A STUDY ON EPIDEMIOLOGY OF CHILDHOOD OBESITY IN DHAKA CITY

PH.D. THESIS

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ABSTRACT

Obesity is a form of malnutrition - a detriment to good health and well being. Obesity amongst children is increasing worldwide at an alarming rate in both developed and developing countries. Obese children are at higher risk of developing coronary heart disease, non-insulin dependent diabetes, respiratory disease etc. The extent of the problem is still unknown in Bangladesh, as there is no such epidemiological data on childhood obesity.

A case-control study, preceded by cross sectional survey was conducted with defined objectives. A multi stage probability proportionate to size (PPS) cluster sampling method was used to obtain the sample. To identify the obese children, a pre-tested questionnaire was used to collect data on age, weight and height among the randomly selected 5000 children of 2-10 years age group from 12 government primary & 23 private elementary schools, 4 hospitals, 8 health centers and 12 immunization centers (on National Immunization Days) from all the 12 thanas (civil administrative sub-districts) of Dhaka city. Survey included a medical history and physical examination to assess the eligibility of the subjects for the study.

Of 5000 children, 380 (7.6%) were identified as obese using the criterion of weight for height >120% as a cut-off point. Obesity was positively correlated with the increase of age in both sexes (r = 0.76). Of all obese children, 216 (56.8%) were boys and 164 (43.2%) were girls. Prevalence of obesity was significantly higher among the boys than girls (P=0. 007).

The study was conducted among the 220 cases of 380 obese children (Wt/Ht > 120%) and 220 randomly selected controls matching age and sex- using a semi-structured questionnaire for identifying the factors associated with the development of childhood obesity. Information also collected from parents of both cases and controls.

Results show childhood obesity was not associated with family structure, family size and birth order. However, family income (P<0.001) and expenditure on food

(P<0.001) were significantly higher among the cases. Data showed that parental obesity was significantly associated with the obesity in children. There was an association between obesity of the children and parents' educational status (P <0.001). There was no difference in the working hours of parents outside households between the cases and controls.

Mean energy intake (2056±751 kcal/d) was found to be significantly higher among the cases (P< 0.001) compared to controls (1508 ± 529 kcal/d). In addition, mean total energy expenditure among the cases was significantly higher than the controls (P<0.001). However, energy balance was significantly higher among the cases. Dose response of energy balance shows, the estimated relative risk of obesity increases with higher levels of energy balance upto a maximum of odds ratio 3.41 (P<0.001). A significant difference (P<0.001) was found in hours of television-video viewing between the cases and controls.

Body composition of both cases and controls were measured using Bioelectrical Impedance Analyzer (BIA) and in 4 sites (biceps, triceps, subscapular and suprailiac) skinfold thickness. It was found that there was a significant difference (P<0.001) in body fat percentage using these two methods.

Parental and child lipid profiles (Triglycerides, Total Cholesterol, Cholesterol-HDL and Cholesterol-LDL) were measured. Significant correlations were found between parental and child lipid profiles. Triglyceride level was significantly higher (P < 0.001) among the cases than controls.

Findings of this study show that the obesity among the children is caused by a positive energy balance over a considerable period, is related to environmental factors including energy intake, energy expenditure and other behavioral aspect. Appropriate interventions like behavioral change regarding energy intake and physical activity are thus recommended to address the childhood obesity- an emerging public health problem in Dhaka city.

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CHAPTER I

INTRODUCTION

INTRODUCTION

Obesity is a form of malnutrition characterized by excessive fatness in the body - a detriment to good health and well being (1,2). In objective terms, obesity can be defined using cut-off points either relative weight index 120% or above the standard weight for height (1,3-7), age and sex specific skin-fold thickness equal or more than 85th percentile (8) or Body Mass Index (BMI) above 30 for adults both male and female (9).

Obesity is not a recent phenomenon. Its historical roots can be traced to the Paleolithic era more than 25000 years ago. Clinical evidence of obesity can be dated as far back as Graeco-Roman times, but little scientific progress was made towards understanding the condition until the 20th century. Throughout most of the human history, weight gain and fat storage have been viewed as signs of health and prosperity (1,9,10).

Obesity may be classified as hypertrophic or hyperblastic or combined on the basis of cell differentiation. According to fat distribution, it may be either generalized or localized. Depending upon onset, it may be classified as childhood onset, adolescent or adult onset. It may be genetic or environmental or combined or may be due to endocrine disorder (11-14).

Obesity is one of the most complex and poorly understood diseases. It is affecting children as well as adults, which has shown increase prevalence, even in those countries where deficiency diseases represent a severe public health problem (9, 15,16). Obesity amongst children has reached epidemic proportions globally, and all these evidences suggest that the situation is likely to get worse in near future (15,17).

The prevalence varies considerably between countries, and between regions within a country. Due to lack of consistency and agreement between different studies in the classification of obesity in children, it is difficult to give an overview of the global prevalence or comparison between countries (9).

In the United States, the prevalence of overweight among the adult population has increased by more than 50% in the last 10 years (18) and it was found that some 20% male and 25% females are obese (9). The prevalence of obesity in Europe is 10-20% in men and 15-25% in women. In most European countries that have reliable data on time trends, the prevalence of obesity seems to be increasing (19). In Japan, prevalence of obesity is 12% in males and 13% in females (9) and it is 3% and 3.8% in male and female respectively in Thailand (20).

Obesity is a critical health concern among children in both developed and developing countries. Approximately, 22 million children under 5 years of age are overweight across the world. In the United States, the number of overweight children and adolescents has doubled in the last two decades, and similar doubling rates are being observed worldwide, including in developing countries (21). A similar trend has been experienced by Japan, where obesity (≥120% SBW) among school children of 6-14 years age group increased from 5% to 10% between 1974 and 1993 (9).

In Thailand, obesity (weight for height >120%) rose form 12.2% in 1991 to 15.6% in 1993(5). A recent study shows, the prevalence of obesity is 15.8% among 6-18 year old male school children in Saudi-Arabia (9). In Malaysia, it is 6.6% & 13.8% among 7 & 10 year-olds respectively. Obesity among 8-10 years old children in 1997 was 12.8% and 12.0% (BMI >85th percentile and 95th percentile respectively) in Philippines (9). In China, about 10% of school children were obese in 1992 (15).

In Bangladesh, a study was conducted in January, 1997 in a school and a MCH clinic in Dhaka city, among 316 the children of 2-10 years age group, the prevalence of obesity (Wt/Ht >120%) was to be found 13%. The study also revealed that obesity was positively correlated with increase of age and family income (22).

Obesity is a recognized disease in its own right and consequently causing a number of chronic, non-communicable and life threatening diseases including non insulin dependent diabetes mellitus, cardiovascular diseases, gall bladder diseases and

hormone-sensitive & gastrointestinal cancers as well as high risk for some non fatal conditions like back pain, arthritis, infertility and poor psychosocial functions including social isolation, peer problems etc (9,15,23-30). The most public health important consequence of childhood obesity is that it commonly tracks into adulthood and persistence rises with age among obese children (9,23,31). It is estimated that 70% of obese children become obese adult (23).

A number of biochemical parameters are altered in obesity. Dyslipidemia, hypertension and insulin resistance are frequently seen in obese children (32-33) and dyslipidemia appears to be related to increased abdominal fat accumulation (34). The characteristic pattern observed consists of elevated serum low-density lipoprotein (LDL) cholesterol and triglycerides and lowered high-density lipoprotein cholesterol levels. But, the association of hypertryglyceridemia with obesity is not strong (2). Studies indicate that atherosclerosis is a life long process that begins in childhood (35,36). For every 10% rise in relative weight, plasma cholesterol rises by 12 mg / dl (37). Hepatic complications with high concentration of liver enzymes particularly characterized by raised serum transaminase levels represent a frequent obesity associated finding in children. Occurrence of cholelithiasis among obese children is less frequent (9).

A few obese children may be suffered from gastro-oesophageal reflux and gastric emptying disturbances, due to raised intra-abdominal pressure caused by increased abdominal fat (9).

Obese is at higher risk of developing hypertension than lean counterpart (38). In India, Gupta and Ahmed found the prevalence of sustained hypertension was 3.4% in obese children, which was 20 times more compared to the controls (39).

Obese children may suffer from orthopedic complications - including slipped capital femoral epiphysis (9), Blount disease (40), knock knee and increased susceptibility to ankle sprains.

Sleep apnea is another consequence of childhood obesity for which aggressive therapy is warranted. Pseudomotor cerebri is a rare condition linked to raised intracranial pressure. Upto 50% of children who present with this syndrome may be obese, but the onset of symptoms does not appear to correlate with weight gain (9).

The economic cost of obesity is an important issue for health care providers and policy makers. In several developed countries, the cost of obesity has been assessed, and it was 2-7% of total health care cost. No such study was conducted in developing countries, but the escalating economic burden of adult non-communicable diseases (NCDs) among the adult population in those countries has already been recognized by a number of international agencies including World Health Organization and the World Bank. In these countries, a large proportion of NCD deaths occur in the productive middle years of life, at ages much younger than are witnessed in developed countries. The health burden attributable to excess weight in transitional societies are likely to be huge due to the absolute numbers at risk, the large reduction in life expectancy and the fact that the problem affects, in particular, individuals with a key role in promoting economic development (9).

Obesity is a nutritional disorder of multifactorial causes. Considering health consequences of childhood obesity, its degree of persistence or tracking over time, and the poor prospects for effective management, it is important to identify its determinants (41). A number of etiological factors like genetic, life styles, emotional over eating, indulgence, parental neglect, medical factors, geographical location, seasonality, socio-economic status, demographic factors have been identified as in different epidemiological studies (9,15,23,42-45).

Genetic factors play an important role in childhood obesity (46-47). It is correlated with family histories of obesity (48). Though fatness does run in families, but the genetic component does not follow simple Mendelian principles and the influence of the genotype on the etiology of obesity may be attenuated or exacerbated by non-genetic factors (49). The relative importance of inherited factors in the development and maintenance of obesity is still being actively debated. The recent discovery of

Simply, obesity is a result of positive energy balance over a considerable period. Body weight is primarily regulated by a series of physiological process but is also influenced by external societal and cognitive factors (9).

Rolland and Bellisle (63) reported that there was no relationship between body mass index (BMI) and skinfold thickness with energy intake in 7 to 12 y old children. Sunnegardh et al also showed no relationship between skinfold thickness and energy intake in 8-y old boys and girls (64). Studies that compared energy intake of lean and obese children have generally not shown that obese children have higher energy intakes than do lean children (65-69).

The recent worldwide increase in the prevalence of childhood obesity may be due impart to a decrease in children's physical activity (70). It is reported that obese children exhibit decreased performance during exercise compared with normal body weight peers (71). Other studies revealed that obese children expend more energy for resting metabolic rate and physical activity than their counterparts (65,66). Studies show that lean children are more physically active (65,72) where as others do not show a significant difference (67,69,73).

Evaluation of body composition is essential for an understanding of the growth and development, and various health disorders including malnutrition in human subjects. A non-invasive, modern method like bioelectrical impedance analysis- may be used to get an accurate and reliable (74) measure to assess the body composition as well as to compare with the traditional methods.

This study was aimed to explore in depth evidences on the prevalence of childhood obesity- to provide a baseline data and to identify the factors contributing to the problem and the pertinent issues requiring further research and to develop recommendations to alert nutrition and health experts to consider it as a public health problem.

CHAPTER II

RATIONALE

RATIONALE

Childhood obesity has become epidemic worldwide including developing countries where it is coexisted with undernutrition (9,15-17).

Obesity in children impacts on their health in both the short and long term (75). Studies have indicated that an important proportion of adult obesity has its origin in childhood (29,36,76). The degree of adiposity in infancy is predictive of fatness in childhood and childhood adiposity, in turn, is correlated with adult obesity (77,78).

Obesity is a potential risk factor for a number of chronic, non-communicable diseases. Child's obesity often results in social isolation, negative body image, low-self esteem, delayed psychosocial development, poor school performance etc. (9,23). Early onset obesity was suggested as a risk factor for mortality in later life (79).

The global epidemic of obesity is a reflection of massive social, economic and cultural upheavals currently facing developing and newly industrialized countries, as well as the ethnic minorities and the disadvantaged section in developed countries (9).

In countries with developing economies, the problem of obesity is emerging to pose a growing threat to the health of the citizens at a time when undernutrition remains a significant problem (9,10). It is evident that in many developing countries, obesity is coexisted with undernutrition, particularly those in rapid transition (9), where an increase in westernization of behavioral and dietary life style is evident (21). It is now replacing the more traditional public health concerns including undernutrition and infectious diseases, as one of the most significant contributors to ill health. It is only a matter of time before the same high mortality rates for such diseases will be seen in developing countries as those prevailing 30 years ago in industrialized countries with well established market economies (9,10).

Co-existence of overweight and obesity with undernutrition constitutes a double burden for the developing nations. These countries do not have the resource to provide anything other than public health and essential clinical services. At present, most individuals with excess weight gain are not treated, and the demand for medical

and dietetic help is expected to rise rapidly. In addition, limited resources will be diverted to pay for slimming diets and other aids to weight loss. In developing societies, the increasing prevalence of obesity is an early indicator of an emerging health burden due to the increasing mortality and morbidity from emerging or accelerating NCDs epidemics. Furthermore, a large proportion of NCD deaths occurs in the productive middle years of life, at ages much younger than those seen in developed countries. The health burdens attributable to excess weight gain in societies in transition are likely to be huge because of the absolute numbers at risk, the large reduction in life expectancy and the fact that the problem affects, in particular, individuals with a key role in promoting economic development (9).

Standards of living including diets and life styles continue to rise- have contributed to the emergence of weight gain, obesity, cardio-vascular disease, diabetes and various form of cancers- posing a growing threat to health in countries all over the world (9). Particularly in developing countries, rapid and widespread urbanization and other social and cultural upheavals erode traditional patterns of diets and life styles. Urban dwellers face powerful economic and social pressures to change to more convenient and more prestigious diets and life styles (80). Urban life and work - lack of exercise, tend to be sedentary. Decreased physical activity has been observed in urban compared with rural groups in Kiribati, Samoa and Fiji . As populations become more westernized, dietary composition changes to include more saturated fat and less fibres (15). The cosmopolitan city- Dhaka, the capital city of Bangladesh- a developing country, is not an exception in this regard.

Khaled studied with Bioelectrical impedance analyzer applied to a subsample of Bangladeshi people belonging to middle and lower middle class-found a marked number of obese-although they did not appear as fatty or chubby. This form of obesity-he termed as hidden obesity (81).

The world is experiencing epidemic of childhood obesity including Bangladesh, India and other developing countries (22,26,82). It is substantially increases the risk of subsequent morbidity whether or not obesity persist into adulthood (82).

In January, 1997, to ascertain the prevalence of obesity among the children of 2-10 years age group, a study was conducted with a small sample size from a school and a MCH clinic in Dhaka city. The study revealed that 41(13%) out of the 316 children were obese (Wt/Ht >120%). The authors recommended that obesity in childhood in the high economic communities could not be ignored in a developing country like Bangladesh. They also recommended for further research to prevent childhood obesity (22).

In Bangladesh, over the past 30 years, the prevalence of diabetes has been increased by some eight folds (83,84). Cardiovascular diseases also show significantly rising trend in the country (85).

Nutrition has been expressed as right in International human rights instruments since 1924 - the Declaration of the Human Rights. Declaration of the Rights of Child, 1959 echoed with the declaration of 1924. Children's right to nutrition receives its fullest and most ringing expression in the Convention on the Rights of the Child, 1989. In the World Summit for Children, 1990, the leaders of the world including Bangladesh committed to "give high priorities to the rights of children (86)."

It is enshrined in the constitution of Bangladesh in article 18(1) that the state shall regard the raising level of nutrition and improvement of public health as among its primary duties.

The Government of Bangladesh has developed national nutrition policy namely, the Bangladesh National Nutrition Policy, 1997, in which one of the most important objectives is to consider the importance of the family unit to provide adequate physical, mental, emotional and social needs of children --------- (87).

One of the objectives of the Bangladesh Plan of Action of Nutrition (NPAN) is promotion of appropriate diets and life-styles to achieve the goal - to improve the nutritional status of the people of Bangladesh to the extent that malnutrition would no longer be a public health problem by the year 2010 (88).

So, it is well documented that the Bangladesh is well committed to attain the proper nutritional status of the people with special attention to the children and other vulnerable groups.

Childhood obesity is almost an untouched aspect in the field of nutrition research in Bangladesh (22). No doubt, undernutrition is an important public health problem in Bangladesh. But the problem of childhood obesity prevails in Bangladesh particularly in Dhaka city has been largely ignored. It may be mentioned, the Dhaka city is in large extent different from the other parts of the country regarding income level, life-styles etc. Among the majority of the city dwellers mostly similar with that of the industrialized/developed countries like USA, UK and others. In recent days, the most of the children of the Dhaka city are engaged in curricular activities even in the afternoon or busy with mostly physically inactive extracurricular activities. Furthermore, the children are loosing their playgrounds in and around their residence as well as at the school (89). This segment of the children deserves the right to grow up with proper physical and psychosocial development. It has been suggested to recognize the problem the problem in the country and to do some thing for it before it gets entirely out of the hand (90).

So far, no study on epidemiological aspect of childhood obesity in this country has been conducted. Therefore, considering the socio-cultural and economic transition of Dhaka city, it was planned to conduct this study, to explore the prevalence of childhood obesity-to provide a baseline data, to identify the factors contributing to the problem and the issues requiring further research and to develop recommendations to alert nutrition and health experts to consider it as a public health problem, and for the policy maker in developing comprehensive public health policies and strategies for the prevention and management of obesity, aimed to save the scarce and limited resources to be devoted for health care for a developing country like Bangladesh for a healthy tomorrow.

CHAPTER III

REVIEW OF LITERATURE

DEFINITION

Obesity is defined simply as a condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired (9).

Histopathologically, obesity may be defined as an abnormal growth of the adipose tissue due to an enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyperblastic obesity) or a combination of both (11).

Most simply, obesity occurs as a long-term positive energy balance (1). In a large majority of cases, it is due to either excessive intake of food or a disorder of metabolism with deficient utilization of one or more food factors (91).

In objective terms, obesity can be defined using cut-off points either relative weight index 120% or above the standard weight for height (1,3-7), age and sex specific skin-fold thickness (SKFT) equal or more than 85^{th} percentile (8) or Body Mass Index (BMI) above 30 for adults both male and female (9).

To date, there has not been the same level of agreement over the classification of overweight and obesity in children and adolescents as in adults. There has been confusion both in terms of a globally applicable reference population and of the selection of appropriate cut-off points for designating a child as obese (9).

HISTORY OF OBESITY

Obesity is not a recent phenomenon. Its historical roots can be traced to the Paleolithic era more than 25000 years ago, since Stone Age artifacts of corpulent women have been found in several sites acted in Europe. Throughout most of the history of mankind the overt manifestations of weight gain and fat storage in children and adults have been viewed as signs of personal health and family wealth and an indicator of economic prosperity of the societies (9,15). Until recently, in most

developing societies, being thin has been equated with poor health and thinness in babies is associated with increased risk of illness (10,15). But nowadays, obesity particularly among women and children is often confronted with social prejudice and discrimination in the most of the countries. However, in some regions such as the Pacific Islands, obesity is still considered desirable and a symbol of wealth and higher social standing (15).

Clinical evidence of obesity can be dated as far back as Graeco-Roman times, but little scientific progress was made towards understanding the condition until the 20th century. In the 19th century, the work of Lavoisier and others indicated that metabolism was similar to slow combustion, and that obese and lean humans obeyed the laws of thermodynamics. Also, the discovery that fat is stored in "cells", the basic unit of biology, led to the idea that obesity could be caused by too many fat cells. Interestingly, the 19th century also saw the publication of the first diet book entitled *Letter on corpulence addressed to the public* by Mr. W. Banting (9).

With the turn of the century, analysis of life insurance data indicated that obesity was associated with an increased death rate. In the 1920s, a familial basis for obesity was suggested along with description of Cushing's Disease and hypothalamic obesity. Later the introduction of thyroid hormone, dinitrophenol and amphetamine as pharmacological treatment of obesity opened the door to the use of drugs, and genetics improved understanding of several specific forms of obesity resulting from genetic defects (9).

Considerable advances have been made in diet, exercise and behavioral approaches to treatment for obesity since their advent in first half of the 20th century, and new drugs with ever-better profiles of pharmacological activity continue to be introduced on a regular basis. Gastric surgery has had the most effective long-term success in treating the severely obese. Despite this progress, however, obesity prevalence continues to increase sharply, and the challenge to public health workers and scientists has never been greater (9).

THEORIES OF OBESITY

The two most common theories about obesity, although controversial, are 'the fat cell theory' and 'the set point theory'. These theories have been proposed by researchers who have to explain why many people have difficulty in controlling their weight even when they are highly motivated (1).

The Fat Cell Theory

When the body stores energy in the form of fat, existing fat cells grow larger (hypertrophy), and sometimes- new fat cells are created (hyperplasia) (92). Once a fat cell is created, it exists for life. Therefore, when an obese person losses weight, fat cells do not disappear, they only shrinks, Proponents of the fat cell theory believe that this cell shrinkage then triggers a strong desire for food. As a result, the individual eats and regains the weight (13).

The Set Point Theory

This theory (1) holds that each individual has a "natural" weight, which is predetermined by a number of biological factors. Attempts to change weight above the set point are thwarted by various mechanisms in the body. In other words, the body defends its set point, even though it may be a weight 10 or 20 pounds greater than what is considered ideal (13). An individual's set point range is apparently determined by genetic factors and (possibly) by factors such as intrauterine environment, resulting in programming of metabolism at birth (50).

