LIPOPROTEIN PROFILE AS A PREDICTOR OF ISCHAEMIC HEART DISEASE IN CHILDREN OF DHAKA CITY

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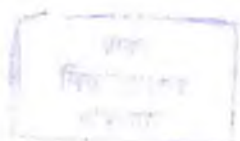
Dedicated to

My Parents & Family



ABSTRACT

Introduction

Lipid levels in young children are useful parameters and are also useful for development of preventive measures of coronary heart diseases. In order to determine the lipid profile among school children of 10-12 years and other determinants of potential ischaemic heart disease (IHD) in children of Dhaka, the present study was carried out in higher, middle and lower economic family.

Methods

A cross sectional study was conducted in 392 school children drawn from three schools of Dhaka city. In order to recommend preventive measures, the children were examined and laboratory tests were carried to identify for determinants of ischaemic heart disease and suggest ways and means to prevent these diseases.

The children were divided into higher (US $ >244 and above) middle (US $122-243.9) and lower (US $24-121.9) socio-economic status by monthly parental income and school standards (English, Bengali and underprivileged groups).

Results

Serum lipids were categorized into desirable and undesirable groups by using conventional cut-off points of TC-C/HDL-C and LDL-C/HDL-C ratios. Higher proportion 31(23.1%) of lower school status children had undesirable limit TC-C/HDL-C ratio as compared with higher school status children 22(16.4%). Similar difference was also observed for LDL-C/HDL-C ratio (17.2% vs. 20.9%). Mean waist hip ratio (WHR) among girls was more (0.86) among lower school status children in our study.

Undesirable lipids ratios among underprivileged children due to higher consumption of carbohydrate and fat. Over and above they don't have facilities for physical exercise. Living in deprived situation children who consumed high carbohydrate and low fat and protein and inadequate exercise, showed low level of mean HDL-C (26.4 ± 26.4). Boys 18(4.6%) and girls 16(4.1%) had lower HDL-C.

Conclusion

Lipid profile in children predicts potential atherosclerotic burden. These findings might help in formulating appropriate programs for controlling long-term preventive programme for IHD and may lead further studies.

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DR.MD.QUMRUL JALIL

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GLOSSARY

Lipoprotein	Lipoprotein is complex particles Comprising of A hydrophilic coat of phospholipids, free cholesterol and specific Polypeptide termed as apolipoproteins around a core of triglyceride and cholesterol ester, which present in varying proportion.
Hyperpercholesterolaemia	**the** lipids that are of relevance In hyperpercholesterolemia are cholesterol, an essential component of membrane and precursor of steroid hormone synthesis and trglycerides, an important energy source.
Low-density lipoprotein (LDL)	**is** the reservoir of 70% of body's Cholesterol, the lipid form correlating Strongly with IHD.
High density lipoprotein (HDL)	**Mainly** phospolipid (50%) correlating Inversely With IHD. Also called 'good' cholesterol, because it finds and rescues struck LDL into pieces and bring back into liver.
Chylomicron	**Mainly** triglyceride, the lowest density Lipoprotein which carry triglyceride derived fat.
Very low density lipoprotein (VLDL)	**Mainly** triglyceride (65%), correlating Conversely with IHD. VLDL becomes LDL after it unloads fat.

KEY WORDS

AIIMS	All India Institute of Medical Science
BMI	Body Mass Index
BMR	Basal Metabolic rate
CHD	Coronery Heart Diasease
CVD	Cardio Vascular Disease
CSD	Clinical Sciences Division
CI	Confidence Interval
DBP	Diastolic Blood Pressure
EFA	Essential Fatty Acid
E CG	Electrocardiogram
FA	Fatty Acid
FBS	Fasting Blood Sugar
FFA	Free Fatty Acid
FFQ	Food Frequency Questionnaire
HDL	High Density Lipoprotein
HDL-C	High Density Lipoprotein
IHD	Ischaemic Heart Disease
ICDDR,B	International Centre for Diarrhoeal Disease Research, Bangladesh
IDL	Intermediate density lipoprotein
LDL-C	Low density lipoprotein
LCT	Long Chain triglycerides
MI	Myocardial Infarction
MAC	Mid Arm Circumference
MRI	Magnetic resonance Imaging
MCT	Medium Chain Tryglycerides
n	Samle size
OR	Odd Ratio
PL	Phospholipid
PUFA	Polyun saturated fatty acid
QI	Quetelet Index
RR	Relative Risk
SBP	Systolic Blood Pressure
TC-C	Total Cholesterol
TG	Triglyceride
TG-C	Triglyceride Cholesterol
Vs	Versus
WAM	Weight for Age Median
WHR	Waist-hip ratio
WHO	World Health Organization

CHAPTER- 1

INTRODUCTION

CHAPTER-1

I. INTRODUCTION

1.1 Background Information

Cardiovascular diseases (CVD) are leading cause of morbidity and mortality in developed countries but they are also emerging as major health problem in developing countries including Bangladesh.

Epidemiological findings have established clear relationships between increased serum cholesterol and Low-density lipoprotein (LDL) level and coronary heart disease (CHD). High-density lipoproteins (HDL) has been recognised as an independent risk factor for coronary disease, but elevated HDL cholesterol a better predictor of CHD risk than either cholesterol or LDL. In the well known Framingham Study, showed after the age of 60, HDL level varies inversely with coronary disease[1]. So, LDL/HDL ratio gives an independent prediction of the risk of coronary heart disease. A close correlation among the extent of pre-atherosclerotic lesion, with serum LDL and total cholesterol concentrations have been shown in children[2] as well as in adults[3] in several studies.

The importance of cholesterol in children is recognised from the epidemiological investigation in children across and within different population. In cross-population studies, children from countries having high incidence of coronary artery disease in adults have higher cholesterol levels than children from countries where adults have

low incidence of coronary artery disease[4]. In a study of the progeny shown that 51% had abnormal levels of lipids or lipoproteins among young victims of IHD. A high serum cholesterol concentration in early adult life is strongly associated with mid life cardiovascular disease[5].

Cardiovascular disease (CVD) has been regarded as the commonest cause of death in industriahsed countries. An increasing trend in the prevalence of CVD, and more specifically, coronary heart disease (CHD) in the developing countries is also observed affecting millions of people by its various forms.

Measures have been taken towards the causal analysis of CVD. Mortality and morbidity rates can be reduced by drugs intervention on CHD risk factors such as arterial hypertension and hypercholesterolemia (hyperbetalipoprotenemia). This implies a long term, often life-long drug treatment (both anti-hypertensive and lipid modifying). It was found that if after the drugs were withdrawn, the risk factors level rapidly restored to its base line. Thus the pharmacological approach for mass prevention of cardiovascular disease (CVD) has low prospects. Results from various hospital, clinic and community based studies shown various risk factors like: dietary habits, family history of ischaemic heart disease (IHD), and its relationship with socio-economic status[6].

People living in Mediterranean countries had lower incidence of cardiovascular disease (CVD) as compared to those in other western countries in spite of their customarily high fat consumption. This might be due to their high consumption of olive oil in their food. Olive oil has the highest level of sequelaen.[7]

It is essential to know the lipid profile among the children. But in Bangladesh there is very little study on this aspect. Early detection and initiation of intervention may be helpful for prevention of cardiovascular disease (CVD) among adult. Hyperlipidaemia in children may lead to hyperlipidaemia with CVD of adult. Our study findings will be helpful in formulating appropriate programs for control of Coronary heart disease (CHD) and also primordial prevention is essential for progression of CHD in future.

The purpose of this study is to know the lipid profile among children and to find out the determinants of cardiovascular disease in selected school of Dhaka city. Coronary heart disease is the commonest form of heart disease and the single most important cause of premature death in the developed world. In the UK one in three men and one in four women die from this disease; an estimated 330 000 people have a myocardial infract each year and approximately 1.9 million people have myocardial infract each year and approximately 1.9 million people have angina. The death rates from coronary heart disease in the UK are declining slowly; unfortunately, the incidence of the condition is increasing rapidly in Eastern Europe and many developing countries[8].

1.2 Rationale of the Study

There is no data on the distribution of lipid profile among the paediatric population in Bangladesh. However, early detection of this health problem might help in early initiation of intervention, thereby arresting the progression of cardiovascular disease (CVD) and ultimately reduces the CV risk among adult. This needs to be investigated, because a finding of hyperlipidaemia in school children could lead to earlier detection and prevention of coronary heart disease (CHD) and to further studies on the relationship of childhood hyperlipidaemia with CVD of adult. Intervention programme aimed at early of life may arrest progression of CVD and reduce the overall morbidity and mortality due to CVD among adult.

1.3 Objective of the study

General Objective:

To study the lipid profile of school children (age 10-12 years) in Dhaka city and asses their potential ischeamic heart disease.

Specific Objective:

i) To determine the blood lipid level of school children aged 10-12 years by a standard method.

ii) To record seven day dietary practices of school children in Dhaka.

iii) To measure anthropometric measurement (Height, Weight, Mid arm circumference and Waist hip ratio) of children in selected schools in Dhaka .

CHAPTER – 2

LITERATURE REVIEW

Chapter-2

LITERATURE REVIEW

A. DIETARY

2.1 Metabolism of Dietary Cholesterol

Nutrition is a powerful environmental factor, which does not only support the vital activities of the human body but is able to influence it either pathologically or preventive measure with drugs. So, dietary prevention was more effective, and absence of side effects, and possibility of long-term application. Over the last decade few institute of preventive cardiology has been involved in research on the diet in the development of CHD and its risk factors among the paediatric population[6].

Samuel and McNamara[9] have reported that significant lumen-mucous exchange does not occur in humans, and uptake of radioactive cholesterol from the lumen release easily reflects the absorption of dietary cholesterol. One puzzling feature in estimations of cholesterol absorption is that a constant fraction of cholesterol tracer appears to be absorbed regardless of the cholesterol intake. Since absorption of cholesterol is not complete, this finding is contrary to what might be expected, namely, that with increasing cholesterol intake, the percent absorption would decrease, as in other biological systems.

Dietary cholesterol is not absorbed completely, this amounts to 25% - 75%, and there is considerable individual variation. Most available techniques use radioactive

tracers, and some investigators doubt whether the rate of tracer uptake by intestinal mucosa accurately reflects absorption of cholesterol. They claimed that luminal cholesterol exchanges with mucosal cholesterol [10,11] and thus the rate of disappearance of radioactivity from the lumen overestimates the mass absorption.

In spite of methodological problems, there is an ample observation that absorption is variable from one person to another. Therefore, it can be required whether fractional absorption of individuals affect their plasma level of cholesterol, In recent investigations, McNamara measured the percentage absorption of dietary cholesterol in a group of men with a range of plasma cholesterol levels and found no correlation between percentage absorption and plasma cholesterol concentration. But Miettinen and his co-workers divided a group of middle-aged Finnish men into three subgroups- - those with high, moderate, or low levels of LDL cholesterol- and estimated the percentage absorption of cholesterol in them. Those with the highest levels of LDL cholesterol had the highest percentage absorption of cholesterol, while those with the lowest levels had the lowest percentage absorption.

Whether an increment in absorption of cholesterol produces a feedback inhibition on the synthesis of cholesterol is yet to be resolved. Peter Edwards reviewed the regulation of cholesterol synthesis as influenced by the activity of several key enzymes. If feedback inhibition were to balance exactly the increment in absorption, the net change in body pools of cholesterol would be zero and dietary cholesterol would not raise the plasma cholesterol level. Increasing amounts of dietary cholesterol does raise the plasma cholesterol in some people indicates that the feedback system is not perfectly regulated. Feedback inhibition of cholesterol synthesis is not the only mechanism preventing hypercholesterolemia after high intakes of cholesterol. Quintao et al demonstrated that a portion of newly absorbed cholesterol is resecreted

into bile. This leads to an increase in biliary cholesterol and serves to rid the body of a fraction of excess dietary cholesterol. Another protective mechanism is increased conversion of cholesterol into bile acids. This serves to prevent hypercholesterolemia in cholesterol-fed rats and dogs, but in humans, enhanced formation of bile acids in response to dietary cholesterol has not been consistently shown[12].

2.2 FAT METABOLISM

Digestion

Fats in the diet are broken down into triglycerides and fatty acids by a pancreatic lipase in the small intestine before they can be absorbed. The processes of fat digestion and absorption are very efficient and over 95% of the consumed foods are digested and absorbed. Up to 250g/day or even more, can be absorbed if the body needs energy.

The newborn baby has to adapt to the relatively high fat content in breast milk after consuming on glucose as an energy substrate in foetal life. He can digest fat, but not as efficiently as the older child or adult, because his pancreatic and biliary secretions are not fully developed[13]. Neonatal fat digestion is aided by the activity of a lipase secreted from the lingual serous glands and in the stomach, hydrolysis occurs, without the need for bile salts, at a pH of around 4.5-5.5[14]. There is also evidence that a lipase in human breast milk contributes to fat digestion in the newborn[15].

Later in life, the process of fat digestion also begins in the stomach, forming emulsion. This is not hydrolysed, but enters the small intestine and is acted upon bile and pancreatic juice[16]. The biliary secretion contains bile acids that are formed in the liver from cholesterol.[17]

As digestion progresses, the lipolytic products pass into "mixed micelles": large molecular aggregates consisting of monoacylglycerols, fatty acids, bile salts and phospholipids. The mixed micelles have hydrophobic which contains the less water-soluble molecules such as cholesterol, the carotenoids, tocopherols and some undigested triacylglycerols.

Absorption

Lipid absorption occurs mainly in the jejunum. The digestion and products pass from the mixed micelles into the enterocyte membrane by passive diffusion. A diffusion gradient is maintained by 1) the presence of a fatty acid binding protein which immediately binds to fatty acids entering the cell, and 2) the rapid re-esterification of fatty acids to the monoacylglycerols, which are the main digestion products crossing the intestinal mucosa[15].

Defects in Digestion and Absorption

Failure to assimilate lipids of dietary origin into the body may arise from maldigestion or absorption[18] (malabsorption). Maldigestion can occur because of incomplete lipolysis. Thus pancreatic insufficiency, from pancreatitis, a pancreatic tumour or in diseases of malnutrition such as kwashiorkor, can lead to a failure to secrete enough lipase. Alternatively there may be enough functional lipase but a failure to produce bile because of biliary disease, with obstruction of the bile duct, or chronic liver disease. The commonest cause of biliary insufficiency in affluent societies, however, arises from bile-salt deficiency induced by surgical resection of

the ileum, where active transport of bile salts occurs. Bile-salt deficiency results in an inability to effect micellar solubilisation of lipolysis products. Gastric disturbances also affect digestive efficiency; thus, mal digestion seems to arise from defects in a variety of organs contributing to from defects in a variety of organs contributing to different aspects of the digestive process. Malabsorption may occur even when digestion is functioning normally, due to defects in the absorptive surfaces of the small intestine. There may be a variety of causes, some common ones being bacterial invasion or sensitisation of the gut to dietary components such as gluten, as in celiac disease. Malabsorption syndromes are characterised by dramatic changes in the morphology of the intestinal mucosa. The epithelium is flattened and irregular, and atrophy of the villi reduces the absorbing surface. A common feature of all fat mal absorption syndromes is a massively increased excretion of fat in the faeces, which arises not only from unabsorbed dietary material but also from the bacterial pollution that proliferates in the gut. Patients with poor fat absorption are at increased risk of deficiencies of energy, fat-soluble vitamins and essential fatty acids.[19]

2.3 Dietary Cholesterol and Risk factors for Coronary Heart Disease

Epidemiological Evidence

Lewis Kuller suggested that observational investigations are potentially valuable experiments of nature. He defined three types of studies: 1) comparison of different populations, 2) time trends of food intake versus trends in mortality, and 3) migration studies. He concluded that all three are appropriate for evaluation of a common source epidemic.

Chicago Western Electric, Zutphen, Boston-Irish and Honolulu Studies

Richard Sheklle reviewed four published studies with reliable assessments of cholesterol intakes. In which adjustments were made for variation in calorie intake: the Chicago Western Electric, the Zutphen, the Boston-Irish, and the Honolulu Studies. All four investigations found a positive association between the cholesterol in-take and subsequent of CHD, and in some studies, this association appeared to be independent of plasma cholesterol concentrations. Sheklle emphasised the limitations of population-based studies in assessing any nutrient, dietary cholesterol in particular: 1) miscalculation of true intake tends to reduce the size of any association and may obscure it entirely, 2) most western populations consume a diet, So high in cholesterol and saturated fatty acids that there is no low risk group for comparison, and 3) the use of clinical end points leads to inaccuracies in quantifying arteriosclerosis, the disease of interest. Post-mortem studies-show that coronary atheroselerosis is common in middle-aged men even when they do not have clinical CHD. They also noted that these four epidemiological studies revealed a positive correlation between dietary cholesterol and development of CHD .

Among the four studies, only the Western Electric Study showed an association between dietary cholesterol and cardiovascular risk independent of dietary fat. Investigators of the Honolulu Heart Study felt that the fact that persons eating high cholesterol die also tend to eat high saturated fat diets precluded meaningful multivariate analysis. The issue of independence was not addressed in the Zutphen or the Boston -Irish Studies.

There has always been great interest in unique populations, but Kuller noted that the problem of confounding variables makes drawing definite conclusions difficult. Populations with unique dietary habits usually differ from comparison populations in several other ways; for instance, unique populations of interest are a Nigerian group that consumes a diet high in saturated fatty acids yet eats little animal fat or cholesterol and vegetarians (compared with so-called pure vegetarians). Comparisons of plasma cholesterol levels and rates of CHD in these populations might help to differentiate between the influence of saturated fatty acids and dietary cholesterol on plasma cholesterol. Turning to trend analysis, Kuller noted the problems of confounding variables; changes in diet may coincide with other changes.

For instance, during the period of declining mortality from CHD in the United States, there were remarkable changes in cigarette smoking, blood pressure control, and treatment of myocardial infarction. All these make it difficult to know the contribution of each factor.

Ni-Hon-San Study

The Ni-Hon-San Study on Japanese men showed that, intakes of dietary cholesterol, levels of plasma cholesterol, and mortality from heart disease increased progressively for men who remained in Japan, moved to Hawaii, or went to San Francisco. This study strongly implicates dietary factors in atherogenesis, although many other changes in lifestyles occurred with migration. Eleanor Pao explain that one-half of the cholesterol foods eaten by American adults comes from meat, poultry and fish. Another one-third is from milk products and eggs. A broad category that includes pastries and cheese paste provides the remainder. Eggs are no longer the primary

source of dietary cholesterol in the United States. Only 15% to 18% of total dietary cholesterol comes from eggs[20].

Socio-Economic Status

The existence of socio-economic inequalities in health has been well established in several studies starting from the behavioural issues upon dietary habits and smoking. Attempts to explain these inequalities have often made reference to the fact that behavioural factors, such as smoking physical activity, and diet, are differentially distributed by socio-economic levels. In addition, psychosocial characteristics, such as depression and cynical hostility, have also been considered as potential explanations for why people at the bottom of the social hierarchy have poorer health. Indeed, considerable evidence has accumulated concerning an inverse relationship between socio-economic status (SES) and the behavioural and psychosocial characteristics, which are important risk factors for poor health. Studies in a variety of industrial countries have shown that lower SES is generally associated with higher rates of smoking 9-32g[21-24], obesity, poor dietary habits [25-30] lower levels of physical activity[31], and higher prevalence of psychosocial that are related to poor health outcome[32-37], while a more complex relationship between SES and alcohol consumption has been reported[24-38,39]. Furthermore, those who occupy lower positions in the socio-economic hierarchy have also been shown to be more resistant to changing risk behaviours than their more advantaged counterparts[40-41].

Health behaviours and psychosocial orientations play an understanding the graded association between lower SES and poorer health, and informing intervention efforts [42,43]. This approach, which views unhealthy behaviours as the consequence of poor

lifestyle management, gained evidence as risk factor epidemiology confirmed that many poor health out comes were associated with the daily conduct of people's lives[44]. Knowles' ideas on individual responsibility for health[45] provided the sort theoretical foundation from which the U.S. Surgeon General, and their counterparts in other countries, could criticise their populations for poor health habits and point out the costs to socio-economic such behavioural transgressions[46].

There are many current examples that at least implicitly reflect this thinking about SES, behaviour, and disease[47]. McGinnis and Foege[48] present and analysis of U.S. data to demonstrate what they characterise as the "root determinants of death and disability". They report that tobacco; alcohol; diet and physical activity are the major contributors to mortality before age 75 year. They also point out that SES is an important factor in premature mortality, they argue that it is difficult to quantify the SES effect independent of smoking, alcohol, diet and physical activity, and they conclude that these public health problems are based in behavioural choices.

The other model of health behaviour agrees that while individuals make choices about how they act, those choices are situated within economic, historical, family, cultural and political contexts. According to this view, these contexts exert important influences on both the process of choice and the types of behavioural options, which are available and, indeed, appropriate. A number of authors have argued that decontextualising behaviour from this real-world setting obscures its socio-economic production and encourages blaming the victims of inequality for their unhealthy lifestyles[49-51]. According to this approach, evidence that socio-economic difference distributes health behaviour should be viewed in a life course perspective, as the cumulative responses of different classes of people to conditions imposed by the social structure[52-54].

In Finland and other industrialized countries found that a clear differences in cardiovascular morbidity and mortality between different social classes. Besides increased morbidity and mortality, lower socio-economic groups also have a more adverse cardiovascular disease risk factor profile.[55,56] The association between socio-economic factors and risk of CVD is partly, but not fully, explained by the traditional risk factors, that the higher levels of smoking, hypertension and cholesterol among people in lower socio-economic groups.[57,58]

B. EPIDEMIOLOGICAL FINDING ON CHD FROM DIFFERENT STUDY AND COUNTRY

2.4 HDL - Cholesterol in Japanese, Americans, and Australians Children

Mortality from CHD in Japan is very much lower than in most developed countries. CHD mortality for both men and women in Australia and the United States is more than five times higher than that in Japan.[59]

Furthermore, CHD mortality in Japan has remained low, as the standard of living has increased, despite an increase in mean plasma TC concentration over the past three decades. Studies conducted in the 1950s reported a mean cholesterol concentration among adults in Japan of 4.0 mmol/L, [60] whereas a national survey conducted in 1989 found that mean concentrations had increased to 5.26 mmol/L for men and 5.14 mmol/L for women 40 to 49 years old, till 60 age-adjusted CHD mortality (range 30 to 69 years) fell by 24% for men and 37% for women over the period 1968 to 1978.[61] Childhood morbidity on CHD has been a contribution of high cholesterol resulting obesity. This has occurred in a setting in which blood pressure for both sexes is higher than in most other developed countries[62] and the proportion of male smokers is much higher than in the US and Australia.[61]

A number of hypotheses have been put forward to explain the apparent inconsistency between increasing westernisation of lifestyle in Japan and the maintenance of low CHD mortality levels.[62] One possibility is that the age cohort whose lifestyle has changed following economic development are not yet old enough to contribute to the CHD mortality statistics.[63] This seems unlikely, because advances in the material standard of living have occurred over a period of at least four decades.[64] That changes in lifestyle have occurred in the "at-risk" age group is reflected in the increase in

serum cholesterol in those >40 as well as <40 years old.[61] Furthermore, in other Asian nations whose economic development occurred later than in Japan, CHD mortality has already risen substantially.[65]

It could be that the high fish intake of Japanese for example, acting via a pathway involving coagulation, may have protected them against the increase in CHD that would have been expected to follow the rise in plasma cholesterol.[66] However, studies in other populations provide estimates for potential risk reduction for consumption of fish that are short of the current difference between Japanese and American CHD mortality[67] **(Annex-4, Table 2.4)** .

Plasma HDL cholesterol and total cholesterol were surveyed in representative populations of schoolchildren in Australia, Japan and US .The mean concentration of plasma HDL- cholesterol (but not total cholesterol) was higher for Japanese schoolchildren than for Australia or US school children ($p < .001$). [68]

2.5 Bogalusa and P DAY Heart Study

An important difference in childhood lipoprotein concentration may explain some of the remaining difference in CHD mortality. Evidence from laboratory and epidemiological studies supports the hypothesis that the concentration of either plasma TC or LDL-C increases risk of CHD, whereas the higher concentration of HDL, reduces risk in adults.[69] In addition, it was shown in the Bogalusa Heart Study[70] and the PDAY study[71] that a higher concentration of both LDL-C and VLDL-C and a lower concentration of HDL-C in children and young adults are associated with a higher risk of premature atherosclerosis. In this article, they compare data on TC and HDL-C in representative samples of the school-age population from Japan and

Australia with data from the Bogalusa study on subjects of the same age in Louisiana. In addition, measures of relevant lifestyle factors, such as diet, and anthropometrical factors that might be expected to influence lipoprotein concentrations are also reported.

