna	$10^3 - 10^5$	
Earthworms	30 - 300	

^a generally considered 15 cm (6 in.) deep except for earthworms.

Due to presence of huge amount microorganisms in surface soil biotransformation of metal and metalloid take place. Microbial transformations of metals occur through redox conversions of inorganic forms and conversions from inorganic to organic form and vice versa, typically methylation and demethylation. Microbial methylation plays an important role in the biogeochemical cycle of metals, because methylated compounds are often volatile.

Formation of As (III) also may lead to the volatilization of arsine (AsH_3) and methylarsines from soils. Under soil conditions of high organic matter, warm temperatures, adequate moisture, and other conditions

conducive to microbial activity, the reaction sequence is driven towards methylation and volatilization [58]. The loss of organic arsenical compounds from the soil was far greater than for the inorganic source of arsenic. . Due to microbial methylation lead and cadmium also converted into volatile dimethylated lead and cadmium [59]. The content of organic matter and fecal coli form bacteria increase in soil along with an increase in the amendment dosages of studied natural organic fertilizers. As a result of bioavailability of As, Cd and Pb in soil as well as in the vegetable plants decreased. The overall results of the studied six natural organic fertilizers represent in the Fig-3.8–3.10.

3.3.6. Concentration of Arsenic, Lead and Cadmium in Vegetables:

It was generally observed that as the amount of natural organic fertilizers was increased, the concentration of the accumulated arsenic, cadmium and lead was also decreased in most cases. The probable reasons are mentioned in section 3.3.4. However, the concentrations of arsenic, cadmium and lead in the studied vegetables as a function of the fertilizer applied are tabulated in Tables-3.3.7, 3.3.8 and 3.3.9, and the variations are represented in Figures 3.8, 3.9 and 3.10. It is to be noted here that the concentrations in dry weight are represented in parentheses.

Table -3.3.7. Concentration of arsenic in the vegetables (mg kg⁻¹).

Type of bed	Concentration of As in vegetables (mg kg ⁻¹) [§]				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control-1	0.149 (0.821)	0.273 (1.616)	0.209 (2.225)	0.136 (2.794)	0.026 (0.437)
Control-2	0.222 (0.900)	0.297 (1.758)	0.144 (1.642)	0.185 (2.791)	0.070 (0.622)
Control-Average	0.185 (0.860)	0.285 (1.687)	0.176 (2.065)	0.160 (2.792)	0.048 (0.529)
CD-10	0.105 (0.396)	0.114 (1.480)	0.189 (1.965)	0.168 (3.165)	0.039 (0.594)
CD-50	0.139 (0.817)	0.083 (1.284)	0.188 (1.912)	0.159 (3.289)	0.087 (0.878)
CD-100	0.104 (0.581)	0.099 (1.359)	0.200 (1.895)	0.170 (3.450)	0.043 (0.496)
CD-200	0.065 (0.523)	0.101 (1.315)	0.165 (1.670)	0.149 (2.816)	0.030 (0.341)
CM-10	0.173 (0.675)	0.279 (3.284)	0.229 (2.318)	0.136 (2.556)	0.059 (0.680)
CM-50	0.156 (0.600)	0.281 (3.191)	0.196 (1.985)	0.081 (1.672)	0.046 (0.558)
CM-100	0.211 (0.817)	0.108 (1.485)	0.165 (1.893)	0.109 (2.744)	0.055 (0.728)
CM-200	0.226 (0.913)	0.157 (1.495)	0.116 (1.190)	0.074 (1.394)	0.046 (0.581)
WH-10	0.104 (0.533)	0.263 (2.894)	0.142 (1.462)	0.226 (4.048)	0.059 (0.760)
WH-50	0.083 (0.431)	0.198 (1.652)	0.178 (2.139)	0.254 (3.966)	0.083 (0.702)
WH-100	0.107 (0.638)	0.225 (1.880)	0.154 (1.632)	0.250 (3.723)	0.083 (0.594)
WH-200	0.062 (0.370)	0.125 (1.368)	0.175 (1.870)	0.151 (2.280)	0.095 (1.096)
DM-10	0.061 (0.421)	0.222 (2.437)	0.213 (2.149)	0.214 (4.100)	0.059 (0.685)
DM-50	0.105 (0.432)	0.122 (1.472)	0.192 (2.068)	0.264 (4.962)	0.043 (0.488)
DM-100	0.082 (0.405)	0.202 (1.409)	0.221 (1.974)	0.199 (3.547)	0.092 (1.047)
DM-200	0.040 (0.396)	0.171 (1.763)	0.246 (2.489)	0.160 (3.180)	0.023 (0.344)
RD-10	0.174 (0.649)	0.110 (1.411)	0.247 (2.139)	0.126 (2.299)	0.057 (0.683)
RD-50	0.165 (0.523)	0.140 (1.546)	0.225 (2.051)	0.206 (3.750)	0.070 (0.935)
RD-100	0.117 (0.447)	0.138 (1.512)	0.281 (2.909)	0.159 (2.647)	0.055 (0.632)
RD-200	0.115 (0.427)	0.142 (1.503)	0.310 (3.137)	0.148 (2.414)	0.038 (0.431)
EW-10	0.117 (0.510)	0.163 (1.348)	0.098 (1.011)	0.231 (4.342)	0.123 (1.411)
EW-50	0.035 (0.274)	0.121(1.196)	0.132 (1.438)	0.430 (7.597)	0.158 (1.730)
EW-100	0.069 (0.440)	0.151 (1.662)	0.179 (1.931)	0.226 (3.899)	0.172 (2.219)
EW-200	0.091 (0.466)	0.113 (1.108)	0.261 (1.819)	0.122 (2.229)	0.173 (1.513)

[§] the values in parentheses represent dry weight.

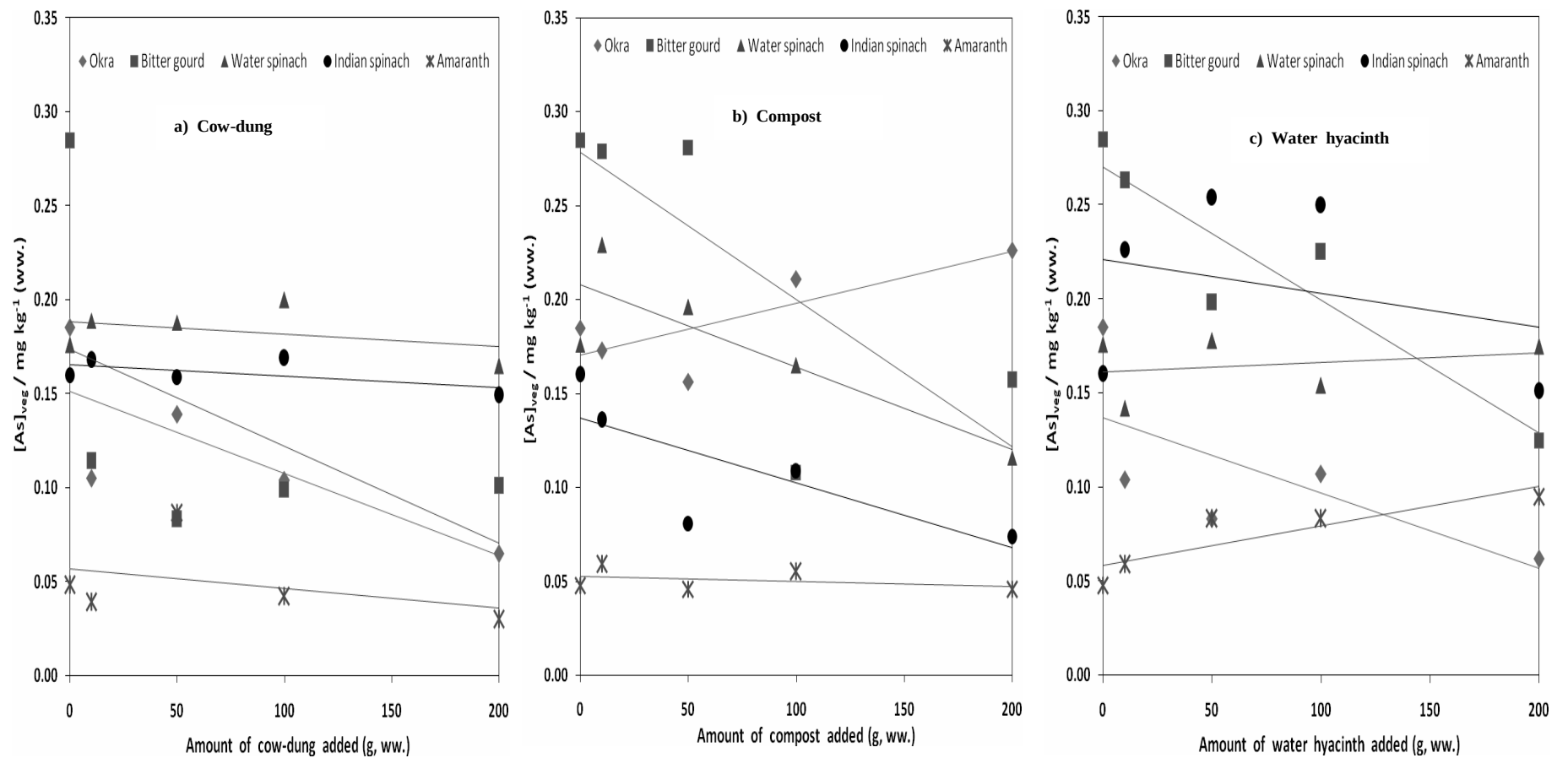


Figure-3.8. Effect of the six fertilizers on accumulation of arsenic in vegetables.

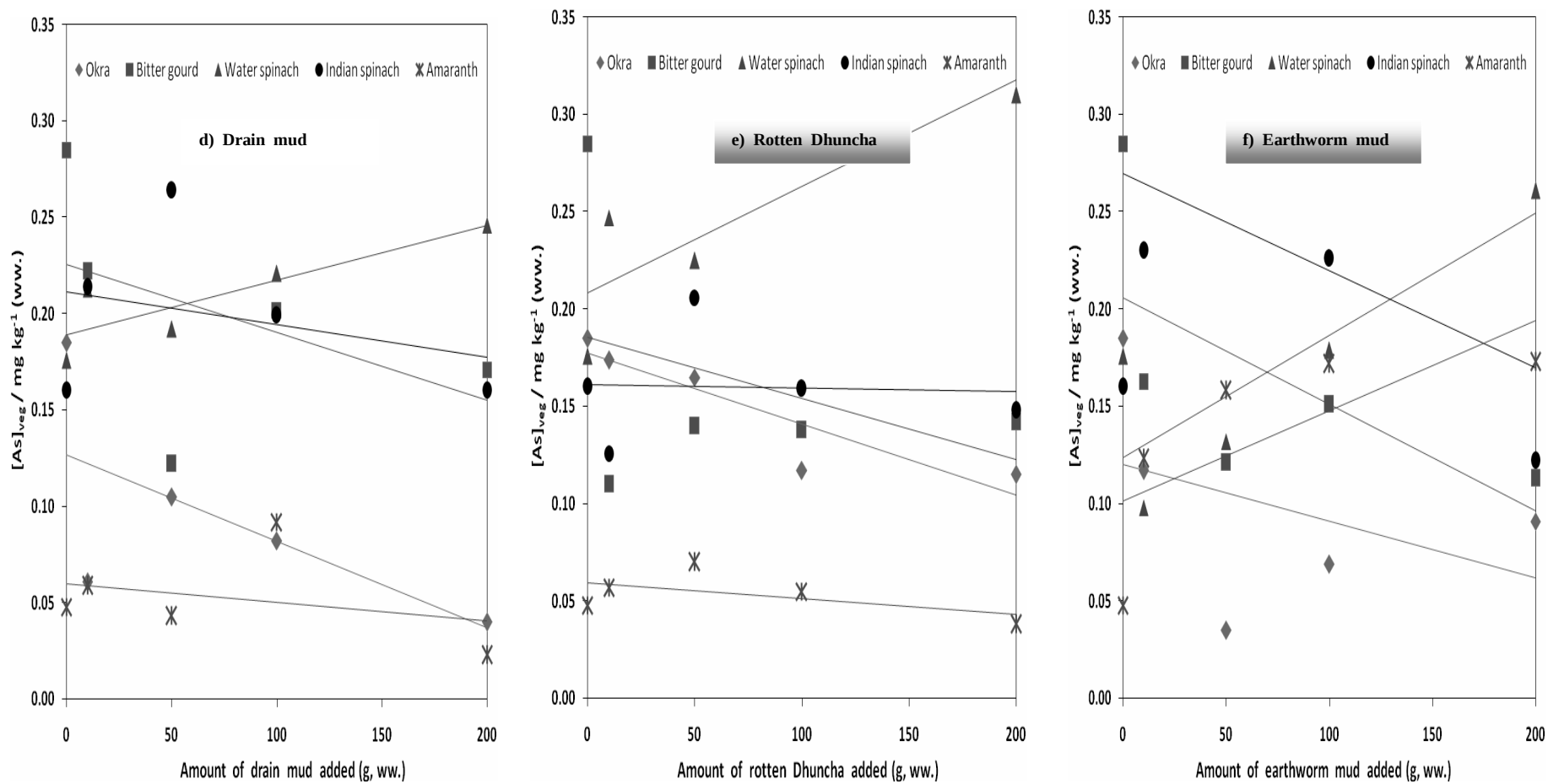


Figure-3.8. Effect of the six fertilizers on accumulation of arsenic in vegetables.

Table -3.3.8. Concentration of cadmium in the vegetables (mg kg⁻¹).

Type of bed	Concentration of Cd in vegetables (mg kg ⁻¹) [§]				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control-1	0.039 (0.214)	0.003 (0.034)	0.002 (0.021)	0.013 (0.249)	0.004 (0.081)
Control-2	0.045 (0.186)	0.003 (0.029)	0.006 (0.069)	0.016 (0.274)	0.022 (0.199)
Control-Average	0.042 (0.200)	0.003 (0.032)	0.004 (0.048)	0.015 (0.262)	0.013 (0.140)
CD-10	0.041 (0.157)	0.001 (0.013)	0.001 (0.020)	0.013 (0.250)	0.004 (0.065)
CD-50	0.033 (0.197)	0.001 (0.012)	0.004 (0.032)	0.018 (0.386)	0.007 (0.073)
CD-100	0.027 (0.155)	0.001 (0.011)	0.003 (0.022)	0.014 (0.264)	0.007 (0.067)
CD-200	0.022 (0.128)	0.001 (0.011)	0.001 (0.012)	0.012 (0.229)	0.004 (0.056)
CM-10	0.050 (0.239)	0.003 (0.029)	0.003 (0.037)	0.004 (0.083)	0.005 (0.063)
CM-50	0.044 (0.218)	0.002 (0.016)	0.002 (0.025)	0.004 (0.069)	0.003 (0.045)
CM-100	0.032 (0.174)	0.001 (0.013)	0.002 (0.021)	0.004 (0.075)	0.010 (0.139)
CM-200	0.016 (0.095)	0.001 (0.012)	0.003 (0.034)	0.003(0.051)	0.018 (0.233)
WH-10	0.048 (0.270)	0.002 (0.023)	0.002 (0.027)	0.019 (0.385)	0.002 (0.025)
WH-50	0.034 (0.189)	0.001 (0.010)	0.003 (0.036)	0.017 (0.296)	0.021 (0.180)
WH-100	0.034 (0.172)	0.001 (0.008)	0.003 (0.035)	0.070 (1.320)	0.008 (0.060)
WH-200	0.024 (0.141)	0.001 (0.010)	0.002 (0.019)	0.013 (0.203)	0.025 (0.249)
DM-10	0.002 (0.016)	0.001 (0.012)	0.006 (0.074)	0.004 (0.072)	0.019 (0.241)
DM-50	0.014 (0.097)	0.001 (0.013)	0.004 (0.045)	0.018 (0.351)	0.022 (0.249)
DM-100	0.016 (0.103)	0.001 (0.010)	0.005 (0.062)	0.022 (0.409)	0.017 (0.235)
DM-200	0.022 (0.165)	0.001 (0.009)	0.003 (0.035)	0.037 (0.738)	0.016 (0.239)
RD-10	0.031 (0.172)	0.001 (0.012)	0.004 (0.050)	0.012 (0.196)	0.006 (0.076)
RD-50	0.045 (0.288)	0.001 (0.009)	0.002 (0.028)	0.014 (0.270)	0.004 (0.061)
RD-100	0.030 (0.133)	0.002 (0.019)	0.002 (0.021)	0.013 (0.181)	0.021 (0.238)
RD-200	0.016 (0.087)	0.001 (0.008)	0.001 (0.018)	0.009 (0.175)	0.005 (0.065)
EW-10	0.030 (0.163)	0.001 (0.013)	0.002 (0.023)	0.016 (0.291)	0.021 (0.259)
EW-50	0.019 (0.143)	0.001 (0.010)	0.002 (0.025)	0.014 (0.264)	0.020 (0.220)
EW-100	0.010 (0.058)	0.001 (0.012)	0.003 (0.033)	0.011 (0.217)	0.017 (0.196)
EW-200	0.001 (0.007)	0.001 (0.009)	0.002 (0.021)	0.008 (0.139)	0.012 (0.154)

[§] the values in parentheses represent dry weight.

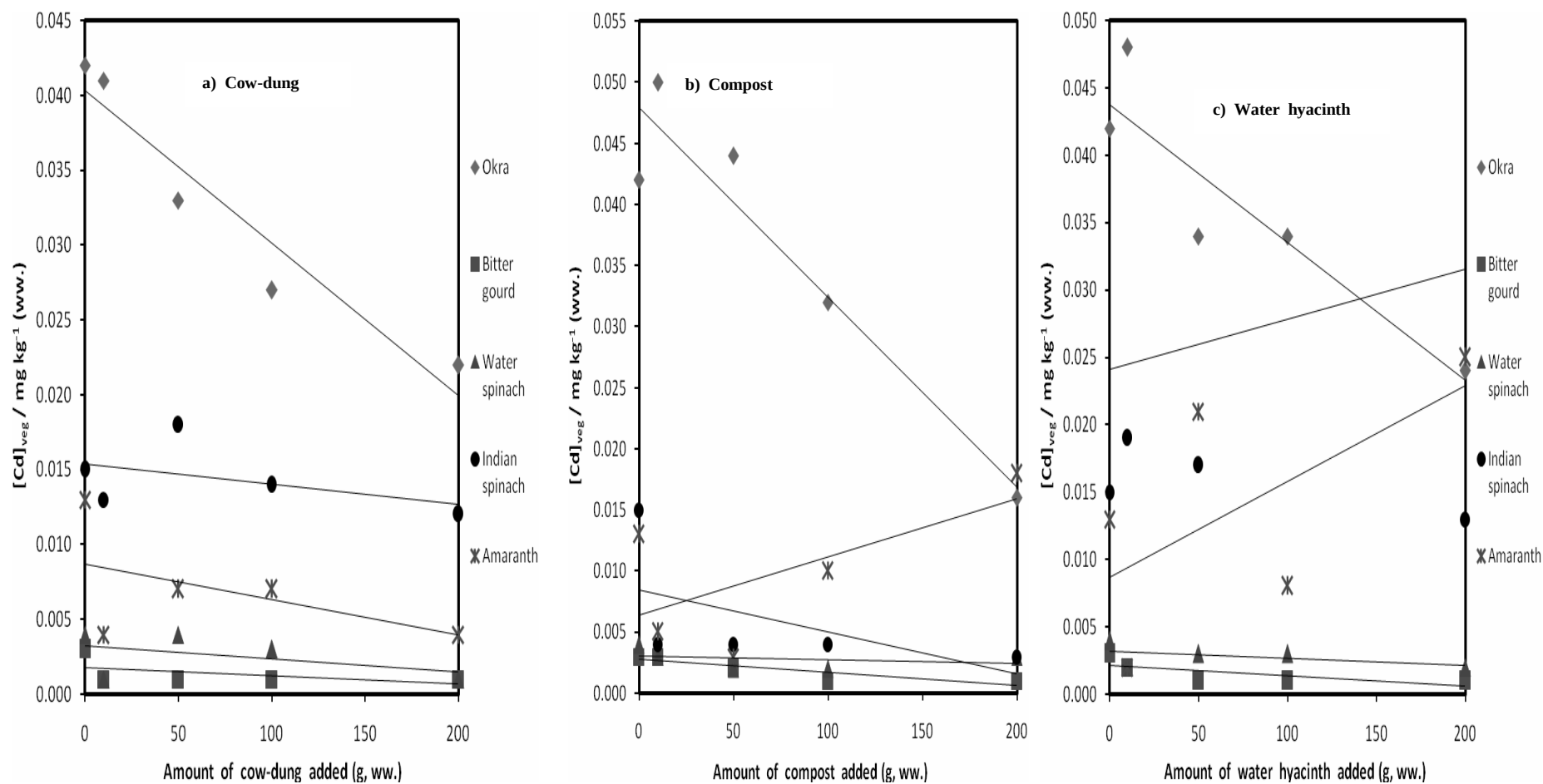


Figure-3.9. Effect of the six fertilizers on accumulation of cadmium in vegetables.