Other Theories

There are other controversial theories that also attempt to explain the development and maintenance of obesity. The malfunctioning hypothalamus theory views obesity as a result of overeating that is caused by a defective hypothalamus. The sluggish metabolism theory holds that certain individuals have inherited bodies that function

on less energy than is normal. The brown fat theory is based on the premise that a special type of fat (brown fat) in the body gets rid of extra calories through heat generation, and obese people have less brown fat than do non-obese individuals (13).

"Fat cell number and size" hypothesis states that overfeeding when adipocytes are forming leads to hyperpalsia and an increase in the overall number of adipocytes, and that individuals with increase cell numbers are more subject to obesity (93).

CLASSIFICATION OR TYPES OF OBESITY

The syndrome of obesity in human being is a complex issue. It has several types or forms with different categories. Some of them are mentioned below-

A. According to cell differentiation obesity can be classified (11-13) as-

a) Hypertrophic

It is a type of obesity in which, abnormal growth of the adipose tissue occurs due to and enlargement of fat cell size (hypertrophy).

b) Hyperblastic

It is a type of obesity in which, abnormal growth of the adipose tissue occurs due to an increase number of fat cell (hyperplasia).

c) Mixed or combined

It is a type of obesity in which, abnormal growth of the adipose tissue occurs due to enlargement of cell size and increase number of fat cells.

Weight gain may be the result of hypertrophy, hyperplasia or a combination of both. Obesity is always characterized by hypertrophy, but only some forms of obesity also involve hyperplasia (94). Obese children have a significantly greater number of fat cells than their lean counterpart (57). Observation in children relating early onset obesity with detectable fat cells numbers were consistent (95,96) with the " fat cell number and size" hypothesis (97). Adiposity hyperplasia occurs mostly during development (98) and is enhanced by overnutrition (99) increasing irreversibly fat storage capacity. It is clear form the studies

on rodents that caloric restriction while decreasing body weight, dies not always decrease body fat to the desired extent (100,101).

In hypertrophic obesity, the number of cells is limited, but they expand to increase body weight. This type of obesity may occur at any time throughout life but is frequently found in adults who gain weight (11,13).

B. Considering clinical usefulness of obesity Garrow (57) has proposed as classification on the basis of Body ness Index for adults-

Grade III	: BMI	> 40.00		
Grade II	: BMI	30	-	40.00
Grade I	: BMI	25	-	29.99
Grade 0	: BMI	20	-	24.90

The major weakness of the use of W/H^2 (originally proposed by Quetelet in 1871) is that some very muscular individuals may be classified as obese when they are not. These numbers will be small, however, BMI is the relative weight index that shows the highest correlation with independent measures of body fat (95).

C. Obesity can be classified according to distribution of body fat in the body-

a) Generalized : In this type, fat distributed all over the body. Usually it is seen in childhood obesity.

b) Localized : There are two major types of regional fat distribution in adult obesity (13,102) -

i) Gynoid type: In some adults, fats stored mainly around the hips, thighs and buttocks, which gives a 'pear shape'. This type is common to women.

ii) Android type: In this type, storage of fats occur primarily in the waist and upper abdomen, producing an 'apple shape', It occurs in both sexes.

iii) Combined type: Combinations of the two types are also seen, particularly in women.

Women with gynoid type of obesity do not develop the impairments of glucose metabolism seen in obese women of the same weight who carry their fat in an android distribution (13).

Android type is characterized by rapid mobilization of free fatty acids and is associated with significant risk for hypertension, cardiovascular diseases and non-insulin dependent diabetes mellitus (13).

Regional fat distribution defines risk of hyperlipidemi in obese children as it does in adults (13).

D. World Health Organization classified obesity in adults according to BMI (9)

Pre- obese	: BMI	25.00	-	29.99
Obese class I	: BMI	30.00	-	34.99
Obese Class II	: BMI	35.00	-	39.99
Obese Class III	: BMI	$\geq$ 40.00		-

E. Obesity can be classified as (11) -

a) Hereditary

When the definitive cause of obesity is not known, heredity probably contributes to the condition. Heredity also dictates where fat is deposited in the body.

a) Endocrine

Obesity caused by hypothyroidism occurs in less than one percent of overweight subjects. In hypothyroidism, the basal metabolic rate (BMR) is lowered because of a deficiency in thyroid secretion. This results in excessive retention of calories, which would normally expended in basal metabolism. Thorough physical examinations are mandatory for optimum weight control results.

F. Obesity also may be classified depending on onset-

a) Childhood onset : This type of obesity begins in childhood.

b) Adolescent onset : This type of obesity begins in adolescent period.

c) Adulthood onset : This type of obesity begins in adulthood.

MAGNITUDE OF OBESITY

Evidence is now emerging to suggest that the prevalence of overweight and obesity is increasing worldwide at an alarming rate in both developed and developing countries (9,75). Easy access to good tasting, high fat foods and decreased physical activity are both likely contributors to the current worldwide epidemic of obesity (103).

In many developing countries, obesity coexists with undernutrition. It is more prevalent in urban than in rural populations. In economically advanced regions, prevalence rate may be as high as in industrialized countries (9).

Determination of prevalence of overweight in children and adolescents is more difficult than in adults due to lack of consistent international criteria for classifying children as overweight or obese (104).

Obesity among adult population

Women generally have higher rates of obesity than men, although men may have higher rates of overweight.

Obesity is now epidemic in developed nations including Australia, New Zealand and Singapore and is rapidly becoming so in many developing populations, particularly Pacific Islanders and certain Asian Nations This rapid process is also seen in minority groups in developed societies e.g. Maoris in New Zealand, Indians in the U.K. Malaysia and Singapore, Australian Aborigines and Torres Straight Islanders (9,15).

Obesity is relatively common in Europe, especially among women and in southern and eastern European countries. The average prevalence of obesity in European centuries participating in the WHO MONICA study between 1983 and 1986 was about 15% in men and 22% in women, although there was great variability both within and between countries. The prevalence of obesity in England-men 6%, women 8% (1980) and men 15% & women16.5% (1995), Germany - men 13.7% women

22.2% (1985) and men 20.5% & women 26.8% (1992), Lithuania - men 22% & women 45%. In the Netherlands, in 1987, 6.0% men & 8.5% women and in 1985 8.4% men & 8.3% women were obese. In Sweden , 7% men and 9% women are obese (9).

The most recent data from individual national studies suggest that the prevalence of obesity in European countries is currently in the range 10-20% in men and 10-25% in women. In agreement with the MONICA data, the prevalence of obesity is generally higher in women than in men (9)

The prevalence of obesity in the USA is those from NHANES III (1988-1994), a recent reanalysis of the data using BMI $\geq$ 30 to classify obesity is particularly valuable for use in global comparisons, and showed that around 20% of all men and 25% of all women in the USA are obese. In Brazil, PNSN (the National Survey on Health and Nutrition), survey indicates that obesity is affecting about 6% of men and 13% of women in 1989 (9,105).

A recent report shows the prevalence of obesity and overweight respectively were 11.1% & 14.3% in U S, 6.0% and 10.0% in Russia and 3.6% and 3.4 in China (75). The prevalence of obesity in general population of Australia and New Zealand appears to be in the range of 10-15% Using Cut-off point of $\geq$ 120% of standard body weight, the prevalence of obesity in Japan is 12% in males and 13% in females (9).

In the countries of the South East Asia Region, good quality, nationally representative, secular trend data on obesity are not available, only limited obesity prevalence data are available. Various studies on nutritional status have been carried out, particularly in India, but these have generally been on undernutrition and on selected population groups and have not used the WHO classification of obesity. As many countries in South East Asia are currently going through the so called "nutrition transition", there is a special need to collect good quality, nationally representative obesity prevalence data. The nutrition transition is associated with a change in the

structure of the diet, reduced physical activity and rapid increased in prevalence of obesity (9,106).

However, data from two studies conducted by the same research center in Thailand do suggest that diet-related chronic diseases, including obesity, are increasing in affluent urban populations. The first study was conducted in 1985 among 35-54 year old Thai officials; it was found that 2.2% of the 2703 men, and 3.0% of the 792 women, had a BMI ≥ 30 (9) .The second study in 1991 was smaller (66 men and 453 women), and had a broader age range (19-61years), but also assessed nutritional factors in affluent urban Thais. Results of this study showed that 3.0% of men and 3.8% of women had a BMI ≥ 30. Prevalence figures for BMI 25-29.9 were considerably higher (15.2% in men and 23.2% in women) (19).

A recent study in Mauritius has shown that a dramatic increase in obesity prevalence over a five years period in both men and women aged 25-74 years. The proportion of obese men increased from 3.4% in 1987 to 5.3% in 1992, while the proportion of obese women increased from 10.4 % to 15.2% in the same period (107).

In developing countries of African region, rural adults still maintaining traditional life styles gained little or no weight with age until relatively recently. However, with the improvement in socio-economic status and increasing changes due to rapid urbanization, the prevalence of obesity among some groups of black women has risen markedly to levels exceeding those in populations in industrialized countries. In fact, approximately 44% of African women living in the Cape Peninsula were estimated to be obese in 1990 (9).

A nationally representative, cross sectional survey was conducted between 1990 and 1993 to study the effects of sex, age and regional distribution on the prevalence of overweight and obesity among 13177 randomly selected adult Saudi-subjects. The prevalence rate was 16% & 24% in male and female respectively, It is 1.5 times higher in urban than rural population for both sexes (9). In the United Arab Emirates, data from the National Nutrition Survey showed that 38% of married women and

15.8% of married men were obese. In Bahrain, obesity was more common in urban (male 9.5% female 30.3%) than rural areas (male 6.5%, female 11.2%) especially in women. In a study in the south of Islamic Republic of Iran revealed that obesity prevalent in adult population, and is more frequent among women than men. The current prevalence of obesity (male 1.2%, female 1.64) in China is probably best documented by the 1992 third Nationwide Nutritional Survey (NNS III) This survey was conducted throughout both urban and rural provinces, and data were collected from a larger representative sample of men (n=14964) and women (n=14590) aged 20-45 year than the China Health and Nutrition Survey cohort (n = 5000 approximately) (9).

Among adults aged 18-60 years in Malaysia, 4.7% of men and 7.9% of women were found to have a BMI above 30. In the women, overweight and obesity problems were more serious in the Indian population; 17.1% of Indian women had a BMI over 30 compared to 8.8% of Malay and 4.3 of Chinese women. Among the Malay population, a considerably higher proportion of both men and women had a BMI over 30 (men: 5.6% urban, 1.8% rural; women: 8.8% urban, 2.6% rural) (9).

The National Nutrition Survey, 1995 of the Republic of Korea found only 1.5% of the population was classified as obese (BMI>30 kg/m^2) and 20.5% were overweight (BMI 25-29.9 kg/m^2) (15).

Obesity among children and adolescents

The worldwide prevalence of obesity in children continues to increase and in many countries it has reached at sufficient proportions to be considered as epidemic (98,108).There is a paucity of data on the prevalence of childhood obesity in the South-East Asian countries. The lack of consistency and agreement between different studies (as different reference data, different cut-off points and different indicators and definitions used) in the classifications of obesity in children and adolescents makes it difficult to give and overview of the global prevalence of obesity or comparison between countries in young age groups (9,104). Nevertheless, irrespective

of the classification system used, studies of obesity during childhood and adolescent have generally reported that if prevalence has increased (9).

In the United States, the prevalence of overweight (defined by the 85th percentile of weight for height) among 5-24 year olds from a biracial community of Louisiana (n=11565) increased approximately two folds between 1973 and 1994. Furthermore, the yearly increases in relative weight and obesity during the latter part of the study period (1983-1994) were approximately 50% greater than those between 1973 and 1982 (108).

In Japan, the prevalence of obese school children (≥120% SBW) aged 6-14 year increased form 5 to 10% during the 20 years between 1974 and 1993. The increase was most prominent among boys of 9-11 years (109).

Childhood obesity is not confined to the developed countries, as high rates are already evident in some developing countries. In Thailand, prevalence of obesity (wt/ht > 120% of the Bangkok reference) among school children aged 6-12 years rose form 12.2% in 1991 to 15.6% in 1993, and in a recent study of 6-18 year old male school children in Saudi Arabia, the prevalence of obesity was found to be 15.8% (9).

Recent Malaysian data demonstrate increasing prevalence of obesity with increasing age: 6.6% among 7 year olds, rising to 13.8% of 10 year-olds (33). Obesity among these 7-10 year olds was higher among boys than girls (12.5% compared to 5.0%). Ethnic differences were also found, especially among boys, where 16.8% of Malays were obese compared to approximately 11.0% of Chinese and Indians. Furthermore, obesity has increased from 1% in 1990 to 6% in 1997 among 13-17 year olds. In Philippines, obesity among 8-10 years old children in 1997 was 12.8% and 12.0% using BMI >85th percentile and 95th percentile respectively. In China, about 10% of school children were obese in 1992. Recent data from Japanese children aged 6-14 years old shows the prevalence of obesity ranging between 5% to 11%. Furthermore, the prevalence of obesity among 9 year old boys has increased from 2.9% in 1970 to 9.7% in 1997; comparative figures among 9 year old girls are 3.4 % and 8.0% (15).

In Bangladesh, to ascertain the prevalence of obesity among the children of 2-10 years age group, a study was conducted in January, 1997, with a small sample size from a school and a MCH clinic in Dhaka city. The study revealed that 41(13%) out of the 316 children were obese (Wt/Ht >120%). The study also revealed that obesity was positively correlated with the increase of age and family income. Statistically no significant difference was found in sex distribution (22).

ETIOLOGY OF CHILDHOOD OBESITY

The etiology of the development of obesity during childhood is complex, multifactorial, and individual and poorly understood and has not been elucidated completely (1). Epidemiological studies have identified several risk factors for obesity in children, including genetics, prenatal malnutrition, birth weight, life style, emotional overeating, high protein intake in infancy, indulgence, parental neglect, medical factors, geographic location, seasonality, education, income, parental obesity, family size, excessive television viewing etc. (24,110). Most obese children exhibit more than one (23) and it is suspected that a complex interaction of genetic, environmental and behavioral factors is responsible (111).

Genetics may increase an individual's susceptibility to obesity, environmental factors are operative, particularly in severe obesity (23). Obesity in humans is observed to be aggregated in families (112), presumably due to both genetic influences and shared life style within the family unit (113,114). Genetic transmission is said to account for approximately 25% of the variation of obesity in the general population. Cultural factors are also believed to be transmitted with family units and have been estimated to account for 30% of the variation in obesity (115). Hence, a sizeable contribution to the familial aggregation observed in human obesity probably stems from shared environmental and behavioral factors within the household as well as inherited factors. Obesity status in the biological parent is a good predictor of the development of obesity in children (116,117).

Although altered energy metabolism does not appear to play a role in the maintenance of the obese state, several studies suggest that reduced energy expenditure is an additional risk factor in the development of obesity in children. Studies suggest that alterations in energy metabolism and/or fat distribution early in life may be risk factors for negative health outcomes in later life (118 -120).

Thus, the environment (nurture) plays a critical and inextricable role in permitting the expression of whatever predisposition (genetic or developmental) may exist towards

obesity. There is a tendency to treat food intake and physical activity as modifiable components of energy balance; with the implication that these behaviors are determined solely by volitional or environmental process (121).

Once children are obese, typically they develop additional difficulties that contribute to their weight gain such as peer problems, social isolation, psychological distress, physical limitation, health problems and skill deficits that preclude their involvement in sports, dance, and other physical activities (23).

Genetic

The genetic factors in weight gain is currently the subject of much research, and the discovery of leptin- a fat derived hormone has led to a renewed interest in genetic and metabolic influences in the development of obesity (9,122).

At a population level, the genetic component of obesity is expressed in terms of heritability. This refers to the proportion of the total variation in a character, which is attributable to genetic factors. The heritability of obesity may be considered either in terms of the total fatness of an individual or the distribution of body fat (9,49). Heritability studies suggest that as much as 70% of the variability in human body weight may be accounted by genetic factors (103,123).

A large number of twin, adoption and family studies on the heritability of different measures of obesity have been conducted. Adoption studies tend to generate the lowest estimates and twin studies the highest (9).

Genetic epidemiology suggests that between one fourth and one half of the variability in body weight in the general population can be explained by genetic factors (124). Whereas external influence undoubtedly creates a pressure for development of obesity, genetic factor seemingly affect the response to external factors and influence the ultimate body weight of an individual. The nature of these genetic influences is largely unknown. This could affect amount of food ingested, the rate of energy

expenditure or both (125). Studies in identical twins strongly imply that body weight in humans is under genetic control (126).

There is strong evidence from twin and adoption studies of a genetic contribution to fatness in human children and adults (127,128). Likewise, there are data that suggest that aspects of energy expenditure are also heritable and predictive of weight gain in children and adults (129). The genes that are primarily responsible for the regulation of body weight in mammals are not known. Pipes and Trahms stated that genetically predetermined growth potential in children can be compromised or accelerated by variation in nutrient intake ranging from undernutrition to overnutrition (45).

The familial resemblances in fatness (130) indicate a significant genetic effect on susceptibility to the diseases. Other studies revealed that familial patterns of adiposity support the hypothesis that variation in body fatness is explained in part by a genetic or heritable component (131-132). Recent evidence suggests that intergenerational transfer of eating styles also contribute to these familial patterns; obese parents may use child-feeding practices that foster the development of obesity. Genetic predisposition may interact synergistically with the physical and social environment to produces inherited patterns of obesity within families. However at present, scant information exists concerning how the family environment shapes the development of children's eating and exercise behaviors and how parents influence their children's eating and activity patterns (133).

Biological parental obesity is a good predictor for the development of obesity in childhood. The probability of a child becoming obese has been estimated to be 7% if neither parent is obese, 40% if one parent is obese, and 80% if both parents are obese (116,117).

The "thrifty gene" hypothesis suggests that obesity result from the introduction of a continuous and ample food supply to people who have developed, through evolution, the ability to store energy millennia of feast (134).

Prenatal nutrition

Fetal life seems to be a critical period for the development of obesity (51). It is well documented that prenatal malnutrition is a determinant of obesity. The Dutch famine was a unique nutritional challenge - occurred at the end of World War II in the western part of the Netherlands. It started and ended abruptly, lasted only 5 months, was preceded and followed by more or less adequate nutrition. At its peak in the first months of 1945, the official daily rations of the general population varied between 400 and 800 kcal. Moreover, the rations for infants were relatively spares, which confines the exposure principally to the prenatal period. The Dutch famine is therefore hardly comparable with other famine period of which in the long - term effects have been studied. For example, a study revealed that people born in Leningrad around the time of the German siege of 1941-1944 did not find an association between prenatal exposure to famine and obesity, but the famine in Leningrad lasted for 28 months and evidently, most people were exposed during the whole period of gestation as well as during infancy (135). Moreover, people born in Amsterdam around the time of the Dutch famine grew up in a time of increasing affluence, whereas the living conditions remained relatively poor for those born in Leningrad.

A study of some 300 000 19 year old conscripts who were exposed in utero to the Dutch famine, showed that the effect to prenatal exposure to famine depended on its timing. The rate of obesity (body weight for height ≥120% according to an external standard) was higher in men exposed in the first half of gestation (2.8%) and lower in men exposed in the last trimester of gestation or the immediate postnatal period (0.8%) than in non-exposed men (1.8%) (135,136).

Ravelli et al measured the body size of 741 people born at term between November 1943 in Amsterdam. They compared people exposed to famine in late, mid, or early gestation (exposed participants) with those born before or conceived after the famine period (non-exposed participants). The body mass index of 50-year-old women exposed to famine in early gestation was significantly higher by 7.4% than that of

non-exposed women. BMI did differ significantly in women exposed in mid gestation or in late. In 50-year-old men, BMI was not significantly affected by exposed to famine during any stage of gestation: BMI differed by 0.4% in men those exposed to famine late gestation, by - 1.2% in those exposed in early mid gestation compared with non-exposed men. Authors concluded that maternal malnutrition with early gestation was associated with higher BMI and waist circumference in 50-y-old women but not in men. These findings suggest that pertubations of central end endocrine regulatory systems that establish in early gestation may contribute to the development of abdominal obesity in later life (7).

Birth weight

Birth weight may be an important predictor of childhood obesity. In a study, it was to be found that prevalence of obesity was higher than the expectation, in the small group of children who weighed less than 2500 gm at birth in small families. Wilkinson et al found that more children than expected were obese at birth, probably because the mothers themselves were heavy. Thus it is possible that being overweight at birth predisposed a few children to later obesity, although other reports on the birth weights of obese children have been contradictory. Higher risk of obesity in children with high birth weight and may be carried on into young adulthood (52,53,137).

Associations between low birth weight, particularly thinness at birth, and increased risk for disease in adulthood, such as type 2 diabetes and cardiovascular disease, are now well established. In many population studies, these associations are enhanced by and even depend on the development of obesity in adulthood. Therefore any relation between small size at birth and obesity in later life may contribute to the pathogenesis underlying the fetal origin hypothesis (138).

A number of studies suggest that low birth weight is associated with a central pattern of fat distribution. Increased central fat deposition, indicated by a high ratio of subscapular to triceps skin folds has been found in 7-12 year old children in the USA who were born small and also in young adult Mexican-Americans (54). Higher waist

circumference and higher waist to hip ratio have both been associated with low birth weight in adults. Birth weight is positively related to body mass index. These relationships were independent of the girls' gestational age at birth and current social class (54).

Sex

A number of physiological processes are believed to contribute to an increased storage of fat in females, Such fat deposits are believed to be essential in ensuring female reproductive capacity. Studies in humans and animals indicate that girls exhibit a stronger preference for carbohydrate before puberty while boys prefer protein. However, after puberty, both males and females display a marked increase in appetite for fat in response to changes in the gonadal steroid level. This rise in fat appetite occurs much earlier and to a greater extent in females (9).