2.6 Muscatine Coronary Risk Factor Survey

In 1971 and 1973 Muscatine Coronary Risk Factor Survey, were selected three group of cases for detailed family study, the high group, with cholesterol levels greater than the 95th percentile twice; the middle group, cholesterol levels between the 5th and 95the percentile; and the Low group, cholesterol levels less than the 5th percentile twice. Coronary mortality determined from death certificates was increased in the young relatives (ages 30-59) of the high group index cases, as follows: twofold excess in high male relatives compared with the middle or low group (p less than 0.05); tenfold excess in the high female relatives compared with the middle and low group combined (p less than 0.01). After correction for years at risk, there was an approximately twofold significantly increased coronary mortality. Stroke mortality was higher, although not significantly, in the older relatives (ages greater than or equal to 60) of the high index cases. This study indicates that school children's cholesterol levels cluster with those of their family members and that persistent hypercholesterolemia in children identifies families at risk for coronary artery disease. [72] Report from expert panel on blood cholesterol levels in children and adolescents was Total cholesterol <170 mg/dl and LDL-C <110 acceptable, 170-199 and 110-129 borderline and ≥200 and ≥ 130 mg/dl high in case of Children and adolescents from families with Hyper cholesterol or premature cardiovascular disease.[73]

2.7 Epidemiological Studies in Framingham (U.S.A) Hawaii, Norway and Israel

Interest in human high-density lipoproteins (HDL) has grown largely because of the finding that plasma HDL cholesterol levels are negatively associated with the incidence of coronary artery disease in man. Epidemiological studies in Framingham (U.S.A), [74] Hawaii[75] Norway, [76] and Israel[77]all support this concept, Originally suggested by the studies of Barr et al.[78] and Gofman et al.[79] It has been postulated that HDL-C may be important in preventing cholesterol deposition or removing cholesterol from tissue thereby reducing the amount of lipid deposition in the arterial wall [80,81] Whether HDL-C is a primary factor in protection against coronary-artery disease of merely reflects an association with other factor remains to be determined.[82] Population studies of healthy people suggest that variations in HDL are largely due to changes in HDL-2 [83,84] Thus the variable component of normolipamic plasma HDL cholesterol appears to be HDL.2 cholesterol.[84] When measured directly by analytic ultracentrifugation, HDL-2 was negatively correlated with very-low-density lipoprotein (VLDL.) in three normal populations.[77, 84,85] Indirect findings comes from four epidemiological studies, [73,75,86] in which HDL. Cholesterol was negatively correlated with fasting plasma-triglyceride levels. Plasma-triglyceride concentrations are directly related to VLDL in the fasting state.[84] similar data have been summarised for single groups of healthy subjects in seven countries.[79]

In the Framingham Heart Study, the Lipid Research Clinic's Mortality Follow-up Study and Coronary Primary Prevention Trial, and the Multiple Risk Factor Intervention Trial, the data were consistent with a 2% to 3% decrease in CHD risk for

each 0.03 mmol/L (1 mg/dL) increase in HDL-C level, after adjustment to control for other risk factors. Similar results were found in men and women. The limited information available on interventions that increase HDL-C suggests that this have a favourable effect on CHD. In studies of atherosclerosis regression, coronary arteriographic changes following interventions that increased HDL-C have generally shown positive results.

The Concept that HDL-C may prevent the entry of cholesterol into the process of atherogenesis or even remove cholesterol from atherosclerotic lesions, so-called reverse cholesterol transport, has been supported by animal experiments. In one, HDL-C was infused into rabbits being fed atherogenic diets, and in another, transgenic mice overexposing human apolipoprotein A-I were fed atherogenic diets. In both cases, there was less rapid progression of atherosclerosis.

Observational studies using case-control methods have consistently shown a strong association of increased TG-C with CHD. Most prospective cohort studies similarly show a strong positive relationship between TG-C and CHD, demonstrating a dose-response relationship. However some studies suggest a specific level must be achieved for increased risk. When these cohort studies are subjected to multivariate analysis, controlling for other risk factors, such as blood pressure, physical activity, and obesity, the effect of TG-C is diminished. The addition of indicators of abnormal glucose metabolism or HDL-C eliminates or significantly reduces TG-C as an independent predictor for risk.

One possible explanation for the variability of these data may be found in the heterogeneity of the TG-C containing lipoprotein and the biological variability of the measurement. A single fasting TG-C may inadequately represent this lipid. Individual TG-C rich lipoproteins, chylomicron remnants, intermediate-density lipoproteins,

very low-density lipoproteins (VLDLS), or particles of differing size and composition may be more closely related to CHD. Postprandial TG-C may be more important than the fasting TG-C levels [74, 295].

Several epidemiological studies have demonstrated that high-density lipoprotein cholesterol (HDL-C) is inversely related to the incidence of coronary heart disease morbidity and mortality[87-93]. Fewer studies have established a relation between HDL-C and specific coronary manifestations, including angina pectoris, coronary insufficiency, and myocardial infarction[88, 89,90]. In addition, some researchers report an uncertain or questionable association[94, 95,96] while most fail to provide data for women[89-96]. In this report, a closer examination of the effect of HDL-C as a determinant of the specific manifestation of myocardial infarction is presented for men and women aged 50 and older. They describe the incidence of myocardial infarction as it occurred for various ranges of HDL-C, after adjusting for age and other cardiovascular risk factors. Further examine the effect of HDL-C within groups of subjects with low and high concentrations of total cholesterol and the resulting implications for screening for total cholesterol along. Finding for this report is based on 12 years of follow-up of a sample of subjects originally enrolled in the Framingham study. The relation between high density lipoprotein cholesterol (HDL-C) and the development of myocardial infraction was examined and after 12 years of follow up, men in the bottom three quartile of HDL-C ($\leq$ 52 mg/dl) experienced a 60% to 70% excess of myocardial infraction as compared to men those HDL-C levels were higher ($p<0.05$). The effect of HDL-C was strong in women.[97]

Several studies had examined the relationship between parental cardiovascular disease/CAD risk factors and associated CAD risk factors in children and young adults. Among school children's cholesterol levels cluster with those of their family

members and that persistent hypercholesterolemia in children identifies families at risk for coronary artery disease. [72]

2.8 Waist: Hip Ratio, Obesity and Physical Activity with Risk of CHD

Overweight in childhood has been shown to be predictive of a wide range of adverse health effects, in particular increased morbidity from coronary heart disease.[98,99] In adults, abdominal adiposity is particularly associated with risk factors for cardiovascular disease. The ratio of waist to hip circumference (waist: hip ratio, WHR) has proven a simple and reliable means of estimating abdominal adiposity in epidemiological studies and is frequently used as an indicator of cardiovascular risk in adults[100]. Boys and girls were comparable in BMI of the parents, total cholesterol, TG –C, HDL-C and WHR ***(Annex-4, Table 2.8)***.

In adults, data from observational studies[101], from dietary manipulation in controlled groups[102, 103], and from clinical trials[104, 105] indicate that dietary fat intake, particularly saturated fat intake, influences total cholesterol and low-density lipoprotein cholesterol levels. In children, uniform and significant associations between nutrient intake and serum lipid levels have not been demonstrated within large groups: Nicklas et al.[106] found few significant correlation's between dietary components and serum lipids; Glueck et al.[107] described some significant relations between diet and lipid levels, but the relations were limited to age and sex sub-groups. Shea et al.[108] found that children with higher intakes of total fat or saturated fat had significantly higher levels of TC and LDL-C, but the sample was small. In the past few years several intervention studies have shown that dietary modifications produce changes in blood lipid levels in children.[109-111]

Evidence indicates that when the children's lipid disorder involves high total cholesterol or low-density lipoprotein cholesterol (LDL-C) levels, the parents are also likely to have high cholesterol as well as an increased incidence of coronary heart disease.[112, 113] Thus, knowledge of their children's high cholesterol levels should be a strong incentive for parent to have their own cholesterol screened. However, when the children's lipid disorder involves high tri-glyceride (TG) or low high-density lipoprotein cholesterol (HDL-C) levels, this situation becomes more complicated. Although high TG levels in parents have been reported to be more likely when high TG is found in their children.[114] a similar association has not been well described for the wider spectrum of lipid disorders incorporating both TG and HDL-C, as well as their interaction with LDL-C. To help describe this association more comprehensively, they used a combination classification scheme, conceptualised before, [115,116] to define the disorders of TG and HDL-C, LDL-C, or a combination. They analysed lipid values from 232 families in the randomly recruited comparison population of large genetics study.[117]

Although no studies have demonstrated that children with elevated lipid or lipoprotein values will die earlier than other children relationships have been found between elevated lipid and lipoprotein values and coronary artery plaques in autopsy studies of children and adolescents. [118, 119] Cardiovascular disease risk factors develop early in life, and risk factors tend to track, or be maintained, from year to year; thus identification during early childhood of variables associated with CVD risk factors, such as serum lipid and lipoprotein values, is needed so that appropriate interventions can be targeted at children who are at risk for adult onset of CVD[119-122.]

Cross-sectional studies have found significant relationships between levels of habitual

physical activity and serum lipid and lipoprotein levels among adolescents[119, 123-125.] How ever, conflicting results have been reported in studies in younger children of the associations among physical activity, exercise capacity, and serum levels of lipids.[124, 125-127] Thus it remains unclear whether physical activity and cardiovascular fitness are related to serum lipid and lipoprotein levels among younger children, and whether these relationships are confounded by body composition, gender, or ethnicity Alternatively, physical activity may have an indirect effect on serum levels of lipids and lipoproteins through the influence it may have on cardiovascular fitness and body composition. Using a prospective cohort design for 1 year, they studied the relationships among indicators of physical activity, physical fitness, body composition, and serum lipid and lipoprotein levels of children beginning at 3 or 4 years of age and ending at 4 or 5 years of age. On the basis of studies of folder children and adolescents. Linder and some scientist explain that one year higher levels of physical activity and physical fitness and the lower levels of fatness would be associated with higher levels of high density lipoprotein and of the subclass HDL-2 and with lower levels of total serum cholesterol, triglycerides, and low density lipoprotein and lower LDL/HDL and TSC/HDL ratios. [118, 122,87]

Obesity is related to abnormal lipid and carbohydrate metabolism and is predictive of subsequent cardiovascular disease[127, 128.] However, the associations have often been fairly weak or have been observed only over long periods of follow-up[129, 130] and obesity may be a risk factor in only specific subgroups of overweight persons[131]. Much evidence suggests that high-risk obese individuals be characterised by their distribution of adipose tissue. Vague[132] was the first to document that the male (android) pattern of upper body obesity, characterised in part by a relatively thick skin fold at the nape of the neck, is more strongly related to increased insulin levels,

diabetes, and atherosclerosis than is the female (gynoid) pattern of lower body obesity.

The adverse effects of upper body obesity have also been studied by using the ratio of waist to hip circumferences (WHR) [133] a relatively simple index that emphasises abdominal obesity[134, 135,136] WHR is associated with an increased prevalence of hypertension and diabetes[137, 138] and with levels of triglycerieds, glucose, insulin, and blood pressure[139, 140,141]

Prospective studies have shown that, even after accounting for Quetelet Index (kg/m^2) or various skin fold thicknesses, the risk of coronary heart disease is greatest in both men[142] and women[143] with an increased WHR.

Although the mechanism by which WHR confers an increased risk of disease is not clear, adipose tissue at different locations exhibits metabolic difference[144], which may influence lipoprotein levels. Although this possibility is supported by reports that WHR is related to adverse levels of lipids and lipoproteins, these studies have examined primarily overweight person[139, 140,141.]

Over the past four decades since serum cholesterol levels were first linked to atherosclerotic disease, a number of additional markers have been identified in an attempt to better characterise the atherogenic potential of the lipid profile. Relationships of cholesterol ester-rich lipoproteins (LDL and HDL) with atherosclerosis have been clearly established. Recent data suggest that TG-rich lipoproteins (chylomicrons, chylomicron remnants and VLDL) may also play a role in atherogenesis.[144-149]

Despite extensive research over the past few decades, it remains unclear whether plasma TG-C, as a marker for the TG-C rich lipoproteins, has independent value in predicting risk of cardiovascular disease. A recent National Institutes of Health

consensus conference concluded that the data to support a judgement of a causal relationship of elevated TG-C with cardiovascular disease are "mixed". [150] Most case-control and prospective cohort studies that have examined the relationship of fasting TG-C on risk of cardiovascular disease have reported strong crude associations.[152-153] There appear to be complex metabolic relationships between cholesterol ester-and triglyceride-rich lipoproteins[146-153] and control for their lipid parameters can substantially attenuate the TG association.[151-153] In particular, control for HDL cholesterol, which is inversely correlated with TG, tends to substantially attenuate the association of TG with CHD.[154]

As previously outlined by Austin et al[152] and Crique et al, [155] the assessment of any relationship of TG with risks of cardiovascular disease is complicated by several methodological issues. First, there is considerable within-individual variability in measured TG levels.[156] Second, the distribution of TG levels in the population is not normal.[157] Third, TG are strongly correlated with other lipid parameters.[158, 159] Fourth, there are complex metabolic relationships between the TG and cholesterol ester-rich lipoproteins that may interact to increase risks of cardiovascular disease.[146-155-162] In an attempt to better understand the complex interactions of TG and cholesterol ester-rich lipoproteins, They examined the interrelationships of the fasting TG level, other lipid parameters, and no lipid risk factors with risk of MI in a study of 340 cases and an equal number of control subjects matched on age, sex, and neighbourhood of residence.

Many foods and nutrients have been implicated as protective factors in the aetiology of ischaemic heart disease (IHD). Early interest centred around the atherogenic potential of saturated fatty acids and dietary cholesterol mainly derived from high fat dairy products, fatty meats, and eggs.[163-165] More recent research has suggested that

oils containing certain unsaturated fatty acids, fruits, vegetables, unrefined cereals and fish might protect against IHD because of their antithrombotic properties or their antioxidant nutrient or dietary fibre (non-starch polysaccharide) content.[162-173] It has been claimed that the beneficial effects of the protective foods might be more powerful than the deleterious effects of foods rich in saturated fatty acids.[162-171] These observations have led to some uncertainty as to the most appropriate emphasis for dietary advice intended to reduce the risk of cardiovascular disease among population and individual at high risk of IHD.

They recently confirmed that reduced IHD death rates in vegetarians compared with meat eaters, [172-173] but vegetarian diets differ from those of meat eaters in many respects and published data have not established which aspects of the vegetarian diet are protective. They present information derived from a semi quantitative food frequency questionnaire completed by more than 11000 vegetarians and meat eaters at recruitment to a prospective study between 1980 and 1981. The study differs from previous prospective studies of diet and IHD in that the volunteers were individuals whose self-selected diet[171] resembled, in nutrient content, current dietary recommendations[172] rather than the relatively high saturated fat diet typical of most affluent societies. The findings may not only help to explain which attributes of a vegetarian diet protect against IHD but also which foods and nutrients are important in the aetiology of IHD in populations who modify their diets along the lines of present guidelines. Information on weight and eight was also collected. An increased risk of IHD in obese subjects has been demonstrated in many studies.[173] In their report the change in IHD risk with increasing body mass index (BMI) within the normal range, as very few volunteers were overweight.

2.9 Serum Lipid and Japanese Children

In 1980, an estimated 48% of deaths in developed countries were attributable to cardiovascular diseases, largely related to atherosclerosis and hypertension. The causal contribution of blood lipids most often studied as total blood cholesterol concentration, to this morbidity and mortality has been well established. This process is already evident in childhood.[174] In Japanese teenagers, data from autopsy studies have revealed fatty streaks and fibrous plaques to be present on intimal surface of coronary arteries and, 22% in aortas[175].

In Japan, as recently as 1985, age-standardised mortality from ischaemic heart disease for adults aged 30-69 was the lowest among 35 industrialized countries; it was only one-sixth as high as in the United States, for both men and women, and one-tenth as high in Japan as in Northern Ireland or Scotland[176]. This exceptionally low ischaemic heart disease mortality has been thought to be due especially to population-wide low dietary fat intakes, and consequently low mean serum cholesterol concentrations. It is significant form this viewpoint that three multi-centre surveys (1960, 1970, 1980) and two governmental nation-wide population-based surveys (1980, 1990) in Japanese adults indicated increasing mean values of serum total cholesterol concentration in recent decades[177-181]. Consistent with these observations, between 1955 and 1988, corresponding dietary changes occurred according to the results of the yearly "National Nutrition Surveys," which were governmental nation-wide population-based surveys in Japan. Energy intake from fat increased from 9% to 26% of old total calories, while the proportion of animal fat increased from 14% to 38% of the total fat

intake over this period. Mean while, total energy intake was stable or might have decreased slightly[182]. These changes in serum total cholesterol concentration and fat intake appear to be real, and apparently the age-standardised mortality from ischaemic heart disease increased threefold 1950 to around 1970 but decreased from 1970 to 1992, remaining at about 1.6 times the 1950 rates as of 1992, for both men and women in Japan[183]. This led to identification and review of 30 qualifying reports from 27 studies conducted from 1977- 1992 **(Annex-4, Table 2.9)**.

Prospective studies in Europe and the United State have shown that serum total cholesterol concentration is positively associated with the subsequent risk of coronary heart disease,[184-188] whereas high-density lipoprotein cholesterol (HDL-C) concentration is inversely associated with the risk[189-195]. Three prospective studies in Japan have shown a direct association between serum total cholesterol and coronary heart disease incidence in Japan[196-198]. A study of Chinese workers also showed a direct association between serum total cholesterol and mortality from coronary heart disease[200]. Other small prospective studies of rural Japanese have failed to show an association between serum total cholesterol and coronary heart disease. The small number of publications on blood lipids and coronary heart disease associations in Japan and other Asian countries is in part due to the low incidence and mortality of coronary heart disease[199,201-203] and low population level of serum total cholesterol [199,200,203].

Coronary heart disease incidence is inversely related to HDL-C in urban Japanese middle – aged men, whose mean total cholesterol (5.10 mmol/L) is relatively low. [203]

Trans-isomers of fatty acids constitute about 5% to 6% of dietary fat in the average US diet[204] mostly derived from the partial hydrogenation of vegetable oils[205,206] . Trans-fatty acids are also formed in the rumen of cattle and comprise about 5% of

dairy and beef fat[207]; however, the predominant isomer, transvaccenic acid, is distinct from those derived from vegetable oils. The trans-fatty acid content of typical margarine's in the US market ranges from 10% to 30% of total fat[208]; however, values as high as 60% have been reported[205]. Levels of trans-fatty acids of more than 10% of total fat are also frequent in cookies, crackers, breads, pastries, and French fried potatoes[205]. Due to the replacement of butter and lard with margarine and vegetable shortening, consumption of trans-isomers increased progressively in the United States during the first half of this century, and it has changed little in the past few decades. [204] An adverse effect of trans-fatty acids was heightened by recent reports that they increase circulating low-density lipoprotein (LDL) cholesterol and reduce high-density lipoprotein (LDL) cholesterol and reduce high-density lipoprotein (HDL) cholesterol.[208, 209] However, direct evidence that intake of trans-isomers affects the incidence of coronary heart disease in humans is limited. In one British case-control study[210], persons dying of coronary heart disease had a higher proportion of trans-fatty acids in their adipose tissue than did those dying of other causes; in that population, the trans-isomers were derived largely from partially hydrogenated marine oil. An increased risk of coronary heart disease was recently reported in a large prospective study limited to women[211] To address the hypothesis that higher intake of trans-isomers formed in the partial hydrogenation of vegetable oils increases the risk of myocardial infarction, we examined this relation in a case-control study conducted in the Boston area in 1982 through 1983.

Trans isomers of fatty acids are formed when liquid vegetable oils are partially hydrogenated to form margarine and shortening. This processed vegetable fat, which many people us instead of animal fats containing saturated fat and cholesterol because of health concerns, contain between 5% and more than 30% of the isomers[212]. Trans

isomers contribute to the hardness of the products; the carbon moieties on the two sides of a double bond provide a straight, closely packed configuration, whereas the cis isomers found in natural vegetable oils have a bent configuration [213]. Trans fatty acids are also formed in the rumen of cattle and make up about 5% of dairy and beef fat[207], however, the predominant isomer is structurally distinct from those derived from vegetable oils. Trans isomers are estimated to constitute 5-6% of dietary fat consumed in the USA[204], but the proportion varies widely, depending on food choices.

There has been concern for some time that high intake of trans isomers could adversely affect risk of coronary heart disease (CHD) [207], partly because the target configuration resembles that of saturated fats. Also, since many trans isomers are derived from the naturally occurring cis, cis-linoleic acid, a precursor of prostaglandin, they could have various effects of platelet activity and other important functions. Some, but not all, studies have shown moderate rises in serum total cholesterol with trans isomers[213].

2.10 American Study

The 1985 review by the Federation of American Societies for Experimental Biology conducted that available laboratory evidence was not sufficient to implicate trans fatty acids as a cause of CHD; however, further investigation was recommended[213]. They report that replacement of naturally occurring fatty acid with trans isomers can increase concentrations of low-density-lipoprotein (LDL) cholesterol[215,216]and lipoprotein (a) [216-217] and lower concentrations of high-density-lipoprotein (HDL) cholesterol [215] in their research work.

To investigate whether a higher intake of trans isomers is associated with an increased

risk of CHD, they studied dietary data collected in 1980 from participants in the Nurses Health Study in relation to subsequent risk of CHD.

International comparisons[218-221] and laboratory data[222] suggest those diets high in saturated fat and cholesterol and low in polyunsaturated fat increase the risk of coronary heart disease. These diets increase blood cholesterol concentration[223-226], which is related to risk of coronary disease[227]. The differences between countries, however, are far larger than one would predict based on effects of cholesterol concentrations. Results of prospective epidemiological investigations and randomised trials have been inconsistent; small size or inadequate dietary assessment may explain most of the discrepancies [228-229]. They examined the association between dietary fat and cholesterol and risk of myocardial infarction in a large cohort of men in United States.

To elucidate these relation s further they identified the foods that contributed most to differences in total trans-isomer intake among study participants. By stepwise regression with energy adjusted total trans-isomer intake as the dependent variable, each food was entered as a predictor variable. The four most important determinants were margarine, beef, pork, or lamb as a main dish, cookies (biscuits), and white bread (total $r^2 = 0.82$). They examined each food in relation to risk of CHD by including them in models with control for age and other CHD risk factors **(Annex-4, Table: 2.10)** .[230]

2.11 European Study

There has been speculation that the high cholesterol and saturated fat content of milk received by infants may influence lipid metabolism throughout life[231]. In humans there is little evidence to support this since comparisons of serum cholesterol

concentrations in people who were breast fed or bottle fed after birth have given conflicting results [232-237]. Nevertheless, some observations give credibility to the speculation. An infant's serum cholesterol concentration, unlike that of an older child or an adult, is strongly related to its intake of cholesterol and saturated fat[234, 238-239]. Follow up studies of children has shown that serum cholesterol concentrations tend to track, so those children maintain the rank order by serum cholesterol concentration over several years.[240-244] Tracking has been observed from the age of 6 months In animals experiments it is clearly shown that different early feeding regimens can lead to permanent changes in serum lipid concentrations and in the metabolic activity of enzymes controlling cholesterol synthesis and excretion[245-247].

They measured serum lipids in a group of men in England, where the method of feeding of all babies was routinely recorded 1911 onwards.[248] The recorded information included whether infants were breast or bottle-fed and whether they were weaned at 1 year of age. They have also examined death rates from ischaemic heart disease in relation to method of infant feeding.

Different infant foods are known to have different immediate effects in the human infant. Babies fed on breast milk or cows' milk have higher serum cholesterol concentrations than those fed on modern formula milks, which have a lower cholesterol content and a higher polyunsaturated: saturated fatty acid ratio[239-240]. This sensitivity of infants to the fat content of the diet contrasts with the low correlation between dietary fat intake and serum cholesterol concentrations in older children and adults[234, 241-242].

Different infant foods also affect the excretion of bile acids. Breast-fed and formula fed infants differ in the quantity of bile acids excreted and in the timing of appearance of secondary bile acids, which depends on the action of gut flora [243]. Breast milk

contains several hormones and growth factors that can influence lipid metabolism, including thyroid hormones and steroids [244-245]. Breast milk provides ideal nourishment for the young infant, but there is evidence that some babies who are exclusively breast fed after 6 months receive inadequate energy[246]. Human breast milk contains low iron concentrations and exclusively breast-fed babies commonly develop low iron stores in the latter half of infancy[247]. Breast milk may also be deficient of vitamins, notably vitamin D, if the mother is poorly nourished[248].

Peterson addressed that the men who had been exclusively bottle fed, who comprised only 5% of the sample, were similar to the not weaned group in having higher death rates from ischaemic heart disease and higher low density lipoprotein cholesterol and apolipoprotein B concentrations. Bottle foods available 70 years ago included patent preparations of dried cows' milk, unmodified cows' milk, diluted condensed milk, and patent foods made from wheat flour or arrowroot [249-250]. Modern formula milks differ from these foods: they are fortified with iron and vitamins; the fat content is mainly unsaturated and the electrolyte contents are similar to that of breast milk. It is therefore difficult to assess the relevance of these findings for bottle fed babies today. They shown that those who had higher birth weights at 1 year had lower serum concentrations of apolipoprotein B. this was an independent of whether or not they had been breast-fed beyond 1 year. Wang and his associate explain that a relation between birth weight and apolipoprotein B concentrations has been shown in infants, through in the opposite direction[251] ***(Annex-4, Table: 2.11)***.

