Nutritional Status among the children of age group 5-14 years in selected arsenic exposed and non-exposed areas of Bangladesh

Dr. Md. Rezaul Karim

Faculty of Post- Graduate Medical Science and Research
University of Dhaka

Nutritional Status among the children of age group 5-14 years in selected arsenic exposed and non-exposed areas of Bangladesh.

Submitted to the University of Dhaka in accordance with the requirement for the degree of Doctor of Philosophy

By

Dr. Md. Rezaul Karim

MBBS, MPH

Faculty of Post- Graduate Medical Science and Research
University of Dhaka

Faculty of Post- Graduate Medical Science and Research
University of Dhaka

This thesis is submitted in fulfillment of the requirement for the degree of Doctor of Philosophy (PhD) under the Faculty of post graduate Medical Science and Research, University of Dhaka. This work has been carried out at the Department of Population Dynamics, NIPSOM. This is an original and innovative type of work and to the best of my knowledge it has not been done any where in Bangladesh.

Dr. Md. Rezaul Karim

Reg. No. Session: 94/ 2007-2008 (Puna)
University of Dhaka

Faculty of Post- Graduate Medical Science and Research

University of Dhaka

Certificate of thesis work

The thesis title 'Nutritional Status among the children of age group 5-14 years in selected arsenic exposed and non-exposed areas of Bangladesh' is submitted by Dr. Md. Rezaul Karim, Reg. No. 94/ 2007-2008 (Puna) in fulfillment of the requirements for the degree of Doctor of Philosophy (PhD) under the University of Dhaka. This is an original and innovative type of work and so far has not yet been done elsewhere in the country. The work is interesting to us and it being approved by:

CO-SUPERVISOR

Prof. Dr. Sk. Akhtar Ahmad
Director
National Institute of Preventive & Social Medicine (NIPSOM)
Mohakhali, Dhaka-1212

DEDICATE TO

MY RESPECTED SISTER,

BELOVED WIFE AND MY CHILDREN

Their sincere encouragement led me to build up my career as a Doctor and Scientist to serve the nation

INDEX

Title	Page no.
Acknowledgement	*i*
Abstract	*iii*
List of Table	*vi*
List of Abbreviation	*xi*
CHAPTER I	
1.1. Introduction	1
1.2. Background	13
Arsenic Chemistry and Biology:	14
Some common Arsenic compounds:	15
Environmental Transport and Distribution:	16
Uses of Arsenic	19
Homicidal use	20
Maximum Permissible Level in Drinking Water	21

Title	Page no.
Arsenic contamination	21
Sources and Reasons of Arsenic Contamination in Ground Water	23
Hydroxy mechanism	24
Arsenic in the human body	26
Exposure:	26
Mechanism	28
Excretion	29
Arsenic Toxicity in Human Body	30
Acute toxicity:	30
Sub-acute toxicity:	31
Chronic toxicity:	32
Symptoms of Arsenic Toxicity:	32
Stages of Clinical Features:	34
Pre-clinical stage:	34
Clinical stage:	35
Stages of complication:	35
Stages of malignancy:	35

Title	Page no.
Clinical manifestation of chronic arsenic toxicity:	35
Skin effects:	35
Gastro intestinal tract effects:	36
Renal system effects:	36
Cardiovascular system effects:	37
Nervous system effects:	37
Hematological effects:	38
Respiratory system effects:	38
Endocrine system effects:	38
Carcinogenicity:	38
Clinical Features of Arsenicosis	39
Pre clinical stage:	39
Initial Stage:	39
Second Stage:	39
Last Stage:	40

Title	Page no.
Clinical Features over Skin due to Chronic Arsenicosis	40
Melanosis:	40
Leucomelanosis:	41
Keratosis:	41
Hyper Keratosis:	41
Treatment and Management:	42
Chelation therapy:	42
Nutritious Diet and Vitamins:	42
Other symptomatic treatment:	43
Spirulina:	43
Social Aspects of Arsenic Contamination	43
Social instability:	43
Superstition:	44
Ostracism:	44
Marriage related problems:	44
Increased poverty:	45
Gender implications of arsenic contamination:	45

Title	Page no.
Measurement of Nutritional Status:	46
Clinical	48
Laboratory	48
Anthropometric	48
Anthropometric assessment	48
The advantages of anthropometry	49
Body mass index (BMI) for age and thinness	52
The use of interpretation of anthropometric measurements	52
Standard deviation or z - score classification	54
1. 3. Justification of the study	57
1.4. Title of the study:	59
1.5. Objectives of the study:	60
1.5.1. General objective:	60
1.5.2. Specific objectives:	60
1.5.3. Ultimate Objective	61
1.6. Research question:	61
CHAPTER II	
2. **REVIEW OF LITERATURE**	62

Title	Page no.
3.15. Key variables	109
3.16. Operational definitions	111
CHAPTER IV	
4. RESULTS	113
A. Socio-demographic characteristics of the respondents	113
B. Environmental condition	128
C. Consumption of water	132
D. Dietary habit	134
E. Nutritional status	139
CHAPTER V	
5. DISCUSSION	196
CHAPTER VI	
6. CONCLUSION	202
CHAPTER VII	
7. BIBLIOGRAPHY:	204
ANNEXURE	
Questionnaire	
Arsenic Map	

Acknowledgement

I express my gratitude to Almighty Allah who gave me the chance, strength and ability to complete the study.

I am very much grateful to Prof. (Dr.) Sk. Akhtar Ahmad, Head of the Department of Occupation and Environmental Health, NIPSOM for his valuable guidance, advice, blessings and co-operation without which completion of this study was not possible.

I express my deep gratitude to Professor (Dr). Md. Abdur Rahama PhD, Director of NIPSOM, for his sympathetic co-operation.

I gratefully acknowledge Professor (Dr). Md. Zaforullah Chowdhury PhD, Ex-Director of NIPSOM for allowing me to carry out this thesis work at NIPSOM.

I like to offer my sincere thanks and gratitude to my teacher, well wisher and guide Professor Md. Shahidullah, Professor of Bio-Statistics, BSMMU, Dhaka for his sincere and helpful advice during this study.

My sincere thanks are due to Dr. Deoraj Caussy (Harry), Environmental Epidemiologist, WHO; who helped me by quick releasing of the funds and giving me support so that I could conduct the study in time.

I am grateful to Mr. Md. Taufiqul Arif, Ag. WHO-EH scientist for his support and co-operation in releasing of fund for this study. I am very much grateful to Dr. Salamat Khandker, Medical Officer, WHO for his kind advice and continuous help and co-operation to complete the work. I offer my special thanks to Mr. Noor Ahmed and Mr. Nazrul Islam of Institute of Nutrition and

Food Science, Dhaka University; for their co-operation in collection and compilation of data. The students of Institute of Nutrition and Food Science, Dhaka University did hard job in the collection of data from the field. My sincere thanks are due to them.

My sincere thanks are due to Thana Health and Family Planning Officers of Sirajdikhan and Sonargaon thanas for their cordial continuous support during the conduction of the study.

I am very much grateful to the people of Sirajdikhan and Sonargaon for their kind help and sincere co-operation, which I shall ever remember.

I am extremely thankful to Dr. Md. Mahmudul Haque, Assistant Professor, Department of Community Medicine, NIPSOM for his untiring help during the different stages of this work.

My warmest thanks to Dr. Manzurul Haque Khan, Assistant Professor, Department of Occupation and Environmental Health, NIPSOM for his generous help, co-operation and support.

I offer my sincere thanks to all of my fellow colleagues working in NIPSOM for their co-operation and encouragement to furnish the work. I am very much grateful to my elder brother Mr. S. M. Jahangir for his managerial support in the data collection work.

Finally, I owe more than I can put into words to my wife, Hamida Karim, my daughter and son for their love, support and encouragement during my stretches of disappointment when I needed it most without which I could not have manage it in entirely.

ABSTRACT

A cross sectional study was conducted on 600 children of arsenic exposed (arsenic concentration of tube well water was above 50 µg /L) and non-exposed (arsenic concentration of tube well water was below 50 µg /L) areas in Bangladesh to find out any difference in the nutritional status. From both exposed and non-exposed areas children of 5-14 years of age, having negative examination report for helminthic infestation and whose guardians were willing to participate in the study were included as respondents. Where as those beyond the age limit and suffering from illnesses like (severe diarrhea, chronic dysentery, acute respiratory infection) were not included in this study. Information was collected on socio-demographic characteristics, duration of water use, daily consumption of water of children, arsenic level of tube-well water, other sources of water intake using pre tested questionnaire. Checklist was used for arsenic level of water, helminthic infestation of stool and anthropometric measurements of the study children.

The study groups (arsenic exposed and non-exposed) were found to be more or less similar in terms of age, gender, number of children (aged 5-14 years) in household, parental education, family type, father's profession, family size, monthly family income, housing condition and type of latrine use ($p > 0.05$).

Dietary history of children showed almost similar intake of carbohydrate, protein and fat in both the exposed and non-exposed groups. The total calorie intake was also found not to be significantly different between the two groups of children. Nutritional status of children was assessed by z-scores of weight for age, height for age and weight for height and also using 5^{th} and 85^{th} percentiles of the body mass index (BMI) for age. Thinness (low BMI for age) was found more among the children of exposed area (49%) than that of non-exposed area (38%). Comparatively children with normal BMI was found to be more in non-exposed area than in exposed area and the difference was found to be significant (χ^2=10.600, p <0.01). The weight for age z-score measurements showed that the children in the exposed area were more underweight than those of the non-exposed area (p< 0.05). Height for age z-score measurements showed that the children in the exposed area were more stunted than those of non-exposed area (p <0.05). Weight for height z–score measurements showed that the children in the exposed areas were more under weight than those of non-exposed area (p <0.05). In this study type of the family (χ^2 =6.516, p < 0.05), number of family (χ^2 =4.688, p =.021) and type of latrine (χ^2 =21.303, p =0.006) had significant association with BMI of children but no association with monthly income (χ^2 =1.873, p = 0.759), occupation (χ^2 = 5.965, p= 0.427) and type of housing (χ^2 =5.795, p = 0.215) was found. The mean BMI of the children was found to be

significantly higher (p= 0.012) in non-exposed children (14.874±2.167) than that of exposed children (14.423±2.208). When compared after grouping the nutritional status (based on BMI) as underweight and normal or overweight an association between nutritional status and arsenic exposed was observed ($p < 0.01$). Such association was observed among children aged 8-10 and 11-14 years but no such association was observed among those who were between 5-7 years of age. These finding suggest that exposure of arsenic might have negative impact on nutritional status among the older age group of children. Chi square tests were performed to see the associations between exposure and effect with confounder's: monthly income and family size. Adjusting the influences of the control variables it was seen that under nutrition was more among the study children with arsenic exposure than the arsenic non-exposed children. The exposure-effect was found to be significantly associated ($p< 0.01$).

Binary logistic regression analysis suggest that malnutrition status was 7.2 times higher among the exposed children in comparison to that in non exposed children and this was positively correlated with frequency intake of pulses, wet rice and number of glasses of water consumed per day. However, the study concludes that nutritional status of the exposed children was significantly lower than that of non-exposed children ($p< 0.05$). The study further suggests that arsenic exposure had a negative impact on the nutritional status of children.

LIST OF TABLES

TITLE	Page no.
Table 1.1: Distribution of the families according to no of 5-14 years aged children and sex of study children	113
Table 1.2: Distribution of the children by age sex and type of subject	115
Table 1.3: Distribution of the children by age and sex	117
Table 1.4: Distribution of the respondents by age of the parents	118
Table 1.5: Distribution of the respondents by sex	120
Table 1.6: Distribution of the respondents by father's level of education	121
Table 1.7: Distribution of the respondents by mother's level of education	122
Table 1.8: Distribution of the respondents by type of family	124
Table 1.9: Distribution of the respondents by main profession of the household	125
Table 1.10: Distribution of the respondents by family size	126
Table 1.11: Distribution of the respondents by monthly family income	127
Table 2.1: Distribution of the respondents by housing condition	128
Table 2.2: Distribution of the respondents by type of latrine used	129

TITLE		Page no.
Table 2.3:	Distribution of the respondents by sources of drinking and cooking water	130
Table 2.4:	Distribution of the respondents by media exposure	131
Table 3.1:	Distribution of the respondents by consumption of water	132
Table 4.1:	Distribution of the respondents by dietary habit	134
Table 4.2:	Nutrients taken per day by the study children	136
Table 4.3:	Minerals and vitamins taken per day by the study children	137
Table 4.4:	Correlation of BMI with principal nutrients taken by the study children	138
Table 5.1:	Age and sex wise mean distribution of weight and height of the children	139
Table 5.2:	Age wise mean distribution of weight and height of the children (boys)	140
Table 5.3:	Age wise mean distribution of weight and height of the children (girls)	142
Table 5.4:	Distribution of the children by age and height for age	144
Table 5.5:	Distribution of the children by age and weight for age	145
Table 5.6:	Distribution of the children by age and weight for height	146
Table 5.7:	Distribution of the children by sex and height for age	147
Table 5.8:	Distribution of the children by sex and weight for age	148

TITLE	Page no.
Table. 5.9: Distribution of the children by sex and weight for height	149
Table 5.10: Distribution of the children by nutritional status	150
Table 5.11: Distribution of the nutritional status of the children according to age group	152
Table 5.12: Mean BMI of the children	153
Table-5.13: BMI of the study children according to income and occupation of the main earning member	154
Table-5.14: BMI of the children according to family type and number of family members	155
Table-5.15: Children's BMI according to type of house they lived in and type of the latrine used by the families	157
Table 5.16: Nutritional status of the children by height for age z-scores according to arsenic exposure	158
Table 5.17: Nutritional status of the children by weight for age z-scores according to arsenic exposure	159
Table 5.18: Nutritional status of the children by weight for height z- scores according to arsenic exposure	160
Table 5.19: Nutritional status of the children by combined z- scores (HAZ, WAZ & WHT) according to arsenic exposure	161
Table 5.20: Nutritional status of the children by BMI according to arsenic exposure	162
Table 5.21: Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI according to arsenic exposure	163

	TITLE	Page no.
Table 5.22:	Nutritional status of the children by height for age z-scores according to arsenic exposure and monthly income of the families	164
Table 5.23:	Nutritional status of the children by weight for age z-scores according to arsenic exposure and monthly income of the families	166
Table 5.24:	Nutritional status of the children by weight for height z- scores according to arsenic exposure and monthly income of the families	167
Table 5.25:	Nutritional status of the children by combined z-scores (HAZ, WAZ & WHT) according to arsenic exposure and monthly income of the families	169
Table 5.26:	Nutritional status of the children by BMI according to arsenic exposure and monthly income of the families	171
Table 5.27:	Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI according to arsenic exposure and monthly income of the families	173
Table 5.28:	Nutritional status of the children by height for age z-scores according to arsenic exposure and family size	175
Table 5.29:	Nutritional status of the children by weight for age z-scores according to arsenic exposure and family size	176
Table 5.30:	Nutritional status of the children by weight for height z- scores according to arsenic exposure and family size	177
Table 5.31:	Nutritional status of the children by combined z-scores (HAZ, WAZ & WHT) according to arsenic exposure and family size	178

	TITLE	Page no.
Table 5.32:	Nutritional status of the children by BMI according to arsenic exposure and family size	179
Table 5.33:	Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI according to arsenic exposure and family size	180
Table 5.34:	Nutritional status of the children by height for age z-scores according to arsenic exposure, monthly income of the families and family size	181
Table 5.35:	Nutritional status of the children by weight for age z-scores according to arsenic exposure, monthly income of the families and family size	183
Table 5.36:	Nutritional status of the children by weight for height z- scores according to arsenic exposure, monthly income of the families and family size	185
Table 5.37:	Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) according to arsenic exposure, monthly income of the families and family size	187
Table 5.38:	Nutritional status of the children by BMI according to arsenic exposure, monthly income of the families and family size	189
Table 5.39:	Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI according to arsenic exposure, monthly income of the families and family size	191
Table 5.40:	Correlates of malnutrition among the children: Binary logistic regression analysis	194

LIST OF ABBREVIATION

ATSDR: Agency for Toxic Substance and Disease Registry

ARI: Acute Respiratory Tract Infection

As: Arsenic

AFS: Atomic Fluorescence Spectrometry

BAMWSP: Bangladesh Arsenic Mitigation and Water Supply Project

BBS: Bangladesh Bureau of Statistics

BC: Before Christ

BGS: British Geological Survey

BITPERHT: Bangladesh Institute of Research for Promotion of Essential and Reproductive Health and Technologies

BMI: Body Mass Index

BMRC: Bangladesh Medical Research Council

CNU: Children's Nutrition Unit

DPHE: Department of Public Health Engineering

DOEH: Department of Occupational and Environmental Health

DMA: Dimethyl Arsenic Acid

GDP: Gross Domestic Product

GR: Glucocoticoid Receptor

GPs: Gram Panchyets

HAZ: Height for Age z-score

HCL: Hydrochloric Acid

IARC: International Agency for Research on Cancer

ICDDR, B: International Centre for Diarrhea Disease Research, Bangladesh

ILO: International Labor Organization

JOPSOM: Journal of Preventive and Social Medicine

MMA: Monomethyl Arsenic Acid

MUAC: Mid Upper Arm Circumference

NAD: Nicotinamide Adenine Dinucleotide

NCHS: National Centre for Health Statistics

PEM: Protein Energy Malnutrition

NIPSOM: National Institute of Preventive and Social Medicine

SOES: School of Environmental Studies

STM: Saha School of Tropical Medicine

SPSS: Statistical Package for Social Science

SD: Standard Deviation

TW: Tube well

TWA: Time Weighted Average

WHO: World Health Organization

WAZ: Weight for Age z-score

WHZ: Weight for Height z-score

µg/L: Microgram per Liter

UN: United Nation

UNICEF: United Nation International Children Emergency Fund

CHAPTER I

INTRODUCTION

1.1. INTRODUCTION

The threat to public health presented by arsenic contamination of drinking water has attracted much attention since the 1990s, largely due to the scale of the problem in Bangladesh, describe as the " the largest poisoning of a population in history" [1]. Water is the most abundant resource in Bangladesh. But arsenic concentration of ground water in our country has become a matter of serious concern. It is the most extensive environmental disaster of the twentieth century. The problem of arsenic contamination of ground water in the subcontinent was recognized in West Bengal, India in 1983 [2, 3, 4, 5]. Though Bangladesh shares a common border with India and similar geomorphologic features in West Bengal, the possibility of having the same problem in Bangladesh was not anticipated by any of the stake holders until 1993 when WHO raised the possibility of arsenic contamination in tube well water in areas adjoining West Bengal [6, 7, 5, 8]. The government of Bangladesh officially recognized the existence of the problem following detection of arsenic contaminated tube wells by the Department of Public Health Engineering (DPHE) in 1993 in the village Chamagram of Baroghoria union under the district of Nawabgonj and reported that water samples from 4 tube wells contained arsenic at levels of 50 μg/L (Bangladesh standard)[1, 4, 5, 9, 10]. However, the Department of Occupational and Environmental

Health (DOEH), National Institute of Preventive and Social Medicine (NIPSOM) identify the first 8 patients of arsenicosis in 1994 in the same area [5, 12, 13].

In Bangladesh installation of tube wells was undertaken with an aim to provide bacteriological safe water to the vast population of the country. About 8 – 12 million tube wells (hand pumps) had been sunk in Bangladesh which ensured access to safe water for it's'97% population [11]. Unfortunately, the identification of arsenic in tube well water put its people in dilemma. Since the detection of arsenic contamination in ground water in Bangladesh the arsenic contamination problem is rapidly emerging and becoming a major public health problem. Till 1995, the arsenic contamination situation in West Bengal of India was believed to be the greatest arsenic disaster in the world. However, in the following years the arsenic contamination of ground water in Bangladesh started unfolding rapidly and the situation has been considered as the largest environmental disaster in the world [14]. In December 1995 it was estimated that 10 million people were at risk of arsenic exposure through tube well water [14]. Now it has been reported that about 30 million to 50 million people are at risk of arsenic exposure [3, 10, 15, 16]. The arsenic contamination in the tube well water has been detected in 61 out of 64 districts [17]. Bangladesh Arsenic Mitigation and Water

supply Project so far has screened tubewells in 271 Upazilla out of 490 Upazilla. Out of 4,946,933 tubewells located in the 271 Upazilla, 29.2% were found to be arsenic contaminated (>50 µg/L) and in 8,540 villages the arsenic contaminated tubewells were more than 80% [17]. In 255 Upazilla, so, far, 38,118 arsenicosis patients had been detected [17]. It has been estimated that about 29 million people in Bangladesh are exposed to drinking water with arsenic exceeding Bangladesh standard 50 µg/L [18, 19].

Not only in Bangladesh but also in many other countries of the world including India, Nepal, Argentina, Thailand, Chili, Finland and Hungry a growing number of people has been identified exposed to high arsenic through drinking water [1,15,19,20,21,21,23].

West Bengal, INDIA

West Bengal is one of the 29 states in India. The area of West Bengal is 89193 sq. km having a population of about 80.1 million. Its administrative structure consists of several districts: each district has several blocks/police stations; each block has several Gram Panchayets (GPs), which are cluster of villages. There are 19 districts, 341 blocks and 37910 villages in West Bengal.

Arsenic crisis in India dates back to as early as 1976 when a preliminary survey on arsenic in dug wells, hand pumps and spring water from Chandigarh and

different villages of Punjab, Patiala, Haryana and Himachal Pradesh in northern India was reported. Officially, arsenic poisoning in West Bengal was first diagnosed by a dermatologist K.C. Saha of School of Tropical Medicine (STM), Kolkata to an outdoor patient of village Ramnagar of Baruipur police station in the district of South 24-Parganas on 6th July, 1983.

Groundwater arsenic contamination status of all 19 districts of West Bengal classified according to the arsenic concentrations found into three categories: Severely affected, mildly affected, and arsenic safe. Nine districts (Malda, Murshidabad, Nadia, North-24-Parganas, South-24-Parganas, Bardhaman, Howrah, Hoogly and Kolkata), where more than 300 μg/L arsenic concentrations were found are categorized as severely affected.

The five districts (Koch Bihar, Jalpaiguri, Darjiling, North Dinajpur and South Dinajpur) where the contaminated tube wells show arsenic concentrations mostly below 50 μg/L (only a few above 50 μg/L but none above 100 μg/L), termed as mildly affected.

The rest five districts (Bankura, Birbhum, Purulia, Medinipur East and Medinipur West), where all the recorded concentrations were below 10 μg/L termed as unaffected or arsenic safe.

People screened for arsenic patients in different districts in West Bengal including children (preliminary survey) were 96,000. Number of adults screened for arsenic patient was 82,000. Number of registered patients with clinical manifestations was 9,356 (9.7%). Number of children screened for arsenic patient was 14,000. Number of children showing arsenical manifestation was 778 (5.6%) [24].

NEPAL

In Nepal, topographically divided in to three regions, the mountainous, hilly and Terai regions. The Terai region is the lowland of Nepal, covering 17% of the total land area of Nepal. About 47% of the population of Nepal resides in the Teari regions. Ninety percent of the populations living in this region use ground water drawn from shallow tube wells. In Nepal 30% of the 200,000 shallow tube wells in the country might have arsenic concentration exceeding 50μg/L. In Nepal it was estimated that one million people are likely to be exposed to arsenic through drinking contaminated water [25].

Arsenic increases the risk of various cancer, and is also associated with may others non-communicable disease such as diabetes, hypertension and other cardiovascular events. Arsenic may have adverse reproductive effects in

humans, reproductive outcomes of arsenic exposure in humans included birth weight, gestational age, placental weight, disorders and others affects [26].

Chronic exposure to arsenic cause dermatologic, neurological, vascular carcinogenic effects and also other organ of system (kidney, liver, urinary bladder). Exposure to arsenic from drinking water increase the risk of skin, lung and bladder cancer possibly that of other site also [1,15,27,28,29,30,31,32,33,34,35,45,70,249]. In a report, WHO has predicted that in most of the southern part of Bangladesh almost 1 in 10 adults' deaths will be a result of cancer triggered by arsenic poisoning in the next decade [10]. Chronic exposure to arsenic through the drinking water may cause severe toxic effects even cancer in human health. It may affect all the organs and systems of the human body [29, 36, 37]. In Bangladesh the skin manifestations of the chronic toxicity are the prime features [8,11,14,38,39,40,41,42,43,44]. In addition to the melanosis, leucomelanosis, bilateral palmo-plantar keratosis and hyperkeratosis [32], non-pitting pedal edema, gangrene of lower limb (black foot disease), hepatomegaly, precancerous dermal lesions, and skin cancers have been detected among the arsenicosis patients [15,27,35,45,46]. Moreover, significantly higher rates of adverse pregnancy outcomes in terms of spontaneous abortion, stillbirth and birth rates [27,47], bladder cancer, hypertension [49], stomach and intestinal cancer [33], anemia and peripheral

neuropathy and respiratory problems [46,50] have also been observed among the arsenic exposed population in Bangladesh [1,26,179]. The clinical manifestation due to arsenic toxicity develop very insidiously after 6 months to 2 years or more depending on the amount of arsenic intake [33,34,51]. Arsenic was reported to be transmitted to the body of the child in mother's womb through placenta and breast milk [15, 23]. Chronic arsenic exposure may increase the risk of death and infant mortality [52]. Infants and Children are considered it be more susceptible to the adverse effects of arsenic exposure. Studies have shown that children are at high risk of arsenic exposure [27]. It has been observed that person taking arsenic contaminated water for 4-6 years may develop arsenicosis [31, 33, 34, 53, 227]. The youngest reported arsenicosis patient in Bangladesh is 4 years old [15].

Chronic arsenic toxin effects in children have been well documented [54, 55, 56]. The effect of arsenic contamination on children has not been widely reported and in addition very little is known about the relationship between arsenic poisoning, children's growth and nutrition. Study conducted was a follow-up infant exposed to arsenic contaminated milk in Japan. The infants, the victims of human error, were exposed to arsenic 1.3 to 3.6 mg /day, which caused sub acute poisoning and 130 deaths. The survivors, who were examined about 15

years after the exposure, were on average, significantly shorter than the non-exposed children [57].

A study carried out in Bangladesh provided evidence that children chronically exposed to arsenic are more stunted in growth than non exposed children [27]. Undernourishments may affect the metabolism of inorganic arsenic and the development of subsequent skin cancers [58, 59]. There is evidence that people of poor socio-economic condition are more prone to arsenicosis. Therefore, it is assumed that poor nutritional status of the people may have more susceptibility to arsenic toxicity [32, 53, 60, 61].

Zhang 1996 showed that persons having accumulation of arsenic in their serum loose their normal nutritional status gradually. Several studies observed the variability of development of arsenic toxicity among the arsenic exposed people with their nutritional status and socio-economic level. Poor nutritional status might increase the susceptibility of human body to arsenic toxicity, leading to decrease methylation of arsenic and increase tissue deposition of arsenic [9, 29].

A case control study concluded that low intake of animal protein, folate and fiber may increase susceptibility to arsenic caused skin lesion [62].

Nutritional status and vitamin A and beta carotene levels are reported to be determinants of the susceptibility of individual to arsenic related health problems [63].

Nutrition plays a decisive role in the prevention or the onset of arsenic related ailments. Low dietary intake of protein and micronutrients (calcium, selenium, vitamins) increase vulnerability to arsenic related diseases. The reason could be that nutritional deficiency might result in slow elimination of arsenic from the body [61,249,250,251].

Undernourishment status, indicated by the long term consumption of dried sweet potato chips and / or low serum level of antioxidant vitamins, has been found to increase the risk of arsenic induced health hazards [59, 62, 63, 64]. Poor arsenic methylation capability, indicated by the low percentage of dimethylarsinic acid in total metabolites of inorganic arsenic in urine, has been documented to significantly associate with the development of arsenicosis [35].

Study conducted in Ronpiboon in Thailand to find out the deficit of intellectual ability in children having high arsenic level in hair. Further the study revealed that high arsenic levels in hair of the children were related to impairment of visual perception [65].

Study Picciotto 2000 conducted in Sweden and in the USA, revealed the strongest evidence regarding synergistic relations on lung cancer and the combined effects of smoking and either occupational or environmental exposure to arsenic. The study clearly indicates that the joint effects of smoking and exposure to arsenic from the industrial sources are greater additive [66].

In humans, there is considerable inter individual variability in the proportion of inorganic arsenic, MMA and DMA excretion in urine. Inorganic arsenic and MMA is enzymatically methylated via one carbon metabolism, a biochemical pathway dependent on folate for recruitment of methyl groups. One carbon metabolism also requires vitamins B_{12} (cobalamin) and B_6 as cofactors. Animal studies have suggested that folate nutritional status may influence arsenic excretion [67].

Smith 2006 in his study in Antofagasta of Chile for the period of 1958 to 1970 in a very high arsenic exposure population, found that exposure to arsenic in drinking water during early childhood or in utero has pronounced pulmonary effects greatly increasing subsequent mortality in young adults from both malignant and nonmalignant lung disease [58].

Kwok 2006 conducted study on reproductive health outcomes in pregnant women. The study shows that there was a small but statistically significant

association between arsenic exposure and birth-defects (odds ratio= 1.005, 95% confidence interval 1.001-1.010), others outcomes such as stillbirth, low birth-weight were not associated with arsenic exposure [68].

While other study found that arsenic affects the outcome of pregnancy with higher rate of spontaneous abortion, stillbirth premature birth in exposed mothers as well as lower birth weight [47, 69].

Different studies found that impact of arsenic exposure to the children [15, 70, 71, 72, 73]. It was found that chronic arsenic exposure may increase the risk of infant motility, affects the outcome of pregnancy with high rate of spontaneous abortion, still birth and premature birth [18, 47, 68, 74, 75, 76, 178]. In Bangladesh the youngest arsenicosis case reported was of age two years and another was of age four years [15, 46].

Bangladesh is a developing country with 150 million people. It belongs to the high-density populated countries in the world with low literacy, very low socio-economic condition and prevalence of morbidity due to malnutrition is 3.32 per 1000. (male: 2.42, female 4.27) [77]. The children of 5-14 years comprise 26.51% of the total population [77]. A vast majority of our population is children, they are the future generation of the Nation they need a healthy environment for their growth and development. So, that in future they become healthy sound

generation to run the country properly. But unfortunately the children are also likely to be exposed to arsenic through drinking water. And still it is not clear that whether arsenic causes any particular effect among them. Studies report that either arsenic causes malnutrition among the children or malnutrition cause more susceptible to arsenic toxicity and amongst our children such study or information could not be collected conclusively.

So, it is an important task for the researcher to explore the state of nutrition and arsenic toxicity among them in order to take appropriate measures for their proper development.

There are many parameters to measure the nutritional status of children. But the most simple and significant parameters are height for age, weight for age, weight for height, and body mass index (BMI) [78]. In this study these parameters had been used to measure the nutritional status of the children with an aim to find out whether arsenic toxicity influence nutritional status of the children or not.

1.2. BACKGROUND

Chronic exposures to arsenic contaminated drinking water for prolonged time starts manifesting a condition known as 'arsenicosis'. It may take 2 – 10 years or more, but generally it appears more in between 4 – 6 years [15,34,51,79]. Occurrence of arsenicosis within 6 – 8 months is also reported [80]. Types of arsenic ingested, level of exposure, body immunity, food habit determine onset of clinical manifestations. Chronic arsenic exposure may result toxicity and may develop various symptoms affecting different organ systems of body such as skin, nervous system, respiratory system and metabolic system [7, 35,61,81,82].

According to WHO, Arsenicosis is the chronic health condition arising from prolonged ingestion of arsenic above the safe dose for at least six months, usually manifested by characteristic skin lesions of melanosis and / or keratosis with or without the involvement of internal organs [83]. Chronic arsenic poisoning may also occur in workers who are exposed to excessive concentration of air borne arsenic compound for a prolonged time. But the common route for chronic toxicity occurs due to prolonged ingestion of arsenic through water, symptoms are partly different from those after inhalation exposure [15].

Arsenic Chemistry and Biology:

Arsenic (As) is a metalloid element, this is brittle in nature and gray or tin white in color [15, 84, 85, 86, 87, 88]. It is a member of group V element with atomic number 33 and atomic weight 74.92. Its specific gravity is 5.73, melting point 817^0 C and sublimes at temperature of 613^0 C. Arsenic exhibits a broad range of chemical reactivity with ability to form alloys with other elements and covalent bonds with carbon, hydrogen and oxygen. It participates readily in oxidation-reduction, methylation-demethylation and acid-base reactions [7, 87]. Arsenic burns in air giving off an odor of garlic and dense white fumes of arsenic trioxide (As_2O_3). Arsenic trioxide is the most important commercial compound of arsenic. The solubility of arsenic trioxide in water is fairly low, about 2% at 25^0 C and 8.2% at 98^0C [15,87,88,89]. It is highly soluble in both hydrochloric acid and alkali [49, 81]. Arsenic occurs naturally in all environmental media: air, soil and water and usually presents in the form of compounds with sulfur and many metals (copper, gold, cobalt, lead, zinc) [8,81,87,88,89,90,91]. Arsenic is present in more than 200 mineral species, the most common of which is arsenopyrite [49,87,88,92]. Chemically arsenic compounds mainly are of two types inorganic and organic [87, 88, 89]. Inorganic arsenic of geological origin is found in ground water used as drinking water in several parts of the world for example in Bangladesh.