Females have a tendency to channel extra energy into fat storage while males use more of this energy for protein synthesis. This pattern of energy usage, or "nutrient partitioning" in females contributes to further positive energy balance and fat deposition for two reasons. First, the storage of fat is far more energy efficient that of protein, and second, it will lead to a lowering of the lean-to-fat tissue ratio with the result that RMR does not increase at the same rate as body mass (9).

Birth order and family size

Childhood obesity is associated with family variables - family size and birth order. Researchers have reported that the prevalence of obesity declines with increasing family size, in small families at least, lastborn tend to have higher rates of obesity than firstborn; most particularly that only children tend to be obese more often than those with siblings (53,139).

Birth order has no consistent effect on prevalence, although some data suggest the greater prevalence occurs among younger children of large families (4,61,140).

A number of studies have been conducted to see the association of first-born child and obesity. Stettler et al found that first-born status independently associated with the development of increased adiposity (141). But contradictory results were found in earlier studies conducted in European and African populations (4,141,142). Study showed, being an only child was one of the common "at risk" factors for obesity among 10-year-old children in Newcastle upon Tyne (53).

Ravelli and Belmont (4) examined a population of young men, whether obesity is related, as in childhood, to family size and birth order. They found birth order and family size effects in that population of young men, using different out comes. They also found that small family size and early birth order were independently advantageous for intellectual competence (143) and school performance (144).

Critical period

Critical or sensitive periods in development have been well described for many physiologic and behavioral processes (145).

Many observations suggested that two and possibly three critical periods exist for the development of obesity and its complications. These include gestation and early infancy, the period of adiposity rebound that occurs between 5 and 7 years of age and adolescence. Follow-up studies of infants exposed to famine prenatally or early in life and of infants of diabetic mothers strongly suggest that prenatal and perinatal over or under nutrition influences the development of fatness in later life.(51)

The prevalence of obesity among children of diabetic mothers at ages 5-9,10-14 and 15-19 years was significantly higher than the prevalence of obesity among children of the same age born to pre - or non diabetic mothers. The prevalence of obesity among the children of diabetic mothers also appeared to be independent of the mother's obesity status at the time of birth. Several other large studies have confirmed the

persistent effect of birth weight for the subsequent risk of obesity at ages 6, 11,15 and 17 years and in adulthood (51,146-148).

The body mass index increases in the first year of life, and subsequently decreases. Beginning at about 5 years of age, BMI again begins to increase. The time at which the second increase occurs has been called the period of obesity rebound. (149,150). Studies also revealed that the preadolescent period between 7 and 11 years attracts particular attention because juvenile obesity may begin during this interval of accelerated growth (60).

Family structure

The characteristics of family structure or family life are closely linked to the development and maintenance of obesity in children .The possible influence of family factors has been debated for several decades (45).

In USA, obesity among the children has been increased by 54 % over the 20 years. The causes of the increase unknown but are thought to be environmental. Some studies suggest that the rise in obesity is the result of a society that does not support children and families effectively. More families have become too removed to be aware of or to fulfill their children's needs. More children have become distressed by isolation, marital disruption, substance abuse, physical or sexual abuse, parental loss, or physical jeopardy. To blunt their difficult feelings children develop excessive appetites for food or inactive pursuits such as television or video games - leads to high intake of food and reduced physical activities causing weight gain (23).

Over the past years, children have become neglected. Indulgence, another form of neglect, has also become more common, with both parents working and away from their children more, exhaustion, guilt, fear of rejection, disempowerment, and unfamiliarity with the child chip away at the parents' ability to provide an authoritative influence in their children's lives. When parents set few limits on behavior, they effectively treat their children as peers, a very unsafe role for the

young. Children may react by developing poor life style habits because of poor guidance or by acting outrageous, flaunting poor food, uncleanness, undone homework, television, and other behavior (23).

Family structure (biological or other parents and number of siblings) did not significantly affect the risk of adult obesity. Parental neglect during childhood predicts a great risk of obesity in young adulthood, independent of age and body mass index in childhood sex, and social background (45).

The association between parental neglect and later obesity is far stronger than those for other psychosocial risk factors, such as parental education or occupation quality of dwelling, or child's school performance (151,152).

The mechanisms, by which these psychosocial factors act, are not known. Dietary habits and physical activity may have roles, but in studies in the general population, neither have shown effects on obesity as large as those observe in this study. Lissau and Sorensen suggest that parental neglect may cause psychological state that affects energy balance by altering behaviors (overeating and physical inactive) or hormone balances, influencing fat storage (corticosteroids, catecholamines or insulin) (45).

Socio-economic status

Socio-economic status (SES) is usually measured in terms of a composite index combining income, education, occupation and, in some developing countries, place of resident (urban/rural). However, individual components of SES may have independent and even opposite effects on dietary intake and physical activity patterns, so that it is often very difficult to make generalizations about the relationship between SES and obesity (9).

Despite these problems, studies have repeatedly shown that high SES is negatively correlated with obesity in developed countries, particularly among women, but positively correlated with it is population of developing countries. Further evidence

suggests that, as the less developed countries attain higher levels of affluence, the positive relationship between SES and obesity is slowly replaced by the negative correlation seen in developed countries (9,153)

Developed countries tend to show an inverse relationship between obesity an SES status even in developing countries (154). A state of food deprivation is now very unusual in any major population groups in the industrialized countries, and the proportion of adults engaged in physical activity at home has fallen markedly with modernization, Thus, the groups of lower SES need to be no more physically active of short of food (in energy terms) than those of higher status. In fact, studies suggest that families belonging to be lower status groups engage in much less physical activity than those in higher ones; for instance, their obesity levels have risen in parallel with rising car ownership and they watch television of many more hours per day (155).

Studies indicate that change in income has little effect on dietary structure in countries where income levels are already quit high in relation to basic food needs; instead, increased in income are spent on more elaborately packaged and processed or higher quality foods rather than on a grate quantity of food. In the poorest income groups, however, food demand is much more price and income sensitive, a many people struggle to obtain enough high quality food for what is considered to be as healthy diet. The diet of household of lower socioeconomic status tends to be energy dense, and high fat intakes are a prominent feature; the more expensive vegetables, fruit and whole grain cereals are eaten more sparingly (9).

In developing countries, the lower obesity rates observed in the population of lower SES are associated with a situation where people are limit in their ability to obtain enough food, yet still engage in moderate to heavy manual work and have little access to public transport. Hence, thin adults are considered poor, and oversight and obesity are a sign of affluence.

However, as per capita income increase, the nature of the diet in traditional societies tends to change in a pervasive and well-documented manner. In particular, intakes of

animal fat and protein increase, those of vegetable fat and protein decrease, those of total, and particularly complex, carbohydrates also decrease, and those of sugar increase (9).

The increase in income may be associated with increased away from home consumption of high fat food items, as in the Philippines, or with increased consumption meat, as in China. However, the over all effect tends to be a greater intake of total fat and an increased prevalence of obesity (153).

Since the 1950s, a number of studies carried out in different parts of the world have shown an association between obesity and various social and economic factors (6,156-160). An association between obesity and SES has been shown in both directions (58). The development of obesity is closely related with the individual's previous SES that illustrates the great importance of environmental factors in the genesis of obesity (161). On the other hand, some studies have also revealed that the presence of obesity in an individual can modify or determine has or her SES (43). One of the main findings of investigation of the relation between SES and obesity has been the importance of gender in this association. In females, most studies in this area have shown a negative association between obesity and SES: the lower the SES the more frequently obesity is found in females. Among males, on the other hand, study results are much less consistent and show both a positive and a negative relation or the absence of any type of association. Moreover, in studies of persons of both sexes where the association was negative in both males and females, the magnitude of the association was greater in the female sex (162).

Data from the 1960s and 1970s have shown that children whose family incomes were below the poverty level were shorter and thinner than children whose family incomes were above the poverty level (62). Dietz and Gortmaker found that the prevalence of obesity was associated with higher income levels (43). However Jones et al found that the NHANES II data indicated that white 6-to 11 year-old girls living below the property level were on average, 1.03 kg heavier than their non-poor counterparts (162). Another study showed that approximately 33% of adult Americans are obese,

with higher prevalence in minority and lower socioeconomic status (SES) Population (163).

Researchers reported that SES to be inversely related it obesity, particularly in white women (164-166). The high prevalence of obesity seen in black women in the United States (167) has been attributed to greater poverty among black Americans. Because young children are dependent on their adult caretakers for food availability and these caretakers also determine the overall family lifestyle, SES may affect children obesity much as it affects adult obesity (168). A comprehensive literature review by Sobal and Stunkard as well as other reports reveal mixed finding on the relation ship between obesity sand SES in children (164,169).

The manner in which low levels of income and education are associated with obesity is not well understood. Most reports deal with the observed inverse relationship between SES and body mass index (BMI) and generally do not provide further insight into the nature of this relation ship. For instance, it is not clear how SES affects behavior (e.g., through overeating or physical inactivity), which then many lead to obesity (165).

Energy intake

Total energy intake refers to all energy consumed as food and drink that can be metabolized inside the body. In simple terms, obesity is a consequence of an energy imbalance -an excess of energy intake over energy expenditure for a considerable period (9,170).

Rolland Cachera and Bellisle reported that there was no relationship between body mass index (BMI) and skinfold thickness with energy in take in 7 to 12 y old children (63). Sunnegardh et al also showed no relationship between skinfold thickness an energy intake in 8-y old boys and girls (64). Studies that compared energy intake of Lean and obese children have generally not shown that obese children have higher energy intakes than do lean children (65-69).

One of the major risk factor for cardiovascular disease is obesity. Because the pathogenic process of cardiovascular diseases begins during childhood (171,172) efforts have begun to identity early antecedents of such diseases among preschool age children (173). Adverse diet is receiving in creasing attention as a possible cardiovascular risk factor in children (174-177).

It is generally accepted that health patterns in childhood (e.g. diet, physical activity) mark the beginnings of many health problems not manifested until much in later life. In light of this, researchers have identified links between dietary patterns in children and cardiovascular risk factors (178).

One proposed explanation for the positive energy balance in obese children is that they eat more than their normal weight peers (179). However, contrary to this popular belief, most studies have found that obese children reportedly do not eat more than their lean counterparts (179,180). It has been concluded that if differences in dietary intake are found, they are in a paradoxical director with obese children eating less than lean children (170). Other studies do not generally support that obesity is caused by excess energy intake (181-183).

The diet is considered to be important variables in controlling weight status, by findings with respect to dietary intake are inconclusive (184). Because studies in adults (185,186) and children (44,69) have failed to find a significant relation between energy intake and adiposity focus has turned to diet composition. It has been suggested that aspects of diet composition, including high fat and low carbohydrate intakes may play a role in overweight (111). Dietary fat has a higher (9 kcal/g) energy density than other macronutrient (protein & carbohydrate 4 kcal/g). This is thought to be largely responsible for the overeating effect, or passive over consumption as it is often called, experienced by many subjects to high-fat foods .The stimulatory effect of fatty foods on energy intake may also be due to the pleasant mouth-feel of fat when eaten. (179). Flatt (184) proposed that fat oxidation occurs as a function of the difference between total energy expenditure and the oxidation of protein and

carbohydrate. Studies in human have confirm that oxidation of protein and carbohydrate is similar to their intake, whereas fat oxidation dies not parallel fat intake (187), Dietary fat intake is therefore hypothesized to pay a key role in the long term regulation of energy balance and the development of obesity (184). The Committee on Nutrition, American Academy of Pediatrics, currently recommends the following dietary goals for all children older than 2 year of age: and average daily intake of 30% of total calories form fat, less than 10% of total calories from SFAs, and less than 300mg of cholesterol per day. A lower fat intake is not recommended. The committee emphasizes that the primary goal of the diet in childhood is to achieve adequate growth and development of children and adolescents (188).

Physical activity

Childhood obesity is increasing worldwide both in the developed and developing countries. It may be due in part to reduce physical activity levels in children (70). Physical activity patterns have an important influence on the physiological regulation of body weight. In particular, they affect total energy expenditure, food intake and balance.

According to World Health Organization (9) - physical activity is a global term referring to "any bodily movement produced by skeletal muscle that results in a substantial increase over the resting energy expenditure". It has three main components -

a) Occupational work: activities undertaken during the course of work.

b) Household and other chore activities undertaken as part of day to day living.

c) Leisure time physical activity: activities undertaken in the individual's discretionary free time. Activity is selected on the basis of personal needs and interests. It includes exercise and sport:

- Exercise: a planned and structure subset of leisure time physical activity that is usually undertaken for the purpose of improving or maintaining physical fitness.

- Sport: defined differently around the world but usual implies a form of physical activity that involves completion. It may also embrace general exercise and a specific occupation.

The time allocated to each of the three components varies considerably between individual and population.

Sedentary behavior

Physical inactivity or sedentary behavior, can be defined as "a state when body movement is minimal and energy expenditure approximates RMR. However-

d) Physical inactivity represent s more than and absence of activity; it also includes participation in physically passive behavior such as televisions viewing, reading. working at a computer, talking with friends on the telephone, deriving a car, medicating or eating .
e) Physical inactivity may contribute to weight gain through means other than a reduction in energy expenditure. Recent studies in adolescent and adults have demonstrated significant relationship between inactivity and other adverse health practices, such as consumption of less healthy foods and an increased fat intake.

Now a days most work is sedentary in nature. Even the physical work of manual jobs is reduced by machines that do the work (189).

Although obesity and related adipose tissue disorder are major contributing cause of metabolic syndrome other factors undoubtedly augment metabolic risk factors. For instance, physical inactivity can worsen insulin resistance and promote development of metabolic abnormalities (190). Body weight is dependent on the balance between

energy intake, in the form of food and drink, and energy expenditure. Daily energy expenditure consists of resting energy expenditure, the energy required to metabolize food (thermic effect of food), and energy expended as a result of activity. When energy intake and expenditure are in balance, weight remains stable. A net excess in energy, whether through greater intake of lesser expenditure leads to weight gain. In children, some of this extra energy may be used for linear skeletal growth; but in both children and adults net excess energy in take leads to increases in both lean body mass an adipose tissue (103). In general population, therefore physical inactivity and obesity go hand in hand in causation of metabolic syndrome. But the relationship between physical activity and obesity in children is not well established, it is thought that physical activity differences in children insufficiently explain the adiposity differences between normal and overweight children (111,179).

The positive energy balance in obese children is that they exercise less than their lean counterparts (191). Earlier studies also suggested that obese children are less physically active compared with lean children (71,192). Similar data from a number of cross-sectional studies reveal an inverse relationship between BMI and physical activity indicating that obese and overweight subjects are less active than their lean counterparts (9). However, such correlations do not demonstrate cause and effect relationships, and it is difficult to be certain whether obese individuals are less active because of their obesity or whether a low level of activity cased the obesity. Davies et al found that high percentages of body fat were associated with low physical activity level in children (193). The secular trend in the increased prevalence of obesity seems to parallel a reduction in physical activity and a rise in sedentary behavior. Rising et al also reported that low physical activity rates were associated with obesity (194).

Although activity of obese is clearly less than of the non obese, the energy expenditure required for comparable activities is often greater because of the greater weight to be moved. When activity was measured directly as energy expenditure, using a 24-hour record of heart rate correlated with oxygen consumption, the daily energy expenditure of obese American girls did not differ significantly from the non-obese.

Results have been inconsistent and have not generally demonstrated a significant inverse relationship between physical activity and body weight or body fat (64,195).

Television and video viewing

Television is a major source of entertainment, information and influence. Television viewing is one of the leading leisure activities of children today, and has now reached every nook and corner of country. All the population of the country is within the reach of television relay now. In addition to local channels, the city dwellers are also enjoying satellite channels round the clock.

A television viewing has a great impact on various aspects of a child's life. Television viewing enhances cognitive development, and conveys knowledge skills and information to the child. It motivates learning and imparts general awareness (196,179). It promotes both conceptual and concrete thinking, and helps in bringing change in behavior and attitudes. It is a matter of great concern to educationists, planner, psychologists and pediatricians all over the world (196).

However, television viewing has negative impacts too. It may place children in passive roles. To make matters worse, physical activity in childhood and adolescence appears to be on the decline. Watching television and playing computer games cuts into time formerly spent in outdoor play. Urban decay and crime make playgrounds unavailable after school. For safety's sake, children often go home immediately after school and lock themselves indoors. Parents at work cannot oversee their children's activities. Children frequently spend their late afternoons watching television and snacking. It may provide escapism from reading, playing, exercise, study etc (197). Dietz and Gortmaker (198) reported that children in the United States spend, on average, as much time watching television each year as that do attending school.

An alarming increase in obesity in children and adolescents can be explained partly be these changes in society and culture (199). It decreases actual social interaction, and provided antisocial or aggressive and destructive roles for imitation (196).

Television imparts a new morbidity in Pediatrics (200). Televisions viewing and its role in pediatric obesity have begun to receive more attention in the past few years.

Time spent watching television was found to be directly associated with prevalence and incidence of obesity among children (179,198,201). Similar relationships have been reported from a national sample of third and fourth graders and in large samples of adults (201-203). The researchers found that men who viewed more than television 3 hours per day are twice as like to be obese as those who viewed less than 1 hour per day. Although it was unclear whether the men were obese because they watched television or whether they watched more because they were obese. Dietz and Gortmaker suggest that former may be the cause (198). Both Tucker & Friedman (201) and Dietz & Gortmaker (198) concluded that there is a definite link between television and increased risk of obesity. In a number of both cross sectional and prospective studies, television viewing was shown to be a significant risk factor for childhood obesity (111,198,201).

Hours spent watching television and video/computer games in 1996 and 1997 were assessed with an 11 item measure; television and video use in 1995 was measured using a 4 item scales by Rosner and Willette (204). They estimated that the relationship between the 11 item measure and repeated 24 hours recall in 1`97 and calculated a deattenuated (205). Correlation for hours of television viewing per day was r=0.27.

Study (206) shows, on average, back girls spent 30% more hours watching television or videos per week than white girls (36.5 vs. 25.1 hours, respectively). Other studies have demonstrated the link between hour spent watching television and obesity in children and adolescents (198, 207). This excess occupation with television and videos is consistent with black girls' reporting a more sedentary lifestyle (208).

The amount of television viewing has also been related to obesity in both cross sectional and longitudinal investigation. For example, watch hourly increment of television viewing by adolescents has been associate with a 2% increase in the prevalence of obesity (198).

A little is known about the way in which television viewing affects energy balance. Television viewing may be related to dietary intake, physical activity metabolic rate, or some combination of these (179).

Disease conditions

a. Genetic disorder

In human, a number of genetically determined conditions result in excess body weight or fatness e.g. Prader-Willi syndrome or Prader-Labhard-Willi syndrome, Bardet-Biedll syndrome, Laurence -Moon-Biedel syndrome etc. But these cases account for only a very small proportion of the obese population (9,49,209).

b. Endocrinological disorders

A number of endocrinological disorders may contribute to obesity such as hypothyroidism, Cushing's syndrome and hypothalamic tumors, etc. However, these are extremely rare cause of obesity (9,49).

Children with pathological obesity for a very small proportion of the total population of obese children. These obese children usually present with short stature or other symptoms before obesity becomes a major problem (170).

Drug treatment

A number of drugs used in different disease conditions can promote weight gain. A number of drugs listed below which causes weight gain (9).

Drug	Main condition treated or other use
Tricyclic antidepressants, lithium	Depression
Sulfonlylureas	NIDDM
β- Adrenergic blockers	Hypertension
Some steroid contraceptives	Contraception
Corticosteroids	Various disease
Insulin	NIDDM
Cyprohepatadine	Allergy, hay fever
Valproic acid, neuroleptics	Epilepsy
Phenothiazine	Psychosis
Pizotifen	Migraine headache

CONSEQUENCES OF OBESITY

Obesity is a nutritional disorder and an important risk factor for a number of chronic, non communicable, life threatening diseases including type-2 diabetes, cardio-vascular diseases and hormone-sensitive and gastro-intestinal cancers. Risks are also higher for some non-fatal conditions such as back pain, arthritis, infertility, and poor psychosocial functions including learning difficulties. Abdominal obesity is of particular concern as it is associated with greater risks to health than the peripheral fat distribution. There is now extensive evidence that links excessive body weight with overall mortality. The overall relationship between mortality and body mass index (BMI) adjusted for age shows a J shaped curve with an acceleration of the mortality risk above a BMI of 30 (9,24,210,211).

During childhood

Obesity in children impacts on their health in both short and long term. Short term consequences including psychosocial, orthopedic, pulmonary, gastroenterologic, endocrinologic, metabbolic, cardiovascular disorders. Long term consequences of child obesity include risks for cardiovascular disease and death that are independent of adult body weight. The most public health important consequence of its persistence into adulthood with all the associated health risks (3,75,212).

Psychosocial effect

The most widespread consequences of obesity in children are psychosocial. Obese children become targets of early and systematic discrimination. Discrimination begins in early childhood and becomes progressively institutionalized. As they mature, the effects of discrimination are more culture bound and insidious. An important sequel of the widespread discrimination and cultural preoccupation with thinness is concern about weight expressed at young ages. The concern becomes part of the culture and is most pronounced among female dancers and gymnasts. Overweight children are ranked lowest as those with whom they would like to be friends. Furthermore,

children ranging in age from 6 to 10 years already associate obesity with a variety of negative characteristics such as laziness and sleepiness (9).