To examine whether method of infant feeding is associated with adult serum lipid concentrations and mortality from ischaemic heart disease. 474 men had died from ischaemic heart disease mortality ratios were 97 (95% CI 81 to 115) in men who had been breast fed and had not been weaned at 1 year, 79 (69to90) in breast fed men who

had been weaned at 1 year, 73(59 to 89) in men who had been breast and bottle fed.[252]

2.12 Canadian Study

Significant reduction of ischeamic heart disease is obtainable with relatively modest lowering of blood cholesterol level of most adult Canadians.[253-256] Reducing other risk factors can further decrease CHD.[257, 258] Reports reference values for plasma lipids, the prevalence of the major lipid and lipoprotein CHD risk factors and their relation to other risk factors determined in statistically probability samples of the population in nine provincial heart health surveys carried out between 1986 and 1990. Data from the provincial heart health surveys provide evidence that hypercholesterolemia begins early in life for men with a steady increase starting in the third decade of life; for women this increase is delayed until the fifth decade, around the time of menopause. Those trends are keeping with well-known mortality statistics, which show that CHD becomes the primary cause of death for men by the fourth decade and for women by fifth decade. The increase with age in total plasma cholesterol, LDL-cholesterol and triglyceride concentrations seen in this study has been observed in other populations. This increase in lipid levels with age is characteristic of industrialized countries. Diets rich in saturated fats and dietary cholesterol and life-styles that predispose people to obesity tend to raise plasma lipid levels.[259-261]

Mean plasma cholesterol levels for women were lower than those for men until the age of menopause, at which point they surpassed the mean values of men. It has been postulated that the sudden rise in plasma cholesterol concentration at the time of menopause is probably due to loss of menopause is probably due to loss of estrogens and consequent decrease in LDL receptor activity.[257] Mean plasma triglyceride values increased with age in both men and women. This is probably associated with

increasing prevalence of overweight with increasing prevalence of overweight with advancing age.[262]

For some specific population groups, including postmenopausal women, older men and subjects with low HDL-C level, triglyceride level appears to play an independent role in the development of CHD.[263]

LDL-C is recognized as the major atherogenic lipoprotein fraction. The increasing mean values of LDL-C with advancing age may be related to the increasing prevalence of overweight in older men and women.[262]

HDL-cholesterol has been found to be a negative risk factor or protective CHD factor well into advanced age .In all age group HDL-cholesterol level was lower in men than in women, a finding that has been observed in other population studies.[262, 264]

Of the study population 46% had total plasma cholesterol levels above 5.2 mmol/L, 15% had LDL-C levels above 4.1 mmol/L, 15% had triglyceride levels above 2.3 mmol/L and 8% had HDL-C levels below 0.9 mmol/L. The age- standardized prevalence of obesity was positively associated with elevation of total plasma cholesterol.[265] The prevalence of obesity Canada has been estimated in the 1971 Nutrition Canada national survey, [266] the 1978 Canada Health survey [267] and the 1981 Canada fitness survey.[268] the pattern of obesity among Canadian Indians has also been assessed.[269] Body mass index (BMI) has been used in this report measure obesity because it is a simple, convenient measure that correlates well with skin-fold and body density measures[270] and has been adopted in the Canadian Guidelines for healthy weights.[271]WHR was used to assess the pattern of fat distribution because it is technically simpler and at least as reliable as skin-fold measurement for this purpose.[271,272] High WHR had been defined as 0.8 or over for women 0.9 or over for men. Although, a cut-off point of 1.0 had been used for men in some reports, [273] they

adopted the level of 0.9 used in the Canada fitness survey.[268]

The prevalence of obesity (BMI ≥ 27) increased with age and was greater in men (35%) than women (27%). Abdominal obesity was likewise higher in men and increased with both age and BMI. Total cholesterol levels increased only with BMI, levels of low-density lipoprotein (LDL) cholesterol and triglycerides and ratio of total cholesterol to high density lipoprotein (HDL) cholesterol increased steadily, while HDL cholesterol decreased consistently with increasing BMI.[274] **(Annex-4, Table-2.12.1a,1b,1c,1d).**

2.13 Netherlands Study

In recent decades both longitudinal and intervention a studies have shown that plasma total cholesterol (TC) is an important risk factor for coronary heart disease (CHD). [275-277] More recently, evidence has accumulated that a low high-density lipoprotein cholesterol level (HDL-C) is an independent risk factor for CHD.[278, 279] Coronary heart disease mortality in the Netherlands has declined during two decades, but it is still the most important cause of Death. [280] To gain insight into the prevalence of and trends in plasma cholesterol levels in the general population of Netherlands, a monitoring project was carried out from 1987 to 1992.The prevalence of hypercholesterolaemia in man ranged from 5% in the youngest (20-29 years) to 29 % in the oldest age group and from 4% to 38% in women. Low HDL-C levels in men ranged from 15% in youngest to 26% in the oldest age group, and in women from 4% in the youngest to 7% in the oldest group. The cut-off point for hypercholesterolaemia was 6.5mmol/L (250mg/dL) and low HDL-C was used 0.9mmol/L (35mg/dL) the lipid profile among higher educational level was more favourable than that of the less

educated.[280] In paediatric population lipid profile 2-20 year age group found different. So, in different study found value range of paediatric_references which resulting feature CHD **(Annex-4, Table- 2.13)**.

2.14 Singapore Study

In a cross – sectional survey of the whole of Singapore, cardiovascular risk factors were measured by standardised techniques. For the analysis in the 18-69 year age group there was adjustment for ethnic group, age, body mass index, alcohol consumption and physical activity. Among serum lipids, high-density lipoprotein (HDL-) cholesterol and fasting triglyceride were inversely related with partial correlation coefficients (r) of males –0.34 ($p< - 0.001$) and females 0.26 ($p< –0.001$). In male smoker HDL-C inversely, even after further adjustment for fasting triglyceride, with compared to non-smokers reductions of 3.4% in light smokers 10.3% in moderate smokers and 13.8% in heavy smokers[281] **(Annex-4, Table- 2.14).**

2.15 Tanzania Study

A community – based survey was used to assess the prevalence of risk factors for coronary heart disease in rural Tanzanians. In all, 8581 subjects aged ≥15 years in eight villages in three regions in rural Tanzania representing a range of socio-economic deprivation were studied. The main outcome measures were serum cholesterol and triglyceride level, blood pressure and prevalence of dyslipidaemia, hypertension, smoking, overweight impaired glucose tolerance (IGT) and diabetes. Mean serum cholesterol levels in men were 4.2, 3.4 and 3.4 and 3.7 mmol/L. and in women 4.4, 3.6 and 3.9 mmol/l in Killimanjaro, Morogoro and Mara regions

respectively. Mean serum cholesterol concentrations were significantly higher in women than in men in all three regions. There was no difference in concentration between sexes and no significant age - related trend. Highest prevalence was found in women >55 years in all regions and men of this age in Killimanjaro[282] **(Annex-4, Table: 2.15).**

2.16 Denmark Study

In Denmark, as in other Western countries, a decline in mortality from ischaemic heart disease (IHD) has been observed. The present study assesses whether the decline in IHD mortality is due to a decrease in incidence or case fatality, and whether parallel changes occurred in the various manifestations of IHD requiring hospitalisation.

During the entire period the age – standardised mortality of AMI decreased in both men and women. Case fatality of AMI decreased in both men and women. It was observed that the declining mortality from IHD in Denmark might be partly due to declining incidence as well as declining case – fatality. [283]

2.17 Indian Study

Review from Asian Indians showed that highest prevalence of CHD. Multiple studies revealed that the usual risk factors like hypertension, hypercholesterolemia, obesity, smoking and a family history of CHD, are not common in South Asians. But posses a different risk factor profile characterised by high triglycerides low HDL, glucose intolerance, insulin resistance, abdominal obesity and increased lipoprotein (a) levels[284]. Some community based study survey conclude that social class 1,2 and 3 in an urban population of India have a higher prevalence of CAD and coronary risk

factors hypercholesterolemia, hypertension, diabetes mellitus and sedentary lifestyle in both sexes. [285]

2.18 Bangladesh Study

In Bangladesh, one clinic based study showed that 51.3% of patients below 60 years and 7.14% above 60 years have high serum cholesterol. Another study in Bangladesh reported that, of the 283 cases of ischemic heart disease (IHD), 72% had high serum cholesterol and 71.3% had increased serum TG. The study indicated that the higher prevalence of type IV hyperlipidemia amongst the IHD cases in Bangladesh could be due to increased intake of carbohydrate in the food [286]. In Bangladesh for a family of six member Taka 2600 per month will fulfil the basic needs for food, clothing, housing, health care and education but Taka 1724 per month will be only enough to keep the family on the borderline of starvation and to pay minimal amounts towards meeting other basic needs. The overall literacy rate for the urban population is higher than the national average. However, the literacy rate for the urban poor is for below this average. Survival is the basic concern of the majority of the poor, little consideration is given to the education of children. Some parents also fear that their children will be alienated from them if they become educated and may not take care of them in their old age. The reasons for the low rate of enrolment in schools are: shortage of books, space and teachers is acute, children are required to supplement family income and are not encourage to go to school, the curriculum in formal school programmes is less appropriate for working children[287].

2.19 Global Prevalence and Secular Trends in Obesity

Only limited obesity prevalence data are available for South East Asian countries. Various studies assessing nutritional status have been carried out. Particularly in

India, but these have generally focused on undernutrition, have been for selected population groups, and have not used the WHO classification of obesity. As many countries in South-East Asia are presently undergoing the so called "nutrition transition", there special need to collect good quality, nationally representative obesity prevalence data. The nutrition transition is associated with a shift in the structure of the diet, reduced physical activity and rapid increases in the prevalence of obesity,[289] the lack of consistency and agreement between different studies over the classification of obesity in children and adolescents has already been acknowledged. For this reason, it is not yet possible to give an overview of the global prevalence of obesity for these younger age groups.

Nevertheless, whatever method is used to classify obesity, studies investigating obesity during childhood and adolescence have generally reported a high prevalence of obesity, and that rate are on the increase. In the USA, for example, the prevalence of overweight (defined by the 85th percentile of weight-for-height) among 5 to 24 year olds from a biracial community of Louisiana increased approximately twofold between 1973 and 1994. Furthermore, the yearly increases in relative weight and obesity during the latter part of the study period (1983 through 1994) were approximately 50% greater than those between 1973 and 1982[290] . A similar trend has been observed in Japan; the frequency of obese schoolchildren (>120% SBW) aged 6-14 years increased from 5% to 10% and that of extremely obese (>140% SBW) children from 1% to 2% during the 20 years (1974 to 1993). The increase was most prominent in male students at ages of 9-11 years. Early obesity leads to an increased likelihood of obesity in later life, as well as an increased prevalence of obesity-related disorders. In the Japanese study, approximately one third of obese children grew into obese adults[291]. Childhood obesity is not only confined to the industrialised countries,

as high rates are already evident in some developing countries. The prevalence of obesity among school children aged 6-12 years in Thailand, as diagnosed by weight-for-height > 120% of the Bangkok reference, rose from 12.2% in 1991 to 15.6% in 1993, [292] and in a recent study of 6 to 18 year old male school children in Saudi Arabia, the prevalence of obesity was found to be 15.8% [293].

The only integrated data currently available that give an overview of the global prevalence of obesity during childhood are those compiled by the WHO program of Nutrition [294,295]. In the WHO analysis, children were classified as obese when they exceeded the NCHS median weight-for-height plus two standard deviations or Z-scores. The reported prevalence of obese children for the age group 0-4.99 years is listed. It should be noted that some children classified as obese under this system might actually have a higher relative weight due to stunting rather than as result of excess adiposity. This is of particular significance in developing countries undergone the nutrition transition, where a higher risk of obesity in stunted children has been described[296].

2.20 Environmental Factor and CHD Study

Barker and his colleagues had focused cardiorespiratory disease epidemiology on the potential origins of adulthood disease risk in processes occurring during early development.[297-299] Lower Birth weight and (among men) poorer growth during the first year of life have been associated with increased coronary heart disease (CHD) mortality over a 65-year follow-up study. Those findings encourage the investigation of the developmental origins of cardiorespiratory disease risk factors, including blood pressure, blood lipids, haemostatic factors, respiratory function, fat distribution and propensity to insulin dependent diabetes. The repeated demonstration that CVD risk

factors such as blood pressure and cholesterol track through childhood into adulthood,[300] these data make a strong prima-facie case that the origins of cardiorespiratory disease should be sought in early life. However there have been few attempts to replicate the basic finding that lower birthweight is associated with increased risk of major coronary morbidity and mortality and both positive and negative findings have been reported in these.[301-303]

The Caerphilly Study is remarkable in this respect, as, in addition to evidence on birth weight and later fatal and non-fatal CHD, prospective data concerning biological and socio-economic risk factors are available for the period prior to the determination of coronary events. They report the first study that considers birth weight, measures of CHD risk factors in adulthood and subsequent CHD.[303]

Report from Health of the Nation[304] advice is to reduce the proportion of men and women aged 16-64 who are obese by at least 25 and 33% by the year 2005'. Reduction in coronary heart disease and stroke was identified as a key area, and obesity was considered as an indirect risk factor, contributing to both raised plasma cholesterol and raised blood pressure.[304] Hence knowledge of trends in weight and prevalence of obesity may help to show whether the target is achievable, to predict future incidence of cardiovascular disease in Britain, and suggest target groups for preventive measures. There have been two reports of recent increases in body mass index in adults. A group of scientist (Gulliford, Rona and Chinn) reported that, trend in young women in England and Scotland from 1973 to 1988, which was not explained by changes in parity. The increase in young men was half that in young women. A comparison of data from the dietary and nutritional survey of British

adults, collected in 1986 and 1987, With that of the height and weight survey of 1980 also showed greater trends in body mass index for women than for men.[305]

Information for children is less recent. Colley[306] compared triceps skin fold thickness in children in 1971 aged between 6 and 14 years with that of children measured in 1959 and found increases in older girls. Peckham et al.,[307] using data from the first two British cohort studies, reported increases in the prevalence of overweight and obesity in 7- year- old children from 1953 to 1965 and in the same children at age 11, i.e. from 1957 to 1969. In the 1970s there was an increase in weight in children aged 5 to 11 years,[308] but slightly less for English children than expected from the increase in height over the same period. Scottish children showed a much greater increase in weight, and an increase in Weight-for-height, so that by 1980 the weight-for-height of Scottish children appeared slightly greater than that for English children. Triceps skin fold thickness also showed greater increases in Scottish children than in English children, but trends for the latter were positive and statistically significant. From this paper they estimate trends in weight-for-height and triceps skin fold thickness, for English and Scottish boys and girls, and compare them with earlier trends. Trends in the two components of weight-of-height were examined, in order to provide a complex picture of the recent changes in growth. Some possible explanations for trends in weight-for-height and skin folds in children, in particular the trends in parental weight-for-height and decreases in family size, were examined, and differences in trends between social groups are considered.[309-313]

Regular physical exercise has a beneficial effect on health and well being. Epidemiological studies confirm the inverse relation between physical activity and the incidence of coronary heart disease (CHD).[314-323] Furthermore, myocardial infarction

not only occurs more often among sedentary people than among active people but also is more often fatal and occurs at lower age.[324-325] Although the epidemiological evidence has been labelled as circumstantial,[317] the statistical consistency of these studies was supported by laboratory results[326-339] and provides a strong argument that regular, vigorous physical activity plays a role in the prevention of myocardial infarction.

The effects of physical training include reduced levels of total cholesterol and low-density lipoprotein cholesterol,[327] increased levels of high-density lipoprotein cholesterol,[328,329] increased insulin receptor density,[330,331] increased glucose clearance,[332] reduced blood pressure[333-335] and increased fibrinolytic activity of plasma.[336-338] Other studies which shown an inverse relation between fitness level and the incidence of CHD as well as the number of risk factors for CHD.[319,333,339] The investigators typically used relatively simple methods to assess fitness and most employed limited samples or population groups, which restricts of their findings.

They have a number of years been involved in the fitness testing and health profiles of federal public servants from across Canada attending management courses in the Ottawa area.[340] Although involvement in the fitness testing session is voluntary, the participation rate for these on-site sessions is usually greater than 90%. The fitness module includes assessment of health status and physical fitness (including cardio respiratory fitness, as assessed with the Canadian Aerobic Fitness Test [CAFT])[341].

Coronary heart disease, a leading cause of morbidity and mortality among adults in the United States, should begin during childhood.[342, 343] Risk factors for coronary heart disease include an elevated low-density lipoprotein cholesterol concentration, a

reduced high-density lipoprotein cholesterol concentration, and an elevated blood pressure. Therefore, appropriate targets for prevention of coronary heart disease include lipoproteins and blood pressure.[343] These factors may be affected by physical activity, defined as "any bodily movement produced by skeletal muscles that results in energy expenditure".[344] Among adults, physical activity has been directly associated with HDL-C concentration[345] and inversely associated with blood pressure.[346] In contrast, among children, associations between physical activity and HDL-C concentration and physical activity and blood pressure are variable.[347-356]

Three cross-sectional studies conducted during late childhood[349-351] reported a direct association between activity and HDL-C concentration. Two studies reported no effect.[347,348] The results of intervention studies are highly variable. Reports have documented a direct association,[352] no association,[353] and an inverse association[354] between physical activity and HDL-C concentration.

The data from the cross-sectional, because factors associated with HDL-C concentration, such as body fatness, estradiol level or Tanner stage and insulin concentration,[357-359] were not controlled.[347-354]

Little work has been reported on the association between physical activity and LDL-C concentration during late childhood. Using a 7-day activity recall, and reported a significant association between physical activity and the HDL-C -to- LDL-C ratio but did not report on the association with LDL-C levels.[347]

Cross-sectional studies of the association between physical activity and blood pressure during late childhood have reported conflicting results.[347,355,356] Two studies among boys and girls aged 11-13 years documented an inverse association between

physical activity and systolic and diastolic blood pressures.[355,356] One reported no significant association.[347] However, these studies did not control for insulin concentration or body mass index, both of which correlate with blood pressure in children.[359,360]

2.21 Role of Trace Elements in CVD

Although high blood pressure, smoking and dyslipidemia are major risk, factors for atherosclerotic cardiovascular diseases, they cannot fully explain variation in the incidence of the diseases and the thus a search for other risk factors continues. Minerals and trace elements have long been suspected as potential risk factors for cardiovascular diseases. Serum calcium had been shown to be positively correlated with several cardiovascular risk factors[361] and survival of people with hypercalcemia is decreased particularly due to increased risk of cardiovascular death[362]. On the other hand deficiency of dietary calcium has been connected to and elevated risk of hypertension[363]. In ecological correlation studies hardness of drinking water is inversely related to cardiovascular mortality and it has been suggested that the probable protective 'water factor' is the high magnesium content of water,[364] although calcium has been implicated also.[365] Low serum magnesium is a risk factor for early death in people with acute myocardial infraction, but the impact of low serum magnesium on long-term prognosis is not well known.[366] The trace metals copper and zinc were of special interest as contributors to cardiovascular risk. According to animal experiments low copper and high zinc content in the diet leads to hypercholesterolemia and cardiovascular complications.[367] In human studies,

however, serum copper has been mostly shown to be raised in patients with cardiovascular diseases, whereas serum zinc has been normal or decreased.[368-371] There are only a few longitudinal studies on the influence of serum calcium, magnesium, copper and zinc on cardiovascular death, which have been controlled for the effect of all other major risk factors. They analysed the association of serum calcium, magnesium, copper and zinc with cardiovascular death in a large prospective population study to determine the independent prognostic value of the serum concentration of each of these elements.

The development of obesity in childhood is a major determinant of the acquisition of cardiovascular risk later in life.[372-374] Children who become more obese are more likely to have elevated blood pressure, elevated concentrations of LDL-C and TG-C, and decreased concentrations of HDL-C as adolescents and young adults. Being overweight as a child is highly predictive of being overweight as an adult.[375]

2.22 Lipoprotein and Immunization

2.22.1 Bacterial Toxins and Lipid Profile in Acute Rheumatic Fever

Cholesterol binds to streptolysin O and related bacterial toxins. In normal serum, only a fraction of the cholesterol attached to lipoprotein is available for binding, probably as a cholesterol-peptide complex formed during catabolic breakdown of the lipoprotein. Cholesterol esterase produced by certain organisms- e.g., *Staphylococcus pyogenes* and *Pseudomonas aeruginosa* augments this fraction both in vitro and in vivo. Endogenous esterase similarly increases the amount of cholesterol-peptide complex, a mechanism that may be activated as a feedback process following binding of toxin to the cholesterol component of the complex. This complex will thus supply

an easily available means of binding bacterial toxin before antibody formation begins; Cholesterol-peptide.[376] It was shown that the lower level of HDL cholesterol in Acute Rheumatic Fever (ARF) with carditis cases possess streptolysin O inhibiting characteristics.[377]

One case-control study results found in Bangladesh shown that in Acute Rheumatic Fever (ARF) with Carditis group, Total cholesterol, HDL and LDL level were significantly low but triglyceride level showed higher values in comparison with control group. It was also true among HDL/TG ratio, which is significantly low between ARF with Carditis group patient.[378]

2.22.2 Altered Lipid Profile after Kawasaki Syndrome

Delineation of lipid values in children after Kawasaki syndrome is important because of the prediction of this disease for coronary arteries. In this study they observed mean high-density lipoprotein cholesterol after clinical recovery tended to be lower in patients with persistent coronary abnormalities than in those without such lesions. Kawasaki syndrome is associated with important abnormalities in lipid metabolism, which is now a cardiovascular problem in some develop country like Japan.[379]

2.23 Food Consumption Habits in Bangladesh

In Bangladesh the basic diet, in all locations consists of rice supplemented by curries. Curries may contain vegetables, fish, meats, unripe fruits or starchy roots, in varying proportions. Pulses, in the form of **dhal** (pulses), a thickened soupy preparation, are also eaten with rice. Thus all foods are considered by the people as supplements to rice. (This is true even for potatoes, which in many parts of the world are generally used as a substitute for cereals, rather than a supplement). The only food taken as a

substitute for rice is wheat, primarily when rice is not available. Seasonally, significant amount of fruits is consumed in summer (May- July). In other seasons fruit consumption is insignificant and only occasional. The basic dietary pattern is rice plus " trimmings", and data would suggest that ideally, in the people's attitudes, rice should constitute the bulk of total intake. Because of high-parboiled rice content of the diet, thiamine and niacin are present in adequate amounts. [380]

Eighty percent of urban poor children are chronically malnourished and 15 percent are acutely malnourished among the urban poor. Extreme poverty, low purchasing power, a perpetual shortage of food and a lack of knowledge about nutrition are the main causes of malnutrition. The children of women who are work outside the home are the most malnourished, as their mothers have little time to looked after them. The calorie intake of the urban poor is even lower than that of the rural poor because the urban poor purchase commercially produced foods, they have difficulty in adjusting to the urban food market and working mothers stop breast-feeding their babies early. It is also noted that urban working conditions for women do not allow them to give proper care to their children.[381]

The average calorie intake was found to be 1975 kcal per person per day in 1981-82 as against 2029 kcal in 1975-76. A foods intake has gone down by about 3% over the two study periods. Average protein intake was about 49gm per person/day for all age and sex groups **(Annex-4, Table- 2.231a, 1b, and 1c).**

The diet of Bangladesh, while food habits vary from region to region and even at the level of individual households, the cooking process generally leads to significant loss of nutrients. Rice is the major food item in our diet. However minerals and vitamins (especially water soluble vitamins B and C) are lost (40% of thiamine and niacin) even during the washing of rice, before the actual cooking (Gopalan 1993).

Bangladesh is a deltaic region with many rivers. Historically rice and fish were abundant. Great expanses of fallow land also enabled people to rear hvestock. Milk and milk products were also available in large quantities. Bangladesh used to export food items to other regions in south Asia till the end of British rule. Evan after that Bangladesh used to export-surplus country for some food items for about two decades. The growth of population, the declaration in production, the increase in inequality, have however led to the current situation of declining in production, the increase in inequality, have however led to the current situation of declining per capita availability of food and increasing inequality in income and consumption. This country has not been able to produce enough food grains for meeting the needs of the people for many decades.[380]

The above cited literature suggest that dietary consumption of saturated fatty acid is 41%(normal range 25%-30%) which is a potential cause of high blood hpids situation exist in Bangladesh .So, what is happening in child is needed to know an urgent basis and for that matter present study is timely designed and conducted. Only 21% rural and about 40% urban people were able to meet their requirement of fat. However, intake of fat consumption increased as compare to 1975-76, 1981-82 surveys.[382] The above cited literature suggest that dietary consumption of saturated fatty acid is 41% which is a potential cause of high blood lipids situation exist in Bangladesh. So, to know urgent basis and from that matter present study is timely designed and conducted .

CHAPTER – 3

MATERIALS AND METHODS

Chapter-3

Study Design and Methodology

3.1 Study Design:

The study was conducted in 392 students drawn randomly from 3 schools of Dhaka city by a predetermined criterion. Age determination: By checking birth record from school register books. Objectives were explained from ensuring co- operation both from school and parents.

This cross-sectional study was conducted between July 1996 to June 1997 in a group of school children.

3.2 Sample Size Estimation

The one-sample problem [383]

Estimating the population proportion

P devotes the true but unknown proportion in the population. The sampling distribution [a] of the sample proportion "P" is approximately normal with mean: [b] E (P) = P, and variance: [c] Var (P) = P(1- P) /n. The sampling distribution may be represented as in (Fig -1).

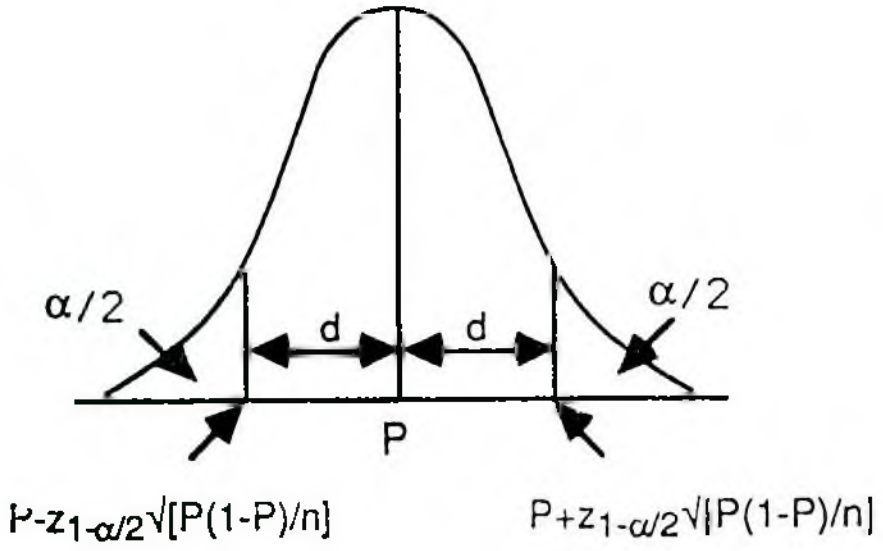

Fig-1 Sampling distribution of the sample proportion

The quantity d denotes the distance, in either direction, from the population proportion and may be expressed as

$$d = Z_1 - \alpha / 2 \sqrt{[P(1-P)/n]}$$

Since there is no such study on lipid profile among children aged 10 –12 years, we are considering that the proportion of hyperlipidaemia among the study children is ≥ 50% to attain maximum sample size . That is P=0.5

We also consider the level of significance 5% at 95% confidence level and then the value of d will be 0.05. (P X allowable error i.e. 0.5 X 0.1 = 0.05). Using the standard sample size estimation formula

$$n = \frac{P(1-P)z^2}{d^2}$$
$$= 306$$

To cover the design effect, 25% more samples were taken and as such the final sample in our study was 392.