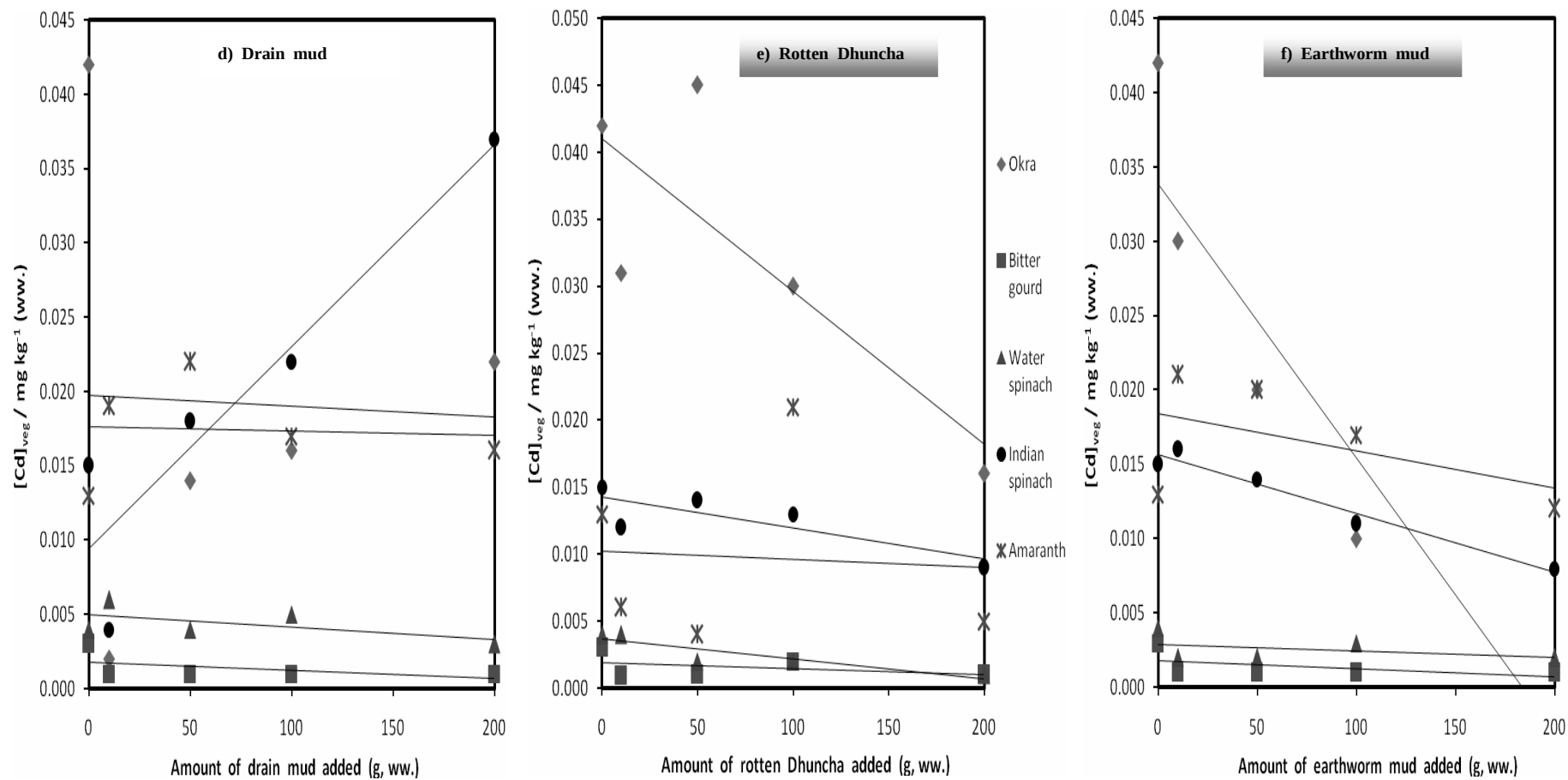


Figure-3.9. Effect of the six fertilizers on accumulation of cadmium in vegetables.

Table -3.3.9. Concentration of lead in the vegetables (mg kg⁻¹).

Type of bed	Concentration of Pb in vegetables (mg kg ⁻¹) [§]				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control-1	0.016 (0.092)	0.013 (0.081)	0.004 (0.042)	0.481 (9.866)	0.460 (7.637)
Control-2	0.029 (0.120)	0.033 (0.380)	0.006 (0.077)	0.564 (8.472)	0.683 (6.001)
Control-Average	0.023 (0.106)	0.023 (0.265)	0.005 (0.060)	0.523 (9.169)	0.572 (6.819)
CD-10	0.028 (0.106)	0.003 (0.049)	0.008 (0.084)	0.484 (8.818)	0.439 (6.643)
CD-50	0.016 (0.099)	0.005 (0.081)	0.004 (0.042)	0.494 (8.830)	0.594 (6.769)
CD-100	0.024 (0.134)	0.006 (0.069)	0.002 (0.028)	0.507 (9.003)	0.551 (6.270)
CD-200	0.015 (0.120)	0.004 (0.063)	0.005 (0.056)	0.482 (8.616)	0.518 (5.983)
CM-10	0.026 (0.124)	0.019 (0.209)	0.004 (0.056)	0.524 (9.367)	0.459 (5.230)
CM-50	0.025 (0.099)	0.016 (0.184)	0.006 (0.060)	0.529 (9.456)	0.504 (6.009)
CM-100	0.033 (0.130)	0.005 (0.077)	0.004 (0.045)	0.534 (9.544)	0.543 (7.152)
CM-200	0.024 (0.099)	0.007 (0.070)	0.012 (0.123)	0.548 (8.653)	0.514 (6.400)
WH-10	0.024 (0.127)	0.018 (0.193)	0.007 (0.092)	0.780 (13.349)	0.600 (6.837)
WH-50	0.021 (0.109)	0.011 (0.095)	0.007 (0.084)	0.541 (8.459)	0.782 (8.923)
WH-100	0.021 (0.127)	0.011 (0.092)	0.009 (0.099)	1.751 (32.223)	0.707 (8.056)
WH-200	0.015 (0.092)	0.008 (0.099)	0.006 (0.074)	0.543 (8.179)	0.783 (8.807)
DM-10	0.011 (0.081)	0.016 (0.195)	0.004 (0.042)	0.502 (9.955)	0.565 (6.438)
DM-50	0.016 (0.092)	0.005 (0.060)	0.004 (0.049)	0.487 (9.118)	0.517 (5.756)
DM-100	0.015 (0.080)	0.012 (0.116)	0.007 (0.063)	0.499 (8.920)	0.554 (6.306)
DM-200	0.013 (0.074)	0.011 (0.094)	0.009 (0.101)	0.432 (7.712)	0.452 (6.657)
RD-10	0.027 (0.148)	0.009 (0.120)	0.008 (0.084)	0.563 (10.229)	0.559 (6.628)
RD-50	0.038 (0.120)	0.008 (0.092)	0.005 (0.056)	0.385 (6.882)	0.504 (5.743)
RD-100	0.016 (0.063)	0.004 (0.046)	0.007 (0.081)	0.451 (8.062)	0.607 (6.863)
RD-200	0.024 (0.092)	0.027 (0.295)	0.009 (0.092)	0.403 (6.784)	0.517 (5.691)
EW-10	0.026 (0.116)	0.013 (0.113)	0.005 (0.062)	0.572 (10.225)	0.600 (6.837)
EW-50	0.015 (0.116)	0.010 (0.106)	0.007 (0.079)	0.639 (11.390)	0.572 (6.232)
EW-100	0.021 (0.134)	0.014 (0.150)	0.009 (0.106)	0.575 (10.278)	0.571 (6.115)
EW-200	0.026 (0.134)	0.014 (0.148)	0.014 (0.123)	0.562 (9.937)	0.686 (7.817)

[§] the values in parentheses represent dry weight.

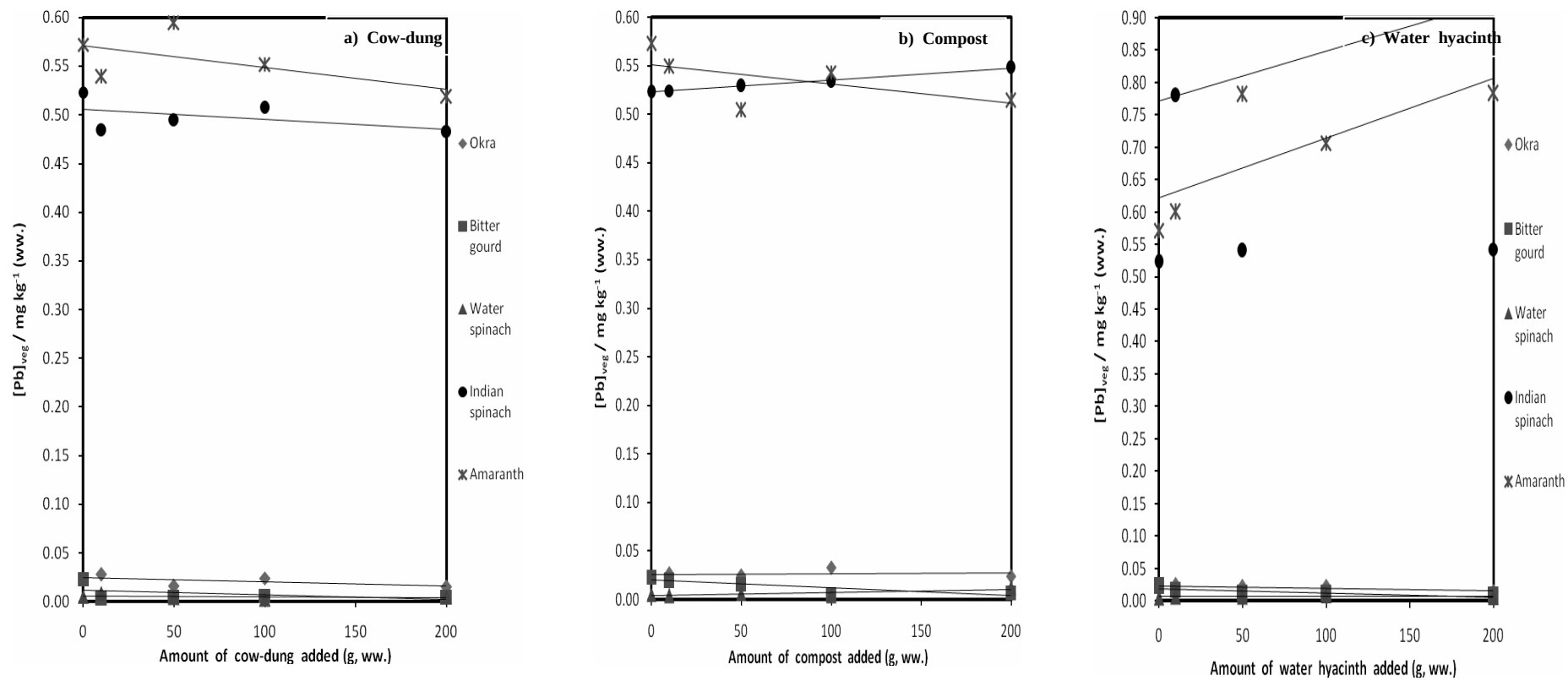


Figure-3.10. Effect of the six fertilizers on accumulation of lead in vegetables.

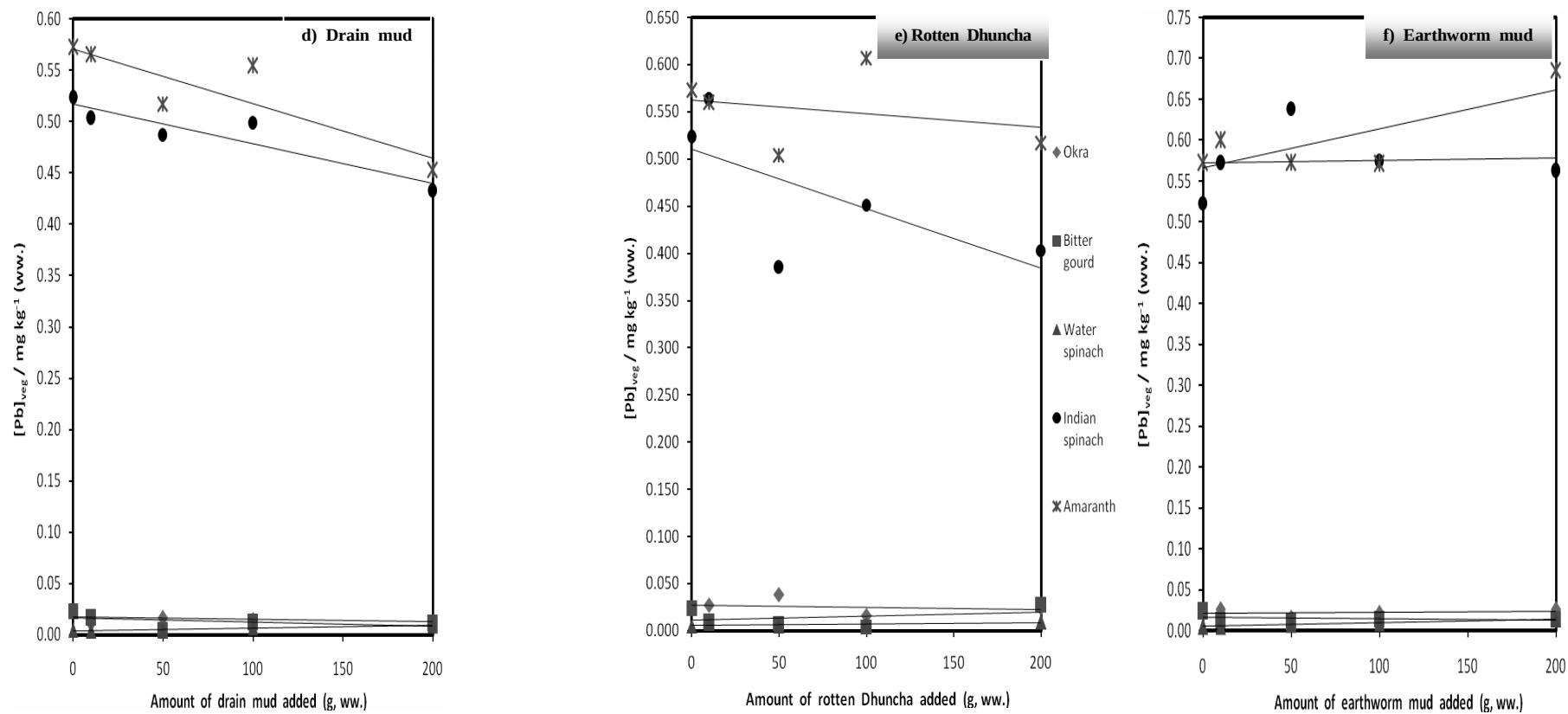


Figure-3.10. Effect of the six fertilizers on accumulation of lead in vegetables.

3.3.7 . Statistical Analyses of Concentrations of Arsenic, Lead and Cadmium in The Vegetables:

The central tendency indicating the location of the distribution were measured by mean and median, while dispersion showing the dissimilarity of the values by range, standard deviation and variance. These were analyzed for each group of the vegetables for arsenic, lead and cadmium using SPSS. The analysis results are represented in Table - 3.3.10.

Table -3.3.10. Statistical analyses of arsenic, cadmium and lead data sets of the vegetables.

Vegetables	Range (Min - Max)	Mean $\pm$ SE	Geomet- ric Mean	Median	Standard Deviation	Variance
Arsenic (As):						
Okra	0.035 – 0.226 (0.274 – 0.913)	0.118 $\pm$ 0.010 (0.554 $\pm$ 0.035)	0.106 (0.529)	0.106 (0.517)	0.053 (0.176)	0.003 (0.031)
Bitter gourd	0.083 – 0.297 (1.108 – 3.284)	0.169 $\pm$ 0.013 (1.709 $\pm$ 0.114)	0.158 (1.636)	0.147 (1.499)	0.066 (0.582)	0.004 (0.339)
Water spinach	0.098 – 0.310 (1.011 – 3.137)	0.194 $\pm$ 0.010 (1.958 $\pm$ 0.090)	0.188 (1.906)	0.191 (1.948)	0.050 (0.457)	0.003 (0.209)
Indian spinach	0.074 – 0.430 (1.394 – 7.597)	0.182 $\pm$ 0.014 (3.294 $\pm$ 0.239)	0.170 (3.108)	0.164 (3.173)	0.072 (1.218)	0.005 (1.484)
Amaranth	0.023 – 0.173 (0.341 – 2.219)	0.072 $\pm$ 0.008 (0.815 $\pm$ 0.089)	0.063 (0.724)	0.059 (0.681)	0.042 (0.452)	0.002 (0.205)
Cadmium (Cd):						
Okra	0.001 – 0.186 (0.007 – 0.288)	0.033 $\pm$ 0.007 (0.153 $\pm$ 0.013)	0.023 (0.126)	0.030 (0.160)	0.034 (0.068)	0.001 (0.005)
Bitter gourd	0.001 – 0.003 (0.008 – 0.034)	0.001 $\pm$ 0.000 (0.014 $\pm$ 0.001)	0.001 (0.013)	0.001 (0.012)	0.001 (0.007)	0.000 (0.000)
Water spinach	0.001 – 0.006 (0.002 – 0.074)	0.003 $\pm$ 0.000 (0.029 $\pm$ 0.003)	0.003 (0.025)	0.003 (0.026)	0.001 (0.016)	0.000 (0.000)
Indian spinach	0.003 – 0.070 (0.051 – 1.320)	0.015 $\pm$ 0.003 (0.286 $\pm$ 0.050)	0.012 (0.220)	0.013 (0.250)	0.013 (0.254)	0.000 (0.065)
Amaranth	0.002 – 0.063 (0.025 – 0.259)	0.015 $\pm$ 0.002 (0.145 $\pm$ 0.016)	0.011 (0.118)	0.014 (0.147)	0.012 (0.084)	0.000 (0.007)
Lead (Pb):						
Okra	0.011 – 0.038 (0.063 – 0.148)	0.022 $\pm$ 0.001 (0.109 $\pm$ 0.004)	0.021 (0.107)	0.023 (0.113)	0.007 (0.022)	0.000 (0.000)
Bitter gourd	0.003 – 0.033 (0.046 – 0.380)	0.012 $\pm$ 0.001 (0.126 $\pm$ 0.015)	0.010 (0.109)	0.011 (0.097)	0.007 (0.078)	0.000 (0.006)
Water spinach	0.002 – 0.014 (0.028 – 0.123)	0.007 $\pm$ 0.001 (0.073 $\pm$ 0.005)	0.006 (0.069)	0.007 (0.075)	0.003 (0.026)	0.000 (0.001)
Indian spinach	0.385 – 1.751 (6.784 – 32.223)	0.570 $\pm$ 0.050 (10.090 $\pm$ 0.923)	0.543 (9.565)	0.527 (9.061)	0.253 (4.709)	0.064 (22.177)
Amaranth	0.439 – 7.152 (5.230 – 8.923)	1.289 $\pm$ 0.397 (6.685 $\pm$ 0.180)	0.754 (6.628)	0.568 (6.533)	2.025 (0.919)	4.101 (0.846)

The values in parentheses represent dry weight. The unit of the parameters used in table 3.3.7 are mgkg^{-1} and $(\text{mgkg}^{-1})^2$.

3.3.8. Efficiency of the Fertilizers:

In order to understand the efficiency of the studied organic fertilizers towards remedial measure of arsenic, cadmium and lead poisoning in vegetable plants, a comparison was made with respect to the control. For this, the average concentrations of arsenic, cadmium and lead found in the studied five vegetables as a function of the applied fertilizers were first tabulated. These are represented in the following three tables.

Table-3.3.11. The average concentration of arsenic in the fresh vegetables grown in fertilizers used bed.

	Average concentration of arsenic (mg kg ⁻¹ , ww.)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	0.185	0.285	0.176	0.160	0.048
Cow-dung	0.103	0.099	0.186	0.162	0.050
Compost	0.192	0.206	0.177	0.100	0.052
Water hyacinth	0.089	0.203	0.162	0.220	0.080
Drain mud	0.072	0.179	0.218	0.209	0.054
Rotten Dhuncha	0.143	0.133	0.266	0.160	0.055
Earth worm mud	0.078	0.137	0.168	0.252	0.157

Table-3.3.12.The average concentration of cadmium in the fresh vegetables grown in fertilizers used bed.

	Average concentration of cadmium (mg kg ⁻¹ , ww.)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	0.042	0.003	0.004	0.015	0.013
Cow-dung	0.031	0.001	0.002	0.014	0.006
Compost	0.036	0.002	0.003	0.004	0.009
Water hyacinth	0.035	0.001	0.003	0.030	0.014
Drain mud	0.014	0.001	0.005	0.020	0.019
Rotten Dhuncha	0.031	0.001	0.002	0.012	0.009
Earth worm mud	0.015	0.001	0.002	0.012	0.018

Table-3.3.13. The average concentration of lead in the fresh vegetables grown in fertilizers used bed.

	Average concentration of lead (mg kg ⁻¹ , ww.)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	0.023	0.023	0.005	0.523	0.572
Cow-dung	0.021	0.005	0.005	0.492	0.551
Compost	0.027	0.012	0.007	0.534	0.528
Water hyacinth	0.020	0.012	0.007	0.904	0.718
Drain mud	0.014	0.011	0.006	0.480	0.522
Rotten Dhuncha	0.026	0.012	0.007	0.451	0.547
Earth worm mud	0.022	0.013	0.009	0.587	0.607

The change in concentration of arsenic, cadmium and lead accumulated in the studied vegetables with respect to the control values were calculated. In the change, increase is indicated by (+) sign and decrease by (-) sign. The results are summarized in metal wise as in Table-3.3.14, 3.3.15 and 3.3.16.

Table-3.3.14. Percent change in concentration of arsenic with respect to control.

	Change in concentration of arsenic with respect to control (%)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	(-) 44.32	(-) 65.26	(+) 5.68	(+) 1.25	(+) 4.17
Cow-dung	(+) 3.78	(-) 27.72	(+) 0.57	(-) 37.50	(+) 8.33
Compost	(-) 51.89	(-) 28.77	(-) 7.95	(+) 37.50	(+) 66.67
Water hyacinth	(-) 61.08	(-) 37.19	(+) 23.86	(+) 30.63	(+) 12.50
Drain mud	(-) 22.70	(-) 53.33	(+) 51.14	(±) 0.00	(+) 14.58
Rotten Dhuncha	(-) 57.84	(-) 51.93	(-) 4.55	(+) 57.50	(+) 227.08
Earth worm mud	(-) 44.32	(-) 65.26	(+) 5.68	(+) 1.25	(+) 4.17

Table-3.3.115. Percent change in concentration of cadmium with respect to control.