Social bias, prejudice

Obesity is highly stigmatized in many industrialized countries, in terms of both of the perceived undesirable bodily appearance and of the character defects that it is supposed to indicate. Even children as young as 6 years of age describe the silhouette of an obese child as 'lazy', 'dirty',' stupid', 'ugly', 'liar' and cheat more often than drawings of other body shapes (9).

Self esteem

However, there is little evidence to suggest that self-esteem is significantly affected in obese young children. Among teenagers, however, there is an inverse relationship between body weight and both overall self-esteem and body image. However, obese adolescents develop a negative self-image that appears to persist into adulthood. One explanation for this apparent discrepancy between children and adolescents is that self-image is derived from parental messages in young children and increasingly from the culture as children (9).

School performance

Preadolescent children associate the shape (or silhouette) of an overweight body with poor social functioning, impaired academic success and reduced fitness and health. An increased prevalence of behavioral and learning difficulties has been observed among children who are gaining weight rapidly. Whether learning difficulties reflect the subtle effects of sleep apnea or psychosocial problem within families requires additional studies (9,23).

Growth

Overweight children tend to be taller, have advanced bone ages, and mature earlier than non overweight children. Longitudinal studies of children who became overweight have shown that herewith gain accelerates or follows shortly after excessive weigh gain. Early maturation, determined by bone age, peak height velocity, and age of menarche, is associated with increased fatness in adulthood (213,214).

Cardio-vascular risk factors

Dyslipidemia, hypertension an insulin resistance are frequently seen in obese children (215,216) and dyslipidemia appears to be related to increased abdominal fat accumulation. The characteristic pattern observed consists of elevated serum low-density lipoprotein (LDL) cholesterol and triglycerides and lowered high-density lipoprotein cholesterol levels (9). Central fat distribution, perhaps through its effect on insulin levels, appears to be an important mediating variable between lipid level and obesity. Elevated serum lipid and lipoprotein levels, blood pressure and plasma insulin in childhood are all carried over into young adulthood, obesity status in childhood at baseline being a significant predictor of adult values (217,218).

Glucose Intolerance

Because obesity is tightly linked to diabetes in animal models of obesity, it is not surprising that glucose intolerance and diabetes are among the most frequent morbid effects of adult obesity. Although few data are available about the frequency of glucose intolerance among obese children and adolescent, the recent observation that non-insulin dependent diabetes mellitus (NIDDM) accounted for one third of all new cases of diabetes in Cincinnati in 1994 suggests that the morbid effect of the recent increases in the prevalence of obesity have already begun, The incidence of NIDDM among adolescents in Cincinnati appears to have increased 10 fold since 1982. the

mean body mass index (BMI) was 37 among the newly diagnosed NIDDM adolescent patients.

Acanthosis nigricans describes increased thick ness and pigmentation of skin in intertriginous folds; it is associates with glucose intolerance in children and adolescents. The prevalence of acanthosis nigricans among obese patients may be as high as 25% consistent with Dietz's consecutive estimates from observation of 100 obese children (9,15,219,220).

Hepatic and gastric complications

Hepatic complications with high concentrations of liver enzymes particularly characterized by raised serum transaminase levels represent a frequent obesity associated finding in children and adolescents. In a large Japanese series, >10% of all obese children seen in a general obesity clinic setting had modest increased of liver enzymes, frequently associated with fatty liver, fatty hepatitis, fatty fibrosis, or cirrhosis Hyperinsulinemia also may play a role in the pathophysiology of steatohepatitis. Weight reduction induces a normalization of hepatic enzymes. Gallstones are a less frequent occurrence among obese children (9).

Gastro-oesophageal reflux and gastric emptying disturbances, which affect a minority of obese children, may be a consequence of raise intra-abdominal pressure due to increased abdominal fat (9).

Hypertension

Hypertension occurs with low frequency in children. In the best community based study of this problem among more than 6600 school children 5 to 18 years of age only 1 % had persistently elevated blood pressure. However, almost 60% of the children with persistently elevated blood pressure had relative weight >120% of the median for their sex, height, and age. Based on the estimated prevalence of obesity in this sample

persistently elevated blood pressure occurred approximately nine times more frequently among the obese (9,38).

Orthopedic complications

It is well documented that obese children can suffer form orthopedic complications, The more serious of these include slipped capital femoral epiphysis and Blount disease (a bone deformity resulting form overgrowth of the tibia) while more minor abnormalities include knock knee (genu valgum) and increased susceptibility to ankle sprains (9).

Sleep apnea

Sleep apnea is another consequences of childhood obesity is characterized by partial airway obstruction for which aggressive therapy is warranted. Some 7% obese children suffer from sleep apnea). Obstructive sleep apnea can cause hypoventilation and even sudden death in severe cases (9,40,221).

Pseudomotor cerebri

Pseudomotor cerebri is a rare condition linked to raised intracranial pressure; it requires immediate medical attention. Upto 50% of children who present with this syndrome may be obese, but the onset of symptoms does not appear to correlate with weight gain (9).

Persistence into adulthood

The most important long-term consequence of childhood obesity as a public health problem is that it commonly tracks into adulthood with all the associate health risk. There is 70% risk of its persistence into adulthood. Persistence is more when its onset is in late childhood or adolescence and when the obesity is server (23,31,76). After the age of 5 yeas, there is a statistically significant tendency for fatty children to

become fatty adults. Must reported that the risk of adult overweight is two-fold greater for individuals who were overweight as children compared with individuals who were not overweight. Risk factors for adult diseases that are associated with childhood obesity may persist into adulthood or increase in prevalence if weight gain occurs (214).

Although critical periods appear to exist for the onset of obesity in childhood, the relative contribution of obesity that begins in the prenatal period, the period of adiposity rebound or in adolescent to the prevalence of adult obesity and its associated complication remains unclear. Odds ratios represent the most useful clinical expression of the likelihood that obesity will persist. In the Fel's sample, the odds ratio for obesity at age 35 years increased from ~2 for males and females who were obese between the ages of I and 6 year to 5 to 10 for children who were obese at ages 10 to 14 years. The odd ratios for subsequent obesity at ages 15 to 18 years ranged form 8 to 57 for males and from 6 to 35 for females. In other studies that tracked obesity, correlation coefficients have ranged from r = .54 to .72 depending on the group sampled. However, these correlation coefficients may be low because of the inclusion of a broad age range. Among the studies that have examined the effects of childhood-onset obesity on ages among children with and without sequelae of obesity has not been evaluated. Most of the adverse effects of obesity are rare in children but common in adults. No study has yet examined the future morbidity of overweight children who lose weight and subsequently gain weight in adulthood. Whether obesity present in childhood or whether treatment of obesity in childhood independent of its effects on weight has an effect on health or psychosocial function therefore remains uncertain (214).

Effects of childhood-onset obesity on adult obesity, at least one has shown that the prevalence of morbid obesity in adults appears to occur with a greater prevalence among individuals who were obese as adolescents. However, only 15% of to 30% of obesity in adults is a result of obesity that was present in childhood or adolescence (222,223).

During adulthood

Relative risk of obesity associated health problems

The non-fatal but debilitating health problems associates with obesity include respiratory difficulties, chronic musculoskekletal problems skin problems and infertility.

Approximate relative risks among the obese for several health problems have recently been reported by WHO (9) -

Table: Health risk associated with obesity

Greatly increased (RR>>3)	Moderately increased (RR 2-3)	Mildly increased (RR 1-2)
Type 2 diabetes Gallbladder diseases Dyslipidemia Metabolic syndrome Breathlessness Sleep apnea	Coronary heart disease Hypertension Oesteoarthritis (Knees and hips) Hyperuricemia and gout	Cancer (breast cancer in postmenopausal women, endometrial cancer and colon cancer) Reproductive hormone abnormalities Polycystic ovary syndrome Impaired fertility Low backpain Increased anesthetic risk Fetal defect associated with maternal

Obesity-related mortality

There has been much controversy about the relationship between obesity and mortality. While a number of studies have found a U or J shaped association, with higher mortality rates at both the upper and lower weight ranges, some have shown a gradual increase in mortality with increasing weight, while others have reported no association at all (9).

Chronic diseases associated with obesity

Cardiovascular disease

CVD encompasses CHD, stroke and peripheral vascular diseases. CHD and stroke account for a large proportion of deaths in men and women in most industrialized countries, and their incidence is increasing in developing countries. Obesity predisposes an individual to a number of cardiovascular risk factors including hypertension, raised cholesterol and impaired glucose tolerance. However, long-term prospective data now suggest that obesity is also important as an independent risk factor for CHD related morbidity an mortality . The Framingham Heart Study ranked body weight as the third most important predictor of CHD among males, after age and dysilipidemia (9). Similarly, in women, a large-scale prospective study in the USA found a positive correlation between BMI and the risk of developing CHD. Weighty gain substantially increased this risk (224). These findings are consistent with data from other countries. A 15 year follow up study of 16000 men and women in eastern Finland concluded that obesity is an independent risk factor for CHD mortality in men and contributes to the risk of CHD in women. CHD risk associates with abdominal obesity than in those with excess fat around the hips and thighs. In addition, mortality form CHD has been shown to be increased in overweight individuals, even at body weights only 10% above the average (225).

Interestingly, Asian Indians have the highest rates of CHD of any ethnic group studied, despite the fact that nearly half this group is lifelong vegetarians. CHD occurs

at an early age and generally follows a server and progressing course. Although the prevalence of classic risk factors is relatively low, there is a substantial prevalence in this population of high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, high lipoprotein (a) levels, hyperinsulinemia and abdominal obesity. These appear to constitute weight-related risk factors in this population that may, in particular, reflect the central distribution of body fat (9).

Hypertension and stroke

A positive linear relationship between adiposity and blood pressure is well documented. Both systolic and diastolic blood pressure increase with BMI and the obese are at higher risk of developing hypertension than lean individual. The risk of developing hypertension increase with the duration of obesity, especially in women, and weight reduction leads to a fall in blood pressure (9).

The reason for the association between increased body weight and elevated blood pressure is unclear. One possibility is that obesity is associated with higher circulating levels of insulin (a consequence of insulin resistance) and consequently with enhanced renal retention of sodium, resulting in increased blood pressure (75). As exercise is known to improve insulin sensitivity this would perhaps explain why exercise also reduces blood pressure. Other possible etiological factors include elevated plasma renin or enhance catecholamine activity (9,226).

Cancer

A number of studies have found a positive association between overweight and the incidence of cancer particularly of hormone dependent and gastrointestinal. Greater risk of endometrial, ovarian, cervical and postmenopausal breast cancer have been documented for obese women, while there is some evidence for and increased risk of prostate cancer among obese men. Lew & Garfinkel revealed that the principal cancer sites of excess mortality associated with obesity were cancer of the colon and rectum among men; and gall bladder, billiary passage, breast, cervix, endometrium, uterus

and ovary among women. The increased incidence of these cancers in the obese individuals greater in those with excess abdominal fats and is thought to be a direct consequent of hormonal changes. The incidence of gastrointestinal cancer, such as colorectal and gall bladder cancer, has also been reported to be positively associated with body weight or obesity in some but not all studies, and renal cell cancer had consistently been associated with overweight and obesity, especially in women. In addition to overall obesity, intra-abdominal fat distribution and adult weight gain have been independently associated with and increased risk of breast cancer. For example, it has been reported that an increase in intra-abdominal fat accumulation increases the risk of postmenopausal breast cancer, independently of relative weight and particularly when there is a family history of the disease. Furthermore, weight gain during adulthood has consistently been associated with increased risk of breast cancer, even in cohort studies that showed no association between baseline relative weight and subsequent risk of breast cancer (9,227).

Diabetes mellitus (Type 2 diabetes)

A positive association between obesity and the risk of developing NIDDM has been repeatedly observed in both cross sectional (228-230) and prospective studies (231,232). The consistency of the association across populations despite different measure of fatness and criteria for diagnosing NIDDM reflects the strength of the relationship. Due to the additive effect of different risk factors and determinants, individuals with high levels of non obesity risk may develop Type 2 diabetes without becoming obese, while in other cases obesity alone may be sufficient to precipitate Type 2 diabetes (15).

Gastro-intestinal diseases

Gall bladder disease is the most common form of digestive disease in obese individuals of all age groups and in both sexes (9,15). Rimm et al in a study of 73,532 obese women in USA and Canada reported a 2.7 fold increase in the prevalence of gall bladder disease. Obesity is a risk factor for gallstones. Gallstones occur three to

four times more often in obese compared with non-obese individuals, and the risk is even greater when excess fat is located around the abdomen (9).

Supersaturation of the bile with cholesterol and reduced motility of the gallbladder, both of which are present in the obese, is thought to be factors underlying gallstone formation. Furthermore, since gallstones enhance the propensity to gallbladder inflammation, acute and chronic cholecystitis is also more common in the obese, Biliary colic and acute pancreatitis are other potential complications of gallstone.

Liver abnormalities, in particular fatty infiltration, are often found in the obese. Associated abnormal liver enzymes may improve with weight loss (15).

Endocrine and metabolic disturbances

Endocrine disturbances

Recent research has shown that adipocytes (fat cells) are more than just fat depots. They also function as endocrine cells producing many locally and distantly acting hormones, and as target cells for a great many hormones. Altered hormonal patterns have been observed in obese patients, especially in those with intra-abdominal fat accumulation. Common hormonal abnormalities associated with intra-abdominal fat accumulation are listed in a table below (9) -

Common hormonal abnormalities associated with intra-abdominal fat accumulation
Insulin resistance and increased insulin secretion
Increased free testosterone and free androstenedione levels associated with decreases sex hormone binding globulin (SHBG) in women
Decreased progesterone levels in women
Decreased testosterone e levels in men
Increased cortisol production
Decreased growth hormone levels.

Metabolic disturbance

Dyslipidemia is common among the obese and characterized by raised plasma triglycerides and lower HDL-cholesterol concentration. This metabolic profile is must often seen in obese patients with a high accumulation of intra-abdominal fat and has consistently been related to an increased risk of CHD. Excessive intra-abdominal fat accumulation is also associated with a greater proportion of small, dense low-density lipoprotein (LDL) particles. The high proportion of these small dense LDL particles may be the result of metabolic disturbances related to the accompanying high triglyceride or low HDL-cholesterol level. Indeed, the hypertriglyceridemic state may be the combined result of an increased production and a reduced breakdown of triglyceride rich lipoproteins. This process results in lower HDL-cholesterol. The triglyceride rich LDL is then enzymically degraded by hepatic lipase to produce small, dense LDL particles. A large proportion of these particles cannot be identified simple by the measurement of total or LDL cholesterol levels because these cholesterol levels are frequently in the normal range in obese individuals. A better indicator of small, dense LDL particle levels is and elevated ration of LDL- apo B to LDL cholesterol. Impaired fat tolerance (i.e. Prolonged and/or exaggerated lipemai following fat ingestion) is now also recognized as a component both of insulin resistance and of atherogenic lipoprotein phenotype (9,233).

Oesteoarthritis and gout

Obesity is associated with the development of oesteoarthritis and gout and, in obese middle aged women at or after menopause, pain at the medial aspect of the knee (adipose dolorosa juxta articulates) Possible factors under lying relationship between obesity and oesteoarthritis include mechanical stresses related to the increased load carried by the obese, metabolic charges associated with increased fatness, and dietary elements (e.g. high fat content) related to the development of obesity. The data indicate that mechanical damage is usually the cause. The increased risk of gout associated with obesity may be related to the accompanying hyperuricemia, although central fat distribution may also be involved, particularly in women (9,234,235)

Pulmonary function

Obesity has an adverse effect on respiratory function and structure, leading to physiological and pathophysiological impairments. Sleep apnea is a common problem among obese individuals. The work of breathing is increased in obesity, mainly as a result of the extreme stiffness of the thoracic cage consequent on the accumulation of adipose tissue in and around the ribs, abdomen and diaphragm (9,15).

Psychosocial problems

Obesity particularly among women and children is often confronted with social prejudice and discrimination (15). Obesity is highly stigmatized in may industrialized counties, in terms both of the perceived undesirable bodily appearance and of the character defect that it is supposed to indicate. The true social and economic costs of the non-fatal health consequences of obesity may therefore be seriously underestimated (9).

Body shape dissatisfaction

Many obese people have an altered body image, i.e. they see their bodies as ugly and believed that others with to exclude them from social interaction this occurs most often in young women of middle and upper socio-economic status, among whom obesity is less prevalent, and in those who have been obese since childhood (9).

Female reproductive health

Gynecological

Obesity is related to several gynecological disorders including polycystic ovary syndrome (PCOS) infertility and general menstrual disorders. Obese women have a higher risk of obstetric complications (9,15,236).

Obstetric

The relative risk of antenatal complications in women whose pre-pregnancy weight was at least 135% of ideal birth weight was reported increased 6.6 fold for the development diabetes, 1.9 fold for pregnancy induced hypertension and 1.4 fold for urinary tract infections (9,15,236).

ECONOMIC COSTS OF OBESITY

The economic costs of overweight and obesity are important issues for health care providers and policy makers alike. To date, there have been only a few attempts to quantify the economic burden of obesity related morbidity and mortality (9).

The economic costs of obesity have been assessed in several developed countries and are in the range of 2-7% of total health care costs. Although there have been no studies of the economic impact of obesity in developing countries, the escalating economic burden of adult NCDs in such countries has already been recognized by a number of international agencies including WHO and the World Bank. The real costs of therapy in developing countries exceed those in developed counties because of the extra burden associated with the use of scarce foreign exchange to pay for imports of expensive equipment and drugs, as well as the need for the specialized training of staff. In view of the existing burdens of endemic deficiency disorders and infectious diseases, obesity prevention is not only crucial but also the only sensible approach to planning public health policies in developing countries. The economic cost (9) of obesity includes -

Direct costs

The cost to the community resulting from the diversion of resources to the diagnosis and treatment of disease directly related to obesity, as well as from the cost of obesity treatment itself (including the cost of providing health care services to patients and their families, and the cost of service providers).

Intangible costs

The cost to individual arising from the impact of obesity on quality of life generally and on health specifically.

Indirect costs

The welfare and economic benefits lost to other members of society through a reduction in the goods and services produced i.e. the impact of the reduced quality of life of obese individual on the productive potential available to the rest of society. These cost are usually measured as the production lost through work related absenteeism and premature death.

Direct costs of obesity in some developed countries are mentioned below (9)-

Country	Year	Population (millions)	Cost (per year)
New Zealand	1996	3.6	NZ $ 135 million
Australia	1994	18.4	AUD $ 464 million
Netherlands	1995	15.7	NG 1 billion
France	1995	58.0	FF 12 billion
USA	1998	274.0	US $ 51.6 billion

ASSESSMENT OF OBESITY

Obesity is a situation of excess fat accumulation in the body and the diagnosis of obesity should ideally be based on an accurate direct or indirect measure of the total body fat mass (237). It is difficult to compare levels of obesity across studies because the estimates are based on different reference data different cut-off points, and different indicators (238). Therefore, determining the correct measure to assess obesity is important because ideal measurements of body fat in populations should be reliable and correlate well with body fat in both sexes and across all ages and ethnic groups. Furthermore, because individuals of different heights or body builds may have similar fat masses yet substantially different proportions total body weight (percentage body fat) is the most relevant measure against which anthropometric measurements should be correlated (239). Absolute cut-off points for assessing obesity not only may vary between reference standards (240-242) but also that within a given population, reference standards may vary in time (242,243) due, for example to secular trends in body fatness or body height.

Weight for Height

Weight for height is a sensitive index of current nutritional status. It is relatively independent of age between one and ten years enhancing its usefulness in areas where the ages of the children are uncertain. Its expression as percentage wt/ht has the advantage of integrating both weight and height in the same measurement and can be easily calculated (244-248).

The high reliability of measurements of height and weight suggests that a variant of weight-for-height provides a more reliable measure of adiposity that can be used to compare adiposity within and between populations (239). The use of weight for height to evaluate obesity is appropriate although this measurement is different from the triceps skinfold measurement used by Dietz and Gortmaker (249). Dietz has noted that Roche et al provided valuable basic comparisons between underwater weighing and triceps skinfold, weight-for-height percentile and relative weight for grading body

fatness, but sample size in the Roche study were small and there was wide variability in the data. Consequently, all three anthropometric measures appeared similar in their ability to estimate percent body fat (250,251). Although percent expected weight for height (% EW) indicates excess weight not excess fat, for practical purpose more than 120% EW indicates significant obesity. To assess obesity many authors have used a cut-off level more than 120% (1,3-7,51,252).

Body composition

Evaluation of body composition is essential for an understanding of the growth and development, and various health disorders including malnutrition of human subjects. Human body consists of three chemically distinct compartments-viz. fat mass (FM), fat free mass (FFM) and total body water (TBW). The chemical composition of FFM is assumed to be constant with a density of 1.1 gm/ml at 37^0 C, TBW of 72-74% of FFM, and FM, stored as triglycerides, has density of 0.9 gm/ml at 37^0 C. Based on these body compartments all body composition methods were developed. Research to establish indirect methods for assessing body composition started by Behnke in 1940. Subsequently, a variety of methods have been introduced (74). So, reliable, non-invasive, rapid and accurate methods for determining body composition are, therefore, highly desirable (253). Some of these methods are described below-

Body Mass Index (BMI)

BMI is the body weight (in Kg) divided by the power of height in meter squared. Individuals with higher BMI are considered overweight, even obese, and those with lower indices are classified as undernourished. Body mass index has achieved international acceptance as a standard for the assessment of obesity in adults and correlates with body fat(r=0.7-0.8) (252,254)

Although, BMI is still widely used to assess the extent of overweightness or leanness. It is not always an accurate index of body composition. For instance, a human subject may weight much more than the average weight for height standards. Yet still be

under fat in terms of body's total amount of fat. The extra weight could simply be due to additional muscle mass (74). Among children, BMI is not a measure of fatness because levels of fatness can change at a constant BMI. This happens because BMI reflects frame size, leg length, and the amount of lean tissue as well as the amount of fat (255,256).