3.3 Study Population

Inclusion Criteria

Apparently healthy school children (boys and girls) aged between 10-12 years of both sex and socio-economic status.

Exclusion Criteria

Children suffering from Diabetes mellitus, hypothyroid, chronic pancreatitis, and nephrotic syndrome were not enrolled.

3.4. Sample Selection

The study children were selected by multistage sampling:

a) At the first stage, a list of the kindergarten or primary schools from the three pre- selected areas was prepared and then one school from each area was selected by simple random sampling. These were:

   i) Kindergarten schools of Dhanmondi area representing population with higher socio-economic status.

   ii) Primary school of Mohammadpur area to represent a population with middle and mixed socio-economic status

   iii) Primary school of Rayer Bazar to represent population with Low Socio- Economic population.

   b) Finally, the sample from each of the selected school at random was proportional to size of school children.

3.5. Sample Size

Following the standard sample size formula a total of 392 children from three different schools were enrolled into the study.

3.6. Data Collection

3.6.1. Variables

a) Independent Variables

Age, Sex, Religion, SES (family size, parental education and their occupation, family income, and housing condition), nutritional status (weight, height, mid arm circumference, triceps skin fold thickness, waist-hip ratio). Daily food intake and Average daily physical activity.

b) Dependant Variables

Serum total cholesterol, serum LDL-C, serum HDL-C, and serum TG-C.

Data Collection Instrument:

a) Structured questionnaire

The questionnaire was a closed type format, which included three sections:

Section I: Personal history with clinical examination

Section II: socio-economic status

Section III: Anthropometric measurements

b) A 7-day food intake check list

c) Laboratory reports form.

The data collection instruments were field tested prior to collection of data at the field level.

3.7. Data Collection Procedure

Prior to data collection the school authorities and parents were briefed about the purpose of the study and informed consent was obtained from the parents. The interviews took place at school. To collect the information on dietary intake, the checklist was provided to the mothers 7 days before the interview who put necessary check marks and submitted the list at the time of interview. The procedure of filling the form was explained to them. If the subject's mother was illiterate then a literate person of the family or neighbourhood completed the form. Mothers were requested to visit the National Centre for Control of Rheumatic fever and Heart Diseases (NCCRFHD) with their children for blood collection, anthropometric measurements and relevant data collection. Estimation of lipid profile was done by spectrophotometric enzymatic method. The results of the serum lipid status of the children were reported to their parents and also supportive measure given on the basis of the result if required.

3.8 Skin Folds Thickness Measurements

3.8.1 The Caliper is so made that a constant pressure of 10 gm/sq.mm of face is exerted at all openings. The instrument was tested and found to be 940gms.

3.8.2 Each small division on the dial was 0.2 mm, but the instrument conveniently read to 0.1 mm interpolation between the markings.

3.8.3 A fold of skin and subcutaneous tissue was picked up between the thumb and forefinger of the left hand and pinched clean away from the underlying muscle. The calipers are applied to the fold a little below the fingers so that the pressure on the fold point measured was exerted by the caliper faces and not by the fingers.

3.8.4 Skinfolds was measured only at sites where a proper fold can be raised. The two most commonly used folds are:

(a) triceps, half-way down left arm between tip of acromion and top of the radius, with the fold picked up in a line passing directly up the arm from the tip of the olecranan process. The arm was hanged relaxed at the side.

(b) Subclavicular, just below the angle of left scapular , the fold being picked up parallel to the natural cleavage line of the skin.

3.8.5 The values with the skinfold caliper gave a markedly non-normal frequency distribution it was most purposes to transform them into a log scale before Use.[384]

3.8.6 The accuracy of measurements of the triceps and subscapular skinfolds was trained individual should duplicate his readings to within ± 5% in two- thirds or more of all repeated measurments.[385]

3.9 Cholesterol Determination Method [386]

3.9.1 Collection of Sample

About 5 ml of venous blood was collected in a dry, test tube from each child.

3.9.2 Preparation and Preservation of Sample

The blood was kept for 1-2 hours at room temperature to allow clot formation. Then it was centrifuged at 4000 rmp for at least 5 minutes. After separation of serum it was preserved in deep freeze at -20^{0} C till the tests were done.

3.9.3 Laboratory Procedure

Sera were tested for the determination of total cholesterol, LDL-C, HDL-C and TG-C by colorimetric method. All the tests were performed at the Clinical Biochemistry Laboratory of the National Centre for Control of Rheumatic fever and Heart Disease (NCCRFHD), Sher e Bangla Nagar, Dhaka, Bangladesh. The tests were performed with commercially available kits **(Boehringer Mannheim testkit, Germany)** and the manufacturer instructions were strictly followed in the performance and interpretation of the tests.

3.10 Test Principle[387]

Total Cholesterol :

$$\text{Cholesterol esters} + H_2O \xrightarrow{\text{cholesterol esterase}} \text{cholesterol} + \text{RCOOH}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{cholesterol oxidise}} \Delta^4 \text{- cholestenone} + H_2O_2$$

$$2H_2O_2 + 4\text{-aminophenazone} + \text{phenol} \xrightarrow{\text{POD}} 4\text{-(}p\text{ benzoquinone-monoimino)- phenazone} + 4\,H_2O$$

HDL-C [388]

By adding phosphotungstic acid and magnesium ions to the sample, precipitates LDL (low-density lipoproteins)and VLDL (very low density lipoproteins). are precipitated Centrifugation leaves only the HDL (high-density lipoproteins) in the supernatant and their cholesterol content is determined enzymatically.

TG-C [389]

$$\text{Triglycerides} + 3\,H_2O \xrightarrow{\text{Lipase}} \text{glycerol} + 3\,\text{RCOOH}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{GK}} \text{glycerol-3-phosphate} + \text{ADP}$$

$$\text{glycerol-3-phosphate} + O_2 \xrightarrow{\text{GPO}} \text{dihydroxyacetone phosphate} + H_2O_2$$

$$H_2O_2 + \text{4-aminophenazone} + \text{4-chlorophenol} \xrightarrow{\text{peroxides}} \text{4-(}p\text{-benzoquinone-mono-imino)-phenazone} + 2\,H_2O + HCl$$

3.11. Materials / Reagents used

(a) Serum at -20^0 C and reagents used ---->(for total cholesterol)

Contents	**MPR 1**	**MPR 2**	**MPR 3**	Preparation and stability of the reagent solution
Bottle 1 Cholesterol reagent	10	10	4	MPR 1: Dissolve contents of one bottle 1 by filling to the mark with redist. water.(about 32 ml) MPR 2: Dissolve contents of one bottle 1 by adding 100-ml redist. water. MPR 3: Dissolve contents of one bottle 1 by adding 500-ml redist. Water. The reagent solution was ready to use after 10 minutes. Stable for four weeks at + 2 to 8^0 C Seven days at + 20 to 25 ^{0}C .

HDL cholesterol precipitant was ready to use. Stable up to the expiry date specified when stored at + 15 to 25 ^{0} C .

3.12 Test Procedure

1) The contents of the kit were brought to room temperature. Sera were taken out of the deep freeze and allowed to reach the ambient temperature.
2) 0.02 ml of sera (sample) and 2.00ml of reagent solution was pipetted into test tubes and mixed well. Another test tube marked 'reagent blank'(RB) contained
3) only 2.0 ml of the reagent solution. Both the test sample and RB were incubated at 37^0 C for 5 minutes.
4) The absorbance of each sample against RB was read within 1 hour = A sample at Hg 546 nm wavelength in an Autoanalyzar Photometer 5010(Boehringer, Germany).

(b) Sample and reagent preparation and test procedure for HDL-C

Assay procedure

Determination of cholesterol using the MPR 1, MPR 2 cholesterol kit, CHOD-PAP method

Wavelength: Hg 546 nm

Spectrophotometer: 500nm

Cuvette: 1cm light path

Incubation temperature: 37^0C

Measure against reagent blank (RB).

One reagent blank for each assay series.

Pipette into test tubes:	RB	Sample
Redistilled Water	100µl	-
Supernatant	-	100µl
Reagent solution	1000µl	100µl
Mix, and incubate RB and sample for 10 min at 37^0C for 5 min, then measure absorbance of sample (A_{sample}) against RB within 1 hour.		

(c) Sample and reagent preparation and test procedure for LDL-C

(d) Sample and reagent preparation and test procedure for TG-C

Sample preparation

The sample stored upto 4^0C

Procedure:

Wavelength: Hg 546 nm

Spectrophotometer 500nm

Cuvette: 1cm light path

Incubation temperature 37^0C

Measure against reagent blank(RB).

One reagent blank for each assay series.

Pipette into test tubes :	
Serum	0.02ml
Reagent solution	2.00ml
Mix , and incubate at 37°C for 10 min. Read absorbance of sample against reagent blank within 69 min= A_{sample}	

3.13. Calculation

(a) The concentration (c) of Cholesterol in the sample

Wave length	c (mg / 100 mL)
Hg 546 nm	1040 x A_{sample}

(b) The concentration of HDL cholesterol in sample -

Wave length	mg/dL	mmol/L
Hg 546nm	c = 853 x A_{sample}	c= 22.1 x A_{sample}

(c) The concentration of LDL cholesterol in the serum -

LDL cholesterol = total cholesterol – $\frac{\text{triglycerides}}{5}$ - HDL cholesterol

(d) The concentration (c) of triglycerides cholesterol (TG) in serum –

Wave length	mg/dL	mmol/L
Hg 546 nm	325.1 x A_{sample}	8.41 x A_{sample}

3.14 Cutoff Point for Hypercholesterolaemia

The cutoff point for hypercholesterolaemia was 2-20 years children was 205 mg/dL for TC-C, 37mg/dL for HDL-C, 145 mg/dL LDL-C and 130mg/dL Triglyceride cholesterol . In this study we followed the above references value for our study. [390]

3.15 Data Analysis

The principal investigators checked the questionnaires and checklists. Data were edited as they were collected and entered into the computer by the co-investigators. Fox pro for data entry and editing Micro diet software system, University of Salford 1983, 1988 mark 7.5 for dietary conversion and SPSS, EPI Info for Statistical analysis. Data were summarised and both the groups were compared.

The significance of differences was evaluated by the chi-square test when the

outcome variable was a categorical one and the differences in mean serum lipid levels among groups were evaluated by student's t-test.

4.16. Micro Diet Software System

"Microdiet" is a DOS based nutrient analysis software package for use by dietitians. The first version was produced fifteen years ago for use on a BBC computer. The first diets to be analysed by "Microdiet" were those of children with food allergies who were eating very few foods. The analysis was necessary each week in order to check that there were no nutrient deficiencies in these very limited diets. Using "Micro diet", dieticians were able to reduce the time taken to analyse one Childs diet from two hours down to ten minutes. Later applications included nutritional monitoring of infants with acute failure to thrive in order to identify simple foods that would meet nutrient deficiencies when incorporated into the Childs existing diet. It was from this application that the idea of developing a linear programming extension of "Micro diet" arose. Recent versions of the software are in use in most health districts in the country. The software is now used for analyses of food intakes of patients with all diet related illnesses. Even though "Micro diet" is still widely used, it is now somewhat old fashioned and will be replaced later this year with a Windows based system.[391]

CHAPTER – 4

RESULTS

Chapter-4

Results

Section -I

Socio-Economic Status

I.1 Age and Sex

Of the 392 school children were selected from three different schools, 61% were male and 39% were female. This children were from 392 families and aged 10-12 years were enrolled into the study. Of them 24%, 45%, and 31% were 10, 11, and 12 years old respectively. Children of 11 years old comprised the largest 45% (Table-I.1). Ninety three percent were Muslims. However, Hindus and Christians comprised 6% and 1% respectively (table not shown).

Table I.I. : Distribution of Age and Sex among study Children (N=392).

Age (Year)	Sex				Total	
	Boys		Girls		n	(%)
	n	(%)	n	(%)		
10	43	(10.9)	51	(13.0)	94	(24)
11	106	(27.0)	72	(18.3)	178	(45)
12	92	(23.4)	28	(7.1)	120	(31)
Total	**241**	**61**	**151**	**39**	**392**	**100**

I.2. Family Size

Of the study children,only 9% members of the family were less than 5 years old (Table not shown).

I.3 Floor Structure

With regard to floor structure, a good proxy indicator of school status, 74% had cemented floor, rest 26% children had non-cemented floor structure indicating that they came from poor underprivileged community (Table not shown).

I.4 Educational Status

Educational qualification of the fathers revealed that 9% children had father with S.S.C. education, and another 7% had fathers with H.S.C. education. However, 21% and 20% children came from families with fathers having graduate and postgraduate education respectively. However,38% were illiterate. 9%, 9%, 12%, and 11% mothers had S.S.C, H.S.C, graduate, and post graduate level of education respectively. However, 38% of them were illiterate discussed in Table-I.4.

Table I.4 : Parental educational status among the study Children.

Qualification	*Father* (n=374)		*Mother* (n=311)	
	Number	Percentage	Number	Percentage
No education	147	39.4	147	47.2
S.S.C	37	9.8	37	12.0
S.S.C-H.S.C	28	7.5	36	11.5
Graduate	82	21.9	47	15.2
Post Graduate	80	21.1	44	14.1
Overall	**374**	**100**	**311**	**100**

I.5. Monthly Family Income

The distribution of monthly family income of the one third study children was income of Tk. 1000-3000. Another one third study children came from middle or high socio-economic background (monthly family income upto Taka 10000 or more) and 34.4% families had income more than taka ten thousand .

Table I.5 : Monthly Family income in taka of the study population (n=392)

Income(Taka)	*Number*	*Percentage*
1000 – 3000	143	36.5
3001 – 10000	114	29.1
10001 & above	135	34.4

I.6. Job Status

Forty-nine percent of parents were in service, 38% had their business, and another 13% had other employment presented in table-I.6.

Table I.6: Job status among parents (n=392)

Job	*Number*	*Percentage*
Service	194	49.5
Business	148	37.8
Other works	50	12.7

I.7. School status

In the sample 31.4% students were from higher status school, 40.6% came from middle status schools, and 28.0% respondents were from schools considered as mixed and low socio-economic status described in Table-I.7.

Table I.7: Percentage of Study Population among three school (n=392)

School status	*Number*	*Percentage*
Higher	123	31.4
Middle	159	40.6
Lower	110	28.0

I.8. Disease Pattern of the Parents

Only 4% fathers' of study children were hypertensive. The prevalence of IHD among them was only in 3% fathers'. As opposed to the fathers', 3% and 2% mothers' had hypertension and IHD respectively (Table-I.8). But in the study children we found only one boys and one girl from higher status has been suffering from hyperlipaedimia (not shown in the table).

Table I.8: History of cardiovascular disease in parents (n=392)

Disease	**Father**		**Mother**	
	Number	Percentage	Number	Percentage
Hypertension	14	3.57	11	2.89
IHD(chest pain)	10	2.55	06	1.53

Section-II

Anthropometrics Measurments

II.1. Gender specific relationships between nutritional parameters.

The mean height of the boys 139.34 cm as opposed to 135.94 cm of girls and mean weight boys was 32.64 kg while girls had a mean of 32.02 kg. MAC of boys was 18.62 cm as opposed to 18.26 cm of girls. In Higher status school the mean was 20.06 cm for boys and 19.64 cm for girls. Among students of middle status schools, mean MAC was 18.80 cm for boys against 17.72 cm of girls and in mixed and lower status schools boys had a mean of 17.43 cm against the 18.16cm of girls (data not shown). Mean Tricep skin fold thickness of boys was 8.34 mm as opposed to 8.47 mm of girls respectively discussed in Table I. However, in schools of students from higher status, the mean value was 11.30 mm and 13.43 mm in boys and girls. For middle status schools the corresponding values were 9.58 mm and 10.53 mm in boys and girls respectively. For boys of low socio-economic status schools the mean skin fold thickness was relatively low for both boys and girls (4.91mm vs. 6.65mm) compared to other type of schools (data not shown). Overall, mean west-hip ratio in boys was 0.84 as opposed to 0.83 in girls shown (Table-II.1). In higher status schools, mean waist-hip ratio was 0.84 in boys and 0.82 in girls. In the middle status group of schools, waist-hip ratios were 0.86 and 0.83 in boys and girls respectively. Similar observations were made in the mixed and lower status schools (data not presented).

Table II.1: Gender specific relationships between nutritional parameters among study children

Sex	Height (cm) Mean ± SD (n=392)	Weight (kg) Mean ± SD (n=392)	MAC (cm) Mean ± SD (n=392)	Tricep skin(mm) Mean ± SD (n=392)	WHR Mean± SD (n=387)
Boys	139.34± 22.78	32.64 ±8.55	18.62 ± 3.64	8.34 ± 4.32	0.84± 6.7
Girls	135.94± 25.23	32.02 ± 8.44	8.26 ±4.38	8.47 ± 5.05	0.83±0.12

II.2 Relationships of abnormal Total cholesterol with Nutritional parameters of by school.

In table-II.2 Relationships of Total cholesterol >205mg/dL study children had mean and SD of higher school status children height, weight, MAC, Tricep skin fold thickness and Waist hip ratio (WHR) were 145.64±14.49, 47.82± 9.46 , 23.45±1.86, 16.59± 3.12 and .83 ± .17 respectively. In middle school status children were 143.67± 7.77 , 32.33± 3.51, 19.0±1.00, 5.8± 5.2 and .85± 7.47 respectively. Anthropometric measurment among lower school status groups were 134 ± 3.84 , 25.20± 3.16 , 16.90±1.66, 5.15± 1.20 and .83± 3.90 . In this table we found weight were more among higher school status (47.82± 9.46 Vs 19.0±1.00 and 16.90±1.66)than mixed and underprivileged (lower) groups. Triceps skin thickness were more among higher school status than other two groups (16.59± 3.12 Vs 5.8± 5.2 and 5.15± 1.20).

Table II.2 : Relationships between normal and abnormal total cholesterol (205mg/dL) with nutritional parameters of by school (Mean ± SD)

School status		Height Mean ± SD	Weight Mean ± SD	MAC Mean ± SD	Triceps Mean ± SD	WHR Mean ± SD
Higher	Normal (n=123)	147.67±18.94	38.82 ± 7.95	19.77± 4.70	11.35±4.74	0.82 ±0.12
	Abnormal (n=11)	145.64±14.49	47.82± 9.46	23.45±1.86	16.59± 3.12	0.83 ±0.17
Middle	Normal (n=114)	135.64± 21.60	31.42±7.34	18.17±3.73	9.06±4.10	0.84± 6.33
	Abnormal (n=3)	143.67± 7.77	32.33± 3.51	19.0±1.00	5.8± 5.2	0.85± 7.47
Lower	Normal (n=70)	136.09 ± 18.49	27.90±7.13	17.72 ± 2.58	5.50±2.10	0.82± 9.80
	Abnormal (n=10)	134 ± 3.84	25.20± 3.16	16.90±1.66	5.15± 1.20	0.83± 3.90

II.3 Relationships of normal and abnormal HDL-C with Nutritional parameters of by school.

In table-II.3 discribed that weight and MAC were significantly higher among higher school status children who were sufferings from low level of HDL-C. WHR among higher school status also higher trend than mixed and lower school status children.

II.4 Relationships of abnormal LDL-C with Nutritional parameters of by school

Among abnormal LDL-C study children MAC and Triceps among higher school status children were 23.10±2.13 and 15.45±3.30 which is more than middle and lower school status children discribed in Table-II.4.It was also true that weight of higher school was higher than two other groups of children.

Table II.3: Relationships between normal and abnormal HDL-C(<37mg/L) with anthropometry.

School statns		Height(cm) Mean ± SD	Weight(kg) Mean ± SD	MUAC(mm) Mean ± SD	Triceps(mm) Mean ± SD	WHR Mean ± SD
Higher	Normal (n=58)	149.96± 17.50	42.57±6.95	20.00 ± 5.44	11.97±5.49	0.81±0.13
	Abnormal (n=7)	152.86± 9.87	45.43±11.96	22.71 ± 22.0	10.00±1.0	0.87±8.15
Middle	Normal (n=39)	132.38± 28.23	30.32±7.52	16.90 ± 4.51	8.47 ±4.36	0.83 ± 8.01
	Abnormal (n=2)	131.50±7.78	27.00±4.24	17.50±2.12	11.25±1.7	0.79±6.45
Lower	Normal (n=37)	140.67 ± 4.51	29.67± 0.58	18.66± 0.57	6.66 ± 2.30	0.91± 4.03
	Abnormal (n=34)	132.32±24.46	26.91±1.80	17.36±1.80	5.17±1.65	0.83±4.86

Table II.4: Relationship of LDL-C(>145mg/dl) with anthropometry.

School status		Height(cm) Mean ± SD	Weight(kg) Mean ± SD	MAC(mm) Mean ± SD	Triceps(mm) Mean ± SD	WHR Mean ± SD
Higher	Normal (123)	147.67±18.94	38.82 ± 7.95	19.77 ± 4.70	11.35 ± 4.70	0.82 ± 0.12
	Abnormal (n=10)	146.40±15.44	46.60 ± 10.71	23.10±2.13	15.45 ± 3.30	0.82 ± 0.18
Middle	Normal (114)	135.64 ± 21.60	31.42 ± 7.34	18.17 ± 3.73	9.06 ± 4.10	0.84 ± 6.33
	Abnormal (n=2)	148.00±2.83	34.00±2.83	18.50± .7	3.7 ± 5.3	0.82 ± 8.03
Lower	Normal (70)	136..09 ± 18.94	27.90 ± 7.13	17.72 ± 2.58	5.50 ± 2.10	0.82 ± 9.80
	Abnormal (n=14)	148.00±4.99	24.86 ± 3.76	16.57 ± 1.65	4.9 ± 1.08	0.83 ± 4.0

II.5. Relationships of abnormal TG-C with Nutritional parameters of children by school status.

Height, weight and MAC were 151.06±7.43, 45.35±8.99 and 22.41± 3.58 among Higher school status children. In mixed groups were 138.00±8.89, 30.40±4.16 and 18.20±1.6. And underprivileged (lower) groups were 136.57±6.62, 27.36±3.34 and 17.64±1.4 respectively. But Triceps skin thickness were significantly more among higher school status children (14.20±5.6 VS 9.7±2.8 and 5.5±1.6) than mixed and lower school status children shown in Table-II.5.

Table -II.5: Relationships of TG-C(>130mg/dl) with Nutritional parameters of Children by school.

School status		Height(cm) Mean ± SD	Weight(kg) Mean ± SD	MAC(mm) Mean ± SD	Triceps(mm) Mean ± SD	WHR Mean ± SD
Higher	Normal (41)	150.00 ± 19.40	41.90 ± 6.87	19.46 ± 5.57	11.64 ± 5.19	0.80 ± .14
	Abnormal (n=17)	151.06±7.43	45.35±8.99	22.41± 3.58	14.20 ± 5.6	0.86±6.17
Middle	Normal (34)	131.50 ± 29.26	30.12 ± 7.80	16.75 ± 4.67	8.45 ± 4.48	0.83 ± 8.33
	Abnormal (n=5)	138.00±8.89	30.40+4.16	18.20±1.6	9.7±2.8	0.80 ± 3.90
Lower	Normal (23)	130.83 ± 27.00	27.00 ± 6.01	17.37 ± 1.94	5.13 ± 1.76	0.83 ± 4.95
	Abnormal (n=14)	136.57±6.62	27.36±3.34	17.64±1.4	5.5±1.6	0.85±5.47

II.6. **Abnormal total cholesterol (> 205mg/dl) and weight for age median (WAM).**

In Higher school upto 84 weight for age, there were none and only 84.6% shown ≥ 84 Weight for age median (WAM). But in middle and lower school status groups we found 18.2% vs 7.7% and 81.8% vs 7.7% of WAM shown in Table-II.6.

Table - II.6: Abnormal Total cholesterol (TC-C) WAM among study children (n =24)

School status	WAM		Total
	Upto 84	> = 84	N(%)
Higher	0.0	84.6%	11(45.8)
Middle	18.2%	7.7%	03(12.5)
Lower	81.8%	7.7%	10(41.7)
Total	**100.0%**	**100.0%**	**24(100)**

II.7. **Abnormal High density lipoprotein(<37mg/dL) and WAM among children.**

In lower status school children who were suffering from abnormal HDL-C were among below WAM level and 50% among higher status group above WAM level. It was also found above WAM level abnormal HDL-C among children in lower status group were also 50%. So, In this study majority suffering from abnormal HDL-C were among lower status group children. (shown in table II.7).

Table II.7 : Abnormal High density lipoprotein (HDL-C) WAM among study children (n =43).

School status	WAM		Total No (%)
	Upto 84	≥ 84	
Higher	0.0	50.0%	7(16.3%)
Middle	6.9%	0.0	2 (4.7%)
Lower	93.1%	50.0%	34(79.1%)
Total	**100.0%**	**100.0%**	**43(100.0)**

II.8. Abnormal Low density lipoprotein WAM among children.