	Change in concentration of cadmium with respect to control (%)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	(-) 26.19	(-) 66.67	(-) 50.00	(-) 6.67	(-) 53.85
Cow-dung	(-) 14.29	(-) 33.33	(-) 25.00	(-) 73.33	(-) 30.77
Compost	(-) 16.67	(-) 66.67	(-) 25.00	(+) 100.00	(+) 7.69
Water hyacinth	(-) 66.67	(-) 66.67	(+) 25.00	(+) 33.33	(+) 46.15
Drain mud	(-) 26.19	(-) 66.67	(-) 50.00	(-) 20.00	(-) 30.77
Rotten Dhuncha	(-) 64.29	(-) 66.67	(-) 50.00	(-) 20.00	(+) 38.46
Earth worm mud	(-) 26.19	(-) 66.67	(-) 50.00	(-) 6.67	(-) 53.85

Table-3.3.16. Percent change in concentration of lead with respect to control.

	Change in concentration of lead with respect to control (%)				
	Okra	Bitter gourd	Water spinach	Indian spinach	Amaranth
Control	(-) 8.70	(-) 78.26	(±) 0.00	(-) 5.93	(-) 3.67
Cow-dung	(+) 17.39	(-) 47.83	(+) 40.00	(+) 2.10	(-) 7.69
Compost	(-) 13.04	(-) 47.83	(+) 40.00	(+) 72.85	(+) 25.52
Water hyacinth	(-) 39.13	(-) 52.17	(+) 20.00	(-) 8.22	(-) 8.74
Drain mud	(+) 13.04	(-) 47.83	(+) 40.00	(-) 13.77	(-) 4.37
Rotten Dhuncha	(-) 4.35	(-) 43.48	(+) 80.00	(+) 12.24	(+) 6.12
Earth worm mud	(-) 8.70	(-) 78.26	(±) 0.00	(-) 5.93	(-) 3.67

The overall change for arsenic, cadmium and lead is shown in Table 3.3.17.

Table-3.3.17. Percent change in concentration of arsenic, cadmium and lead with respect to control.

Overall change (%)			
	As	Cd	Pb
Cow-dung	(-) 19.70	(-) 40.67	(-) 19.31
Compost	(-) 10.51	(-) 35.34	(+) 0.80
Water hyacinth	(+) 3.11	(-) 0.13	(+) 15.50
Drain mud	(-) 6.26	(-) 5.77	(-) 17.65
Rotten Dhuncha	(-) 2.06	(-) 38.73	(-) 2.58
Earthworm mud	(+) 34.05	(-) 32.50	(+) 10.11

The findings can be represented in the following Figure 3.11.

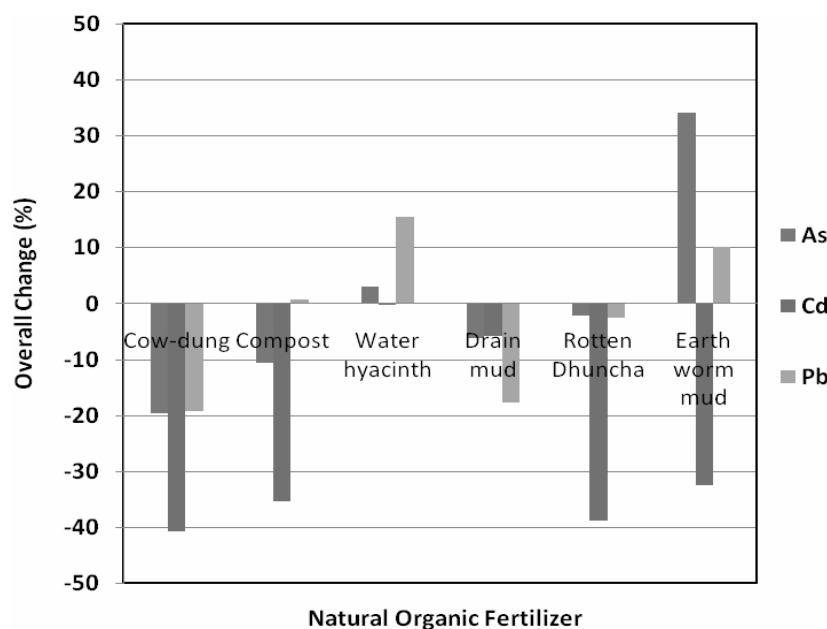


Figure-3.11. Efficiency of the studied six natural organic fertilizers to reduce toxic ions.

The above Table and Figure show that the studied six natural organic fertilizers can be arranged in the following order in terms of their efficiencies to reduce the toxic ions.

For Arsenic: Cow-dung > Compost > Drain mud > Rotten Dhuncha > Water hyacinth > Earthworm mud

For Cadmium: Cow-dung > Rotten Dhuncha > Compost > Earthworm mud > Drain mud > Water hyacinth

For Lead: Cow-dung > Drain mud > Rotten Dhuncha > Compost > Earthworm mud > Water hyacinth

Therefore, the overall order for all the fertilizers are

Cow-dung > Drain mud > Rotten Dhuncha > Compost > Earthworm mud > Water hyacinth

The above findings suggest that the first four natural organic fertilizers of overall order, namely, cow-dung, drain mud, rotten dhuncha and compost, can be utilized in the agricultural soil for simultaneous reduction of arsenic, cadmium and lead toxicity of vegetables plants. Fortunately, in Bangladesh the usual practice of the farmers is to use the mentioned four fertilizers. They sometimes replace drain mud by pond mud or ditch mud. However, earthworm mud and water hyacinth are not frequently used due to their relatively low availability.

Inspection of the carbon to nitrogen ratio (C:N) of the studied fertilizers (Table 3.3.3) show that the overall average C:N ratio of the fertilizers is 55.44 (C as 20.51% and N as 0.68%). The overall average organic matter (OM) of the fertilizers is 38.83%, a moderate level. Both of these reveal that the fertilizers are good for use in agricultural purpose. It is also reported that the studied fertilizers are very good in restoring soil texture, good soil condition for bacterial growth and many other properties. This is supported in our data.

3.4. CONCLUSIONS:

Analyses of irrigation water, cultivated soils, the natural organic fertilizers (rotten cow-dung, rotten Dhuncha tree, compost, drain mud, dried water hyacinth and earthworm mud) and edible plant tissues of the vegetables (Okra, Bitter gourd, Water spinach, Indian spinach and Amaranth) lead to the following conclusions:

1. The overall average organic matter (OM) of the fertilizers (38.83%), average pH of 7.16 and overall average C:N ratio of the fertilizers (55.44) indicate that the studied fertilizers are good for use in agricultural purpose, especially for developing desired soil texture and bacterial growth.
2. Analysis of sand, clay and silt shows that the studied soil is of silt loam type. The acidity of the soil is increased slightly after being amended with the fertilizers. Its final average pH of 6.15 is very suitable for vegetable plant growth, since the majority of vegetable plants prefer a soil pH of around 6 to 7 (characteristic soil of moist climate).
3. When the studied natural organic fertilizer is mixed with soil, its organic matter (OM) content is increased to 57%. Microbiological examination shows that amongst the bacteria, only *Fecal Coliform* is increased to about 10^4 fold when fertilizers are added.
4. The irrigation water is hard and nearly neutral in nature, has higher value of electrical conductivity and contains no Fecal Coliform bacteria. But it also contains arsenic, cadmium and lead as 5 mg L^{-1} each (fortified).
5. Generally, as the amount of applied natural organic fertilizers were increased, the plant height as well as percent yield were both increased. The increment roughly follows a linear pattern. This is due to improvement in plant health as more fertilizers are added. This may be caused by increment in organic matter (OM) of soil

that has a tendency to adsorb the toxic ions of As, Pb and Cd, and enrichment in bacteria which facilitate reduction, bio-methylation, etc. of ions of As, Pb and Cd to generate their volatile gases.

6. After harvesting with contaminated water, total As, Pb and Cd content in soil is decreased with respect to control in most cases, although 120 mg of each of As, Pb and Cd is added in 2.25 sq. ft area through irrigation and the fertilizers themselves contain considerable amount of As, Pb and Cd.
7. Although plant phytotoxicity differ from metal to metal, bitter gourd is least exposed to As, Cd and Pb toxicity while Indian spinach is most exposed of those.
8. The overall efficiency of the studied fertilizers in simultaneous reduction of arsenic, cadmium and lead toxicity in vegetables is as follows: Cow-dung > Drain mud > Rotten Dhuncha > Compost > Earthworm mud > Water hyacinth.

The concluding remarks refer that the first four fertilizers, namely, cow-dung, drain mud, rotten Dhuncha and compost, could be recommended for remedy of arsenic, cadmium and lead toxicity in vegetables. Fortunately, in Bangladesh and in many Asian countries, the usual practice of the farmers is to use the mentioned four fertilizers.

3.5 References:

[1]	Zhang XW, Yan XJ, Zhou ZR et al. Arsenic Trioxide Controls the Fate of the PML-RAR{alpha} Oncoprotein by Directly Binding PML. <i>Science</i> 2010; 328(5975):240-243.
[2]	Baker A. J. M., (1981). Accumulators and excluders-strategies in the response of plants to heavy metals. <i>J. Plant Nutr.</i> 3 643–654.10.1080/01904168109362867
[3]	Temmerman De. L., Ruttens A., Waegeneers N., 2012. Impact of atmospheric deposition of As, Cd and Pb on their concentration in carrot and celeriac. <i>Environ pollut</i> 166, 187-195.
[4]	Azizur Rahman M., Hasegawa H., 2011. Aquatic arsenic: Phytore-mediation using floating macrophytes. <i>Chemosphere</i> 83, 633–646.
[5]	Marschner H(1995) Mineral nutrition of higher plants. Academic Press, London.
[6]	Moreno-Jiménez E., Esteban E., Peñalosa J.M., (2012). The fate of arsenic in soil-plant systems. <i>Rev Environ Contam Toxicol</i> 215, 1-37.
[7]	Silva Gonzaga M.I., Ma L.Q., Pacheco E.P., Santos Dos W.M., 2012. Predicting arsenic bioavailability to hyperaccumulator <i>Pteris Vittata</i> in arsenic-contaminated soils. <i>Int J Phytorem</i> 14, 939-949.
[8]	Ghosh P., Rathinasabapathi B., Ma L.Q., 2011. Arsenic-resistant bacteria solubilized arsenic in the growth media and increased growth of arsenic hyperaccumulator <i>Pteris vittata</i> L. <i>Bioresource Technol</i> 102, 8756-8761.
[9]	Di Lonardo, S., Capuana, M., Arnetoli, M., Gabbrielli, R., Gonnelli, C. 2011. Exploring the metal phytoremediation potential of three <i>Populus alba</i> L. clones using an in vitro screening. <i>Environ Sci Pol-lut Res Int</i> 18(1), 82-90.
[10]	Afrous, A., Manshouri, M., Liaghat, A., Pazira, E., Sedghi, H. 2011. Mercury and arsenic accumulation by three species of aquatic plants in Dezful, Iran. <i>Afr J Agric Res</i> 6(24), 5391-5397.
[11]	Ma, L.Q., Komar, K.M., Tu, C., Zhang, W., Cai, Y., Kennelly, E.D. 2001. A fern that hyperaccumulates arsenic. <i>Nature</i> 409, 579.
[12]	Smith, P.G., Koch, I., Reimer, K.J. 2008. Uptake, transport and transformation of arsenate in radishes (<i>Raphanus sativus</i>). <i>Sci Total Environ</i> 390, 188-197.
[13]	Bergqvist, C., Greger, M. 2012. Arsenic accumulation and specia-tion in plants from different habitats. <i>Appl Geochem</i> 27, 615–622.
[14]	Zhao, F.J., Ma, J.F., Meharg, A.A., McGrath, S.P. 2009. Arsenic uptake and metabolism in plants. <i>New Phytol</i> 181, 777-794.

[15]	Azizur Rahman, M., Kadohashi, Maki, T.K., Hasegawa. 2011. Transport of DMAA and MMAA into rice (<i>Oryza sativa</i> L.) roots. <i>Environ Exp Bot</i> 72, 41-46.
[16]	Moreno-Jiménez, E., Esteban, E., Peñalosa, J.M. (2012). The fate of arsenic in soil-plant systems. <i>Rev Environ Contam Toxicol</i> 215, 1-37.
[17]	Anastasia F.B. and Kender W.j. Influence of soil arsenic on the growth of lowbush blueberry. <i>J. Environ. Qual.</i> , 2 (3) (1973) 335-337.
[18]	Liu G.L., Cheng F.X., gao S. D. and Li M. Q , Effect of arsenic in soil on plants. <i>Zhongguo Nongye Kexue (Beijihg)</i> , 4(1985)9-16.
[19]	Pitten F.A., Miiller G, Konig P., Schmidt D., Thurow K. and Krameer A., Risk assessment of a former military base contaminate with organoarsenic-based warfare fagents: uptake of arsenic by terrestrial plants. <i>The Science of the Total Environment</i> , 226(1999) 237-245.
[20]	Milton A. and Johnson M. Arsenic in the food chains of a revegetated metalliferous mine trailing pond. <i>Chemosphere</i> , 39(5) (1999) 765-779.
[21]	Williams K. T. and Whetstone R, R,. Arsenic dist6ribution in soils and its presence in certain plants. U. S. Department of Agriculture, 1940.
[22]	Huq S.M.I. and Naidu R. Arsenic in groundwater of BangladeshL contamination in food chain. Papers presented on “Arsenic contamination in Bangladesh, May 18-23, 2002.
[23]	Liu G.L. and Gao S.R., Effect of arsenium in red soil on crop. <i>Turang Tonghao</i> , 18(5)(1987) 231-233.
[24]	Ali M. A, Badruzzaman A.B, M., et al, Arsenic in plant-sil environment in Bangladesh. United Nations University, ToKyo, japan, 1987, pp 85-112.
[25]	CAL Limei, HUANG Lanchun, ZHOU Yongzhang, XU Zhencheng, PENG Xiaochun, YAO Ling'ai, ZHOU Yang and PENG Ping'an: Heavy metal concentrations of agriculture soils and vegetables from Dongguan, Guangdong. Important National Science & Technology Specific Projects of China, No.2007zxo7211.
[26]	Jump up ^ Gilluly, Waters, Woodford (1975). <i>Principles of Geology</i> (4 th ed.). USA: W.H. Freeman. ISHN 978-0716702696.
[27]	Jump up Chesworth, Ward, ed. (2008). <i>Encyclopedia of soil science</i> .Dordrecht,Netherlands: Springer. xxiv. ISB 1-4020-3994-8.
[28]	Jump up to:“Sources. Negative charge:”. Jan.ucc.nau.edu. Retrieved 2012-11-07.

[29]	Available Jump up ^ http://www.nature/journal/v433/n7023/full/433204a.html
[30]	Recommended Soil pH for Growing Garden Fruits and Vegetables. http://www.thegardenhelper.com/soilPH.htm
[31]	Roger Nelson's soil texture triangle: Hydraulic properties calculator. http://staffweb.wilkes.edu/brian.oram/soilwatr.htm
[32]	People's report 2002-2003, Bangladesh Environment Volume :II Database.
[33]	Composition of Organic Matter. In: Plant Pathology. My Agriculture Information Bank. http://agriinfo.in/?page=topic&superid=5&topicid=169 [accessed November 25, 2012]
[34]	N. C. Brady, The nature and properties of soils. 10 th Edition, Prentice-Hall of India Private Limited, New Delhi, 1999.
[35]	BGS/DPHE, Arsenic contamination of groundwater in Bangladesh. <i>BGS Technical Report WC/00/19</i> , Vol 2: Final report. British Geological Survey (UK) and Department of Public Health Engineering (Bangladesh), 2001, pp. 77-207.
[36]	US EPA. 1992. Ground Water Issue. Behavior of Metals in Soils. Office of Research and Development. Office of Solid Waste and Emergency Response. EPA/540/S-92/018. United States Environmental Protection Agency.
[37]	Gardea-Torresdey, J.L; Tiemann; K.J; Gamez, G.D; 1999. Effect of chemical composition for multi-metal binding by <i>Medicago Sativa(Alfalfa)</i> . J.Hazard. Mater. B 69, 41-51.
[38]	Horsfall, M.Jnr; Abia, A.A; 2003. Sorption of Cadmium (II) and Zinc (II) ions from aqueous solutions by cassava waste biomass. Water Research, 37, 4913-4923.
[39]	Horsfall, M. Jnr; Spiff, A.I; 2005. Kinetics studies on the sorption of lead and cadmium ions from aqueous solution by <i>Caladium bicolor</i> (wild Cocoyam) Biomass. Bull. Chem.Soc. Ethiop. 19(1), 89-102.
[40]	Xuejun Guo and Fuhua Chen, Removal of Arsenic by Bead Cellulose Loaded with Iron Oxyhydroxide from Groundwater. College of Environmental Science and Engineering, Nankai University, Tianjin, China 300071.
[41]	Mohammad Mosaferi, ^{1,2} Sepideh Nemati, ³ Alireza Khataee, ⁴ Simin Nasseri, ² and Ahmad Asl Hashemi ⁵ ; Removal of Arsenic (III, V) from aqueous solution by nanoscale zero-valent iron stabilized with starch and carboxymethyl cellulose.

[42]	Stamberg, J.; Peska, J. Preparation of porous spherical cellulose. <i>React. Polym.</i> 1983 , <i>1</i> , 145-147.
[43]	Boeden, H. F.; Pommerening, K.; Becker, M.; Rupprich, C.; Holtzhauer, M. Bead cellulose derivatives as supports for immobilization and chromatographic purification and proteins. <i>J. Chromatogr.</i> 1991 , <i>552</i> , 389-414.
[44]	Wolf, B. Bead cellulose products with film formers and solubilizers for controlled drug release. <i>Int. J. Pharm.</i> 1997 , <i>156</i> , 97-107.
[45]	Kendorff, H., and M. Schnitzer. 1980. Sorption of metals on humic acid. <i>Geoch. Cosmochim. Acta</i> 44:1701–1708.
[46]	Elliot, H.A., M.R. Liberati, and C.P. Huang. 1986. Competitive adsorption of heavy metals by soils. <i>J. Environ. Qual.</i> 15(3):214–219.
[47]	Jin, X., G.W. Bailey, Y.S. Yu, and A.T. Lynch. 1996. Kinetics of single and multiple metal ion sorption processes on humic substances. <i>Soil Sci.</i> 1996(8):509–520.
[48]	. Suave, S., M. Mc Bride, and W. Hendershot. 1998. Soil solution speciation of lead (II): Effects of organic matter and pH. <i>Soil Sci. Soc Am. J.</i> 62:618–621.
[49]	Daniel G. Strawn and Donald L. Sparks. Effects of Soil Organic Matter on the Kinetics and Mechanisms of Pb(II) Sorption and Desorption in Soil. <i>Soil Sci. Soc. Am. J.</i> 64:144–156 (2000).
[50]	Manceau, A., M.C. Bosset, G. Sarret, J.-L. Hazemann, M. Mench, P. Cambier, and R. Prost. 1996. Direct determination of lead speciation in contaminated soils by EXAFS spectroscopy. <i>Environ. Sci. Technol.</i> 30(5):1540–1552.
[51]	Piotr Skodowski, Alina Maciejewska and Jolanta Kwiatkowska, The effect of organic matter from brown coal on bioavailability of heavy metals in contaminated soils. <i>Soil and water Pollution Monitoring, Protection and remediation</i> , 3-23. 2006 Springer.
[52]	Fytianos, K., Katsianis, G., Triantafyllon, P., and Zachariadis, G., 1999, Accumulation of heavy metals in vegetables grown in an industrial area in relation to soil, <i>Bull. Environ. Contam. Toxicol.</i> 67:423-430.
[53]	Ge, Y., Murray p., and Hendershot, W. H., 2000, Trace metal speciation and bioavailability in urban soils, <i>J. Environ. Pollut.</i> 107:137-144.
[54]	Romkens, P.F.A.M., Bouwman, L.A., and Boon, G.T., 1999, Effect of plant growth on copper solubility and speciation in soil solution samples. <i>Environ. Pollut.</i> 106:315-321.

[55]	Khan, A. g., Kuek,C., Chandhry, T.M., Khoo, C.S., and Hayes, W. phere J., 2000, Role of plants, mycorrhizae and phytochelators in heavy metal contaminated land remediation, <i>Chemospher</i> , 41:197-207.
[56]	Dudley, L. M., J. E. McLean, R. C. Sims and J. J. Jurinak.1988. Sorption of copper and cadmium from the water-soluble fraction of an acid mine waste by two calcareous soils. <i>Soil Sci</i> . 145:207- 214.
[57]	Dudley, L. M., J. E. McLean, T. H. Furst, and J. J. Jurinak.1991. Sorption of Cd and Cu from an acid mine waste extract by two calcareous soils: column studies. <i>Soil Sci</i> .151:121-135.
[58]	Woolson,E.A.1977a Generation of alkylarsines from soil. <i>Weed Sci</i> 25, 412-416.
[59]	Pongratz, R., Heumann, K.G. 1999. Production of methylated mercury, lead and cadmium by marine bacteria as a significant natural source for atmospheric heavy metals in polar regions. <i>Chemosphere</i> 39, 89-102.