B. Organic Arsenic: [92]

Monomethyl arsenic acid	Methane arsenic acid	Less toxic than inorganic form
Dimethyl arsenic acid	Cacodylic acid	
Arsenobetaine		
Arseno choline		

Environmental Transport and Distribution:

Arsenic is a ubiquitous element that is found in the atmosphere, in the aquatic environment, in soils and sediments and in organisms. The arsenic contents of the earth's crust is 1.5 to 2 mg/kg, it ranks 20th in abundance in relation to other elements [23, 61, 92]. Arsenic is associated with igneous and sedimentary rocks, particularly with sulfide ores. Natural phenomena such as weathering, biological activity, volcanic activity and anthropogenic activity (e.g, fossil fuel combustions, smelting operations) are responsible for the emission of arsenic into the atmosphere [15, 89]. Arsenic is mainly transported in the environment by water [95]. Sedimentation of arsenic in association with iron and aluminum may sometimes be considerable. In oxygenated water, arsenic usually occurs as arsenate. But under reducing condition for instance, in deep well water, arsenite predominates. Methylation of inorganic arsenic to methyl and dimethyl arsenic acid is associated with biological activity in water [96, 97]. Air borne concentrations

of arsenic in urban areas may range from about 0.002 mg/m^3 to .03 mg/$m.^3$ Mean total arsenic concentration in air from remote and rural areas range from 0.02 to about 4 ng/m^3, much higher concentrations (>1000 ng /m^3) have been measured in the vicinity of industrial sources, although in some areas this is decreasing because of pollution abatements measures. Concentration of arsenic in open ocean seawater is typically 1-2 μg/ liter. Arsenic is widely distributed in surface freshwaters, and concentration in rivers and lakes are generally below 10 μg/ liter, although individual may range up to 5 mg/liter near anthropogenic sources. Arsenic levels in groundwater average 1-2 μg/ liter except in areas with volcanic rock and sulfide mineral deposits where arsenic levels can range up to 3 mg/liter. Mean sediment arsenic concentrations range from 5 to 300 mg/kg, with the higher levels occurring in areas of contamination. Background concentrations in soil range from 1 to 40 mg/kg, with mean values often around 5mg/kg. Naturally elevated levels of arsenic in soils may be associated with geological substrate such as sulfide ores. Anthropogenically contaminated soils can have concentrations of arsenic up to several grams per 100 ml. In oxygenated soil inorganic arsenic is usually present as pentavalent form (arsenate). In the soil, biomethylation of inorganic arsenic occurs and results in the release of methylarsines into the air [1, 61, 96].

Arsenic concentrations in freshwater and terrestrial biota are usually less than 1mg/kg (fresh weight). Terrestrial plants may accumulate arsenic by root uptake from the soil or by adsorption of airborne arsenic deposited on the leaves. Arsenic levels are higher in biota collected near anthropogenic sources or in areas with geothermal activity. Some species accumulate substantial levels, with mean concentration of up to 3000 mg/kg at arsenical mine sites. Arsenic levels in natural waters are usually low (a few μg / liter), there are several areas in the world where humans consume drinking water containing > 100 μg As / liter resulting from natural geochemical activities [9,23,85,92].

Marine organisms normally contains arsenic residues ranging from < 1 to more than 100mg/ kg, predominately as organic arsenic species such as arsenosugars and arsenobetaine (intervertbrates and fish). Some marine organisms have been shown to transform inorganic arsenic into more complex organic compounds, such as arsenobetaine, arsenocholine and arsonium phospholipids [23, 92].

Uses of Arsenic

Arsenic compounds are mainly used in agriculture, forestry and industrial processes. Arsenic (III) oxide is used in manufacture of agricultural chemicals (pesticides), glass and glassware, industrial chemicals, copper and lead alloys and pharmaceuticals. In agriculture, arsenic compounds such as lead arsenate, coppreracetoarsenite, sodium arsenite, calcium arsenate and organic arsenic compounds are used as pesticides[85, 88, 89, 98]. Substantial amount of methyl arsenic acid and diethyl arsenic acid are used as selective herbicides. Chromate copper arsenite, sodium arsenate and zinc arsenate are used as wood preservatives. Some phenyl arsenic compounds such as arsenic acid are used as feed additives for poultry and swine. Small amount of arsenic compounds continue to be used as drugs in some countries. As medicine, arsenic is used since the fifth century BC, when Hippocrates recommended the use of an arsenic sulfide for the treatment of abscess. Arsenic preparations were used for the treatment of skin disorder, tuberculosis, leukemia, asthma, leprosy, syphilis, amoebic dysentery etc. Homeopaths are also using arsenic as drug [51, 85, 89,91,98,99,100,101].

Homicidal Use

The time of Roman Empire through the Middle Ages and the Renaissance, arsenic was the king of poisons. A Greek physician in the court of the Roman emperor Nero, describe arsenic as a poison in the first century. During the fourth century BC, the Romans consider use of poisons in politics; reported in history. Among the most infamous of poisoners was a woman known as Toffana who made arsenic laced cosmetic and instructed women on their use. In Italy, during the middle ages, the most widely accused of poisoners were the Borgias, Pope Alexander VI, and his son, Cesare. There have long been rumors to suggest that the final agonies of Napoleon Bonaparte in 1821 were due to the repeated administration of arsenic by someone in his cortege. Claire Booth Luce was the United State ambassador to Italy, she become victim of arsenic poisoning [102,103].

In the middle ages, arsenic was commonly used as a suicidal and homicidal agent. A well-known case in Sweden is King Erik XIV, the eldest son of Gustav Vasa, who died in 1577, after a week of chest and stomach pains, it was probably Erik's brother Johan III who ordered the assassination. Much later, the French Emperor Napoleon Bonaparte was sent into exile on the island of St. Helena, following his defeat at the battle of Waterloo. Upon the death of Napoleon in 1821, it was announced that he died of stomach cancer, but many

minerals are formed. Arsenic minerals are formed as a part of this geological process in the magic rocks or intrusions. So, the primary sources of arsenic are the igneous-metamorphic complex rock bodies. The earth surface is experiencing a complex system of weathering, erosion, transportation and sedimentation. As a result of these geological processes along with other elements, the arsenic minerals and arsenic are experiencing all those processes. Bangladesh is situated in the lower end of the three great river systems, the Ganges, the Brahmaputra and the Meghna [8, 93,109,110]. The catchment areas of the three great river systems (Ganges, Brahmaputra and Meghna) is about 1.5 million square kilometers (600,000 sq. miles) comprising the Himalayan mountain systems, the Indian Shield and the Shillong Plateau and the great Genetic Plains. Due to geologic conditions, the sediments produced in the catchment areas are very high. The sediment carried annually by these three great river systems has been estimated to be about 2.4 billion tons, which are passed through the rivers in Bangladesh.

As because a large part of the country is under normal inundation and affected by major floods, the sediments are being deposited in the fluvio-deltaic areas. It is conceived that these sediments that contain arsenic have been deposited throughout the geologic time. The catchment areas expose Archean basement

rocks, Paleozoic (Gondwana) rocks, Jurassic to creteceous Trap rocks (Deccan and Rajmahal) and all the rocks ranging in age from Paleocene to the recent age [32,93,100,111,112,113, 118,164,253].

Bangladesh is mostly covered by Holocene and recent sediments. Most of the sediments in the Bengal basin may contain arsenic that has been deposited along with other sediments laid down by the three great river systems throughout the geologic time [100].

The Gangetic alluvium comprises the districts of Kushtia and Jessore and parts of Rajshahi, Pabna, Faridpur, Khulna, Barisal and Dhaka. It covers an area of 27,000 sq. km. The soils are moderately fertile and texture of soils ranges from clay loam to sandy loam. Brahmaputra alluvium comprises the districts of Comilla, Noakhali, and Sylhet except hilly areas and parts of greater Mymensingh, Dhaka and Chittagong. It occupies an area of 40,000 sq.km the dominating texture for the soil is sandy loam, the soils are naturally fertile [24,100,107,127].

Sources and Reasons of Arsenic Contamination in Ground Water

Arsenic contamination in ground water in West Bengal has been found in 7 districts. Most of the arsenic contaminated areas of West Bengal are in the region of alluvial formation. The source of arsenic contamination in these 7

districts is noted to be geological. Bangladesh is geographically attached with West Bengal and having similar aquifers and socioeconomic background. The alluvial formation in Bangladesh has close geological affinity as those encountered in West Bengal. In Bangladesh, the ground water is also been drawn through deep tube well vigorously for irrigation purposes particularly during drought when riverbeds become dry. The studies carried out in West Bengal clearly indicate that in the region of alluvial sediment there is existence of pyrite and this pyrite is rich in arsenic. Due to heavy ground water withdrawal and fluctuation of water table from pro monsoon to post monsoon and also due to thousands of bore holes, the underground aquifer is aerated and the pyrite decomposes, the acid released due to this decomposition leaches out arsenic from pyrite [24, 93,100].

Hydroxy Mechanism

In nature, arsenic bearing minerals undergo oxidation and release arsenic to water. This could be one explanation for the problems of arsenic in the groundwater of West Bengal and Bangladesh. In these areas the groundwater usage is very high. It has been estimated that there are about 4-10 million tube wells in Bangladesh. The excessive withdrawal and lowering of the water table for rice irrigation and other requirements lead to the exposure and subsequent

oxidation of arsenic-containing pyrite in the sediment. As the water table recharges after rainfall, arsenic leaches out of the sediment into the aquifer.

However, recent studies seem to favor the reduction of Fe/As ox hydroxides as the source for arsenic contamination in groundwater [114,115,116]. Arsenic forms co-precipitates with ferric oxyhydroxide. Burial of the sediment, rich in ferric oxyhydroxide and organic matter, has led to the strongly reducing groundwater conditions. The process has been aided by the high water table and fine-grained surface layers which impede the penetration of air to the aquifer. Microbial oxidation of organic carbon has depleted the dissolved oxygen in the groundwater. The highly reducing nature of the groundwater explains the presence of arsenite (<50%) in the water. The "pyrite oxidation" hypothesis is therefore unlikely to be a major process, and the "oxyhydroxide reduction" hypothesis is probably the main cause of arsenic contamination in groundwater [116,117]. Although the oxyhydroxide reduction hypothesis requires further validation, there is no doubt that the source of arsenic in West Bengal and Bangladesh is geological, as none of the explanations for anthropogenic contamination can account for the regional extent of ground water can account for the regional extent of ground water contamination. During the past 30 years, the use of phosphate fertilizers has increased threefold in this region. The wide

spread withdrawal of groundwater may have mobilized phosphate derived from fertilizers and from the decay of natural organic materials in from fertilizers and from the decay of natural organic materials in shallow aquifers. The increase in phosphate concentration could have promoted the growth of sediment biota and the adsorption of arsenic from sediments, and the combined microbiological and chemical process might have increased the mobility of arsenic [117].

Arsenic in the Human Body

Exposure:

Susceptibility to arsenicosis can vary in individuals – often within the same family drinking from the same water source – depending on their state of health, nutritional status and on how much arsenic contaminated water they drink for how long and the concentration of arsenic in water. Arsenicosis is not contagious. An individual cannot get arsenicosis or its symptoms by touching or embracing a person who is suffering from it [104].

Non- occupational human exposure to arsenic in the environment is primarily through the ingestion of food and water. Of these, food is generally the principal contributor to the daily intake of total arsenic. In some areas arsenic in drinking water is a significant source of exposure to inorganic arsenic. In these cases, arsenic in drinking- water often constitutes the principal contributor to the daily

arsenic intake. Contaminated soils such as mine tailings are also potential sources of arsenic exposure. The daily intake of total arsenic from food and beverages is generally between 20 and 300 μg / day. Limited data indicate that approximately 25% of the arsenic present in food is inorganic, but this depends highly on the type of food ingested. Inorganic arsenic levels in fish and shellfish are low (<1%). Foodstuffs such as meat, poultry, dairy products and cereals have higher levels of inorganic arsenic. Pulmonary exposure may contribute up to approximately 10 μg / day in a smoker and about 1 μg / day in a non-smoker, and more in polluted areas[33, 61, 92, 99].

In workplaces with up-to-date occupational hygiene practices, expose generally does not exceed 10 μg / m^3 (8-h time weighted average (TWA). However, in some places workroom atmospheric arsenic concentrations as high as several milligrams per cubic meter have been reported [92].

The total daily intake of arsenic by the general population is usually less than 20 μg /day, but is greatly influenced by amount of seafood in the diet. Drinking water ordinarily may contain a few micrograms of arsenic per liter. Arsenic level of drinking water ≥ 50 μg /L may lead to health effects [100].

Mechanism

Absorption, excretion and retention of arsenic in the human body is influenced by the amount and the chemical forms in which it is ingested. Arsenic in the forms that are ordinarily present in food and the organic compounds of arsenic acid are well absorbed. Following absorption, arsenic is distributed rapidly and widely to all tissues of the body e.g. liver, kidney, spleen, heart, jejunum, marrow, lungs, pancreas, muscles, stomach, thyroid, skin, brain and spinal cord [15, 89,100].

In human, arsenic can be reduced to the inorganic pentavalent from the inorganic trivalent form and methylation of inorganic to organic monomethyl (MMA) and dimethyl (DMA) forms. Methylation is mediated by cytosolic enzymes, primarily in the liver and in the main detoxification mechanism, as either MMA or DMA bind strongly to biological molecules [104,119].

In the human, arsenic can be reduced to the inorganic pentavalent from the organic trivalent form. Inorganic arsenic is methylated to organic form Dimethylarsenic Acid and Monomethyl Arsenic acid [120]. The methylation inorganic arsenic in the body is a detoxification process, which reduces the affinity of the compounds for the tissues. The arsenic (III) in the body combines

with sulphydryl containing substances and inhibits the activity of many enzymes of the group. It interferes with cell enzymes, cell respiration and mitosis [100].

Arsenic is slightly absorbed through the skin when administered in a lipid vehicle and when inhaled approximately 45% excrete in the urine and 2.5% in the faces after 10 days. About 50-100% of soluble forms of arsenic are readily absorbed following ingestions. The excessive arsenic intake can lead to health/physical problems in the body of the consumers of arsenic affected drinks and food. Other than ingestion, arsenic causes toxicity through inhalation. Arsenic in solution is more toxic than undissolved arsenic. The toxicity of arsenic decreases in the order: Arsine► Inorganic arsenic (III) ►Organic arsenic (III) ►Inorganic arsenic (V) ►Organic arsenic (V) ►Arsonium compounds and elemental arsenic [92].

Excretion

Both trivalent and pentavalent inorganic arsenic in solution are readily absorbed after ingestion. When arsenic is absorbed into the body, the major portion is excreted mainly through urine and a small portion through faces. Arsenic is also eliminated through skin, hair, nail and to some extent through bronchial secretions. After administration, arsenic appears in urine within 2 to 8 hours. About 25% being excreted in 24 hours and about 75% within 7 days of

Patient first of all complains of a feeling of faintness, nausea and severe burning pain in the throat and stomach with increased salvation. Intense thirst and severe vomiting are constant symptoms [93,123].

Purging is usually accompanied by tenesmus, pain and irritation about the anus. The stool is expelled frequently and involuntarily. Initially the stools are dark coloured, foetid and bloody but later becomes colourless, odourless and watery resembling rice water stool of cholera. The urine is suppressed and scantly. There may be serious cramps in calf muscles. Skin becomes cold and clammy, face becomes cyanosed, eyes are shunken, and pulse is feeble, irregular and frequent. The respiration becomes laboured and lastly convulsion and coma precede death [80, 93,101,121,123,124,125].

Sub-Acute Toxicity:

This is a condition where arsenic is administered in a small dose at repeated intervals [80,81,87,88]. The symptoms are at first dyspepsia, cough and tingling in the throat, then vomiting and purging with abdominal pain and tenesmus with foul tongue, dry and congested throat and a feeling of depression and languour. The motions are bloody. The symptoms of neuritis are pronounced. Severe cramps on the muscles which are tender on pressure. Patient becomes restless and cannot sleep and ultimately collapse sets in and results in death.

Chronic Toxicity:

The clinical manifestations due to chronic arsenic toxicity develop very insidiously after six months to two years or more depending on the amount of arsenic intake [15,100, 110.] Chronic toxicity of arsenic is best discussed in terms of the organ system affected-the skin, nervous system, liver, cardiovascular system and respiratory tract [15,87,88,89,101,122].

Symptoms of Arsenic Toxicity:

The clinical manifestation due to chronic arsenic toxicity develop very insidiously after six months to two years or more depending on the amount of arsenic intake[93,110,126]. The period for development of clinical manifestation differs from patient to patient depending on the amount of arsenic ingested, nutritional status of person, immunity level of the individual and the total time period of arsenic ingestion. The higher the concentration above the maximum permissible level $\geq$ 50 μg /L the amount of daily water intake, the earlier is the onset of clinical features.

The apparent symptoms of arsenicosis may be said to have manifested themselves as melanosis and keratosis mainly.

Darkening of skin (diffuse melanosis) in the body or in palm is the earliest symptom spotted pigmentation (spotted melanosis) is usually seen on chest, back or limbs. This is a very common symptom [15, 93,110].

Leucomelanosis or raindrop pigmentation, which is white and black spots side by side, is also seen on many patients. Leucomelanosis is common in persons who have stopped drinking arsenic contaminated water but had spotted melanosis earlier [7,110].

The hardening and thickening of palms and soles is called keratosis[15]. Keratosis is palpable and in most cases distribution is symmetric. Diffuse with nodular keratosis on palms and soles is a sign of moderate to severe toxicity. Rough dry skin often with palpable nodules (spotted keratosis) in dorsum of hands, feet and legs are the symptoms seen in severe cases.

Combination of pigmentation (melanosis) and nodular rough skin (spotted palmoplanter keratosis) almost points of arsenic toxicity excluding hundreds of causes of isolated pigmentation and nodular rough skin. Other symptoms sometimes found are: Conjunctival congestion, non-petting swelling (solid oedema) of feet, bronchitis, gastro-enteritis, peripheral neuropathy [15,28,29,30,31,32,33,34,87,133].

Several cases like liver enlargement, spleen enlargement and fluid in abdomen have been seen as complications of arsenicosis[15,87,88,89]. Due to arsenic toxicity, limbs may be affected by ulcer or gangrene, which in some cases have to be amputated. Squamous cell carcinoma, basal cell carcinoma, Bowen's disease, carcinoma affecting lung, bladder, genitourinary tract of other sites are often seen in advanced neglected cases suffering for long days [11, 15, 16, 28, 29, 30, 31, 32, 33, 34, 43, 108, 121, 122, 125, 126, 127, 128, 133]

Stages of Clinical Features:

In Bangladesh skin manifestations are prime and common[15, 43,108,129]. The clinical manifestations develop in four stages viz Pre-clinical, Clinical, Complication and Malignancy[97, 127]. In Bangladesh majority of the patients are found in the clinical and complication stages. The stages of clinical manifestations can be briefly described as follows:

Pre-clinical stage:

This may be subdivided into chemical phase: High arsenic concentration in urine. Sub-clinical: High arsenic concentration in body tissues with no apparent clinical symptoms.

Clinical stage:

Clinical symptoms are spotty pigmentation of skin of the body, keratosis of palms and soles, bronchitis and gastroenteritis. These are confirmed by detection of arsenic concentration measured in nail, hair and skin.

Stages of complication:

Symptoms of clinical phase are associated with different complications as the other organs like lung, liver, muscles, eye, vessels are affected[15, 129,130]. Clinical symptoms are associated with biochemical, evidence of organ dysfunction as well as histological, histochemical abnormalities and high concentrations of arsenic in different organs involved.

Stages of malignancy:

Malignancy of skin, lung, bladder or other organs may develop[7, 11, 15, 79, 110].

Clinical manifestation of chronic arsenic toxicity:

Skin effects:

A number of skin lesions have been attributed to chronic exposure to arsenic compounds [7, 15, 35,129]. Symmetric hyperkeratosis of the palms and soles is a characteristic finding after long term ingestion of inorganic arsenic in drinking water. Hyper pigmentation (melanosis) of the skin is commonly encountered

and occurs throughout the body. Melanosis is not always associated with keratosis but keratosis is always associated with melanosis. Melanosis is also observed in tongue and buccal mucus membrane[7, 11, 14, 15,42,50,79,108].

Gastro intestinal tract effects:

The chronic absorption of arsenic occasionally produces hepatocellular toxicity which may be the result of an inhibition of the enzymes by arsenic involved in cellular respiration. Trivalent arsenic binds readily to sulfuric groups of enzymes and has been shown to inhibit private dehydrogenate function that alternation has been correlated with the swelling and distortion of the hepatic mitochondria's. Chronic exposure to arsenic has been reported to produce liver enragement and has been associated with cirrhosis of the liver. Non-cirrhotic portal hypertension may develop in some cases obstruction of bile ducts may cause jaundice. Gastric symptoms like nausea, vomiting, loss of appetite, salvation constipation or sometimes diarrhea occurs with loss of weight.

Renal system effects:

Urine may be coloured or green and in some cases dysuria and anuria develop from renal tubular necrosis,

Cardiovascular system effects:

Peripheral disease has been observed among person in Chile and in Taiwan who had chronic exposure to arsenic in drinking water. Early symptoms included acrocyanosis and Raynaud's phenomenon. Those changes were associated with hyper pigmentation and hyperkeratosis. These progressed in severe cases to frank gangrene of the extremities associated with endarteritis obliterans. Myocardial degeneration and cardiac failure may result from chronic arsenic poisoning.

Nervous system effects:

Peripheral neuropathy affecting primarily sensory function has been encountered in several studies of persons with chronic exposure to arsenic. Hearing loss possibly reflecting arsenic toxicity to the eighth cranial nerve was reported in a study.

Symptoms accompanying arsenical neuritis are burning, tingling sensation pain and tenderness in the affected limb. Severe weakness is observed in legs and feet leading to difficulty in walking. The extremities show a decrease in touch, pain and temperature sensation. Tendon reflexes are absent or diminished. Knee jerks are usually lost.

Headache, drowsiness, confusion and convulsion are seen in both acute and chronic arsenic intoxication.

Hematological effects:

Chronic exposure to arsenic has been associated with disturbed erythropoiesis and megaloblastic formation has been noted. These changes may reflect the inhibitory effects of arsenic on cellular respiration, Anemia, leucopenia. Thrombocytopenia usually developed.

Respiratory system effects:

In early stages of arsenic intoxication respiratory infection is found to be associated with other clinical manifestations.

Endocrine system effects:

Diabetes mellitus and Goiter have also been reported in association with prolonged ingestion of arsenic through drinking water.

Carcinogenicity:

Arsenic has been found to cause cancer of the skin, liver, lung, urinary bladder, prostate and possibly of heamopoietic and lymphatic tissues. Inorganic arsenic has indicated an association with lung cancer [84, 86,131,132].

Arsenic exposure has been associated with three types of skin cancers-Bowen's disease, Basal cell carcinoma and Squamous cell carcinoma. These cancers frequently multiple in origin and develop primarily from arsenical keratosis. The prevalence of arsenic related skin cancer appears to depend upon total absorbed dose of arsenic.

Clinical Features of Arsenicosis

Sign and symptoms of chronic arsenicosis differ in manifestation in different countries. In Bangladesh skin manifestation are prime and common.

The clinical manifestations are categorized in the following stages [18,43,51,93]:

Pre clinical stage: Not detectable by clinical manifestation.

Initial Stage: Melanosis (spotted, diffuse)

Keratosis (spotted, Diffuse)

Conjunctivitis (Conjunctival Congestion)

Bronchitis

Gastro-enteritis

Second Stage: Depigmentation (leucomelanosis-rain drop pigmentation)

Hyperkeratosis

Edema of legs (non-pitting)

Peripheral neuropathy

Nephropathy (early stage)

Hepatopathy (early stage)

Last Stage: Nephropathy (late stage)

Hepatopathy (late stage)

Gangrene

Cancer (skin, bladder and lung)

In Bangladesh majority of the patients are found in the initial and second stages.

Clinical Features over Skin due to Chronic Arsenicosis

Melanosis:

Blackening/ darkening of the skin diffuse or spotted due to deposition of black pigment (melanin) in the skin and mucous membrane due to stimulation of melanocyte.

Mild- blackening of skin (melanosis), thinly distributed in palm, trunk, gum, tongue, lips etc (both spotted and diffuse).

Moderate- melanosis densely affecting gums, palm and trunk (spotted and diffuse) with leucomelanosis (rain drop pigmentation).

Severe- melanosis densely and extensively affecting gum, palm, trunk and whole body with leucomelanosis.

Leucomelanosis:

Depigmentation in hyperpigmeted area characterized by whitish / pallor patch in rain drop manner, due to exhaust melanocyte.

Keratosis:

Rough, dry, hard and thickening of epithelium due to increased keratinization. Keratosis is palpable and in most cases distribution is symmetric.

Mild- Just palpable keratosis (spotted and diffuse) but not clearly visible

Scatteredly affecting palm and sole.

Moderate- Palpable and visible keratosis (spotted and diffuse) affecting palm and sole.

Severe- wart-like keratosis (spotted and diffuse) on hands, legs and feet.

Hyper keratosis:

Densely and extensively distributed keratosis affecting whole palm and sole.

Treatment and Management:

So far there is no specific treatment for chronic arsenicosis. Withdrawal of further intake of arsenic contaminated water and taking arsenic free water improve the cases [11,42,43,50,51,100,122,128,129,134,135,136,200]. Chelation therapy and vitamins and nutritious diet enhance the recovery.

Chelation therapy:

Chelation therapy for the treatment of arsenicosis may consider to be the specific therapy for relief of systemic clinical manifestations and reduction of arsenic stores in the body, decreasing subsequent cancer risk.

Nutritious Diet and Vitamins:

Symptoms are improved by good diet and vitamins high protein diet helps in the clearance of inorganic arsenic by increased methylation and protects against toxic effect of arsenic. The antioxidants vitamins-A E and C play an important role for management of cases. Vitamin C reduces the toxicity of arsenic and deficiency of vitamin A increases sensitivity to arsenic [7,11,15,79,110].

Excessive intake of vitamin A may produce chronic toxicity in the body such as appetite loss, dry skin, bone and joint pain, enlarged liver and spleen, abnormal skin pigmentation Vitamin E is relatively non-toxic. Excessive dose of vitamin C, 2gm or more may produce side effects.

People should be advised to take more protein and vitamin rich food like beans, peas, pulse, lentils, wheat, soybeans, green and leafy vegetables.

Other symptomatic treatment:

Keratosis of palms and soles can be treated by local application of keratolytic ointment-20% urea and 10% to 20% salicylic acid in cream or vaseline, cryosurgery can also be done to remove keratosis. Treatment of associated fungal infection with ointment and medicine also improve the cases.

Spirulina:

Sprirulna is a multicellular, filamentous, cynobacterium belonging to aiga of cyno-phyta. It is rich in protein (65%), carbohydrate (20%), fat (5%), minerals (7%), vitamins and natural pigments. In a preliminary report, beneficial effects of spriluna in chronic arsenic poisoning patients were found [137,138,139,140].

Social Aspects of Arsenic Contamination [141].

Social instability:

Lack of proper knowledge about arsenic contamination and unavailability of arsenic safe drinking water as well as proper treatment are creating extreme instability in the social life of the people in the arsenic-prone areas of Bangladesh.

Superstition:

Superstitions and prejudices are created surrounding arsenic patients. For example in the north-eastern district of Kushtia, arsenic is considered as a "curse of Allah" or the work of evil spirits. People stay away from arsenic victims, neglect them, or become scared of them because of these superstitions.

Ostracism:

Arsenic patients are often identified by the society as patients of leprosy and as a result they remain ostracized, at either the household or the village level. Children of arsenic patients are not allowed to attend social or religious functions. The patients as well as their close relatives are not allowed to use public tube wells and village ponds. Often family members, like husbands or wives, abandon the arsenicosis victims.

The problem is more serious in the case of children. The entry of arsenic affected children into schools becomes restricted. Some may be denied the opportunity to go to school. They also are subject to social ostracism by their friends and classmates.

Marriage related problems:

Arsenic has an adverse impact on marital relationships. People are reluctant to develop marital relationships with families whose members suffer from

arsenicosis. This has caused serious anxiety for parents of unmarried adult children particularly in daughter. Many women are divorced or abandoned by their husbands due to arsenicosis.

Increased poverty:

Those having poverty are the main victims of arsenic contamination as they are compelled to drink contaminated tube well water. Researchers believe that the severity of arsenicosis is very much related to nutritional deficiency. Malnutrition makes them easy victims. Due to poverty, victims are deprived of proper treatment. When seeking treatment, the costs become a burden to them. As arsenicosis decreases the victim's ability to work, he or she often suffers from a reduced income. Due to ostracism, arsenic patients lose their jobs. Thus, arsenic negatively contributes to the poverty situation in Bangladesh.

Gender implications of arsenic contamination:

In Bangladesh women perform the majority of the household work, but their work remains relatively invisible and unrecognized in society. Among many other tasks, collecting and carrying water for household use, particularly in the rural areas, is the responsibility of women and girls. Arsenic contamination in nearby drinking water source often compels them to collect and carry water from a long distance, imposing an additional burden on them. Because of socio-

Nutritional status is the end result of utilization of energy in the body, which means whether the particular individual is nourished, malnourished, or over nourished, or whether he/ she has an imbalance of nutrition [143,144].

Nutritional status may also be termed as the condition of health of the individual as influenced by the utilization of nutrients. It can be determined adequately by the correlation of information obtained through a careful medical and dietary history, through physical examination and appropriate investigations[143, 144].

There are various methods for assessing and analysis nutritional status. Methods of nutritional assessment are as follows: [98,121,145,146,147,148,149,150,151]

- Clinical
- Laboratory
- Anthropometric
- Dietary

Clinical

It includes assessment of clinical signs and symptoms pertaining to specific nutrient deficiency. Often non–specific.

Examples: Kwashiorkor, Marasmus, Vitamin A deficiency, Anemia.

Laboratory

By laboratory method nutritional assessment is done by biochemical or functional parameters, viz. Hemoglobin level in blood. Serum retinol in vitamin A deficiency, Thyroid hormone level in iodine deficiency, of individual.

Anthropometric

Anthropometric assessment

Anthropometric examination is the most simple, most practical and ascertaining nutritional status, uses measurements of the physical dimension and the gross composition of the human body, provides information on protein and energy imbalances, identifies degree of malnutrition and provides information about past nutritional history, which is not possible by other methods.

Developmental impairment is the most extensive public health problem among children in many developing countries including Bangladesh. It arises from the complex of nutritional, biological and social deprivation and is manifest as ill

health, wasting, and growth retardation resulting in stunting (short stature), functional disadvantages, and high mortality rates. The use of anthropometry has increased rapidly in recent years. Physical development impairment may be assessed by anthropometric measurement.

The advantages of anthropometry

- Simple, safe, non- invasive procedure.
- Applicable to large sample size.
- Requires inexpensive, portable and durable equipment, which can be made or purchased locally.
- Methods are precise and accurate if standardized techniques are used.
- Information on past long term nutritional history can be obtained.
- Helps identification of mild, moderate or severe degree of malnutrition.
- Helps evaluation of nutritional status over time and from generation to generation.

Anthropometric measures include

⇒ Measurement of Growth.

⇒ Body fat

⇒ Fat free mass (skeletal muscle, non- skeletal muscle, soft lean tissues, skeleton).

Measurement of Growth: The most widely used anthropometric measurements of growth are:

- Height or length.
- Body weight.
- Head circumference (often measured).

Indices derived from growth measurements are:

- Height for age
- Weight for age
- Weight for height
- Head circumference for age

Significance of different indices derived from growth measurements are:

Weight for age

Body weight represents the sum of protein, fat, water, and bone mineral mass, and does not provide any information on relative changes in four chemical components.

Weight for age in children from 6 months to 7 years of age is an index to assess acute malnutrition, and is widely used to assess protein energy malnutrition (PEM) and over nutrition, especially in infancy when the measurement of length is difficult.

A major limitation of it as an index of PEM is that it does not take into account height differences. As a result children with low weight for age are not necessarily wasted.

To interpret a single measurement of weight in relation to the reference data, the exact age of the children must be known.

Height for age

May be used as an index of nutritional status of population groups as it estimates past or chronic nutritional status.

It can be used as index of stunting which is the end result of a reduced linear growth.

Stunting occurs from extended periods of inadequate food intake and increased mobility and generally found in countries of poor economy.

Weight for height

- It is a sensitive index of current nutritional status.
- It is relatively independent of age between 1 and 10 years, enhancing its usefulness in areas where the ages of the children are uncertain.
- Weight for height is also useful in evaluating the benefits of intervention programs, as this index is more sensitive to changes in nutritional status than height for age.
- It also helps identification of wasting in hospital patients.
- Edema and obesity may complicate the interpretation of weight for height measurements.

Body mass index (BMI) for age and thinness

BMI has been recommended as the best of the anthropometric indicators of thinness and overweight. The WHO expert committee on physical status has recommended this.