Among lower status school group it was found that significant number of children suffering from different grade of malnutrition 92.9% below weight for height (median) value. But among higher status children who are suffering from abnormal LDL-C were above WAM value (Shown in table-II.8).

Table II.8: Abnormal Low density lipoprotein (LDL-C) WAM among study children (n =26).

School status	WAM %		Total N(%)
	Upto 84	≥ 84	
Higher	0.0	83.3%	10 (38.5)
Middle	7.1%	8.3%	02 (7.7)
Lower	92.9%	8.3%	14 (53.8)
Total	**100.0**	**100.0**	**26 (100)**

II.9. Abnormal Triglyceride cholesterol and WAM

In Table-II.9 discussed that, Abnormal Triglyceride cholesterol among higher status children were more suffering from above level of weight for age median. But lower

status school children were suffering from below level of WAM which is very much conflicting in our study. It might be both groups suffering from over nutrition and under nutrition in different grade.

Table II.9 : Abnormal Triglyceride cholesterol(TG-C) WAM among study children(n =36).

School status	WAM		Total N(%)
	Upto 84	≥ 84	
Higher	0.0	89.5%	17(47.2)
Middle	23.5%	5.3%	5(13.9)
Lower	76.5%	5.3%	14(38.9)
Total	**100.0**	**100.0**	**36(100)**

II.10. **Height for age (Z-score) among total cholesterol (TC-C) subject.**

In Table- II.10 the height for age Z-scores shows that in higher school status 1(9.1%) children found Z score < - 2 SD or below among abnormal group but it was not true among middle and lower school status children who were suffering from abnormal total cholesterol. In case of moderate malnutrition it was found that Z score < -2 SD were only 1(10.0%) among lower school status children among abnormal group but it is not true among other two groups in our study.

Table–II.10: Distribution of Total cholesterol-C among the subject by their height for age Z-score.

School status	Height for age (Z-score)		Overall N(%)
	Stunting (< - 2.00 SD)	Normal (≥ - 2.00 SD)	
Higher			
Normal	2(1.8)	109(98.2)	111(100)
Abnormal	1(9.1)	10(90.9)	11(100)
Middle			
Normal	16(14.5)	94(85.5)	110(100)
Abnormal	0 (0.0)	3(100.0)	3(100)
Lower			
Normal	5(8.7)	54(91.3)	59(100)
Abnormal	1(10.0)	9(90.0)	10(100)

II.11. **Height for age (Z-score) among HDL-C groups among school children**

Height for age among abnormal HDL-C more prevalent among lower school status children 1(7.1%) among them ≥ - 2.00 13(92.9). But none were suffering from abnormal HDL-C with <-2.0 SD (malnutrition) among middle status school children in our study shown in Table-II.11.

Table-II.11. Distribution of HDL-C among the subject by their Z-score of height for age.

School status	Height for age (Z-score)		Overall N(%)
	Stunting <-2.00 SD n(%)	Normal ≥ -2.00 SD n(%)	
Higher			
Normal	2 (1.8)	110 (98.2)	112(100)
Abnormal	1(10.0)	9 (90.0)	10(100)
Middle			
Normal	16(14.4)	95(85.6)	111(100)
Abnormal	0(0.0)	2(100.0)	2(100)
Lower			
Normal	5(9.1)	50(90.9)	55(100)
Abnormal	1(7.1)	13(92.9)	14(100)

II.12. **Height for age (Z-score) among LDL-C subjects**

In higher school status group (Table-II.12), among the abnormal LDL-C children, none were stunted. However, 100% and 6% children were stunted among abnormal LDL-C children in middle and lower school status group respectively.

Table II.12: Distribution of LDL-C among the subject by Height for age Z-score.

School status	Height for age(Z-score)		Overall N (%)
	Stunting <-2.00 SD n(%)	Normal ≥-2.00 SD n(%)	
Higher			
Normal	1(2.0)	50(98.0)	51(100)
Abnormal	0(0.0)	7(100.0)	7(100)
Middle			
Normal	4(11.1)	32(88.9)	36(100)
Abnormal	2(100.0)	0 (0.0)	2(100)
Lower			
Normal	0(0.0)	3(100.0)	3 (100)
Abnormal	2(6.1)	31(93.9)	33(100)

II.13. **Height for age (Z-score) among TG-C groups.**

In the middle school status abnormal subjects were 3(60.0%) found stunted (<-2.00 SD) .But it was not true in other school status groups. It was also true that among ≥ -2.00 SD among abnormal TG-C groups were 17(100.0) among higher and 14(22.2) were among lower status school children shown in table II.13.

Table II.13: Distribution of TG-C among the subject by their Z-score of H/A.

School status	Height for age (Z-score)		Overall N(%)
	Stunting <-2.00 SD n(%)	Normal ≥-2.00 n(%)	
Higher			
Normal	3(2.9)	102(97.1)	105(100.0)
Abnormal	0(0.0)	17(100.0)	17(100.0)
Middle			
Normal	13(12.0)	95(88.0)	108(100.0)
Abnormal	3(60.0)	2(40.0)	5(100.0)
Lower			
Normal	6(10.9)	49 (89.1)	55(79.7)
Abnormal	0 (0.0)	14(100)	14(100)

II.14 **Distribution of WHR among hyper lipidemic subjects.**

Waist-hip ratio among hyper lipidemic middle status boys were more than higher and lower status school boys. It was only true for girls of higher status schools shown in Table –II.14.

Table-II.14: Distribution of WHR among abnormal subjects.

School status	*Boys*	*Girls*
Higher	0.82	0.87
Middle	0.89	0.84
Lower	0.82	0.86

II.15. **Distribution of WHR study children among boys.**

In Table- II.15 shown Waist Hip Ratio (WHR) among boys were 30(15.5%) belong to <.80 WHR 140(72.5%) belong to .80 -.89 WHR and 23(11.9%) within and above .90 WHR. In this table shown above .90 WHR were 12(52.2%), 9(39.1%) and 2(8.7%) in higher, middle and lower school status children respectively.

Table II.15: Distribution of Waist Hip Ratio (WHR) among study boys.

School status	WHR for Boys			Total (%)
	<0.80	0.80-0.89	0.90 +	
Higher	15(17.2%)	60(69.0%)	12(52.2%)	87(45.1)
Middle	6(20.0%)	50(76.9%)	9(39.1%)	65(33.7)
Lower	9(22.0%)	30(73.2%)	2(8.7%)	41(21.2%)
Overall	**30(15.5%)**	**140(72.5%)**	**23(11.9%)**	**193(100.0)**

II.16. **Distribution of WHR among study girls.**

In Table- II.16 shown Waist Hip Ratio (WHR) among girls were 5(4.4%) belong to <0.70 WHR 34(30.1%) belong to 0.70 - 0.79 WHR and 74(65.5%)within and above 0.80 WHR. In this table shown above 0.80 WHR were 18(50.0%), 33(68.8%) and 23(79.3%)in higher, middle and lower school status school of Dhaka city respectively.

Table II.16: Distribution of Waist Hip Ratio among study girls.

School status	WHR for Girls			Total (%)
	<0.70	0.70-0.79	0.80+	
Higher	2(5.6%)	16(44.4%)	18(50.0%)	36(31.9)
Middle	2(40.0%)	13(38.2%)	33(68.8%)	34(30.1%)
Lower	1(20.0%)	5((14.7%)	23(79.3%)	29(25.7%)
Total	**5(4.4%)**	**34(30.1%)**	**74(65.5%)**	**113(100.0)**

II.17. **Distribution of body mass index (BMI) of school children**

In table-II.17. distribution of mean BMI among boys had higher than girls. In both sexes BMI <20 was predominant than other category. It was also found that BMI 20-24 range 16(6.8) boys and 15(10.1) among girls. But none of the boys and girls was present in 24-29 groups. In study children we found >25 BMI were 5(2.1%) boys and 4(2.7%) were girls. In a given category, the percentage of both sexes with a centripetal distribution of fat due to increased with age (not shown in the table).

Table II.17: Distribution of BMI of school children by sex

Sex	No of subjects	BMI Mean±SD	BMI % of subjects		
			<20 n(%)	20-24 n(%)	25- 30+ n(%)
Boys	237	18.02 (29.86)	216(91.1)	16 (6.8)	5(2.1)
Girls	148	16.90 (6.39)	129(87.2)	15(10.1)	4(2.7)
Overall	**385**		**345(89.6)**	**31(8.1)**	**9(2.4)**

II.18. **Distribution of body mass index (BMI) of children by school status**

Body mass index among school children shown in table-II.18. The prevalence of extreme leanness (BMI < 20) is greater among lower and middle status school children. But it was not true in the higher school children.

Table II.18: Distribution of BMI by school status.

School status	BMI % Subjects		
	<20 n (%)	20-24 n (%)	25-30+ n (%)
Higher	98 (80.3)	21(17.2)	3(2.4)
Middle	101(89.4)	7(6.2)	5(4.4)
Lower	69(100)	0(0)	0(0)

Section III

III.I. 7-Day Food Frequency Recall

Table III.1 shows the mean of number of feeds regardless of quantity and quality taken by a students based on a 7-day food frequency recall. The students from higher status schools, gave a mean of 26.62 in girls against that of 23.91 in boys. In the middle status schools, girl children took the feeds on an average of 24.39 times while that was reported to be 21.61 times for boys. Such food frequency was low for students of either sex from schools of lower socio-economic status, which is discussed in the table.

Table III.1: Number of meals consumes and average 7 days calorie Intake by children

School status	*Boys*	*Girls*
Higher	23.91	26.62
Middle	21.61	24.39
Lower	21.72	19.42

III.2 Mean Calorie, Carbohydrate, Protein and fat consumption by study children. (n=392)

Among study children we found maximum intake of calorie, carbohydrate, protein and fat intake by girls population. It was also found mean calorie intake among both population not significant. Carbohydrate, Protein and Fat consumption were almost same between both populations. (Shown in table III.2).

Table-III.2: Mean Calorie, Carbohydrate, Protein and fat consumption by study children(n=392).

Nutrients	Boys/day				Girls /day			
	Mean	Min	Max	SD	Mean	Min	Max	SD
Energy Kcal	1171.88	1115.76	1248.25	67.12	1289.07	1135.75	1482.83	177.03
Carbohydrate	108.27	99.13	117.93	9.41	119.71	110.15	131.01	10.53
Protein N X 6.25	38.32	33.20	46.94	7.50	40.82	34.39	49.14	7. 55
Fat	66.65	61.86	75.48	7.65	73.47	63.31	86.57	11.90

III.3 **Average daily calorie Intake.**

In Table- III.3 Describes the daily calorie Intake of students attending at schools of different Socio-economic status. Mean calorie intake was same in both sexes.

Table- III.3: Average daily calorie intake by the study population

School status	*KCal/day*	
	Boys	Girls
Higher	1246.25	1482.83
Middle	1153.65	1248.65
Lower	1115.76	1135.75

III.4. **Carbohydrate**

Daily carbohydrate Intake for students from schools of different socio-economic status showed that girls of higher status schools consumed more carbohydrate containing foods than boys. However, the intake was similar among students of either sex of middle and lower status schools.(Table- III.4) .

Table III.4: Daily Consumption of carbohydrate among study subject (n=392)

School status	*Boys/gm*	*Girls/gm*
Higher	99.13	131.01
Middle	117.93	117.55
Lower	107.75	110.15

III.5 **Protein**

With regard to mean daily consumption of protein for students from different schools, male children of high school status consumed 46.94g against 49.14g consumed by girls. However, consumption was relatively less in students from low school status shown in table III.5.

Table III.5: Daily Consumption of protein of the study subjects(n = 392)

School status	Boys/gm	Girls/gm
Higher	46.94	49.14
Middle	34.83	38.95
Lower	33.20	34.39

III.6. **Fat**

Mean daily fat consumption by students from higher status group was less in boys than girls (75.84 gm vs. 86.57 gm). As expected, fat consumption was less in both groups of students among schools of lower school status groups (Table –III.6).

Table III.6: Daily Consumption of fat of the study subject (n=392).

School status	Boys/gm	Girls/gm
Higher	75.84	86.57
Middle	61.86	70.55
Lower	62.63	63.31

III.7. **Retinol, Carotene, Vitamin C, and Vitamin E consumption**

It was found both boys and girls of lower school status children had relatively low intake of retinol, Only girls of lower school status consume less carotene than girls of other schools. Vitamin C and vitamin E intake among children almost similar between all groups shown in Table-III.7.

Table III.7: Daily Consumption of antioxidant by the study population (n=392).

School status	Retinol		Carotene		Vit.C		Vit.E	
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
Higher	636.80	627.40	841.36	842..36	13.36	13.49	1.04	2.17
Middle	383.30	470.02	795.87	904.80	11.58	12.58	2.14	2.11
Lower	418.59	424.25	908.48	698.44	12.73	10.53	1.87	1.95

III.8 **Average daily consumption of minerals and trace elements.**

The middle and lower status boys had relatively lower consumption of trace elements than higher status school boys. Among girls of higher status school minerals and trace elements consumption was more than middle and lower status groups in our study shown in Table-III.8.

Table –III.8: Average daily consumption of minerals and trace elements among school boys and girls.

Schools status	Sex	Calcium /mg	Magnesium /mg	Copper /mg	Zinc/mg
Higher	Boys	355.31	133.22	0.62	3.81
	Girls	305.60	131.74	0.63	3.88
Middle	Boys	275.48	110.79	0.50	3.13
	Girls	275.48	101.30	0.44	2.86
Lower	Boys	242.71	101.41	0.46	2.80
	Girls	202.39	98.28	0.45	2.58

III.9 . Distribution of food intake (Animal source) in study children (Normal and abnormal groups)

Distribution of animal source among children found higher and middle school status children consume more than lower school status children .But in case chicken it was found that higher school status children consume more than other two groups among normal serum cholesterol.(Table-III.9) In table II-9 shown that egg, milk and milk product consume more among higher school status children who were high total cholesterol but it was not true in other two groups. Again it was found that beef, chicken, mutton and fish were more consume by both groups among those who had high total cholesterol.

III.10 . Distribution of food intake (Plant source) in study children normal and abnormal groups

Distribution of plant source (Beans, Fruits, Rice, Parata, Bread, Leafy Vegetable . and other vegetables) almost same among both school status groups. In Table III.10 Among abnormal groups (TC-C>205mg/dl) it was found that only "other vegetable" were more consume by lower school status group. But this was not true in other two groups. In this study we found that high total cholesterol groups were consume same amount of plant sources.

Table-III.9 Distribution of food intake (Animal sources) in study children

School status		Food groups (number) Mean ± SD						
		Egg	Milk	Milk product	Beef	Mutton	Chicken	Fish
Higher	Normal	(59) 19.03± 8.5	(52) 73.83± 39.2	(52) 59.75± 30.8	(52) 15.60±7.3	(24) 10.89±7.6	(50) 360.0±1794	(59) 22.57±11.3
	Abnormal	(2) 19.29± 3.0	(1) 125.0 ± 0	(2) 44.64±12.63	(1) 11.43±0	---	(2) 428.57±151.5	(1) 28.57±0
Middle	Normal	(57) 14.29±8.8	(53) 74.46 ±41.6	(32) 55.80± 37.0	(38) 13.68± 6.58	(16) 7.50±6.4	(33) 237.01±157.9	(63) 25.40±8.9
	Abnormal	(2) 21.43±.00	(3) 101.19±20.6	(3) 65.48±37.17	(2) 4.29±.00	(2) 4.29±.00	(2) 321.43±151.5	(3) 26.67±8.7
Lower	Normal	(24) 8.04± 4.2	(16) 45.76 ± 36.2	(23) 45.03± 29.9	(24) 9.29±5.00	(1) 4.29±0	(11) 233.77±150.1	(42) 31.97±34.2
	Abuormal	(6) 7.86±3.2	(5) 28.57±15.9	(6) 29.76 ± 0	(4) 15.71±5.47	(1) 4.29±0	(2) 321.43±151.5	(10) 24.57±10.7

Table- III.10. Distribution of food intake (Plant source) in study children normal and abnormal groups .

School Status		Food groups/gm (number) Mean ± SD						
		Beans	**Fruits**	**Rice**	**Pratea**	**Bread**	**Leafy veg.**	**Other Veg**
Higher	Normal	(62) 75.12±23.1	(57) 66.67± 28.8	(63) 116.73± 9.1	(48) 11.61± 8.5	(56) 72.12± 30.8	(55) 6.84 ± 4.8	(56) 105.23± 37.2
	Abnormal	(2) 57.14±40.4	(2) 42.86±40.4	(2) 120.00±.00	(2) 4.29±.00	(2) 78.57±22.2	(1) 6.00±.00	(2) 75.00±75.7
Middle	Normal	(62) 67.51±27.4	(60) 69.29±26.9	(63) 117.55±9.1	(37) 13.90± 9.0	(54) 64.31±34.4	(54) 7.48 ±3.97	(51) 105.88± 45.0
	Abnormal	(3) 66.67±16.5	(3) 95.24±8.2	(3) 120.00±.00	(2) 10.71±9.0	(3) 62.86±15.7	(3) 7.33± 1.1	(3) 107.14± 56.6
Lower	Normal	(41) 70.73±26.7	(40) 63.93±28.3	(42) 119.18±3.6	(13) 7.91±5.4	(31) 63.36± 36.4	(36) 8.50± 3.7	(37) 130.31±30.4
	Abnormal	(10) 60.00±34.8	(10) 60.00±25.9	(10) 114.86±11.5	(4) 12.86± 12.1	(6) 70.71± 29.4	(9) 7.56± 4.9	(10) 120.00± 39.3

Fig. III.1: **Comparison between amount of food intake (plant sources) among study population**

When our data of food intake from plant sources were compared with that of National Nutrition Survey (1995-96), we observed substantially less amount of intake of rice and leafy vegetables by our study population (Fig. III.1).

Fig. III.2 : **Comparison between amount of food intake(Animal sources) among study population**

Our study population was observed to consume higher amount of egg, milk, milk product, and beef than that of the population that participated in National Nutrition Survey (1995-96). However, their mutton and fish intake was less than the National figures(Fig. III.2).

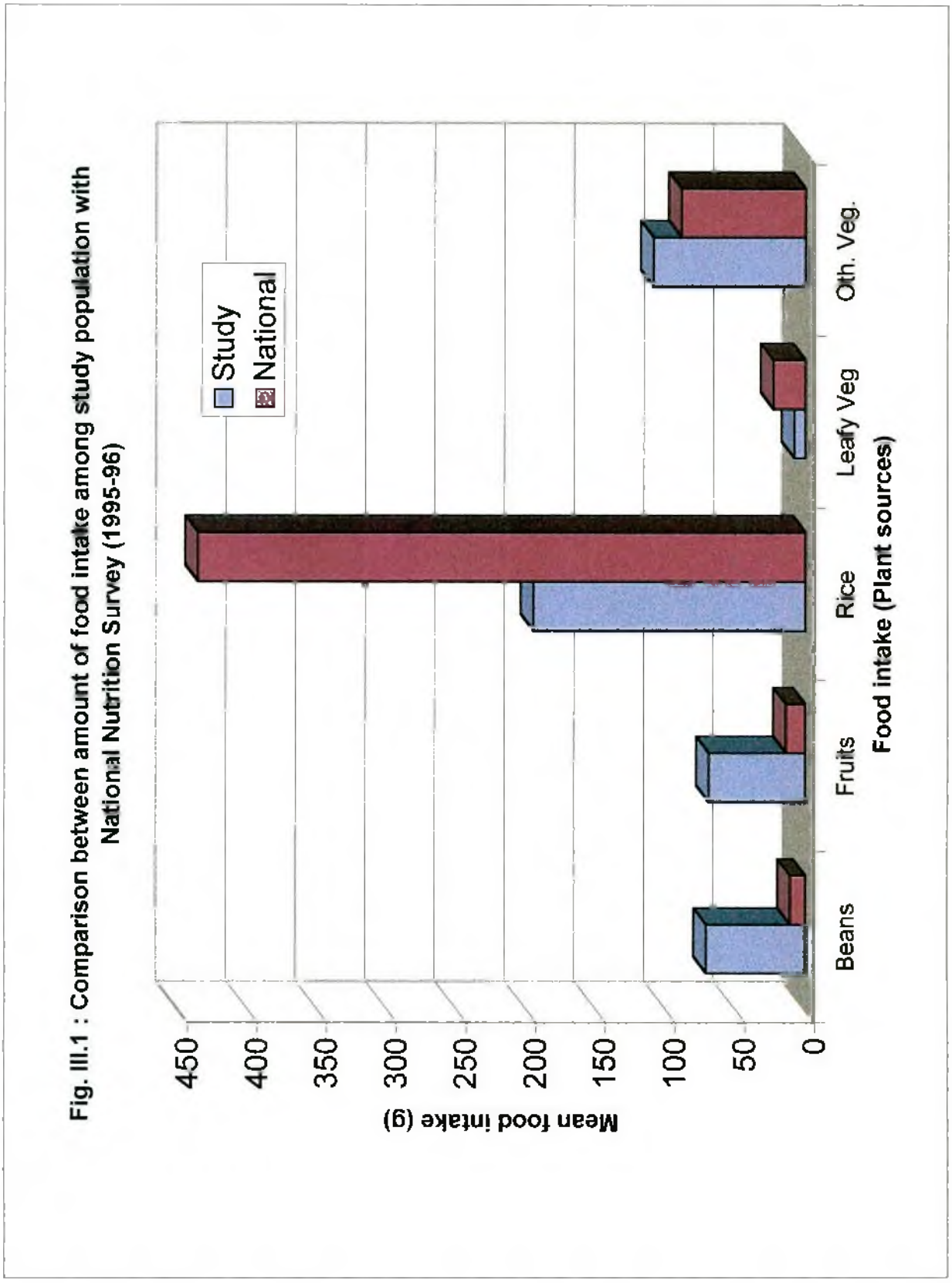

Fig. III.1 : Comparison between amount of food intake among study population with National Nutrition Survey (1995-96)

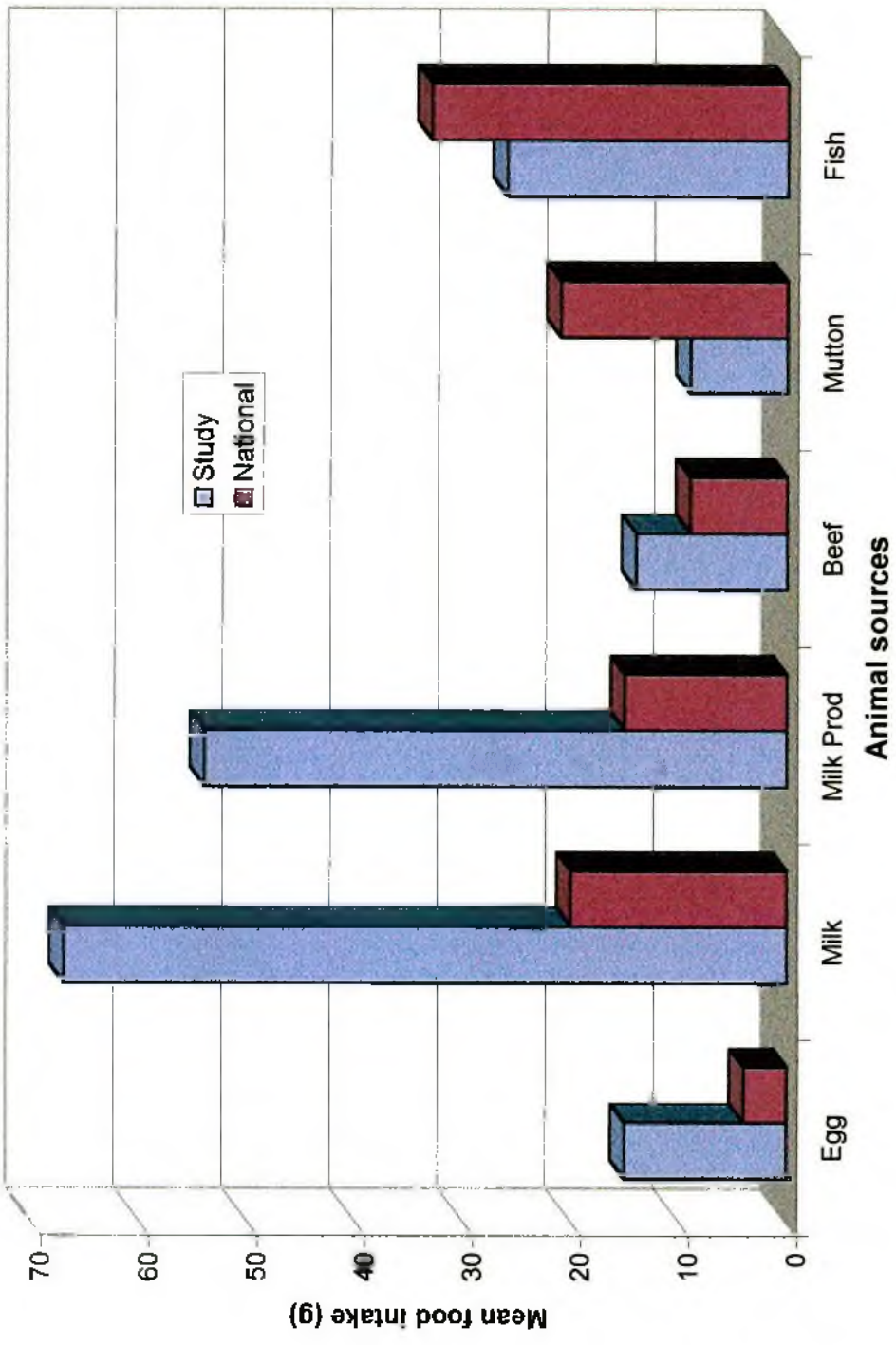

Fig. III.2 : Comparison between amount of food intake among study population with national Nutritional Survey (1995-96).