**CHAPTER FOUR: RISK ASSESSMENT TO
ARSENIC, CADMIUM AND LEAD EXPOSURE.**

4. INTRODUCTION

Risk assessment has been defined as “the characterization of the potential adverse health effects of human exposures to environmental hazards” [1]. In a risk assessment, the extent to which a group of people has been or may be exposed to a certain chemical is determined, and the extent of exposure is then considered in relation to the kind and degree of hazard posed by the chemical, thereby permitting an estimate of the present or potential health risk to the group of people involved. Risk assessment typically involves four steps: exposure assessment, toxicity assessment, risk characterization, and uncertainty analysis. The first step is to determine the potential health effects of toxic endpoints that may result from excess exposure to a metal. This is followed by dose-response studies, either conducted through large scale human epidemiologic studies on human populations with a broad range of human exposures or based on animal studies [2]. Human health risk assessment is also defined as the process to estimate the nature and probability of adverse health effects in humans who may be exposed to chemicals in contaminated environmental media, now or in the future, by the United States Environmental Protection Agency (USEPA). Health risk assessment sprang up during the 1980s. It involves identifying the potential of a risk source to introduce risk agents into the environment, estimating the amount of risk agents that come into contact with the human-environment boundaries and quantifying the health consequence of exposure. Since the birth of health risk assessment, it has drawn a lot of attention from many scientists across the world and many assessments relating to drinking purpose and human health have been reported. Alam et al. carried out a study of the water quality conditions of Sylhet city of Bangladesh and its restaurants and assessed the risks to human health. At the same time in China, massive studies have been done on health risk assessment. Li and Tian analyzed health risk caused by heavy metals in a water source and found the health risk comparisons caused by gene toxic substances was $\text{Cr}^{6+} > \text{As} > \text{Cd}$ and risk comparisons caused by body toxic substances was $\text{Pb} > \text{Cu} > \text{Hg}$ [3]. Heavy metals are an important source of food contamination

and health hazard. The main threats to human health are associated with exposure to arsenic, cadmium, lead and mercury and copper. Absorption of heavy metals through food has been shown to have serious consequences on health and thereby economic development associated with a decline in labour productivity as well as the direct costs of treating illnesses such as kidney disease, damage to the nervous system, diminished intellectual capacity, heart disease, gastrointestinal diseases, bone fracture, cancer and death [4]. Trace elements are very harmful because of their non biodegradable nature, long biological half-lives, and their potential to accumulate in different body parts. High concentrations of trace elements (Cu, Cd, and Pb) in vegetables and fruits were related to prevalence of upper gastrointestinal cancer high [5]. Millions of people worldwide consume arsenic-contaminated rice; however, little is known about the uptake and bioavailability of arsenic species after arsenic-contaminated rice ingestion [6]. Environmental risk factors include unsafe water, sanitation and hygiene, indoor and outdoor air pollution, chemicals, wastes, recreational environment, water resources management, land use, noise, radiation, occupation, climate change, and others. Water-pollutants are a major source of morbidity and mortality in developing countries [7]. Heavy metals constitute a very heterogeneous group of elements widely varied in their chemical properties and biological functions. Heavy metals are kept under environmental pollutant category due to their toxic effects in plants, human and food. Arsenic (As), Cadmium (Cd), Lead (Pb), Mercury (Hg) are cumulative poisons. These heavy metals are persistent, accumulate and are not metabolized in other intermediate compounds and do not easily break down in environment. These metals are accumulating in food chain through uptake at primary producer level and then through consumption at consumer level. However, subsequent metal contamination discovered by testing water necessitated mitigation programmes [8]. As trace elements, some heavy metals (e.g. copper, selenium, zinc) are essential to maintain the metabolism of the human body. However, at higher concentrations they can lead to poisoning [9]. The International Agency for Research on Cancer (IARC) classified lead as a

possible human carcinogen in 1987 [10]. Causes of arsenic toxicity include ingestion of arsenic (found in insect poisons), skin contact (e.g. some linseed oils) and even drinking water. Arsenic is typically considered a heavy metal and shares many toxic characteristics with the other heavy metals (eg, lead, mercury) [11]. Humans may come in contact with arsenic in contaminated dusts, fumes, or mists. They may eat food contaminated with arsenical pesticides or grown with arsenic-contaminated water or in arsenic-rich soil [12]. Initially arsenic enters into the human body through ingestion, inhalation, or skin absorption. After entering into the body it is distributed in a large number of organs including the lungs, liver, kidney and skin [13]. The body has need for approximately 70 friendly trace element heavy metals, but there are another 12 poisonous heavy metals, such as Lead, Mercury, Aluminum, Arsenic, Cadmium, Nickel, etc., that act as poisonous interference to the enzyme systems and metabolism of the body. No matter how many good health supplements or procedures one takes, heavy metal overload will be a detriment to the natural healing functions of the body. Some metals are naturally found in the body and are essential to human health. Iron, for example, prevents anemia, and zinc is a cofactor in over 100 enzyme reactions. Magnesium and copper are other familiar metals that, in minute amounts, are necessary for proper metabolism to occur. Toxic heavy metals have no function in the body and can be highly toxic. Heavy metals are taken into the body via inhalation, ingestion, and skin absorption. If heavy metals enter and accumulate in body tissue faster than the body's detoxification pathways can dispose of them, a gradual buildup of these toxins will occur. Metals are particularly toxic to the sensitive, rapidly developing systems of fetuses, infants, and young children. Some metals, such as lead and mercury, easily cross the placenta and damage the fetal brain. Childhood exposure to some metals can result in learning difficulties, memory impairment, damage to the nervous system, and behavioral problems such as aggressiveness and hyperactivity. At higher doses, heavy metals can cause irreversible brain damage. Children may receive higher doses of metals from food than adults, since they consume more food for their body weight than

adults [14]. Currently, millions of people living in and around Bangladesh are at risk for organ dysfunction and cancer from chronic arsenic poisoning from the water supply [15]. There are some heavy metals that are necessary to support life in very small doses, but in large amounts are toxic and can present a health hazard if they build up in the system. Zinc is important to the body, as is cobalt atoms found in vitamin B-12. Iron is another good heavy metal, and so is copper, manganese, chromium and selenium. On the other hand lead, mercury and cadmium are heavy metals that are very toxic to humans, and have no known benefit to the body. Lead, mercury, cadmium, and arsenic are the most common examples of toxic metal exposure [16]. Toxicity is of the utmost concern in dealing with hazardous substances. This includes both long term chronic effect from continual or periodic exposure to low levels of toxicants and acute effects from a single large exposure [17]. Cadmium and cadmium compounds are, compared to other heavy metals, relatively water soluble. The major route of exposure to cadmium for the non-smoking general population is via food; the contribution from other pathways to total uptake is small. A major part of cadmium in the human diet comes from agricultural products. The pathway of human exposure from agricultural crops is susceptible to increases in soil cadmium. Cadmium accumulates in the human body and especially in the kidneys. The accumulation of cadmium in the kidney (in the renal cortex) leads to dysfunction of the kidney with impaired reabsorption of proteins, glucose, and amino acids [18]. In the general non-smoking adult population, the major exposure pathway of lead is from food and water. Airborne lead may contribute significantly to occupational exposure and exposure of smokers. For infants and young children lead in dust and soil often constitutes a major exposure pathway and this exposure has been one of the main concerns as to the exposure of the general population. The intake of lead will be influenced by the age and behavioral characteristics of the child and the bioavailability of lead in the source material. In adult humans approximately 10% of the dietary lead is absorbed. However, in infants and young children as much as 50% of dietary lead is absorbed. Depending upon the type of lead compounds, particle

size, and solubility in body fluids, up to 50% of inhaled lead compounds may be absorbed. Current studies indicate that even minute levels of toxic elements have negative health consequences; however, these vary from person to person. Nutritional status, metabolic rate, the integrity of detoxification pathways (ability to detoxify toxic substances), and the mode and degree of heavy metal exposure all affect how an individual responds. Children and the elderly, whose immune systems are either underdeveloped or age-compromised, are more vulnerable to toxicity [19]. Although some investigators prefer to determine lead and cadmium etc in the liver of fish, an investigation was carried out in the edible tissue only as the dietary habit of the people of Bangladesh excludes liver of fishes. The levels of Pb vary between $0.29 \mu\text{g g}^{-1}$ in *Mystus vittatus* and $10.055 \mu\text{g g}^{-1}$, *Sicamugil cascasisa* (Kachki) and those of Cd, between $0.03 \mu\text{g g}^{-1}$, *Mastacembelus armatus* and $0.16 \mu\text{g g}^{-1}$, *Ompok pabda* (Pabda). These variations are likely to be due to the living nature and dietary habits of the different species of fish. Shorputi is the bottom living fish. *Tenualosa ilisha* (Ilish) is a fish of migrating nature. This fish is largely available in the river, the Meghna at the mouth of the Bay of Bengal. The levels of Pb and Cd in this fish are 1.64 and $0.10 \mu\text{g g}^{-1}$. In two medium size popular fish species, *Labeo rohita* (rui) and *Pangasius pangasius* (Pangas), Pb levels range between 1.25 and $3.09 \mu\text{g g}^{-1}$ and those of Cd, 0.09 and $0.13 \mu\text{g g}^{-1}$. Due to dietary difference between these fish species and the other fish samples is unlikely to be the source of higher metal [20]. Concern has been raised by Bangladeshi and international scientists about elevated levels of arsenic in Bengali food, particularly in rice grain. That was the first inclusive food market-basket survey from Bangladesh, which addresses the speciation and concentration of arsenic in rice, vegetables, pulses, and spices. Three hundred thirty aman and boro rice, 94 vegetables, and 50 pulse and spice samples were analyzed for total arsenic, using inductivity coupled plasma mass spectrometry (ICP-MS). The districts with the highest mean arsenic rice grain levels were all from southwestern Bangladesh: Faridpur (boro) $0.51 >$ Satkhira (boro) $0.38 >$ Satkhira (aman) $0.36 >$ Chuadanga (boro) $0.32 >$ Meherpur

(boro) $0.29 \mu\text{g As g}^{-1}$. The vast majority of food ingested arsenic in Bangladesh diets was found to be inorganic; with the predominant species detected in Bangladesh rice being arsenite (As^{III}) or arsenate (As^{V}) with dimethyl arsenic acid (DMA^{V}) being a minor component. Vegetables, pulses, and spices are less important to total arsenic intake than water and rice. Predicted inorganic arsenic intake from rice is modeled with the equivalent intake from drinking water for a typical Bangladesh diet. Daily consumption of rice with a total arsenic level of $0.08 \mu\text{g As g}^{-1}$ would be equivalent to a drinking water arsenic level of $10 \mu\text{g L}^{-1}$ [21]. Environment is defined as the totality of circumstances surrounding an organism or group of organisms especially, the combination of external physical conditions that affect and influence the growth, development and survival of organisms. It consists of the flora, fauna and the biotic, and includes the aquatic, terrestrial and atmospheric habitats.

The environment is considered in terms of the most tangible aspects like air, water and food, and the less tangible, though no less important, the communities we live in. A pollutant is any substance in the environment, which causes objectionable effects, impairing the welfare of the environment, reducing the quality of life and may eventually cause death. Such a substance has to be present in the environment beyond a set or tolerance limit, which could be either a desirable or acceptable limit. Hence, environmental pollution is the presence of a pollutant in the environment; air, water and soil, which may be poisonous or toxic and will cause harm to living things in the polluted environment [22]. Heavy metal may enter the body through inhalation of dust, direct ingestion of soil, and consumption of food plants grown in metal contaminated soil [23].

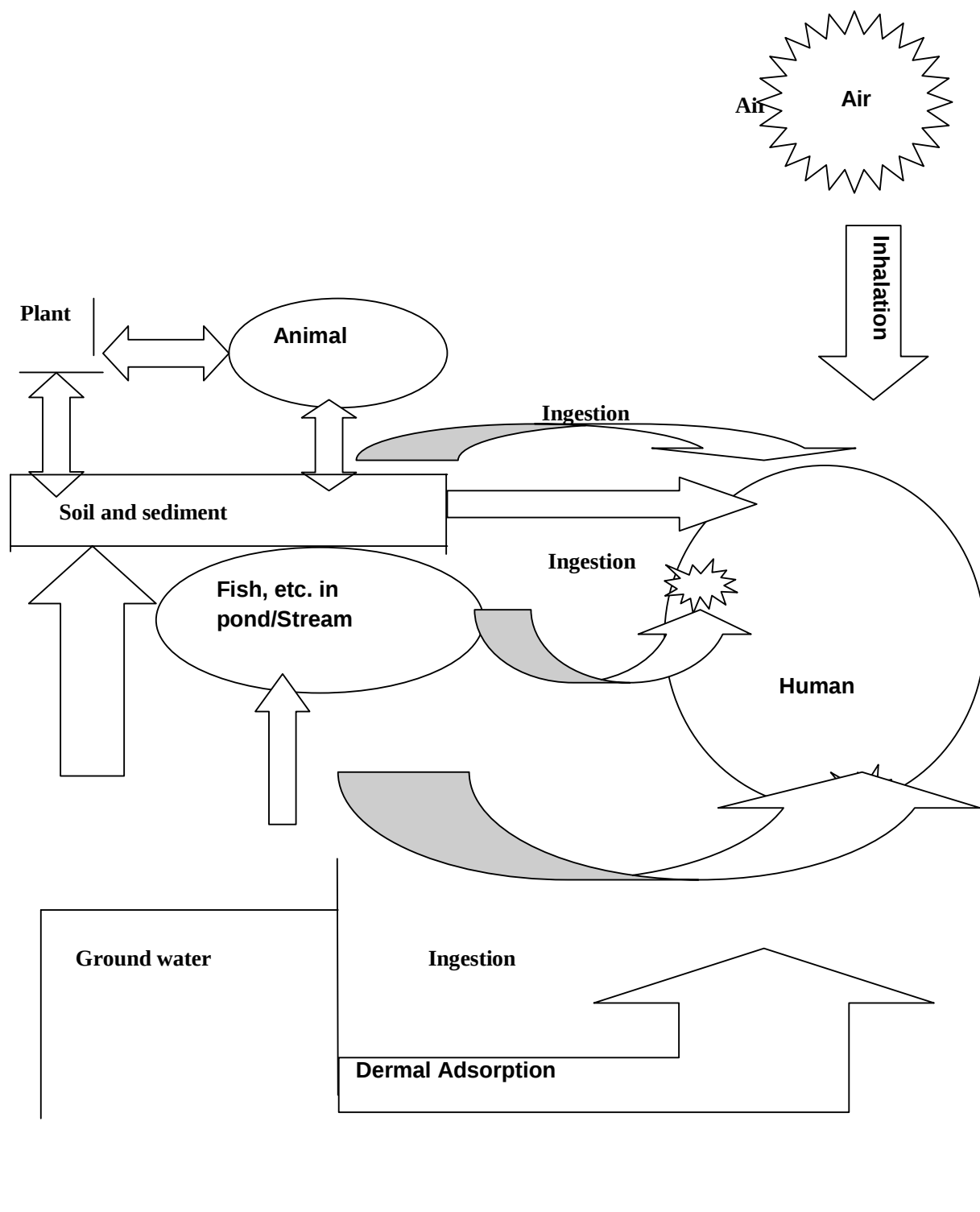


Fig 4.1 Exposure pathways of heavy metal to human beings in nature.

So it is important to determine the human health risk assessment in Rajshahi City Corporation. For this purpose a risk assessment of metal exposure to population subgroups living on, and growing food on, urban site was performed. Uptake of Arsenic, Lead, and Cadmium was modeled for a selection of commonly available food in Rajshahi City Corporation Population subgroups were including Children, Average Persons, and Senior.

4.1 EXPERIMENTAL PROCEDURES

4.1.1 Materials

The materials employed for the investigation is described in section 2.1.

4.1.2 Methods

The methods of fertilizers and soil preparation, and irrigation [20] were the same as mentioned in chapter 2. Water, vegetables and soil/dust sampling and analysis procedures [21-24] are discussed elaborately in the previous chapter, and hence not mentioned here.

4.1.3 Result and discussion

The environment is considered in terms of the most tangible aspects like air, water and food, and the less tangible, though no less important, the communities we live in. A pollutant is any substance in the environment, which causes objectionable effects, impairing the welfare of the environment, reducing the quality of life and may eventually cause death. Such a substance has to be present in the environment beyond a set or tolerance limit, which could be either a desirable or acceptable limit. Hence, environmental pollution is the presence of a pollutant in the environment; air, water and soil, which may be poisonous or toxic and will cause harm to living things in the polluted environment [22]. Trace elements are very harmful because of their no

biodegradable nature, long biological half-lives, and their potential to accumulate in different body parts. High concentrations of trace elements (Cu, Cd, and Pb) in vegetables and fruits were related to prevalence of upper gastrointestinal cancer high [5]. Cadmium may promote skeletal demineralization and increase bone fragility and fracture risk.

In Fudkipara and Kumarpara moholla of Rajshahi City Corporation, all the people are educated and well established. Few houses were very old and others are new or built before ten years ago. The house owner's someone employer, someone businessman and others are unemployed. Maximum house have 5/6 member's. All house member use tube-well water for drink and tap water for cook their food. They collect their daily foods from nearer market. Each house owner's monthly income is minimum 13,000 and maximum 25,000Tk. Some house has tube-well inside in and others are collecting their water from far away tube-well. Some house has children of 6-7 years old. Some people in each house are between 35 to 40 years old. Two houses have $\frac{1}{2}$ member are between 55 to 60 years old. The entire houses are made with bricks, sand and cement. All the houses have colored by limbo. The roads of these places are always busy with many types of vehicles like rickshaws, auto-rickshaws, buses. Besides these moholla there are some shops. So always these places are gathered by various kinds of people. There are no ponds or canals near these places only the river Padma beside Fudkipara and Kumarpara. The houses are very nearest to each other. All house member use gas/kerosene to cook their food. All house owners have capacity to buy meat/fish in a week. The samples were collected from their rice, fish vegetable, water and dust. Dusts were collected from the floor of each house. The study area located roughly between 24° 19' N to 24° 21' N and 88° 36' E. The studied households are marked in Table 4.1.

Table 4.1 Profile of studied household owners.

Ward No	Village	Owner's Name	GPS	Sample Code
12	Futki Para	Polash	24°19'60"N 88°43'10"E	R-BF12-01
		Manik	24°19'60"N 88°43'10"E	V-BF12-02
		Robi	24°19'60"N 88°43'10"E	F-BF12-03
		Somvunath	24°19'60"N 88°43'10"E	W-BF12-04
		Protim	24°19'60"N 88°43'10"E	D-BF12-05
	Kumarpara	Kousik Ghosh	24°22'04.26"N 88°36'05.99"E	R-BR12-01
		Sunil	24°22'04.63"N 88°36'06.26"E	V-BR12-02
		Hamid	24°22'04.14"N 88°36'06.27"E	F-BR12-03
		Ronjit	24°22'02.39"N 88°36'02.95"E	W-BR12-04
		Soumen	24°22'01.91"N 88°36'03.60"E	D-BR12-05

Toxic heavy metals easily enter into food commodities from various sources of polluted ecosystem. The aim of this study was to monitor the heavy metals in fruits and vegetables. The results obtained so far are shown in Table 4.3 - 4.29.

For estimating the exposure of arsenic, cadmium and lead for consumers of studied sample, we first calculated Average Daily Dose (ADD) using equation (1.1) as suggested by U.S.EPA Guidelines for Exposure Assessment:

$$ADD = \frac{\bar{C}_{veg} \cdot IR \cdot ED}{BW \cdot AT} \dots\dots\dots 1.1$$

Where $\bar{C}_{veg}$ the average concentration of arsenic in vegetables, IR is ingestion rate for vegetables and is nearly constant, ED is exposure duration and is the sum of the exposure duration for all events, BW is the body weight and AT is averaging time (the time period over which the dose is averaged, converted to days).

Similar ADD would be obtained from Rice, Fish, Water and dust. Dust Inhalation would be calculated using the equation:

$$ADD = \left(\frac{M_i R_{in}}{B} \right) F_{ex} \dots\dots\dots 1.2$$

Where M_i is the inhaled metal concentration (μgm^{-3}), R_{in} is the inhalation rate (m^3d^{-1}), B is the body weight of the exposed subject (kg) and F_{ex} is the fraction exposure (defined as the ratio of the exposure duration to an averaging time i.e $F_{ex} = ED/AT$)

For soil ingestion the average daily dose (ADD) would be calculated using equation 1.3.

$$ADD = \left(\frac{M_{soil} R_{in}}{B} \right) F_{ex} \dots\dots\dots 1.3$$

where R_{in} is the soil ingestion rate (kgd^{-1}) that were delivered from literature studies, M_{soil} is the metal concentration in soil (mgkg^{-1}). Values for B and F_{ex} were the same as in equation 1.2.

For three population subgroups the values for the parameters used in equation 1.1, 1.2 and 1.3 were taken from standard exposure factors [34, 35] and Bangladesh Bureau of statistics [33, 40] presented in table 4.2 (activity budget).

$$ADD_{As} = ADD_{Rice} + ADD_{Veg} + ADD_{Fish/Meat} + ADD_{Dust} + ADD_{Water} \dots\dots 1.3$$

$$HQ_{As} = \frac{ADD_{As}}{RfD} \dots\dots\dots 1.4$$

Similar, HQ_{Pb} and HQ_{Cd} would be calculated.

Finally, HI would be estimated using

$$HI = HQ_{As} + HQ_{Pb} + HQ_{Cd} \dots\dots\dots 1.5$$

Risk may be characterized using a hazard quotient (HQ). This is the ratio of the average daily dose (ADD; milligrams per kilogram per day) of a chemical to a reference dose (RfD, milligrams per kilogram per day) defined as the maximum tolerable daily intake of a specific metal that does not result in any deleterious health effects.

If $HQ > 1.00$, then the ADD of a particular metal exceeds the RfD, indicating that there is a potential risk associated with that metal. In the United Kingdom, DEFRA and the Environment Agency have published

reference doses for Cd and Ni (DEFRA and Environment Agency 2002d, 2002e). Reference doses for Cu, Pb, and Zn were derived using the framework recommended for U.K. risk assessments. Index doses (to assess cancer risk) for Cd and Ni were not employed because they are set at a level similar to background concentrations and because they were derived for compounds of Cd and Ni, which are rarely present in a no occupational environment and are difficult to assess from measures of total metal concentration. The RfDs used in this study are given in Table no.1. The RfDs may be derived from a no observed adverse effect level (NOAEL) or a lowest observed adverse effects level (LOAEL) which could lead to inconsistencies in the final RfD estimation. Essential elements (Cu and Zn in this study) have greater RfD values because of their lower toxicity.

Table 4.2. Parameters involved in the risk assessment model (activity budget).