Skinfold thickness

Skinfold thickness measurements are said to provide an estimate of the size of the subcutaneous fat depot, which in turn provides an estimate of the total body fat. Such estimates are based on two assumptions- i) the thickness of the subcutaneous adipose tissue reflects a constant proportion of the total body, and ii) the skinfold sites selected for measurement, either singly or in combination, represent the average thickness of the entire subcutaneous adipose tissue (257). The measurement of skinfold thickness can be made at several places of a human body, such as chest, axila, triceps, subscapula, abdomen, thigh and suprailiac. There are several mathematical equation available for the prediction of body fatness, ie. Fat mass, from the skinfold thickness measurement (258). Moreover, since the distribution of fat in the human body could be different from one individual to another, the use of skinfold equation to predict body composition is restricted to position from whom these equation are delivered. Furthermore, the precision of measurement of skinfold thickness is dependent upon the skill of anthropometrist/investigator and the site of measurement (74).

However, even if total body fat mass or total body fat percentage is known, the definite of obesity is to some extent arbitrary i.e. a body fat percentage exceeding 25%, 30% or 35% but estimates of body fat percentage may be used as a basis for a more consistent diagnosis of obesity (237).

As in adults skinfold thickness measurements in children give an estimate of the size of the subcutaneous fat layer. The problem is to relate this estimate to an accurate estimate of total body fat mass or total body fat percentage. Skinfold thickness

measurements can be easily and reliably obtained in children and there is an abundance of data on skinfold thicknesses in children of a wide age range (240,259).

Bioelectrical Impedance Analysis

Bio-electrical impedance analysis (BIA) is a widely used method for estimating body composition that is relatively simple, quick, non-invasive and highly reliable. It is highly correlated with total body water and fat free mass and has been validated in children. Despite a general public perception that BIA measures body fat, the technique actually is used to measure the electrical impedance of body tissues, which provides an estimate of total body water (TBW). Using values of TBW derived from BIA, one can then estimate fat-free mass (FFM) and body fat. In addition to its use in estimating adiposity, BIA is being used in the estimation of body cell mass and TBW in various clinical conditions (260).

In BIA, a small alternating current is applied to the body, and the resistance or impedance of the body to that current is measured (261). Of the different body components, only water with its dissolved electrolytes can conduct the current [6]; thus, body impedance is a measure of body water (262).

Alternating electrical current flows through the body by several different physical mechanisms. Current flows through physiologic fluids by the movement of ions. This movement is opposed by viscosity and other effects, which can be modeled electrically as a resistance. In addition, the applied current will charge cell membrane and other interfaces, which can be modeled electrically as capacitors. Thus, the impedance of the human body (and of material in general) can be modeled by a combination of capacitive and resistive elements (263).

A capacitance and a resistance can be combined in two ways in series or in parallel. In either case, the components can be chosen to produce the same impedance of the circuit; however, the component values will differ (263).

The impedance (or resistance and capacitance) of the human body is an extensive quantity, which like weight or volume depends on body size. It is also useful to consider the intrinsic electrical properties of body tissues, expressed as intensive quantities that (as with temperature) do not depend on body size. Two intensive electrical properties of matter are conductivity (θ) and permittivity (ε). Conductivity is a measure of the amount of current that will flow when an electric field is impressed across the material; permittivity is a measure of the amount of charge that will be induced at interfaces such as cell membranes by the field. Conductivity and permittivity are defined in terms of a parallel-equivalent model because the two electrical processed (conduction of current and accumulation of charge at interfaces) occur independently, i.e. in parallel. Resistivity (ρ), which is expressed as Ω m, is the inverse of conductivity and is also defined with respect to a parallel circuit model. Most discussion of the electrical properties of tissue in the biophysical literature are in terms of conductivity; we currently use resistivity because of its more common use in the whole-body impedance literature (263).

Impedance (Z) is the frequency-dependent opposition of a conductor, animate or inanimate, to the flow of an administered alternating electrical current. This opposition has two components or vectors termed resistance (R) and reactance (Xc). Resistance is pure opposition of the conductor to the flow of current. In practical terms, R is the inverse of conductance or the ability of an object to transmit an electrical current. According to Ohm's Law, R equal the voltage (E) divided by current (I), or $R = E / I$. In a biological conductor, I is transmitted principally by ions in aqueous solution. Thus, the amount of electricity that can be conducted, or the conductivity, is proportional to the number of ions in a volume of a conductor (264).

Reactance is the reciprocal of capacitance, or the voltage stored by a condenser for a brief period of time. In the body, reactance is associated with several types of polarization (separation of charges or electrochemical gradients) that may be produce by cell membranes and tissue interfaces. Capacitance causes the administered current to lag behind the voltage and creates a phase shift that is represented geometrically as the phase angle (ϕ) or the arc tangent of the ratio of X/R (264).

Assuming that the body has the simple geometric shape of a cylinder, the theoretical relation between body water and impedance is of the form TBW= ρ x H^2 /Z, TBW is total body water, ρ is specific resistively of the body fluid is body height, and Z is the measured impedance value. When TBW is assumed to be a fixed part of the fat-free mass, BIA also enables the assessment of FFM (261,262).

Prerequisite factors or factors influencing impedance measurement are -

A concern with any indirect method for the assessment of human body composition is the requirement to minimize interferences associated with the measurement technique. With the BIA, some factors have been identified that affect the validity of measurement-

1. **Measurement technique**

   The subject should be placed in supine position on a non-conductive surface. The arms and legs should not be in contact with other body parts because disturbance of the applied electric current, and hence the electric field, will occur, resulting in artifactually low *R* and *Z* values (264).

2. **Placement of electrodes**

   Standardized placement locations enhanced reproducibility of the measurements and facilitated the general use of the method. The use of sites on the distal limbs specifically the dorsal surfaces of the hands and feet has been suggested.

3. Environmental factors

A variety of environmental factors disrupt circulatory homeostasis and affect impedance measurements of the body. All the factors significantly affect the R measurements. Some important factors (260,261) are mentioned here-

a) Acute exercise and heat exposure

Acute exercise and heat exposure increase peripheral blood flow compared with euthermic conditions result in decreased control R values with minimal effect on Xc.

b) Fluid intake and acute cold exposure

Voluntary restriction of fluid intake and acute cold exposure tend to increase R values.

Exercise can affect BIA measurements by at least three hypothesized mechanisms. *1*) The hemodynamic response to exercise consists of increased cardiac output and blood flow to skeletal muscles. Increased vascular perfusion and warming of muscle tissue will reduce Z and muscle resistivity (ρ). 2) The process of heat dissipation includes increased cutaneous blood flow and vasodilatation, increased skin temperature, and sweating. These changes should also reduce Z. 3) Sensible and insensible fluid looses result in dehydration, loss of TBW, and in increase in Z. Thus, BIA measurements should vary depending on the muscle groups involved and the intensity of exercise changes in skin blood flow and heat production, and amount of fluid loss.

c) Body temperature

Fever causes alteration in the BIA measurements.

d) Dehydration

Dehydration significantly affects the prediction of TBW and ECF.

e) Consumption of food or beverage:

Consumption of food or fluids before BIA measurements are made could influence Z by changing TBW and ECW volumes. However, depending on the timing of measurement and the amount of food or fluid ingested. Usually subjects are encouraged to refrain from eating or drinking for a 2-hour period before testing.

CHAPTER IV

HYPOTHESIS

HYPOTHESIS

Socio-economic status, dietary habit and life style factors are associated with the development of childhood obesity.

CHAPTER V

OBJECTIVES

OBJECTIVES

a. General

1. To identify the socio-economic status, dietary habit, life style factors and parental characteristics for the development of childhood obesity.

b. Specific

1. To assess the association of obesity with age, sex, birth order and family size distribution .

2. To assess the association of socio-economic status and environmental factors with the development of childhood obesity.

3. To assess the association of parental obesity with that of children.

4. To assess the food habits and life styles of children.

5. To measure body fat content by indirect method.

6. To assess the lipid profile of a representative subgroup of children and its relationship with their parents.

CHAPTER VI

MATERIALS AND METHODS

MATERIALS AND METHODS

Study design : A case control study preceeded by a cross sectional survey.

Study Location : Dhaka City

All 12 thana (Civil administrative-subdistrict) at-

a) 12 Government primary schools
b) 23 Private elementary schools
c) 4 Hospitals
d) 8 Maternal and Child Health Centres
e) 12 Immunization Centers (on National immunization days)

Study period : 1999-2001

Study population: 2-10 years age group

Inclusion criteria

1. Case : Children in terms of weight for height more than 120 % of the NCHS/WHO reference (obese).

   Control: Non obese children as controls matching age and sex .

2. Age: 2-10 years

3. Sex: Both sexes.

4. Parental consent

Exclusion criteria

1. Child suffering from any illness for last one month.
2. Handicapped children.
3. Child taking drug like steroid, cyproheptadine, phenothiazine etc. causing weight gain.
4. Parents unwilling for invasive investigations.

Estimation of sample size

Referring the study (22) and considering the prevalence rate 10 % or less, using the ***Inverse sampling method for rare items***, 5000 children of 2-10 years age group was surveyed to obtain the cases (265). An equal number of non-obese children were selected randomly matching age and sex as controls. Sample size has been calculated as below-

P = Proportion of obese children ≤ 0.10 (10%)

$$V(p) = \frac{mp^2Q}{(m-1)^2}$$

$$\hat{p} = p = \frac{m-1}{n-1}$$

Here, m = number of rare items = number of obese children

n = sample size

$$CV(p) = \frac{(mQ)^{\frac{1}{2}}}{m-1} \qquad < \sqrt{m}/(m-1)$$

m = 500

Here,

P = 0.10

Q = 1- 0.10 = .90

$$p = \frac{m-1}{n-1}$$

$$= 4990$$

$$n-1 = \frac{m-1}{p}$$

$$= \frac{500-1}{0.10}$$

n = 4990+1

= 4991

≈ 5000

∴ Sample size = 5000

Sampling:

A three stage probability proportionate to size (PPS) cluster sampling method was used to obtain representative sample of 5000 children aged 2-10 years using the current census data. The design provided a systematic sampling based on the current population census of 12 thanas of Dhaka city (104,266-270).

Stage-I : Sampling involved a stratified random selection of schools, health centers or hospitals. If a school health center/hospital declined to participation, it was replaced by another school health center/hospital

Stage-II : Class rooms/section of the class out patient department/unit were randomly selected from each school/health center or hospital.

Stage-III : At school randomly selected students and at health center or hospital every alternate child was sampled.

Survey included a medical history and physical examination to assess the eligibility of the subjects for the study.

Of 5000 children, 380 (7.6%) were identified as obese using the criterion of weight for height >120% of NCHS/WHO reference as a cut-off point. An equal number of non obese children (<120% of NCHS/WHO reference as controls were selected randomly matching age, sex and stratum of sampling (51).

Due to shifting of accommodation including transfer of place of works of parents, unwillingness to participate in the study by the parents and illness of either parents and/or children (during case- control study period) a total of 160 obese children could not be included in the study. Even with this exclusion, the total number of cases and controls were within statistically sound sample size [α =0.05 two sided, B = 0.20, Po

0.15 and R=2 and the estimated sample size ≈ 207 using table for estimation sample size for case- control study] (271).

Finally, the case control study part of the study was conducted among the 220 cases and 220 controls. Data were collected at the school or laboratory or at prefixed places. For this, parents were communicated through school authority or by the investigator himself. A semi-structured questionnaire was used for identifying the factors associated with the development of childhood obesity. Data on socio-economic, demographic, anthropometry, body composition, 24 hour dietary recall, 24 hour physical activity recall and lipid profile were collected. Information was also collected from parents of both cases and controls.

Data collection

Socio-economic and demographic information

Age of the child as years and months (eg. 6 years 6 months) have been recorded where designated month is the last completed month (272). Later on, the age of the both cases & controls were checked by the parents.

Information on demographic characteristics like birth order, family structure and family size and other family information like monthly total family income, monthly family expenditure on foods were collected through interviewing the parents.

Measurement of height

Height was measured with to the nearest 0.5 cm (without shoes & cap/hat or false hair that would interfere with height measurement) using locally made wooden stadiometer (as per WHO guidelines) (273). The heels together, toe apart at a 45^0 angle, and head in a Frankfort horizontal plane (274).

Measurement of weight

Weight was measured using a pre-calibrated bathroom scale (272) with the subject wearing light indoor cloths, with emptied pockets and without shoes. Since the measurements were done in summer, the cloths of parents & children were very light and no weight correction was needed. Weight was measured to the nearest 0.1kg (275).

Assessment of energy intake

A 24-hour dietary recall was used to asses the dietary intake of the both cases and controls. The parents and their child were interviewed together to complete the recall all foods and beverages consumed by the child during the previous 24 hours- a typical day of a week. Mothers were the primary source of recall, but spouses were encouraged to collaborate in providing information about the child's dietary intake (272,275). In case of non-housewives mothers, caregiver of the child participated to provide information on child's dietary intake.

It has been shown that collaborative recalls of dietary intake have a higher degree of validity than if children or parents independently completed the measure (273).

A semi-structured questionnaire was used to obtain a complete description as possible, including brand name and recipes. Calibrated utensils including containers, glasses, plates measuring cups & spoons and different fruits, fast foods, snacks & vegetables and various types of food models were used as memory aids and/ or to assist the respondent for quantification of portion size of food items consumed (272,274,275). Vitamins and minerals supplement used was also noted, if any (274).

Dietary intake data were analyzed using the locally prepared software.

Estimation of total energy expenditure

A simple, pictorial chart, depicting 24-hour physical activities commonly performed by children was used to assist the recall. These activities including, sleeping, brushing teeth, washing hands & mouth, breakfast, bathing/showering, dressing, walking, climbing/stepping down stairs, sitting in class room, reading, writing, walking to and fro, taking meal, lying, sports like foot-ball, cricket, badminton, carom, skipping, bicycles, house-hold sports, watching television, video, video games, computer uses and other sedentary activities were recorded using a semi-structured questionnaire. The participants checked off the amount of daily time spent on various activities.

Basal Metabolic Rate (BMR) was estimated using Schofield's equation (276).

24-hour activities were classified into light, moderate and heavy according to different published literatures and put appropriate activity factor (Multiples of BMR).Total energy expenditure of 24 hours was calculated by summing the products obtained from the BMR multiplied by appropriate activity factor and the duration of the respective physical activity (276-279).

Estimation television video viewing hours

Television and video viewing was measured among the both cases and controls. They were asked the time spent on watching television, movies, videos each day of previous week-a typical week. They also asked to recall dividing a day into 4 quarter. To assist the recall, the programs of different TV channels published in local newspapers were shown. Parents were also asked to assist the recall. On the basis of daily TV video viewing weekly total hours spent on TV video was calculated (207,280,281).

Assessment of body composition

Measurement of skinfold thickness

Skinfold thicknesses on 4 sites (Biceps, Triceps, Subscapular and Suprailiac) were measured, with the subject seated on a stool, on the right side of the body. The sites selected were as follows. (1) Biceps: over the mid point of the muscle belly with the arm resting supinated on the subject 's thigh (2) Triceps: over the mid point of the muscle belly, mid way between the olecranon and tip of the acromion, with the upper arm hanging vertically (3) Subscapular: just below the tip of the inferior angle of the scapula, at and angle of about 45^0 to the vertical. (4) Suprailiac: just above the iliac crest in the mid axillary line. At these four sites, the skinfold was pinched up firmly between the thumb and forefinger of the left hand of the investigator and pulled away slightly from the underlying muscle before applying the calipers for the measurement(257,282).

The instrument used was the Harpenden Skinfold Calipers (British Indicators Ltd. St Albans, Herts, and England) which exert a constant pressure of 10 gm/sq. mm of face is exerted at all openings of the jaws. The width of the opening is read off on a scale incorporated in the apparatus.

Three measurements of height, weight and skinfold were taken for each variable. The average of the three measurements was used in data analysis.

To compare the body composition derived from bioelectrical impedance analysis as well as different equations for estimating body composition from skinfold thicknesses was used in the study.

To predict the body density (kg/l) from calculated total 4 site skinfold thicknesses following equations (282,283) were used -

a) Equations of Durnin and Rahman -

Body density (Y) from the log of the sum of 4 sites skinfold thickness in mm (X)

For boys , $Y = 1.1533 - 0.0643 X \pm .0.0083$

For girls, $Y = 1.1369 - 0.0598 X \pm 0.0081$

The body fat % was estimated using equation of Siri[2]

Fat % [(4.95/density)-4.5] x 100

Body fat % was also estimated using of a modification of the Siri as proposed by Westrate and Deurenberg (283).

b) Equations of Brook

Predicted density, D

For boys, D=1.1660 - 0.0070 x (log sum of 4 skinfols)

For girls, D=1.1440 - 0.060 x (log sum of 4 skinfols)

Body fat % was estimated using of a modification of the Siri as proposed by Westrate and Deurenberg (283)

c) Equations Deurenberg et al

For boys, D=1.1133 - 0.0561 x (log sum of 4 skinfols)+1.7(agex 10^{-3})

For girls, D=1.1187 - 0.063 x (log sum of 4 skinfols)+1.7(age x 10^{-3})

Body fat % was estimated using of a modification of the Siri as proposed by Westrate and Deurenberg (283)

Bioelectrical Impedance Analysis

A bioelectrical impedance analyzer (Model 101A; RJL Systems, Detroit, Michigan, USA) which applies a 50 kHz oscillating current of 800 UA) was used to measure the body impedance-the total body resistance*(R)* and reactance*(Xc)*.

All subjects were refrained from eating, drinking and exercising for 2h before testing. Furthermore, subjects were instructed to void urine prior (within 30 min of the test) to impedance measurement. Body temperature of each subject was measured orally with a mercury thermometer just before the impedance testing. Dehydration of the subject was also checked. Subject with body temperature above normal was excluded from testing. Clothing was removed from the subject except for dry underwear. Shoes and socks were removed. The child was positioned to lie quietly supine on a non-conducting examination table, with arms slightly apart from the body; legs were separated (abducted approximately 30^0 from the body) so that the thighs were not touching each other (284-287).

Contact areas were scrubbed with alcohol immediately before electrode placement. Electrodes were placed on right side. One pair of electrodes was placed on the back of the hand, the distal one over the distal end of the metacarpal and the proximal one over an arbitrary midline between the radius and ulna at the wrist joint. Another pair was placed at the distal end of the metatarsal and between medial and lateral malleoli at the ankle (286). A distance of 3 cm has been shown to be the minimum required for sufficient separation of electrodes (287,288).

Total Body Water (TBW) was calculated using the following equation, where R is resistance (286).

$$TBW\ (kg) = 0.76 + 0.18\ ht^2/R + 0.39\ wt$$

Fat Free Mass (FFM) was derived from age and sex specific reference value (hydration factor)of the child according to Fomon et al (287). Fat mass was calculated from the difference between body weight and fat free mass.

Assessment of lipid profile

Venous blood was obtained after an over night fast of at least 14 hours (289).Standard procedures and precautions were followed from collection sample to analysis of the samples.

Serum triglycerides & serum total cholesterol were estimated by enzymatic colorimetric test and HDL- cholesterol was estimated by precipitation method. For these, commercially available test kits produced by Human, Germany were used. If TG less than 400mg/dl LDL-cholesterol was calculated using formula-

Total Cholesterol- HDL Cholesterol -TG/5 gm/dl.

In case of TG equal or more than 400gm, LDL-cholesterol was estimated by enzymatic colorimetric test using commercially available test kits produced by Human, Germany (290).

Assessments of body composition using BIA and lipid profile were done among 50 cases and 50 controls and their parents. For this part of the study, either parents suffering from chronic illness were excluded from the study.

Age and sex specific reference values and classification of lipid disorders were used according to different published literatures both for children and parents (291-293).

Data analysis

All statistical analyses were done using SPSS PC. Chi-square tests were done to see the association between categorical variables. Comparisons between groups were performed by t tests and non-parametric, Mann-Whitney tests. Correlations between continuously distributed variables were assessed by using Pearson correlation analyses. Logistic regression was used to examine factors associated with obesity. Other appropriate statistical tests were done for analyses of the data. Two tailed *P values* <0.05 were considered significant.

Limitations of the study

1. Lack of national or international standard reference for classification of obesity in children

2. Lack of comprehensive reference value for estimating energy expenditure in children.

CHAPTER VII

RESULTS

Table 1: Thana and age group wise distribution of study children (n=5000)

Thana	Total	Age of the child (Years)								
		2-2+	3-3+	4-4+	5-5+	6-6+	7-7+	8-8+	9-9+	10
Kotowali	285	29	32	29	36	33	30	31	28	37
Sutrapur	416	43	46	42	53	48	44	45	40	55
Lalbagh	445	46	49	45	57	51	47	48	44	58
Motijheel	718	74	80	72	92	82	76	78	70	94
Ramna	264	27	29	26	34	30	28	29	26	35
Demra	364	38	41	37	46	41	39	39	35	48
Cantonment	404	42	44	41	52	46	43	44	39	53
Gulshan	286	30	32	29	36	33	30	31	28	37
Mohd.pur	428	44	48	43	55	49	45	46	42	56
Dhanmondi	274	28	30	28	35	31	29	30	27	36
Mirpur	818	85	91	82	104	93	87	89	80	107
Tejgaon	298	31	33	30	38	34	32	32	29	39
Total	**5000**	**517**	**555**	**504**	**638**	**571**	**530**	**542**	**488**	**655**

Table shows, the thana and single age distribution of study population.