Section-IV

Epidemiology of Lipid profile among study subjects.

IV.1 Lipid profile of study subjects.

In our study (Table IV.1) shows mean and standard deviation and minimum and maximum lipids among the children where TC-C minimum and maximum value 99mg/dL and 208 mg/dL, HDL-C 22 mg/dL and 72 mg/dL, LDL-C 37 mg/dL and 221 mg/dL and TG-C found 36 mg/dL and 215 mg/dL respectively.

Table-IV.1: Lipid profile minimum and maximum value of study subjects .

Lipid profile	*Mean ± SD*	*Minimum value*	*Maximum value*
TC-C mg/dL	174.52 ± 19.85	99	280
HDL-Cmg/dL	38.74 ± 5.29	22	72
LDL-C mg/dL	64.74 ± 41.18	37	221
TG-C mg/dL	60.47 ± 39. 24	36	215

IV.2 Mean and SD of Lipid profile among three status study children.

The mean total cholesterol, HDL, LDL, and TG levels in students of higher and middle status schools lied within the normal ranges. This was true for TC, LDL, and TG levels of students from lower strata schools. However, HDL level in low school status school students was significantly low ($p<0.05$) compared to that of students from higher and middle status students.

Mean total cholesterol level was 181.91 mg/dL among children those included higher school status schools. That was 159.18 mg/dL and 179.14 mg/dL in students from

middle and lower school status schools respectively (**Table not shown**).

The mean serum triglyceride levels of students regardless of their school status were within the normal range. The level of TG in middle class school students was lower than other groups (***Table not shown***).

IV.3. **Abnormal lipoprotein among school children.**

In this study a total of 392 school children were enrolled. Among these 392 school children, 24(6.12%) children were hyperlipidemic, 43(10.95%) children had low level of High density lipoprotein (HDL),26(6.63%) had high LDL and 36(9.18%) children had high level of Triglyceride cholestrol. Children with abnormal (hyperlipidemic) lipid profile, were considered as hyperlipidemic case.

Table IV.3 : Abnormal lipoprotein among school children (N=392)

Cholesterol	Number	Percentage
TC (>205mg/dL)	24	6.1
HDL (<37mg/dL)	43	10.9
LDL (>145mg/dL)	26	6.6
TG (>130mg/dL)	36	9.1

IV.4 **Mean total cholesterol among normal and abnormal groups**

Total cholesterol among higher and lower status students was higher than middle status school students shown in Table –IV.4

Table IV.4: Mean and standard deviation of total cholesterol among children normal and abnormal groups.

School status	*TC-Cholesterol*	
	Normal(Mean±SD)	**Abnormal (Mean± SD)**
Higher status	169.83 ± 19.38	233.55 ± 21.31
Middle status	154.81 ± 20.70	211.67 ± 3.51
Lower status	161.22 ± 29.08	227.76 ± 23.36
All school	**162.80 ± 23.26**	**228.29 ± 21.49**

IV.5 Mean and standard deviation Low density lipoprotein (LDL) among normal and abnormal school children.

Among the abnormal lipids children, mean plasma level of LDL was more among higher and lower schools status than that of middle status schools. This was also true for normal LDL level less than 150 mg/dL shown among middle status school status children. (Table-IV.5).

Table –IV.5: Mean and SD Low Density Lipoprotein (LDL) among normal and abnormal among children.

School Status	*LDL-Cholesterol*	
	Normal (Mean± SD)	*Abnormal (Mean± SD)*
Higher	107.98 ± 20.65	172.50 ± 20.37
Middle	91.30 ± 24.15	149.50 ± 0.71
Lower	102.96 ± 25.07	168.00± 19.90
All school	**101.19± 23 .84**	**168.31 ± 19.77**

IV. 6. Mean and standard deviation High density lipoprotein (HDL) among Normal and Abnormal group.

Abnormal HDL-C were significantly ($p<0.05$) lower among lower status school children than that of other two school status children. This was also true for HDL-C level among normal subjects which was also abnormal finding in our study among lower school status (Table- IV.6).

Table IV.6: Mean and SD High density Lipoprotein (HDL) among normal and abnormal group.

School status	*HDL- Cholesterol*	
	Normal (Mean± SD)	*Abnormal(Mean± SD)*
Higher	41.07± 5.20	35.00 ± 0.92
Middle	46.23 ± 5.65	35.50 ± 0.50
Lower	27.38 ± 4.87	26.38 ± 3.66
All school	**43.79±5.65**	**28.21 ± 4.89**

IV.7 Mean and standard deviation serum Triglyceride(TG) among normal and abnormal

In the table-IV.7 it was found that in the both school status mean TG-C was more than 150mg/L among abnormal groups. But in the normal groups overall mean TG-C was 89.33 ± 19.33 found in our study.

Table IV.7: Mean and standard deviation Triglyceride cholesterol (TG-C) among normal and abnormal

School status	*TG-Cholesterol*	
	Normal (Mean± SD)	*Abnormal(Mean± SD)*
Higher	89.85 ± 16.92	155.47± 27.30
Middle	85.85 ± 21.54	150.40 ± 16.98
Lower	93.52 ± 19.83	153.97 ± 24.74
All school	**89.33 ± 19.33**	**153.97 ± 24.58**

IV.8. **Mean Lipoprotein Among Abnormal Groups of Children**

Higher and lower status school children had proportionately more total cholesterol, and LDL. However, HDL of lower status school children was lower than that of higher and middle status school children. Serum TG level was same in students of schools of diverse status discussed in the Table-IV.8.

Table IV.8: Mean and Standard Deviation of Abnormal Lipoprotein Among School Children

School and Lipids Status	*T.Chol. (Mean± SD) (n=24)*	*HDL (Mean±SD) (n=43)*	*LDL (Mean±SD) (n=26)*	*TG (Mean± SD) (n=36)*
Higher	233.6 ± 21.4	35.0±0.9	172.5±20.4	155.5±27.3
Middle	211.7 ± 3.5	35.5± 0.5	149.5±0.7	150.4±17.0
Lower	227.8 ± 23.4	26.4± 3.7	168.0 ±19.9	154.0 ± 24.7
Overall	228.2±21.5	28.2±4.9	168.3±19.8	154.0±24.5

NB: The cutoff value for hypercholesterolaemia (abnormal Lipid Profile) was 2-20 years children was 120-205 mg/dL TC-C, 37-73 mg/dL, HDL-C, 66-145 mg/dL LDL-C and 110-130 mg/dL Triglyceride cholesterol . In this study we followed the above references value for our study. [390]

IV.9 Distribution of abnormal lipoprotein among the study subjects by their gender

Boys and girls of both higher and lower status schools had similar level of total cholesterol (TC-C). Both boys and girls of lower status schools had proportionately more low level of HDL than that of higher school status. However, this was not true for LDL-C and TG-C level was same in boys and girls regardless of their school status(Shown in Table- IV.9).

Table IV.9: Distribution of normal and abnormal lipoprotein by their gender

School Status		TC-C		HDL-C		LDL-C		TG-C	
		Boys N(%)	Girls N(%)	Boys N(%)	Girls N(%)	Boys N(%)	Girls N(%)	Boys N(%)	Girls N(%)
Higher	Normal	33 (51.6)	14 (30.4)	37(66.1)	14(40.0)	34(54.8)	14(30.4)	30(50.0)	11(28.2)
	Abnormal	8 (53.3)	3 (33.3)	4(17.4)	3(15.0)	7 (41.2)	3(33.3)	11 (55.0)	6(37.5)
Middle	Normal	18 (28.1)	18 (39.1)	18 (32.1)	19(54.3)	18(29.0)	19 (41.3)	16 (27.1)	18(46.2)
	Abnormal	1 (6.7)	2 (22.2)	1(4.3)	1(5.0)	1 (5.9)	1 (11.1)	3 (15.0)	2(12.5)
Lower	Normal	13 (20.3)	14 (30.4)	1 (1.8)	2 (5.7)	10(16.1)	13 (28.3)	13(22.0)	10(25.6)
	Abnormal	6 (40.0)	4 (44.4)	18(78.3)	16(80.0)	9(52.9)	5 (55.6)	6 (30.0)	8(50.0)

IV.10 Distribution of High TC-C /HDL-C Ratio among study subjects.

Total Cholesterol/HDL ratio more than >4.5 (undesirable) among higher school boys were 13(21.7), girls were 9 (52.9). Undesirable level among lower status school boys were 17(89.5) and girls were 14(77.8) respectively. But Total Cholesterol / HDL ratio <4.5 (desirable) among higher school were 28(68.3%) boys and 8(47.1%) were girls. In middle status boys were 16(84.2%) and girls were 18(90.0%) but lower status boys were 2(10.5%) and 4(22.2%) girls respectively shown in table-IV.10.Among study children 76(56%) desirable and 58(44%) undesirable level of Total Cholesterol/ HDL cholesterol ratio found in this study.

Table-IV.10 Distribution of high TC-C / HDL-C Ratio among school children.

School status	Number (%)		Overall Number (%)
	Desirable* <4.5	Undesirable* > 4.5	
Higher			
Boys	28(68.3)	13(21.7)	41(100.0)
Girls	8(47.1)	9 (52.9)	17(100.0)
Middle			
Boys	16(84.2)	3(20.7)	19(100.0)
Girls	18(90.0)	2(16.7)	20(100.0)
Lower			
Boys	2(10.5)	17(89.5)	19(100.0)
Girls	4(22.2)	14(77.8)	18(100.0)
Overall	**76(56.0)**	**58(44.0)**	**134(100.0)**

*Reference following by Charlotte C, Cook Fuller Nutrition 94/95; 6th edition 1994 [392]

IV.11 **Distribution of high LDL/HDL-C Ratio among study children**

In table IV.11 high LDL/HDL– C ratio more than >3 (Undesirable) among three different school children were 13(31.7) were boys and 10(58.8) were girls among higher school status , 16(84.3) boys and 12(66.6) girls among lower school status respectively but among middle status children undesirable level was less than other two groups. Overall ratio among different level LDL/HDL cholesterol were 77(57.4%) desirable, and 57(42.6%) undesirable level respectively. It was also found that among lower status children were more undesirable level than other two groups which is our new study findings.

Table-IV.11: Distribution of high LDL/HDL-C Ratio among school children

School status	Number (%)		Overall Number (%)
	Desirable* <3	Undesirable > 3	
Higher			
Boys	28(68.3)	13(31.7)	41(100.0)
Girls	7 (33.4)	10(58.8)	17(100.0)
Middle			
Boys	15(78.9)	4(21.1)	19(100.0)
Girls	18(90.0)	2(10.0)	20(100.0)
Lower			
Boys	3(15.8)	16(84.3)	19(100.0)
Girls	6(33.3)	12(66.6)	18(100.0)
Overall	**77(57.4)**	**57(42.6)**	**134(100.0)**

*Reference following by Charlotte C, Cook Fuller Nutrition 94/95; 6th edition 1994 [392]

IV.12. **Average physical activity among hyper cholesterol study group.**

In this table physical activity among hyper cholesterol group who had minimum and maximum activity per minute has shown. The boys of lower and higher groups had poor physical activity (shown in table IV.12).

Table IV.12 : Mean physical activity among normal and abnormal Total cholesterol school children

School status		Mean ± SD	Min. activity/min	Max.activity/min
Higher	Normal (n=41)	57.9 ± 44.4	30	180
	Abnormal (n=11)	95.4 ± 44.1	30	180
Middle	Normal (n=33)	107.2 ± 30.8	60	180
	Abnormal (n=3)	120 ± 0.0	120	120
Lower	Normal (n=25)	78.00 ± 45.0	30	240
	Abnormal (n=10)	57 ± 12.2	30	120

IV.13 **Physical activity among Abnormal lipid profile group.**

Daily physical activity among middle status school boys and girls was higher than that of two other groups (shown in table-IV.13).

Table-IV.13 Physical activity among Abnormal lipid profile group.

School status	*Boys/min*	*Girls/min*
Higher	105	70
Middle	120	120
Lower	70*	30*

*P=<0.001

IV.14. Physical activity and Total Cholesterol among normal and abnormal group

Physical activity below 60 minutes were 182(59.2%), Above 60+ minutes activity were 5(50.0%) were abnormal total cholesterol among higher school status children, 2(100.0%) were middle status school children and 3(21.4%) were among lower school status respectively. It was also found that physical activity among higher and lower school children were very poor than middle status children.(shown in Table-IV.14).

Table IV.14: Physical activity and Total Cholesterol among normal and abnormal group

School status	Physical activity/minute/day		Total N (%)
	<60 n(%)	>60 n(%)	
Higher			
Normal	89(78.8)	24(21.3)	113(100.0)
Abnormal	5(50.0%)	5(50.0%)	10(100.0)
Middle			
Normal	35(31.3%)	77(68.8)	112(100.0)
Abnormal	0.0	2(100.0%)	2(100.0)
Lower			
Normal	42(75.0%)	14(25.0)	56(100.0)
Abnormal	11(78.6%)	3(21.4%)	14(100.0)
Overall	**182(59.2%)**	**125(40.8)**	**307(100.0)**

IV.15 Physical activity and HDL-Cholesterol among normal and abnormal group

In Table- IV.15 shown that among HDL cholesterol groups 83(61.9%) among <60 minute physical activity, 51(38.1) were above>60+ minutes physical activity Among

them < 60 minutes physical activity 5(71.4%) suffering from abnormal HDL cholesterol in higher status school children, 25(73.5%) were among lower status groups but none were among middle status groups. In our study majority were suffering from abnormal HDL–C among <60 minute physical activity which is our new finding in the study.

Table IV.15: Physical activity and HDL-Cholesterol among normal and abnormal group

School status	Physical activity/minute		Total N (%)
	<60 n(%)	>60 n(%)	
Higher			
Normal	39(76.5%)	12(23.5)	51(100.0)
Abnormal	5(71.4%)	2(28.6%)	7(100.0)
Middle			
Normal	11(29.7)	26(70.3)	37(100.0)
Abnormal	0(0.0)	2(100.0)	2(100.0)
Lower			
Normal	3(100.0%)	0(0.0)	3(100.0)
Abnormal	25(73.5%)	9((26.5%)	34(100.0)
Overall	**83(61.9%)**	**51(38.1)**	**134(100.0)**

IV. 16 Physical activity and LDL -Cholesterol among normal and abnormal group

In Table- IV.16 Physical activity and LDL – Cholesterol found 182(59.2%) among <60 minute activity and 6(1.9%) for 60-100 minutes physical activities and 119(38.7%) were above 100 minutes physical activities. Among higher school status children abnormal LDL-C groups were 12(70.6%), Middle school status children were 1(20.0%) and lower school status children were 10(71.4%) had <60 minutes physical activities. In 60-100 minutes physical activity were no abnormal groups LDL-C. Above 100 minutes physical activity it was 5(29.4%), 4(80.0%) and 4(28.6%) respectively in different school status children.

Table-IV.16: Physical activity and LDL -Cholesterol among normal and abnormal group.

School status	Physical activity/minute		Total N (%)
	<60 n(%)	>60 n(%)	
Higher			
Normal	89(79.5%)	23(20.6)	112 (100.0)
Abnormal	5(45.5%)	6(54.5%)	11 (100.0)
Middle			
Normal	35(31.5%)	76(68.5)	111 (100.0)
Abnormal	0.0	3(100.0)	3 (100.0)
Lower			
Normal	45(75.0%)	15(25. 0)	60(100.0)
Abnormal	8(80.0%)	2(20.0%)	10(100.0)
Overall	**182(59.2%)**	**125(40.8)**	**307(100.0)**

IV.17 Physical activity and TG -Cholesterol among normal and abnormal group

Below 60 minutes physical activity among different school status children were

(Abnormal TG-C) were 12(70.6%), 1(20.0%) and 10(71.4%). But above >60+ minutes 5(29.4%), 4(80.0%) and 4(28.6%) among higher, middle and lower school status children shown in Table-IV.17.

Table IV.17 : Physical activity and TG -Cholesterol among normal and abnormal group.

School status	Physical activity/minute/ day		Total N(%)
	<60 n(%)	>60 n(%)	
Higher			
Normal	82(77.4)	24(22.7)	106(100.0)
Abnormal	12(70.6)	5 (29.4)	17(100.0)
Middle			
Normal	34(31.2)	75(68.8)	109(100.0)
Abnormal	1(20.0)	4 (80.0)	5(100.0)
Lower			
Normal	43(76.8)	13(23.2)	56(100.0)
Abnormal	10(71.4)	4(28.6%)	14(100.0)
Overall	**182(59.2%)**	**125 (40.6)**	**307(100.0)**

IV.18 Logistic regression analysis (Dependant variable TC-C mg/dL)

Logistic regression analysis among total cholesterol found not significant among school status and other independent variable shown in table IV.18.

Table IV.18: Results of Logistic Regression Analysis (Dependent variable TC-C/ mg/dL)

Independent variables	Co- efficient	Odd Ratios	95% CI	P value
1. Sex	.2066	1.2295	0.491-3.076	.6589 (NS)
2. BCG	.4766	1.6106	0.426-6.095	.4827 (NS)
3. DPT	-.7871	.4552	0.007-31.327	.7154 (NS)
4. POLIO	.5552	1.7422	0.031-99.475	.7879 (NS)
5. Physical Activity	-.0046	.9954	0.986-1.005	.3481 (NS)
6. WHR	-.5778	.5611	0.007-45.458	.7966 (NS)
7. School status	-.3720	.6893	0.251-1.891	.4700 (NS)
-2 log likelihood 166.15 P value not significant (NS).				

IV.19 **Logistic Regression Analysis (dependent variable HDL-C mg/dL)**

In table IV.21 shown independent variable like BCG, Polio vaccine and school status among HDL-C found significant. Student of higher school found four times more likely to have high (95% CI for Odds ratio) HDL-cholesterol p<00.1 highly significant than lower and middle status school. Polio vaccine immunised group are less likely to developed abnormal HDL cholesterol in this study. BCG immunised group have higher risk of abnormal HDL cholesterol than those who were not immunised.

Table IV.19: Results of Multiple logistic Regression Analysis (Dependent variable HDL-C mg/dL)

Independent variables	Co- efficient	Odd Ratios	95% CI	P value
1. Sex	.4543	.8139	0.334-1.983	.6503
2. BCG	.6664	4.6957	1.272-17.336	.0203*
4. POLIO	.7954	.1147	0.029-0.457	.0021**
5. Physical Activity	.0050	1.0022	0.992-1.012	.6564
6. WHR	2.7612	.2905	0.001-65.086	.6544
7. School status	.5577	4.3389	1.454-12.946	.0085**
-2 Log Likelihood 132.156				
** p<00.1 =highly significant.				
* p< 0.05= significant				

IV.20 Logistic Regression Analysis (Dependent variable LDL-C mg/dL)

Logistic regression analysis among LDL cholesterol found not significant among school status and other independent variable shown in table IV.20.

Table IV.20: Results of Logistic Regression Analysis (Dependent variable LDL-C/ mg/dL).

Independent variables	Co- efficient	Odd Ratios	95% CI	P value
1. Sex	.4529	.8970	0.370-2.175	.8100 (NS)*
2. BCG	.5409	1.6986	0.588-4.904	.3273 (NS)
4. POLIO	.5528	.5852	0.198-1.729	3324 (NS)
5. Physical Activity	.0049	.9993	0.990-1.009	8939 (NS)
6. WHR	2.0240	1.2523	0.024-66.156	9115 (NS)
7. School status	.5075	.9614	0.356-2.600	9383 (NS)
-2 Log Likelihood 176.812 *NS= not significant				

IV.21 Logistic regression analysis (dependent variable TG-C mg/dL).

In table IV.21 shown higher risk for lower status school than that of higher school (p<.074) significant at 10% .

Table IV.21: Results of Logistic Regression Analysis (dependent variable TG-C mg/dL).

Independent variables	Co- efficient	Odd Ratios	95% CI	P value
1. Sex	.3843	1.6916	0.797-3.593	.1714
2. BCG	.4935	1.7218	0.655-4.530	.2709
4. POLIO	.5217	.4300	0.155-1.196	.1058
5. Physical Activity	.0043	.9974	0.989-1.006	.5404
6. WHR	2.5845	.0169	0.0001-2.683	.1145
7. School status	.4491	.4494	0.186-1.084	.0749*
-2 Log Likelihood 214.129 * significant at 10%				

IV. 22 Percentiles of Lipid profile among study Children

In table-IV.24 Shown plasma total Cholesterol, LDL -C, HDL-C and TG-C levels among study school children, and their median and percentiles. However, Median among higher and lower school status children were increasing trend than middle status children. But HDL-C of lower status school children were lower than that of higher and middle status school children. Serum TG level was same in students of diverse status.

Table IV.22: Percentiles distribution of Lipid profile among different school

School status Lipids	25th		Median		75th		90th		99th	
Higher status	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls
TC-C mg/dL	160	160	183	174	200	200	231	231	256	280
LDL-C mg/dL	93	100	120	112	137	132	166	171	195	211
TG-C mg/dL	93	83	100	85	142	133	203	162	215	200
HDL-mg/dL	38	39	39	40	41	43	51	46	72	48
Middle status										
TC-C mg/dL	144	138	155	153	169	171	206	200	215	212
LDL-C mg/dL	81	78	92	93	101	112	140	139	149	150
TG-C mg/dL	61	61	92	92	107	123	137	138	169	169
HDL-mg/dL	45	40	49	45	52	48	55	52	56	55
Lower status										
TC-C mg/dL	156	140	175	183	204	215	222	230	280	260
LDL-C mg/dL	92	100	121	144	161	163	175	185	188	221
TG-C mg/dL	85	70	118	118	150	135	170	200	185	200
HDL-mg/dL	***25***	***23***	***28***	***25***	***33***	***26***	***37***	***35***	***40***	***39***

CHAPTER – 5

DISCUSSION

Chapter-5

Discussion

Lipids are a substrate for production of metabolic energy and in young children it has been shown to predict value to prevent future development of coronary heart diseases. A cross sectional study was conducted in 392 school children drawn from three schools of Dhaka city and findings are discussed below.

Socio-economic status

The subjects in this study represented a group of healthy primary school children in Dhaka city. Kindergarten schools of Dhanmondi area representing population with higher socio-economic status, primary school (government) of Mohammadpur represented a population with middle and mixed socio-economic status, and the rest of the study children were drawn from poor socio-economic status from UCEP school, Rayer bazar area, Dhaka.

The monthly income distribution was almost similar to the observed of Bangladesh reports. A comparative study among urban poor in Bangladesh Study Report explain that in Bangladesh for a family of four, the basic needs for foods, clothing, housing, health care and education were calculated to be Tk.2600 per month; but Tk.1724 per month will be enough to keep the family on the borderline of starvation and to pay minimal amounts towards meeting other basic needs.[287] The study children were above these the monthly income category. Occupation of the parents was 49.7% service, 37.9% business and rest 12.8% other worker. In this study 32% students from higher status school, 40% from middle status school and 28% respondents were

from the under privileged schools (Table-I.5, 1.6 and I.7).

Nutritional Status of Children

Barker and his colleagues[297-299] focused on cardio- respiratory disease and related it with lower birth weight and (among men) poorer growth during the first year of life have been associated with increased coronary heart disease (CHD) mortality shown over a 65-year follow-up in a retrospective study. Those findings encourage the investigation of the developmental origins of cardio respiratory disease risk factors, including blood pressure, blood lipids, haemostatic factors, respiratory function, fat distribution and propensity to insulin dependent diabetes. The CVD risk factors such as blood pressure and cholesterol track through childhood into adulthood.[300] These data make a strong prima-facie case that the origins of cardio respiratory disease should be sought in early life. However there have been few attempts to replicate the basic finding that lower birth weight is associated with increased risk of major coronary morbidity and mortality and both positive and negative findings have been reported.[301-303] The mean weight of the study subjects were 32.64±8.01 kg, which was close to national figure indicate that birth weight was not normal i.e. there was increase low birth weight children in the sample.

It was found that weight and mid arm circumference (MAC) were significantly higher among higher school status (45.43 ± 11.96 Vs 27.00 ± 4.24 and 26.91 ± 1.80) than mixed and underprivileged children. Higher trend waist hip ratio (WHR) also among higher status school than mixed and lower status school children (Table-II.3), indicating children of higher status is likely to be at risk of coronary heart disease in future. Between abnormal LDL-C study children's MAC and triceps skin fold

thickness among higher school status children was 23.10 ± 2.13 and 15.45 ± 3.30, which was more than middle and lower status school children (Table-II.4). The same pattern also exists in case of triglyceride cholesterol in our study. So, study reveals that children with abnormal lipoprotein pattern among higher school status children had Height, Weight, MAC, Triceps and other parameters were more than that of middle and lower school status children (Table-II.5).

A group of scientist like Colley and others[306] compared that triceps skin fold thickness in children in 1971 aged between 6 and 14 years with that of children measured in 1959 and found an increase in older girls. Peckham et al,[307] using data from the first two British cohort studies, reported increases in the prevalence of overweight and obesity in 7-year-old children from 1953 to 1965 and in the same children at age 11 found 1957 to 1969. In the 1970s there was an increase in weight in children aged 5 to 11 years,[308] but slightly less for English children than expected from the increase in height over the same period. Scottish children showed a much greater increase in weight, and an increase in Weight-for-height, so that by 1980 the weight-for-height of Scottish children appeared slightly greater than that for English children. Overall observation was weight of higher school status was more than two other groups of children.

Triceps skin fold thickness also showed greater increases in Scottish children than in English children, but trends for the latter were positive and statistically significant. From their paper the estimated trends in weight-for-height and triceps skin fold thickness, for English and Scottish boys and girls, and compare them with earlier trends. Trends in the two components of weight-for-height were examined, in order to provide a complex picture of the recent changes in growth. Some possible

explanations for trends in weight-for-height and skin folds in children, in particular the trends in parental weight-for-height and decreases in family size, were examined, and differences in trends between social groups were considered.