Factors	Unit	Population sub-group			References
		Children (0-6 yr)	Average Person	Senior	
Age	yr	6	39.5	60.3	
Body weight (BW)	kg	18.6	80.8	82.6	
Rice ingestion rate	g (ww.) d ⁻¹	100	344	300	
Vegetable ingestion rate	g (ww.) d ⁻¹	60	155	120	
Fish ingestion rate	g (ww.) d ⁻¹	40	60	60	
Water ingestion rate	L d ⁻¹	1.4	2.5	2	
Soil and dust ingestion rate	mg (ww.) d ⁻¹	100	50	50	
Dust inhalation rate	m ³ d ⁻¹	10.1	16	15.7	
Exposure frequency (EF)	d yr ⁻¹	350	350	350	
Year	d	365.25	365.25	365.25	
Averaging time (AT)	yr	6	30	50	
Exposure duration (ED)	yr	6	30	50	
RBAF [‡] of As	(unit less)	0.4			
RBAF of Pb	(unit less)	0.6			
RBAF of Cd	(unit less)	0.6			

As reference dose (^{As} RfD)	mg kg ⁻¹ d ⁻¹	0.0003	0.0003	0.0003	
Pb reference dose (^{Pb} RfD)	mg kg ⁻¹ d ⁻¹	0.0005	0.05	0.001	
Cd reference dose (^{Cd} RfD)	mg kg ⁻¹ d ⁻¹	0.0005	0.0005	0.0005	

[‡] RBAF stands for relative bio-availability factor.

4.2.1 Concentration of Arsenic, Cadmium and Lead in Dietary Products, Drinking Water and Dust:

The concentrations of arsenic, cadmium and lead in the studied dietary products, drinking water and dust are enlist in table 4.3, 4.4 and 4.5. Each data corresponds to mean of triplicate measurements.

Table 4.3 Concentrations of arsenic in dietary products, drinking water and dust.

Village	Owner's name	Concentration of As ($\mu\text{g g}^{-1}$) [†]					
		Rice	Vegetable	Fish	Dust	Air	Water
Futki Para	Polash	0.056(0.062)	0.007(0.109)	0.073(0.433)	1.097(1.103)	9.23×10^{-06}	0.0023
	Manik	0.064(0.071)	0.028(0.121)	0.089(0.434)	1.004(1.032)	8.44×10^{-06}	0.0029
	Robi	0.047(0.051)	0.034(0.137)	0.125(0.552)	1.019(1.022)	8.57×10^{-06}	0.0019
	Somvunath	0.051(0.058)	0.0329(0.165)	0.089(0.436)	1.039(1.141)	8.74×10^{-06}	0.0037
	Protim	0.045(0.050)	0.037(0.203)	0.138(0.491)	1.146(1.151)	9.64×10^{-06}	0.0022
Kumar Para	Kousik Ghosh	0.090(0.098)	0.009(0.122)	0.141(0.590)	0.703(0.722)	5.91×10^{-06}	0.0032
	Sunil	0.066(0.073)	0.009(0.119)	0.227(0.862)	0.702(0.702)	5.90×10^{-06}	0.0027
	Hamid	0.075(0.079)	0.021(0.084)	0.091(0.361)	0.859(0.862)	7.22×10^{-06}	0.0028
	Ronjit	0.064(0.069)	0.033(0.154)	0.527(2.966)	1.040(1.054)	8.75×10^{-06}	0.0025
	Soumen	0.059(0.067)	0.021(0.220)	0.088(0.282)	1.029(1.036)	8.65×10^{-06}	0.0023

[†] the values in parentheses represent dried mass. Estimated based on the mentioned model, concentrations of air and water are expressed in $\mu\text{g m}^{-3}$ and $\mu\text{g L}^{-1}$ respectively.

Table 4.4. Concentrations of Cadmium in dietary products, drinking water and dust.

Village	Owner's name	Concentration of Cd($\mu\text{g g}^{-1}$) [†]					
		Rice	Vegetable	Fish	Dust	Air	Water
Futki Para	Polash	0.00039(0.0004)	0.0004(0.0056)	0.0012(0.0072)	0.0004(0.0004)	3.36×10^{-07}	0.0065
	Manik	0.0003(0.0004)	0.0030(0.0131)	0.0034(0.0168)	0.0003(0.0003)	3.83×10^{-07}	0.0052
	Robi	0.0003(0.0004)	0.0033(0.0132)	0.0039(0.0171)	0.0004(0.0004)	1.13×10^{-07}	0.0054
	Somvunath	0.0004(0.0005)	0.0024(0.0126)	0.0027(0.0133)	0.0004(0.0004)	$5. \times 10^{-08}$	0.0058
	Protim	0.0003(0.0004)	0.0016(0.0088)	0.0038(0.0135)	0.0005(0.0005)	1.12×10^{-07}	0.0054
Kumar Para	Kousik Ghosh	0.0103(0.0112)	0.0012(0.0163)	0.0033(0.0138)	0.0009(0.0009)	7.57×10^{-09}	0.0042
	Sunil	0.0100(0.0111)	0.0006(0.008)	0.0029(0.0109)	0.0006(0.0006)	5.05×10^{-09}	0.0048
	Hamid	0.0114(0.0121)	0.0021(0.0084)	0.0065(0.0257)	0.0004(0.0004)	3.36×10^{-09}	0.0049
	Ronjit	0.0170(0.0184)	0.0022(0.0102)	0.0017(0.0096)	0.0004(0.0004)	3.36×10^{-09}	0.0061
	Soumen	0.0072(0.0082)	0.0012(0.0123)	0.0019(0.0061)	0.0004(0.0004)	3.36×10^{-09}	0.0047

[†] the values in parentheses represent dried mass. Estimated based on the mentioned model, concentrations of air and water are expressed in $\mu\text{g m}^{-3}$ and $\mu\text{g L}^{-1}$ respectively.

Table 4.5. Concentrations of lead in dietary products, drinking water and dust.

Village	Owner's name	Concentration of Pb ($\mu\text{g g}^{-1}$) [†]					
		Rice	Vegetables	Fish	Dust	Air	Water
Futki Para	Polash	0.063(0.070)	0.003(0.043)	0.009(0.056)	0.040(0.040)	3.36×10^{-09}	0.001
	Manik	0.024(0.027)	0.012(0.051)	0.016(0.077)	0.046(0.047)	2.52×10^{-09}	0.002
	Robi	0.018(0.020)	0.013(0.051)	0.010(0.045)	0.013(0.013)	3.36×10^{-09}	0.001
	Somvunath	0.030(0.034)	0.009(0.047)	0.010(0.047)	0.007(0.008)	3.36×10^{-09}	0.001
	Protim	0.036(0.040)	0.009(0.047)	0.018(0.064)	0.013(0.013)	4.21×10^{-09}	0.002
Kumar Para	Kousik Ghosh	0.051(0.0552)	0.003(0.043)	0.010(0.043)	0.029(0.030)	1.76×10^{-06}	0.001
	Sunil	0.046(0.051)	0.003(0.034)	0.013(0.051)	0.025(0.026)	1.50×10^{-06}	0.001
	Hamid	0.056(0.0595)	0.012(0.047)	0.019(0.074)	0.013(0.013)	7.98×10^{-07}	0.002
	Ronjit	0.051(0.0552)	0.010(0.047)	0.008(0.047)	0.013(0.013)	7.92×10^{-07}	0.001
	Soumen	0.049(0.0552)	0.005(0.049)	0.016(0.051)	0.020(0.020)	1.19×10^{-06}	0.002

[†] the values in parentheses represent dried mass. Estimated based on the mentioned model, concentrations of air and water are expressed in $\mu\text{g m}^{-3}$ and $\mu\text{g L}^{-1}$ respectively.

In the studied samples arsenic concentrations in rice, vegetables, fish, drinking water and dust ranged from 0.045-0.075, 0.007-0.037, 0.073-0.527, 0.0023-0.0037 and 0.702-1.146 $\mu\text{g g}^{-1}$ respectively on fresh weight basis. Obviously arsenic concentration was highest in dust (1.146 $\mu\text{g g}^{-1}$, ww) and lowest in drinking water (0.002 $\mu\text{g L}^{-1}$). Cadmium concentrations in rice, vegetable, fish, drinking water and dust ranged from 0.0003-0.0170, 0.0004-0.0033, 0.0012-0.0065, 0.0042-0.0065 and 0.0003-0.0009 $\mu\text{g g}^{-1}$ (ww) respectively. It is clear that cadmium concentration was highest in rice (0.0170 $\mu\text{g g}^{-1}$, ww) and lowest in dust (0.0003 $\mu\text{g g}^{-1}$, ww) respectively. It was also found that lead concentration in rice, vegetables, fish, drinking water and dust ranged from 0.018-0.063, 0.003-0.013, 0.008-0.019, 0.001-0.002 and 0.007-0.046 $\mu\text{g g}^{-1}$ respectively. This reflects that lead concentration was highest in rice (0.063 $\mu\text{g g}^{-1}$, ww) and lowest in drinking water (0.001 $\mu\text{g L}^{-1}$).

In the investigation, the concentrations of heavy metals in the studied samples follow the following order: Dust > Fish > Rice > Vegetable > Drinking water for As; Rice > Fish > Vegetable > Dust > Drinking water for Cd; Dust > Fish > Rice > vegetable < Drinking water for Pb. Generally the concentrations of the concerned metals in the studied samples follow the order: Dust > Rice > Fish > Vegetable > Drinking water.

Williams et al. [24] found that the mean arsenic level of Bangladesh rice was 0.13 (range 0.03-0.30) mg/kg which is in good agreement with our findings. A survey of the levels of cadmium, lead and arsenic in different types of rice available on the Swedish retail market was carried out in 2001-03 [25]. The types of rice included long and short grain, brown, white, and parboiled white rice. The mean levels found were as follows: total As: 0.20 mg kg⁻¹, (inorganic As: 0.11 mg kg⁻¹); Cd: 0.024 mg kg⁻¹; and Pb: 0.004 mg kg⁻¹. Our studied values for As in rice ranged from 0.045 to 0.276 $\mu\text{g g}^{-1}$, values for Cd in rice ranged from 0.0003 to 0.0143 $\mu\text{g g}^{-1}$ and values for Pb in rice ranged from 0.018-0.063 $\mu\text{g g}^{-1}$. Afshar et al [26] determined Cd content in Amol rice (a variety of Iranian rice) found that the mean Cd concentration in Amol rice was 0.09 $\mu\text{g g}^{-1}$.

But in highly contaminated areas the levels of As, Pb and Cd are considerably higher. For example, in Samota village, Jashor, Bangladesh (a highly arsenic contaminated village) the arsenic concentration in snake gourd vegetables was 0.401 (range 0.306-0.489) $\mu\text{g g}^{-1}$ [27]. In highly contaminated areas from Copsa Mica, Zlatna and Baia Mare of Romania [28] the cadmium concentration ranged 0.10-0.27 $\mu\text{g g}^{-1}$ and lead concentration ranged 1.0-5.50 $\mu\text{g g}^{-1}$ (fresh matters) in root vegetables grown.

Air lead levels were recorded as being very high in Dhaka, Bangladesh between 1997-2000 [6]. In a cross-sectional study of 49 children from three areas in Dhaka, the mean $\pm$ SD of blood lead levels was $17.6 \pm 4.9 \mu\text{g/dL}$ [7].

4.2.2 Statistical analysis of arsenic, cadmium and lead in

Environmental samples: Statistical analysis show that average value of arithmetic and geometric mean of the studied samples are 0.278 $\mu\text{g g}^{-1}$ and 0.261 $\mu\text{g g}^{-1}$ for As, 0.003 $\mu\text{g g}^{-1}$ and $2.06 \times 10^{-03} \mu\text{g g}^{-1}$ Cd, 0.022 $\mu\text{g g}^{-1}$ and $1.49 \times 10^{-02} \mu\text{g g}^{-1}$ for Pb. Standard deviation and variance for As, Cd and Pb are also include in table 4.6, 4.7 and 4.8 respectively.

Table 4.6. Statistical analyses of arsenic data sets[§] of the studied samples

Sample	Concentration mg/kg					
	Range (Mini-Maxi)	Mean $\pm$ SE	Geometric	Median	Standard Deviation	Variance
Rice	0.045-0.090 (0.050-0.098)	0.062 $\pm$ 0.004 (0.068 $\pm$ 0.004)	0.060 (0.067)	0.062 (0.068)	0.014 (0.014)	0.000 (0.000)
Vegetable	0.007-0.037 (0.084-0.220)	0.023 $\pm$ 0.004 (0.143 $\pm$ 0.013)	0.020 (0.138)	0.025 (0.130)	0.012 (0.043)	0.000 (0.002)
Fish	0.073-0.527 (0.282-2.966)	0.159 $\pm$ 0.043 (0.741 $\pm$ 0.252)	0.130 (0.567)	0.108 (0.464)	0.137 (0.797)	0.019 (0.636)
Drinking water	0.002-0.004	0.003 $\pm$ 0.0002	0.003	0.003	0.001	0.000
Dust	0.702-1.146 (0.702-1.151)	0.964 $\pm$ 0.049 (0.983 $\pm$ 0.052)	0.951 (0.969)	1.024 (1.034)	0.156 (0.164)	0.024 (0.027)

[§] the values in parentheses represent dry weight

Table 4.7. Statistical analyses of cadmium data sets[§] of the studied samples

Sample	Concentration mg/kg					
	Range (Mini- Maxi)	Mean±SE	Geometric Mean	Median	Standard Deviation	Variance
Rice	0.0003-0.017 (0.0004-0.018)	0.006±0.002 (0.006±0.002)	0.002 (0.002)	0.004 (0.004)	0.006 (0.007)	0.000 (0.000)
Vegetable	0.0004-0.003 (0.006-0.016)	0.002±0.0003 (0.011±0.001)	0.002 (0.010)	0.002 (0.011)	0.001 (0.003)	0.000 (0.000)
Fish	0.001-0.007 (0.006-0.026)	0.003±0.0005 (0.013±0.002)	0.003 (0.012)	0.003 (0.013)	0.001 (0.006)	0.000 (0.000)
Drinking water	0.004-0.007	0.005±0.0002	0.005	0.005	0.001	0.000 (0.000)
Dust	0.0003-0.0009 (0.00030.0009)	0.0005±0.0005 (0.0005±0.0005)	0.0004 (0.0004)	0.0004 (0.0004)	0.0002 (0.0002)	0.000 (0.000)

[§] the values in parentheses represent dry weight

Table 4.8. Statistical analyses of lead data sets[§] of the studied samples

Sample	Concentration mg/kg					
	Range (Mini- Maxi)	Mean±SE	Geometric Mean	Meadian	Standard Deviation	Variance
Rice	0.018-0.063 (0.020- 0.070)	0.042±0.005 (0.047±0.005)	0.040 (0.044)	0.048 (0.053)	0.015 (0.016)	0.000 (0.000)
Vegetable	0.003-0.013 (0.034- 0.051)	0.008±0.001 (0.046±0.002)	0.007 (0.046)	0.009 (0.047)	0.004 (0.005)	0.000 (0.000)
Fish	0.008-0.019 (0.0430.077)	0.013±0.001 (0.056±0.004)	0.012 (0.054)	0.012 (0.051)	0.004 (0.012)	0.000 (0.000)
Drinking water	0.001-0.002	0.001±0.0002	0.001	0.001	0.001	0.000 (0.000)
Dust	0.007-0.046 (0.0080.047)	0.022±0.004 (0.022±0.004)	0.019 (0.019)	0.017 (0.017)	0.013 (0.013)	0.000 (0.000)

[§] the values in parentheses represent dry weight

The maximum permissible limits (MPL) of arsenic, cadmium and lead concentrations in drinking water were suggested by World Health Organization, WHO [8] as 0.01, 0.003 and 0.01 mgL⁻¹ respectively. In Bangladesh MPL of As, Pb and Cd concentration in drinking water was suggested by Department of Environment of Govt. of Bangladesh as 0.05, 0.005 and 0.05 mgL⁻¹ respectively. Obviously the MPLs of Bangladesh is roughly 5 times higher than those of international standard.

The Joint FAO/WHO Expert Committee on Food Additives (JCEFA) has proposed a maximum level of $0.2 \mu\text{g g}^{-1}$ Cd in rice but the community warned that people who eat a of rice from regions containing the higher levels of cadmium could be significantly exposed. And also JCEFA has set the Provisional Tolerable Weekly Intake (PTWI) for the cadmium and lead equal to 7 and $25 \mu\text{g g}^{-1}$ of body weight, respectively [42].

Permissible limits of Copper, Lead, Cadmium and Chromium in vegetables as recommended by WHO/FAO are 40, 0.3, 0.2, 2.3 mg kg⁻¹ respectively [43].

Levels of cadmium and lead Concentration in fishes are 0.030 mg kg^{-1} and 0.050 mg/kg as reported in WHO [44]

4.2.3 Individual Contribution from As, Cd and Pb Exposure

For estimating the exposure of arsenic, lead and cadmium for consumers of the studied samples, we first calculated Average Daily Dose (ADD) using equation (1.1) as suggested by U.S.EPA in Guidelines for Exposure Assessment [30].

$$ADD = \frac{\bar{C}_{veg} \cdot IR \cdot ED}{BW \cdot AT} \dots\dots\dots 1.1$$

Where $\bar{C}$ the average concentration of As, Cd or Pb in the sample, IR is ingestion rate, ED is the exposure duration, BW is the body weight and AT is averaging time. The details of ADD calculation are described in Method section. It is to be noted that some data were adapted from the standard exposure factors [34-35] and data of Bangladesh survey and standards [25-27].

The Average Daily Dose (ADD) were estimated for three population subgroups: '*Children*', '*Average Person*' and '*Senior*'. The children of age 0-6 years are referred to as '*High Exposed Individuals (HEIs)*'. The average persons and seniors are considered as of 39.5 and 60.3 years. The details are represented in table 4.2. The ADD for arsenic, cadmium and lead for the three population subgroups are shown in the following nine Tables.

Table 4.9. Average daily dose (ADD) of arsenic for children population sub-group.

Village	Owner's name	Average Daily Dose (ADD) of As / mg kg ⁻¹ d ⁻¹						
		^{As} ADD _{rice}	^{As} ADD _{veg}	^{As} ADD _{fish}	^{As} ADD _{water}	^{As} ADD _{dust}	^{As} ADD _{Air}	^{As} ADD _{total}
Fudki Para	Polash	1.15×10 ⁻⁰⁴	8.66×10 ⁻⁰⁶	6.02×10 ⁻⁰⁵	6.64×10 ⁻⁰⁵	2.26×10 ⁻⁰⁶	1.92×10 ⁻⁰⁶	2.54×10 ⁻⁰⁴
	Manik	1.32×10 ⁻⁰⁴	3.46×10 ⁻⁰⁵	7.34×10 ⁻⁰⁵	8.37×10 ⁻⁰⁵	2.07×10 ⁻⁰⁶	1.76×10 ⁻⁰⁶	3.28×10 ⁻⁰⁴
	Robi	9.69×10 ⁻⁰⁵	4.20×10 ⁻⁰⁵	1.03×10 ⁻⁰⁴	5.48×10 ⁻⁰⁵	2.10×10 ⁻⁰⁶	1.78×10 ⁻⁰⁶	3.01×10 ⁻⁰⁴
	Somvunath	1.05×10 ⁻⁰⁴	3.96×10 ⁻⁰⁵	7.34×10 ⁻⁰⁵	1.07×10 ⁻⁰⁴	2.14×10 ⁻⁰⁶	1.82×10 ⁻⁰⁶	3.29×10 ⁻⁰⁴
	Protim	9.27×10 ⁻⁰⁵	4.57×10 ⁻⁰⁵	1.14×10 ⁻⁰⁴	6.35×10 ⁻⁰⁵	2.36×10 ⁻⁰⁶	2.01×10 ⁻⁰⁶	3.20×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.85×10 ⁻⁰⁴	1.11×10 ⁻⁰⁵	1.16×10 ⁻⁰⁴	3.08×10 ⁻⁰¹	1.50×10 ⁻⁰⁶	1.23×10 ⁻⁰⁶	3.08×10 ⁻⁰¹
	Sunil	1.36×10 ⁻⁰⁴	1.11×10 ⁻⁰⁵	1.87×10 ⁻⁰⁴	7.79×10 ⁻⁰⁵	1.50×10 ⁻⁰⁶	1.23×10 ⁻⁰⁶	4.15×10 ⁻⁰⁴
	Hamid	1.55×10 ⁻⁰⁴	2.61×10 ⁻⁰⁵	7.50×10 ⁻⁰⁵	8.08×10 ⁻⁰⁵	1.80×10 ⁻⁰⁶	1.50×10 ⁻⁰⁶	3.39×10 ⁻⁰⁴
	Ronjit	1.32×10 ⁻⁰⁴	4.08×10 ⁻⁰⁵	4.34×10 ⁻⁰⁴	7.21×10 ⁻⁰⁵	2.10×10 ⁻⁰⁶	1.82×10 ⁻⁰⁶	6.83×10 ⁻⁰⁴
	Soumen	1.22×10 ⁻⁰⁴	2.61×10 ⁻⁰⁵	7.26×10 ⁻⁰⁵	6.64×10 ⁻⁰⁵	2.10×10 ⁻⁰⁶	1.80×10 ⁻⁰⁶	2.91×10 ⁻⁰⁴

Table 4.10. Average daily dose (ADD) of Cadmium for children population sub-group