The use of interpretation of anthropometric measurements

- A low BMI indicates significant thinness or underweight in relation to height.

- BMI less than the fifth percentile indicate short term nutritional deficiencies, which could represent acute weight loss due to short term illness, inadequate intake or excessive work burden.
- BMI –for –age $<5^{th}$ percentile is recommended as a provisional cut- off point for thin or undernourished.

In this study nutritional status was measured by the following anthropometric assessment methods:

- ❖ Weight for age
- ❖ Height for age
- ❖ Weight for height

Several systems are available for classifying individuals as malnourished based on anthropometric indices. All utilized at least one or more cut off points based on appropriate reference data.

These are:-

- Percentiles
- Percentage of reference median
- Standard deviation scores or Z scores

STANDARD DEVIATION OR Z - SCORE CLASSISIFICATION

$$\text{Z- Score} = \frac{\text{Individual value} - \text{median value of reference population}}{\text{Standard deviation value of reference population.}}$$

Superiority of Z-score: [152].

- Z- Scores are useful because they have the statistical property of being normally distributed.
- This allows calculation of meaningful average value and S.D. for a population.
- It has greater capacity to determine the proportion of population that falls below extreme values to percentiles.
- If a population has a mean Z score value of O, it means that it has the same median value as the reference population.
- 2.0% of the reference population falls below -2S.D. score.

Standard deviation value and median value of reference population calculated by transforming the original National Centre for Health Statistics (NCHS) reference data. Normal Z- score varies from (– 2SD to + 2SD).

$$\text{HAZ} = \frac{\text{(Observed height) – (Median height of reference for given age and sex)}}{\text{Standard deviation of height of reference population}}$$

$$\text{WAZ} = \frac{\text{(Observed weight) – (Median weight of reference for given age and sex)}}{\text{Standard deviation of weight of reference population}}$$

$$\text{WHZ} = \frac{\text{(Observed weight for height) – (Median weight for height of reference for given sex and height)}}{\text{Standard deviation of weight for height reference population}}$$

The above cut-off points categorized nutritional status, severe and moderate forms of under-weight, stunting, wasting, over-weight or obesity.

Weight for age, Height for age, Weight for height Z- score gives indication on severity of nutritional status of children[152].

Z- score values	**Weight for age: (WAZ)**	**Height for age: (HAZ)**	**Weight for height: (WHZ)**
+2sd to –2sd	Normal weight	Not stunted	Normal
Less than –2sd to –3sd	Moderately underweight	Moderately stunted	Moderately wasted
Less than –3sd	Severely underweight	Severely stunted	Severely wasted

1. 3. JUSTIFICATION OF THE STUDY

Bangladesh in recent times is passing a crucial moment due to arsenic toxicity in ground water. Prolonged consumption of water containing high concentration of arsenic affects on health. The effect is more serious with increasing length of exposure and with increasing concentration. Other factors such as sensitivity, nutritional status of the individual, genetic cause, immunity also play important roles.

Dietary regimens that reduce methylation capacity, such as protein malnutrition may have a role in explaining individual variation in susceptibility to arsenic cell damage and carcinogenicity.

Poor nutritional status of the people favors the toxicity to arsenic[32, 53, 60, 61].

Arsenic effects on the antioxidant defense system. Arsenic lowers the plasma level of uric acid and increases plasma triglycerides content without increasing vitamin E level in serum. As a result there is an oxidative stress on human antioxidant defense.

Zhang in 1996 showed that persons having accumulation of arsenic in their serum lost their normal nutritional status gradually [176].

Bangladesh is a country populated with 150 million people, high density populated area, less literacy, very low socio-economic condition with prevalence of morbidity (malnutrition) (both sexes 3.32, male 2.42, female 4.27 per1000 population)[77]. Along with the infectious disease, malnutrition is also loaded with the health problem of the people of this country.

Now a new burden of arsenic toxicity is being overloaded with the usual health problem of the people of this country.

In recent times Bangladesh is facing real catastrophe due to arsenic contamination in tube-well water. So, it is important to explore the relationship between nutritional status and arsenic toxicity.

The children of 5-14 years comprise 26.51% of the total population[77]. They are the future generation of the nation. At present it is an important task to explore that the state of nutrition and arsenic toxicity among them in order to take appropriate measures for the health development.

The study attempts to provide information about the nutritional status of both the arsenic exposed and non-exposed group of children. The community will be benefited if we know the results whether arsenic contaminated water plays any role on the nutritional status of our children.

1.5. OBJECTIVES of the study:

1.5.1. General objective:

To assess and compare the nutritional status of children aged 5-14 years in arsenic exposed and non- exposed areas.

1.5.2. Specific objectives:

1. To determine height and weight of the children in arsenic exposed and non- exposed area.
2. Compare the nutritional status of children between the two areas.
3. To assess the dietary intake in terms of 24-hours recall basis of children of the two areas.
4. To determine the type of water consumed by the children of the study areas.
5. To compare the socio-demographic characteristics of the two areas.

1.5.3. ULTIMATE OBJECTIVE

1. Provide results to the planners and policy makers to control arsenic related health problems.
2. To disseminate the findings of the study to the professional gathering in the form of seminar.
3. To publish research articles in peer reviewed national and international scientific journals.

1.6. Research question:

What is the difference in nutritional status between arsenic exposed and non-exposed children?

CHAPTER II

LITERATURE REVIEW

2. REVIEW OF LITERATURE

Arsenic is a chemical element that has the symbol As and atomic number 33. Arsenic was first written about by Albertus Magnus (Germany) in 1250. Its atomic mass is 74.92 [153].

Arsenic is a naturally occurring element widely distributed in the earth's crust. In the environment, arsenic is combined with oxygen, chlorine, and sulfur to form inorganic arsenic compounds. Arsenic in animals and plants combines with carbon and hydrogen to form organic arsenic compounds [84, 86].

Several studies have shown that ingestion of inorganic arsenic can increase the risk of skin cancer and cancer in the liver, bladder, and lungs. Inhalation of inorganic arsenic can cause increased risk of lung cancer. The international agency for research on cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans [84, 86,131,132,154,155].

There is some evidence that exposure to arsenic in the womb and early childhood may increase mortality in young adults [84,156].

There is some evidence that inhaled or ingested arsenic can injure pregnant women or their unborn babies. Arsenic can cross the placenta and has been found in fetal tissues. Arsenic is found at low levels in breast milk [84,156].

Poor nutritional status may increase as individual's susceptibility to arsenic toxicity, or alternatively that arsenicosis may contribute to poor nutritional status. It was observed in different study from Taiwan, Chile, West Bengal India, Nepal, Japan and Bangladesh [63,157,158,159,160,161,162,163]. Limited studies have indicated that poor nutritional status may increase the risk of arsenic related health effects.

Mazumder (2006) had done a study on chronic exposure of arsenic (>50 μg/L) in Indian population. The study showed that the age adjusted prevalence of keratosis and hyper pigmentation were strongly related to arsenic level of water. The study shows that the various other non carcinomatous clinical manifestation of 3512 and 4171 people drinking water containing arsenic ≤ 50 μg/L respectively. Incidences of weakness, hepatomegly, chronic lung disease, dyspepsia and neuropathy were found to be among 1.37, 2.99, 7.74, 31.81 and 2.73 percent in the former group while 4.99, 1021, 11.68, 27.84 and 4.7 percent in the later group respectively. The study also shows that arsenic dose response relationship in regard to hepatomegly, lung disease and weakness were also carried out. The overall age adjusted prevalence of hepatomegly among females gradually rose from 3.3 per 100 in the lowest exposure category (<50 μg/L) to 18.6 per 100 in the highest exposure category (<800 μg/L). A strong trend

appeared in males, the age adjusted prevalence increased from 3.6 per 100 in the lowest category to 29.8 per 100 in the highest exposure category. The prevalence of hepatomegly was greater among males than females. Results of pulmonary effects manifested by cough, crepitation and shortness of breath among non smokers were analyzed excluding 819 smokers because of their small numbers and potential confounding. The overall prevalence in females for each respiratory out come was close to 2.5 per 100. Clear trends of increasing prevalence by arsenic water concentration can be seen for cough and crepitation. Among males, the overall age adjusted prevalence of cough (5.2 per 100) and crepitation (4.4 per 100) were nearly twice as high as among females but once again there were trend of increase with increasing arsenic content in children of both sexes in the age range of 10-19 years. The age adjusted prevalence of weakness was also found to be increased with increasing arsenic water concentration in both sexes[164].

Choprapawon (2006) assess the prevalence of cancer from cancer registry, Ronpibool (arsenic contaminated for last ten years) district (2.22/1000) ranked fifth among the 23 districts. The study revealed the prevalence of cancer sites in different districts arsenic contaminated area named Hausai (3.19/1000), Lansaka (2.29/1000), Phomkiri (2.23/1000), Kanom (2.23/1000), and Ronpibool

(2.20/100). Lung cancer was the highest in Hausai, Lansaka, Phomkiri, while cancer of oral cavity (includes cancer of tongue, plate, mouth, saliva gland and tonsil) found highest in Kanom. Lymphoma (includes Hodgkin's, Follicular node, Diffuse non-Hodgkin's, T-cell) and breast cancer had highest proportion (13.5% of all sites), followed by lung cancer (10.2%). Ronpibool had highest proportion of skin cancer (3.4%) [165].

In Romania and Hungry the study was done in the period of (1981-1991) to estimate the incidence of morbidity between the arsenic exposed and non-exposed population. The arsenic concentration of exposed group was above 100 μg /L and controlled group was < 50 μg /L. The study revealed that high rate of bladder cancer among the exposed population (adults). Lung, kidney, leukemia, and others lymphatic and hemathopoetic cancers were not elevated in the exposed as against the unexposed population. The study also revealed that incidence of anemia, high blood pressure, peripheral neuropathy were high in exposed groups, increase prevalence of cutaneous disorders (hyperpigmentosis, hyperkeratosis, epithelioma), peripheral neuropathy (parestesis in the extremities) and also cardiovascular diseases (hypertension, myocardial infraction, strokes) in exposed group versus the control [166].

Mahmood (2002) review in different papers and noted that according to WHO 13 lakh children of under-five age group, mostly in developing countries, die annually for causes related to unsafe drinking water, poor sanitation and hygiene. The report says that "Arsenic problem" will have major economic impacts throughout Bangladesh, affecting agriculture and other related industries, water management, public health and the overall National economy. Arsenic has been known as a human carcinogen; at high levels it can damage nerves, the stomach, intestines and skin. The clinical manifestations due to chronic arsenic toxicity develop very insidiously after six months to two years or more depending on the amount of arsenic intake. The most common early signs of chronic arsenic poisoning are muscle weakness and aching, skin pigmentation, garlic odor in breath and perspiration, excessive salivation and sweating, stomatitis, generalized itching, sore throat, coryza, lacrimation and conjunctivitis. Patients with chronic arsenicosis often complain of gastrointestinal symptoms such as anorexia, nausea, vomiting, excessive salivation, pain in abdomen, constipation or sometimes diarrhea and weight loss. It has been found that arsenic toxicity is more pronounced among poor and malnourished groups of people. The arsenic contamination is not only causing serious health hazard to the people, but also affecting the environment creating social problems and overburdening our limping economy because it is creating a

vicious cycle of malnutrition. Arsenic and other heavy metals used both in agriculture and in industry, can cause, cancer, damage to the liver, kidney and intestines, sterility, malnutrition and genetic abnormalities in human. Although thousands of people have already been diagnosed with poisoning symptoms, much of the 'at-risk' population particularly among children and adolescents has not yet been assessed for arsenic related health problems. One study done in three villages in Northern Argentina with high level of arsenic in drinking water indicated that children are more sensitive to arsenic induced toxicity than adults [4, 33].

Dhaka community hospital reviews article and suggest that toxicity for arsenic in human body is manifested in three manners [32].

a) Arsenic in urine (for recent exposure)

b) Arsenic in blood (for instant exposure)

c) Arsenic in skin, hair and fingernails (for chronic exposure)

Manifestation of arsenic toxicity depends on dose of ingestion:

- Large doses for arsenic can produce human deaths.
- Lower levels impact many different body tissues or systems: result in 'system effects.

- Oral intake has greater harmful effects than dermal or inhalation exposure.

Intake of arsenic affects different system of human body:

- Skin abnormalities (dark/light sports in skin,' crons' on palms and sole: progress to skin cancer.
- Irritation of digestive tract, leading to pain, nausea, vomiting, diarrhea
- Fetus damage during pregnancy
- Blood vessel damage
- Increase cancer risk, especially in liver, bladder, kidney and lung
- Liver and/or kidney damage
- Abnormal heart function
- Decreased production of red and white bloods cells
- Aggravated malnutrition affectations
- Impaired nerve functioning pins and needles feeling in feet and hands

Arsenic and certain arsenic compounds are known carcinogens. Amounts of arsenic intake required to cause harmful effect depends on the chemical and physical from of arsenic. Inorganic from of arsenic is more toxic than organic forms. Water-soluble forms are more toxic. Humans are most sensitive to arsenic than animals.

Cross sectional study was done by Maharjan (2007) on adults villagers (both male and female) in an area of lowland Nepal. The study shows that arsenic intake was negatively correlated with body mass index and consequentially increased the prevalence of underweight individuals, among whom the prevalence of skin manifestations was 1.65 fold higher than normal weight individuals. The study also revealed that the prevalence of skin symptoms was consistently higher in the underweight than in the normal groups when exposure level was considered [157].

A cross sectional study was conducted by Tun (2003) in Myanmar on arsenic exposed populations. The study shows that high arsenic in drinking water can cause skin symptoms and other clinical manifestation in exposed groups and clinical manifestation became fading after stopping consumption of arsenic contaminated water [32].

Maharjan (2005) done community based dose response study on arsenic exposed community in lowland in Nepal. The study revealed that prevalence of arsenic induce skin manifestation was 80% and males had prevalence twice as high as females [21].

Kadono (2002) done study to assess the prevalence of arsenic contamination of skin manifestation (Skin manifestation are sensitive marker of arsenicosis). The

study revealed that 50% of the study population showed skin manifestation and prevalence of skin manifestation was quit high and males were more vulnerable to arsenic exposed [167].

Maharjan (2001-2004) surveys done on arsenicosis cases in Terai, Nepal, study was conducted in six arsenic contaminated district of terai namely Nawalparasi, Bara, Parsa, Rautahat, Rupandehi and Kapilavastu. In these districts, arsenic contamination in tube wells varied from 2.1% to 25.7%. The prevalence of arsenicosis was on average, 2.2% and varied from 0.7% in Kapilavastu district to 3.65 in Nawalparasi district. In the community based study the highest prevalence (18.6%) of arsenicosis was found in Patkhouli village of Nawalparasi, where 95.8% of tube wells were contaminated with arsenic. The prevalence of arsenicosis was higher in older age-groups (>50 years) of both the sexes. Males suffered more from arsenicosis than females (odds ratio: 2.50, 95% confidence interval 1.80-3.47). Skin, manifestations, such as melanosis and keratosis, were the common symptoms of arsenicosis [168].

Cross sectional study done by Guha (2006) in arsenic contaminated area in West Bengal shows that morbidity from skin lesions due to chronic arsenic toxicity has been found in almost one-third of the study populations (aged more then 15 years). The study also revealed that higher prevalence of arsenic skin lesions

among males. There was a significant association of the disease with the exposure levels and to the dose index ($p<0.05$) [169].

Different studies shows that arsenic related skin lesions associated with water containing arsenic level, consumption and duration of water intake [11,14,21,25,38,39,41,43,44,51,168,170].

Smith (2000) has done cross sectional study in Chile. The exposed and non exposed population taking water was 750 to 800 and 10 µg/L respectively. The study population also taking fruits, vegetables, red-meat and chicken. The study revealed that arsenic induced skin lesion occurs despite good nutrition [58].

Islam (2004) carried out a study in arsenicosis patients of arsenic exposed area. The mean arsenic concentrations in the drinking water among exposed and non-exposed population were 218 µg /L and 11.3 µg /L respectively. The study shows that incidence of leukemia and anemia was more in exposed group. The study also found that body mass index was lower in about 28% of the arsenicosis cases compared to 15% of the control population [171].

A study was conducted by Rahman (2006) to find out skin lesion among male and female population. The concentration of drinking water was 143 µg /L (male) and 155 µg /L (female). The study shows that males had a higher risk of developing skin lesions than females [30].

A cross sectional study was done by Ahmad (1999) to compare pregnancy outcomes in women exposed to arsenic through drinking water (>0.1μg /L) to outcomes of women who were exposed to arsenic level <0.02 μg /L. The study found that rate of spontaneous abortion, stillbirth, and preterm birth were 2.9, 2.24, 2.54 times higher respectively in the exposed group than in the non-exposed group[14].

Watanabe (2001) conducted a surveys over arsenic exposed population in rural area (water arsenic level was > 50 μg /L) in Bangladesh. The study suggested that male were more sensitive in developing hyperkeratosis and melanosis than female [172].

Rich (2006) done cohort study on arsenic exposed area to observed the pregnancy outcomes among pregnant women. The concentration of arsenic level was 50 μg / L. The study shows that arsenic affects birth weight, gestational age, and placental weight of arsenic exposed pregnant women [26].

Meharg (2003) conducted study in Bangladesh to examine the arsenic assault on food chain. The study shows that low dietary intake of protein and micronutrients increases vulnerability to arsenic related diseases[173].

A study was conducted by Rahman (2001) on fifty districts of Bangladesh and nine districts in West Bangle over adults and children population. The arsenic concentration of that area was above 50 µg /L. The study shows that symptoms of chronic arsenic toxicity developed insidiously after 6 month to 2 years exposure. The study also found that the onset of arsenic toxicity depend on the concentration of arsenic in drinking water intake and nutritional status of individuals [34].

Chen (2006) carried out series of study on arsenic exposed population. The study shows that under nutritional status, the low serum level of antioxidant vitamins increased the risk of arsenic induced health hazards [174].

Smith (2006) carried out study on the role of nutritional status on human susceptibility to the health effects of arsenic in drinking water in Argentina, Chile, India and Taiwan. The study shows that nutritional deficiency (like: selenium, B-carotene, methionine) has susceptibility to arsenic related effects [175].

Milton (2004) conducts study to assess the nutritional status among the chronic arsenic exposed and unexposed population. The study revealed that mean arsenic concentration in drinking water for the exposed and unexposed population was 641.15 and 13.5 µg / L respectively. Body mass index was found to be lower than 18.5, the cut off point malnutrition, in 57(41.315%) out of 138 exposed arsenicosis cases and 31(21.53%) out of 144 unexposed individuals. The crude prevalence ratio was 1.92(95%) CI=1.33, 2.78) for poor nutritional status among the arsenicosis cases compare to the unexposed populations. The findings of this study add to the evidence that poor nutritional status may increase an individual's susceptibility to chronic arsenic toxicity, or alternatively that arsenicosis may contribute to poor nutritional status [71].

Rahman (2001) study done in different districts of Bangladesh and West Bengal. The study shows that symptoms of chronic arsenic toxicity depend on the concentration of arsenic in drinking water, volume of intake and the health and nutritional status of individuals [34].

A study was conducted by Zhang (1996) among arsenic exposed group of population suffering from chronic renal disease. The study found that there was accumulation of arsenic in their serum and they lost their normal nutritional status gradually [176].

Miamato (2005) conducted a study to find out the association of nutritional status (wasting) among the arsenic exposed and non-exposed children of 7-14 years of age. In the study area the arsenic concentration of tube well water was above 0.01µg/L. The study showed that children in exposed area was significantly more wasted than non-exposed area (mean difference = 0.361, $p < 0.001$) [177].

Chronic arsenic poisoning associated with groundwater contamination has been reported from many developing countries, where poor nutritional status is concomitantly found. Although it has been suggested that poor nutritional status affects the toxicity and metabolism of arsenic [54, 55], a few systemic reports dealing with this issue exit. A case control study showed that malnourished individuals are more often found among patients with arsenicosis than among the non-exposed population [71]. An extensive study conducted in West Bengal reported heightened susceptibility to arsenic toxicity among individuals with lower body weight, because the groups with different exposure level were recruited from different areas, the modifying effects of nutritional status could have been mediated by other unidentified (environmental) factors such as dietary habit or socioeconomics status. There are some other studies [46, 21]

describing the effects of nutrition on arsenic toxicity, with very limited quantitative information.

Participants with poor nutritional status (weight below 80% of standard body weight for their age and sex) were reported from West Bengal, India to have overall 1.6 fold- increase (for males = 1.5, females = 2.1) in the prevalence of keratosis, suggesting that malnutrition may increase the susceptibility to arsenic toxicity [161]. Arsenic affected people of southwestern Taiwan [63], West Bengal India [158,159,160] and the Antofagasta region in north Chile [162,163] were reported to have a poor nutritional status.

Arsenic intake was negatively correlated with body mass index and substantially increased the prevalence of underweight individuals, among whom the prevalence of skin manifestations was 1.65 fold higher than normal weight individuals. When exposure level was considered, the prevalence of skin symptoms was consistently higher in the underweight than in normal individuals [157].

Chung (2006) determines whether differences in micronutrient status contribute to susceptibility to arsenic induces skin lesions. The study revealed that lower micronutrients (especially lower selenium, vitamin E and beta carotene) increase risk of arsenic induced skin lesions [158].

Dhar (1997) conducted study on arsenic effected children. The study shows that all the arsenicosis children were very poor and ill nourished [8].

Maharjan (2007) conducted a cross sectional study in a population of almost all are farmers, share a similar socioeconomic and lifestyle. The study found that arsenic intake was negatively correlated with body mass index and substantially increased the prevalence of underweight individuals, among whom the prevalence of skin manifestations was 1.65- fold higher than normal weight individuals. When exposure level was considered, the prevalence of skin symptoms was consistently higher in the underweight than in the normal groups [157].

A cross sectional study done by Mitra (2002) the study revealed that the mean arsenic concentration in water was significantly associated with the severity of the disease and body mass index correlated inversely ($r = -0.298$, $p=0.013$) with the duration of disease after controlling age [180].

Kabir (2001) in a case control study among adult population of similar socioeconomic status and also both were drinking same water source observed that BMI of exposed assessed group was significantly lower than that of non-exposed group (19.71± 2.16 vs. 22.02±1.81, $p< 0.001$). Mean ±SD weight and height of the arsenicosis patients were significantly lower than non-arsenicosis patients (weight: 47.17±6.83 vs.54.35± 6.03, $p<0.001$; height: 154.39±10.38 vs. 157.07, $p<0.05$) [73].

Ahmad (2002) done in a cross sectional study among the children of 6-12 years of exposed and non-exposed group. The study found that exposed groups were shorter compared to non-exposed groups ($p< 0.05$) [104].

Islam (2004) in a case control study among arsenic exposed adult population showed that the 28% of the cases were underweight (malnourished) compared to that of 15% of the control. The mean arsenic concentration level of exposed (case) and non-exposed (control) were 218.1µg/L and 11.3µg/L respectively [171].

Milton (2004) in a cross sectional comparative study in an adult population 6-70 years observed that malnutrition (BMI <18.5) was 1.92 times higher among those having arsenicosis compared to those not having arsenic exposure (mean arsenic was >50 µg/L) [71].

A study was conducted by Mitra (2004) showed that there was no significant difference in terms of BMI between those having signs of arsenic toxicity and those not having such sign but having similar exposure [160].

Study done Minamoto (2005) among the arsenic exposed and non-exposed children of 7-14 years of age showed that the nutritional status (wasting status) in exposed children was lower than those in non-exposed children [160].

Mitra (2004) study done on a population with mean age of ~ 41 year observed that there was no difference in terms of BMI between those having signs of arsenic toxicity and those not having such sign but having similar exposure [160].

In Japan a follow up study was observed in infants exposed to arsenic (1.3- 3.6 mg/day) through intake of contaminated powdered milk showed that teenagers were on average shorter and of lower weight [57].

In animals a low intake of protein, choline or methionine has been shown to result in a decrease arsenic methylation and a marked increase in tissue retention [181].

Earlier experimental studies suggest that poor nutrition results in decreased methylation of arsenic owing to decreased supply of the methylation substrate, eventually leading to enhanced arsenic toxicity (ATSDR 2000). This is supported by a recent human study showing that a low dietary intake of protein,

iron, zinc and niacin was associated with enhanced accumulation of monomethyl arsenic acid in a US population [159].

A study was done by Akter (2005) to assess the effect of arsenic concentration of water, seafood and food grains or vegetables. The study showed that the concentration of arsenic in seafood is higher but most of the arsenic is in forms that are non-toxic to humans: i.e. the bioavailability is low. Therefore, that is perfectly safe to eat. The study also revealed that a food that has high bioavailability of arsenic is probably more toxic. The study concluded that the concentration of water by arsenic is probably more harmful to human than arsenic in food grains or vegetables, because arsenic bioavailability in water is generally higher than its bioavailability in food [182].

A study was done by Almela (2002) in Spain to assess different metal like: total arsenic, inorganic arsenic, lead, cadmium and mercury contents of 18 algae food products. The study found that arsenic concentration in algae foods was higher [183].

A study conducted by Munoz (2000) in Spain to assess the total arsenic and inorganic arsenic contents in 153 samples of seafood products: fish, mollusks, crustaceans and preserved fish. The study found that the content of arsenic in seafood product was higher [184].

A study was conducted by Rahman (2007) to investigate the accumulation and distribution of arsenic in different fractions of rice grain collected from arsenic affected area of Bangladesh. It was observed that regardless of rice varieties, arsenic content was higher in root then that of shoot and raw rice grains. The study also showed that arsenic content was higher in non-parboiled rice grain then that of parboiled rice[185].

A study was conducted by Rahman (2007) to assess the concentration of arsenic on varies types of leafy and non-leafy vegetables grown in arsenic contaminated area. The study showed that the highest mean arsenic values observed were 463 mug/kg for sweet gourd leaf, 429 mug/kg for red amaranth and 369 mug/kg for green amaranth. The highest mean arsenic levels were detected for arum root (1167 mug/kg) followed by water spinach (1001 mug/kg) and radish leaf (962 mug/kg) in Muradnagar. The maximum mean arsenic concentrations found were 850 mug/kg for marsh herb, 437 mug/kg for arum root and 407 mug/kg for sweet potato leaf in Nabinagar. The study observed that daily dietary intake of arsenic from sampled vegetables was 86.5, 221.0 and 93.0 mug for adults (both males and females) and 51.9, 132.6 and 55.8 mug for children around 10 years for Laksam, Muradnagar and Nabinagar, respectively[186].

A study was conducted by Signes (2007) to estimate the total and inorganic arsenic intakes through both drinking water and cooked rice. The result showed that arsenic concentration in cooked rice is higher than that of in raw rice (range 227 to 1641 micro kg (-1) [187].

Devesa (2007) on his study observed that food is generally consumed in processed forms, after a preservation treatment or cooking, which may alter the concentration and chemical forms of arsenic. The study revealed that all the arsenic present in cooking water may be retained during cooking of rice, increasing the contents of this metalloid to significant levels [188].

What is the Practical Importance of reduced Body Mass Index by Arsenic?

Arsenic can affect insulin related sugar metabolism [189] or disturb glucocorticoid functions [190,191] both which compromise the energy metabolism. Alternatively, systemic disease status caused by exposure to arsenic may lead to malnutrition[192, 193,194].

Arsenic and many of its compounds are especially poisons. Arsenic disrupt ATP production though several mechanisms. At the level of the citric acid cycle, arsenic inhibits pyruvate dehydrogenase and by competing with phosphate it uncouples oxidative phophorylation, thus inhibiting energy-linked reduction of NAD+ mitochondrial respiration and ATP synthesis. Hydrogen peroxide production is also increased, which might form reactive oxygen species and oxidative stress. These metabolic interferences lead to death from multi- system organ failure probably from nercotic cell death, not apoptosis [195].

Arsenite has high affinity for sulfhydryl groups and thus can form covalent bonds with the disulfide bridges in the molecules of insulin, insulin receptors, glucose transporters (GLUTs) and enzymes involve glucose metabolism(eg: pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase). As a result, the normal functions of these molecules can hamper [189].

Arsenic can disrupt the puruvate and succinate oxidation pathways. This inhibition effectively blocks the Krebs cycle, interrupting oxidative phosphorylation, which results in marked depletion of ATP stores. Arsenic also can produce a thiamine deficiency by preventing transformation of thiamine into acetyl-coenzyme A (COA) and succinyl-COA [196].

Studied done JHA (2004) in the seedlings of two rice cultivars (Malviya-36 and Pant-12) the effect of increasing levels of arsenic in situ on the content of sugars and the activity of several enzymes of starch and sucrose metabolism: α-amylase (EC 3.2.1.1), β-amylase (EC 3.2.1.2), starch phosphorylase (EC 2.4.1.1), acid invertase (EC 3.2.1.26), sucrose synthase (EC 2.4.1.13) and sucrose phosphate synthase (EC 2.4.1.14). During a growth period of 10-20 d As_2O_3 at 25 and 50 μM in the growth medium caused an increase in reducing, non-reducing and total soluble sugars. An increased conversion of non-reducing to reducing sugars was observed concomitant with arsenic toxicity. The activities of α-amylase, β-amylase and sucrose phosphate syntheses declined, whereas starch phosphorylase, acid invertase and sucrose syntheses were found to be elevated. Results indicate that in rice seedlings arsenic toxicity causes perturbations in carbohydrate metabolism leading to the accumulation of soluble sugars by

altering enzyme activity. Sucrose syntheses possibly play a positive role in synthesis of sucrose under arsenic toxicity [197].

Management of Arsenicosis

Ahmad (1998) conducted study among the adult populations having melanosis (93.5%), keratosis (68.3%), hyperkeratosis (37.6%) and depigmentation (leucomelanosis) (39.1%). The study observed that improvement of arsenic toxicity on withdrawal of intake contamination water, taking protein rich food and vitamin A, E and C [11].

Ahmad (1998) conducted study on the management of arsenocosis by taking vitamin A, E, C and regime. The study shows that there was improvement of keratosis 90.9% and melanosis 86.4% by these regimes [79].

Hassanuzzaman (2001) in a cross sectional study showed that there was significant difference of mean serum vitamin A, E, C and zinc level between arsenic exposed and non-exposed populations [198].

Sikder (1999) conducted study in a population of different age (6-54) groups. The study observed that melanosis (100%), hyperkeratosis (80%), legs cramp (20%), conjunctivitis (12%) improved with avoidance of arsenic contamination water, local application of keratolytic ointment (20% urea and 10% to 20% salicylic acid or Vaseline) and oral use of vitamin A, E and C [40].

Khan (1997) in their study mentioned that symptom of arsenicosis patient had been improved by taking good diet and vitamins A, E, and C and used arsenic safe water [43].

Steinmasu (2005) in his study observed the effect of the diet and arsenic metabolism. The study revealed that people with diets deficient in protein and other nutrients are more susceptible than others to arsenic caused cancer [159].

Huq (2000) conducted study on patient aged between 18-65 years having chronic arsenic poisoning. The study revealed that all the patients were improved clinically and there was significant reduction ($p < 0.0001$) of the excretion of urinary arsenic with spirulina [138]. This is in line with other studies conducted in Bangabandu Sheikh Mujib Medical University, Dhaka[137, 138,140,199].

A study was done by Khan (1999) all over Bangladesh from 1994 to 1998. It was observed that there was improvement of initial stage and second stage of arsenicosis patients by the withdrawal of arsenic contaminated water and taking vitamin A, E and C and used keratolytic agent [105].

Few studies done on the patients having chronic arsenicosis evaluate the effectiveness of management by vitamin A, E, C regimen and withdrawal of arsenic contamination water. The study revealed that there was clinical improvement of patients[43, 69, 79, 200, 248].

Different studies show that there was clinical improvement of chronic arsenicosis by the treatment of spirulina and withdrawal of arsenic contamination water[100, 138, 139, 140, 244].

Intestinal Parasite

Malnutrition is a worldwide problem and the problem is more marked in developing country like Bangladesh, where children are the main victims [57]. The causes of malnutrition are complex; in one side the causes include nutritional deficiency like vitamin A, calcium, iron, iodine, helminthic infestation etc. and in other side consumption of environmental toxin like lead, arsenic, mercury etc. through water can seriously deplete stores of iron, vitamin C and essential nutrients [57].

In Bangladesh helminthic infestation was one of the causes of malnutrition. Different studies observed that malnutrition and low hemoglobin levels are widely prevalent among those children suffering from intestinal parasite [102,111, 201].

Helminthic infestation is a serious public health problem, especially in areas of low environmental quality and of people of low nutritional status. In the growing stage, children are more susceptible to the ill effects of parasitic attacks, as their need for nutrients is high. In young children, physical and

mental development may be effected by mal-absorption, blood and protein loss and diarrhea generated often by presence of several types of worms in the gut. There is also the risk of complications. Many parasites interfere with the process of intestinal absorption of nutrients. They feed on the nutrients, depriving the children of its sources of nutrition. The child is thus thrown into a state of acute and chronic malnutrition[204, 205,208,246,247].