Figure 1 : Sex distrition of children of 2-10 years(n=5000)

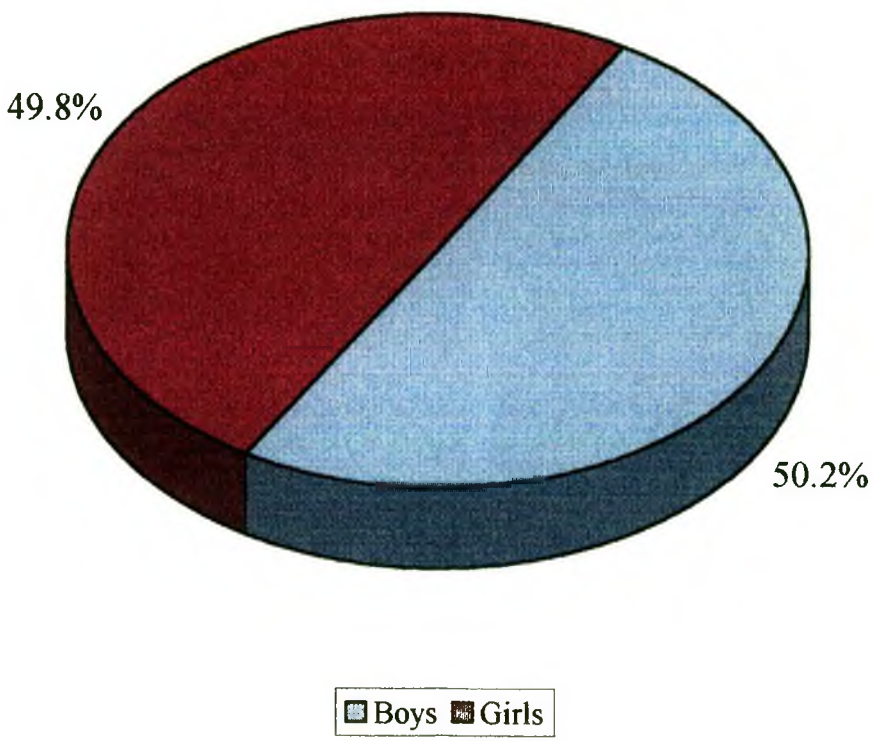


Figure shows that, out of total 5000 children of 2-10 years age group, 2510 (50.2%) were boys and 2490 (49.8%) were girls.

Figure 2 :Nutritional status of the study children (n=5000)

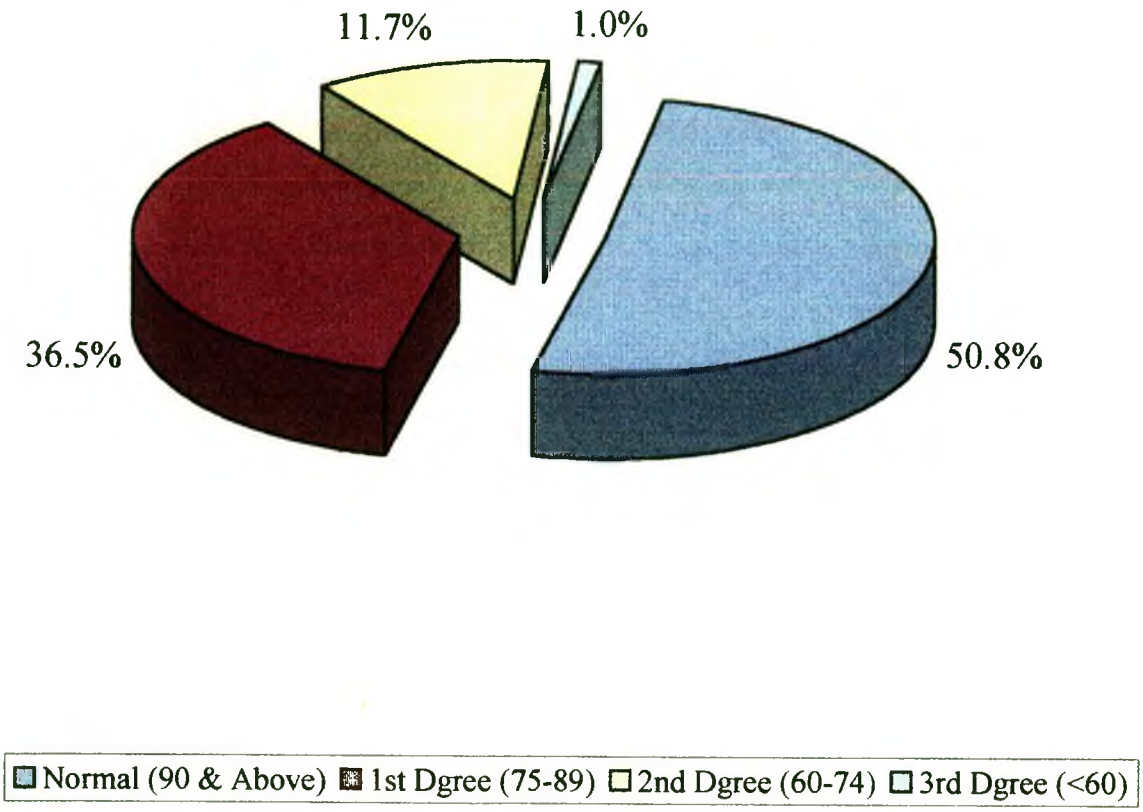


Figure shows that, according to Gomez classification (wt/age), of 5000 children some 51% were nutritionally normal. 36.5%, 11.7% and 1% children were suffering from 1st degree, 2nd degree and 3rd degree malnutrition respectively.

Table 2: Nutritional status of children by SD scores (n=5000)

Indicators	-3.00 & below	-2.99 to -2.00	-1.99 to -1.00	-0.99 to +0.99	+1.00 to +1.99	+2.00 & above	Total
Ht/Age	150 (3.0%)	388 (7.8%)	928 (18.6%)	2662 (53.2%)	598 (12.0%)	274 (5.5%)	5000 (100%)
Wt/Ht	110 (2.2%)	594 (11.9%)	1434 (28.7%)	2196 (44.0%)	378 (7.6%)	280 (5.6%)	5000 (100%)
Wt/Age	93 (1.9%)	635 (12.7)	1500 (30.0%)	2185 (43.7%)	355 (7.1%)	232 (4.6%)	5000 (100%)

Table shows nutritional status of 5000 children of 2-10 years age group, using SD scores.

Table 3:Distribution of weight, height, Z scores and median values of children (n=5000)

Age group		Wt(kg)	Ht(cm)	HAZ	WAZ	WHZ	HAM	WAM	WHM
24-71 months (n_1= 2216)	Mean	14.60	98.17	-0.43	-0.81	-0.63	98.29	92.15	94.93
	SD	3.77	11.87	1.64	1.34	1.25	6.53	16.00	1206
	Skew	1.26	0.07	0.09	0.93	0.72	0.09	1.42	1.09
71-120 months (n_2= 2784)	Mean	24.63	126.03	-0.17	-0.48	-0.64	99.26	96.03	96.73
	SD	7.18	9.74	1.21	1.44	1.51	5.44	23.60	16.80
	Skew	1.20	0.09	0.09	0.72	0.77	0.10	1.17	1.31
24-120 months (n= 5000)	Mean	20.19	113.68	-0.28	-0.63	-0.63	98.83	94.31	95.93
	SD	7.73	17.52	1.42	1.40	1.39	5.97	20.67	14.91
	Skew	1.13	-0.27	0.01	0.81	0.76	0.05	1.36	1.36

Table shows weight, height, height for age Z scores(HAZ), weight for age Z score(WAZ),weight for height Z score (WHZ),height for age median, weight for age median and weight for height median of children in two age group as well as total children.

Figure 3 :Nutritional status of study children (n=5000)

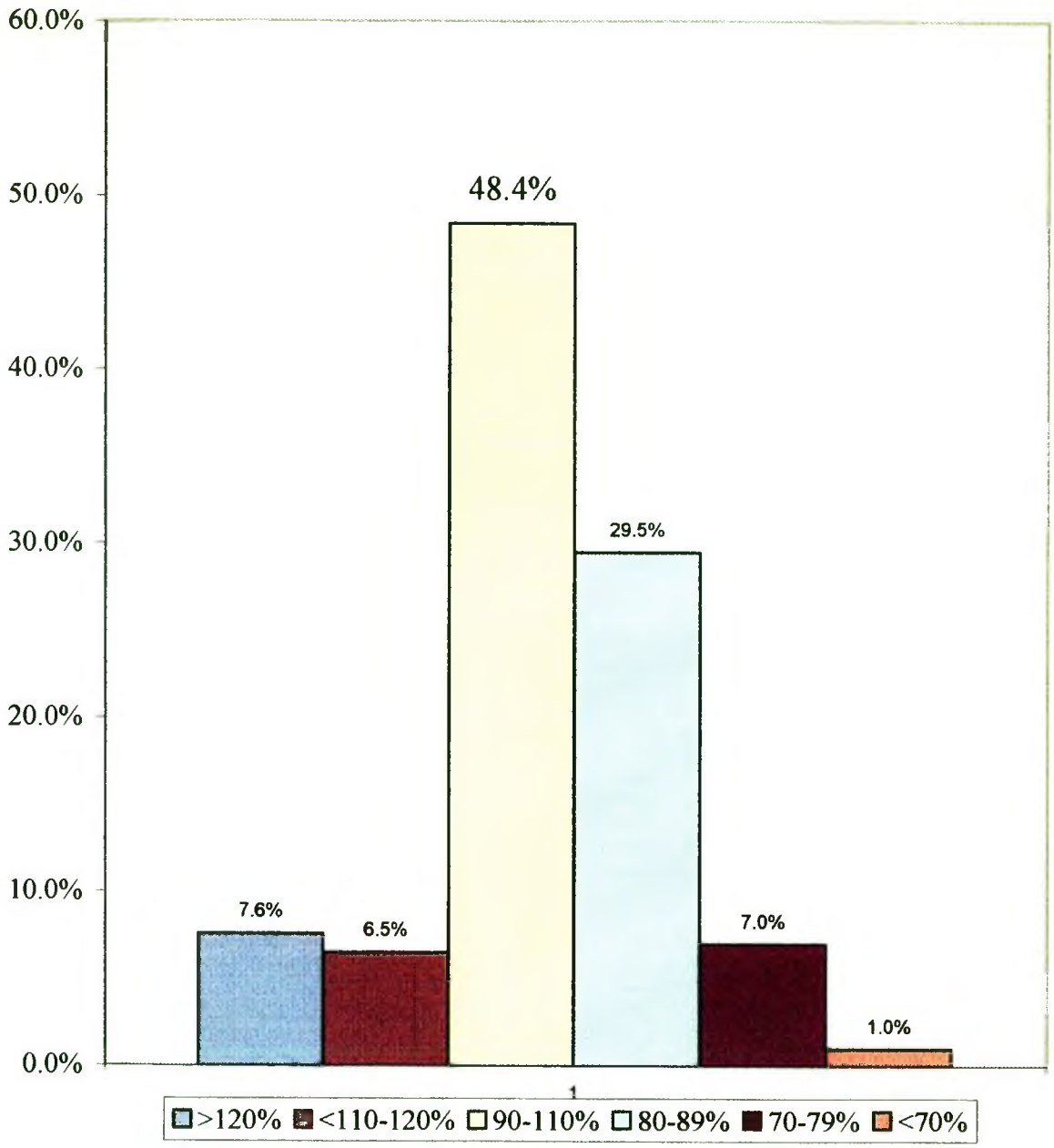


Figure shows, using weight for height indicator, 7.6 %children having wt/ht> 120% of NCHS (obese)and 6.5%, (overweight) having wt/ht 110-120%

Figure 4 :Distribution of obese and non obese children (n=5000)

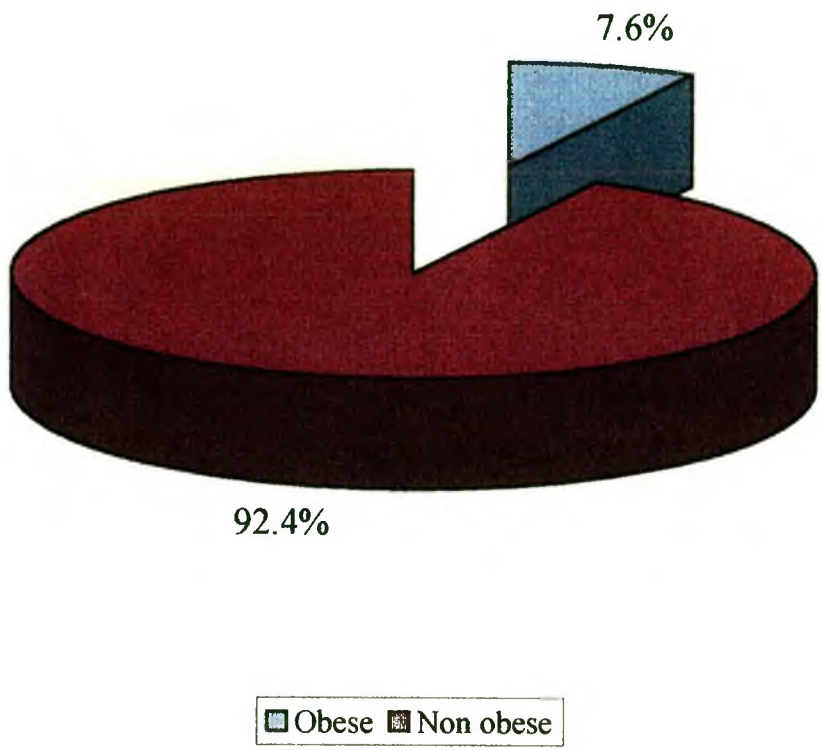


Figure shows that out of 5000 children, 380 (7.6%) children were obese (wt / ht >120%) and 4620 were non obese.

Figure 5 : Distribution of obese children by sex (n=380)

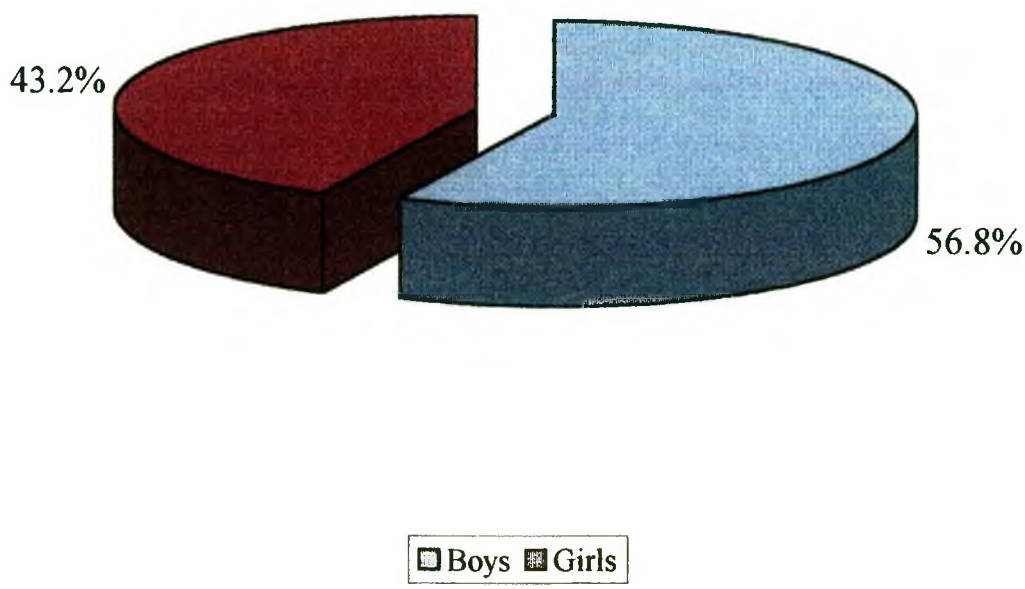


Figure shows that out of 380 obese children, 216 (56.8%) were boys and 164 (43.2%) were girls.

Figure 6: Distribution of age and obesity by sex of the children(n=5000)

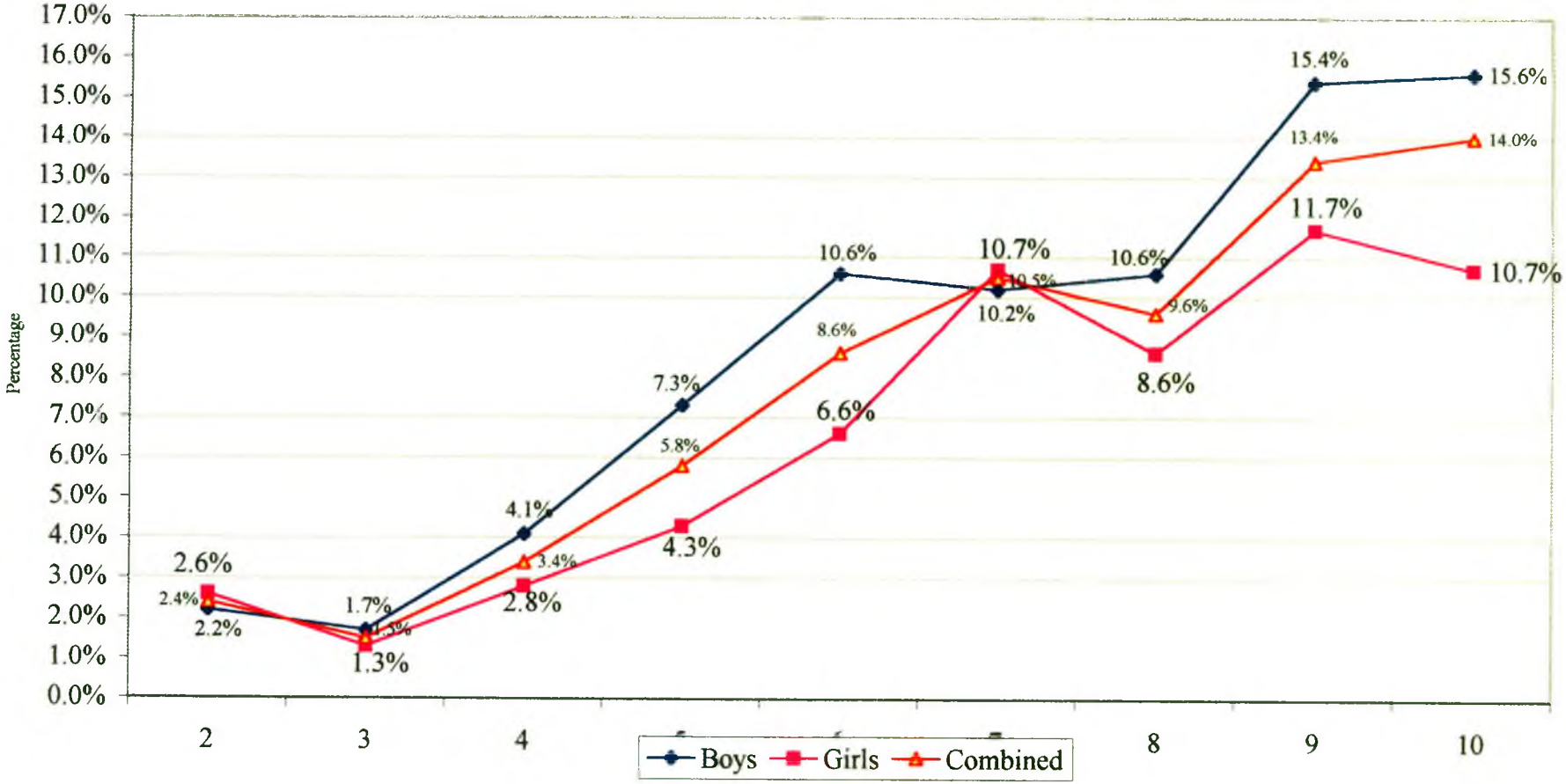
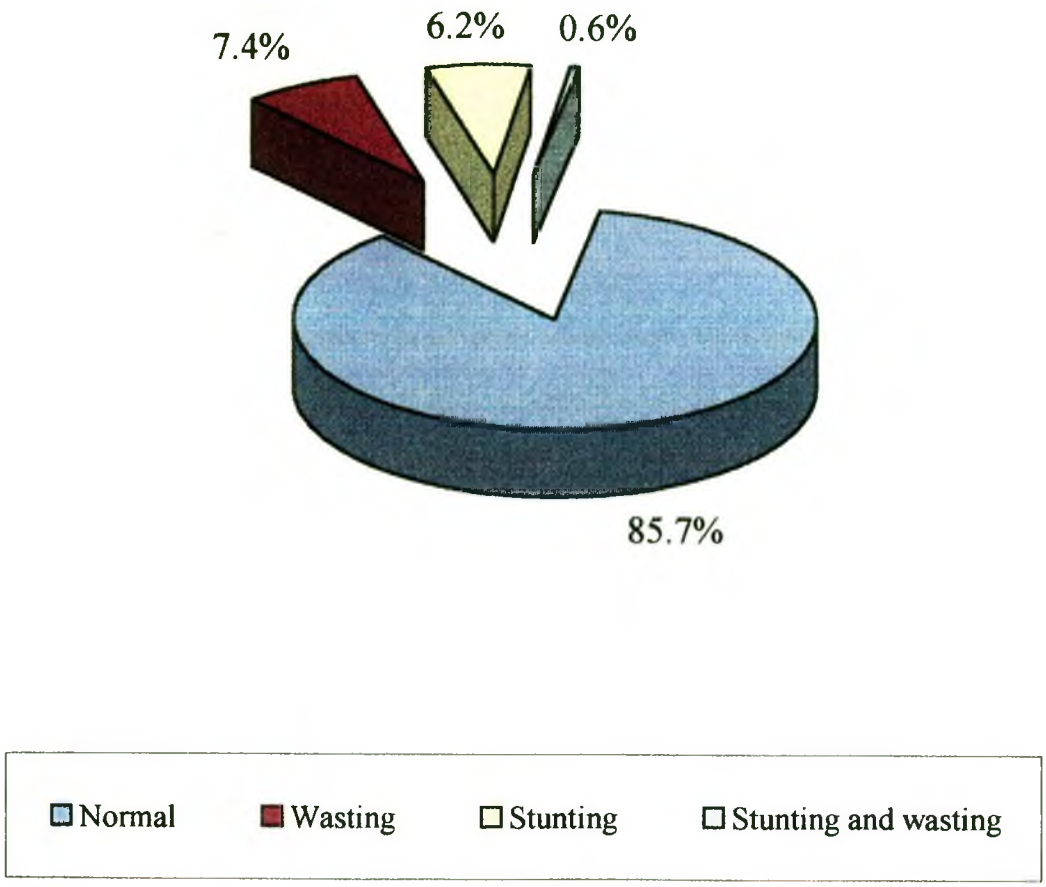


Figure shows, with increase of age, obesity among children also increased (r=0.76 both sex), which is more higher among boys/girls

Figure 7 : Prevalence of stunting and wasting (n=5000)



According to Waterlow classification, 85.7%were normal, 7.4% wasted, 6.2% stunted and 0.6% were both stunted and wasted

Table 4: Distribution of stunting and obesity

Sex	Stunting	Obese	Non obese	Total	Total
Boys	Stunted	5	149	154	2510
	Not Stunted	211	2145	2356	
Girls	Stunted	9	180	189	2490
	Not Stunted	155	2146	2301	
Total		380	4620	5000	

Table shows that among 2510 boys, only 5 stunted children were obese against 211 non stunted. Among 2490 girls, 9 stunted girls were obese against 155 non-stunted. Chi square test shows, stunting is negatively associated ($P<0.002$) with obesity among the study population.