Village	Owner's name	Average Daily Dose (ADD) of Cd/ mg kg ⁻¹ d ⁻¹						
		CdADD _{ite}	CdADD _{veg}	CdADD _{fish}	CdADD _{water}	CdADD _{dust}	CdADD _{Air}	CdADD _{total}
Fudki Para	Polash	1.24×10 ⁻⁰⁶	7.42×10 ⁻⁰⁷	1.48×10 ⁻⁰⁶	2.82×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.07×10 ⁻⁰⁴	3.92×10 ⁻⁰⁴
	Manik	9.27×10 ⁻⁰⁷	5.56×10 ⁻⁰⁶	4.20×10 ⁻⁰⁶	2.23×10 ⁻⁰⁴	8.14×10 ⁻¹⁰	8.05×10 ⁻¹⁵	2.34×10 ⁻⁰⁴
	Robi	9.27×10 ⁻⁰⁷	6.12×10 ⁻⁰⁶	4.82×10 ⁻⁰⁶	2.33×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.07×10 ⁻¹⁴	2.45×10 ⁻⁰⁴
	Somvunath	1.24×10 ⁻⁰⁶	4.45×10 ⁻⁰⁶	3.34×10 ⁻⁰⁶	2.49×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.07×10 ⁻¹⁴	2.58×10 ⁻⁰⁴
	Protim	9.27×10 ⁻⁰⁷	2.97×10 ⁻⁰⁶	4.70×10 ⁻⁰⁶	2.35×10 ⁻⁰⁴	1.36×10 ⁻⁰⁹	1.34×10 ⁻¹⁴	2.44×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	3.18×10 ⁻⁰⁵	2.23×10 ⁻⁰⁶	4.08×10 ⁻⁰⁶	1.82×10 ⁻⁰⁴	2.44×10 ⁻⁰⁹	2.44×10 ⁻⁰⁹	2.20×10 ⁻⁰⁴
	Sunil	3.09×10 ⁻⁰⁵	1.11×10 ⁻⁰⁶	3.59×10 ⁻⁰⁶	2.06×10 ⁻⁰⁴	1.63×10 ⁻⁰⁹	1.63×10 ⁻⁰⁹	2.42×10 ⁻⁰⁴
	Hamid	3.52×10 ⁻⁰⁵	3.89×10 ⁻⁰⁶	8.04×10 ⁻⁰⁶	2.13×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.09×10 ⁻⁰⁹	2.60×10 ⁻⁰⁴
	Ronjit	5.25×10 ⁻⁰⁵	4.08×10 ⁻⁰⁶	2.10×10 ⁻⁰⁶	2.64×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.09×10 ⁻⁰⁹	3.32×10 ⁻⁰⁴
	Soumen	2.23×10 ⁻⁰⁵	2.23×10 ⁻⁰⁶	2.35×10 ⁻⁰⁶	2.02×10 ⁻⁰⁴	1.09×10 ⁻⁰⁹	1.09×10 ⁻⁰⁹	2.29×10 ⁻⁰⁴

Table 4.11. Average daily dose (ADD) of Lead for children population sub-group

Village	Owner's name	Average Daily Dose (ADD) of Pb / mg kg ⁻¹ d ⁻¹						
		^{Pb} ADD _{ice}	^{Pb} ADD _{veg}	^{Pb} ADD _{fish}	^{Pb} ADD _{water}	^{Pb} ADD _{dust}	^{Pb} ADD _{Air}	^{Pb} ADD _{total}
Fudki Para	Polash	1.95×10 ⁻⁰⁴	5.01×10 ⁻⁰⁶	1.15×10 ⁻⁰⁵	4.02×10 ⁻⁰⁵	1.05×10 ⁻⁰⁷	1.05×10 ⁻⁰⁷	2.52×10 ⁻⁰⁴
	Manik	7.48×10 ⁻⁰⁵	2.19×10 ⁻⁰⁵	1.94×10 ⁻⁰⁵	6.79×10 ⁻⁰⁵	1.19×10 ⁻⁰⁷	1.19×10 ⁻⁰⁷	1.84×10 ⁻⁰⁴
	Robi	5.66×10 ⁻⁰⁵	2.34×10 ⁻⁰⁵	1.25×10 ⁻⁰⁵	4.37×10 ⁻⁰⁵	3.52×10 ⁻⁰⁸	3.52×10 ⁻⁰⁸	1.36×10 ⁻⁰⁴
	Somvunath	9.18×10 ⁻⁰⁵	1.69×10 ⁻⁰⁵	1.17×10 ⁻⁰⁵	4.11×10 ⁻⁰⁵	1.71×10 ⁻⁰⁸	1.71×10 ⁻⁰⁸	1.62×10 ⁻⁰⁴
	Protim	1.12×10 ⁻⁰⁴	1.58×10 ⁻⁰⁵	2.21×10 ⁻⁰⁵	7.75×10 ⁻⁰⁵	3.49×10 ⁻⁰⁸	3.49×10 ⁻⁰⁸	2.27×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.56×10 ⁻⁰⁴	5.75×10 ⁻⁰⁶	1.26×10 ⁻⁰⁵	4.41×10 ⁻⁰⁵	7.72×10 ⁻⁰⁸	1.00×10 ⁻⁰⁷	2.19×10 ⁻⁰⁴
	Sunil	1.43×10 ⁻⁰⁴	4.82×10 ⁻⁰⁶	1.66×10 ⁻⁰⁵	5.80×10 ⁻⁰⁵	6.56×10 ⁻⁰⁸	5.00×10 ⁻⁰⁸	2.23×10 ⁻⁰⁴
	Hamid	1.74×10 ⁻⁰⁴	2.17×10 ⁻⁰⁵	2.31×10 ⁻⁰⁵	8.09×10 ⁻⁰⁵	3.49×10 ⁻⁰⁸	5.00×10 ⁻⁰⁸	3.00×10 ⁻⁰⁴
	Ronjit	1.58×10 ⁻⁰⁴	1.85×10 ⁻⁰⁵	1.03×10 ⁻⁰⁵	3.59×10 ⁻⁰⁵	3.47×10 ⁻⁰⁸	5.00×10 ⁻⁰⁸	2.23×10 ⁻⁰⁴
	Soumen	1.50×10 ⁻⁰⁴	8.72×10 ⁻⁰⁶	1.97×10 ⁻⁰⁵	6.88×10 ⁻⁰⁵	5.23×10 ⁻⁰⁸	5.00×10 ⁻⁰⁸	2.47×10 ⁻⁰⁴

Table 4.12. Average daily dose (ADD) of arsenic for Average population sub-group.

Village	Owner's name	Average Daily Dose (ADD) of As / mg kg ⁻¹ d ⁻¹						
		^{As} ADD _{ice}	^{As} ADD _{veg}	^{As} ADD _{fish}	^{As} ADD _{water}	^{As} ADD _{dust}	^{As} ADD _{Air}	^{As} ADD _{total}
Fudki Para	Polash	9.14×10 ⁻⁰⁵	5.15×10 ⁻⁰⁶	2.08×10 ⁻⁰⁵	2.73×10 ⁻⁰⁵	7.00×10 ⁻⁰⁷	7.00×10 ⁻⁰⁷	1.46×10 ⁻⁰⁴
	Manik	1.04×10 ⁻⁰⁴	2.06×10 ⁻⁰⁵	2.53×10 ⁻⁰⁵	3.44×10 ⁻⁰⁵	6.41×10 ⁻⁰⁷	6.41×10 ⁻⁰⁷	1.86×10 ⁻⁰⁴
	Robi	7.67×10 ⁻⁰⁵	2.50×10 ⁻⁰⁵	3.56×10 ⁻⁰⁵	2.25×10 ⁻⁰⁵	6.50×10 ⁻⁰⁷	6.50×10 ⁻⁰⁷	1.61×10 ⁻⁰⁴
	Somvunath	8.32×10 ⁻⁰⁵	2.35×10 ⁻⁰⁵	2.53×10 ⁻⁰⁵	4.39×10 ⁻⁰⁵	6.43×10 ⁻⁰⁷	6.63×10 ⁻⁰⁷	1.77×10 ⁻⁰⁴
	Protim	7.34×10 ⁻⁰⁵	2.72×10 ⁻⁰⁵	3.93×10 ⁻⁰⁵	2.61×10 ⁻⁰⁵	7.32×10 ⁻⁰⁷	7.32×10 ⁻⁰⁷	1.67×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.47×10 ⁻⁰⁴	6.62×10 ⁻⁰⁶	4.01×10 ⁻⁰⁵	3.80×10 ⁻⁰⁵	4.49×10 ⁻⁰⁷	4.50×10 ⁻⁰⁶	2.37×10 ⁻⁰⁴
	Sunil	1.08×10 ⁻⁰⁴	6.62×10 ⁻⁰⁶	6.46×10 ⁻⁰⁵	3.20×10 ⁻⁰⁵	4.48×10 ⁻⁰⁷	4.50×10 ⁻⁰⁶	2.16×10 ⁻⁰⁴
	Hamid	1.22×10 ⁻⁰⁴	1.54×10 ⁻⁰⁵	2.59×10 ⁻⁰⁵	3.32×10 ⁻⁰⁵	5.48×10 ⁻⁰⁷	5.40×10 ⁻⁰⁶	2.02×10 ⁻⁰⁴
	Ronjit	1.04×10 ⁻⁰⁴	2.43×10 ⁻⁰⁵	1.50×10 ⁻⁰⁴	2.96×10 ⁻⁰⁵	6.64×10 ⁻⁰⁷	6.60×10 ⁻⁰⁶	3.15×10 ⁻⁰⁴
	Soumen	9.63×10 ⁻⁰⁵	1.54×10 ⁻⁰⁵	2.50×10 ⁻⁰⁵	2.73×10 ⁻⁰⁵	6.57×10 ⁻⁰⁷	6.60×10 ⁻⁰⁶	1.71×10 ⁻⁰⁴

Table 4.13. Average daily dose (ADD) of Cadmium for Average population sub-group.

Village	Owner's name	Average Daily Dose (ADD) of Cd/ mg kg ⁻¹ d ⁻¹						
		CdADD _{ite}	CdADD _{veg}	CdADD _{fish}	CdADD _{water}	CdADD _{dust}	CdADD _{Air}	CdADD _{total}
Fudki Para	Polash	7.34×10 ⁻⁰⁷	4.41×10 ⁻⁰⁷	5.12×10 ⁻⁰⁷	1.17×10 ⁻⁰⁴	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	1.19×10 ⁻⁰⁴
	Manik	7.34×10 ⁻⁰⁷	3.31×10 ⁻⁰⁶	1.45×10 ⁻⁰⁶	9.27×10 ⁻⁰⁵	2.87×10 ⁻¹⁰	2.87×10 ⁻¹⁰	9.82×10 ⁻⁰⁵
	Robi	7.34×10 ⁻⁰⁷	3.64×10 ⁻⁰⁶	1.67×10 ⁻⁰⁶	9.67×10 ⁻⁰⁵	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	1.03×10 ⁻⁰⁴
	Somvunath	9.79×10 ⁻⁰⁷	2.65×10 ⁻⁰⁶	1.15×10 ⁻⁰⁶	1.03×10 ⁻⁰⁴	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	1.08×10 ⁻⁰⁴
	Protim	7.34×10 ⁻⁰⁷	1.76×10 ⁻⁰⁶	1.62×10 ⁻⁰⁶	9.76×10 ⁻⁰⁵	4.79×10 ⁻¹⁰	4.79×10 ⁻¹⁰	1.02×10 ⁻⁰⁴
Kumar Para	Kousik ghosh	2.52×10 ⁻⁰⁷	1.32×10 ⁻⁰⁶	1.41×10 ⁻⁰⁶	7.55×10 ⁻⁰⁵	8.62×10 ⁻¹⁰	8.60×10 ⁻¹⁰	7.85×10 ⁻⁰⁴
	Sunil	2.45×10 ⁻⁰⁷	6.62×10 ⁻⁰⁷	1.24×10 ⁻⁰⁶	8.57×10 ⁻⁰⁵	5.75×10 ⁻¹⁰	5.75×10 ⁻¹⁰	8.78×10 ⁻⁰⁴
	Hamid	2.79×10 ⁻⁰⁷	2.32×10 ⁻⁰⁶	2.78×10 ⁻⁰⁶	8.86×10 ⁻⁰⁵	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	9.40×10 ⁻⁰⁴
	Ronjit	4.16×10 ⁻⁰⁷	2.43×10 ⁻⁰⁶	7.26×10 ⁻⁰⁷	1.09×10 ⁻⁰⁴	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	9.40×10 ⁻⁰⁴
	Soumen	1.76×10 ⁻⁰⁷	1.32×10 ⁻⁰⁶	8.11×10 ⁻⁰⁷	8.39×10 ⁻⁰⁵	3.83×10 ⁻¹⁰	3.83×10 ⁻¹⁰	8.62×10 ⁻⁰⁴

Table 4.14. Average daily dose (ADD) of Lead for Average population sub-group

Village	Owner's name	Average Daily Dose (ADD) of Pb / mg kg ⁻¹ d ⁻¹						
		^{Pb} ADD _{ite}	^{Pb} ADD _{veg}	^{Pb} ADD _{fish}	^{Pb} ADD _{water}	^{Pb} ADD _{dust}	^{Pb} ADD _{Air}	^{Pb} ADD _{total}
Fudki Para	Polash	1.54×10 ⁻⁰⁴	2.98×10 ⁻⁰⁶	3.97×10 ⁻⁰⁶	1.67×10 ⁻⁰⁵	3.82×10 ⁻⁰⁸	3.82×10 ⁻⁰⁸	1.78×10 ⁻⁰⁴
	Manik	5.92×10 ⁻⁰⁵	1.30×10 ⁻⁰⁵	6.70×10 ⁻⁰⁶	2.82×10 ⁻⁰⁵	4.36×10 ⁻⁰⁸	4.36×10 ⁻⁰⁸	1.07×10 ⁻⁰⁴
	Robi	4.48×10 ⁻⁰⁵	1.39×10 ⁻⁰⁵	4.31×10 ⁻⁰⁶	1.81×10 ⁻⁰⁵	1.28×10 ⁻⁰⁸	1.28×10 ⁻⁰⁸	8.11×10 ⁻⁰⁵
	Somvunath	7.27×10 ⁻⁰⁵	1.00×10 ⁻⁰⁵	4.06×10 ⁻⁰⁶	1.71×10 ⁻⁰⁵	6.22×10 ⁻⁰⁹	6.22×10 ⁻⁰⁹	1.04×10 ⁻⁰⁴
	Protim	8.86×10 ⁻⁰⁵	9.37×10 ⁻⁰⁶	7.64×10 ⁻⁰⁶	3.22×10 ⁻⁰⁵	1.27×10 ⁻⁰⁸	1.27×10 ⁻⁰⁸	1.38×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.24×10 ⁻⁰⁴	3.42×10 ⁻⁰⁶	4.35×10 ⁻⁰⁶	1.83×10 ⁻⁰⁵	2.82×10 ⁻⁰⁸	2.82×10 ⁻¹⁰	1.50×10 ⁻⁰⁴
	Sunil	1.13×10 ⁻⁰⁴	2.87×10 ⁻⁰⁶	5.72×10 ⁻⁰⁶	2.41×10 ⁻⁰⁵	2.39×10 ⁻⁰⁸	2.40×10 ⁻¹⁰	1.46×10 ⁻⁰⁴
	Hamid	1.38×10 ⁻⁰⁴	1.29×10 ⁻⁰⁵	7.98×10 ⁻⁰⁶	3.36×10 ⁻⁰⁵	1.27×10 ⁻⁰⁸	1.28×10 ⁻¹⁰	1.92×10 ⁻⁰⁴
	Ronjit	1.25×10 ⁻⁰⁴	1.10×10 ⁻⁰⁵	3.54×10 ⁻⁰⁶	1.49×10 ⁻⁰⁵	1.26×10 ⁻⁰⁸	1.27×10 ⁻¹⁰	1.54×10 ⁻⁰⁴
	Soumen	1.19×10 ⁻⁰⁴	5.18×10 ⁻⁰⁶	6.79×10 ⁻⁰⁶	2.86×10 ⁻⁰⁵	1.91×10 ⁻⁰⁸	1.91×10 ⁻¹⁰	1.60×10 ⁻⁰⁴

Table 4.15. Average daily dose (ADD) of arsenic for senior population sub-group.

Village	Owner's name	Average Daily Dose (ADD) of As / mg kg ⁻¹ d ⁻¹						
		^{As} ADD _{rice}	^{As} ADD _{veg}	^{As} ADD _{fish}	^{As} ADD _{water}	^{As} ADD _{dust}	^{As} ADD _{Air}	^{As} ADD _{total}
Fudki Para	Polash	7.80×10 ⁻⁰⁵	3.90×10 ⁻⁰⁶	2.03×10 ⁻⁰⁵	2.13×10 ⁻⁰⁵	6.72×10 ⁻⁰⁷	6.72×10 ⁻⁰⁷	1.25×10 ⁻⁰⁴
	Manik	8.91×10 ⁻⁰⁵	1.56×10 ⁻⁰⁵	2.48×10 ⁻⁰⁵	2.69×10 ⁻⁰⁵	6.15×10 ⁻⁰⁷	6.15×10 ⁻⁰⁷	1.58×10 ⁻⁰⁴
	Robi	6.54×10 ⁻⁰⁵	1.89×10 ⁻⁰⁵	3.48×10 ⁻⁰⁵	1.76×10 ⁻⁰⁵	6.24×10 ⁻⁰⁷	6.24×10 ⁻⁰⁷	1.38×10 ⁻⁰⁴
	Somvunath	7.10×10 ⁻⁰⁵	1.78×10 ⁻⁰⁵	2.48×10 ⁻⁰⁵	3.43×10 ⁻⁰⁵	6.37×10 ⁻⁰⁷	6.37×10 ⁻⁰⁷	1.49×10 ⁻⁰⁴
	Protim	6.26×10 ⁻⁰⁵	2.06×10 ⁻⁰⁵	3.48×10 ⁻⁰⁵	2.04×10 ⁻⁰⁵	7.02×10 ⁻⁰⁷	7.02×10 ⁻⁰⁷	1.40×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.25×10 ⁻⁰⁴	5.01×10 ⁻⁰⁶	3.93×10 ⁻⁰⁵	2.97×10 ⁻⁰⁵	4.31×10 ⁻⁰⁷	4.20×10 ⁻⁰⁶	2.04×10 ⁻⁰⁴
	Sunil	9.19×10 ⁻⁰⁵	5.01×10 ⁻⁰⁶	6.32×10 ⁻⁰⁵	2.51×10 ⁻⁰⁵	4.30×10 ⁻⁰⁷	4.20×10 ⁻⁰⁶	1.90×10 ⁻⁰⁴
	Hamid	1.04×10 ⁻⁰⁴	1.17×10 ⁻⁰⁵	2.53×10 ⁻⁰⁵	2.60×10 ⁻⁰⁵	5.26×10 ⁻⁰⁷	5.40×10 ⁻⁰⁶	1.73×10 ⁻⁰⁴
	Ronjit	8.91×10 ⁻⁰⁵	1.84×10 ⁻⁰⁵	1.47×10 ⁻⁰⁴	2.32×10 ⁻⁰⁵	6.37×10 ⁻⁰⁷	6.30×10 ⁻⁰⁶	2.85×10 ⁻⁰⁴
	Soumen	8.21×10 ⁻⁰⁵	1.17×10 ⁻⁰⁵	2.45×10 ⁻⁰⁵	2.13×10 ⁻⁰⁵	6.31×10 ⁻⁰⁷	6.30×10 ⁻⁰⁶	1.47×10 ⁻⁰⁴

Table 4.16. Average daily dose (ADD) of Cadmium for senior population sub-group

	Owner's name	Average Daily Dose (ADD) of Cd/ mg kg ⁻¹ d ⁻¹						
		CdADD _{ice}	CdADD _{veg}	CdADD _{fish}	CdADD _{water}	CdADD _{dust}	CdADD _{Air}	CdADD _{total}
Fudki Para	Polash	6.26×10 ⁻⁰⁷	3.34×10 ⁻⁰⁷	5.01×10 ⁻⁰⁷	9.08×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	9.23×10 ⁻⁰⁵
	Manik	6.26×10 ⁻⁰⁷	2.51×10 ⁻⁰⁶	1.42×10 ⁻⁰⁶	7.18×10 ⁻⁰⁵	2.76×10 ⁻¹⁰	2.76×10 ⁻¹⁰	7.64×10 ⁻⁰⁵
	Robi	6.26×10 ⁻⁰⁷	2.76×10 ⁻⁰⁶	1.63×10 ⁻⁰⁶	7.49×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	7.64×10 ⁻⁰⁵
	Somvunath	8.35×10 ⁻⁰⁷	2.00×10 ⁻⁰⁶	1.13×10 ⁻⁰⁶	8.00×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	8.40×10 ⁻⁰⁵
	Protim	6.26×10 ⁻⁰⁷	1.34×10 ⁻⁰⁶	1.59×10 ⁻⁰⁶	7.56×10 ⁻⁰⁵	4.60×10 ⁻¹⁰	4.60×10 ⁻¹⁰	7.91×10 ⁻⁰⁵
Kumar Para	Kousik Ghosh	2.15×10 ⁻⁰⁵	1.00×10 ⁻⁰⁶	1.38×10 ⁻⁰⁶	5.85×10 ⁻⁰⁵	8.27×10 ⁻¹⁰	8.25×10 ⁻¹⁰	8.24×10 ⁻⁰⁴
	Sunil	2.09×10 ⁻⁰⁵	5.01×10 ⁻⁰⁷	1.21×10 ⁻⁰⁶	6.64×10 ⁻⁰⁵	5.51×10 ⁻¹⁰	5.50×10 ⁻¹⁰	8.90×10 ⁻⁰⁴
	Hamid	2.38×10 ⁻⁰⁵	1.75×10 ⁻⁰⁶	2.71×10 ⁻⁰⁶	6.86×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	9.68×10 ⁻⁰⁴
	Ronjit	3.55×10 ⁻⁰⁵	1.84×10 ⁻⁰⁶	7.10×10 ⁻⁰⁷	8.48×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	1.23×10 ⁻⁰⁴
	Soumen	1.50×10 ⁻⁰⁵	1.00×10 ⁻⁰⁶	7.94×10 ⁻⁰⁷	6.50×10 ⁻⁰⁵	3.68×10 ⁻¹⁰	3.68×10 ⁻¹⁰	8.18×10 ⁻⁰⁴

Table 4.17. Average daily dose (ADD) of Lead for senior population sub-group.