In a cross sectional study conducted by Oberhelman (1998) showed correlation between intestinal parasites, physical growth and psychomotor development among study children. The study showed that the prevalence of malnutrition was 14.6% weight for age (WFA), 8.4 % weight for height (WFH), 36.3% height for age (HFA). Helminthic infestation was more prevalent in malnourished children. Developmental status associated with low weight for age (WFA) and intestinal helminthes. The study also revealed that intestinal helminthes may be contributory factor for developmental disabilities [202].

Salviva (1990) conducted study to observe the association between stunting status and helminthic infestation in children. The study showed that there was a positive association between intestinal parasite and stunting status in studied children [203].

Chan (1992) conducted study by three anti- helmithic interventions to observe the effect on the prevalence and intensity of intestinal nematode infections. The study shows that the repeated chemotherapeutic intervention reduced both prevalence and intensity levels of intestinal nematode infections [204].

A study was done by Kumar (2003) to evaluate the prevalence of anemia with worm infestation on school going girls of age 6-10 years. The study showed that prevalence of worm infestation was 86.66%, 68.16% and 82.9% in mild, moderate and severely anemic groups[205].

A study was done by Ananthakrishnan (2001) to assess different disease pattern among the school children age 5-15 years. The study revealed that anemia was 57.1%, under nutrition 57.6%, worm infestation (46.4%) and skin infections 8.7% [206].

Oliveros (2005) in the first National conference on intestinal helminth at the Asian institute of management said that different study showed that children age one to five are infected with soil intestinal helminthasis. He also said that worm infestation have adverse effects on the nutritional status [207].

Rao (2005) study done on adolescent boys and girls aged 11-19 years. The study revealed that intestinal parasites was observed in 59.5% adolescents there was

high prevalence of under nutrition in terms of under weight 61.7%, stunting 51.7%, wasting 32.8%. The study also showed that 82.3% study children were anemic [208].

A cross sectional study was conducted by Awasthi (1997) in preschool children. The study showed that 67.6% were underweight, 62.8% were stunted, 26.5% were wasted and hemoglobin levels of the children were 9.1 g / dL, 9.3g/ dl and 9.6 g/ dL respectively. The study also showed that malnutrition and low hemoglobin levels are more prevalent among those children suffering from intestinal parasite [201].

Cross sectional study conducted by Oberhelman (1998) to assess the correlation between malnutrition and intestinal parasite shows that the prevalence of malnutrition was 14.6% weight for age, 8.4% weight for height and 36.3% height for age among those who were suffering from helminthic infestation [202].

Malnutrition a Major Public Health Problem in Bangladesh

The United Nation International Children Emergency Fund report shows statistical information on children's nutritional status for 187 countries, where it is evident that Bangladesh has the worst nutrition situation among all countries. About 56% of the children (<5 years) are under weight and another 55% are short for their height. Each figure is the highest in the world, the lowest world figure for the former is 3% and the later is only 1%. The percentage of acutely malnourished children (wasted) is 18% in Bangladesh, only next to the highest figure in the world is 25% in Afghanistan. Prevalence of low birth weight appears also at the highest level (50%). The 4th report on world's nutrition situation, published by a UN body, claims that iron deficiency anemia itself causes a productively loss of 2% of GDP in Bangladesh[209].

A study was conducted by Hassan (1998) to assess the nutritional status among the children aged 0-12 years. The result shows that only 6% were found to have normal weight for age and 75% were considered as suffering from malnutrition of 2nd and 3rd degree. The weight for height was normal in 43%, where as 41% were found to have been suffering from mild under nutrition. Sixteen percent were considered as wasted (weight for height less than 80% of the standard). Almost 65% had height for age less than 90% of standard (stunted). When

wasting and stunting were combined, only 30% could be considered normal and 10% had severe malnutrition [210].

Hassan (1993) done another study on nutritional status of 1108 children of Dhaka city of aged 0-12 years, was assessed by measuring weight for age, weight for height and height for age from 11 locations. The study observed that only 12% of children were found to have normal weight for age and 58% was considered was suffering from malnutrition of second and third degree. The weight for height was normal in 39% and 21% was considered as wasted (weight for height <80% of standard). The percentage of stunted children increased with age. When wasting and stunting were combined, 47% of the children could be considered normal and 12% severe malnutrition [211].

Jahan (1994) conducted cross-sectional study among the hundred house holds in a village of Narayanganj district between 1988 and 1989. The study indicted that the children were mostly deficient in energy (54%), protein (49%), vitamin A (77%) and riboflavin (73%). About 70% of children were either wasted or stunted or both. Almost all of them (98%) were found to suffer from various degrees of malnutrition. 49% of the children were anemic [212].

Kabirullah (1990) conducted study in the anthropometric nutritional status of pre-school children in a rural village, has been analyzed to determine the

influence of various socio-economic factors, out of a target population of 2136. 82.5% were available for the study. The average weight for age of the children was 73.1% and height for age was 89.7% of the National Center for Health Statistics (NCHS) standard. The effect of age on the nutritional status of the children was significant but not linear, Weight for age was better at the age was better at the age of 5-12 months compared to other age groups. Level of education of the parent affected the nutritional status almost linearly. Nutritional status of the children was independent of the occupation of their parent. But annual family income exerted a significant influence on the nutritional status. The size of the family had no effect. The results are discussed in the light of the situation in Bangladesh to determine the indicators of the nutritional problems of the country [213].

Sultana (1985) has analyzed medical records of 2137 children admitted to children's nutrition unit (CNU) in Dhaka to find out the risk factors contributing to protein energy malnutrition (PEM). Most of them come from urban slums of Dhaka city. The patients were admitted over a period of 6 years 18% were marasmic, 20% had kwashiorkor and 62% had marasmic – Kwashiorkor according to welcome classification [214].

Study conducted Keramat (1977) among 227 students of 5-13 years boys and girls to examine their nutritional status. The study revealed that among the boys 11.4% were suffering from varies degree of malnutrition at the age of 5-6 years, 15.7% at the age of 7-8 years, 8.5% at the age of 9-10 years and there was no cases of malnutrition above 10 years of age. Among the girls 11.5% were suffering from varies degree of malnutrition at the age of 5-6 years, 38.5% at the age of 7-8 years, 20% at the age of 9-10 years and 20% at the age of 11-12 years [215].

Nahar (1998) conducted prospective study in urban affluent, slum and rural communities of Bangladesh during February 1994 to February 1995. From each community 250 pregnant mothers were recruited in this study and at the end total 660 live births were studied to determine the incidence and risk factors of low birth weight. The study found that incidence of low birth weight was highest in urban slum (36.8%), followed by rural area (20.9%) and lowest in urban affluent community (18.3%) [216].

Kaneta (2000) carried out study the gender inequality in nutritional status and the effects of various soci-economic, demographic and health-programme factors on gender inequality in a remote rural area of Bangladesh. Measurements of mid-upper arm circumference (MUAC) were taken from 2016 children aged

then 5 years (50.8% male, 49.2% female) in 1994. Average MUAC for all children was 130mm; 33% were severely malnourished. Of the severely-malnourished children, 54.2% were female and 45.8% were male [217].

Rahman (1995) conducted study on socio-economic factors influencing the nutritional status of under five children in a rural village. Among the study children the average weight for age was 68.95% and height for age was 86.29% and weight for height was 4.67% of the National Center for Health Statistics (NCHS) standard [34].

CHAPTER III

METHODOLOGY

3. METHODS AND MATERIALS

3.1. Types of study:

This was a cross sectional comparative study.

3.2. Place of study:

The study was conducted in arsenic contaminated and non- contaminated villages of two selected Upazilla. The arsenic contaminated villages were in Sanmondi union of Sonargoan Upazilla under the district of Narayangonj. The villages are: Mirar Char (Jidder gaon), Mog Bazer (Mirer Char), Nagirpur, Bhabani Pur and Viti Khandi. The Arsenic non-contaminated villages were in Sirajdikhan Upazilla under the district of Munshigong. The arsenic non-contaminated villages are: Kusumpur, Chandandul, Shialdi, Rajdia and Tanguripara under Ischapura union.

3.3. Study Population:

Children of 5-14 years of age from arsenic contaminated and non-contaminated areas were included as study population. Exposed children were those who drank water from the tube wells having arsenic level > 50 μg / L. On the other hand, non-exposed group of children were those who drank water from the tube wells having arsenic level below 50 μg / L.

3.4. Selection Criteria:

Total one thousand thirty (1030) tube wells were examined for arsenic contamination. Out of 1030 tube wells 485 tube wells water having arsenic level > 50 µg / L, the rest of the tube wells arsenic level was below 50 µg / L. Children of 5-14 years from selected arsenic non-exposed and arsenic exposed areas were enlisted. Total 920 children from arsenic non-exposed area and 910 children from arsenic exposed area were included as study population. According to the stool examination report the children who were not suffering from helminthic infestation were included in this study. Total 1015(510 arsenic non-exposed and 505 arsenic exposed) were included as the respondent.

i. Inclusion Criteria:

Exposed children of 5-14 years of age irrespective of sex who were taking arsenic contaminated tube wells water > 50 µg/ L and stool examination report for helminthic infestation was found to be negative.

Non-exposed children, aged 5-14 years irrespective of sex who were taking tube wells water having arsenic level below 50 µg/ L and stool examination report was found to be negative for helminthic infestation.

ii. Exclusion Criteria:

Those children who suffered from severe diarrhea, amoebic dysentery, blood dysentery, measles any others major illness (Acute respiratory tract infection , Typhoid fever) within last three month, unwillingness of guardians of the children to participate in the study.

3.5. Sample Size and Sampling Technique:

Selection Procedure: Department of Public Health Engineering (DPHE) Tube well (TW) screening report► District ► Upazilla ►Union ►Village.

Tube well screening report was collected from assigned section of Department of Public Health Engineering (DPHE). From the DPHE Tube well screening report arsenic contaminated and non-contaminated district were listed. From the list one arsenic exposed (Munshigonj) and arsenic non-exposed (Narayangonj) district were selected. With in selected district the most affected Upazilla (proportion of arsenic contamination of tube well was highest) was selected as the arsenic contamination area which is Sonargaon. On the other hand the Upazilla having less arsenic contamination in the district, the least effected Upazilla was selected as the arsenic non- contaminated area which is Sirajdikhan. From selected Upazilla initially arsenic exposed and non-exposed villages were selected based on the DPHE.

Tube well screening report by Bangladesh Arsenic Mitigation and Water Supply Project [17]. According to the guideline of DPHE Tube well screening report arsenic contaminated village of Sonargaon Upazilla under Narayangonj district was selected purposively. Similarly an arsenic non-contaminated village of Sirajdikhan Upazilla under Munshigonj district was also selected. To locate arsenic contamination tube well, information was collected from local DPHE office for the particular village. In particular village to confirm the arsenic contamination tube wells were again examined for arsenic content. The children of the households whose Tube well was found to be arsenic contamination (>50 µg/L) after examination of water were included as study population of exposed area. Similarly the children of the household whose Tube well was found to be non-contamination (below 50 µg/L) after examination of water were included as the study population of non-exposed area.

NIPSOM Field Kit was used for determination of arsenic content of tube-well water in the field level. The result was reconfirmed by laboratory analysis by using AFS method (Atomic Fluorescence Spectrometry) [218].

Amongst the study population those who fulfilled the inclusion criteria were listed and from the list a total of 300 children from arsenic exposed group and 300 children from non-exposed group were selected as respondent of the study.

3.6. Respondents:

The selected respondent children of 5-14 years of age were interviewed for data collection. The parents of the selected children were also included for interview to assist their children during interview.

3.7. Data collection Instruments [52]:

Designed questionnaire was used for collection of data. The questionnaire was pre-tested before final use in the field. Information was collected on socio-demographic characteristics, duration of water use, daily consumption of water of children, arsenic level of tube-well water, other sources of water intake. Checklist was used for arsenic level of water, helminthic infestation of stool and anthropometrics measurements of the study children.

Interviewers:

Before data collection, interviewers were trained by the researcher on various aspect of data collection including questionnaire. A supervisor was selected to supervise the overall field work. After completion of training the interviewers collected data from the fields.

3.8. Data Collection Procedure:

The questionnaire was filled up by interview of children as well as their parents.

3.8.1. Interview:

Children and parents were interviewed. Face to face interview was carried out through a pre-tested questionnaire to collect information. The information was collected on age, sex, education, dwelling type, family size, monthly income, education of parents, sources of water intake, duration of use of tube-well water from the past sources, daily consumption of water of children, other sources of water intake, morbidity, dietary practice through 24- hours dietary recall questions.

3.8.2. Interviews of the Children-

After completion of interview, anthropometric measurements of the studied children were taken.

3.8.3. Anthropometric Measurements-

Data on height and weight of the children was taken through standard instruments. The procedures followed for measurement are described below

Weight:

A weighing scale was used to measure weight. Child was asked to stand on the weighing scale with minimum clothing, without shoes and without any weight in hand or touching or catching things. Three measurements were taken for each child and the mean was recorded.

Height:

Height was measured by height and weight machine. The height was recorded in a standing position of the subject without foot wear, feet together, knees straight and heel, buttocks and shoulder blades in contact with the vertical wall. Three measurements were taken for each child and the mean was recorded.

Body mass index (BMI):

BMI was calculated using the available weight and height by the following formula: [52,212,220,221].

$$BMI = \frac{\text{Weight in kg}}{(\text{Height in m})^2}$$

Classification of Nutritional status according to BMI: [145,212]

The nutritional status of the children was classified according to their BMI into three groups as below:

Sl no.	(Body mass index) BMI	Classification of Nutritional status
1	BMI is less than 5th percentile of BMI for the age and sex of reference population	Thinness (Having low BMI for the age and sex)
2	BMI is within 5th to 85th percentile of BMI for the age and sex of reference population	Normal (Having normal BMI for the age and sex)
3	BMI is greater than 85th percentile of BMI for the age and sex of reference population	Overweight (Having high BMI for the age and sex)

Formula for calculation of Z-score

In this study Z-score of weight for age, height for age and weight for height were used to assess the different aspects of nutritional status of the study children. Z-scores were calculated by the following formula:

$$\text{Z- Score} = \frac{\text{(Individual value) – (median value of reference population)}}{\text{Standard deviation value of reference population.}}$$

Standard deviation value and median value of reference population was calculated by transforming the original NCHS reference data. Normal Z- score varies from (– 2SD to + 2SD).

$$\text{HAZ} = \frac{\text{(Observed height) – (Median height of reference for given age and sex)}}{\text{Standard deviation of height of reference population}}$$

$$\text{WAZ} = \frac{\text{(Observed weight) – (Median weight of reference for given age and sex)}}{\text{Standard deviation of weight of reference population}}$$

$$\text{WHZ} = \frac{\text{(Observed weight for height) – (Median weight for height of reference for given sex and height)}}{\text{Standard deviation of weight for height reference population}}$$

The Z- score of weight for age, height for age and weight for height were categorized by the following cut off level to determine the nutritional status of the children [152].

Z- score values	Weight for age: (WAZ)	Height for age: (HAZ)	Weight for height: (WHZ)
+2sd to –2sd	Normal weight	Not stunted	Normal
Less than –2sd to –3sd	Moderately underweight	Moderately stunted	Moderately wasted
Less than –3sd	Severely underweight	Severely stunted	Severely wasted

3.9. Collection procedure and estimation of arsenic concentration of tube well water in field:

In the field arsenic level of Tube well water was analyzed by NIPSOM Field Kit method. Sample water was collected for further analysis for its arsenic level. Water was collected in a plastic bottle mixed with nitric acid. All these samples were properly labeled with the identification number of the corresponding patients.

3.9.1. Estimation of Arsenic:

The collected water sample was examined by atomic fluorescence spectrometry (AFS) at the Department of Occupational and Environmental Health, NIPSOM for reconfirmation of arsenic concentration.

Procedure of the Arsenic Test in Tube well Water by NIPSOM Field Kit [42].

1. First place the disc paper between the flattened surfaces of two glass flanges and secure the flanges with the clip.
2. Take 15 ml. of the water sample in the test tube (up to the lower mark).
3. Add 0.1 gm of reagent No. 1 (Potassium iodide), 0.5 gm of reagent No. 2 (Zinc) and 0.1 gm of reagent No. 3 (Stannous chloride) to the water sample taken in the test tube.
4. Now add 4 ml of 1:1 Hydrochloric acid (HCI) into the test tube (up to the higher mark) and insert the appropriate end of the flange into the test tube.
5. Then knock gently the lower end of the test tube for few seconds.
6. Allow the test tube to stand for 5 minutes, by this time the expected reactions will have taken place.
7. Remove the secured/clipped flanges from the test tube, remove the clip, separate the flanges and bring out the disc paper.

8. Compare the color change obtained on the disc paper with the color scale and record the content of arsenic in the format.

3.10. Assessment of Daily Water Consumption:

Daily consumption of water by each child was measured by assessing the number of glasses of water the children drinks per day according to the sample glass. The sample glass contains 250 ml of water.

3.11. Stool Examination:

To exclude children having helminthic infestation stool of the participant children were collected in a supplied plastic container mixed with 10% formalin. All these samples were properly labeled with the identification number of the corresponding children. The sample was examined under microscope to see ova of intestinal parasite in the laboratory of Microbiology and Parasitology, NIPSOM.

3.12. Dietary Assessment:

Dietary intakes of the study children were assessed using 24-hours recall method and details of all foods and drinks. Consumed foods and drinks by the children were recorded. The participants were shown various utensils such as measuring cups, spoons, glasses and plates to get nearest approximation of the amounts of food consumed. The serving weight of different food items was calculated using

Software FORTRAN 77. Equivalent raw food weight was calculated by using a conversion table for Bangladeshi foods formulated by the Institute of Nutrition and Food science [78,223,224,225,226].

3.13. Consent and Confidentiality:

At the beginning of interview, objectives of the study were briefly discussed among the respondents and their parents. It was also informed that the researcher would maintain confidentially of everything and the collected information would be used for research purpose only. The interview started after getting verbal consent of the respondent and their parents.

3.14. Analysis of Data:

All the data, which were cheeked for integrity and collected from the field, were entered for analysis by using SPSS software program in 11.5 versions. 24-hours recall food frequency data were analyzed in the Department of Institute of Nutrition and Food Science, Dhaka University.

3.15. Key Variables

- Age of the Children
- Father's Age
- Mother's Age
- No. of Family Member
- No. of children aged 5-14 years
- Qualification of Father
- Qualification of Mother
- Occupation of Father
- Occupation of Mother
- Monthly Income of the Family
- Family Type
- House Type
- Latrine Type
- Present Source of Drinking Water
- Source of Cooking Water

- Duration of Using Current Drinking Water Source (Tube well)
- Knowledge about Meaning of Red and Green Color of Tube Well
- Method of Rice Cooking (Letting gruel out or not)
- Frequency of Taking Pulses
- Any attack of Diarrhea, Dysentery, Measles or any major illness by the Children during last three months
- Any history of taking anti helminthic by the Children during last three months
- Arsenic Level of Present Tube Well Water
- Height of the Children
- Weight of the Children
- Presence or absence of worm in stool of the Children
- Dietary intake in last 24 hours

3.16. Operational definitions

- Arsenic Exposed Area: - Area where arsenic level in water is found to be > 50 µgm/liter.
- Arsenic Non-Exposed Area: - Area where arsenic level in water is found to be < 50 µgm/liter.
- Basha Bhat: - Cooked rice in which gruel is not let out.
- Cereal: - Cereals include rice and rice flour, wheat and wheat flour, maize and various products made of rice and wheat.
- Earning member: - A person who bring material in cash or kind for services rendered and for the use of goods. Services imply labor and organization of production while Goods imply land property and capital.
- Family member: - A family member is a person who depends on the family.
- Fan Gala Bhat: - Cooked rice in which gruel is let out.
- Household: - A dwelling unit where one or more persons live and eat together under a common cooking arrangement.
- Household Head: - A person who is the decision maker regarding different activities of the household.

- Income: - Material return in cash or kind received in exchange of Goods and Services in a particular period.
- Non-sanitary Latrine: - Open air Latrine or Latrines which are fixed but have no water seal.
- Sanitary Latrine: - Fixed Latrine which is water sealed.

CHAPTER IV

RESULT

4. RESULTS

A. Socio-demographic characteristics of the respondents

Table 1.1: Distribution of the families according to no of 5-14 years aged children and sex of study children

Characteristics	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
No. of 5-14 yr aged children							
1	158	52.7	154	51.3	312	52.0	0.188
2	115	38.3	105	35.0	220	36.7	
3	26	8.7	36	12.0	62	10.3	
4	1	.3	5	1.7	6	1.0	
Sex (index child)							
Male	152	50.7	154	51.3	306	51.0	0.870
Female	148	49.3	146	48.7	294	49.0	

*p value reached from chi square test

Table 1.1 shows information about the study children. Among the exposed group 52.7% families had one 5-14 year aged child, 38.3% families had two, 8.7% families had three and 0.3% families had four 5-14 year aged children. On the other hand among the non exposed group 51.3% families had one 5-14 year aged child, 35.0% families had two, 12.0% families had three and 1.7% families had four 5-14 year aged children. No significant difference of number of 5-14 years children was seen between the groups ($p>0.05$).

Regarding sex of the index children it was seen that among the exposed group 50.7 children were male and 49.3% were female. The proportions of male and female index children among the exposed group were 51.3% and 48.7% respectively. No statistically significant difference of sex of the index children was seen between the groups ($p>0.05$).

Table 1.2: Distribution of the children by age sex and type of subject

Characteristics	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
Age in years							
5-6.99	81	27.0	84	28.0	165	27.5	
7-9.99	114	38.0	131	43.7	245	40.8	
≥10	105	35.0	85	28.3	190	31.7	
***Mean ± SD (Range)**	8.8±2.6 (5-14)		8.4±2.4 (5-14)		8.6±2.5 (5-14)		0.110*
Sex							
Boys	154	51.3	152	50.7	306	51.0	0.870
Girls	146	48.7	148	49.3	294	49.0	

*p value reached from unpaired student's t test
p value reached from chi square test

Age and sex of the index children were described in table 1.2. Among the exposed families 27.0% of the study children were having age 5-6.99 years, 38.0% aged 7-9.99 years and the rest 35.0% aged ≥10 years. On the other hand among the non exposed families 28.0% of the study children were having age 5-6.99 years, 43.7% aged 7-9.99 years and the rest 28.3% aged ≥10 years. Similar age group was observed between the exposed and non exposed groups.

Regarding sex of the index children it was seen that among the exposed group 51.3% children were male and 48.7% were female. The proportions of male and female index children among the exposed group were 50.7% and 49.3% respectively. No statistically significant difference of sex of the index children was seen between the groups ($p>0.05$).

Table 1.3: Distribution of the children by age and sex

Age in years	*Sex*				*Total (n=600)*		*p value*
	Boys(n=306)		Girls (n=294)				
	No.	%	No.	%	No.	%	
5-6.99	83	27.1	82	27.9	165	27.5	
7-9.99	120	39.2	125	42.5	245	40.8	
≥10	103	33.7	87	29.6	190	31.7	
Mean ± SD (Range)	8.7±2.5 (5.0-14.0)		8.5±2.5 (5.0-13.9)		8.6±2.5 (5.0-14.0)		0.437

*p value reached from unpaired student's t test

Of the total 600 index children 306 were boys and 294 were girls. Among the boys 27.1% had age 5-6.99 years, 39.2% had age 7-9.99 years and the rest 33.7% had age ≥10.99 years. On the other hand among the girl index children 27.9% had age 5-6.99 years, 42.5% had age 7-9.99 years and the rest 29.6% had age ≥10.99 years. The mean age ±SD was 8.7±2.5 years among the boys and 8.5±2.5 years among the girls. No significant difference was seen between the boys and girls ($p > 0.05$) (Table 1.3).

Table 1.4: Distribution of the respondents by age of the parents

Characteristics	*Subjects*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
Age in years (Father)							
<35	35	11.7	33	11.0	68	11.3	
35-44	157	52.3	167	55.7	324	54.0	
45-54	87	29.0	85	28.3	172	28.7	
≥55	21	7.0	15	5.0	36	6.0	
Total	**300**	**100**	**300**	**100**	**600**	**100**	
Mean ± SD	41.2±6.9 (29-70)		41.6±6.6 (28-65)		41.4±6.8 (28-70)		0.530
Age in years (mother)							
<30	93	31.0	80	26.7	173	28.8	
30-39	157	52.3	172	57.3	329	54.8	
≥40	50	16.7	48	16.0	98	16.3	
Total	**300**	**100**	**300**	**100**	**600**	**100**	
Mean ± SD	32.5±5.9 (22-55)		32.5±5.7 (20-52)		32.5±5.8 (20-55)		0.922

*p value reached from unpaired student's t test

Mean age of the fathers was 41.4 years with SD ± 6.8 years. It was 41.2±6.9 among the exposed and 41.6±6.6 was among the non-exposed. Highest 54.0% was in the age group 35-44 years, which was among exposed group 52.3% and

in the non exposed group 55.7%. Non difference of age was seen between the groups ($p>0.05$)

Among the mothers mean age was 32.5 years with SD ± 5.8 years. It was 32.5±5.9 years among the exposed and 32.5±5.7 years among the non-exposed. In the combined group highest 54.8% was in the age group 30-39 years. In the exposed group 52.3% and in the non exposed group 57.3% aged 30-39 years. No statistically significant difference was observed between the groups ($p>0.05$) (Table 1.4).

Table 1.5: Distribution of the respondents by sex

Sex (respondent)	*Subjects*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Male	50	16.7	47	15.7	97	16.2	0.739
Female	250	83.3	253	84.3	503	83.8	

*p value reached from chi square test

Table 1.5 shows sex of the respondents. Out of the total 600, 97 (16.2%) were male and 503 (83.8%) were female. In the exposed group male and female proportions were 16.7 % and 83.3%. On the other hand male and female proportions in the non exposed group were 15.7% and 84.3% respectively. No significant difference of sex was found between exposed and non exposed groups ($p>0.05$).

Table 1.6 Distribution of the respondents by father's level of education

Characteristics	*Subjects*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
Level of education (Father)							
No education	62	20.7	67	22.3	129	21.5	
Primary	141	47.0	112	37.3	253	42.2	
Secondary	63	21.0	67	22.3	130	21.7	
Passed SSC	21	7.0	36	12.0	57	9.5	
Higher Secondary	5	1.7	4	1.3	9	1.5	
Passed HSC	4	1.3	10	3.3	14	2.3	
Graduate and above	4	1.3	4	1.3	8	1.3	
Level of education (Father)							
Illiterate	62	20.7	67	22.3	129	21.5	0.619
Literate	238	79.3	233	77.7	471	78.5	

*p value reached from chi square test

About education of the fathers it was seen that out of the total 600 maximum 42.2% had primary level of education, which was 47.0% among the exposed group and 37.3% among the non exposed group. Of the total 600, 129(21.5%) were illiterate and 471(78.5%) were literate. The proportions of illiterate were 20.7% among the exposed group and 22.3% among the non exposed group. On the other hand the proportions of literate were 79.3% and 77.7% among the exposed and non exposed groups respectively. No significant difference of education was observed between the groups ($p>0.05$) (Table 1.6).

Table 1.7: Distribution of the respondents by mother's level of education

Characteristics	*Subjects*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Level of education (Mother)							
No education	71	23.7	79	26.3	150	25.0	
Primary	163	54.3	133	44.3	296	49.3	
Secondary	52	17.3	66	22.0	118	19.7	
Passed SSC	9	3.0	16	5.3	25	4.2	
Higher Secondary	1	.3	1	.3	2	.3	
Passed HSC	2	.7	5	1.7	7	1.2	
Graduate and above	2	.7	0	.0	2	.3	
Level of education (Mother)							
Illiterate	71	23.7	79	26.3	150	25.0	0.451
Literate	229	76.3	221	73.7	450	75.0	

*p value reached from chi square test

Regarding mothers' education it was seen that out of the total 600 maximum 49.3% had primary level of education. Which was 54.3% among the exposed group and 44.3% among the non exposed group. Of the total 600, 150(25.0%) were illiterate and 450(75.0%) were literate. The proportions of illiterate were 23.7% among the exposed group and 26.3% among the non exposed group. On the other hand the proportions of literate were 76.3% and 73.7% among the exposed and non exposed groups respectively. No significant difference of education was observed between the groups ($p>0.05$) (Table 1.7).

Table 1.8: Distribution of the respondents by type of family

Type of family	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Joint	193	64.3	176	58.7	369	61.5	0.154
Nuclear	107	35.7	124	41.3	231	38.5	

*p value reached from chi square test

Type of family of the respondents is shown in Table 1.8. Of the total 600 families 369(61.5%) families were joint families and 231(38.5%) were nuclear families. Among the exposed group 64.3% were joint families and 35.7% were nuclear families. On the other hand among the non exposed group the proportions of joint and nuclear families were 58.7% and 41.3% respectively. The types of family was found to be similar between the groups ($p<0.05$).

Table 1.9: Distribution of the respondents by profession of the household

Main profession of the household	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Farmer	134	44.7	136	45.3	270	45.0	0.995
Laborer	36	12.0	37	12.3	73	12.2	
Business	73	24.3	72	24.0	145	24.2	
Service	57	19.0	55	18.3	112	18.6	

*p value reached from chi square test

Table 1.9 reveals the profession of the respondents. Among the exposed group 44.7 % were Farmer s, 12.0 % were Laborer, 24.3 % were doing Business and 19.0 % Services. On the other hand non-exposed group the proportions of Farmers, Laborers, Businessmen and Service holders were 45.3%, 12.3%, 24.0% and 18.3% respectively. No significant difference of profession was observed between the exposed and non exposed groups ($p>0.05$).

Table 1.10: Distribution of the respondents by family size

Family size	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Up to 4	80	26.7	94	31.3	174	29.0	
5	129	43.0	91	30.3	220	36.7	
6	80	26.7	95	31.7	175	29.2	
≥7	11	3.7	20	6.7	31	5.2	
Mean ± SD (Range)	5.04±.9 (3-7)		5.11±1.0 (3-8)		5.08±.9 (3-8)		0.411

*p value reached from unpaired student's t test

Mean family size was found to be 5.08±.9. It was 5.04±.9 among the exposed group and 5.11±1.0 among the non exposed group. Student's t-test reveals that there was no statistically significant difference of family size between the exposed and non exposed groups ($p>0.05$) (Table 1.10).

Table 1.11: Distribution of the respondents by monthly family income

Monthly Family Income (Tk.)	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
<3000	30	10.0	48	16.0	78	13.0	
3000-5999	227	75.7	220	73.3	447	74.5	
≥6000	43	14.3	32	10.7	75	12.5	
Mean ± SD (Range)	4015.0±1246.6 (2000-7000)		3861.7±1348.7 (1500-15000)		3938.3±1299.9 (1500-15000)		0.149

*p value reached from unpaired student's t test

Table 2.1 describes the monthly income of the families. Among the exposed group 10.0% families had monthly income of <3000 taka, 75.7% had 3000-5999 taka and the rest 14.3% families had monthly income of taka ≥6000. On the other hand among the non exposed group 16.0% families had monthly income of <3000 taka, 73.3% had 3000-5999 taka and the rest 10.7% families had monthly income of taka ≥6000. Monthly income was found to be similar between the exposed and non exposed groups. No statistical difference was observed ($p>0.05$) (Table 1.11).

B. Environmental condition

Table 2.1: Distribution of the respondents by housing condition

Housing condition	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Brick built	43	14.3	42	14.0	85	14.2	0.985
Tin roof, tin wall & mud floor	143	47.7	145	48.3	288	48.0	
Tin roof, brick wall & mud floor	114	38.0	113	37.7	227	37.8	

*p value reached from chi square test

Table 2.1 describes the housing condition of the families. Among the exposed families 14.3% families had brick built houses. Tin roof, tin wall and mud floored houses were seen among 47.7 % families and 38.0 % of them had Tin roof, brick wall and mud floored houses. On contrary among the non exposed families 14.0% had brick built houses, 48.3 % tin roof, tin wall and mud floor, and 37.7 % families had tin roof, brick wall and mud floor houses. There was no significant difference in the housing condition of the families ($p>0.05$).

Table 2.2: Distribution of the respondents by type of latrine used

Type of latrine used	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Sanitary	163	54.3	168	56.0	331	55.2	0.681
Non sanitary	137	45.7	132	44.0	269	44.87	

*p value reached from chi square test

Regarding type of the latrines the proportion of families with sanitary latrines among the exposed group was 54.3%. It was 56.0% among the non-exposed. Again non sanitary latrines were possessed by 45.7% exposed and 44.0% non exposed families. No statistically significant difference was found in latrine type of the families ($p>0.05$) (Table 2.2).

Table 2.3: Distribution of the respondents by sources of drinking and cooking water

Sources of water	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Sources of drinking water							
Tube well	298	99.3	300	100.0	598	99.7	-
Pond	2	.7	0	.0	2	.3	
Sources of cooking water							
Tube well	49	16.3	232	77.3	281	46.8	0.001
Others	251	83.7	68	22.7	319	53.2	

*p value reached from chi square test

Table 2.3 shows sources of water by the families. Among the exposed families 99.3% uses tube well water for drinking and 0.7% used pond water. On the other hand all of the non exposed families used tube well water for drinking. Regarding cooking water it was seen that 16.3% of the exposed families used tube well water for cooking and 83.7% used other sources. Among the non exposed families 77.3% used tube well water for cooking and the rest 22.7% used other sources.