In this study weight-for-height though not compared with NCHS yet corresponding parameter for nutritional status found higher in higher school status group.

The subjects in our study population compares well with nutritional parameter found among children in 1975-76,1981-82 and 1995-96 surveys. It appears that nutritional status has improved from that of 1975-76. In 1981-82 about 6% of 0-11 year old children was found to be normal, 20% was observed to have 1st degree, 51% 2nd degree and 23% 3rd degree malnutrition. According to the 1989-90 and 1992 Child Nutrition Surveys, prevalence of wasting actually increased from 14.7% to 16.7%. Stunting also remained more or less static at around 65% (Bangladesh Bureau of Statistics, Child Nutrition Survey of Bangladesh 1992). About a third of the population in Bangladesh consumed less than 1805 calories per adult equivalent per day during 1990(BBS, 1993 year Book).[382] Height for age Z score ≥-2.00+ shown in low HDL-C was more prevalent among lower school status children 13(92.9%). But none had low HDL-C with Z score <-2.0 SD (stunting) among middle status school children in our study.

But, height for age among (Z -score <-2.00 SD) lower school status had higher LDL-C (6.1 %) and ≥-2.00+ Z-score (94 %) but abnormal LDL-C was less between other two groups (Table-II.12). However, in this study the subjects have better nutritional status and abnormal lipid profile status was different in different school status categories. The study observed that children of higher status school of Dhaka city needs further long term follow up for detection of ischaemic heart disease initiated.

Waist-hip ratios among hyperlipaedemic boys from medium socio-economic class were more than those among hyperlipaedemic boys from higher and lower school status which indicated that beef is more or only source of protein energy and fat source in diet, it was only true for girls of higher status schools (Table – II.14)

Overweight in children had been shown to be predictive of a wide range of adverse health effects, in particular increased morbidity from coronary heart disease.[98, 99] in adults, abdominal adiposity is particularly associated with risk factors for cardiovascular disease. The ratio of waist to hip circumference (waist: hip ratio, WHR) has proven a simple and reliable means of estimating abdominal adiposity in epidemiological studies and is frequently used as an indicator of cardiovascular risk in adults.[100] The adverse effects of upper body obesity have also been studied by using the ratio of waist to hip circumferences (WHR) [133] a relatively simple index that emphasises abdominal obesity[134, 135,136] WHR is associated with an increased prevalence of hypertension and diabetes[137, 138] and with levels of triglycerieds, glucose, insulin, and blood pressure[139, 140,141] Prospective studies have shown that, even after accounting for Quetelet Index (kg/m^2) or various skin fold thickness, the risk of coronary heart disease is greatest in both men[142] and women[143] with an increased WHR.

Although the mechanism by which WHR confers an increased risk of disease is not clear, adipose tissue at different locations exhibits metabolic difference[144], which may influence lipoprotein levels. Although this possibility is supported by reports that WHR is related to adverse levels of lipids and lipoproteins, their findings proved primarily overweight persons[139, 140,141.]

Distribution of mean BMI showed that boys had higher BMI than girls. In both sexes BMI <20 was predominant than other category. In a given category, the percentage of

both sexes with a centripetal distribution more among higher school status. The prevalence of extreme leanness (BMI < 20) is greater among lower and middle status school children. But it was not true in the higher status school children.

In Canada prevalence of obesity (BMI ≥ 27) increased with age and was greater in men (35%) than women (27%). Abdominal obesity was likewise higher in men and increased with both age and BMI. Total cholesterol levels increased only with BMI, levels of low density lipoprotein (LDL) cholesterol and triglycerides and ratio of total cholesterol to high density lipoprotein (HDL) cholesterol increased steadily, while HDL cholesterol decreased consistently with increasing BMI .[274] It is partially true in our study those who were suffering from abnormal lipoprotein among higher school status children. Our result showed similar trend of BMI prevalence among boys and girls of higher status school children.

Among adolescents of urban Bangladesh, only 4.3% of males and 2.9% of females have a BMI greater than 26.87, which is equivalent to the 85th centile of the standard reference population and indicative of overweight. However, among urban adults, 12.1% of males and 24.1% of females have a BMI over 25.0 corresponding to the 85th centile of the standard reference population indicative of overweight. On the other hand, among urban adults, 27.4% of males and 25.5% of females have a BMI less than 18.5, indicative of underweight[382] but in this study we found low HDL-C among lower status school children who had BMI < 20.0 and a little higher BMI among middle status school children. Studies in UK reported highest prevalence of CHD among Asian Indians.[284] Multiple studies revealed that the usual risk factors like hypertension, hypercholesterolemia, obesity, smoking and a family history of CHD, were not common in South Asians. But they posses a different risk factor profile characterised by high triglycerides, low HDL-C, glucose intolerance, insulin

resistance, abdominal obesity and increased lipoprotein (a) levels.[284] Obesity is related to abnormal lipid and carbohydrate metabolism and is predictive of subsequent cardiovascular disease[127, 128.] However, the associations have often been fairly weak or have been observed only over long periods of follow-up[129, 130] and obesity may be a risk factor in only specific subgroups of overweight persons.[131] We observed the same from higher school status children. Much evidence suggests that high-risk obese individuals be characterised by their distribution of adipose tissue. Vague[132] was the first to document that the male (android) pattern of upper body obesity, characterised in part by a relatively thick skin fold at the nape of the neck, is more strongly related to increased insulin levels, diabetes, and arteriosclerosis than is the female (gynoid) pattern of lower body obesity. The development of obesity in childhood is a major determinant of the acquisition of cardiovascular risk later in life.[399-401]

Role of Food Habits and Cholesterol

Among our study children we found maximum consumption of calorie, carbohydrate, protein and fat intake by girl population. Mean calorie intake among girls was higher than boys, however, the difference was not statistically significant. Carbohydrate, Protein and Fat consumption were almost same between both populations (Table- III.2).

There has been a persistent concern that dietary cholesterol may have an atherogenic action independent of its effect to raise the fasting level of LDL. Zilversmit[11] first suggested the possibility that fasting lipoproteins containing dietary cholesterol were more atherogenic. More recently, evidence from animal model works and in vitro studies suggest that remnants of triglyceride - rich lipoproteins could be atherogenic. Several Investigators are still speculating that induction of cholesterol rich chylomicron remnants by dietary cholesterol may be an atherogenic factor that is

independent of fasting cholesterol levels.

Dietary cholesterol likewise may increase the cholesterol content of VLDL remnants or reduce their atherogenic potential as well. Although this concept does not see radical, the clinical implications may be profound. New Information about fasting lipoproteins might explain the epidemiological linkage between dietary cholesterol and CHD. In this study we have tried to identify lipid abnormality in school children and risk factors from a nested case-control design. In this study we found no significant association between dietary cholesterol and 7 days food intake, WHR, BMI, School status and other nutritional parameter among study children.

In Bangladesh few studies hypothesised that the higher prevalence of type IV hyperlipidemia amongst the IHD cases could be due to increased intake of carbohydrate in the foods.[286]

In Bangladesh one nutritional survey explain that basic food pattern is dominated by rice plus "trimmings" and data would suggest that ideally, in the people's attitudes, rice should constitute the bulk of total intake.[380] It was also observed that in our study results girls were consumed more carbohydrate containing foods than boys among higher status school (Table-III.3). The reason may be socio-cultural, hormonal influence is another confounder and in most of the cases girls of this age (10-12 years) spend more time in their home, thereby, they might consume more food with relatively less expenditure of energy.

Food Habits Among Children

Food habits are also important factors in the causation of coronary heart disease, consumption of diet rich in animal fat may cause an increase in serum total cholesterol, and so it is expected that a diet rich in animal fat will increase the risk of CHD. Cooking oil, such as soybean or mustard oil, is often adulterated in our markets,

and that might have contributed to trans-fatty acid formation thereby raising serum cholesterol. In this study school children from low socio-economic status are observed to have hyperlipedemia. But fat consumption among them was low (Table- III.4). So there are some confounders behind this abnormal lipoprotein. Triglyceride was more among one clinic based study, and incremental consumption of more carbohydrate in Bangladeshi culture.[286]

Samuel and McNamara[9] have reported that significant lumen-mucous exchange does not occur in humans, and uptake of radioactive cholesterol from the lumen release easily reflects the absorption of dietary cholesterol. He also examined that variability of cholesterol synthesis in mononuclear cells from patients loaded with an excess of dietary cholesterol. Most of this patient did not show a detectable rise in plasma cholesterol, and he concluded that such patients efficiently suppress cholesterol synthesis. About 20% of the patients did respond with higher plasma cholesterol levels, suggesting a lack of an efficient feedback mechanism.

Another puzzling feature in estimations of cholesterol absorption is that a constant fraction of cholesterol tracer appears to be absorbed regardless of the cholesterol intake. Since absorption of cholesterol is not complete, this finding is contrary to what might be expected, namely, that with increasing cholesterol intake, the percent absorption would decrease, as in other biological systems.

Dietary cholesterol is not absorbed completely, this amount to 25% to 75%, and there is considerable individual variation. Most available techniques use radioactive tracers, and some investigators doubt whether the rate of tracer uptake by intestinal mucosa accurately reflects absorption of cholesterol. They claimed that luminal cholesterol exchanges with mucosal cholesterol[10, 11] and thus the rate of disappearance of radioactivity from the lumen overestimates the mass absorption.

In spite of methodological problems, there is an ample observation that absorption is variable from one person to another. Therefore, it can be required whether fractional absorption of individuals affect their plasma level of cholesterol, In recent investigations, McNamara measured the percentage absorption of dietary cholesterol in a group of men with a range of plasma cholesterol levels and found no correlation between percentage absorption and plasma cholesterol concentration. But Miettinen and his co-workers[9] divided a group of middle-aged Finnish men into three subgroups those with high, moderate, or low levels of LDL cholesterol and estimated the percentage absorption of cholesterol in them. Those with the highest levels of LDL cholesterol had the highest percentage absorption of cholesterol, while those with the lowest levels had the lowest percentage absorption.

Whether an increment in absorption of cholesterol produces a feedback inhibition on the synthesis of cholesterol is yet to be resolved. Peter Edwards reviewed the regulation of cholesterol synthesis as influenced by the activity of several key enzymes. If feedback inhibition were to balance exactly the increment in absorption, the net change in body pools of cholesterol would be zero and dietary cholesterol would not raise the plasma cholesterol level. Increasing amounts of dietary cholesterol does raise the plasma cholesterol in some people indicates that the feedback system is not perfectly regulated. Feedback inhibition of cholesterol synthesis is not the only mechanism preventing hypercholesterolemia after high intakes of cholesterol. Quintao et al[12] demonstrated that a portion of newly absorbed cholesterol also resecreted into bile. This leads to an increase in biliary cholesterol and serves to rid the body of a fraction of excess dietary cholesterol. Another protective mechanism is increased conversion of cholesterol into bile acids. This serves to prevent hypercholesterolemia in cholesterol-fed rats and dogs, but in humans, enhanced

formation of bile acids in response to dietary cholesterol has not been consistently shown[12].

The average daily consumption among school children's was of less than recommended dose calcium, magnesium, copper and zinc (Table III.7,8). World Health Organisation (WHO) has recommended the daily dose of calcium would be 500mg/day. In USA it is 800mg/day for adults and in Australia 1000mg/day for postmenopausal women. Average calcium requirements were 180mg/day for adolescent growth it reaches 400mg/day. Some authors[361] narrated that calcium; magnesium, copper and zinc have long been suspected as potential risk factors for cardiovascular diseases. Although high blood pressure, smoking and dyslipidemia are major risk factors for atherosclerotic cardiovascular diseases they cannot fully explain variation in the incidence of the diseases and the thus a search for other risk factors continues. Minerals and trace elements have long been suspected as potential risk factors for cardiovascular diseases. Serum calcium has been shown positively correlated with several cardiovascular risk factors and survival of people with hypercalcemia is decreased particularly due to increased risk of cardiovascular death.[362] On the other hand deficiency of dietary calcium has been linked with elevated risk of hypertension.[363]

In ecological correlation studies hardness of drinking water is inversely related to cardiovascular mortality and it has been suggested that the probable protective 'water factor' is the high magnesium content of water,[364] although calcium has been implicated also.[365] Low serum magnesium is a risk factor for early death in people with acute myocardial infraction, but the impact of low serum magnesium on long-term prognosis is not well known.[366] The trace metals copper and zinc were of special

interest as contributors to cardiovascular risk. According to animal experiments low copper and high zinc content in the diet leads to hypercholesterolemia and cardiovascular complications.[367] In human studies, however, serum copper has been mostly with cardiovascular diseases, whereas serum zinc has been normal or decreased.[368-371]

Food rich in these micronutrients for example egg, milk and milk product consumptions are more among higher school status children who had high total cholesterol. Again it was found that Beef, chicken, mutton and fish consumption was more among those who had high total cholesterol (Table III.11).

In Table III.13 Among abnormal groups (TC-C>205mg/dL) it was found that only "other vegetable" were more consume by lower status school groups. But this was reverse in other two groups. In our study we found that increases total cholesterol (TC-C) groups consumed same amount of plant sources. In Bangladesh food consumption per day in rural and urban areas in 1995-96 data indicates that cereals accounted for the largest share of foods (62% in rural and 53% in urban areas). But non - leafy vegetables followed by roots and tubers were next in total amount consumed by rural people, and a little more than three-fifth of that consumed by urban people. Protein and other micronutrient rich food like fish, meat and milk and milk products, fat and oils and pulses, accounted for less than 10% of the food consumed by rural people versus 16% among urban residents.[382]

Nicklas et al.[106] found few significant correlations between dietary components and serum lipids; Glueck et al.[107] described some significant relations between [108]diet and lipid levels, but the relations were limited to age and sex sub-groups. Shea et al. found that children with higher intakes of total fat or saturated fat had significantly higher

levels of TC and LDL-C, but their sample size was small. In the past few years several intervention studies have shown that dietary modifications produce changes in blood lipid levels in children.[109-111]

It could be that the high fish intake of Japanese for example, acting via a pathway involving coagulation, may have protected them against the increase in CHD that would have been expected to follow the rise in plasma cholesterol.[66] However, studies in other populations provide estimates for potential risk reduction for consumption of fish that are short of the current difference between Japanese and American CHD mortality.[67] Plasma HDL cholesterol and total cholesterol were surveyed in representative populations of schoolchildren in Australia, Japan and US .The mean concentration of plasma HDL-cholesterol (But not total cholesterol) was higher for Japanese school children than for Australia or US schoolchildren ($p<.001$). [68]

High fat consumption from both animal and plant sources was observed in children with abnormal total cholesterol. The findings explain some link between food intake and abnormal lipids level among children.

High Density Lipoprotein

Several epidemiological studies have demonstrated that high-density lipoprotein cholesterol (HDL-C) is inversely related to the incidence of coronary heart disease morbidity and mortality[87-93]. Fewer studies have established a relation between HDL-C and specific coronary manifestations, including angina pectoris, coronary insufficiency, and myocardial infarction[88, 89,90.] Abnormal HDL-C was significant ($p<0.05$) among lower school status children's than that of higher and middle school status. This was also true for HDL-C level of normal subjects of lower school status children (Table-IV.10). In addition, some researchers report an uncertain or

questionable association[94, 95,96] while most fail to provide data for women.[89-96] From Framingham study report, it was observed that the effect of HDL-C as a determinant of the specific manifestation of myocardial infarction was shown for men and women aged 50 and older. The relation between high density lipoprotein cholesterol (HDL-C) and the development of myocardial infraction was examined and after 12 years of follow up, men in the bottom three quartile of HDL-C (≤ 52 mg/dl) experienced a 60% to 70% excess of myocardial infraction as compared to men those HDL-C levels were higher ($p<0.05$). The effect of HDL-C was strong in women.[97]

It is also interesting to note that boys and girls from the higher and middle socio-economic classes had very low levels of HDL–C.

In Netherlands it was evident that a low high density lipoprotein cholesterol level (HDL-C) is an independent risk factor for CHD.[279, 280] In our study we observed that the low level HDL-C was more among lower status school children but it was not similar in Netherlands.Evidence has accumulated that a low HDL-C is an independent risk factor for CHD.[278, 279] Coronary heart disease mortality in the Netherlands has declined during two decades, but it is still the most important cause of Death.[280]

In our country, some researchers have also found a lower level of HDL-C among middle and old age groups. Low levels of HDL-C have also been observed more frequently in South East Asian countries. In India multi-centre studies have revealed that the usual risk factors like hypertension, hypercholesterolemia, obesity, smoking and a family history of CHD, are not common in South Asians. But this posses a different risk factor profile characterised by high triglycerides, low level HDL, glucose intolerance, insulin resistance, abdominal obesity and increased lipoprotein (a) levels[285].

Coronary Risk Among Study population

Coronary heart disease (CHD) risk prediction based on serum lipid levels total "Cholesterol/HDL ratio" is well known. This figure if less than 4.5 indicates remote change of developing CHD.[419] Distribution of TC-C/HDL-C ratio among study children was found desirable (<4.5) level in 56% of children and borderline to undesirable level (>4.5-5.5) in 43.2%. Undesirable limit was more among lower school status than other two groups (16.4%). An undesirable level of LDL-C/HDL-C ratio was found more often among lower status children than the other two school categories, which is one of the surprising findings of our study (Table-IV.10). It is necessary to identify how much of our population is suffering from undesirable levels of (>5) of TC-C/HDL-C or this ratio.

Several studies have indicated that the LDL/HDL ratio gives an independent prediction of the risk for coronary disease. A close correlation between the extent of pre-atherosclerotic lesion with serum LDL and total cholesterol concentrations in children as well as in adults have been shown in several studies.[2, 3]

Table IV.11 high levels LDL/HDL – C ratio was found among lower and higher status school children. Total ratio among undesirable (>3) limit of LDL/HDL cholesterol ratio was 15.6%. It was also found that lower status children more often had undesirable levels of LDL/HDL cholesterol ratio compared to the other two groups of children. So, it needs further investigation for long-term follow-up of these children to know the risk determinant of coronary heart disease and other pathophysiologic abnormality in our community. Little work has been reported on the association between physical activity and LDL-C concentration during late childhood. Using a 7-day activity recall, Sallis et al[343] reported a significant direct association between physical activity and the HDL-C -to-LDL-C ratio but did not report on the

association with LDL-C levels.

Physical Activity and Prediction of CHD

Daily physical activity among middle status schoolboys and girls was higher than that of two other groups (Table-IV.13). Three cross-sectional studies[349-351] reported that a direct association exist between physical activity and HDL-C concentration. Two studies reported no effect.[347,348] The results of intervention studies are highly variable. Reports have documented a direct association,[352] no association,[353] and an inverse association[354] between physical activity and HDL-C concentration. Physical activities found among hypercholesterolaemic groups were low in lower status school children that are significantly associated with low HDL cholesterol. Linder and his associate explain that one year higher levels of physical activity and physical fitness and the lower levels of fatness would be associated with higher levels of high density lipoprotein and of the subclass HDL2 and with lower levels of total serum cholesterol, triglycerides, and low density lipoprotein and lower LDL/HDL and TSC/HDL ratios. [118, 122,87] Coronary heart disease, a leading cause of morbidity and mortality among adults in the United States,[342] should begin during childhood.[343] Risk factors for coronary heart disease include an elevated low-density lipoprotein cholesterol concentration, a reduced high-density lipoprotein cholesterol concentration, and an elevated blood pressure. Therefore, appropriate targets for prevention of coronary heart disease include lipoproteins and blood pressure.[343] These factors may be affected by physical activity.[344] Among adults, physical activity has been directly associated with HDL-C concentration[345] and inversely associated with blood pressure.[346] In contrast, among children, associations between physical activity and

HDL-C concentration and physical activity and blood pressure are variable.[347-356]

In the present study, we investigate only lipid profile among selected school and pattern of hyperlipaedemia among the paediatric population; the result of physical activity and HDL-C in different school status gives different results. We observed some relation between physical activity and HDL-C, but it needs further thoughtful idea among the subjects and their daily physical activity.

Risk of Total Cholesterol, HDL-C and TG-C

However, Median of TC-C, HDL-C, LDL-C and TG-C among higher and lower school status children were increasing trend than middle status children. But HDL-C among lower status school population was found low than that of higher and middle status school children. Serum TG level was same between students of diverse status (Table- IV. 24).

A close findings than that of Muscatine Coronary Risk Factor Survey, where selected three group of cases for detailed family study, the high group with cholesterol levels greater than the 95th percentile twice; the middle group cholesterol levels between the 5th and 95the percentile; and the low group cholesterol levels less than the 5th percentile twice.

Coronary mortality determined from death certificates was increased in the young relatives (ages 30-59) of the high group index cases, as follows: twofold excess in high male relatives compared with the Middle or low group (p less than 0.05); tenfold excess in the high female relatives compared with the middle and low group combined (p less than 0.01) correction for years at risk, there was an approximately twofold significantly increased coronary mortality.

The said study indicates that children's cholesterol levels cluster with those of their family members and that persistent hypercholesterolemia in children identifies

families at risk for coronary artery disease.[72] Cholesterol levels above the 75th percentile should be considered hypercholesterlemic and at risk for future adult heart disease. Children with total Cholesterol levels below 170mg/dL require no intervention other than that recommended for the general population, and should be re-evaluated in 5 years.

Report from expert panel on blood cholesterol levels in children and adolescents was total cholesterol <170 mg/dL and LDL-C <110 acceptable, 170-199 and 110-129 borderline and ≥200 and ≥ 130 mg/dL high in case of children and adolescents from families with hyper cholesterol or premature cardiovascular disease. [74]

In our study the cut-off value of lipoprotein profile was not similar than that of Muscatine Coronary Risk Factor Survey and the design was totally different from our study.

Critical Analysis Between School Children

Delineation of lipid values in children after Kawasaki syndrome is important because of the predelineation of this disease for coronary arteries. In this study they observed that mean high-density lipoprotein cholesterol after clinical recovery tended to be lower in patients with persistent coronary abnormalities than in those without such lesions (p=0.085). Kawasaki syndrome is associated with important abnormalities in lipid metabolism.[379]

In Bangladesh one case-control study[378] results reported that in Acute Rheumatic Fever (ARF) with carditis group, total cholesterol, HDL and LDL level were significantly low but triglyceride level was higher values in comparison to control group. It was also true among HDL/TG ratio, which was significantly low in ARF with carditis groups.

To identify variables independently associated with total cholesterol, HDL-C, LDL-C, and TG-C after simultaneous adjusting for confounders or variables thought to be of importance from biomedical point of view, we performed multivariate analyses. Thus four different models were constructed keeping independent variables (sex, physical activity, waist-hip ratio, school status, and BCG and polio immunisation constant. Students of higher school-status were found to be four times more likely to have higher HDL-C than children of lower and middle-status school children (OR 4.3, 95% CI 1.5-13.0, and p=0.00). Children who received BCG immunisation had 5 times higher risk against non-immunised children (OR 4.7, 95% CI 1.3-17.3, and p=0.02). However, children with history of polio immunisation had 89% reduced risk of HDL-C than those who were not immunised after controlling for potential confounding variables (Table IV.18, 19, 20, 21).

CHAPTER – 6

SUMMARY AND CONCLUSION

Chapter- 6

Summary and Conclusion

The lipid abnormalities start at early life, give many cardiovascular fatal disease in adulthood. The early detection helps in early initiation of intervention and succeeds in arresting the progression of cardiovascular diseases (CVD) and ultimately reduces the occurrences of CVD risk among adults. This study was conducted among a groups of school children to predict the lipids abnormality in our children, which will help in the prevention of future development of atherosclerotic disease.

The lipid profile among children of 10-12 years and identify lipids and other determinants of potential ischaemic heart disease in children of Dhaka was studied. A total of 392 children from three schools were selected. The children were clinically examined and, laboratory tests were carried out. The study children were selected by multistage sampling. The three pre-selected areas was prepared and selected and, these were higher, middle and lower socio-economic school status population. The sample from each of the school was selected randomly. The variables were age, sex, religion, school status (family size, parental education and their occupation, family income, and housing condition), nutritional status (weight, height, mid arm circumference, triceps skin fold thickness, waist-hip ratio). Consecutive seven days food intake, and average daily physical activity also included in our study. Dependant variables were serum Total cholesterol (TC-C), Low-density Cholesterol (LDL-C),High-density cholesterol (HDL-C), and Triglyceride cholesterol (TG-C). Serum was tested for the determination of total cholesterol, LDL-C, HDL-C and TG-C by colorimetric method. All the tests were performed at the clinical biochemistry laboratory with commercially available kits (Boehringer Mannheim testkit, Germany).

Among the study population the prevalence rate of abnormal total cholesterol 24 (6.12%), low level of HDL-C 43 (10.95%), high LDL-C 26 (6.63%) and high level of TG-C 36 (9.18%).Plasma high-density lipoprotein (HDL) cholesterol concentration was found to be significantly ($p<0.05$) lower among lower status school children than high and middle status school children. High total cholesterol (>205 mg/dl) and low HDL (<37 mg/dl) was also detected among lower status school children of Dhaka city. In this finding among study population it was found that desirable level was 76(56%) and undesirable level of Total Cholesterol/ HDL cholesterol ratio 58(44%). It was also found that among lower status school children were more undesirable level of LDL/HDL cholesterol than two other group. In the study population, children who had below 60 minutes/day physical activity, majority were suffering from abnormal HDL–C (<37m/dL) in the study period. None were suffering from abnormal HDL-C with <-2.0 SD (stunting) among middle status school children in our findings. Waist-hip ratio among abnormal cholesterol in middle status schoolboys was more than higher and lower status schoolboys. It was only true for girls of higher status schools. Children we found ≥ 25 Body Mass Index (BMI) among boys 2.1% and girls 2.7% respectively. In a given category, the percentage of both sexes with a centripetal distribution of fat due to increased with age. Polio vaccine immunised group were less likely to be developed abnormal HDL cholesterol in our study finding. BCG vaccine immunised group have higher risk from abnormal HDL cholesterol than those who were not immunised. Percentiles distribution of lipid profile among different school children Median among higher and lower school status group and were increasing trend than middle status children. But HDL-C of lower status

school children was lower than that of higher and middle. Serum TG-C level was same in students of diverse status. Undesirable lipids ratios among underprivileged children due to higher consumption of carbohydrate and fat. Over and above they don't have facilities for physical exercise because of cultural reasons and inadequate system in school authority concern. Living in poverty situation those children who consumed high carbohydrate and low fat and protein and little exercise showed low level of mean HDL-C.