Village	Owner's name	Average Daily Dose (ADD) of Pb / mg kg ⁻¹ d ⁻¹						
		^{Pb} ADD _{ice}	^{Pb} ADD _{veg}	^{Pb} ADD _{fish}	^{Pb} ADD _{water}	^{Pb} ADD _{dust}	^{Pb} ADD _{Air}	^{Pb} ADD _{total}
Fudki Para	Polash	1.32×10 ⁻⁰⁴	1.13×10 ⁻⁰⁶	3.88×10 ⁻⁰⁶	1.29×10 ⁻⁰⁵	3.67×10 ⁻⁰⁸	3.67×10 ⁻⁰⁸	1.50×10 ⁻⁰⁴
	Manik	5.05×10 ⁻⁰⁵	4.93×10 ⁻⁰⁶	6.56×10 ⁻⁰⁶	2.19×10 ⁻⁰⁵	4.18×10 ⁻⁰⁸	4.18×10 ⁻⁰⁸	8.40×10 ⁻⁰⁵
	Robi	3.82×10 ⁻⁰⁵	5.26×10 ⁻⁰⁶	4.22×10 ⁻⁰⁶	1.41×10 ⁻⁰⁵	1.23×10 ⁻⁰⁹	1.23×10 ⁻⁰⁸	6.18×10 ⁻⁰⁵
	Somvunath	6.20×10 ⁻⁰⁵	3.80×10 ⁻⁰⁶	3.97×10 ⁻⁰⁶	1.32×10 ⁻⁰⁵	5.97×10 ⁻⁰⁹	5.97×10 ⁻⁰⁹	8.30×10 ⁻⁰⁵
	Protim	7.56×10 ⁻⁰⁵	3.55×10 ⁻⁰⁶	7.48×10 ⁻⁰⁶	2.49×10 ⁻⁰⁵	1.22×10 ⁻⁰⁹	1.22×10 ⁻⁰⁸	1.12×10 ⁻⁰⁴
Kumar Para	Kousik Ghosh	1.06×10 ⁻⁰⁴	1.29×10 ⁻⁰⁶	4.26×10 ⁻⁰⁶	1.42×10 ⁻⁰⁶	2.70×10 ⁻⁰⁹	1.35×10 ⁻⁰⁸	1.31×10 ⁻⁰⁴
	Sunil	9.65×10 ⁻⁰⁵	1.09×10 ⁻⁰⁶	5.60×10 ⁻⁰⁶	1.87×10 ⁻⁰⁶	2.30×10 ⁻⁰⁹	1.15×10 ⁻⁰⁸	1.05×10 ⁻⁰⁴
	Hamid	1.18×10 ⁻⁰⁴	4.89×10 ⁻⁰⁶	7.81×10 ⁻⁰⁶	2.60×10 ⁻⁰⁵	1.22×10 ⁻⁰⁹	6.10×10 ⁻⁰⁹	1.57×10 ⁻⁰⁴
	Ronjit	1.07×10 ⁻⁰⁴	4.18×10 ⁻⁰⁶	3.47×10 ⁻⁰⁶	1.16×10 ⁻⁰⁶	1.21×10 ⁻⁰⁹	6.05×10 ⁻⁰⁹	1.16×10 ⁻⁰⁴
	Soumen	1.01×10 ⁻⁰⁴	1.96×10 ⁻⁰⁶	6.64×10 ⁻⁰⁶	2.21×10 ⁻⁰⁵	1.83×10 ⁻⁰⁹	9.15×10 ⁻⁰⁹	1.32×10 ⁻⁰⁴

Amongst the ingestion and inhalation processes, dust driven air includes in inhalation and the others are in ingestion processes. Studied metal exposure contribution for the three population sub-groups are shown in table 4.18. It is clear that ingestion processes cover more contribution than inhalation process towards metal exposure. On an average, 98.26 % exposure is contributed from ingestion processes, while the rest (1.73 %) from inhalation process.

Table 4.18 Metal exposure contribution for three population sub-groups.

Population sub-groups	Exposure Contribution (%)		
	Metal	Ingestion	Inhalation
Children	As	94.18	5.820
	Cd	99.99	0.003
	Pb	99.92	0.075
Adult	As	96.23	3.770
	Cd	99.99	0.0023
	Pb	99.96	0.042
Senior	As	94.18	5.820
	Cd	99.99	0.003
	Pb	99.93	0.075

Average total HQ value ($^M\text{HQ}_{\text{Total}}$) of arsenic, cadmium and lead contents for children are 1.43, 0.49 and 0.45, respectively (Table 4.19). The table reflects that for the same diet and atmosphere, As, Cd and Pb contribute to 60.79%, 20.89% and 18.32% respectively for children. But for average person, the As, Cd and Pb contributes is 78.86%, 20.78% and 0.36%. For senior person, the As, Cd and Pb contributes is 70.15%, 17.50% and 12.38%. In total exposure the order is $\text{As} > \text{Cd} > \text{Pb}$ for all population sub-groups.

Table 4.19 Average HQ contribution in HI value.

Population sub-groups	Average metal wise HQ value			Contribution of metal HQ in HI value (%)		
	$As_{HQ_{Total}}$	$Cd_{HQ_{Total}}$	$Pb_{HQ_{Total}}$	$As_{HQ_{Total}}$	$Cd_{HQ_{Total}}$	$Pb_{HQ_{Total}}$
Children	1.43	0.49	0.45	60.79	20.89	18.32
Adult	0.80	0.21	0.003	78.86	20.78	0.36
Senior	0.68	0.17	0.12	70.15	17.50	12.38

For all population sub-groups metal contribution towards HQ values followed the order; for arsenic: rice (50.52) > fish (18.99) > drinking (14.25) > vegetable (11.32) > dust (4.61); for cadmium: drinking (86.26) > rice (10.61) > vegetable (1.61) > fish (1.53) > dust (0.002) and for lead: rice (68.55) > drinking (19.99) > vegetable (5.89) > fish (5.52) > dust (0.056) (Table 4.18). Table 4.19 and 4.20 summarize the HQ values for different population sub-groups that is shown in Tables 4.21-4.29.

Table 4.20 Average contribution of HQ value in Average total HQ value (%).

Population Sub groups	Metal	Sample wise contribution in Average total HQ value (%)				
		Rice	Vegetable	Fish	Water	Dust
Children	As	39.76	11.35	24.60	17.81	5.82
	Cd	6.72	1.32	1.67	90.30	0.002
	Pb	58.34	7.50	7.58	26.52	0.075
Average person	As	55.98	12.00	15.10	13.02	3.78
	Cd	12.04	1.80	1.31	84.88	0.002
	Pb	71.83	6.93	4.07	17.12	0.042
Senior Person	As	55.82	10.62	17.26	11.91	4.23
	Cd	13.06	1.71	1.62	83.61	0.002
	Pb	75.47	3.24	4.90	16.33	0.050

Table 4.21. Arsenic contributed hazard quotient (HQ) for children population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		^{As} HQ _{rice}	^{As} HQ _{veg}	^{As} HQ _{fish}	^{As} HQ _{water}	^{As} HQ _{soil}	^{As} HQ _{dust}	^{As} HQ _{total}
Fudki Para	Polash	0.385	0.029	0.201	0.221	0.008	0.064	0.907
	Manik	0.440	0.115	0.245	0.279	0.007	0.059	1.144
	Robi	0.323	0.140	0.343	0.183	0.007	0.059	1.056
	Somvun-ath	0.350	0.132	0.245	0.356	0.007	0.061	1.150
	Protim	0.309	0.152	0.379	0.212	0.008	0.067	1.127
Kumar Para	Kousik Ghosh	0.618	0.037	0.387	0.308	0.005	0.041	1.396
	Sunil	0.453	0.037	0.624	0.260	0.005	0.041	1.420
	Hamid	0.515	0.087	0.250	0.269	0.006	0.050	1.177
	Ronjit	0.440	0.136	1.448	0.240	0.007	0.061	2.332
	Soumen	0.405	0.087	0.242	0.221	0.007	0.060	1.022

Table 4.22. Cadmium contributed hazard quotient (HQ) for children population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$^{Cd}HQ_{rice}$	$^{Cd}HQ_{veg}$	$^{Cd}HQ_{fish}$	$^{Cd}HQ_{water}$	$^{Cd}HQ_{soil}$	$^{Cd}HQ_{dust}$	$^{Cd}HQ_{total}$
Fudki Para	Polash	0.0019	0.0015	0.0030	0.5643	2.55×10^{-06}	2.17×10^{-06}	0.5706
	Manik	0.0019	0.0111	0.0084	0.4466	1.91×10^{-06}	1.63×10^{-06}	0.4680
	Robi	0.0019	0.0122	0.0096	0.4656	2.55×10^{-06}	2.17×10^{-06}	0.4894
	Somvunath	0.0025	0.0089	0.0067	0.4977	2.55×10^{-06}	2.17×10^{-06}	0.5157
	Protim	0.0019	0.0059	0.0094	0.4700	3.19×10^{-06}	2.71×10^{-06}	0.4872
Kumar Para	Kousik Ghosh	0.0504	0.0026	0.0028	0.1509	6.48×10^{-07}	1.72×10^{-06}	0.207
	Sunil	0.0490	0.0013	0.0025	0.1714	4.32×10^{-07}	1.15×10^{-06}	0.224
	Hamid	0.0558	0.0046	0.0056	0.1772	2.88×10^{-07}	7.66×10^{-07}	0.243
	Ronjit	0.0832	0.0049	0.0015	0.2188	2.88×10^{-07}	7.66×10^{-07}	0.308
	Soumen	0.0352	0.0026	0.0016	0.1678	2.88×10^{-07}	7.66×10^{-07}	0.207

Table 4.23. Lead contributed hazard quotient (HQ) for children population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		^{Pb} HQ _{rice}	^{Pb} HQ _{veg}	^{Pb} HQ _{fish}	^{Pb} HQ _{water}	^{Pb} HQ _{soil}	^{Pb} HQ _{dust}	^{Pb} HQ _{total}
Fudki Para	Polash	0.390	0.010	0.023	0.080	2.39×10^{-06}	0.0002	0.504
	Manik	0.150	0.044	0.039	0.136	2.73×10^{-06}	0.0002	0.368
	Robi	0.113	0.047	0.025	0.087	8.04×10^{-07}	0.0001	0.272
	Somvunath	0.184	0.034	0.023	0.082	3.90×10^{-07}	0.0000	0.323
	Protim	0.224	0.032	0.044	0.155	7.98×10^{-07}	0.0001	0.455
Kumar Para	Kousik Ghosh	0.313	0.011	0.025	0.088	1.76×10^{-06}	0.0002	0.438
	Sunil	0.286	0.010	0.033	0.116	1.50×10^{-06}	0.0001	0.445
	Hamid	0.348	0.043	0.046	0.162	7.98×10^{-07}	0.0001	0.600
	Ronjit	0.316	0.037	0.021	0.072	7.92×10^{-07}	0.0001	0.445
	Soumen	0.300	0.017	0.039	0.138	1.19×10^{-06}	0.0001	0.495

Table 4.24. Arsenic contributed hazard quotient (HQ) for adult population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$^{As}HQ_{\text{rice}}$	$^{As}HQ_{\text{veg}}$	$^{As}HQ_{\text{fish}}$	$^{As}HQ_{\text{water}}$	$^{As}HQ_{\text{soil}}$	$^{As}HQ_{\text{dust}}$	$^{As}HQ_{\text{total}}$
Fudki Para	Polash	0.305	0.017	0.069	0.091	0.001	0.023	0.506
	Manik	0.348	0.069	0.084	0.115	0.001	0.021	0.638
	Robi	0.256	0.083	0.119	0.075	0.001	0.022	0.555
	Somvunath	0.277	0.078	0.084	0.146	0.001	0.022	0.609
	Protim	0.245	0.091	0.131	0.087	0.001	0.024	0.579
Kumar Para	Kousik Ghosh	0.490	0.022	0.134	0.127	0.001	0.015	0.787
	Sunil	0.359	0.022	0.215	0.107	0.001	0.015	0.719
	Hamid	0.408	0.051	0.086	0.111	0.001	0.018	0.675
	Ronjit	0.348	0.081	0.500	0.099	0.001	0.022	1.051
	Soumen	0.321	0.051	0.083	0.091	0.001	0.022	0.570

Table 4.25. Cadmium contributed hazard quotient (HQ) for adult population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$^{Cd}HQ_{rice}$	$^{Cd}HQ_{veg}$	$^{Cd}HQ_{fish}$	$^{Cd}HQ_{water}$	$^{Cd}HQ_{soil}$	$^{Cd}HQ_{dust}$	$^{Cd}HQ_{total}$
Fudki Para	Polash	0.0015	0.0009	0.0010	0.2343	2.88×10^{-07}	7.66×10^{-07}	0.238
	Manik	0.0015	0.0066	0.0029	0.1854	2.16×10^{-07}	5.75×10^{-07}	0.196
	Robi	0.0015	0.0073	0.0033	0.1933	2.88×10^{-07}	7.66×10^{-07}	0.205
	Somvunath	0.0020	0.0053	0.0023	0.2066	2.88×10^{-07}	7.66×10^{-07}	0.216
	Protim	0.0015	0.0035	0.0032	0.1951	3.60×10^{-07}	9.58×10^{-07}	0.203
Kumar Para	Kousik Ghosh	0.0504	0.0026	0.0028	0.1509	6.48×10^{-07}	1.72×10^{-06}	0.207
	Sunil	0.0490	0.0013	0.0025	0.1714	4.32×10^{-07}	1.15×10^{-06}	0.224
	Hamid	0.0558	0.0046	0.0056	0.1772	2.88×10^{-07}	7.66×10^{-07}	0.243
	Ronjit	0.0832	0.0049	0.0015	0.2188	2.88×10^{-07}	7.66×10^{-07}	0.308
	Soumen	0.0352	0.0026	0.0016	0.1678	2.88×10^{-07}	7.66×10^{-07}	0.207

Table 4.26. Lead contributed hazard quotient (HQ) for adult population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$PbHQ_{rice}$	$PbHQ_{veg}$	$PbHQ_{fish}$	$PbHQ_{water}$	$PbHQ_{soil}$	$PbHQ_{dust}$	$PbHQ_{total}$
Fudki Para	Polash	0.003	0.0001	0.0001	0.0003	2.87×10^{-07}	7.64×10^{-07}	0.004
	Manik	0.001	0.0003	0.0001	0.0006	3.28×10^{-07}	8.71×10^{-07}	0.002
	Robi	0.001	0.0003	0.0001	0.0004	9.65×10^{-08}	2.57×10^{-07}	0.002
	Somvunath	0.001	0.0002	0.0001	0.0003	4.68×10^{-08}	1.24×10^{-07}	0.002
	Protim	0.002	0.0002	0.0002	0.0006	9.58×10^{-07}	2.55×10^{-07}	0.003
Kumar Para	Kousik Ghosh	0.002	0.0001	0.0001	0.0004	2.12×10^{-07}	5.63×10^{-07}	0.003
	Sunil	0.002	0.0001	0.0001	0.0005	1.80×10^{-07}	4.79×10^{-07}	0.003
	Hamid	0.003	0.0003	0.0002	0.0007	9.58×10^{-08}	2.55×10^{-07}	0.004
	Ronjit	0.003	0.0002	0.0001	0.0003	9.50×10^{-08}	2.53×10^{-07}	0.003
	Soumen	0.002	0.0001	0.0001	0.0006	1.43×10^{-07}	3.81×10^{-07}	0.003

Table 4.27. Arsenic contributed hazard quotient (HQ) for senior population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		^{As} HQ _{rice}	^{As} HQ _{veg}	^{As} HQ _{fish}	^{As} HQ _{water}	^{As} HQ _{soil}	^{As} HQ _{dust}	^{As} HQ _{total}
Fudki Para	Polash	0.260	0.013	0.068	0.071	0.001	0.022	0.435
	Manik	0.297	0.052	0.083	0.090	0.001	0.021	0.543
	Robi	0.218	0.063	0.116	0.059	0.001	0.021	0.478
	Somvunath	0.237	0.059	0.083	0.114	0.001	0.021	0.515
	Protim	0.209	0.069	0.128	0.068	0.001	0.023	0.498
Kumar Para	Kousik Ghosh	0.418	0.017	0.131	0.099	0.001	0.014	0.679
	Sunil	0.306	0.017	0.211	0.084	0.001	0.014	0.632
	Hamid	0.348	0.039	0.084	0.087	0.001	0.018	0.576
	Ronjit	0.297	0.061	0.489	0.077	0.001	0.021	0.947
	Soumen	0.274	0.039	0.082	0.071	0.001	0.021	0.487

Table 4.28. Cadmium contributed hazard quotient (HQ) for senior population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$Cd_{HQ_{rice}}$	$Cd_{HQ_{veg}}$	$Cd_{HQ_{fish}}$	$Cd_{HQ_{water}}$	$Cd_{HQ_{soil}}$	$Cd_{HQ_{dust}}$	$Cd_{HQ_{total}}$
Fudki Para	Polash	0.0013	0.0007	0.0010	0.1815	2.80×10^{-07}	7.35×10^{-07}	0.184
	Manik	0.0013	0.0050	0.0028	0.1437	2.10×10^{-07}	5.51×10^{-07}	0.153
	Robi	0.0013	0.0055	0.0033	0.1498	2.80×10^{-07}	7.35×10^{-07}	0.160
	Somvunath	0.0017	0.0040	0.0023	0.1601	2.80×10^{-07}	7.35×10^{-07}	0.168
	Protim	0.0013	0.0027	0.0032	0.1512	3.50×10^{-07}	9.19×10^{-07}	0.158
Kumar Para	Kousik Ghosh	0.0430	0.0020	0.0028	0.1169	6.30×10^{-07}	1.65×10^{-06}	0.165
	Sunil	0.0418	0.0010	0.0024	0.1328	4.20×10^{-07}	1.10×10^{-06}	0.178
	Hamid	0.0476	0.0035	0.0054	0.1373	2.80×10^{-07}	7.35×10^{-07}	0.194
	Ronjit	0.0710	0.0037	0.0014	0.1696	2.80×10^{-07}	7.35×10^{-07}	0.246
	Soumen	0.0301	0.0020	0.0016	0.1300	2.80×10^{-07}	7.35×10^{-07}	0.164

Table 4.29. Lead contributed hazard quotient (HQ) for senior population sub-group.

Village	Owner's name	Hazard Quotient (HQ)						
		$Pb_{HQ_{rice}}$	$Pb_{HQ_{veg}}$	$Pb_{HQ_{fish}}$	$Pb_{HQ_{water}}$	$Pb_{HQ_{soil}}$	$Pb_{HQ_{dust}}$	$Pb_{HQ_{total}}$
Fudki Para	Polash	0.1318	0.0011	0.0039	0.0129	1.40×10^{-05}	3.67×10^{-05}	0.150
	Manik	0.0505	0.0049	0.0066	0.0219	1.59×10^{-05}	4.18×10^{-05}	0.084
	Robi	0.0382	0.0053	0.0042	0.0141	4.69×10^{-06}	1.23×10^{-05}	0.062
	Somvunath	0.0620	0.0038	0.0040	0.0132	2.28×10^{-06}	5.97×10^{-06}	0.083
	Protim	0.0756	0.0035	0.0075	0.0249	4.66×10^{-06}	1.22×10^{-05}	0.112
Kumar Para	Kousik Ghosh	0.1057	0.0013	0.0043	0.0142	1.03×10^{-05}	2.70×10^{-05}	0.125
	Sunil	0.0965	0.0011	0.0056	0.0187	8.75×10^{-06}	2.30×10^{-05}	0.122
	Hamid	0.1176	0.0049	0.0078	0.0260	4.66×10^{-06}	1.22×10^{-05}	0.156
	Ronjit	0.1067	0.0042	0.0035	0.0116	4.62×10^{-06}	1.21×10^{-05}	0.126
	Soumen	0.1015	0.0020	0.0066	0.0221	6.97×10^{-06}	1.83×10^{-05}	0.132

4.3 Risk assessment

$$HI = HQ_{As} + HQ_{Pb} + HQ_{Cd}$$

Hazard Index (HI) was calculate to showing greatest potential for risk indication and used it produce map. Standard risk assessment procedure is to evaluate the sum of HQ for the individual metals to provide a hazard index (HI); (Hampshire Reearch Institute 1995).

Values of HI were used to produce maps of the studied area thus showing where the greatest potential for risk was located. Children may receive higher doses of metals from food than adults, since they consume more food for their body weight than adults. The hazard quotients in table are calculated for the most sensitive life stage.

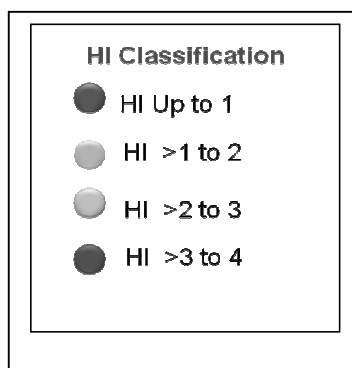
The highest hazard quotients in the table are calculated for As, Cd, Pb these being respectively. Hazard Quotients is a complex parameter used for the estimation of potential health risks associated with long term exposure to chemical pollutants. The hazard index which is the sum of hazard quotients, is a screening risk assessment technique commonly used to judge whether there is concern for additive effects between chemicals. If the hazard index is less than unity (1.0) it indicates that even if all the metals acted on the same organ or interacted in some other way to cause health effects the risk of these effects would be low. The hazard quotients and hazard index were calculated to assess potential health risk to local populations through dietary product (rice, vegetable, fish) drinking water and dust consumption. Based on the HQ values in this study, due to the HQ value ($HQ > 1$) for children of Futkipara and Kumarpara have danger of As. But HQ value of Cd and Pb is safe.