Table 2.4: Distribution of the respondents by media exposure

Media exposure	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Present	202	67.3	187	62.3	389	64.8	0.200
Absent	98	32.7	113	37.7	211	35.2	

*p value reached from chi square test

Table 2.4 shows that 67.3% of the exposed families had media exposure i.e. they possessed radio and/or TV. On the other hand of the non exposed families 62.3% was exposed to media. No significant difference was observed between the groups ($p>0.05$).

C. Consumption of water

Table 3.1: Distribution of the respondents by consumption of water

Characteristics	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		**Non Exposed (n=300)**				
	No.	**%**	**No.**	**%**	**No.**	**%**	
Duration of consumption of water (years)							
<15	45	15.0	13	4.3	58	9.7	
15-19	107	35.7	103	34.3	210	35.0	
20-24	90	30.0	149	49.7	239	39.8	
≥25	58	19.3	35	11.7	93	15.5	
Mean ± SD (Range)	19.5±5.1 (1.0-33.0)		20.0±3.2 (11.0-27.0)		19.8±4.3 (1.0-33.0)		0.143
Amount of water consumed (ml)							
Up to 1500	147	49.0	63	21.0	210	35.0	
1501-2000	84	28.0	117	39.0	201	33.5	
2001-2500	41	13.7	82	27.3	123	20.5	
≥2501	28	9.3	38	12.7	66	11.0	
Mean ± SD (Range)	1775.0±532.6 (1000.0-3250.0)		2060.0±503.5 (1000.0-3000.0)		1917.5±537.1 (1000.0-3250.0)		0.001

*p value reached from unpaired student's t test

Water consumption by the families is described in the table 3.1. Among the exposed group 15.0% of the families were using the current water source for less than 15 years. 35.7% families are using the current water source for 15-19 years, 30.0% for 20-24 years and the rest 19.3% for ≥25 years. On the other hand among the non- exposed group 4.3% of the families were using the current water source for less than 15 years. 34.3% families were using the current water source for 15-19 years, 49.7% for 20-24 years and the rest 11.7% for ≥25 years. The mean duration of uses was 19.5±5.1 among the exposed families and 20.0±3.2 among the non exposed families. No statistically significant difference was seen regarding duration of use of the current water sources between the groups ($p>0.05$).

Regarding water consumption it was seen that among the exposed group 49.0% consumed up to 1500 ml of water daily, 28.0% consumed 1501-2000 ml, 13.7% consumed 2001-2500 ml and 9.3% consumed ≥2501 ml of water daily. On the other hand among the non exposed 21.0% consumed up to 1500 ml of water daily, 39.0% consumed 1501-2000 ml, 27.3% consumed 2001-2500 ml and 12.7% consumed ≥2501 ml of water daily. Mean water consumption was 1775.0±532.6 ml among the exposed and 2060.0±503.5 among the non exposed ($p<0.01$)

D. Dietary habit

Table 4.1: Distribution of the respondents by dietary habit

Characteristics	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
No. of wet rice taken per week by children							
0	106	35.3	61	20.3	167	27.8	
1-2	162	54.0	128	42.7	290	48.3	
3-4	28	9.3	93	31.0	121	20.2	
≥5	4	1.3	18	6.0	22	3.7	
Mean ± SD (Range)	1.3±1.2 (0-7)		2.1±1.5 (0-7)		1.7±1.4 (0-7)		0.001
Frequency of taken pulses per week by children							
0	10	3.3	0	.0	10	1.7	
1-2	150	50.0	51	17.0	201	33.5	
3-4	106	35.3	134	44.7	240	40.0	
5+	34	11.3	115	38.3	149	24.8	
Mean ± SD (Range)	2.7±1.4 (0-7)		4.2±1.8 (1-7)		3.5±1.8 (0-7)		0.001

*p value reached from unpaired student's t test

Among the exposed group 35.3% do not take wet rice. 54.0% take wet rice 1-2 times, 9.3% takes 3-4 times and 1.3% takes wet rice 5 or more times per week. On the other hand among the non exposed group 20.3% did not take wet rice. 42.7% take wet rice 1-2 times, 31.0% takes 3-4 times and 6.0% takes wet rice 5 or more times per week. Significant difference of taking wet rice between the groups was seen ($p<0.01$).

Among the exposed group 3.3% did not take pulses, 50.0% take pulses 1-2 times per week. 35.3% take pulses 3-4 times per week and 11.3% take pulses 5 times or more per week. On the contrary among the non-exposed 17.0% take pulses 1-2 times per week. 44.7% take pulses 3-4 times per week and 38.3% take pulses 5 times or more per week. Significant difference of taking pulses was observed between the groups ($p<0.01$) Table 4.1.

Table 4.2: Nutrients taken per day by the study children

Principal nutrients	Amount of principal nutrients taken per day		P value
	Exposed (Mean ± SD)	Non-exposed (Mean ± SD)	
Total weight (g)	533 ± 282	530 ± 240	>0.05, df 598
Energy (kcal)	1018 ± 448	1012 ± 394	>0.05, df 598
Protein (g)	33 ± 28	30 ± 24	0.212, df 598
Fat (g)	9 ± 8	11 ± 22	0.124, df 598
Carbohydrate (g)	224 ± 76	222 ± 76	>0.05, df 598

P-value reached from student's t-test

Table 4.2 shows the average amount of nutrients taken by the studied children per day. For collecting information about the intake of foods from the studied children of the groups, 24-hours recalled questionnaire was used. Independent sample t-tests were performed to find out any difference of mean intake of principal nutrients by the studied children. It was observed that there was no significant difference of intake of nutrients by the study children between the two groups.

Table 4.3: Minerals and vitamins taken per day by the study children

Nutrients	*Subjects*		*p-value*
	Exposed (Mean ± sd)	**Non-Exposed (Mean ± sd)**	
Ca (mg)	290.1382±249.39022	305.0086±214.16056	.963
Iron (mg)	9.1868±6.91798	9.2718±7.31282	.241
Ribo (mg)	.4494±.28100	.4931±.66097	.205
Thia (mg)	.7175±.30978	.7983±.30221	.728
Zinc (gm)	4.3653±3.12078	4.3429±2.51017	.492

p-value reached from student's t-test

Table 4.3 shows the average amount of minerals and vitamins taken by the studied children per day. It was observed that there was no significant difference of intake of nutrients by the study children between the two groups.

Table 4.4: Correlation of BMI with principal nutrients taken by the study children

Correlation of BMI with principal nutrient		
Principal nutrients	**Pearson Correlation**	**p**
Energy	0.277	<0.01
Protein	0.125	<0.01
Carbohydrate	0.293	<0.01
Fat	0.095	<0.01
Vit. A	0.297	<0.01
Vit. C	0.145	<0.01

The food intake of the children was studied by 24-hour recall questionnaire. The amount of the principal nutrients was calculated. BMI of the study children were found to have correlation with intake of six important nutrients at 0.01 levels (Table 4.4).

E. Nutritional status

Table 5.1 Age and sex wise mean distribution of weight and height of the children

Age in yrs	no	*Weight (kg)*					Height (cm)				
		Boys		Girls	p value		Boys		Girls	p value	
		Mean ±SD	no.	Mean ±SD		no.	Mean ±SD	no.	Mean ±SD		
5-6.99	83	16.1±2.6	82	15.7±2.3	0.349	83	106.8±6.6	82	106.1±6.8	0.459	
7-9.99	120	20.4±3.0	125	19.8±3.8	0.166	120	120.2±6.0	125	119.6±7.3	0.429	
≥10	103	29.4±6.9	87	32.5±7.6	0.004	103	136.4±9.3	87	138.9±8.4	0.053	

Weight and height of the study children according to sex and age group are shown in table 5.1. Mean weight ±SD of the boys in the age group 5-6.99 years, 7-9.99 years and ≥10 years were 16.1±2.6 kg, 20.4±3.0 kg and 29.4±6.9 kg respectively. Again mean height ±SD of the boys in the age group 5-6.99 years, 7-9.99 years and ≥10 years were 106.8±6.6 cm, 120.2±6.0 and 136.4±9.3 cm respectively. On the other hand mean weight ±SD of the girls in the age group 5-6.99 years, 7-9.99 years and ≥10 years were 15.7±2.3 kg, 19.8±3.8 kg and 32.5±7.6 kg respectively. Again mean height ±SD of the girls in the age group 5-6.99 years, 7-9.99 years and ≥10 years were 106.1±6.8 cm, 119.6±7.3 and 138.9±8.4 cm respectively.

Table 5.2 Age wise mean distribution of weight and height of the children (boys)

Age in yrs	*Weight (kg)*					Height (cm)				
	Exposed		Non exposed		p value	Exposed		Non exposed		p value
	no.	Mean ±SD	no.	Mean ±SD		no.	Mean ±SD	no.	Mean ±SD	
5-6.99	45	15.9±2.7	38	16.3±2.3	0.443	45	105.9±6.3	38	108.0±6.9	0.150
7-9.99	62	20.0±2.8	58	20.9±3.1	0.095	62	119.2±5.7	58	121.4±6.2	0.049
≥10	45	27.5±6.2	58	30.9±7.0	0.010	45	134.5±7.7	58	137.9±10.2	0.069

Among the 306 male children 83 had age between 5-6.99 years. Of them 45 were in the exposed group and they had mean weight 15.9 kg with SD ±2.7 kg and mean height 105.9 cm with sd ±6.3 cm. In this age group 5-6.99 years there were 38 boys who were not exposed to arsenic contamination having mean weight 16.3 kg with SD±2.3 kg and mean height 108.0 cm with Sd ±6.9 cm. Of the total 120 male children of age group 7-9.99 years 62 were exposed to arsenic contamination and they had mean weight 20.0 kg with SD ±2.8 kg and mean height 119.2 cm with SD ±5.7 cm. In this age group there were 58 boys who were not exposed to arsenic and they had mean weight 20.9 kg with SD ±3.1 kg

and mean height 121.4 cm with SD ±6.2 cm. Among the boys of age ≥10 years 45 were in the exposed group having mean weight 27.5 kg with SD ±6.2 kg and mean height 134.5 cm with SD ±7.7 cm. The rest 58 non-exposed boys of age group 10-14 years had mean weight 30.9 kg with SD ±7.0 kg and mean height 137.9 cm with SD ±10.2 cm (Table 5.2).

to 14 years 40 were in the exposed group having mean weight 31.6 kg with SD ±7.5 kg and mean height 137.6 cm with SD ±8.5 cm. The rest 47 exposed girls of age group ≥10 years had mean weight 33.3 kg with SD ±7.7 kg and mean height 140.1 cm with SD ±8.2 cm (Table 5.3).

Table 5.4 Distribution of the children by age and height for age

Age in years	*Height for age*						*p value*
	Normal (n=423)		Moderate stunting (n=141)		Severe stunting (n=36)		
	No.	%	No.	%	No.	%	
5-6.99	111	67.3	42	25.5	12	7.3	0.746
7-9.99	179	73.1	54	22.0	12	4.9	
≥10	133	70.0	45	23.7	12	6.3	

Stunting status of the children according to age group was shown in Table 5.4. In the age group 5-6.99 years 67.3% were having normal height for age, 25.5% had moderate stunting and 7.3% had severe stunting. In the age group 7-9.99 years 73.1% were having normal height for age, 22.0% had moderate stunting and 4.9% had severe stunting. And in the age group ≥10 years 70.0% were having normal height for age, 23.7% had moderate stunting and 6.3% had severe stunting. No significant difference in the status of height for age was seen by age group ($p>0.05$).

Table 5.5 Distribution of the children by age and weight for age

Age in years	*Weight for age*						*p value*
	Normal (n=547)		Moderate underweight (n=52)		Severe underweight (n=1)		
	No.	%	No.	%	No.	%	
5-6.99	141	85.5	23	13.9	1	0.6	
7-9.99	227	92.7	18	7.3	0	0.0	0.14 df=2
≥10	179	94.2	11	5.8	0	0.0	

Underweight status of the study children according to age group was shown in Table 5.5. In the age group 5-6.99 years 85.5% were having normal weight for age, 13.9% had moderate underweight and 0.6% had severe underweight. In the age group 7-9.99 years 92.7% were having normal weight for age, 7.3% had moderate underweight and 0.0 % had severe underweight. And in the age group ≥10 years 94.2% were having weight for age, 5.8 % had moderate underweight and 0.0% had severe underweight. The underweight status was found to be associated with the age group of the children ($p<0.05$).

Table 5.6 Distribution of the children by age and weight for height

Age in years	*Weight for height*						*p value*
	Normal (n=566)		Moderate wasting (n=29)		Severe wasting (n=5)		
	No.	%	No.	%	No.	%	
5-6.99	147	89.1	16	9.7	2	1.2	
7-9.99	231	94.3	11	4.5	3	1.2	0.002
≥10	188	98.9	2	1.1	0	.0	

Wasting status of the study children according to age group was shown in Table 5.6. In the age group 5-6.99 years 89.1% were having normal weight for height, 9.7% had moderate wasting and 1.2% had severe wasting. In the age group 7-9.99 years 94.3% were having normal weight for height, 4.5% had moderate wasting and 1.2% had severe wasting. And in the age group ≥10 years 98.9% were having normal weight for height, 1.1% had moderate wasting and none had severe wasting. The wasting status was found to be associated with the age group of the index children ($p<0.05$)

Table 5.7 Distribution of the children by sex and height for age

Sex	*Height for age*						*p value*
	Normal (n=423)		**Moderate stunting (n=141)**		**Severe stunting (n=36)**		
	No.	**%**	**No.**	**%**	**No.**	**%**	
Boys	222	72.5	68	22.2	16	5.2	0.490
Girls	201	68.4	73	24.8	20	6.8	

Stunting status of the study children according to sex was shown in Table 5.7. Among the boys 72.5% were having normal height for age, 22.2% had moderate stunting and 5.2% had severe stunting. On the other hand among the girls 68.4% were having normal height for age, 24.8% had moderate stunting and 6.8% had severe stunting. No significant difference in the status of height for age was seen by sex of the children ($p>0.05$).

Table 5.8 Distribution of the children by sex and weight for age

Sex	*Weight for age*						*p value*
	Normal (n=547)		Moderate underweight (n=52)		Severe underweight (n=1)		
	No.	%	No.	%	No.	%	
Boys	288	94.1	18	5.9	0	.0	0.027
Girls	259	88.1	34	11.6	1	.3	

Underweight status of the study children according to sex was shown in Table 5.8. Among the boys 94.1% were having normal weight for age, 5.9% had moderate underweight and none had severe underweight. On the other hand among the girls 88.1% were having normal weight for age, 11.6% had moderate underweight and 0.3% had severe underweight. Statistically significant difference of weight for age was seen by sex of the children ($p<0.05$).

Table. 5.9 Distribution of the children by sex and weight for height

Sex	*Weight for height*						*p value*
	Normal (n=566)		Moderate wasting (n=29)		Severe wasting (n=5)		
	No.	%	No.	%	No.	%	
Boys	289	94.4	15	4.9	2	.7	0.883
Girls	277	94.2	14	4.8	3	1.0	

Wasting status of the study children according to sex was shown in Table 5.9. Among the boys 94.4% were having normal weight for height, 4.9% had moderate wasting and 0.7% had severe wasting. On the other hand among the girls 94.2% were having normal weight for height, 4.8% had moderate wasting and 1.0% had severe wasting. No significant difference in the status of weight for height was seen by sex of the index children ($p>0.05$).

Table 5.10 Distribution of the children by nutritional status

Characteristics	*Subject*				*Total (n=600)*		*p value*
	Exposed (n=300)		Non Exposed (n=300)				
	No.	%	No.	%	No.	%	
Height for age							
Normal	199	66.3	224	74.7	423	70.5	0.025
Stunting	101	33.7	76	25.3	177	29.5	
Weight for age							
Normal	264	88.0	283	94.3	547	91.2	0.006
Underweight	36	12.0	17	5.7	53	8.8	
Weight for height							
Normal	277	92.3	289	96.3	566	94.3	0.034
Wasting	23	7.7	11	3.7	34	5.7	

p value reached from chi square test

Stunting, Underweight and Wasting status of the children according to the areas (exposed and non-exposed) are shown in Table 5.10.

Out of the 300 exposed children 66.3% were with normal height for age and 33.7% had stunting. On the other hand out of the 300 non-exposed children, 74.7% had normal height for age and 25.3% were having stunting. Stunting was found associated with exposure ($p<0.01$).

Regarding weight for age it was seen that among the exposed children 88.0% were normal and 12.0% were underweight. On the other hand in the non-exposed area 94.3% children had normal weight for age and 5.7% were underweight. Here also significant difference in weight for age found to be associated with exposure ($p<0.05$).

In the exposed area 92.3% had normal weight for height and 7.7% were having wasting. On the other hand about 96% of the non-exposed children had normal weight for height and 4% had some degree of wasting. Wasting status was found to be associated with exposure ($p<0.05$).

Table 5.11 Distribution of the nutritional status of the children according to age group

n=600

Age group	Nutritional Status	Areas		Total	χ^2	df and p value
		Exposed	Non-exposed			
5–7 years	Underweight	70	55	125	χ^2= 0.196	p=.658 df=1
	Normal & overweight	80	70	150		
8–10 years	Underweight	53	39	92	χ^2=4.398	p < 0.05 df=1
	Normal & overweight	42	57	99		
11–14 years	Underweight	24	19	43	χ^2=5.708	p < 0.05 df=1
	Normal & overweight	31	60	91		
Total 5–14 years	Underweight	147 (49.0)	113 (37.7)	260 (100.0)	χ^2= 7.846	p < 0.01 df=1
	Normal and overweight	153 (51.0)	187 (62.3)	340 (100.0)		

*Figures in the parentheses show percentages

The effects of arsenic on nutritional status of the children in the two areas were examined within the age groups of the children. It was seen that in the exposed area out of the total 300 children 153 (51.0%) children were having normal BMI for age or had high BMI (overweight) and 147 (49.0%) children were underweight. On the other hand in the non-exposed group out of the total 300 children 187 (62.3%) children had normal BMI or were overweight and the rest 113 (36.7%) children were underweight. The difference of nutritional status in the two groups was found statistically significant ($p < 0.01$). In the age group of 5-7 years difference was not statistically significant, but the difference was statistically significant in higher age group 11 – 14 years, (Table-5.11).

Table 5.12 Mean BMI of the children

Area	Mean	Std. Deviation	Std. Error Mean
Exposed	14.4231	2.20768	.12746
Non-exposed	14.8740	2.16722	.12512

Mean BMI of the children in the exposed group was 14.42 ± 2.21 kg/m^2. It was 14.87 ± 2.17 kg/m^2 in the non-exposed group. BMI of the non-exposed group children were found to be significantly higher than of the exposed group children ($p<0.05$).

Table-5.13 BMI of the study children according to income and occupation of the main earning member

BMI group of the children	Monthly income of the families (taka)			Occupation of the main earning member			
	<3000	3000-5999	≥6000	Farmer	Laborer	Business	Service
Thinness (Low BMI for Age), n=260	32	194	34	95	38	65	62
Normal BMI for Age, n=320	45	236	39	110	59	70	81
Overweight (High BMI for Age), n=20	1	17	2	4	2	7	7
Total	78	447	75	209	99	142	150
χ2 and p value	χ^2=1.873, p=.759			χ^2=5.965, p=.427			

BMI of the children was studied according to the monthly income of the families which is shown in table-5.13. It revealed that out of a total 260 children with low BMI, 32 were from families having monthly income of taka less than 3000. The relation of BMI with monthly income of the families was found to be non significant (χ^2=1.873, p=.759).

Four categories of occupation were found as the source of income among the members of the families. No association was found between BMI of the studied children with the occupation (χ^2=5.965, p=.427).

Table-5.14 BMI of the children according to family type and number of family members

BMI group of the children	Type of the family		Number of family members			
	Joint family	Nuclear family	Up to 4	5 persons	6 persons	≥7 person
Thinness (Low BMI for Age), n=260	171 (65.77%)	89 (34.23%)	71	107	73	9
Normal BMI for Age, n=320	190 (59.37%)	130 (40.63%)	92	106	100	22
Overweight (High BMI for Age), n=20	8 (40.0%)	12 (60.0%)	11	7	2	0
Total	369(61.5%)	231(88.5%)	174	220	175	31
χ2 and P value	χ^2=6.516, p=.038		χ^2=14.868, p=.021, df=6			

Table-5.14. Shows relationship of BMI of the children with the type of the families. Majority (65.77%) of the children having thinness (Low BMI for Age) were from joint families where as majority (60.00%) of the children having

overweight (High BMI for Age) were from nuclear families. The association was found to be significant (χ^2=6.516, p <0.05).

Out of the total 320 children with normal BMI, 92 came from families having of family members up to 4, 106 came from families having of 5 members, 100 came from families having of 6 members and rest 22 were from families having members ≥ 7 persons. Overweight (High BMI for Age) children were mostly from families with less number of family members. The relation of BMI of children with number of family members was found statistically significant (χ^2=14.868, p=.021).

Table-5.15 Children's BMI according to type of house they lived in and type of the latrine used by the families

BMI group of the children	House type of the respondent			Type of latrine used	
	Brick built	Tin roof, tin wall & mud floor	Tin roof, brick wall & mud floor	Sanitary	Non-Sanitary
Thinness (Low BMI for Age), n=260	26	168	66	53	207
Normal BMI for Age, n=320	41	201	78	208	112
Overweight (High BMI for Age), n=20	4	15	1	8	12
Total	71	384	145	269	331
$\chi 2$ and P value		χ^2=5.795, p=.215		χ^2=115.644, p=.000	

BMI of the children was not found to be statistically related with the house type of the families Tables-5.15, (χ^2=5.795, p=0.215).

Out of 320 children with normal BMI, 208 were from families having sanitary latrine and 112 from families with non-sanitary latrines. BMI of the children had significant relation with latrine used by the families ($p < 0.001$).

Table 5.16: Nutritional Status of the Children by Height for Age Z-Scores according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Stunting [a]	Normal [b]		
Exposed	101 (33.7%)	199 (66.3%)	300 (100.0%)	
Non-Exposed	76 (25.3%)	224 (74.7%)	300 (100.0%)	χ^2=5.009, p= 0.025
Total	177 (29.5%)	423 (70.5%)	600 (100.0%)	

a (HAZ < –2 sd for the age and sex)

b (HAZ +2sd to –2 sd for the age and sex)

Nutritional status of the children was measured by z-score scales. It was seen that out of the 300 children with arsenic exposure 101 (33.7%) had stunting and the rest 66.3% were normal. On the other hand among the 300 arsenic non-exposed children 76 (25.3%) had stunting and the rest 224 (74.7%) were normal. It was found that stunting was significantly higher (χ^2 =5.009, p= 0.025) among the exposed children then the non-exposed children.

Table 5.17: Nutritional Status of the Children by Weight for Age Z-Scores according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Underweight [a]	Normal [b]		
Exposed	36 (12.0%)	264 (88.0%)	300 (100.0%)	
Non-Exposed	17 (5.7%)	283 (94.3%)	300 (100.0%)	χ^2=7.471, p= 0.006
Total	53 (8.8%)	547 (91.2%)	600 (100.0%)	

a (WAZ < –2 sd for the age and sex)

b (WAZ +2sd to –2 sd for the age and sex)

Table 5.17 shows Nutritional status of the study children by Weight for Age z-scores according to arsenic exposure. Out of the total 300 children who were exposed to arsenic contaminated water 36 (12.0%) were underweight and 264 (88.0%) were normal. On the other side 53 (8.8%) of the 300 non-exposed children were underweight and 283 (94.3%) were normal. It was found that under weight was significantly higher (χ^2 =7.471, p= 0.006) among the exposed children then the non-exposed children.

Table 5.18: Nutritional Status of the Children by Weight for Height Z- Scores according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Wasting [a]	Normal [b]		
Exposed	23 (7.7%)	277 (92.3%)	300 (100.0%)	
Non-Exposed	11 (3.7%)	289 (96.3%)	300 (100.0%)	χ^2=4.490, p= 0.034
Total	34 (5.7%)	566 (94.3%)	600 (100.0%)	

a (WHZ < –2 sd for the age and sex)

b (WHZ +2sd to –2 sd for the age and sex)

Weight for Height z- scores measurements shows that 7.7% of the exposed children were having wasting and 92.3% were normal. Again only 3.7% of the non-exposed children were wasted and the rest 96.3% children of this group were normal. It was found that wasting was significantly higher (χ^2 =7.471, p= 0.006) among the exposed children then the non-exposed children.

Table 5.19: Nutritional Status of the Children by Combined z- scores (HAZ, WAZ & WHT) according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Under nutrition [a]	Normal [b]		
Exposed	119 (39.7%)	181 (60.3%)	300 (100.0%)	
Non-Exposed	84 (28.0%)	216 (72.0%)	300 (100.0%)	χ^2=9.120, p= 0.003
Total	203 (33.8%)	397 (66.2%)	600 (100.0%)	

a (Abnormal in any z- score scale)

b (Normal in all z- score scales)

Table 5.19. Reveals that out of the total 300 arsenic exposed children 119 (39.7%) were with under nutrition in any of the z- score scales (HAZ, WAZ or WHT) and 181 (60.3%) were normal in all z- score scales. On the other hand 84 (28.0%) of the 300 non-exposed children were with under nutrition in any of the z- score scales (HAZ, WAZ or WHT) and the rest 216 (72.0%) were normal in all z- score scales. Nutritional status of the children by combined z- scores (HAZ, WAZ & WHT) was found associated with arsenic exposure ($p<0.01$).

Table 5.20: Nutritional Status of the Children by BMI according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Thinness [a]	Normal [b]		
Exposed	147 (49.0%)	153 (51.0%)	300 (100.0%)	
Non-Exposed	113 (37.7%)	187 (62.3%)	300 (100.0%)	χ^2 =7.846, p= 0.005
Total	260 (43.3%)	340 (56.7%)	600 (100.0%)	

a (BMI <5th centile for the age and sex)

b (BMI ≥85th centile for the age and sex)

Table 5.20 shows nutritional status of the children by BMI. A total of 147 (49.0%) children among the exposed group were having thinness (BMI <5th centile for the age and sex) and 153 (51.0%) were normal (BMI ≥85th centile for the age and sex). Again it was seen that out of the total 300 non-exposed children 113 (37.7%) had thinness (BMI <5th centile for the age and sex) and 187 (62.3%) were normal (BMI ≥85th centile for the age and sex). BMI based nutritional status of the children was also found to be associated with the exposure to arsenic ($p<0.01$).

Table 5.21: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHZ) and BMI according to Arsenic Exposure

Area	Nutritional Status		Total	χ^2, p value
	Under nutrition [a]	Normal [b]		
Exposed	200 (66.7%)	100 (33.3%)	300 (100.0%)	
Non-Exposed	158 (52.7%)	142 (47.3%)	300 (100.0%)	χ^2 =12.217, p= 0.000
Total	358 (59.7%)	242 (40.3%)	600 (100.0%)	

a (Abnormal in any of the scales)

b (Normal in all z- score scales and BMI scale)

It was seen from Table 5.21: that 200 (66.7%) children of the exposed group had under nutrition in any of the z- score scale or BMI scale. In this group 100 (33.3%) children were normal in all z- score scales and BMI scale. On the other hand out of the total 300 non-exposed children 158 (52.7%) had under nutrition in any of the z- score scale or BMI scale and the rest 142 (47.3%) were Normal in all z- score scales and BMI scale. Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI was found associated with arsenic exposure ($p<0.001$).

Table 5.22: Nutritional Status of the Children by Height for Age Z- Scores according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Stunting [a]	Normal [b]		
<3000 Taka	Exposed	12 (40.0%)	18 (60.0%)	30 (100.0%)	χ^2=0.357, p= 0.550
	Non-Exposed	16 (33.3%)	32 (66.7%)	48 (100.0%)	
	Total	28 (35.9%)	50 (64.1%)	78 (100.0%)	
3000-5999 Taka	Exposed	75 (33.0%)	152 (67.0%)	227 (100.0%)	χ^2=4.378, p= 0.036
	Non-Exposed	53 (24.1%)	167 (75.9%)	220 (100.0%)	
	Total	128 (28.6%)	319 (71.4%)	447 (100.0%)	
6000 Taka or Above	Exposed	14 (32.6%)	29 (67.4%)	43 (100.0%)	χ^2=1.039, p= 0.308
	Non-Exposed	7 (21.9%)	25 (78.1%)	32 (100.0%)	
	Total	21 (28.0%)	54 (72.0%)	75 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=5.205, df=1, p=.023**			

a (HAZ < –2 sd for the age and sex)

b (HAZ +2sd to –2 sd for the age and sex)

Significant tests were performed to see the associations between exposure and effect with confounder monthly income. Of the three strata monthly income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by Height for Age z- scores was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka ($p<0.05$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=5.205, df=1, p=.023].

Table 5.23: Nutritional Status of the Children by Weight for Age Z- Scores according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Underweight [a]	Normal [b]		
<3000 Taka	Exposed	2 (6.7%)	28 (93.3%)	30 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.005 p=1.000
	Non-Exposed	3 (6.3%)	45 (93.8%)	48 (100.0%)	
	Total	5 (6.4%)	73 (93.6%)	78 (100.0%)	
3000-5999 Taka	Exposed	28 (12.3%)	199 (87.7%)	227 (100.0%)	χ^2=7.548, p= 0.006
	Non-Exposed	11 (5.0%)	209 (95.0%)	220 (100.0%)	
	Total	39 (8.7%)	408 (91.3%)	447 (100.0%)	
6000 Taka or Above	Exposed	6 (14.0%)	37 (86.0%)	43 (100.0%)	χ^2=0.364, p= 0.546
	Non-Exposed	3 (9.4%)	29 (90.6%)	32 (100.0%)	
	Total	9 (12.0%)	66 (88.0%)	75 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=6.179, df=1, p=.013**			

a (WAZ < –2 sd for the age and sex)

b (WAZ +2sd to –2 sd for the age and sex)

Of the three strata monthly income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by weight for age z- scores was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka ($p<0.05$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=6.179, df=1, p=.013].

Table 5.24: Nutritional Status of the Children by Weight for Height Z- Scores according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Wasting [a]	Normal [b]		
<3000 Taka	Exposed	3 (10.0%)	27 (90.0%)	30 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=4.928 p=0.053
	Non-Exposed	0 (.0%)	48 (100.0%)	48 (100.0%)	
	Total	3 (3.8%)	75 (96.2%)	78 (100.0%)	
3000-5999 Taka	Exposed	15 (6.6%)	212 (93.4%)	227 (100.0%)	χ^2=0.527, p= 0.468
	Non-Exposed	11 (5.0%)	209 (95.0%)	220 (100.0%)	
	Total	26 (5.8%)	421 (94.2%)	447 (100.0%)	
6000 Taka or Above	Exposed	5 (11.6%)	38 (88.4%)	43 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=3.934 p=0.047
	Non-Exposed	0 (.0%)	32 (100.0%)	32 (100.0%)	
	Total	5 (6.7%)	70 (93.3%)	75 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=3.487, df=1, p=.062**			

a (WHZ < –2 sd for the age and sex)

b (WHZ +2sd to –2 sd for the age and sex)

Significant tests were performed to see the associations between exposure and effect with confounder monthly income. Of the three strata monthly Income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by Weight for Height z- scores was found to be associated with the exposure (arsenic contamination) in neither group. In group with monthly income<3000 Taka the relation was nearly significant (p=0.053). Adjusting the influence of the control variables the exposure effect was not found to be associated [Mantel-Haenszel Chi-Squared=3.487, df=1, p=.062].

Table 5.25: Nutritional Status of the Children by Combined Z-Scores (HAZ, WAZ & WHT) according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Under nutrition [a]	Normal [b]		
<3000 Taka	Exposed	13 (43.3%)	17 (56.7%)	30 (100.0%)	χ^2 =0.489, p= 0.484
	Non-Exposed	17 (35.4%)	31 (64.6%)	48 (100.0%)	
	Total	30 (38.5%)	48 (61.5%)	78 (100.0%)	
3000-5999 Taka	Exposed	88 (75.2)	139 (61.2%)	227 (227.0)	χ^2 =6.664, p= 0.010
	Non-Exposed	60 (27.3%)	160 (72.7%)	220 (100.0%)	
	Total	148 (33.1%)	299 (66.9%)	447 (100.0%)	
6000 Taka or Above	Exposed	18 (41.9%)	25 (58.1%)	43 (100.0%)	
	Non-Exposed	7 (21.9%)	25 (78.1%)	32 (100.0%)	
	Total	25 (33.3%)	50 (66.7%)	75 (100.0%)	
Tests of Conditional Independence:				**Mantel-Haenszel Chi-Squared=9.149, df=1, p=.002**	

a (abnormal in any z- score scales)

b (Normal in all z- score scales)

Of the three strata monthly income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by combined z- scores was found to be associated with the exposure (arsenic contamination) in group having Monthly Income 3000-5999 Taka ($p<0.05$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=9.149, df=1, p=.002].