Table 5: Childhood obesity by different Indicators

Indicator	Obesity		Percent of total population (5000)
	Cut off value	No. of obese children	
Wt/ht	>120%	380	7.6%
WHZ	≥2.0	280	5.6%

Table shows category of obese and non-obese children. Number of obese children are 380 and 280 using cut off level weight for height above 120% weight for height Z scores equal or above +2SD.

Table 6: Distribution of age of the cases and controls

Age (yrs)	Cases	Controls	Total
4 - 4+	6	6	12
5 - 5+	7	7	14
6 - 6+	13	13	26
7 - 7+	25	25	50
8 - 8+	44	44	88
9 - 9+	90	90	180
10	35	35	70
Total	**220**	**220**	**440**

Table shows that out of 440 study children (220 cases & 220 controls), single age distribution in 4-10 years children were 6, 7, 13, 25, 44, 90, and 35 in each group. The mean age in each group was 8.3± 1.4 years.

Table 7: Sex distribution of cases and controls. (N=440)

Sex	Cases	Controls	Total
Boys	149	149	498
Girls	71	71	142
Total	220	220	440

Out of total 440 study children, 220 were cases (standard weight for height >120%) and 220 were controls (standard weight for height ≤120%). Of them 149 were boys and 71 were girls in each group.

Table 8: Distribution of birth order and childhood obesity

Birth order	Cases (n=220)	Controls (n=220)	Total (n=440)
1st	113	106	219
2nd	68	58	126
3rd & above	39	56	95
Total	220	220	440

Out of 220 cases, 113 were 1st issue, 768 2nd issue, and 39 were 3rd & above issue, But among 220 controls, 1st, 2nd and 3rd & above birth order were 106, 58, 56 respectively. Chi-square test shows that, there was no significant ($\chi^2 = 4.060$ $P = 0.131$) association between birth order and obesity of the children.

Table 9: Distribution of family size and childhood obesity

Family size	Cases (n=220)	Controls (n=220)	Total (n=440)
Upto 4	74	59	133
5-6	86	106	192
7-8	35	31	66
Above 8	25	24	49
Total	220	220	440

Table shows 74 cases and 59 controls were from the family having members upto 4, 86 cases & 106 controls from the family of 5-6 members, 35 of cases and 31 controls were from the family of 7-8 members and, of family size of above 8, 25 were cases and 24 were controls. There was no significant association ($\chi^2 = 4.038$, $P = 0.257$) between family size and childhood obesity.

Table 10: Distribution of family structure and childhood obesity

Family Structure	Cases (n=220)	Controls (n=220)	Total (n=440)
Nuclear family	166	170	336
Joint family	54	50	104
Total	220	220	440

Considering family structure, among the cases (n=220) 166 from nuclear & 54 from joint family and, among the controls (n=220), 177 from nuclear & 50 from joint family. It is evident that obesity of children is not significantly ($\chi^2 = 0.201$ $P = 0.654$) associated with family structure.

Table 11: Distribution of fathers' education level and childhood obesity

Educational level	Childhood obesity		
	Case (n=220)	Control (n=220)	Total (n=440)
Below SSC	10	57	67
SSC & HSC	57	80	137
Graduation	63	46	109
Masters & above	90	37	127
Total	220	220	440

Out of 220 parents of cases, 10, 57, 63 & 90 had have education, below Secondary School Certificate (SSC) level, Secondary & Higher Secondary School Certificate (HSC) level, Graduation level and Masters & above respectively. Among the 220 parents of controls, 57 were below SSC, 80 were SSC & HSC holders, 46 were graduates and 27 were Masters & above. It is evident from the table, with increase of fathers' level of education, obesity of the children also increased. So, it may be interpreted, there is a significant association ($\chi^2 = 61.60$, $P < 0.001$) between fathers' education level and childhood obesity.

Table 12: Distribution of mothers' education level and childhood obesity

Educational Level	Childhood obesity		
	Cases (n=220)	Controls (n=220)	Total (n=440)
Below SSC	34	102	136
SSC & HSC	99	81	180
Graduation	54	25	79
Masters & above	33	12	45
Total	220	220	440

Table shows that with increase of level of mothers' education, obesity of children also increased. There is a significant (χ^2=56.246, P <0.001) association between mother's education level and childhood obesity.

Table 13: Distribution of fathers' occupation and childhood obesity

Occupation	Cases (n=220)	Controls (n=220)	Total (n=440)
Day labourers, Hawkers, Rickshaw Pullers, Sweepers	9	51	60
Service holders	73	61	134
Professionals	36	15	51
Businessmen	81	47	128
Small Traders	21	46	67
Total	220	220	440

It is evident from the above table, out of 51 children of professionals, 70.6% were obese; followed by 63.3% obese children among 128 children of businessman parents, out of 134 children of service holder parents 54.5% children were obese ($\chi^2 = 57.48$, P<0.001).

Table 14: Distribution of mothers' occupation and childhood obesity

Mothers' occupation	Cases (n=220)	Controls (n=220)	Total (n=440)
Housewives	185	197	382
Non Housewives	35	23	58
Total	220	220	440

Table shows among the mothers of cases (220), 185 were housewives & 35 were non-housewives (day labourer, sweepers, hawkers, service holders. professionals, businessman etc.) No statistical significant (χ^2 =2.4 P < 0. 12) association was to be found between mothers' occupation and childhood obesity.

Table 15: Distribution of fathers' working hours outside house-hold and childhood obesity

Working hours/day	Cases (n=220)	Controls(n=220)	Total (n=440)
<10 hours	106	98	204
>10 hours	114	122	236
Total	220	220	440

Table shows 114 fathers of cases worked more than 10 hours daily outside household against 122 fathers of controls. It is evident from the Chi-square test, no significant ($\chi^2 = 0.585$,$P = 0.444$) association between fathers working hours outside and obesity in children.

Table 16: Distribution of mothers' working hours outside house-hold and childhood obesity

Working hours/day	Cases (n=220)	Controls (n=220)	Total (n=440)
<3 hours	186	196	382
>3 hours	34	24	58
Total	220	220	440

Table shows 186 mothers of cases worked less than 3 hours in a day outside household against 196 mothers of controls. Out of mothers of 58 children, 34 mothers of cases worked more than 3 hours outside household daily against 24 mothers of controls. Chi-square test shows, no significant (χ^2 = 1.986, P = 0.159) association between mothers working hours outside household and obesity in children.

Table 17: Difference of parents' working hours outside house-holds among cases and controls

Parents' working Hrs	Subjects	Mean (hrs)	"t" value	*P* value
Fathers' working Hrs outside Household	Cases (n=220)	10.93 ± 2.34	1.665 (Special "t" test as F Significant)	0.097 *Not Significant*
	Cotrols (n=220)	11.11± 2.13		
Mothers' working Hrs outside Household	Cases (n=220)	1.18±2.91	0.83 (Simple "t" test as F Not Significant)	0.407 *Not Significant*
	Controls (n=220)	0.76±0.76		

Table shows, the mean working hours in a day outside households, were 10.93 hrs & 11.11 hrs of fathers' of cases and controls respectively. Independent sample "t" test shows, no significant difference of working hours between the two groups of fathers (*P*=0.47). Similarly, no significant difference of working hours outside household of mothers of cases & controls was to be found. So, it may be concluded here, parents' working hours outside household has no significant effect on obesity in children in the study population.

Table 18: Distribution of monthly family income of cases and controls

Monthly family income (Tk.)	Cases (n=220)	Controls (n=220)	Total (n=440)
Upto 10,000	41	91	132
10,001-20,000	96	94	190
20,001-30,000	52	23	75
30,001 & above	31	12	43
Total	220	220	440

Table shows, with increase of monthly family income level, the number of obese children also increased. It is seen from the chi-square for trend test, that there is an significant (χ^2 for trend = 36.48, $P<0.001$) association between family income level and obesity of children.

Table 19: Distribution of monthly family expenditure on food and childhood obesity

Monthly family expenditure on food (Tk.)	Cases(n=220)	Controls (n=220)	Total (n=440)
Upto 4,000	13	57	70
4,001-8,000	111	112	223
8,001-12,000	77	46	123
12,001 & above	19	5	24
Total	220	220	440

Table shows that, with increase of monthly family expenditure on food, the number of obese children also increased. There is a significant (χ^2 for trend =39.82 $P <$ 0.001) association between monthly family expenditure on food and obesity among children.

Table 20: Difference of monthly family income and monthly family expenditure on food between cases and controls.

Variables	Subjects	Mean ± SD	t value	*P* value
[a]Monthly family income(Tk)	Case (n=220)	23720±17576	t= 4.752 (Special ' t' test as F is significant	*P*<0.001
	Control (n=220)	16652±13338		
[c]Monthly family expenditure (Tk.)	Case (n=220)	8678±4316	t= 5.489 (Simple 't' test as F not significant)	*P*<0.001
	Control (n=220)	6616±3531		

a) Non parametric test Mann – Whitney test (mean rank :case 260.65 & control 180. 35) *P*<0.001

b) Non Parametric test: Mann-Whitney test (mean rank: case 260.33 & control 180. 67) *P* < 0.001

The table shows that mean difference of monthly family income, monthly per capita income and monthly family expenditure on food between cases and controls are statistically significant (p<0.001)

Table 21: Difference of mean energy intake and energy expenditure among cases and controls

Variables	Subjects	Mean±SD	T value	*P* value
Energy intake (kcal/day)	Cases (n=220)	2056±751	8.048 (Special "t" test as F Significant)	*P*<0.001
	Controls (n=220)	1558±529		
Energy expenditure (kcal/day)	Cases (n=220)	1868±313	14.878 (Special "t" test as F Significant))	*P*<0.001
	Controls (n=220)	1495±200		
Physical Activity Level(PAL)	Cases (n=220)	1.35±0.144	14.878 (Simple "t" test as F not significant))	*P*<0.001
	Controls (n=220)	1.40±0.09		

Table shows, the mean energy intake of cases & controls were 2056 ± 751 Kcal & 1558 ± 529 Kcal respectively. The mean energy intake was higher among cases, which is statistically significant ($p<0.001$). Energy expenditure was also significantly ($p<0.001$) higher among the cases. Physical activity level (PAL) is significantly higher among the non-obese children ($p<0.001$).

Table 22: Relation of Energy balance of children to childhood obesity

Energy Balance Kcal/day	Cases	Controls	Odds ratio
0	105	110	1
1-300	31	49	0.66
301-600	34	31	1.15
601-900	22	15	1.53
901-1200	10	8	1.31
1201-1500	5	3	1.75
≥1501	13	4	3.41

Table shows data on energy balance (Energy intake - energy expenditure) of obese (cases) and non-obese (controls) children. Taking as the reference group children who had not energy balance, the estimated relative risk of obesity increases with higher levels of energy balance upto a maximum of odds ratio 3.41. The Chi-square test for trend χ^2 46.3.8, $p<0.001$.

Table 23: Paired Sample 't' test Energy intake & Energy expenditure and Energy requirement & Energy intake among cases and controls

Paired Variables	Subjects	Paired difference: Mean	t value	*P* value
Energy intake - Energy Expenditure (kcal/day)	Cases (n=220)	188±835	3.347	*P*=0.001
	Controls (n=220)	63±544	1.706	*P*=0.089
Energy requirement - Energy intake (kcal/day)	Cases (n=220)	-44±777	-0.840	*P*=0.402
	Controls (n=220)	454±555	12.12	*P*<0.001

Table containing paired sample "t" tests show that, among the cases, there is significant difference between Energy intake & Energy expenditure (*P*=0.001). But the controls took energy less than their daily requirement, which is statistically significant (p<0.001).

Fig. 8. Relationship between energy intake and fat mass

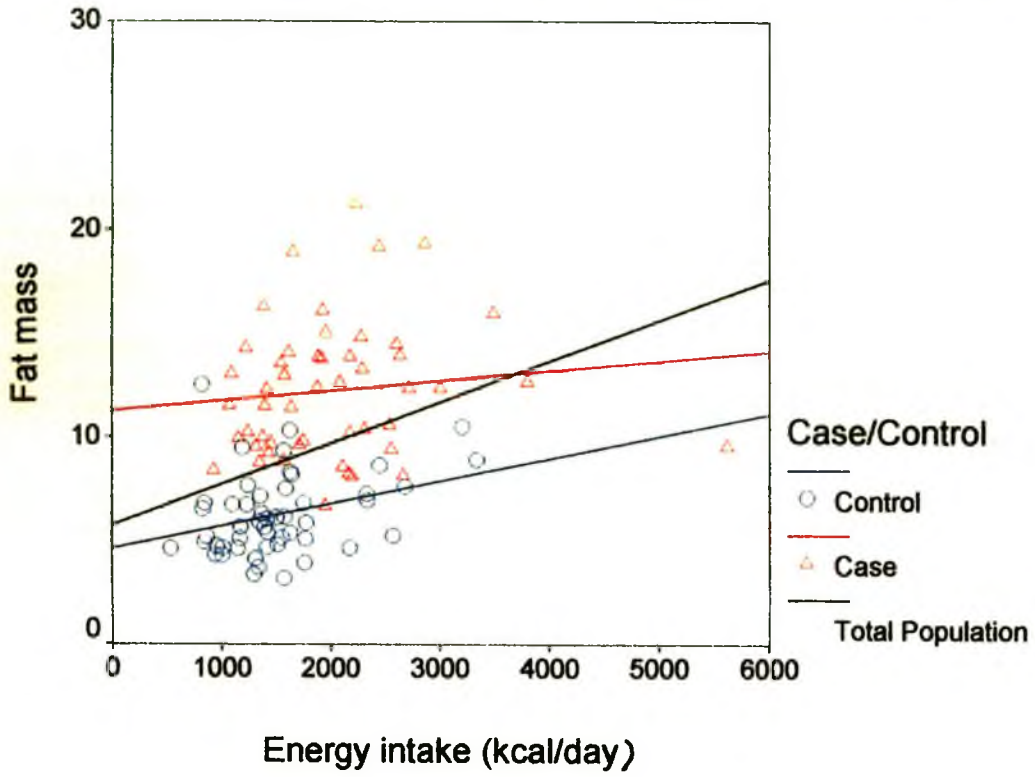


Figure shows the relationship between energy intake and mass of cases and controls (r=0.37, P=0.001)

Table 24: Distribution of hours of TV-Video viewing and childhood obesity

Weekly Hrs of TV-video viewing	Cases (n=220)	Controls (n=220)	Total (N=440)
Upto 7	20	33	53
8-14	76	93	169
15-21	85	76	161
22-above	39	18	57
Total	**220**	**220**	**440**

Table shows, the weekly total hours of television and video viewing of children. It is seen that with increase hours of TV video viewing, number of obese children also increased. Chi-square test shows there is a statistically significant (χ^2 = 13.13869 P=0.004) association between duration of TV video viewing and childhood obesity.

Table 25: Difference of mean TV-Video viewing among cases and controls

	Cases/Controls	Mean ± SD (Hrs)	t value	*P* value
Weekly TV-Video Viewing	Cases (n=220)	16.31± 6.37	3.929 (Simple "t" test as F Not Significant)	*P*<0.001
	Controls (n=220)	14.01± 5.89		

Table shows the mean hours of TV-Video viewing in a week is 16.31 ± 6.37 hrs and 14.01± 5.89 hrs among cases and controls children respectively. Cases enjoy TV-Video more hours than their controls, which is statistically significant (*P*=0.001).

Table 26: Distribution of fathers' obesity (using wt/ for ht %) and childhood obesity

Fathers' obesity	Child obesity		
	Cases (n=220)	**Controls (n=220)**	**Total (n=440)**
Obese (wt/ht >120%)	34	12	46
Non Obese (wt/ht <120%)	186	208	394
Total	220	220	440

Table shows out of 46 obese fathers, 34 having obese children, 12 non obese children. Furthermore, out of 394 non obese fathers, 186 have obese children. Chi square test shows, that there is an significant ($\chi^2 = 10.70625$, $P < 0.001$) association between fathers obesity and child's obesity.

Table 27: Distribution of mothers' obesity (using wt/ht %) and childhood obesity

Mothers' obesity	Child obesity		
	Cases (n=220)	Controls (n=220)	Total (n=440)
Obese (wt/ht >120%)	96	50	146
Non Obese (wt/ht <120%)	124	170	294
Total	220	220	440

Table shows out of 146 obese mothers having 96 obese children & 50 non-obese children. Furthermore, out of 294 non-obese mothers, 124 have obese children. It is evident from the Chi square test, there is a significant (χ^2 = 20.75762, $P < 0.001$) association between mothers obesity and obesity in children.

Table 28: Distribution of parents' obesity (using BMI) and childhood obesity

Parents' obesity	Childhood obesity			
	Case (n=220)	**Control (n=220)**	**Total (n=440)**	***P* value**
Mother Obese (BMI >30%)	25	5	30	$P<0.001$
Father Obese (BMI >30%)	8	1	9	$P<0.01$
Both obese (BMI >30%)	7	2	9	$P=0.08$
Both non-obese (BMI >30%)	180	212	392	
Total	220	220	440	

Table shows obese mothers have more obese children than the obese fathers. Both parents obese having non significant number of obese children.

Table 29: Lipid profile of children

Case/Controls	Level	TG	Total-cholesterol	HDL-cholesterol	LDL-cholesterol
Obese (n=50)	Normal	6	36	13	22
	Abnormal	44	14	37	8
	Total	**50**	**50**	**50**	**50**
Non Obese (n-50)	Abnormal	28	33	9	44
	Normal	22	17	41	6
	Total	**50**	**50**	**50**	**50**

Table 30: Difference of mean of TG, Total-cholesterol, HDL -cholesterol and LDL -cholesterol among the cases and controls

Variable	Subjects	Mean (mg/dl)	t value	*P* value
TG	Case (n=50)	182	5.937 (special "t" test as F Significant)	*P*<0.001
	Control (n=50)	99		
Total-cholesterol	Case (n=50)	117	1.058 (special "t" test as F Significant)	*P*<0.298
	Control (n=50)	104		
HDL-cholesterol	Case (n=50)	29	0.155 (special "t" test as F Not Significant)	*P* <0.574
	Control (n=50)	26		
LDL-cholesterol	Case (n=50)	61	0.266 (special "t" test as F Not Significant)	*P* <0.791
	Control (n=50)	57		

Table shows, there is statistically significant difference in the mean value of tryglycerides only. ($p<0.001$) between cases and controls.

Table 31: Correlation of Fathers' lipid profile with children lipid profile

Father's lipid profile	TG	T-Cholesterol	HDL	LDL
TG	r=0.09 p=0.375	r=0.831 p=0.01	r=0.783 p=0.01	r=0.785 p=0.01
Total-cholesterol	r=0.20 p=0.05	r=0.937 p=0.01	r=0.898 p=0.01	r=0.879 p=0.01
HDL-cholesterol	r=0.012 p=0.90	r=0.84 p=0.01	r=0.88 p=0.01	r=0.76 p=0.01
LDL-cholesterol	r=0.146 p=0.146	r=0.225 p=0.05	r=0.11 p=0.278	r=0.222 p=0.05

Table shows that fathers' TG, Total- cholesterol & HDL- cholesterol are highly positively correlated with child Total - cholesterol, HDL- cholesterol and LDL-cholesterol. But, TG of children poorly correlated with Fathers' lipid profile. There is a non-significant negative correlation between Fathers' LDL- cholesterol with child's HDL- cholesterol.