Risk assessment strategies are often aimed at population subgroups. It is common practice to identify vulnerable people in society, such young children or elderly, and assess potential risk to the health of these population subgroups[Dhuka and Miller 1999; Government /Research councils Initiative on risk Assessment and Toxicology (GRCIRAT) 1999]. Ryan and Chaney (1995) considered young children to be highly exposed individuals (HEIs). Thus risk assessment can usually focus on highly exposed subpopulations on the basis that if the risk to the HEI is acceptable then most of the population is protected [12]. Figure 4.2, 4.3 and 4.4 shows the HI maps for the children, average person and senior population subgroups for the studied area. The average HI value was 2.36 for children. The HI values for children population subgroups reflect that all children, since $HI > 1.00$ is in risk. For the population subgroup (Figure 4.1) 50% (5 out of 10) provides $HI > 2$ to 3; 40% (4 out of 10) provides $HI > 1$ to 2 and 10% (1 out of 10 provides $HI > 3$ to 4.

Hazard Index for all population sub-groups.

Table 4.30 Hazard Index for Children population sub-groups.

Village	Owner's name	Hazard Index (HI)			
		^{As} HQ _{total}	^{Pb} HQ _{total}	^{Cd} HQ _{total}	HI
Futki Para	Polash	0.907	0.504	0.570	1.981
	Manik	1.144	0.368	0.468	1.980
	Robi	1.056	0.272	0.489	1.817
	Somvunath	1.150	0.323	0.516	1.989
	Protim	1.127	0.455	0.487	2.069
Kumar Para	Kousik Ghosh	1.396	0.440	0.438	2.274
	Sunil	1.420	0.484	0.445	2.349
	Hamid	1.177	0.521	0.600	2.298
	Ronjit	2.332	0.645	0.445	3.422
	Soumen	1.022	0.458	0.495	1.975



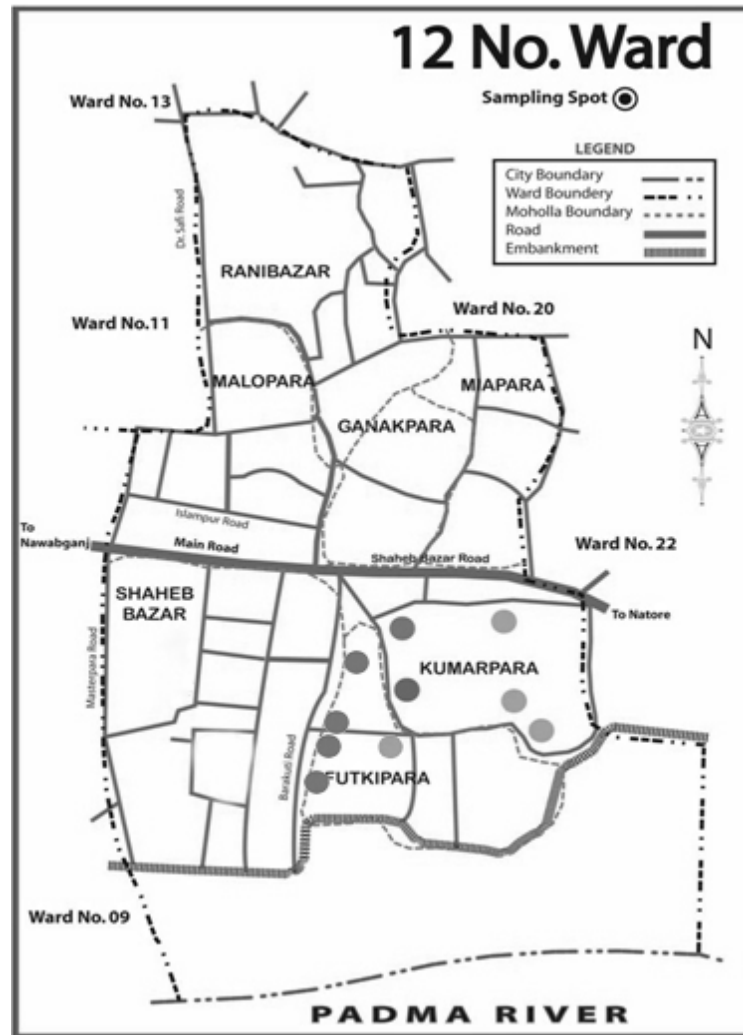


Figure 4.2 Map of HI values for the children population sub-group of the Fudkipara and Kumarpara .

Table 4.31. Hazard Index for Average population sub-groups.

Village	Owner's name	Hazard Index (HI)			
		$^{As}HQ_{total}$	$^{Pb}HQ_{total}$	$^{Cd}HQ_{total}$	HI
Futki Para	Polash	0.506	0.004	0.238	0.748
	Manik	0.638	0.002	0.196	0.836
	Robi	0.555	0.002	0.205	0.762
	Somvunath	0.609	0.002	0.216	0.827
	Protim	0.579	0.003	0.203	0.785
Kumar Para	Kousik Ghosh	0.787	0.207	0.003	0.997
	Sunil	0.719	0.224	0.003	0.946
	Hamid	0.675	0.243	0.004	0.922
	Ronjit	1.051	0.308	0.003	1.362
	Azizul Haque	0.685	0.003	0.216	0.904

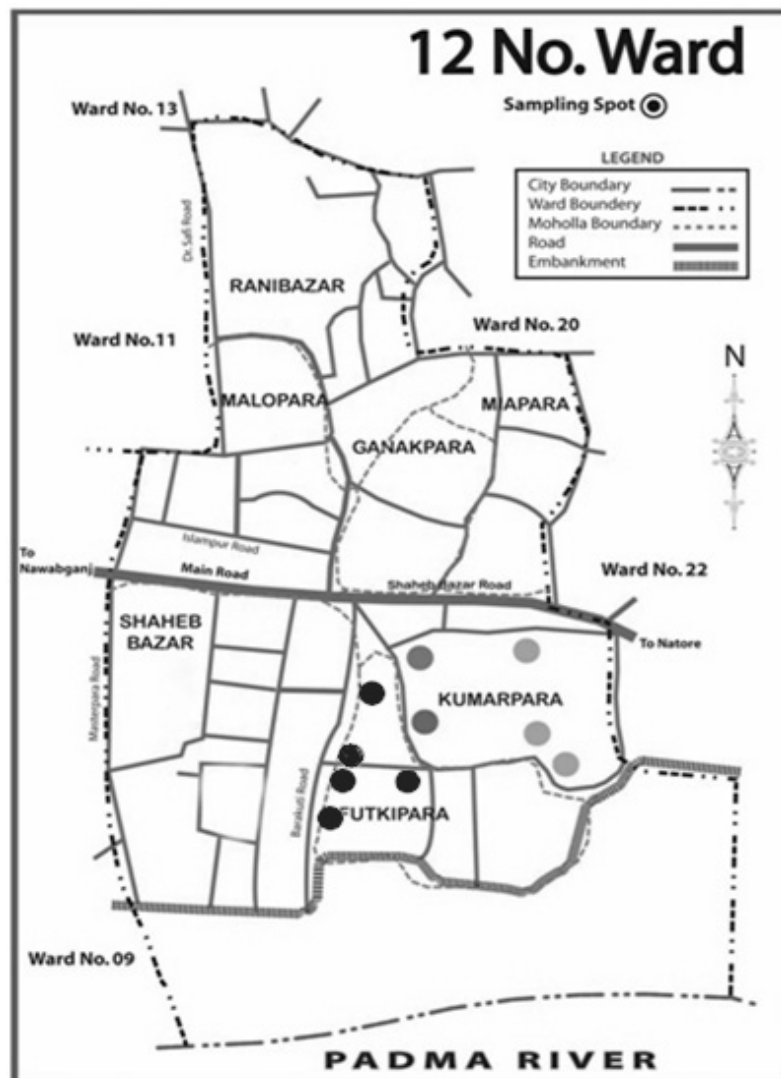
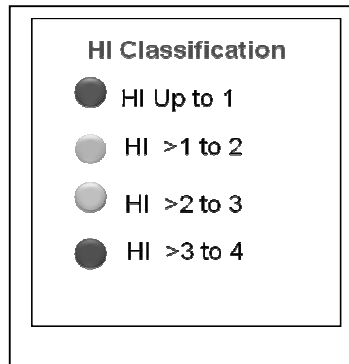


Figure 4.3 Map of HI values for the average population sub-group of the Futkipara and Kumarpara .

Table 4.32. Hazard Index for Senior population sub-groups.

Village	Owner's name	Hazard Index (HI)			
		$^{As}HQ_{total}$	$^{Pb}HQ_{total}$	$^{Cd}HQ_{total}$	HI
Fudki Para	Polash	0.435	0.150	0.184	0.769
	Manik	0.543	0.084	0.153	0.780
	Robi	0.478	0.062	0.160	0.700
	Somvunath	0.515	0.083	0.168	0.766
	Protim	0.498	0.112	0.158	0.768
Kumar Para	Kousik Ghosh	0.679	0.165	0.125	0.969
	Sunil	0.632	0.178	0.122	0.932
	Hamid	0.576	0.194	0.156	0.926
	Ronjit	0.947	0.246	0.126	1.319
	Soumen	0.487	0.164	0.132	0.783

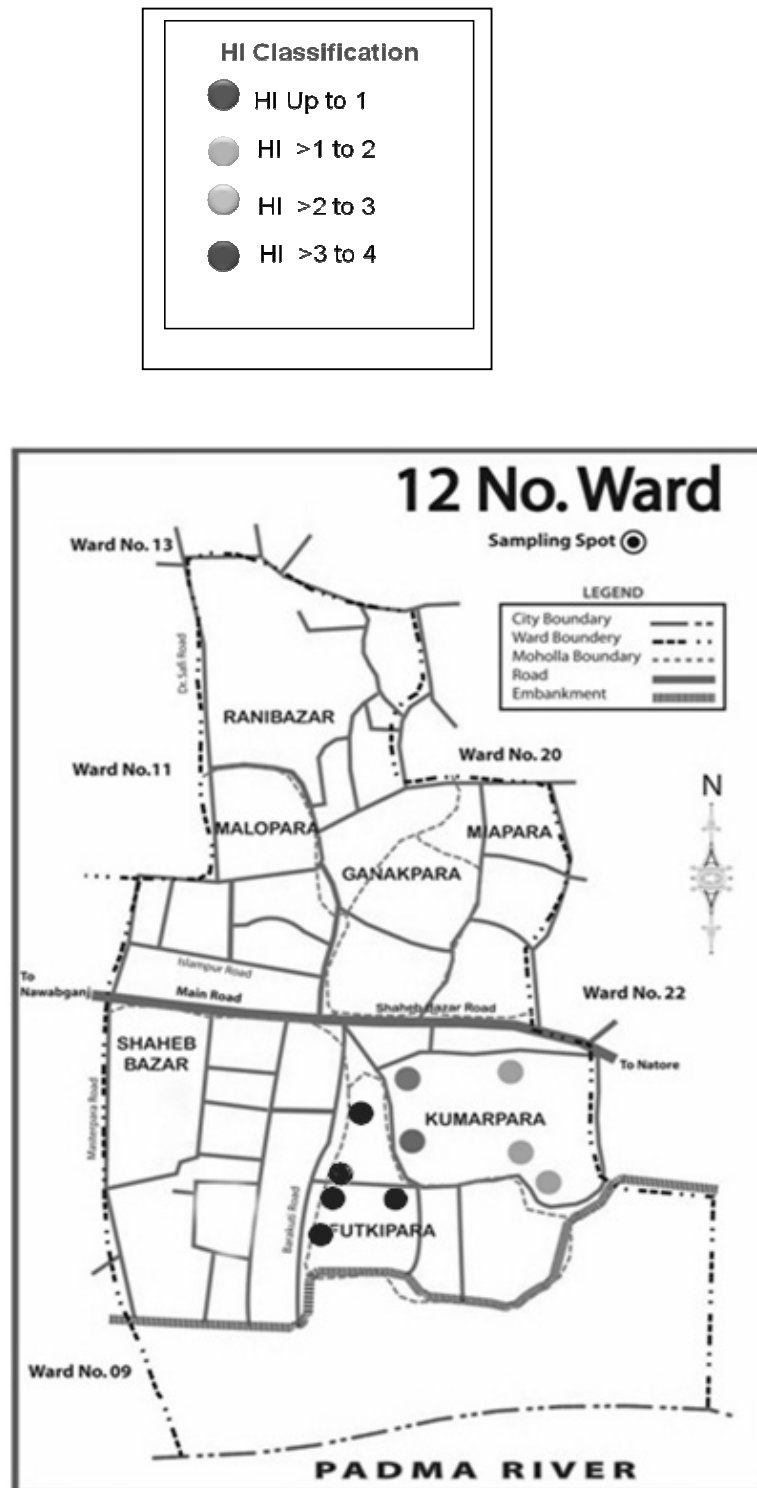


Figure 4.4 Map of HI values for the senior population sub-group of the Fudkipara and Kumarpara

4.4 Comparison

From map study the comparison of HI value follow the following order

Children > Senior person > A verage population.

4.5 Conclusion

1. As concentration range was (0.002 - 2.741) $\mu\text{g g}^{-1}$, Cd concentration (0.0003- 0.0143) $\mu\text{g g}^{-1}$ and Pb concentration range was (0.001 - 0.0398) $\mu\text{g g}^{-1}$
2. Generally the concentrations of the concerned metals in the studied samples follow the order: Dust > Rice > Fish > Vegetable > Water.
3. Metal exposure contribution order is Ingestion (98.26%) > Inhalation (1.73%).
4. Sample wise contribution order in average total HQ value of mental is as follows: In general: Rice > Fish > Water > Veg. > Dust for Pb.
5. Generally metal HQ value follows the following order: 0.97(As) > 0.204(Cd) > 0.191(Pb).
6. Metal HQ contribution follows the following order in total HI value: As (69.87%) > Cd(19.67%) > Pb(10.31%).
7. In case of children population sub-group minimum, maximum and average HI value is 1.87, 4.042 and 2.36 respectively. As a result all children have in risk of the study area.
8. In case of children population sub-group minimum, maximum and average HI value is 0.747, 2.331 and 1.016 respectively. As a result four fifths of average population is at safe level and one fifth of average population is at risk level.
9. In case of children population sub-group minimum, maximum and average HI value is 0.766, 2.067 and 0.097 respectively. As a result four fifth of senior population is at safe level and one fifth of senior population is at risk level.

4.6 Reference

- [1] Human health effects of metals, U.S. Environmental protection Agency. Risk Assessment Forum. 2004, Pp-17.
- [2] Robert Goyer, Contributors: Mari Golub, Harlal Choudhury, Michael Hughes, Elaina Kenyon, Marc Stifelman, Issue paper on the Human health effects of metals. U.S. Environmental Protection Agency Risk Assessment Forum. August 19, 2004, Pp-17
- [3] . Li P. Y, Qian H., Human health risk assessment for chemical pollutants in drinking water source in shizuishan city, J. Environ. Health. Sci. Eng., Vol. 8, No. 1, 2011, pp. 41-48.
- [4] Jarup L, Hazards of heavy metal contamination. British Medical Journal, 2003, 68: 167-182.
- [5] Jasim Uddin Ahmad and Md. Abdul Goni, Heavy metal contamination in water, soil, and vegetables of the industrial areas in Dhaka, Bangladesh, Earth and Environmental Science Environmental Monitoring and Assessment, Volume 166, Numbers 1-4, pp. 347-357,
- [6] Juhasz A.L., Smith E., Weber J., Rees M., Rofe A, Kuchel T., et al. In Vivo Assessment of Arsenic Bioavailability in Rice and Its Significance for Human Health Risk Assessment. Environ Health Perspect, 3 August 2006, 114:1826-1831.
- [7] Avaiabe at: http://www.wpro.who.int/NR/rdonlyres/5FE0751B-0123-4BFC-AC2E FD51D6F36764/0/09_Chapter4Environmentaltrends.pdf [accessed september 20, 2011]
- [9] Mukesh K., Raikwar, Puneet Kumar, Manoj Singh and Anand Singh, Toxic effect of heavy metals in livestock health, Indian Veterinary World, January 2008, Vol.1(1):28-30, pp-28.
- [10] Avaiabe at: <http://www.tip2000.com/health/waterpollution.asp> [accessed September 20, 2011]

- [11] Avaiabe at: <http://emedicine.medscape.com/article/812953-overview> [accessed September 20, 2011]
- [12] Saha J.C., Dikshit A. K. and Bandyopadhyay M.A., Saha K. C., A review of arsenic poisoning and its effects on human health, Tropical Medicine, pp-2.
- [13] Avaiabe at: http://www.epa.gov/region04/foiapggs/readingroom/hercules_inc/a_review_of_arsenic_poisoning_and_its_effects_on_human_health_3v.pdf [accessed September 20, 2011]
- [14] Avaiabe at: <http://www.psr.org/environment-and-health/confronting-toxics/heavy-metals/> [accessed September 20, 2011]
15. Avaiabe at: <http://emedicine.medscape.com/article/814960-overview>, [accessed September 20, 2011]
16. Avaiabe at: <http://www.healthtestingcenters.com/heavy-metal-blood-test.aspx> [accessed September 20, 2011]
17. Manahan & Stanleye, Environmental chemistry, 2000, 7th edition, pp-613.
18. Avaiabe at: http://ec.europa.eu/environment/waste/studies/pdf/heavy_metalsreport.pdf [accessed September 20, 2011]
- [19] Violent, A. and Pigna, M, Competitive sorption of arsenate and phosphate on different clay minerals and soils. Soil Sci, 2002, pp. 1788-1796.
- [20] Avaiabe: <http://www.articlesbase.com/science-articles/levels-of-lead-and-cadmium-in-17-species-of-fresh-water-fish-of-bangladesh-3582236.html> [accessed September 20, 2011]
- [21] Williams PN, Islam MR, Adomako EE, Raab A, Hossain SA, Zhu YG, Feldmann J, Meharg AA. Increase in rice grain arsenic for regions of Bangladesh irrigating paddies with elevated arsenic in ground waters. Environ Sci Technol. 2006 Aug 15; 40(16):4903-8.
- [22] **Duruibe, J. O, Ogwuegbu, M. O. C and Egwurugwu, J. N**, Heavy metal pollution and human biotoxic effects, International Journal of Physical Sciences Vol. 2 (5), ISSN 1992 - 1950 © 2007 Academic Journals, May, 2007, pp. 112-118.
- [23] Ejaz ul Islam, Xiao-e Yang, Zhen-li He and Qaisar Mahmood, J Zhejiang Univ Sci B. Journal of Zhejiang University Science, 2007 January; 8(1): 1–13

- [24] Williams PN, Price AH, Raab A, Hossain SA, Feldmann J, Meharg AA, Variation in arsenic speciation and concentration in paddy rice related to dietary exposure. Environ Sci. Technol. 2005 Aug 1; 39 (15): 5531-40.
- [25] Jorhem L, Astrand C, Sundström B, Baxter M, Stokes P, Lewis J, Grawé KP. Elements in rice from the Swedish market: 1. Cadmium, lead and arsenic (total and inorganic). Food Additives and contaminations: Part A: Chemistry, Analysis, Control, Exposure, and Risk Assessment. 2008 Mar; 25(3): 284-92.
- [26] Gholam reza jahed khanikil and Muhammad ali zazoil, Cadmium and lead Contents in Rice (*Oryza sativa* in the North of Iran, International journal of agriculture &biology, Int.J.Agric. Biol., Vol.7,No.6, 2005, pp-1027, Available at [http:// www.ijab.org](http://www.ijab.org).
- [27] M. G. M Alam et al. The science of the total environment, 308, pp 83-96, 2003.

CHAPTER FIVE:

RECOMMENDATIONS FOR FUTURE WORK

5.1 RECOMMENDATIONS FOR FUTURE STUDY

Heavy metals such as arsenic, cadmium, lead, chromium and mercury are some of the important environmental pollutants. Their significant presence in the atmosphere, soil and water may cause serious problems to all organisms. Their bio-accumulation in the food chain can be treated as a potential threat to human health. So human health risk assessment involves a lot of variables. Besides these, there are some potential ways to take remedial measure against those. With regard to the considerations, the following studied could be carried out in future.

Studies related to remedy of environmental pollution:

1. In this investigation, we were confined in five natural organic fertilizers that the farmers of Bangladesh use frequently. There are some other similar fertilizers, namely, rotten weeds, baggage (Obtained from crushing sugarcane), poultry stool, rotten used tea leaves, etc. that could be studied for remedial measure against significant bio-accumulation of toxic elements in vegetables.
2. The mentioned study of remedial measure using natural organic fertilizers could also be applied to several types of corns like paddy, maize, wheat, pulse, etc.
3. It was observed that entrance of heavy metal pollutants in vegetable plants affected adversely the yield of vegetables. The same adverse effect was reported for rice by arsenic. This is a national as well as a global threat towards food security. Therefore, this study should be extended to affected areas extensively. That is, more site-specific studies should be carried out.
4. Some cations and anions may affect the uptake of toxic elements in

vegetables. To understand this, more studies might be carried out that include the influence of foreign cations such as Na^+ , Ca^{2+} , Fe^{3+} , etc. and foreign anions such as NO_3^- , SO_4^{2-} , PO_4^{3-} , etc. The kinetic studies related to these are also important.

Studies related to human health risk assessment:

1. Now-a-days, organic pesticides and insecticides like D.D.T., Organophosphates, N-Methyl Carbamates, Organochlorines, etc. are used to control pests and insects. These herbicides and fungicides enter into the human body through food chain. For prolonged exposure a considerable amount of the pesticides and insecticides retained in human body that might cause serious health hazards. For example, D.D.T. remains undegraded in human muscles for 40 years! With this regards the study may be extended.
2. British Geological survey in 2000 reports that toxic elements like fluoride, iodide, nickel, strontium, uranium, etc. are found in plenty in groundwater of Bangladesh. So Hazard Index (HI) for human health risk assessment should include those elements in addition.
3. In this investigation, two villages are considered for collection of some selected samples with the aim to assess the levels and determined hazard quotient and hazard index for human risk assessment of toxic elements (arsenic, cadmium and lead). Site-specific integrated risk assessment should be made covering all possible exposure pathways. This will provide the actual total risk due to exposure of the toxic elements.
4. More diet like soft drinks, beer, whisky, fast-foods, appetizers, etc. might contain toxic elements which may pose a risk to the human health. More study might be carried out with this regards.