Table 5.26: Nutritional Status of the Children by BMI according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Thinness [a]	Normal [b]		
<3000 Taka	Exposed	13 (43.3%)	17 (56.7%)	30 (100.0%)	χ^2 =0.107, p= 0.743
	Non-Exposed	19 (39.6%)	29 (60.4%)	48 (100.0%)	
	Total	32 (41.0%)	46 (59.0%)	78 (100.0%)	
3000-5999 Taka	Exposed	110 (48.5%)	117 (51.5%)	227 (100.0%)	χ^2 =4.803, p= 0.028
	Non-Exposed	84 (38.2%)	136 (61.8%)	220 (100.0%)	
	Total	194 (43.4%)	253 (56.6%)	447 (100.0%)	
6000 Taka or Above	Exposed	24 (55.8%)	19 (44.2%)	43 (100.0%)	χ^2 =4.467, p= 0.035
	Non-Exposed	10 (31.3%)	22 (68.8%)	32 (100.0%)	
	Total	34 (45.3%)	41 (54.7%)	75 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=7.145, df=1, p=.008**			

a (BMI <5th centile for the age and sex)

b (BMI ≥85th centile for the age and sex)

Of the three strata monthly income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by BMI was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and 3000-5999 Taka ($p= 0.028$ and $p= 0.035$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=7.145, df=1, p=.008].

Table 5.27: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHZ) and BMI according to Arsenic Exposure and Monthly Income of the Families

Monthly Income	Area	Nutritional Status		Total	χ^2, p value
		Under nutrition [a]	Normal All [b]		
<3000 Taka	Exposed	19 (63.3%)	11 (36.7%)	30 (100.0%)	χ^2=0.383, p= 0.536
	Non-Exposed	27 (56.3%)	21 (43.8%)	48 (100.0%)	
	Total	46 (59.0%)	32 (41.0%)	78 (100.0%)	
3000-5999 Taka	Exposed	152 (67.0%)	75 (33.0%)	227 (100.0%)	χ^2=8.293, p= 0.004
	Non-Exposed	118 (53.6%)	102 (46.4%)	220 (100.0%)	
	Total	270 (60.4%)	177 (39.6%)	447 (100.0%)	
6000 Taka or Above	Exposed	29 (67.4%)	14 32.6% ( )	43 (100.0%)	χ^2=5.355, p= 0.021
	Non-Exposed	13 (40.6%)	19 (59.4%)	32 (100.0%)	
	Total	42 (56.0%)	33 (44.0%)	75 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=11.840, df=1, p=.001**			

a (Abnormal in any of the scales)

b (Normal in all z- score scales and BMI scale)

Of the three strata Monthly Income<3000 Taka, 3000-5999 Taka and 6000 Taka or above; Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and 3000-5999 Taka ($p= 0.004$ and $p= 0.021$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=11.840, df=1, p=.001].

Table 5.28: Nutritional Status of the Children by Height for Age Z- Scores according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Stunting [a]	Normal [b]		
≤ 4	Exposed	26 (32.5%)	54 (67.5%)	80 (100.0%)	χ^2=0.149, p= 0.700
	Non-Exposed	28 (29.8%)	66 (70.2%)	94 (100.0%)	
	Total	54 (31.0%)	120 (69.0%)	174 (100.0%)	
>4	Exposed	75 (34.1%)	145 (65.9%)	220 (100.0%)	χ^2=6.031, p= 0.014
	Non-Exposed	48 (23.3%)	158 (76.7%)	206 (100.0%)	
	Total	123 (28.9%)	303 (71.1%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=4.732, df=1, p=.030**			

a (HAZ < –2 sd for the age and sex)

b (HAZ +2sd to –2 sd for the age and sex)

Significant tests were performed to see the associations between exposure and effect with confounder family size. Of the two strata family size ≤ 4 persons and >4 persons; Nutritional status of the children by Height for Age z-scores was found to be associated with the exposure (arsenic contamination) in group family size >4 persons. Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=5.205, df=1, p=.023].

Table 5.29: Nutritional Status of the Children by Weight for Age Z- Scores according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Underweight [a]	Normal [b]		
≤ 4	Exposed	13 (16.3%)	67 (83.8%)	80 (100.0%)	χ^2=4.326, p= 0.038
	Non-Exposed	6 (6.4%)	88 (93.6%)	94 (100.0%)	
	Total	19 (10.9%)	155 (89.1%)	174 (100.0%)	
>4	Exposed	23 (10.5%)	197 (89.5%)	220 (100.0%)	χ^2=3.789, p= 0.052
	Non-Exposed	11 (5.3%)	195 (94.7%)	206 (100.0%)	
	Total	34 (8.0%)	392 (92.0%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=7.027, df=1, p=.008**			

a (WAZ < –2 sd for the age and sex)

b (WAZ +2sd to –2 sd for the age and sex)

Of the two strata family size ≤ 4 persons and >4 persons; Nutritional status of the children by Weight for Age z- scores was found to be associated with the exposure (arsenic contamination) in group family size ≤ 4 (p= 0.038) and it was found nearly associated in group family size >4 persons (p= 0.052). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=7.027, df=1, p=.008].

Table 5.30: Nutritional Status of the Children by Weight for Height Z- Scores according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Wasting [a]	Normal [b]		
≤ 4	Exposed	7 (8.8%)	73 (91.3%)	80 (100.0%)	χ^2 =0.350, p= 0.554
	Non-Exposed	6 (6.4%)	88 (93.6%)	94 (100.0%)	
	Total	13 (7.5%)	161 (92.5%)	174 (100.0%)	
>4	Exposed	16 (7.3%)	204 (92.7%)	220 (100.0%)	χ^2 =5.330, p= 0.021
	Non-Exposed	5 (2.4%)	201 (97.6%)	206 (100.0%)	
	Total	21 (4.9%)	405 (95.1%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=4.029, df=1, p=.045**			

a (WHZ <–2 sd for the age and sex)

b (WHZ +2sd to –2 sd for the age and sex)

Of the two strata family size ≤ 4 persons and >4 persons; Nutritional status of the children by Weight for Height z-scores was found to be associated with the exposure (arsenic contamination) in group Family >4 persons (p= 0.021). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=4.029, df=1, p=.045].

Table 5.31: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHT) according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Under nutrition [a]	Normal [b]		
≤ 4	Exposed	32 (40.0%)	48 (60.0%)	80 (100.0%)	χ^2=0.922, p= 0.554
	Non-Exposed	31 (33.0%)	63 (67.0%)	94 (100.0%)	
	Total	63 (36.2%)	111 (63.8%)	174 (100.0%)	
>4	Exposed	87 (39.5%)	133 (60.5%)	220 (100.0%)	χ^2=9.206, p= 0.002
	Non-Exposed	53 (25.7%)	153 (74.3%)	206 (100.0%)	
	Total	140 (32.9%)	286 (67.1%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=8.848, df=1, p=.003**			

a (Abnormal in any z- scores scale)

b (Normal in all z- score scales)

Of the two strata family size ≤ 4 persons and >4 persons; Nutritional status of the children by combined z- scores (HAZ, WAZ & WHT) was found to be associated with the exposure (arsenic contamination) in group Family >4 persons (p= 0.002). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=8.848, df=1, p=.003].

Table 5.32: Nutritional Status of the Children by BMI according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Under nutrition [a]	Normal [b]		
≤ 4	Exposed	35 (43.8%)	45 (56.3%)	80 (100.0%)	χ^2 =0.532, p= 0.466
	Non-Exposed	36 (38.3%)	58 (61.7%)	94 (100.0%)	
	Total	71 (40.8%)	103 (59.2%)	174 (100.0%)	
>4	Exposed	112 (50.9%)	108 (49.1%)	220 (100.0%)	χ^2 =7.891, p= 0.005
	Non-Exposed	77 (37.4%)	129 (62.6%)	206 (100.0%)	
	Total	189 (44.4%)	237 (55.6%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=7.172, df=1, p=.007**			

a (BMI <5th centile for the age and sex)

b (BMI ≥85th centile for the age and sex)

Of the two strata Family size ≤ 4 persons and >4 persons; Nutritional status of the children by BMI was found to be associated with the exposure (arsenic contamination) in group family size >4 (p= 0.005). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=7.172, df=1, p=.007].

Table 5.33: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHZ) and BMI according to Arsenic Exposure and Family Size

Family size	Area	Nutritional Status		Total	χ^2, p value
		Under nutrition [a]	Normal [b]		
≤ 4	Exposed	51 (63.8%)	29 (36.3%)	80 (100.0%)	χ^2=0.718, p= 0.379
	Non-Exposed	54 (57.4%)	40 (42.6%)	94 (100.0%)	
	Total	105 (60.3%)	69 (39.7%)	174 (100.0%)	
>4	Exposed	149 (67.7%)	71 (32.3%)	220 (100.0%)	χ^2=13.113, p= 0.000
	Non-Exposed	104 (50.5%)	102 (49.5%)	206 (100.0%)	
	Total	253 (59.4%)	173 (40.6%)	426 (100.0%)	
Tests of Conditional Independence:		**Mantel-Haenszel Chi-Squared=11.711, df=1, p=.001**			

a (abnormal in any z- score scales and BMI scale)
b (Normal in all z- score scales and BMI scale)

Of the two strata Family size ≤ 4 persons and >4 persons; Nutritional status of the children by combined z- scores (HAZ, WAZ & WHZ) and BMI was found to be associated with the exposure (arsenic contamination) in group family size >4 (p= 0.000). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=11.711, df=1, p=.001].

Table 5.34: Nutritional Status of the Children by Height for Age Z- Scores according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Stunting [a]	Normal [b]		
<3000 Taka	≤4	Exposed	2 (25.0%)	6 (75.0%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=4.928 p=0.053
		Non-Exposed	6 (42.9%)	8 (57.1%)	14 (100.0%)	
		Total	8 (36.4%)	14 (63.6%)	22 (100.0%)	
	>4	Exposed	10 (45.5%)	12 (54.5%)	22 (100.0%)	χ^2=1.497, p= 0.221
		Non-Exposed	10 (29.4%)	24 (70.6%)	34 (100.0%)	
		Total	20 (35.7%)	36 (64.3%)	56 (100.0%)	
3000-5999 Taka	≤4	Exposed	21 (35.0%)	39 (65.0%)	60 (100.0%)	χ^2=0.305 p= 0.581
		Non-Exposed	21 (30.4%)	48 (69.6%)	69 (100.0%)	
		Total	42 (32.6%)	87 (67.4%)	129 (100.0%)	
	>4	Exposed	54 (32.3%)	113 (67.7%)	167 (100.0%)	χ^2=4.991, p= 0.025
		Non-Exposed	32 (21.2%)	119 (78.8%)	151 (100.0%)	
		Total	86 (27.0%)	232 (73.0%)	318 (100.0%)	
6000 Taka or Above	≤4	Exposed	3 (25.0%)	9 (75.0%)	12 (100.0%)	χ^2=1.011, p= 0.315
		Non-Exposed	1 (9.1%)	10 (90.9%)	11 (100.0%)	
		Total	4 (17.4%)	19 (82.6%)	23 (100.0%)	
	>4	Exposed	11 (35.5%)	20 (64.5%)	31 (100.0%)	χ^2=0.272 p= 0.602
		Non-Exposed	6 (28.6%)	15 (71.4%)	21 (100.0%)	
		Total	17 (32.7%)	35 (67.3%)	52 (100.0%)	
Tests of Conditional Independence:					**Mantel-Haenszel Chi-Squared=5.327, df=1, p=.021**	

a (HAZ < –2 sd for the age and sex)

b (HAZ +2sd to –2 sd for the age and sex)

Significant tests were performed to see the associations between exposure and effect with confounder monthly income and family size. Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size> 4; Nutritional status of the children by Height for Age z- scores was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and family size> 4 ($p<0.05$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=5.327, df=1, p=.021].

Table 5.35: Nutritional Status of the Children by Weight for Age Z- Scores according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Underweight [a]	Normal [b]		
<3000 Taka	≤ 4	Exposed	0 (.0%)	8 (100.0%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.571 p=1.000
		Non-Exposed	1 (7.1%)	13 (92.9%)	14 (100.0%)	
		Total	1 (4.5%)	21 (95.5%)	22 (100.0%)	
	> 4	Exposed	2 (9.1%)	20 (90.9%)	22 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.204 p=0.652
		Non-Exposed	2 (5.9%)	32 (94.1%)	34 (100.0%)	
		Total	4 (7.1%)	52 (92.9%)	56 (100.0%)	
3000-5999 Taka	≤ 4	Exposed	10 (16.7%)	50 (83.3%)	60 (100.0%)	χ^2=2.772 p= 0.096
		Non-Exposed	5 (7.2%)	64 (92.8%)	69 (100.0%)	
		Total	15 (11.6%)	114 (88.4%)	129 (100.0%)	
	> 4	Non-Exposed	6 (4.0%)	145 (96.0%)	151 (100.0%)	χ^2=5.235 p= 0.022
		Exposed	18 10.8% ( )	149 (89.2%)	167 (100.0%)	
		Total	24 (7.5%)	294 (92.5%)	318 (100.0%)	
6000 Taka or Above	≤ 4	Exposed	3 (25.0%)	9 (75.0%)	12 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=3.025 p=0.082
		Non-Exposed	0 (.0%)	11 (100.0%)	11 (100.0%)	
		Total	3 (13.0%)	20 (87.0%)	23 (100.0%)	
	> 4	Exposed	3 (9.7%)	28 (90.3%)	31 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.255 p=0.613
		Non-Exposed	3 (14.3%)	18 (85.7%)	21 (100.0%)	
		Total	6 (11.5%)	46 (88.5%)	52 (100.0%)	
Tests of Conditional Independence:					Mantel-Haenszel Chi-Squared=6.520, df=1, p=.011	

a (WAZ < –2 sd for the age and sex)

b (WAZ +2sd to –2 sd for the age and sex)

Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size > 4; Nutritional status of the children by Weight for Age z- scores was found to be associated with the exposure (arsenic contamination) in group with monthly income 3000-5999 Taka and family size >4 only. However, adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=6.520, df=1, p=.011].

Table 5.36: Nutritional Status of the Children by Weight for Height Z- Scores according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Wasting [a]	Normal [b]		
<3000 Taka	≤4	Exposed	1 (12.5%)	7 (87.5%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=1.750 p=0.186
		Non-Exposed	0 (.0%)	14 (100.0%)	14 (100.0%)	
		Total	1 (4.5%)	21 (95.5%)	22 (100.0%)	
	>4	Exposed	2 (9.1%)	20 (90.9%)	22 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=3.148 p=0.076
		Non-Exposed	0 (.0%)	34 (100.0%)	34 (100.0%)	
		Total	2 (3.6%)	54 (96.4%)	56 (100.0%)	
3000-5999 Taka	≤4	Exposed	3 (5.0%)	57 (95.0%)	60 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.670 p=0.413
		Non-Exposed	6 (8.7%)	63 (91.3%)	69 (100.0%)	
		Total	9 (7.0%)	120 (93.0%)	129 (100.0%)	
	>4	Exposed	12 (7.2%)	155 (92.8%)	167 (100.0%)	χ^2 =2.352 p=0.125
		Non-Exposed	5 (3.3%)	146 (96.7%)	151 (100.0%)	
		Total	17 (5.3%)	301 (94.7%)	318 (100.0%)	
6000 Taka or Above	≤4	Exposed	3 (25.0%)	9 (75.0%)	12 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=3.025 p=0.082
		Non-Exposed	0 (.0%)	11 (100.0%)	11 (100.0%)	
		Total	3 (13.0%)	20 (87.0%)	23 (100.0%)	
	>4	Exposed	2 (6.5%)	29 (93.5%)	31 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=1.382 p=0.240
		Non-Exposed	0 (.0%)	21 (100.0%)	21 (100.0%)	
		Total	2 (3.8%)	50 (96.2%)	52 (100.0%)	
Tests of Conditional Independence:					**Mantel-Haenszel Chi-Squared=3.764, df=1, p=.052**	

a (WHZ < –2 sd for the age and sex)

b (WHZ +2sd to –2 sd for the age and sex)

Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size> 4; Nutritional status of the children by Weight for Height z-scores was found to be associated with the exposure (arsenic contamination) in neither group. However, adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=3.764, df=1, p=.052].

Table 5.37: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHZ) according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Under nutrition [a]	Normal [b]		
<3000 Taka	≤4	Exposed	3 (37.5%)	5 (62.5%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.058 p=0.810
		Non-Exposed	6 (42.9%)	8 (57.1%)	14 (100.0%)	
		Total	9 (40.9%)	13 (59.1%)	22 (100.0%)	
	>4	Exposed	10 (45.5%)	12 (54.5%)	22 (100.0%)	χ^2=0.978 p=0.323
		Non-Exposed	11 (32.4%)	23 (67.6%)	34 (100.0%)	
		Total	21 (37.5%)	35 (62.5%)	56 (100.0%)	
3000-5999 Taka	≤4	Exposed	24 (40.0%)	36 (60.0%)	60 (100.0%)	χ^2=0.374 p=0.541
		Non-Exposed	24 (34.8%)	45 (65.2%)	69 (100.0%)	
		Total	48 (37.2%)	81 (62.8%)	129 (100.0%)	
	>4	Exposed	64 (38.3%)	103 (61.7%)	167 (100.0%)	χ^2=7.715 p=0.005
		Non-Exposed	36 (23.8%)	115 (76.2%)	151 (100.0%)	
		Total	100 (31.4%)	218 (68.6%)	318 (100.0%)	
6000 Taka or Above	≤4	Exposed	5 (41.7%)	7 (58.3%)	12 (100.0%)	χ^2=3.159 p=0.076
		Non-Exposed	1 (9.1%)	10 (90.9%)	11 (100.0%)	
		Total	6 (26.1%)	17 (73.9%)	23 (100.0%)	
	>4	Exposed	13 (41.9%)	18 (58.1%)	31 (100.0%)	χ^2=0.964 p=0.326
		Non-Exposed	6 (28.6%)	15 (71.4%)	21 (100.0%)	
		Total	19 (36.5%)	33 (63.5%)	52 (100.0%)	
Tests of Conditional Independence:					**Mantel-Haenszel Chi-Squared=9.386, df=1, p=.002**	

a (Abnormal in any z- scores scale)

b (Normal in all z- score scales)

Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size > 4; Nutritional status of the children by combined z-scores (HAZ, WAZ & WHZ) was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and family size>4 ($p<0.01$) . Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=9.386, df=1, p=.002].

Table 5.38: Nutritional Status of the Children by BMI according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Under nutrition [a]	Normal [b]		
<3000 Taka	≤4	Exposed	4 (50.0%)	4 (50.0%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.100 p=0.752
		Non-Exposed	6 (42.9%)	8 (57.1%)	14 (100.0%)	
		Total	10 (45.5%)	12 (54.5%)	22 (100.0%)	
	>4	Exposed	9 (40.9%)	13 (59.1%)	22 (100.0%)	χ^2=0.040 p=0.841
		Non-Exposed	13 (38.2%)	21 (61.8%)	34 (100.0%)	
		Total	22 (39.3%)	34 (60.7%)	56 (100.0%)	
3000-5999 Taka	≤4	Exposed	23 (38.3%)	37 (61.7%)	60 (100.0%)	χ^2=0.182 p=0.670
		Non-Exposed	29 (42.0%)	40 (58.0%)	69 (100.0%)	
		Total	52 (40.3%)	77 (59.7%)	129 (100.0%)	
	>4	Exposed	87 (52.1%)	80 (47.9%)	167 (100.0%)	χ^2=7.881 p=0.005
		Non-Exposed	55 (36.4%)	96 (63.6%)	151 (100.0%)	
		Total	142 (44.7%)	176 (55.3%)	318 (100.0%)	
6000 Taka or Above	≤4	Exposed	8 (66.7%)	4 (33.3%)	12 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=7.640 p=0.005
		Non-Exposed	1 (9.1%)	10 (90.9%)	11 (100.0%)	
		Total	9 (39.1%)	14 (60.9%)	23 (100.0%)	
	>4	Exposed	16 (51.6%)	15 (48.4%)	31 (100.0%)	χ^2=0.384 p=0.535
		Non-Exposed	9 (42.9%)	12 (57.1%)	21 (100.0%)	
		Total	25 (48.1%)	27 (51.9%)	52 (100.0%)	
Tests of Conditional Independence:					**Mantel-Haenszel Chi-Squared=6.871, df=1, p=.009**	

a (BMI <5th centile for the age and sex)

b (BMI ≥85th centile for the age and sex)

Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size > 4; Nutritional status of the children by BMI was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and family size >4 ($p<0.01$) . Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=6.871, df=1, p=.009].

Table 5.39: Nutritional Status of the Children by Combined Z- Scores (HAZ, WAZ & WHZ) and BMI according to Arsenic Exposure, Monthly Income of the Families and Family Size

Monthly Income	Family size	Area	Nutritional Status		Total	χ^2, p value
			Under nutrition [a]	Normal All [b]		
<3000 Taka	≤4	Exposed	5 (62.5%)	3 (37.5%)	8 (100.0%)	Fisher's Exact Test Linear-by-Linear Association=0.179 p=0.673
		Non-Exposed	10 (71.4%)	4 (28.6%)	14 (100.0%)	
		Total	15 (68.2%)	7 (31.8%)	22 (100.0%)	
	>4	Exposed	14 (63.6%)	8 (36.4%)	22 (100.0%)	χ^2=1.005 p=0.316
		Non-Exposed	17 (50.0%)	17 (50.0%)	34 (100.0%)	
		Total	31 (55.4%)	25 (44.6%)	56 (100.0%)	
3000-5999 Taka	≤4	Exposed	37 (61.7%)	23 (38.3%)	60 (100.0%)	χ^2=0.009 p=0.926
		Non-Exposed	42 (60.9%)	27 (39.1%)	69 (100.0%)	
		Total	79 (61.2%)	50 (38.8%)	129 (100.0%)	
	>4	Exposed	115 (68.9%)	52 (31.1%)	167 (100.0%)	χ^2=11.352 p=0.001
		Non-Exposed	76 (50.3%)	75 (49.7%)	151 (100.0%)	
		Total	191 (60.1%)	127 (39.9%)	318 (100.0%)	
6000 Taka or Above	≤4	Exposed	9 (75.0%)	3 (25.0%)	12 (100.0%)	χ^2=7.425 p=0.006
		Non-Exposed	2 (18.2%)	9 (81.8%)	11 (100.0%)	
		Total	11 (47.8%)	12 (52.2%)	23 (100.0%)	
	>4	Exposed	20 (64.5%)	11 (35.5%)	31 (100.0%)	χ^2=0.766 p=0.405
		Non-Exposed	11 (52.4%)	10 (47.6%)	21 (100.0%)	
		Total	31 (59.6%)	21 (40.4%)	52 (100.0%)	
Tests of Conditional Independence:					**Mantel-Haenszel Chi-Squared=11.835, df=1, p=.001**	

a (Abnormal in any of the z- score scales and BMI scale)

b (Normal in all z- score scales and BMI scale)

Of the six strata, monthly income<3000 Taka and family size ≤ 4, monthly income<3000 Taka and family size >4, monthly income3000-5999 Taka and family size ≤ 4, monthly income 3000-5999 Taka and family size >4, monthly income 6000 Taka or Above and family size ≤ 4, monthly income 6000 Taka or Above and family size > 4; Nutritional status of the children by combined z-scores (HAZ, WAZ & WHZ) and BMI was found to be associated with the exposure (arsenic contamination) in group monthly income 3000-5999 Taka and family size >4 ($p<0.01$) and in group monthly income6000 Taka or Above and family size ≤ 4 ($p<0.01$). Adjusting the influence of the control variables the exposure effect was found to be significantly associated [Mantel-Haenszel Chi-Squared=11.835, df=1, p=.001].

Correlates of malnutrition: Binary logistic regression analysis:

To assess the factors influencing the malnutrition among the children binary logistic regression analysis was carried out in which the dependent variable, 'nutritional status' was dichotomized (malnourished/normal). For prediction of influencing factors for malnutrition, variables that showed significant association with nutritional status, in chi-square analysis were entered into logistic regression model. The nutritional status of the children was assessed by z- score of weight for age, height for age and weight for height. Children having any of the parameter in terms of under weight, stunting and wasting was considered as malnutrition.

The dependent and independent variables are shown in the box:

Dependent variable: Nutritional status
0= Normal
1= Malnourished

Independent variables

Subjects	Age in years (father)	Age in year's (mother)
0= Non exposed	0=<40	0=<30
1=Exposed	1=≥40	1=≥30
Duration of tube well water use		No. of wet rice taken per week
0= <7		0=<2
1=≥7		1=≥2
Sources of water		Frequency of pulses taken per week (Continuous)
0=Tube well		No. of glass of water taken daily (continuous)
1=other sources		

Table 5.40: Correlates of malnutrition among the children: Binary logistic regression analysis

Independent variables	*β*	*df*	*p value*	*Odds ratio*	*95.0% C.I*
Subject					
Non exposed (RC)	-	-	-	-	-
Exposed	1.978	1	.000	7.230	2.915-17.931
Sources of water					
Tube well (RC)	-	-	-	-	-
Others sources	.305	1	.471	1.357	.592-3.111
Age in years (Father)					
<40 (RC)	-	-	-	-	-
≥40	-.845	1	.053	.429	.183-1.010
Age in years (Mother)					
<30 (RC)	-	-	-	-	-
≥30	-.213	1	.648	.808	.324-2.015
Duration of Tube well water use (yrs)					
<7 (RC)	-	-	-	-	-
≥7	1.263	1	.000	3.537	1.771-7.063
No. of wet rice taken per week by children					
<2					
≥2	1.449	1	.000	4.261	2.122-8.556
Frequency of pulses taken per week	1.432	1	.000	4.186	3.207-5.464
No. of glass of water taken per day	.626	1	.000	1.869	1.596-2.189
Model Chi square	498.898	5	0.001		
df	5				
Significance	0.001				
Constant	-12.145				

RC= Reference category

Out of 8 variables, 5 variables showed significant association in binary logistic regression analysis. The analysis showed that subjects exposed to arsenic contamination, duration of tube well water use, frequency of taken per week and number of glass of water drink per day appeared to be the main prediction of malnutrition among the children ($p<0.001$). Data analysis indicated that the malnutrition was found to be 4.2 times higher among the children who consumed wet rice more than 2 times per week, 7.2 times higher among the children exposed to arsenic in water. In the logistic model, showed that the malnutrition of exposed group was significantly positively correlated with frequency of pulses, wet rice and number of glass of water drinks per day indicating the children were more exposed to arsenic contamination through pulses, wet rice and also arsenic contaminated water.

CHAPTER V

DISCUSSION

5. Discussion

This cross sectional comparative study was conducted to find out if there was any difference in nutritional status of arsenic exposed and non-exposed children aged 5-14 years. A total 300 arsenic exposed children were selected from villages under Narayangonj district and another 300 non-exposed children were selected from villages under Munshigonj district respectively. A pre-tested questionnaire was used to interview the respondents and anthropometric measurements (height and weight) were used to assess the nutritional status of the children. Nutrients taken per day by the children were assessed by using 24-hours recall questionnaire.

Chronic arsenic poisoning associated with groundwater contamination has been reported from many developing countries, where poor nutritional status is concomitantly found [54, 55, 56].

Poor nutritional status may increase an individual's susceptibility to arsenic toxicity, or alternatively that arsenicosis may contribute to poor nutritional status. Such observations have been made in different studies from Taiwan, Chile, West Bengal India, Nepal, Japan and Bangladesh [63,157,158,159,160,161,162,163].

Few studies have related some toxic effect of arsenic exposure with nutritional status of exposed population. In an arsenic exposed population in West Bengal

body weight was found to be negatively associated arsenical keratosis (Mazumder 1998) and in Taiwan vascular effects (black foot disease) was found to be associated with under nourishment [62]. In Taiwan a low serum β-carotene concentrated was associated with higher prevalence of arsenic related skin cancer [228] and ischemic heart disease [59].

Studies [126,229] have shown that socioeconomic condition had influence nutritional status. Studies [231,232,233,234] have also revealed numbers of the family and type of the family influence nutritional status.

The exposed and non-exposed group of this study was found not to be statistically different in terms of age and gender ($p>0.05$). Furthermore no remarkable difference in overall socio-economic status (e.g. income, education, occupation etc.) between the groups was found. Type of the family and number of the family members no significant difference was observed between the two groups. Therefore age, gender, income, education, occupation, family type and family size are least likely to have any effect on the difference of nutritional status between the groups.

Dietary intake is an important determinant of nutritional status. 24- hours recall for dietary intake assessment has been under taken in studies carried out by Shaaben (2005) [237] in Egypt, Chen (2004)[235] in Bangladesh, Mitra (2004)[160] in

India, Brunner (2001)[236] in UK. In this study 24- hours recall dietary intake generally used as a tool for assessed dietary intake.

The amount of the nutrients (Energy, Protein, Carbohydrate, Fat, Vitamin-A and Vitamin-C) were subsequently calculated. Independent sample t-tests were performed to find out any difference of mean intake of nutrients (Total weight, Energy, Protein, Fat and Carbohydrate) taken by the study children. It was observed that there was no significant difference of intake of nutrients by the studied children between the two groups.

Anthropometry measurements are widely accepted tool for assessing the nutritional status of children. Different studies [237,238,239] support these tools for assessing nutritional status of children.

Body mass index is considered to be the more nutritionally than genetically related (Khongsdier 2001), thus it is appropriate to use BMI as an indicator of the nutritional status. Body mass index may be influenced by age, gender [240] and socio-economic status [241]. The factors that can influence BMI, includes dietary intake, and socioeconomic status has intra population variations [242].

The nutritional status of children was assessed by z-scores of weight for age, height for age and weight for height and also using 5th and 85th percentiles of the body mass index (BMI) for age. Using the z-score Height for age (HFA),

Weight for age (WFA) and weight for height (WFH) the children were grouped as normal or stunted; normal or underweight and normal or wasted respectively. Stunting, underweight and wasting were found to be significantly higher in exposed then in non- exposed group ($p<0.05$).

Thinness (low BMI for age) was found to be more among the children of exposed area (49 %) than that of non-exposed area (38%). Comparatively children with normal BMI was found to be more in non-exposed area than in exposed area and the difference was found to be significant ($p< 0.05$).

The mean BMI of the children was found to be significantly higher ($p= 0.012$) in non-exposed children (14.874±2.167) than that of exposed children (14.423±2.208). When compared after grouping the nutritional status as underweight and normal or overweight (based on BMI) an association between nutritional status and arsenic exposed was observed ($p<0.01$). Such association was observed among children aged 8-10 and 11-14 years but no such association was observed among those who were between 5-7 years of age. In both the analysis nutritional status was found to be significantly low among the arsenic exposed children particularly among the older children of 8-10 years and 11-14 years. Different studies [136,157,172,180] showed that BMI in exposed group was significantly lower than that of non-exposed group.

Previous studies showed that socio-economic condition (in term of income, occupation, type of family and size of family) may influence nutritional status of study population [124,177,243]. Bi-variate analysis showed that between BMI with monthly income of family and occupation no association was found ($p > 0.05$) but significant association with type of family and number of the family was observed ($p < 0.05$).

In both the group (exposed and non-exposed) monthly income and family size were found to be similar, but in families of similar size, variation in monthly income can influence nutritional status. Similarly in families with similar monthly income, variation in family size may influence nutritional status. Considering these influences of arsenic exposure on nutritional status after adjusting for monthly family income and family size was explore and it was found that under nutrition was still significantly higher ($p<0.01$) in exposed group then in non-exposed group.

To assess the factors influencing the malnutrition among the children binary logistic regression analysis was carried out. The nutritional status of the children was assessed by z- score of weight for age, height for age and weight for height. Children having any of the parameter in terms of under weight, stunting and wasting was consider as malnutrition. The analysis suggests that malnutrition

status was 7.2 times higher among the children exposed to arsenic contaminated water. That is consistent with previous studies [71,157,160,171,172,177,193,194].

Few studies showed that frequency of taking pulses, wet rice and number of glass of arsenic contain drinking water per day had similar type of correlation on nutritional status[71,157,160,162,171,177,180,193]. Analysis also revealed that malnutrition was significantly positively correlated with frequency of taking pulses, wet rice and number of glass of water drinking per day indicating the children were more exposed to arsenic contamination through wet rice, pulses and also arsenic contaminated water.

From the finding of the current study it can be concluded that there was significant difference of nutritional status of the children between of exposed and non-exposed areas. The study shows that the children exposed area had lower nutritional status compared to that of non-exposed area.

CHAPTER VI

CONCLUSION

6. CONCLUSION

This cross sectional study was designed to compare the nutritional status of 5-14 years age children of selected arsenic non-exposed and arsenic expose areas. Probable determinants (socio-demographic characteristics, principal dietary intake) were considered to assess any relation with nutritional status. The exposed groups of children were consuming water from sources having arsenic level above 50 µg /L. On the other hand non-exposed children were consuming water with arsenic level below 50 µg /L. The study findings suggest that there was no significant difference in overall socio-economic status (e.g. income, education, occupation) between exposed and non-exposed areas. Dietary consumption also did not show any significant difference between these two groups. However, the study showed significantly higher number of under weight children in the exposed area in comparison to non- exposed area. It was further observed that as the age increased the number of underweight children was found more in the exposed area. Chi square tests were performed to see the associations between exposure and effect with confounder's: monthly income and family size. Adjusting the influences of the control variables it was seen that under nutrition was more among the study children with arsenic exposure than

the arsenic non-exposed children. The exposure-effect was found to be significantly associated ($p< 0.01$).