This research findings might help in formulating appropriate programs for control of IHD. Based on the correlation test, regression analysis, chi-square test, odds ratio, 95% confidence interval and likelihood ratio, it may be concluded that measurement of lipoprotein profile by standard laboratory test is still better measurement for community based study as well as clinic based study.

Increased BMI and abdominal obesity among school children may be responsible for the increasing trend for abnormal lipids in future. Genetic cause might be another important reasons for abnormal lipids between different school status children. This study also support that food habit, physical activity, socio cultural and family history may generate hypothesis for future studies for early diagnosis of abnormal serum lipids among paediatric population.

CHAPTER – 7

LIMITATION AND FURTHER RESEARCH

Chapter -7

Limitation and Further Research

The study was conducted in a group of school student of Dhaka city. There was constrain both internal and external for collection and presentation of blood samples for further analysis. Measurement of Apo A-1 and Apo B could be more informative for our population. Unfortunately we could not measure them. We could go for other parameter of lipoproteins, which was essential for exploring genetic factors. It was not possible to measure subscapular skin fold thickness, even some of the girls did not allow to measure other related variables due to socio-religious reasons. In our study, we used a food frequency questionnaires (FFQ) for a week by a structured food list table. We could not make a quantitative assessment due to lack of trained manpower and time. Additionally, the validity of FFQ was not tested. This limitation also did not allow the researcher to compare the situation of potential predictors of ischaemic heart disease, more accurately, among children of survey area.

Blood cholesterol levels are determined by both environmental and genetic factors, such as apolipoprotein genes.[393] A quantitative dietary assessment and analysis of genetic factors could give us an insight into the lipid abnormalities specific to our population. The cloning of apoA-1 has made it possible to apply restriction fragment length polymorphism analysis[83] of the human gene in normal individuals. In the other hand monoclonal antibodies against LDLs were used extensively in research on the immunochemical heterogeneity apo-lipoprotein (apo) B containing lipoproteins, the development for techniques for quantitative and qualitative assays of plasma LDL content, and analysis of the sites in the protein moiety of LDLs involved in its recognition by cell receptors. The facilities for DNA analysis in our laboratories should be developed for advance research in our country and our centre.

Hyperlipidaemic children should be observed for long term to find out appropriate measures to control such abnormalities in our children. Present study was done in urban children. So, distribution of lipids in rural children should be examined to get more representative results of Bangladesh at large.

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ANNEXURE

Annex-1

Bangladesh Medical Research Council

Mohakhali,Dhaka-1212, Bangladesh
Tel: 871395

Application For Ethical Clearance

1. Principal Investigator (s):

2. Co-Investigator (s):

3. Place of the Study/Institute (s):

4. Title of Study:

5. Type of Study:

6. Duration:

7. Total Cost:

■ Check documents being submitted herewith to committee:

- ❒ Umbrella Proposal
- ❒ Proposal Summary
- ❒ Abstract for Ethical Review Committee as per attachment(Obligatory)

- ❒ Informed Consent form for Subjects
- ❒ Verbal Consent Form for Subjects
- ❒ Procedure for maintaining confidentiality
- ❒ Questionnaire or interview Schedule

1. A description of the areas to be covered in the questionnaire or interview which could be considered either sensitive or be asked in the sensitive or which would constitute an invasion of privacy.

2. Examples of the type of specific question to be asked in the sensitive areas.

3. An indication as to whom the questionnaire will be presented to the committee for review.

We agree to obtain approval of the Ethical Review Committee for any change involving the rights and welfare of subjects or any changes of Methodology before making any such change.

Principal Investigator/Leader/ Coordinator

Other Investigators

Annex-2

Consent Form for Subjects (+ Bengali translations)

সম্মতি পত্র

"বাতজ্বর ও বাতজ্বরজনিত হৃদরোগ পরীক্ষার জন্য ছেলে মেয়েদের গলার লালা ও রক্ত পরীক্ষা অত্যন্ত প্রয়োজন। আপনার অনুমতি পেলে আমরা আপনার ছেলে/মেয়েদের উক্ত পরীক্ষা নির্দিষ্ট দিবসে শুরু করিব"।

এ ব্যাপারে আমি সম্মতি প্রদান করছি। এ ব্যাপারে যদি কোন অসুবিধা দেখা দেয় তবে ডাক্তারগন যথাযথ চিকিৎসা প্রদান করিবেন।

স্বাক্ষর :----------

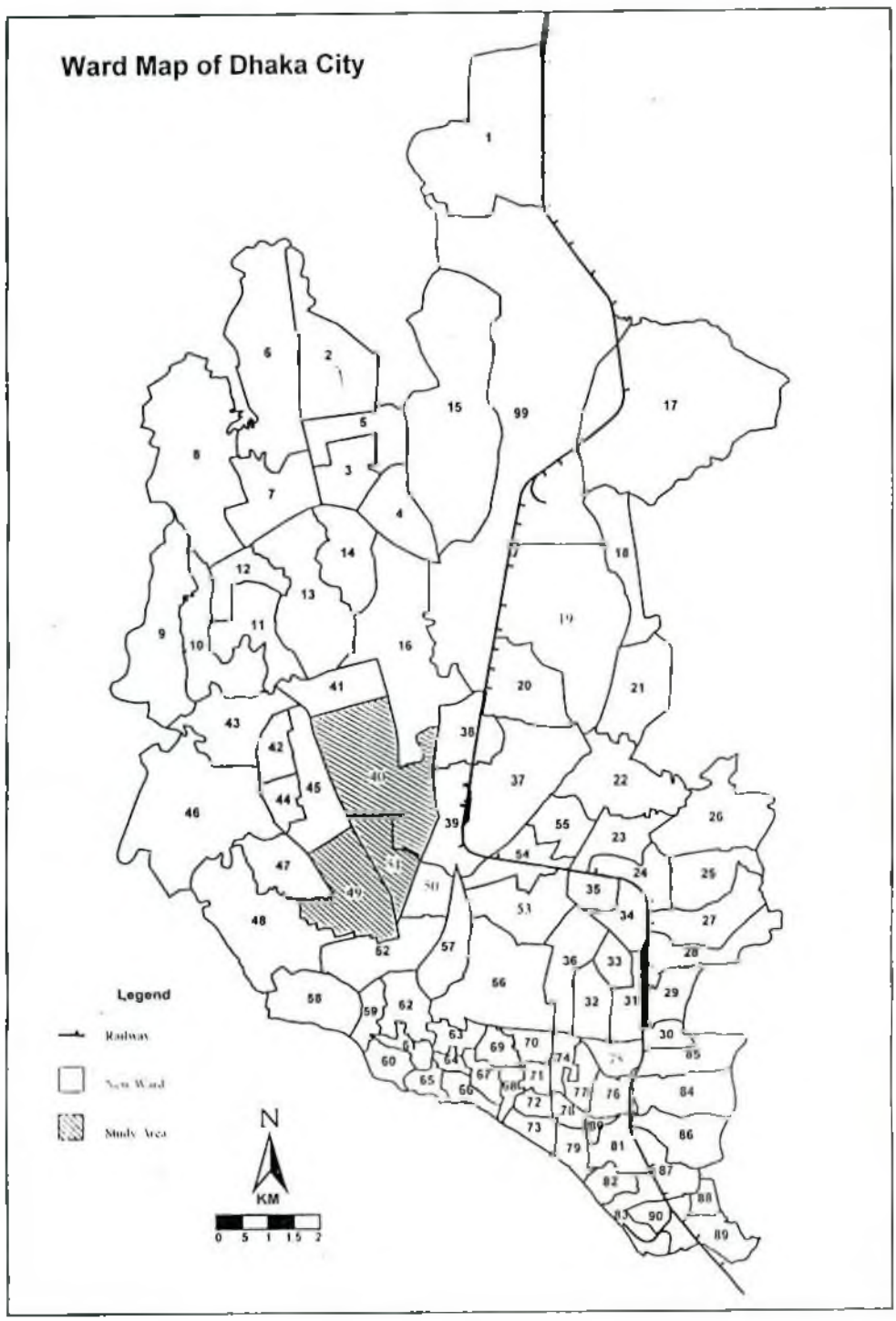
পুরানাম :----------

ঠিকানা : ----------

ফোন নম্বর :----------

Annex-3

Ward Map of Dhaka City



Annex -4

Tables

Table 2.4: CHD Mortality rate in US, Australia and Japan 1992

Sex	United States	Australia	Japan
Men	161.80*	143.23	26.76
Women	63.22	53.63	9.93

* Death rates per 100 000 age-adjusted to the World standard population age 25-74 years.

Table 2.8: Study variables for boys and girls

Variable	Boys	Girls	P value
BMI(kg/m^2)	26 ± 2.6*	25.5 ± 3.2	$p = 0.003$
BMI(kg/m^2)mother	27.9 ± 5.9	27.6 ± 5.8	ns
BMI(kg/m^2)father	26.0 ± 3.0	27.2 ± 3.6	ns
Total cholesterol(mmol/l)	4.5 ± 0.7	4.4 ± 0.7	ns
HDL-C(mmol/l)	1.1 ± 0.2	1.2 ± 0.2	$p = 0.001$
TG-C(mmol/l)	1.2 ± 0.1	0.9 ± 0.4	ns
WHR	0.92 ± 0.07	0.81 ± 0.08	$p = 0.0001$

* Data are mean ± SD. Statistical differences were evaluated by Mann-Whitny U – test.

Table 2.9: Describtive (Limited table shown) features of cholesterol studies in Japanese children.

Study number	Reference number	Study year	Study location; prefecture	Other serum lipid measurments[a]
9b[d]	27	1977	Iwate	- -
10	28	1982	Osaka	TG HDL-C
5	21	1984-1985	Nara	TG HDL-C
15[b]	33	1990[c]	Osaka	TG HDL-C
26	44	1992	Tokyo	- HDL-C

[a] HDL-C = high density lipoprotein cholesterol; TG = triglyceride. Parentheses means age-and sex-specific numerical mean values and/or numbers of participants are not available in at least three age groups.

[b] Studies 7 and 15 are presumed to have determined lipid concentrations in serum.

[d] In study 9, the information for 6-9 years of age is from study 9a and that for 10-17 years is from study 9b.

Table 2.10: Intake of foods in relation to risk of CHD among 85,095 women*

Foods	Relative risk (95% CI)						
	<1-per month	1-4per month	2-4 per wk	5-6 per wk to 1 per day	2-3 per day	> 4 per day	P (trend)
Margarine	1-0	0.83(0.50-	1.03(0.71-	1.07(0.80-1.42)	1.03(0.77-	1.66(1.10-	0.02
(teaspoon; 5ml)	1-0	1.38)	1.50)	1.22(0.61-2.43)	1.38)	2.49)	0.86
Beef, pork,	1-0	1.16(0.59-	0.95(0.48-	1.49(1.09-2.04)	-----	---	0.007
lamb	1-0	2.26)	1.86)	1.26(0.96-1.66)	1.55(1.02-	---	0.003
(main-dish)		1.05(0.81-	1.02(0.75-		2.34)	---	
Cookie (1)		1.36)	1.39)		1.43(1.08-		
White-bread		0.85(0.59-	1.07(0.78-		1.90)		
(1slice)		1.24)	1.47)				

* From separate proportional hazards models, controlling for age, time, body mass index, alcohol intake, smoking, menopausal hormone use, family history of myocardial infarction before age 60 yr, history of hypertension, multivitamin use.

Table 2.11: Mean Serum low-density lipoprotein cholesterol concentration (mmol/l) in men aged 59-70 according to weight at 1 year, birth weight, and whether weaned at 1 year.

	Weight at 1 year(lb)			
Birth weight (lb)	≤21	-21	>23	All
Weaned:				
≤7	4.8(45)	4.8(29)	4.6(30)	4.8(84)
-8.25	4.8(41)	4.5(47)	4.5(39)	4.6(127)
>8.25	4.8(22)	4.6(45)	4.5(64)	4.6(131)
All*	4.8(108)	4.6(121)	4.5(113)	4.6(342)
Not weaned:				
≤7	4.8(18)	4.1(7)	3.9 (1)	4.6(26)
-8.25	4.9(13)	4.9(12)	5.1(10)	4.9(35)
>8.25	5.7 (8)	5.3(9)	5.2(12)	5.4(29)
All †	5.0(39)	4.8(28)	5.1(23)	5.0(90)

* Overall standard deviation =1.1

† Overall standard deviation =1.2

Table 2.12.1a,1b: Distribution of subjects by Total plasma cholesterol and HDL-C level by sex and age

Sex; age, yr	Total plasma cholesterol levels mmol/L: % of subjects		
	<5.2	5.2-6.1	>6.2
Men			
18-24	86	11	3
25-34	66	26	8
35-44	42	36	22
45-54	32	32	33
55-64	39	36	25
65-74	39	40	25
All	52	30	18
Women			
18-24	82	14	4
25-34	77	18	5
35-44	69	23	8
45-54	40	43	17
55-64	20	43	38
65-74	20	36	44
All	57	27	16
Total	55	29	17

Sex; age, yr	HDL-cholesterol level; mmol/L: % of subjects		
	<0.9	0.9-1.9	≥1.3
Men			
18-24	9	56	35
25-34	11	56	33
35-44	15	53	32
45-54	15	60	25
55-64	15	50	35
65-74	15	55	30
All	13	55	32
Women			
18-24	3	41	56
25-34	4	33	63
35-44	4	35	61
45-54	4	33	64
55-64	3	37	60
65-74	4	40	55
All	4	36	60
Total	8	45	46

Table-2.12.1c, 1d : Distribution of subjects by LDL-C and TG-C level by sex and age.

Sex ; age, yr	LDL-cholesterol level; mmol/L: % of subjects		
	<3.4	3.4-4.0	≥4.1
Men			
18-24	88	10	3
25-34	70	21	10
35-44	53	27	20
45-54	43	31	26
55-64	47	32	21
65-74	45	29	26
All	60	24	16
Women			
18-24	87	10	3
25-34	83	12	5
35-44	76	17	7
45-54	58	25	17
55-64	39	28	33
65-74	32	28	40
All	67	18	14
Total	**63**	**21**	**15**

Sex ; age, yr	TG-cholesterol level; mmol/L: % of subjects	
	<2.3	≥2.3
Men		
18-24	91	9
25-34	88	12
35-44	78	22
45-54	68	32
55-64	74	26
65-74	79	21
All	80	20
Women		
18-24	95	5
25-34	95	5
35-44	93	7
45-54	87	13
55-64	78	22
65-74	79	21
All	89	11
Total	**85**	**15**

Table2.13: Mean, Standard deviation (SD) and prevalence of high-risk levels of total and HDL-cholesterol in men and women aged 20-59 by 10 years age strata. Monitoring project on CVD risk factors, The Netherlands, 1987-1992.

	MEN				WOMEN			
	20-29	30-39	40-49	50-59	20-29	30-39	40-49	50-59
No.	3531	5110	5622	5405	4070	5687	6062	6135
Total cholesterol mean (mmol/l)*	4.75	5.35	5.82	5.98	4.87	5.03	5.50	6.23
SD(mmol/l	0.94	1.06	1.10	1.06	0.88	0.91	0.99	1.13
%≥6.5mmol/L	4.5	13.6	24.1	29.2	4.2	6.2	14.5	37.7
HDL-C								
Mean(mmol/l)	1.14	1.12	1.11	1.09	1.38	1.36	1.39	1.36
SD(mmol/l	0.25	0.26	0.28	0.28	0.30	0.31	0.33	0.34
%	15.1	20.3	22.2	25.6	3.8	4.8	5.1	7.1
%≥0.9mmol/L								

*1 mmol/l= 38.7mg/dl.

Table 2.14: Partial correlation cofficients (r) among serum lipids adjusting for ethnic group,age,body mass index, alcohol consumption and physical activity, both sexes aged 18-69 years.

Relationship	Males No	(p)	r	Females No	(p)	r
LDL-C and HDL-C	1051	0.00	(0.907)	1038	-0.08	(0.010)
LDL-C and fasting triglyceride	971	0.03	(0.386)	993	0.11	(<0.001)
HDL-C and fasting triglyceride	971	-0.34	(<0.001)	993	-0.26	(<0.001)

LDL-C – low density lipoprotein cholesterol. HDL-C = High density lipoprotein cholesterol.

Table 2.15: Mean serum total cholesterol concentrations(mmol/l) by region and gender

Age (years)	No[a]	Killimanjaro Mean	SD	No	Morogoro Mean	SD	No	Mara Mean	SD
Men	1472	4.2	1.2	1239	3.4	1.0	430	3.7	0.9
Women	2271	4.4	1.2	1317	3.6	1.0	543	3.9	0.9

[a] Age –adjusted.

Table 2.23.1a: In 1975-76 study report of Energy intake in Kcal by activity

Age(10-12year) and Sex	*Light active/ Kcal.*	*Moderately active/ Kcal.*	*Very active/ Kcal*
Boys	2340	2600	3042
Girls	2115	2350	2750

Source: Nutrition survey 1975-76. INFS (DU).

Table 2.23.1b: In 1981-82 study report of Energy intake in Kcal by activity

Age(10-12years) And Sex	*Light active*	*Moderately active*	*Very active*
Boys	2579	2463	2511
Girls	2286	2234	2233

Source: Nutrition survey 1981-82.INFS(DU).

Table 2.23.1c : Daily per capita food intake in rural and urban areas of Bangladesh in 1995-96.*

Food group(gm)	Rural	Urban	National
Cereal	452.0	379.0	436.0
Roots and tubers	70.0	77.0	72.0
Pulses	10.0	16.0	11.0
Leafy vegetables	21.0	31.0	23.0
Non Leafy vegetable	92.0	77.0	89.0
Fruits	13.0	18.0	14.0
Meat	6.0	21.0	9.0
Fish	32.0	37.0	33.0
Milk and milk products	13.0	20.0	15.0
Eggs	3.0	7.0	4.0
Fats and oil	6.0	13.0	8.0
Sugar	6.0	9.0	7.0
Others	6.8	8.0	7.1
Total(gm)	730.8	713.0	728.1

***Source: Nutrion survey 1995-96. INFS (DU) .**

Annex-5

Lipid Profile Study Questionnaire

Serial No. :_____ ID No. : _____

Interviewer's name :_____Date of interview:_____

Name of School :_____

Section I: Personal History

Name : _____ Father's Name : _____

Address :_____

1. Age (year): 10 yrs =1, 11 yrs=2, 12 yrs=3 ☐
2. Gender : (1=Girl, 2=Boy) ☐
3. Religion: Muslim=1, Hindu=2, Christian=3 other(specify)=7 ☐
4. BCG: (1=Yes, 2=No, 3= Don't know, 9= N/A) ☐
5. DPT: (1=Yes, 2=No, 3= Don't know, 9= N/A) ☐
6. Polio: (1=Yes, 2=No, 3= Don't know, 9= N/A) ☐
7. Physical Activities per day (hour minute) ☐☐ ☐☐

SECTION II: SOCIO-ECONOMIC STATUS

8. Number of Family Member ☐☐
9. < 5 years ☐
10. > = 5 years ☐
11. Educational status of Mother : < SSC =1, SSC-HSC=2, Graduate=3, Post graduate=4 No education=5 ☐
12. Educational status of Father: < SSC =1, SSC-HSC=2, Graduate=3, Post graduate=4 No education=5 ☐
13. Occupation of Mother 1=Housewife, 2=Service, Others=7 (specify) ☐
14. Occupation of Father : 1=Service, 2=Business, Others=7 (specify) ☐
15. Approx. Monthly Family Income (Taka) ☐

15. Approx. Monthly Family Income (Taka) ☐
    1=1000-3000, 2=3001-5000, 3=5001-10000
    4= >1000

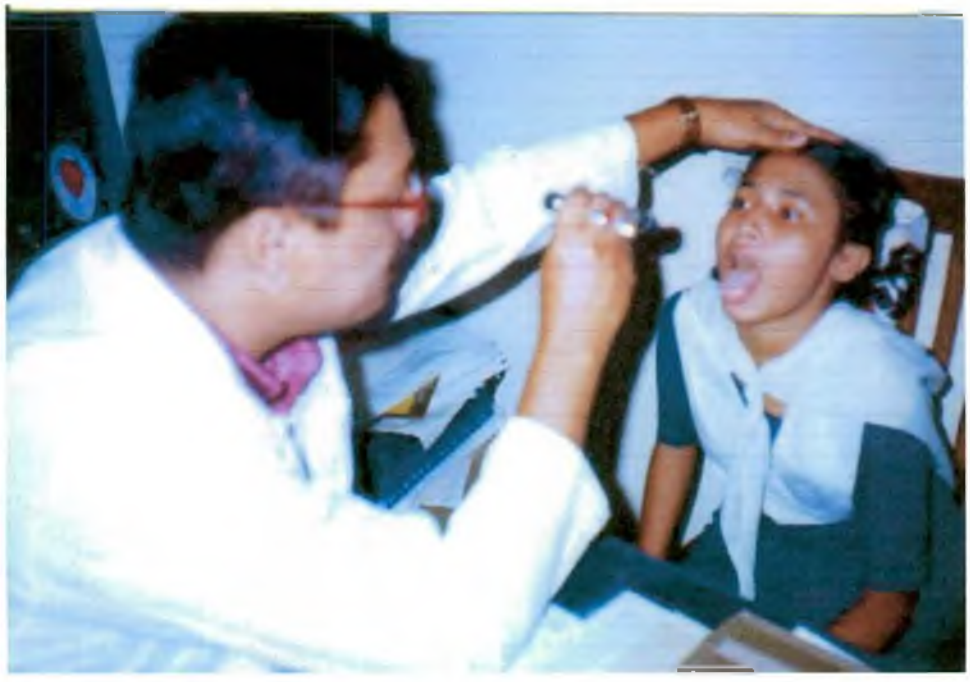
History of Parents

16. Mother : ☐
    1= Hypertension, 2= Diabetics, 3=Goitre, 4= Kidney diseases
    5=IHD
17. Father: ☐
    1= Hypertension, 2= Diabetics, 3=Goitre, 4= Kidney diseases
    5=IHD
18. Floor structure: ☐
    1=Cemented, 2=Non Cemented
19. Wall structure : ☐
    1=Brick, 2 =Bamboo fence, 3= Ordinary tin
    4=Courugated tin, 5=Straw, 6= Jute stick,
    7=Mixed, 8=Mud, 9=Other
20. Roof structure : ☐
    1=Concrete/Pucca, 2=Bamboo fence, 3= Ordinary tin
    4=Courugated tin, 5=Straw, 6= Polythin,
    7=Mixed, 8=Other
21. Suffering from any disease during last 5 years :
    a. Father ☐
    b. Mother ☐
    1= Hypertension, 2= Diabetics, 3=Goitre, 4= Kidney diseases,
    5=IHD 6=Other(specify)

SECTION III: ANTHROPOMETRIC MEASUREMENTS

22. Height (cm) ☐☐☐☐☐
23. Weight (kg) ☐☐☐☐☐
24. MAC (cm) ☐☐☐☐
25. Triceps Skinfold thickness (mm) ☐☐☐☐
26. Subscapular Skinfold thickness (mm) ☐☐☐☐
27. Waist (cm) ☐☐☐☐
28. Hip (cm) ☐☐☐☐
29. Ratio (Waist : Hip) ☐☐☐☐

Photograph 1: Principal investigator examining a school girl.



Photograph 2: Principal investigator, Statistical Officer and Health assistant.

Photograph 3: Members of the laboratory research team with Principal investigator

Photograph 4: Principal investigator, Sr. laboratory technicians and clinical pathologist in the National Centre for Control of Rheumatic Fever and Heart Disease (NCCRFHD)

এক সপ্তাহের খাদ্য তালিকা:

Annex -6

রোগীর নাম :

কে লিখেছেন :

তারিখ	দৈনিক খাদ্য গ্রহণ	ডিম	দুধ	দুগ্ধজাত খাদ্য	গরুর মাংশ	খাসির মাংশ	মুরগীর মাংশ	মাছ	ডাল	ফল	ভাত	সালাদ	রুটি	পাতাযুক্ত শাকসব্জি	অন্যান্য সব্জি	রান্নার ব্যবহৃত তৈল	মন্তব্য

জমা দেওয়ার তারিখ :

জমা গ্রহন কারীর স্বাক্ষর :

Annex -7

BIO-CHEMICAL REPORT

Date of collection : /____/____/____/

Patient' ID No. : _____ Laboratory No. : _____

Name of the Patients : _____ Age(yr) _____ Sex : _____

Laboratory Results :

TC-C : _____ mg/dL

LDL-C : _____ mg/dL

HDL-C : _____ mg/dL

TG-C : _____ mg/dL

OTHER PARAMETERS :

ASO Titre : _____ IU

C-reactive protein : _____

Throat swab culture (BHS) : _____ Group

Others(Specify) : _____

Lab. Comments :

_____ _____

Lab. Technician Lab. Head