In this study out of 8 variables, 5 variables showed significant association in binary logistic regression analysis. The analysis showed that subject exposed arsenic contamination, duration of tube well water use, frequency of taken pulses per week and number of glass of water drink per day appeared to be the main prediction of malnutrition among the children ($p<0.001$). Data analysis indicated that the malnutrition was found to be 4.2 times higher among the children who consumed wet rice more than 2 times per week, 7.2 times higher among the children exposed to arsenic in water. The study showed that the malnutrition was positively correlated with frequency of pulses intake per week and number of glass drinking intake consumed per day indicating the children were more exposed to arsenic contamination through foods and also water. So, the lower nutritional status observed among the arsenic exposed children compared to non-exposed children in this study seems to be related to arsenic exposure.

CHAPTER VII

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Smith L. Arsenic in drinking water. WELL fact sheet. WELL- resource centre network for water. Sanitation and Environmental Health; 2005: 1-5.

2. Ahmad SA, Sengupta MK, Mukherjee A, Hossain MA, Das B, Nayak B. Arsenic groundwater contamination and its health effects in the state of Uttar Pradesh (UP) in upper and middle Ganga Plain, India: a sever danger: 2006.

   http: // www. sciencedirect.com/science/journal/00489697. (Accessed on 1st Jan 2006.

3. Chakraborti D, Mukherjee SC, Pati S, Sengupta MK, Rahaman MM, Chowdhury UK. Arsenic groundwater contamination in middle Ganga Plain, Bihar, India: a future danger? Environ Health Perspect.2003; 111: 1194-201.

4. Mahmood SAI. The silent killer is engulfing Bangladesh. Environmental Articles archive. Human Health; 2002: 1 – 5.

5. United Nation International children's Emergency Fund. Arsenic poisoning in Bangladesh and West Bengal (internet). WHO: 1999.

   http:// www.who.int.org (Accessed 5th November 2005).

6. Chowdhury UK, Rahaman MM, Mandal BK, Paul K, Lodh D, Biswas BK. Groundwater arsenic contamination and human suffering in West Bengal, India and Bangladesh..Environ Sci.2001; 8: 393-415.

7. Ahmad SA, Sayed MHSU, Hadi SA. Arsenic: Pani Dushan O Shastha Samasya. 1st edition; 2000: 35.

8. Dhar RK, Biswas BK, Samanta G, Mandal BK, Chakraborti D, Roy S, Jafar A. Groundwater arsenic calamity in Bangladesh. Current Sci.1997; 73 (1): 48-59.

9. Chowdhury M. Ground water tragedy and our duties. The Bangladesh Observer.2006.Sunday; March 19: 1 – 3.

10. NGOs. Arsenic Information and support unit (NAISU), NGO forum for drinking water supply and sanitation. An overview of arsenic issues and mitigation initiatives in Bangladesh; 2003: 7-8.

11. Ahmad SA, Sayed MHSU, Khan MH. Arsenicosis: Neoplastic manifestations of skin. Journal of Preventive and Social Medicine (JOPSOM). 1998; 17(2):110-115.

12. Ahmad SA, Muyeen-us-Safa AKM, Sayed MHSU. Arsenic in drinking water and urinary level of arsenic. Environmental Science. 2001; 8 (5): 417 – 423.

13. Ohtsuka PR, Yamamoto PK, Watanabe DC, Inaoka DT. Effect of chronic ingestion of arsenic contaminated water in rural communities of Bangladesh. Massachusetts Institute of Technology. University of Tokyo. Swiss Federal Institute of Technology; 2001:1 – 4.

14. Ahmad SA, Sayed MHSU, Hadi SA, Farequee MH, Khan MH, et al. Arsenicosis in a village in Bangladesh. International Journal of Environmental Health Research. 1999; 9: 187-198.

14. BGS and DPHE. Arsenic contamination of ground water in Bangladesh/ Kininburgh DG and Smedley PL ed. Vol. 2: Final report. British geological surveys report WC/ 00/19 key worth, UK. British Geological Survey: 2001.

15. Ahmad SA. Arsenic: water contamination and health hazard. 1st ed. Rajshahi. Udayan Press; Bangladesh; 2000: 32-37.

16. Smith AH, Lingas EO, Rahman M. Contamination of drinking water by arsenic in Bangladesh. A public health emergency. Bulletin of the World Health Organization. 2000; 78(9): 1093-1103.

17. BAMWSP. Data Book. Bangladesh Arsenic Mitigation Water Supply Project; 2004:5.

18. Barabhuiya MAK, Sayed MHSU, Khan MH, Ahmad SA. Arsenic contamination and its consequence on human health: A review. International workshop on arsenic mitigation, Dhaka. Research studies on health impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 2002: 295-300.

19. World Health Organization. Arsenic in drinking water (internet). Fact sheet no.210. Bangladesh: 2001.

http: //www. chelationtheraphyonline.com /articles/ p99.htp (Accessed 1st March 2007).

20. Sun G, Li X, Pi J, Sun Y, Li B, Jin Y, et al. Current research problems of chronic arsenicosis in China. Journal of Health, Population Nutrition .2006; 24(2):1.

21. Maharjan M, Watanabe C, Ahmad SA, Ohtsuka R. Arsenic contamination in drinking water and skin manifestations in lowland Nepal. The first community based survey. Am.J.Trop. Med. Hyg.2005; 73(2): 477-479.

22. Tun KM, Nyi S, Myint T, Htut T, Nyunt MT, Thein M, et al. Arsenic contamination of ground water: an emerging health concern. Regional health from, regional health from WHO South East Asia region. 2003; (7): 1 – 6.

23. Susheela AK. Arsenic in drinking water: A public hazard. Nutrition Foundation of India; 1998: 1 – 3.

24. School of Environmental Studies (SOES). Jadavpur University, Ground water arsenic contamination in West Bengal-India (20 years study); 2006: 1-6.

25. Ahmad SA, Maharjan M, Watanabe C, Ohtsuka R. Arsenicosis in two villages in Terai, lowland Nepal. Environmental science.2004.11; 3:180.

26. Rich CH, Peralta C, Browning SR, Huang B, Ferreccio C, Caminero AG, et al. Arsenic exposure and reproductive outcomes: preliminary report from a pregnancy cohort study in Chile. arsenic/conferences/sandiego/abstract; 2000: 2, 5.

27. Ahamed S, Sengunta MK, Mukherjee SC, Pati S, Mukherjee A, Rahman MM et al. An eight-year study report on arsenic contamination in groundwater and health effects in eruani village Bangladesh and an approach for its mitigation. J Health Popul Nutr. 2006; 24 (2):129 – 141.

28. Li D, An D, Zhou Y, Liu J, Waalkes MP.Current status and prevention strategy for coal-arsenic poisoning in guizhou, china. J Health Popul Nutr.2006. Xxx.24 (0); 000-000:1 – 3.

29. Moinuddin. Arsenic contamination as a major health hazard in Bangladesh. Part three. Drinking death in groundwater; 2002: 1 – 5.

http:// www.acdis.uiuc.edu/Research/Ops/Moinuddin/Contents/ Part3.html.

30. Rahman M, Vahter M, Sohel N, Yunus M, Wahed MA, Streatield PK, et al. Arsenic exposure and age and sex specific risk for skin lesion: A population based case referent study in Bangladesh; 2006: 1- 6.

31. Health and Environment. Arsenic invasion. Newsletter from the Centre for Science and Environment.2005; (3):1, 1 – 5.

32. Dhaka Community Hospital. Arsenic pollution in ground water of Bangladesh; 2003: 1 – 7.

33. Mahmood SAI. Water is life but arsenic there is the silent killer. The Daily Star. 2002. Thu, July23:1-3. (Internet):http:www.eng-consult.com /arsenic /as249.txt (Accessed 20th Oct 2006).

34. Rahman MM, Chowdhury UK, Mukherjee SC, Mondal BK, Paul K, Lodh D,et al. Chronic arsenic toxicity in Bangladesh and west Bengal India – A review and commentary. J Toxicol Clin Toxicol. 2001; 39(7): 683 – 700.

35. Chen CJ, Tseng MP, Hsu Li, Tseng CH. Individual susceptibility to arseniasis;2000: 1 – 2.

36. Rahaman MM, Sengupta MK, Ahmad S, Chowdhury UK, Lodh D, Hossain MA. Arsenic contamination in groundwater and its health impact on residents in a village in West Bengal, India. Bulletin World Health Organization. 2005; 83: 49-57.

37. Wasserman GA, Liu X, Parvez F, Ahsan H, Litavak PF,vanGreen A, et al. Water arsenic exposure and children's intellectual function in Araihazar Bangladesh. Environ Health Perspect. 2004; 112: 1329-33.

38. Ahsan H, Perrin M, Rahman A, Parvez F, Stute M, Zheng Y, et al. Associations between drinking water and urinary arsenic levels and skin lesions in Bangladesh. JOEM. 2000; 42 (12): 187-200.

39. Milton AH, Rahman M. Environmental pollution and skin involvement pattern of chronic arsenicosis in Bangladesh. J Occop Health.1999; 41: 207-208.

40. Sikder MS, Maidul AZM, Momin A. Clinical manifestation of chronic arsenic toxicity in Bangladesh-A 250 case study. Bangladesh J. Dermatol. Venereol. Leprol. 1999; 16(1): 6-8.

41. Tondel M, Rahaman M, Magnuson A, Chowdhury IA, Raruqee MH, Ahmad SA. The relation ship of arsenic levels in drinking water and the prevalence rate of skin lesions in Bangladesh. Environmental Health Perspectives. 1999; 107(9): 83-85.

42. Ahmad SA, Sayed MHSU, Hadi SA, Khan AW. Modified arsenic field test kit: A cheap and easy device for detection of arsenic in water. Journal of Preventive and Social Medicine (JOPSOM). 1997; 16 (2): 145-150.

43. Khan AW, Ahmad SA, Sayed MHSU, Hadi SA, Khan MH, Jalil MA, et al. Arsenic contamination in ground water and its effects on human health with particular reference to Bangladesh. Journal of Preventive and Social Medicine (JOPSOM).1997); 16(1): 65-73.

44. Maidul AZM, Momin A, Akramullah SM, Ahmed Z, Afsar S, Salauddin A, et al. Arsenical keratosis (Chronic Arsenism) -22 cases study. Bangladesh J. Dermatol. Venereol. Leprol.1996; 13(1): 1-4.

45. Hsueh YM, Cheng GS, Wu MM, Chen CJ. Multiple risk factors associated with arsenic induce skin cancer. Environmental Science.1988; 71(1):109-14.

46. Smith AH. Nutritional modulation of arsenic health risks: 2000.

http://phys4.harvard.edu/~wilson/ arsenic / conferences / sandiego / abstract. 2, 3.

47. Ahmad SA, Sayed MHSU, Hadi SA, Barua S, Khan MH, Faruquee MH, et al Arsenic in drinking water and pregnancy outcomes. Environmental Health Perspectives. 2001; 109 (6): 629-631.

48. Rahman M, Tondel M, Ahmad SA, Axelson O. Diabetes mellitus associated with arsenic exposure in Bangladesh. Am J Epidemol. 1998; 148(2): 198-203.

49. Rahman M. Non-malignant health effects of arsenic exposure. In Division of occupational and environmental Med. Dept.of health and env. Faculty of health science. Linkoping Uni. Eds. Linkoping University Medical Dissertation. Sweden: Linkoping University Press. 1999; 189 (11): 55-120.

50. Ahmad SA, Bandarnyake D, Khan AW, Hadi SA, Uddin G, Halim MA. Arsenic in ground water and arsenicosis in Bangladesh. Int. J. Environ. Health Res. 1997; 7: 271-6.

51. Khan AW, Ahmad SA, Hadi SA, Farquqee MH, Khan MH. Arsenic: Silent catastrophe. Environment on Health.1997; 1: 32-38.

52. Akhtar HH, Karim F, Chowdhury MEEK, Rahaman MH. A study to identify the risk factors affecting nutritional status of adolescent girls in Bangladesh. Bangladesh Institute of Research for Promotion of Essential and Reproductive Health and Technologies (BIRPERHT).1998; xvii-xx: 7-12.

53. Jakaria M. The use of alternative safe water options to migrate the arsenic problem in Bangladesh. Community Perspective. BRAC Research and Evaluation Division Dhaka. Bangladesh; 2003: 4,23,24,26.

54. National research council. Arsenic in drinking water. Washington. DC; academic press: 1999.

55. National Research Council. Arsenic in drinking water 2001 update, Washington DC; National Academic Press; 2001: 2-7.

56. Smith AH, Smith MMH. Arsenic drinking water regulations in developing countries with extensive exposure. Toxicology. 2004; 198: 39-44.

57. Minamoto K, Taylor CGNM, Moji K, Karim E, Rahaman M. A study on arsenic contamination water and extent of acute childhood malnutrition (Wasting) in rural Bangladesh. Environmental Science. 2005; 12(5): 283-291.

58. Smith AH, Arroyo AP, Mazumder DNG, Kosnett MJ, Hernandez AL, Berris M, et al. Arsenic induces skin lesions among Atacameno people in North Chile despite good nutrition and centuries of exposure. Environmental Health Perspective. 2000.1081 ;(7): 617-620.

59. Hsueh YM, Huang YL, Huang CC, Wu WL, Chen HM, Yang MH, et al. Urinary levels of inorganic and organic metabolites among residents in an arseniasis hyperendemic area in Taiwan. J. toxicol. Environ.Health. 1998; 54(6): 431-44.

60. ICDDR' B. Arsenic exposed and its impact on health in children. Periodical Journal of Health, Population and Nutrition. 2006. 24; (2): 1-3.

61. Health and Environment. Lead story. News Letter from the Centre for Science and Environment. 2005; Jan-Feb: 1 – 4.

62. Chen CJ, Wu M, Lee SS. Atherogenicity and carcinogenicity of high arsenic well water. Multiple risk factors and related malignant neoplasm of black foot disease. Arteriosclerosis.1988; 8(5): 452-60.

63. Hsueh M, Chen GS, Wu MM, Kuo TL, Chen CJ. Multiple risk factors associated with arsenic induced skin cancer. Effects of chronic liver diseases and malnutrition status.Br.J.Cancer.1995; (71):109-14.

65. Siripitayakunkit U, Lue S, Choprapawan C. Possible effects of arsenic on visual perception and visual motor integration of children in Thailand. Arsenic / conferences / sandiego / abstract.2000; 2: 4.

66. Picciotto IH. Interactions between arsenic and other exogenous exposures in relation to health outcomes: 2000. http:// phys4.harvard.edu /~wilson/Arsenic/ conferences / sandiego / abstract: 2, 4.

67. Gamble MV, Liu X, Ahsan H, Pilsner JR, Ilievski V, Slavkovich V, et al. Folate, homocysteine, and arsenic metabolism in arsenic exposed individuals in Bangladesh. Environ Health Perspect.2005; 113(12):1683-8.

68. Kwok RK, Kaufmann RB, Jakaria M. Arsenic in drinking –water and reproductive health outcomes: A study of participants in the rural Bangladesh integrated nutrition programme. J health Popul Nut.2006; 24(2): 190-205.

69. Biswas S, Talukder G, Sharma A. Prevention of cytotoxic effects of arsenic by short term dietary supplementation with selenium in mice in vivo.muta.res. 1999; 44 (1):155-60.

70. Chakraborti D, Sengupta MK, Rahaman MM, Ahmad S, Chowdhury UK, Hossain MA. Groundwater arsenic contamination and its health effects in the Ganga- Megna- Bramoputra Plain. J Environ Monti. 2004; 6: 74N-83N.

71. Milton AH, Hasan Z, Shahidullah SM, Sharmin S, Jakariya MD, Rahaman M, et al. Association between nutritional status and arsenicosis due to chronic arsenic exposure in Bangladesh. International Journal of Environmental Health Research.2004; 14(2):99-108.

72. Shahidullah S, Sharmin S, Jakaria M, Rahman M, Dear K, Smith W, et al. Association between nutritional status and arsenicosis due to chronic arsenic exposure in Bangladesh. International Journal of Environmental Health Research. 2004; 14 (2): 99-108 (10).

73. Kabir MI, Rahman M, Flora MS, Azad AK. Arsenicosis and body mass index in a selected area of Bangladesh. Journal of Preventive and Social Medicine (JOPSOM). 2001; 20(1):6- 12.

74. Rahman A, Vahter M, Ekstrom EC, Rahman M, Mustafa AHMG, Wahed MA, et al. Association of arsenic exposure during pregnancy with fetal loss and infant Death: A cohort study in Bangladesh. American Journal of Epidemiology; 2007:1-9.

75. von Ehrenstein OS, Mazumder DNG, Smith MH, Ghosh N, Yuan Y, Windham G. Pregnancy outcomes, Infant mortality and arsenic in drinking water in west Bengal, India. American Journal of Epidemiology. 2006; 163(7):662-669.

76. Hopenhayn C, Ferreccio C, Browing SR, Huang B, Peralta C, Gibb H, et al. Arsenic exposure from drinking water and birth weight. Epidemiology. 2003; 14(5): 593-602.

77. Statistical Pocket Book of Bangladesh. Bangladesh bureau of statistics planning division. Ministry of Planning Government of the people's Republic of Bangladesh; 2002:3,126, 363.

78. Ali SMK, Pramanic MMA. Conversion factors and dietary calculation Dhaka. Institute of Nutrition and Food Science. University of Dhaka; 1999:9-11.

79. Ahmad SA, Faruquee MH, Sayed MHSU, Khan MH, Jalil MA, Ahmed R, et al. Chronic arsenicosis: Management by Vitamin A, E, C regimen. Journal of Preventive and Social Medicine (JOPSOM).1998; 17(1): 19-26.

80. International Labour Organization. Encyclopedia of Occupational Diseases. 4th Ed. Geneva; ILO. 1983; 179-182.

81. Chen CJ, Chion HY, Hung WI, Chen SY, Hsuch YM, Tseng CH, et al. Systemic non-carcinogenic effects and development toxicity of inorganic arsenic. In: Ed Abemathy co, Calderon RL, Chappell WR, Eds. Arsenic exposure and health effects. Madras: Chapman and Hall; 1997:124-134.

82. Mazumder DNG, Gupta JD, Santra A, Ghose A, Sanker S, Chattopadhaya, et al. Non-cancer effect's chronic arsenicosis with special reference to Liver damage. In: Ed Abemathy CO, Calderon RL, Chappell WR, Eds. Arsenic exposure and health effects. Madras: Chapman and Hall; 1997: 112-123.

83. Caussy D. Facilitator guide, Detection, Management and Surveillance of Arsenicosis in South East Asia Region. WHO technical publication no.30. ISBN 92 9022 264 5; 2005: 1-136.

84. Agency for Toxic Substances and Disease Registry. Toxicological profile for arsenic. Atlanta, GA: ATSDR, US Department of Health and Human Services, Public Health Services: 2000.

85. Rahman M, Tondel M, Ahmad SA, Chowdhury IA, Faruquee MH, Axelson O. Hypertension and arsenic exposure in Bangladesh. Hypertension. 1999; 33: 74-78.

86. Agency for toxic substance and disease registry (ATSDR). Toxicological profile for arsenic (draft). U.S Public health service, U.S. Department of Health and Human Services, Atlanta, GA: 1998.

87. World Health Organization. Air Quality Guidelines. 2nd Ed. Geneva. WHO; 1988: 171-178.

88. International Labour Organization. Encyclopedia of Occupational Health and Safety. 3rd Ed. ILO, Geneva. 1983; 1: 1179-1182.

89. Klasen CD. Casarette and Dowell's Toxicity: The basic sequence of poison 6th Ed. Sanfrancisco: Becker and Sutherland; 1992: 818-822.

90. Smith AH, Rahman M. Arsenic in drinking water: A public health point of view. Medicine Digest. 2001; 3(1): 3-7.

91. Clayton GD, Clatton FE, Patty's. Industrial Hygiene and Toxicology. 3rd Rev. Ed. New York, John Willie Sons .1981; 24: 1517-1531.

92. Environmental Health Criteria 224. Arsenic and arsenic compounds. 2nd Ed. World Health Organization; 2001: 3, 4, 7, 103.

93. Khan AW. Health effects on arsenic toxicity. Consultation on arsenic in drinking water and resulting arsenic toxicity in India and Bangladesh. New Delhi, India. World Health Organization (South East Asia).Research studies on Health Impact of Arsenic Exposure. Bangladesh Medical Research Council (BMRC); 1997:325-329.

94. World Health Organization. Arsenic, Environmental Health Criteria 18. Geneva; 174: 1981.

95. Rantnaike RN. Acute and chronic arsenic toxicity, Postgrad, Med.J. 2003; 79:391- 396.

96. Panthi SR, Sharma S, Mishra AK. Recent statues of arsenic contamination in groundwater of Nepal- A review, Kathmandu University of Science and Technology. 2006; 11(1):1-2.

97. Shama G. Removal of arsenic from groundwater. The Katmandu post. 2003. Nepal; Sun, October: 1 – 2.

98. World Health Organization. Early detection of occupational diseases. Geneva: World Health Organization; 1986: 3-11.

99. Smith R. Toxic Metals: Arsenic: A murderous mistory, Dartmouth toxic metals research program. A program of the center for environment health science of Dartmouth; 2001:1-5.

http://www.dartmouth.edu /~toemetal /TXSHas.shtml.

100. Khan AW, Ahmad SA. Arsenic in drinking water: Health effects and management. Research Studies on Health Impact of Arsenic Exposure. Bangladesh Medical Research Council (BMRC); 2002:338-350.

101. World Health Organization (1981). Environmental health criteria 18: arsenic Geneva;World Health Organization: 1-3.

102. Smith H, Dekaminsky R, Niwas S, Soto R, Jolly P. Prevalence and intensity of Ascaris lumbricoides and Trichuris trichiura and associate socio-economic variables in four rural Honduran communities. Mem Inst Oswaldo Cruz. 2001; 96(3): 303-14.

103. Rahaman MM. Socio-economic factors influencing the nutritional status of under five children in a rural village. Northern Medical Journal. 1995; 4(1): 10-14.

104. Ahmad TS. Effects of chronic arsenic exposure on nutritional status of children 6-12 years in a selected area of Bangladesh. Research studies on health impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 2002: 396-397.

105. Khan AW, Ahmad SA, Sayed MHSU, Hadi SA, Faruque MH, Khan MH, et al. Groundwater arsenic contamination and its effects on human health in Bangladesh. Journal of Diarrheal Disease Research.1999; 17(2):1.

106. Smith AH. Excerpts from 'Technical report and action plan for arsenic in drinking water in Bangladesh focusing on health'. In consultation on arsenic in drinking water and resulting arsenic toxicity in India and Bangladesh. New Delhi. WHO; 1997: 3-9.

107. Chatterjee A, Das D, Mandal BK, Chowdhury TR, Samanta G, Chakraborti D. Arsenic in ground water in six districts of West Bengal, India: the biggest arsenic calamity in the world. Part-1. Arsenic species in drinking water and urine of the affected people. Analyst.1995; 120:643-50.

108. Hotta N. Clinical aspects of chronic arsenic poisoning due to pollution in and around a small refining spot. Jap. J. Cons. Med. 1989; 53 (1.2): 49-70.

109. Smith AH. Cancer risks from arsenic in drinking water: A public health point of view. Medicine Digest.1992; 1(3):1-3.

110. Ahmad SA, Sayed MHSU, Hadi SA, Farequee MH, Khan MH, Jalil MA, et al. Health's effects due to arsenic toxicity in Bangladesh. The 3rd forum on arsenic contamination of ground water in Asia. Miyazaki University research group for arsenic contamination in ground water. Research studies on health impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 1998:304-306.

111. Ahmed F, Khan MR, Akhtaruzzman M, Karim R, Marks GC, Banu CP, et al. Efficacy of twice –weekly multiple micronutrient supplementation for improving the hemoglobin and micronutrient status of anemic adolescent schoolgirls in Bangladesh. American Journal of Clinical Nutrition.2005. Vol. 82; No.4: 829-835.

112. Smith AH, Lingas EO, Rahman M. Contamination of drinking water by arsenic: A public health emergency. Bulletin of the WHO. 2000; 78(9): 1093-1103.

113. Chowdhury QI. Arsenic poising: we are at risk. The Daily Star.2002.Thu, Jan10; 3(836):1-6. http:www.thedailystar.net /daily star news /20001 /10/ n2011009.htm (Accessed 22nd October 2006).

114. BGS and DPHE. Arsenic contamination of ground water in Bangladesh/ Kininburgh DG and Smedley PL ed. Vol. 2: Final report. British geological surveys report WC/ 00/19 key worth, UK. British Geological Survey: 2001.

115. British Geological Survey (BGS): 2000.

http// www. bgs.ac.uk/ Arsenic/ bphase 1/ B find.htm#.

116. Nickson R, McArthur J, Burgess W, Ahmed M, Ravenscroft P, Rahman M. Arsenic poisoning of Bangladesh ground water (letter). Nature.1999; 395 (700): 338.

117. Acharyya SK, Chakraborty P, Lahiri S, Raymashay BC, Guha M, Bhowmik A. Arsenic poisoning in the Ganges delta. Nature 401.1999; (6753): 545.

118. Hussain AZMI. Arsenic contamination of ground water in Bangladesh. A briefing paper Ministry of Health and Family Welfare in Bangladesh; 1999:1 – 5.

119. Yamauchi H, Kinoshita J, Nagai N, Shimazaki K, Kasamatu M, Saito S, et al. Study on the metabolism of arsenic in the patients with acute arsenic poisoning by intake of arsenic trioxide in wakayama city, Japan. Arsenic / conferences / sandiego / abstract; 2000: 2, 5.

120. Crecelics EA. Change in the chemical speciation of arsenic following ingestion by man. Environmental Health Perspectives.1977; 19: 147-150.

121. World Health Organization. Use and interpretation of anthropometric indicators of nutritional status. Report of WHO working group. Bulletin of the World Health Organization. 1986; 64(6): 929-941.

122. Landrigan PJ. Arsenic-state of the art. Am.J Ind Med. 1981; 2: 5-14.

123. Kilburn KH. Neurobehavioral impairment from long-term residential arsenic exposure. In Ed Abemathy CO, Calderon RL, Chappell WR, Eds. Arsenic exposure and health effects. Madras: Chapman and Hass; 1997:159-175.

124. Rahman M, Vahter M, Wahed MA, Sohel N, Yunus M, Stereatfield PK, et al. Prevalence of arsenic exposure and skin lesions. A population survey in Matlab, Bangladesh.J Epidemiol Community Health; 2000: 1.

125. Rom WN. Editor. Environmental and Occupational Medicine. 2nd Ed. New York; Little: Brown and Company; 1992: 5-9.

126. Rahman M, Quamruzzaman Q, Das R, Biduyt JY, Shohel AE, Rahman RCMA, et al. Health hazards of arsenic poisoning, In: Ahmed MF, editor. Bangladesh environment 2000. International Conference on Bangladesh. Dhaka. Bangladesh; 2000: 135-150.

127. Mandal BK, Chowdhury TR, Samanta G. Arsenic in ground water in seven districts of West Bengal, India- The biggest arsenic calamity in the World. Curr. Sci.1996; 70(11): 976-986.

128. Saha KG. Chronic arsenical dermatosis from tube well water in West Bengal during 1983-87. Ind. J. Dermatol. 1995; 40:1-12.

129. Mazumder DNG. Treatment of chronic arsenic toxicity as observed in West Bengal. J. Ind. Med. Asso.1996; 94(2): 41-42.

130. Valentine JL, Reisbord LS, Lachenbrunch PA. Health response by questionnaire in arsenic-exposed population. J. Clin. Epidemiol. 1992; 45(5): 487 – 494.

131. Agency for toxic substance and disease registry (ATSDR). Case studied in environmental medicine. Arsenic toxicity. Public health service, U.S. Department of Health and Human Services, Atlanta, GA: 1990.

132. U.S. Environmental protection agency. Integrated risk information system (IRIS) on arsenic. National centre for environmental assessment, Office of the research and development, Washington, DC: 1999.

133. Hussain AZMI. Arsenic contamination of ground water in Bangladesh. A briefing paper Ministry of Health and Family Welfare in Bangladesh; 1999:1 – 5.

134. Ahmed MF. An assessment of arsenic problems in Bangladesh. International workshop on arsenic mitigation, Dhaka. January 14-16. Research studies on health impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 2002: 269-277.

135. Smith AH, Mahfujar R. Arsenic in drinking water: A public health point of view. Medicine Digest. 2001; 1(3): 1-2.

136. Smith AH. Do nutritional deficiencies increase susceptibility to arsenic induced health effects? National Institute of environmental health science. Environmental Health News; 2006: 1-2.

http:// www.niehs.nig.gov /dert /profiles/hilites/2004/nutridef.htm.

137. Khan MAK, Choudhury SAR, Misbauddin M, Maidul AZM, Sahjahan M. Effects of spirulina in the treatment of chronic arsenic poisoning in Bangladesh. Bangladesh Journal of Medical Science; 2001: 7(1).

138. Huq MA, Misbauddin M, Choudhury SAR. Spirulina in the treatment of chronic arsenic poisoning. Bangladesh J Physiol Pharmacol. 2000; 16(1): 15-16.

139. Chowdhury KK, Hanifi MA, Sabrina R, Bhuiya. A. gender inequality and server malnutrition among children in a remote rural area of Bangladesh. J Health Popul. Nutie.2000; 18 (3): 123-130.

140. Sikder MS, Maidul AZM, Khan MAK, Huq MA, Choudhury SAR, Misbauddin M. Effects of spirulina in the treatment of chronic arsenicosis. Bangladesh J. Dermatol. Venereol. Leprol. 2000; 17(1): 9-13.

142. Corinne H, Robinson, Marilyn R, Lawler. Food and its relation to health, normal and therapeutic nutrition. 15th edition; 1995:3- 4.

143. Gibson R. Methods used in Nutritional Assessment. Principals of Nutritional Assessment; 1990: 4-5.

144. Gibson SR. Anthropometric assessment, in principles of nutritional assessment. Oxford University press, New York, Oxford; 1990: 155-207.

145. Frisancho AR. Anthropometric stands for the assessment of growth and nutritional status. Published in the United State of America by the University of Michigan press manufacture in the united state of America; 1999: 39-43.

146. Bangladesh Bureau of Statistics. Child nutrition survey of Bangladesh. (1995-96). Ministry of Planning (statistics division). GOB; 1997: 7-12.

147. Waterlow JC, Buzina R, Keller W. The presentation and use of height and weight data for comparing the nutritional status of groups of children under the age of 10 years. Bulletin of the World Health Organization. 1977; (55):489-498.

148. Faruquee DMH. Study on case management of chronic arsenicosis. Bangladesh Medical Research Council; 2002:385.

149. Ali SMK, Malek MA, Salamatullah Q. Deshio Khaddo Drabber Pushtiman (original in Bangali) Dhaka. Institute of Food and Nutrition Science; University of Dhaka; 1992: 1-7.

150. Beaton G, Kelly J, Kevany R. Appropriate uses of anthropometrics indices in children 1990 December. Acc/scn state of the art series on nutrition policy discussion paper no. 7. United Nations. Administrative committee on coordination/subcommittee on nutrition; 1990:1-51.

152. Garrow JS, James WPT, Ralph A. Human nutrition and dietetics. Churchill Livingstone Publisher Limited; 2000: 518.

153. Emsley, John. Nature's Building Blocks: An A-Z Guide to the Elements. Oxford: Oxford University Press; 2001:43,513, 529.IBSN o-19-850341-5.

154. IARC. Metal and Metallic compounds. IARC Monographs on the evaluation of carcinogenic risk of chemicals to humans. vol 23. Lyon, France: International Agency for Research on Cancer; 1990: 438.

155. IARC. Some inorganic and Organic metallic Compounds. Monographs on the evaluation of carcinogenic risk of chemicals to humans, vol 2.Lyon, France: International Agency for Research on Cancer; 1973: 181.

156. Frangstrom B, Moore S, Nermell B, Kuentl L, Goessler W, Grander M, et al. Breast- feeding protects against Arsenic Exposure in Bangladesh Infants. Environmental Health Perspective. 2008. Vol 116; Num 7: 1.

157. Maharjan M, Watanabe C, Ahmad SA, Umezaki M, Ohtsuka R. Mutual interaction between nutritional status and chronic arsenic toxicity due to groundwater contamination in an area of Terai, lowland Nepal. Journal of Epidemiology and Community Health .2007; 61: 389-394.

158. Chung JS, Haque R, Guha Mazumder DN, Moore Le, Ghosh N, Samanta S,et al. Blood concentration of methionine, selenium, beta carotene, and other micronutrients in a case control study of arsenic induced skin lesion in west Bengal, India. Environ. Res.2006; 101(20): 230-7.

159. Steinmaus C, Carrigan K, Kalman D, Atallah R, Yuan Y, Smith Ah. Dietary intake and arsenic methylation in a US. Population. Environ Health Perpect.2005; 113 (9): 1153-9.

160. Mitra SR, Mazumder DNG, Basu A, Block G, Haque R, Samanate S, et al. Nutritional factors and susceptibility to arsenic caused skin lesions in west Bengal, India. Environ Health Perspective. 2004; 112(10): 1104-9.

161. Mazumder DN, Haque R, Ghosh N. Arsenic levels in drinking water and the prevalence of skin lesions in west Bengal, India. Int.J.Epidemiol.1998 ;(27):871-872.

162. Borgono JM, Vicent P, Venturino H, Infante A. Arsenic in the drinking water of Antofagasta. Epidemiological and clinical study before and after the installation of the treatment plant. Environ, Health Perspect.1997; (45):5895-9.

163. Zaldivar R, Gullier A. Environmental and clinical investigations on the endemic chronic arsenic poisoning in infants and children. Zbl. Bakt. Hyg. I.Abt. Orig.b. 1997; 165:226-34.

164. SOES. International conference on arsenic in ground water: causes effect and remedy. School of Environmental Studies (SOES), Jadavpur University Calcutta; 1995: 11-23.

165. Choprapawon C, Porapakkham Y. Occurrence of cancer in arsenic contaminated area.Ronpibool district, Nakorn Srithmmarat Province, Thailand. Arsenic / conferences / sandiego/ abstract.2000; 2:3.

166. Arsenic exposure from drinking groundwater transylvania, Romania. An overview eugens, gurzau. Environmental Health centre. cluj napoca, Romania. arsenic / conferences / sandiego / abstract. 2000; 2: 6.

167. Kadono T, Inaoka T, Murayama N, Ushijima K, Nagano M, Nakamura S, et al. Skin manifestations of arsenicosis in two villages in Bangladesh. International Journal of Dermatology. 2002; 41(12): 841, 846.

168. Maharjan M, Shretha RR, Ahmad SA, Watanabe C, Ohtsuka R. Prevalence of arsenicosis in Terai, Nepal. Journal of Health, Population Nutrition.2006; 24(2): 1.

169. Guha M, Chattapadyay A, Nagdeva DA. Environmental health catastrophe in eastern India: A case study of arsenic morbidity in west Bengal: The Internet Journal of Third World Medicine. 2006; vol 3: no 2.

170. Hore SK, Rahman M, Yunus M, Das CS, Yeasmin S, Ahmad SA, et al. Detection arsenic related skin lesions: Experience from a large community based survey in Bangladesh. International Journal of Environmental Health Research. 2007; 17(20: 141-149.

171. Islam LN, Nabi AHMN, Rahman MM, Khan MA, Kazi A. Association of clinical complications with nutritional status and the prevalence of leucopenia among arsenic patients in Bangladesh. International Journal of Environmental Research and Public Health. 2004; 1 (2): 74-84.

172. Watanabe C, Miyazaki K, Ohtsuka R, Inaoka.T. Arsenic contamination of ground: a Bangladesh survey riken. Review no.35. Focused on new trends in bio trace elements research; 2001: 19-22.

173. Meharg AA, Rahman MM. Arsenic contamination of Bangladesh paddy field soils: implications for rice contribution to arsenic consumption. Environ Sci Technol.2003; 37: 229-234.

175. Smith AH, Marhall G, Yuan Y, Ferreccio C, Liaw J, von Ehrenstein O, et al. Increase mortality from lung cancer and bronchiectasis in young adults after exposure to arsenic in utero and in early childhood. Environ Health Perspect. 2006; 114(8): 1293-6.

176. Zhang X, Cornelis R, De-Kimpe J. Accumulation of arsenic species in serum of patients with chronic renal disease. Clinical Chemistry. 1996; 42(8): 1231-1237.

177. Minamoto K, Taylor CGNM, Moji K, Karim E, Rahaman M. Arsenic contaminated water and extent of acute childhood malnutrition (wasting) in rural Bangladesh. Environmental Science.2005; 12 (5): 283-291.

178. Nordstrom S, Beckman L, Nordeson I. Occupational and environmental risks in and around a smelter in Northern Sweden V: Spontaneous abortion among female employees and decreased birth weight in their offspring. Herrditas. 1997; 90:291-296.

179. Newman PE. Can reduced folic acid and vitamin B12 levels cause deficient DNA methylation producing mutations which initiate atherosclerosis? Med. Hypothesis.1999; 53:421-4.

180. Mitra AK, Bose Bk, Humayan K, Das BK, Hussain M. In arsenic related health problems among hospital patient in Southern Bangladesh. Journal of Health, Population and Nutrition.2002; 20 (3):1- 2.

182. Akter KF, Owens G, Davey DE, Naidu R. Arsenic speciation and toxicity in biological systems. Rev Environ contam toxicol, 2005; 184: 97-149.

183. Almela C, Algora S, Benito V, Clemente MJ, Devesa V, Súñer MA, et al. Heavy metal, total arsenic, and inorganic arsenic contents of algae food products. Institute de Agroquímica y Tecnología de Alimentos (CSIC), Apartado 73, 46100 Burjassot (Valencia), Spain. J Agric Food Chem. 2002. Feb 13; 50 (4):918-23 11829668.

184. Munoz O, Devesa V, Suner MA, Velez D, Montoro R, Urieta I et al. Total and inorganic arsenic in fresh and processed fish products. J agric food chem. 2000; 48(9); 4369-76.

185. Rahman MA, Hasegawa H, Rahman MM, Rahman MA, Miah MAM. Accumulation of arsenic in tissues of rice plant (Oryza sativa L.) and its distribution in fractions of rice grain. Graduate School of Natural Science and Technology, Kanazawa University, Kakuma, Kanazawa 920-1192, Japan. Chemosphere. 2007; Jun 26: 17599387.

186. Rahman MM, Owens G, Mallavarapu M, Naidu R. Arsenic contamination of vegetables grown in arsenic contaminated areas of Bangladesh and estimation of arsenic intake from vegetables by the local population. Environ Int. 2007;Nov 28: 18054080.

187. Signes A, Mitra K, Burló F, Barrachina AAC. Contribution of water and cooked rice to an estimation of the dietary intake of inorganic arsenic in a rural village of West Bengal, India. Food Addit Contam. 2007; Aug 31:1-10 17852383.

188. Devesa V, Vélez D, Montoro R. Effect of thermal treatments on arsenic species contents in food. Food Chem Toxicol. 2007; Aug 23: 17928121.

189. Tseng CH. The potential biological mechanism of arsenic –induce diabetes mellitus. Toxical Appl Pharmacology. 2004; 197(2): 67-83.

190. Bodwell JE, Kinglev LA, Hamilton JW. Arsenic at very low concentrations alters glucocorticoid receptor (GR) mediated gene activation but not GR- mediated gene repression: complex dose- response effects are closely correlated with levels of activated GR and require a functional GR DNA binding domain. Chem Res Toxical. 2004; Aug 17 (8): 1064-76.

191. Kaltreider RC, Davis AM, Lariviere JP, Hamilton JW. Arsenic alters the function of the glucocorticoid receptor as a transcription factor. Environ Health Prepect. 2001; Mar, 109(3): 245-51.

192. Zierold KM, Knobeloch L, Anderson H. Prevalence of chronic disease in adults exposed to arsenic contaminated drinking water. American Journal of public Health 1936-1937. 2004. Nov, Vol 94; No: 11.

193. Yoshida T, Yamauchi H, Sun FN. Chronic health effects in people exposed to arsenic via the drinking water: dose-response relationships in review. Toxical Appl Pharmacol. 2004. Aug 1; 198 (3): 243-52.

194. Guha M, Chattopadhyay A, Nagdeve DA. Environmental health catastrophe in eastern India: a case study of arsenic morbidity in west Bengal. The International Journal of Third World Medicine; 2006: 1 – 2.

195. Casartt, Doull's. Essentials of Toxicology; 2003:23-29.

196. Website: www carbonbased.com, copyright: 2003 carbon based corporation.

197. JHA AB, Dubey RS. Carbohydrate metabolism in growing rice seeding under arsenic toxicity. Journal of plant physiology. ISSN 0176-1617 CODE JPPHEY. vol. 161, no 7; 2004:867-872.

198. Hasanuzzaman TM. Nutritional status among the arsenic exposed and non-exposed persons in some selected areas in Bangladesh. Research studied on health impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 2001: 401.

199. Chowdhury PSAR, Khan MMAK, Misbahuddin PM, Islam PAZMM, Shajahan DM. Role of Spirulina in the treatment of chronic arsenic poisoning. Bangladesh Armed Forces Medical Journal; XXVII: 2000.

200. Anwar DJ. The poor in Bangladesh suffers effects and treatment of arsenic poisoning. International workshop on arsenic mitigation, Dhaka: 14-16 January. Research studies on Health Impact of arsenic exposure. Bangladesh Medical Research Council (BMRC); 2002: 285-294.

201. Awasthi S, Pande VK. Prevalence of malnutrition and intestinal parasites in preschool slum children in Lucknow. Indian Pediatr. 1997; 34(7):599-605.

202. Oberhelman RA, Guerrero ES, Fernandez ML, Silio M, Mercado D, Comiskey N, et al. Correlations between intestinal parasite, physical growth and psychomotor development among infants and children from rural Nicaragua. Am J Trop Med Hyg .1998; 58(4):470-5.

203. Saldiva SR, Silveira AS, Phillipi ST, Torres DM, Mangini AC, Dias RM, et al. Ascaris-Trichuris association in Brazilian children. Paediatr Perinat Epidemiol. 1999; 13(1): 89-98.

204. Chan L, Kan SP, Bundy DA. The effect of repeated chemotherapy on the prevalence and intensity of Ascaris lumricoides and Trichuris trichura infection. Southeast Asia J Ptro Med Public Health.1992; 23(2): 228-34.

205. Kumar VCS, Kumar AH, Sunita V, Kapur I. Prevalence of anemia and worm infestation in school going girls at Gulbarga, Karnataka. Indian Pediatrics.2003; 40: 70-72.

206 Anathakrishnan S, Pani SP, Nalini P. A comprehensive study of morbidity in school age children. Indian Pediatrics. 2001; 38: 1009.

207. Oliveros Y. 5M Pinoy kids have intestinal worms: health. Sun Star. 2005. Manila.Fri. Oct: 1-2.

208. Rao VG, Aggrawal MC, Yadav R, Das SK, Sahare LK, Bondley MK, et al. Intestinal parasitic infestations, anemia and under nutrition among Tribal adolescent of Madhy Pradesh. Indian Journal of community. 2005; 28(1):1-5.

209. United Nation International Children Emergency Fund. The UNICEF report, The State of the World's Children; 2004: 1-5.

210. Hassan N, Ahmed K. Nutritional status of under – 12 children of Bangladesh. Bangladesh J. Nutr.1988; 1 (1): 1-6.

211. Hassan N, Ahmed K. Nutritional status of under – 12 urban children of Bangladesh J. Nutor.1993; 6 (2): 1-15.

212. Jahan K, Hossain M. Nature and extend of malnutrition in Bangladesh. Bangladesh National Nutritional survey. Institute of Nutrition and Food Science University of Dhaka. 1998; part-1: 112-144.

212. Jahan K, Hassan N. Nutritional status of children and the relationship with some characteristics of mother and children. Bangladesh. 1994; 7(11):1-8.

213. Kabirullah M, Ali MM, Ibrahim M. Socio-economic factors influencing the nutritional status of pre-school children in rural village in Bangladesh. Bangladesh J child health. 1990; 14 (4): 125-131.

214. Sultana K. Factors contributing to protein energy malnutrition in urban Dhaka. Bangladesh of child health. 1985; 9(2):80-89.

215. Ali S, Keramat M. Health survey of students of a primary school. Bangladesh Medical Council (BMRC). 1977. Bulletin; III (2): 124-129.

215. Keramat. Health survey of students of a primary school. BHRC Bulletin.1977. Dec Vol. 111 No; 2:124-129.

216. Nahar N, Afroza S, Hossain M. Incidence of low birth weight in three communities in Bangladesh. Bangladesh Medical Research Council Bull. 1998; (18):157-164.

217. Kaneta CK, Hanif MA, Sabrina R, Bhyiya A. Gender inequality and server malnutrition among children in a remote rural area of Bangladesh. J Health Popul. Nutie. 2000; Dec, 18(3): 123-130.

218. Samanta G, Chowdhury TR, Mandal BK, Biswas BK, Chowdhury UK, Basu BK. Flow injection hydride generation atomic absorption spectrometry for determination of arsenic in water and biological samples from arsenic affected districts of West Bengal, India and Bangladesh. Microchem J. 1999; 62: 174-91.

219. Yasmin F, Badrunahar, Haque MA, Haque MM, Karim MR. Factors influencing intake of the elderly. Journal of Preventive and Social Medicine (JOPSOM). 2004; 23(2):51-60.

222. Gallagher D, Visser M, Sepulvida D, Pierson RN, Harris T, Heymsfield SB. How useful is body mass index for comparision of body fatness across age, sex and ethnic group? Am.J.Epidemol.1996; 143: 228-39.

223. Acheson KJ, Campell IT, Edholm OG, Miler DS, Stock MJ. The measurement of food and energy intake in man- an evaluation of some techniques. American Journal of Clinical Nutrition. 1980; (33): 1147-1154.

224. Gersovitz M, Madden JP, Smickiklas-wright H.Validity of the twenty-four-hour dietary recall and seven-day record for group comparisons. Journal of American Dietetic Association.1978 ;(73): 48-55.

225. Health and Welfare Canada. Nutrition Canada National Survey. Health and Welfare, Ottawa: 1973.

226. Samuelson G. An epidemiological study of child health and nutrition in a Northern Swedish country. Methodological study of the recall technique. Nutrition and metabolism. 1970; 12: 321-340.

227. Assam Public Health Engineering Department, Government of Assam. Problem of arsenic (Internet):2006.

http:/ aphe.nic/topfrl.htm (Accessed 2nd November 2006).

228. Hsueh YM, Chiou HY, Huang YL, Wu WL, Huang CC, Yang MH, et al. Serum beta carotene level, arsenic methylation capability and incidence of skin cancer. Cancer Epidemiol. Biomarkers prev.1997; 6(8):589-96.

229. Sukla H, Gupta PC, Mehta HC, Hebert JR. Descriptive epidemiology of body mass index of an urban adult population in Western India. Journal of epidemiology and community health: 2002.

230. Reyes H, Cuevas RP, Sandol A, Castillo R, Santos JI. The family as a determinant of stunting in children living in conditions of extreme poverty: a case-control study. BHC Public Health. 2004; 4:57.

231. James WPT, ferro-Luzzil A, Weterlow JC. Define of chronic energy deficiency in adults. Report of working party of IDECG. 1998; 42: 969-81.

232. Cook J, Altman D.G, Moore DMC, Topp SG, Holland WW, Elliott A. A survey of the nutritional status of school children: relationship between nutrient intake and socio-economic factors. Brit. J. Pre. Soc. Med: 1973; 27: 91.

233. Douglas J. WB, Simpon HR. Height in relation to poverty, family size and social class: a longitudinal study. Milbank mem. Fd.Quari.1964.42; no.3: 20.

234. Grant MW. Rate of growth in relation to birth rank and family size. Brit. J. Pre. Soc. Med; 1964:18, 35.

235 Chen Y, Ahsan H, Parves F, Howe GR. Validity of food frequency questionnaire for a large prospective cohort study in Bangladesh. Br J Nutr.2004; 92(5):851-19.

236. Brunner E, Stallone D, Juneja M, Bingham S, Mramote M. Dietary assessment in Whitehall II: comparison of 7 d diet diary and food frequency questionnaire and validity against biomarkers.Br J Nutr.2001; 86(3):405-14.

237. Mei Z, Strawn LMG. Standard deviation of anthropometric Z-scores as a data quality assessment tool using the 2006 WHO growth standards: a cross country analysis. Bulletin of World Health Organization.2007; 85: 441-448.

237. Shaaben SY, Marzouk D, Nassar MF, Ezzat NI, Mohamed I. Early detection of protein energy malnutrition in sharkia goernorate. J egupt public health assoc.2005; 80(5-6):665-85.

238. Singh J, Sengupta S. Nutritional status of Sonowal Karachi Children of Dibrugarh District, Assam. Anthropologist. 2007; 9(3): 233-235.

239. Tarozzi A. Growth reference charts and the nutritional status of Indian children. Duke University; 2007: 1-22.

240. Khongsdier R. Body mass index of adult males in 12 populations of Northeast India.Ann.Hum.Boil.2001; 28(4):374-83.

241. Nube M, Asenso-Okyere WK, van den Boom GJ. Body mass index as indicator of standard of living in developing countries. Eur.J.Clin. Nutr. 1998; 52(2): 136-44.

242. Hindmarsh JT, Abernathy CO, Peters Gr. Environmental aspects of arsenic toxicity. In: Sarker B, ed. Heavy metals in the environment. 1st edn. New York: Marcel Dekker; 2002:217-229.

243. Rousham EK. Socio-economic influences on gender inequalities in child health in rural Bangladesh. Eur J Clin Nutr. 1996; 50(8): 560-4.

244. Haque MS. Arsenic: need for use of surface water. Feature of human health.2002; Sep: 1–2.

http://www.bcas.net/Env.Features/Huma health/September 2002/15% 20%to2030.htm.

245. Spallholz JE, Boylan LM, Rahman MM. Environmental hypothesis: is poor dietary selenium intake an underlying factor for arsenicosis and cancer in Bangladesh and West Bengal, India? Science of the Total Environment. 2003; 323 (2004): 21 – 32.

246. Ezeamama AE, Friedman JF, Acosta LP, Bellinger DC, Langdon GC, Manalo DL, et al. Helminth infection and cognitive impairment among Filipion children. J. Trop. Med. Hyg; 2005:540-548.

247. Kutty VR, Soman CR, Kumar V. Pattern of helminthic in primary school children of Thiruvanthapuram district. Kerala research programme on local level development, Centre for development studies. Prasanth Nagar, Ulloor, Thiruvanthapuram; ISBN no: 81-87621-19-2; 2006:1-3.

248. Faruqee DMH. Study on case management of chronic arsenicosis. Bangladesh Medical Research Council; 2002: 385.

249. Mazumder DNG, Ghosh N, De BK, Santra A, Das S, Lahiri S, et al. Epidemiological study of chronic arsenic toxicity in West Bengal.2000. http://phys4.harvard.edu / ~ Wilson /arsenic/conferences/sandiego/abstract. 2:2.

250. Ruan Y, Peterson MH, Wauson EM, Wase JG, Finnell RH, Vorce RL. Folic acid protects SWV/ fnn embryo fibroblasts against arsenic toxicity. Toxicol, Lett. 2000; 117 (3): 129-37.

251. Vahter M, Marafante E. Effects of low dietary intake of methionone, choline or protein on the biotransformation of arsenite in the rabbit.Toxical. Lett. 1987; 37: 41-6.

252. Ferdousi Y, Karim MR, Haque A, Haque M. Factor's influencing nutrient intake of the elderly. Journal of Preventive and Social Medicine (JOPSOM) .2004; 23(2):51-61.

ANNEXURE

জাতীয় প্রতিষেধক ও সামাজিক চিকিৎসা প্রতিষ্ঠান (নিপসম)
মহাখালী, ঢাকা-১২১২।

আমি ডাঃ মোঃ রেজাউল করিম, সহকারী অধ্যাপক, নিপসম, মহাখালী, ঢাকা। আমি ঢাকা বিশ্ববিদ্যালয়ের অধীনে একজন পিএইচডি বিভাগের শিক্ষার্থী গবেষক। আমার গবেষনার জন্য আপনার কাছ থেকে কিছু তথ্যের প্রযোজন। আপনার দেয়া তথ্যাদির গোপনীয়তা রক্ষা করা হবে এবং শুধু মাত্র গবেষনার কাজে ব্যবহার করা হবে। সঠিক তথ্য প্রদান করে আপনি সহযোগিতা করবেন বলে আশা করি।

প্রশ্নমালা

ক্রমিক নং

তারিখঃ

গ্রামঃ

ইউনিয়নঃ

থানাঃ

জেলাঃ

১। আপনার নাম কি? ----------

২। আপনার বয়স কত?---------- ☐

৩। আপনার পরিবারের মোট সদস্য সংখ্যা কত? ☐

৪। আপনার পরিবার কি ধরনের? ☐
১. একান্ন ২. পৃথক

৫। আপনার পরিবারে(৫-১৪) বৎসরের কয়টি ছেলেমেয়ে আছে ☐

৬। আপনার সন্তানদের(৫-১৪বৎসরের) বিবরন দিন:

ক্রমিক নং	নাম	বয়স	লিংগ	শিক্ষাগত যোগ্যতা
১.				
২.				
৩.				
৪.				

৭। আপনার ধর্ম কি? ----------

৮। আপনার শিক্ষাগত যোগ্যতা কি?

১. নিরক্ষর ২. প্রাথমিক (১ম-৫ম) ৩. মাধ্যমিক (৬ষ্ঠ-১০ম)

৪. এস,এস,সি পাশ ৫. উচ্চ মাধ্যমিক (১ম-২য় বর্ষ)

৬. এইচ,এস,সি পাশ ৭. স্নাতক ও উর্দ্ধে পাশ ।

৯। আপনার পেশা কি? ১. গৃহিণী ২. চাকুরী ৩. ব্যবসা

৪. কৃষিকাজ ৫. শ্রমজীবি ৬. অন্যান্য

১০। আপনার পরিবারে উপার্জনকারী কে?

১. নিজে ২. স্ত্রী ৩. সন্তান ৪. অন্যান্য

১১। উপার্জনকারীদের পেশা কি?

১. কৃষিকাজ ২. শ্রমজীবি ৩. ব্যবসা ৪. চাকুরী ৫. অন্যান্য

১২। আপনার পরিবারের মাসিক গড় আয় কত?

১৩। আপনি কি ধরনের বাড়ীতে বসবাস করেন?

১. ইটের তৈরী বাড়ী ২. টিনের চাঁদ, টিনের দেয়াল, কাচা মেঝে

৩. টিনের চাঁদ, বাঁশের দেয়াল, কাচা মেঝে ৪.অন্যান্য

১৪। আপনারা কি ধরনের পায়খানা ব্যবহার করেন?

১.পাকা = সেপটিক ট্যাংক ২. পাকা = ওয়াটার সিল্ড ৩.পাকা = ঢাকা নয়

৪. কাচা = স্থায়ী জায়গা ৫. খোলা পায়খানা

১৫। আপনার বাড়ীতে কি বিদ্যুত আছে ?

১. হ্যাঁ ২. না

১৬। আপনারা কি বসা ভাত খান, নাকি ফেনগালা ভাত খান?

১. বসা ভাত ২. ফেন গালা ভাত

১৭। আপনার ছেলেমেয়েরা সপ্তাহে কতদিন পান্তাভাত খায়?..... দিন

১৮। আপনার ছেলেমেয়েরা সপ্তাহে কতদিন ডাল খায়?..... দিন

১৯। আপনার ছেলেমেয়ে গত তিন মাসে ১. ডাইরিয়া ২. আমযুক্ত পায়খানা ৩. রক্তযুক্ত পায়খানা ৪.হাম রোগে ভুগেছে কি? ☐

১. হ্যা ২. না

২০। আপনার ছেলেমেয়েরা গত তিন মাসে অন্য কোন অসুখে ভুগেছে কি? ☐

১. হ্যাঁ ২. না

(হ্যাঁ হলে বর্ননা করুন)

অসুখের বিবরণ----------

২১। আপনার ছেলেমেয়েরা গত তিনমাস আগে কোন ধরনের কৃমির বড়ি খেয়েছে কি? ☐

১. হ্যা ২. না

২২। বর্তমানে আপনি খাবার পানি কোথা থেকে সংগ্রহ করেন? ☐

১. পুকুর ২. কুয়া ৩. নদী ৪. নলকুপ

৫. গভীর নলকুপ ৬. বৃষ্টির পানি ৭. পি,এস,এফ

৮. অন্যান্য

(যদি উত্তর নলকুপের পানি না হয় তবে ২৪ নং প্রশ্নে যান)

২৩। আপনি নলকুপের পানি কি ভাবে পান করেন? ☐

১. সবাসরি ২. ফুটিয়ে ৩. ফিটকিরি দিয়ে ৪. অন্যান্য

২৪। রান্নার কাজে ব্যবহৃত পানি কোথা থেকে সংগ্রহ করেন? ☐

১. নলকুপ ২. নদী ৩. পুকুর ৪. বৃষ্টির পানি ৫. অন্যান্য

২৫। বর্তমান নলকুপ থেকে আপনি কতদিন ধরে খাবার পানি সংগ্রহ করছেন? ☐

..... বৎসর।

২৬। বর্তমান নলকুপে আর্সেনিক দুষন আছে কিনা?
১. হ্যাঁ ২. না

২৭। বর্তমান নলকুপের পূর্বে আপনি অন্য কোন নলকুপের পানি পান করতেন কিনা?

১. হ্যা ২. না

২৮। হ্যাঁ হলে কত দিন?.....বৎসর

২৯। পূর্বের নলকুপে আর্সেনিকের দুষন ছিল কিনা?

১. হ্যাঁ ২. না

৩০। আপনাব ছেলেমেযেরা প্রতি জনে প্রতি দিন কত গ্লাস খাবার পানি পান করেন?
(১ গ্লাস = ২৫০ মিঃলিটার.)

ক্রমক নং	নাম	বযস	লিংগ	কত গ্লাস
১.				
২.				
৩.				
৪.				

৩১। আপনি কি জানেন লাল ও সবুজ চিহ্নিত নলকুপ কি?

১. হ্যাঁ ২. না

৩২। আপনি কি জানেন আপনার গ্রামের নলকুপ লাল ও সবুজ রং করা হয়েছে?

১. হ্যাঁ ২. না

চেকলিষ্ট

ক্রমিক নং- তারিখঃ

পরিবার প্রধানের নামঃ

ঠিকানাঃ- গ্রামঃ

ইউনিয়নঃ

থানাঃ

জেলাঃ

১। বর্তমান নলকুপের আর্সিনিকের মাত্র ..... মিঃ গ্রাম/লিটার

২। পূর্বের নলকুপের আর্সিনিকের মাত্র ..... মিঃ গ্রাম/লিটার

৩। ৫-১৪বৎসরের ছেলে মেয়েদের বিবরন ঃ

ক্রমিক নং	নাম	বয়স	লিংগ	ওজন (কেজি)	উচ্চতা (সে.মি.)
১.					
২.					
৩.					
৪.					

৪। ছেলেমেয়ের পায়খানা পরীক্ষার বিবরণঃ-

(পেটে কোন ধরনের কৃমি আছে কি না)

ক্রমিক নং	নাম	বয়স	লিংগ	পরীক্ষার ফলাফল কৃমির উপস্থিতি + / -
১.				
২.				
৩.				
৪.				

গত ২৪ ঘন্টার পরিবারের সদস্যদের পৃথক পৃথক ভাবে খাদ্য গ্রহণ সংক্রান্ত আন্তপারিবারিক তথ্য (ফিফল পদ্ধতি)

তারিখঃ | | | | | | |

গ্রামঃ ইউনিয়নঃ থানাঃ জেলাঃ

সদস্যের নামঃ বয়সঃ লিংগঃ ১= পুরুষ, ২= মহিলা

খাওয়ার সময়	খাদ্য তালিকা ও বর্ণনা	রান্না করা খাদ্য					রান্না ছাড়া খাদ্য		গৃহীত খাদ্যের খাদ্যোপযোগী কাঁচা ওজন (গ্রাম)
		পারিবারিক মাপ	সম্পূর্ণ ওজন (গ্রাম)	উপকরণ	কাচা ওজনে রুপান্তরের গুনীতক	উপাদানের কাঁচা ওজন (গ্রাম)	পারিবারিক মাপ	সম্পূর্ণ ওজন (গ্রাম)	
সকালের নাস্তা									
মধ্যবর্তী সময়ে গৃহীত খাবার									

খাওয়ার সময়	খাদ্য তালিকা ও বর্ণনা	রান্না করা খাদ্য					রান্না ছাড়া খাদ্য		গৃহীত খাদ্যের খাদ্যোপযোগী কাঁচা ওজন (গ্রাম)
		পারিবারিক মাপ	সম্পূর্ণ ওজন (গ্রাম)	উপকরণ	কাঁচা ওজনে রূপান্তরের গুনীতক	উপাদানের কাঁচা ওজন (গ্রাম)	পারিবারিক মাপ	সম্পূর্ণ ওজন (গ্রাম)	
দুপুরের খাবার									
বিকালের নাস্তা									
রাতের খাবার									

7. Annexure

Translated from Bengali

National Institute of Preventive and Social Medicine (NIPSOM)
Mohakhali, Dhaka-1212.

The questionnaire to assess nutritional status among the children of age group 5-14 years in selected arsenic exposed and non-exposed area in Bangladesh.

Exposed/ Non-exposed

I----------, have come to you on behalf of Dr. Md. Rezaul Karim, Assistant Professor, NIPSOM to collect some information for research on "Nutritional status among the children of 5-14 years in arsenic exposed and non-exposed areas". All of your information will be used for research purposes. If you allow me I can start interviewing. Thank you.

Interview Questionnaire

Serial No: ☐ Date:

Vill: Union: Thana: Dist:

1. Respondent: ☐

I. Father II. Mother

2. What is your name (respondent)? ----------

3. How old are you? ----------

4. What is the name of your spouse? ----------

5. How old is your spouse? ----------

6. How many members' are there in your family? ----------

7. How many children do you have in the family between 5-14 years? ----------

8. Can you please give following information about your children between 5-14 years of age?

Sl no.	Name of the children	Age	Sex	Educational qualification

9. What is your educational qualification?
I. Primary II. Secondary III. S.S.C IV. H.S.C V. Graduation

10. What is the educational qualification of your spouse?
I. Primary II. Secondary III. S.S.C IV. H.S.C V. Graduation

11. What is your religion?
I. Islam II. Hindu III. Buddhists IV. Christian. V. Others

12What is your occupation?
I. Agriculture II. Business III. Service IV Others.

13. What is the occupation of your spouse?
I. House wife II. Agriculture III. Business IV. Service V. Others

14. What is the monthly income of your family?

15. What is the type of your family?
I. Nuclear II. Joint III. Others

16. What is the type of house you live in?
I. Complete brick II. Tin roof, tin wall and kacha floor
III. Tin roof, bamboo wall and kacha floor
IV. Complete kacha.

17. What type of latrine do you use?
    I. Pucca: septic tank II. Pucca: water sealed
    III Pucca: not sealed. IV.Kacha: permanent place V. Open latrine

18. What is the present source of drinking water?
    I. Tube-well II. Pond III. Rainwater IV. River water V. Other

19. How you drink tube-well water (If answer is tube-well water)?
    I. Directly II. Boiling III. Use fitcari IV. Other

20. What is the source of water you use for cooking purpose?
    I. Tube-well water II. Pond III. Rainwater IV. River water V. Other

21. How long have you been drinking water from the current source (tube-well)?

22. Have you heard about arsenic contamination in your current source of water (tube-well)?
    I. Yes II. No

23. Except the current tube well did you drink water from any other tube well?
    I. Yes II. No

24 .If yes how many yeas? ----------

25. Do you know whether the previous tube well was arsenic contaminated?
    I. Yes II. No

26. Do you know the meaning of red and blue marks of tube well?
    I. Yes II. No

27. Have you heard about red or green mark of tube well in your village?

    I. Yes II. No

28. How do you process rice for eating?
    I. Basha bhat II. Fen gala bhat

29. How many days in a week do your children take panta bhat? ----------

30. How many days in a week do your children take dal? ----------

31. How many glasses of water do your children drink in a day (1 glass=250ml)

Sl no.	Name of the children	Age	Sex	How many glasses

32. Had any of your children of 5-14 years of age been suffered from any of the Following disease during last three months?

I. Diarrhea II. Amoebic Dysentery III. Blood Dysentery IV. Measles v. Any major illness (Typhoid fever, acute respiratory tract infection).

33. Had any of your children of 5-14 years of age been suffered from any other diseases?

I. Yes II. No

If yes, please explain ----------

---

34. Did any of your children between 5-14 years of age take any antihelmintic in last three months?

I. Yes. II. No

Signature of the interviewer---------- Date:

Name of the interviewer:

Exposed/ Non- exposed

Check List

Serial no: Date:

Vill: Union: Thana: Dist:

1. Arsenic level of present tube-well----------mg/L

2. Anthropometric measurements:

Information about the children between 5-14 years of age of the respondent family.

Sl no.	Name of the children	Age	Sex	Weight (kg)	Height (cm)

3. Stool findings (worm infestation) of the children between 5-14 years of the respondent family?

Sl no.	Name of the children	Age	Sex

Signature of the interviewer---------- Date:

Name of the interviewer:

Dietary intake in last 24 hours

Meal	Item	Serving size	Cooked weight (gm)	Food code	Raw weight(gm)
Breakfast					
Mid morning					
Lunch					
Afternoon					
Supper					

Signature of the interviewer---------- Date: ----------