Table 32: Correlation of mothers' lipid profile with children's lipid profile

Mothers' lipid profile	TG	T-Cholesterol	HDL Cholesterol	LDL Cholesterol
TG	r=0.345 p=0.01	r=0.811 p=0.01	r=0.755 p=0.01	r=0.752 p=0.01
T- Cholesterol	r=0.227 p=0.05	r=0.951 p=0.01	r=0.913 p=0.01	r=0.899 p=0.01
HDL-Cholesterol	r=0.039 p=0.698	r=0.868 p=0.01	r=0.894 p=0.01	r=0.785 p=0.01
LDL-Cholesterol	r=0.20 p=0.845	r=0.385 p=0.01	r=0.233 p=0.05	r=0.407 p=0.01

Table shows, there are highly positive correlation between mothers' lipid profile with child's lipid profile except for Father's TG & LDL-cholesterol with child lipid profile.

Table 33: Distribution of parents' lipid disorder with child lipid profile

Lipid disorders	Cases (obese) n=50)								Cases (obese) n=50)							
	TG		Total -Chol		HDL-Chol		LDL Chol		TG T-Chol		Total -Chol		HDL Chol		LDL Chol	
Father's disorder	2	8	8	2	3	7	9	1	16	6	15	7	4	18	19	3
Mother's disorder	1	5	3	3	4	2	4	2	1	1	1	1	-	2	2	-
Both disorders	2	14	14	2	-	16	14	2	5	5	5	5	1	9	8	2
Both no disorder	1	17	11	7	6	12	15	3	6	10	12	4	4	12	15	1

Table shows no significant association between parents' lipid disorder with child's lipid profile.

Table 34: Distribution of body fat percentage obtained from Bioelectrical Impedance Analysis

Body fat %	Subjects		Total (n=100)
	Case (n=50)	Control (n=50)	
Upto 20%	0	2	2
20.01-25.00%	0	25	25
25.01-30.00	17	23	40
above 30.00	33	0	32
Total	50	50	100

Table shows that 33 (66%) children of total 50 obese children having body fat percentage above 30%. 50% of non obese children having body fat percentage between 21-25.00%.

Table 35: Paired sample "t" test for body fat percentage obtained from BIA method and body fat percentage calculated from 4 sites skinfold thickness using different equations (n=100).

Paired BIA & other methods	Mean	Paired difference mean	Paired sample correlation	"t" value	*P* value
BIA-Durnin & Rahaman	27.63 ± 3.8 23.71 ± 6.8	3.9± 3.9	0.886	10.116	*P*<0.001
BIA-Durnin & Rahaman (modified)	27.63 ± 3.8 17.35 ± 7.5	10.25±4.5	0.889	22.73	*P* <0.001
BIA-Brook	27.63 ± 3.8 19.56 ±9.6	8.08 ±6.6	0.858	12.17	*P* <0.001
BIA-Durenberg	27.63 ± 3.8 22.44 ± 6.6	5.20± 3.8	0.88	13.835	*P* <0.001

Table of Paired 't' tests show, even the mean difference is statistically significant, body fat parentage calculated using formulae of Durnin & Rahaman with that of value obtained from Bioelectrical Impedance Analysis -is the lowest among other equations used.

Table 36: Paired sample "t" test for body fat percentage obtained from BIA method and body fat percentage calculated from 4 sites skinfold thickness using different equations among the obese children (n=50).

Paired BIA & other methods used for body fat % estimation	Mean	Paired difference mean	Paired sample correlation	"t" value	*P* value
BIA-Durnin & Rahaman	29.13±3.2 30.74±1.9	1.56±2.3	0.683	4.759	*P*<0.001
BIA-Durnin & Rahaman (modified)	30.74±1.9 23.25±3.6	7.48±2.6	0.723	20.313	*p*<0.001
BIA- Brook	30.74±1.9 27.56±4.3	3.17±3.5	0.609	6.419	*P* <0.001
BIA- Durenberg	30.74±1.9 27.99±3.0	2.75±2.4	0.624	8.252	*p*<0.001

Table of Paired 't' tests show, even the mean difference is statistically significant, body fat parentage calculated for the obese children using formulae of Durnin & Rahaman with that of value obtained from Bioelectrical Impedance Analysis -is the lowest among other equations used.

Table 37: Regression analysis

Variables	Beta value (Intercept)	Wald χ^2	Significance	Exponent (Beta)
Age of the child	-1.175	36.572	0.000	0.309
Mother's age	.006	.014	.904	1.006
Birth order	-.120	.248	.618	.887
Father's education	.249	3.634	.057	1.282
Mother's education	-.045	.131	.718	.956
Father's occupation	-	1.474	.831	-
Day laborer & others	-.046	.003	.958	.955
Service holders	-.494	.912	.340	.610
Professionals	-.124	.032	.857	.883
Businessmen	-.580	.748	.387	.560
Small traders	-.509	.277	.599	.601
Mother's occupation	-	1.133	.951	-
Housewives	-1.326	.446	.504	.266
Non housewives	-.638	.603	.437	.528
Family structure	-.999	2.477	.116	.368
Family size	-.116	.941	.332	.890
Family income	.000	2.46	.620	1.000
Energy expenditure	.015	79.754	.000	1.015
Energy intake	.001	13.114	.000	1.001
TV viewing	.203	.763	.382	1.226
Father's obesity	-.065	.512	.474	.937
Mother's obesity	.117	3.156	.076	1.124
Constant	1.389	.114	.735	4.012

Table shows highest significant level of energy expenditure.

CHAPTER VIII

DISCUSSION

DISCUSSION

This study has been conducted among 2-10 years age group in Dhaka city. Children's age less than 2 years and above 10 years may be heavier and fatter for development and physiological reasons (13). So far, no epidemiological data on childhood obesity in Dhaka city is available. The prevalence of obesity reported in this sample of Dhaka city is 7.6%, using weight for height more than 120% of NCHS/WHO reference. There is a significant difference (P<0.007) between the prevalence of obesity between boys (56.8%) and girls (43.2%). This difference is similar to the data reported in Italian and Malaysian population (33,294). In this study, highest prevalence (14.0%) of obesity was found in 9-10 years old children, but no statistically significant difference was to be found between boys and girls (P=0.067) in this subgroup, which is inconsistent with the finding of Kotani et al among the Japanese children (109). It has been reported that obesity among 8-10 year old children of Philippines in 1997 was 12.0% (15).

Data of this study show, prevalence of obesity among the study population increased with age (r=0.76, chi-square for age trend 117.61 P<0.001. The trend is higher among the boys ((χ^2 for trend 69.03) than girls (χ^2 for trend 44.25).

The age trend of this study is consistent with the other studies (15,22,43,56,57). The mean age of the obese children was 8.3 years. Wilkinson et al have been investigated a total 161 children (94 girls and 67 boys) of New Castle Upon Tyne and found that 29% of the boys and 54% of the girls had weights for height above the 90th percentile by 5 years of age, the figure for the controls being 4%. The remaining 71% of boys and 46% girls developed their obesity after the age of 5 years. The result of the present study is consistent with the study of Wilkinson et al (53).

Among the 5000 study children of 2-10 years age group, the mean weight and height of the 6-10 years subgroup are 24.63 ± 7.18 kg and 126.03 ± 9.74 cm respectively (Table 3) . Bangladesh National Nutrition Survey 1995-96 revealed that the mean

weight and height of 6-9 years group in national representative sample of urban children were 18.78 ± 3.94 kg and 115 ± 9.04 cm respectively (295).

Childhood nutritional stunting, an indicator of chronic undernutrition (296-298), has been suggested as one factor contributing to high rates of obesity in developing countries because of the observed positive association between stunting and adolescent and adult obesity (299-304). In this study, the association between obesity and stunting in children is not statistically significantly. Amongst the 5000 study children, 2510 were boys. Of the total boys, 154 were stunted and only 5 (3.25%) boys of them were obese, against 9 (4.76%) girls were obese amongst 189 stunted girls. Sawaya et al found that stunted girls appeared to be at even greater risk of obesity than boys, with a 35% prevalence of obesity compared with 11% in stunted adolescent boys. In the present study, though the association between stunting and obesity is not significant but it was to be found that girls were more obese than their stunted boys counterpart, which is similar with findings of Sawaya et al (299). Hoffman reported that stunting increases the risk of obesity in developing countries, particularly in girls and women (305).

Both birth order and family size have been related to childhood obesity. But in this study no significant association was to be found between childhood obesity and birth order or family size. Finding of this study is inconsistent with other studies in which the prevalence of obesity is high among single children and declines with increasing family size. (53,139) . Birth order has no consistent effect on prevalence of obesity (4), although some data suggest greater prevalence among younger children of large families (61,140)

Stettler et al found that first-born status independently associated with the development of increased adiposity (141). But contradictory results were found in earlier studies conducted in European and African populations (4,141,142)

It was observed that the risk of obesity in 10 years old girls in USA decreased as the number of siblings increased (141). Other studies show that (306,307), some one third

of obese children were only children an another one third were last born. Wilkinson found (53) that being an only child was one of the commons 'at risk' factors for obesity.

The characteristic of family structure or family life are closely linked to the development and maintenance of obesity in children (45) . In this study, obesity in children is not significantly associated with family structure, which is inconsistent with the findings of Wilkinson et al. (53). Intact parental relationships are also important for the growth and development of the children.

Despite the frequent use of education as a determinant of socio- economic class, its effect on rates of obesity had rarely been examined as independent variable. No available studies establish the relation of parental education to prevalence of obesity among children (61).

In this study, there is a significant association between obesity in children and parents education level. With increase of parents' education level, obesity also increased among children. Occupation and education levels are related to income level. Most of the parents with higher education are engaged in high earning occupation, which may influence on family expenditure on food.

In addition to education, income, occupation, place of resident etc. are the determinants of socio-economic status. In developed countries, socio-ecomic status is inversely correlated with the obesity, But in developing countries, it is positively correlated.(9,153).

In this study, childhood obesity is significantly associated with the higher level of family income (χ^2 for trend 36.48 $P< 0.001$). Results also show, the monthly family expenditure on food (χ^2 for trend 39.82 $P< 0.001$) is directly associated with the obesity among the children.

The finding of this study regarding income level is consistent with the other studies (9,62), but inconsistent with the findings revealed by other authors (154,163). It is evident that with increase of income level, the expenditure on food also increased, which eventually causes higher energy intake. It may be mentioned here, parents' occupation also having influence on childhood obesity in this study. Children of parents having occupation of higher income are more obese than their low earning occupation counterpart.

The effect of income level on obesity had received much attention by the researchers. Nevertheless, the two largest North American surveys that examined economic status in relation to childhood obesity presented conflicting data. In USA, the ten state Nutrition Survey found that white children of higher socio-economic classes were fatter than those with lower incomes, although an inverse relationship was present among girls in late adolescence (308). In contrast, a study of 3,300 white school children found more obesity among lower class children than at upper socio-economic levels. Despite economic data from several other studies (55,111,309), no consistent class trends are apparent.

Socio-economic status of the families of obese children was higher in contrast to their non-obese counterpart, which is similar to other developing countries where childhood obesity is emerging or increasing due to socio-ecnomic transition.

Body weight is dependent on the balance between energy intake, in the form of food and drink, and energy expenditure. Daily energy expenditure consists of resting energy expenditure, the energy required to metabolize food (thermic effect of food), and energy expended as a result of activity. When energy intake and expenditure are in balance, weight remains stable. A net excess in energy, whether through greater intake of lesser expenditure leads to weight gain. In children, some of this extra energy may be used for linear skeletal growth; but in both children and adults net excess energy intake leads to increase in both lean body mass and adipose tissue.

In this study, energy intake is significantly ($P<0.001$) higher among the obese children (2056±751 Kcal/day.) than the non obese counterpart (1558±529 kcal/day). But there is no statistically significant difference between boys and girls regarding calorie intake for both cases and controls.

In this study, energy expenditure among the obese children is significantly higher than their non-obese counterpart (1868 ± 313 kcal Vs 1495± 200). Amongst the cases, energy expenditure is significantly higher in the boys than the girls (P=0.015). Comparing with energy expenditure, energy balance is higher among the obese children. Here, physical activity level has been calculated. Physical activity level is significantly higher among the non obese children against their obese counterpart (1.40 ± 0.09 Vs 1.35± 0.14)

Energy balance (in context of energy intake and expenditure) is more among obese children. Paired sample t test shows (Table-23), the energy balance is not statistically significant among the controls (P<0.089). Children, who have not energy balance, taking them as the reference group, the estimated relative risk of obesity increases with higher levels of energy balance upto a maximum of odds ratio 3.41. The Chi-square test for trend χ^2 46.3.8, p<0.001. (Table-22). Furthermore, energy intake among the control was significantly less than their requirement (P< 0.001).

Only a few large epidemiological studies have examined the relationship between energy intake and obesity in children. Rolland-Cachera and Bellisle (263) reported no relationship between body mass index (BMI) and skinfold thickness with energy intake in 7-to 12-year-old children. Sunnegardh et al (64) also showed no relationship between skinfold thickness and energy intake in 8-year-old boys and girls and 13-year-old boys, although a significant negative correlation was reported for the 13-year-old girls. Other studies that compared energy intake of lean and obese children have generally not shown that obese children have higher energy intakes than do lean children (65-69). Negative results from the latter may be due to the limitation of self-reports of energy intake as a measure of energy requirements.

Goran et al (310) found no relation between energy expenditure and obesity in a 4 year follow-up of 75 white preadolescent children. The main predictors of change in fat mass relative to fat free mass in their study were gender, initial fatness, and parental fatness. Similarly Maffeis et al. (311) reported that neither physical activity nor energy and nutrient intakes significantly affect the change in BMI over 4 years in 112 prepubertal children with a mean age of 8.6 years at baseline after parental obesity was taken into account. BMI at baseline was the only variable retained in multivariate analyses.

In contrast, two longitudinal studies of younger preschool children have provided evidence for an association. Among 97 White children aged 3-5 years at baseline in the Framingham Children's Study those with low levels of physical activity gained substantially more fat that did more active children as they were folded into first grade (312). Similarly, Klesges et al (41) reported that dietary intake and physical activity accounted for more of the variance in changes in BMI than did non modifiable variables such as parental obesity in 146 White, middle class, preschool children over a 3 years follow-up.

Results have been inconsistent and have not been generally demonstrated a significant inverse relationship between physical activity and body weight or body fat (64,195). Tanasescu et al reported that obese children had similar total energy intakes but lower energy intakes per kilogram of body weight compared with non-obese children (137).

Inconsistent results have also been reported in studies comparing lean and obese children. Some studies show that lean children are more physically active (71,192,313), whereas others do not show a significant difference (111,179).Some reports indicated that television watching is linked with obesity in children. Tanasescu et al reported that obese children performed significantly lower levels of physical activity in the cold season compared with controls and marginally lower levels in the warm season (137).

It has been reported (314-316)) that the lack of consistent or inconclusive results in previous studies could be due to the large measurement errors associated with the assessment of energy intake in general and under reporting among obese children and adults. Physical activity, particularly in children, or inadequate sample sizes could have been inadequate.

Few studies have examined the relationship between obesity and percent of energy from fat. In this study, significantly higher amount of calorie had come from fat ($P<0.05$).Eck et all (176) reported that children at high risk of obesity, based on parental obesity, had a similar total energy intake but higher percent of energy intake from total fat than did children at low risk of obesity. However, the two groups of children were at the same relative weight, and the relationship between relative weight and percent of energy intake from fat was not reported. Studies have shown that the diets of obese children have a higher proportion of fat than those of normal weight children (317). Similar findings also reported by Gazzaniga and Burns (318) that a significant relationship between percent body fat and dietary fat as percent of energy intake in 9-to 11-y-old children. Thus, evidence is beginning to support the idea that dietary fat is associated with body fatness in children.

Television viewing has become not only one of the most important daily activities that competes with being active but also one that influences child development. (179). Dietz and Gortmaker reported that children in the United States spend, on average, as much as time watching television each year as they do attending school (198).

Researchers have identified television viewing as one of the prominent potential modifying factors that can lead to obesity in childhood. Each hourly increment of television viewing by adolescents has been associate with a 2% increase in the prevalence of obesity (198).

In this study, the television and video viewing is significantly associated with the obesity among the children. Obese children viewed television significantly ($P<0.001$)

more hours than controls (16.31 Vs 14.01 hrs). The mean duration of television exposure was 16.31 hours per week among the obese children, which was almost equal to children of India as reported by Gupta et al. (196). Similar agreement with a number of studies (198,207).

Dietz and Gortmaker reported that the association between television viewing and obesity was statistically significant and persisted when other potential variables were controlled (198).

In contrast, cross-sectional epidemiological studies have consistently found relatively weak positive association between television viewing and child and adolescent adiposity (198,208,319). The consistently weak association was to be found in an epidemiological study might be due to the measurement error in self-report of television viewing (320).

Tucker and Friedman (201) reported that television viewing might have a powerful influence on adult obesity as well. These researchers found that men who viewed television more than 3 hours per day were twice as like to be obese as those who viewed less than 1 hour per day. Although, it was unclear whether the men were obese because they watched television or whether they watched more because they were obese. Dietz and Gortmaker's study (198) suggests that the former may be the cause.

A little is known regarding the way in which television viewing affects energy balance. Television viewing may be related to more dietary intake including increased opportunity for snacking, less physical activity, metabolic rate, or some combination of these (179).

Tanasescu et al (137) have identified from their case-control study among Puerto Rican prepubertal children that hours of daily TV viewing was associated with obesity, which is consisted with the present study. They reported that TV viewing levels correlated positively with intake of sweets and snacks and negatively with

maternal rating of child's level of physical activity in whole sample. Among girls, there was no correlation between TV viewing and sweets and snacks intake, but significant negative correlations were found between TV viewing and activity expressed both as maternal rating and as physical activity score. Among boys, the correlation of TV viewing with sweets and snacks consumption was significant, whereas there was no correlation with activity levels. In this study, only the hours of television viewing has been investigated. No intake of foods likes snacks and sweets during the TV viewing have been recorded.

Obese parents are more likely to have obese children and are independent of the sex of the parent or child. This pattern exists even when children are reared apart from their biological parents. Parents provide both the genes and environmental context for their children's growth & development and familial patterns of adiposity are the results of gene-environment interaction (137,321).

In this study, parental obesity is significantly associated with obesity in children. Maternal obesity is more significantly ($P<0.001$) associated than the fathers' obesity ($P<0.01$). Findings of this study are consisted with the findings of other studies (322,323). It is widely accepted that parental behaviors and practices shape many aspects of children's development (61) The parents' food preferences, the quantities and variety of foods in the home, the parents' eating behavior, and the parents' physical activities patterns work in concert to establish an emotional environment (324,325) and weight outcomes. In this study, most of the mothers were housewives and primary caretakers of the children, typically provide with structure for meals, by offering foods, and others and by using child feeding practices that provide information to the child about how much and what to eat and mothers' behaviors, life styles etc. influence children. Due to this maternal influence, mothers' obesity is more significantly associated with children obesity.

But children of both obese parents are not statistically significant ($P=0.08$) which is inconsistent with the study of Lake et al (211). Lake et al reported that children with two obese parents are more likely to be obese compared with children with only one

obese parent, who in turn are more likely to be obese than children without obese parent. The tracking of obesity into adulthood is stronger in children when both parents are obese.

In this study, there is statistically significant ($p<0.001$) association between obesity and serum triglycerides among children. Triglyceride level was significantly ($p<0.001$) higher among the obese children (182 ± 53 mg/dl). This finding is consistent with other studies (326-329). But no significant difference was to be found regarding serum total cholesterol, HDL - cholesterol and LDL - cholesterol.

It has been reported that not obese individuals have raised serum lipids. Hitchcock and Gracey revealed that obesity (>120% of ideal body weight) was no more a feature of mothers or children with high cholesterol levels (330).

Serum total cholesterol, HDL cholesterol and LDL cholesterol of children are positively highly correlated with fathers' triglycerides, total cholesterol and HDL cholesterol. There is highest correlation of child total cholesterol with fathers' total cholesterol which is statistically significant ($r = 0.937$, $P= 0.01$). Similar finding also was to be found between mothers and children. But it can be mentioned here, correlation of parents' serum total cholesterol and serum total cholesterol of children, it is more correlated with mothers than fathers ($r = 0.951$ vs. 0.931). Finding of this study is similar with the finding of Godfrey et al (330). Regarding the more correlation with mothers, it may be concluded that role of mothers in controlling the family diet, particularly that younger children might explain the close relationship.

Laskarzewski et al studied among children of 4-20 year age group from both the hyper lipidemic and random recall groups, BMI was inversely related to HDL - cholesterol and positively related to LDL - cholesterol and triglyceride. Frerichs et al echoed with the findings of Laskarzewski for Bogalusa children of 5-14 year age group (329,326).

There is no statistically significant association of parents' lipid disorders with abnormal profile of lipid of children. Finding of this study is inconsistent with Heinle et al and Tamir et al (331,332).

Body composition was obtained from bioelectrical impedance (BIA) analysis and estimated from 4 sites skin fold thicknesses using different equations. Among the obese (wt/ht >120%) children, body fat percentage obtained from BIA, two third of children had fat percentage above 30 fat and another 44% had between 25-30%. Of the controls, half of the children had body fat percent between 20-25%. Among the equations used, the equation of Durnin and Rahaman (257) the estimated mean fat percentage of the study children is significantly closest to fat percentage obtained from BIA. Similar result also found in case obese children. Use of the equation of Durnin and Rahaman for estimation of body fat for obese children may be validated through further large sample studies.

Logistic regression was used to examine the factors associated with the obesity in the study population. Of the factors significantly associated with the obesity in children as indicated earlier, the regression analysis shows, the significant association of age of the children, energy intake and energy expenditure. Energy expenditure ranked the highest significant level (Table-37).

The findings of this study support the hypothesis that socio-economic status, dietary habit and life style factors are associated with the development of obesity in children.

CHAPTER IX

CONCLUSION

CONCLUSION

The findings of this study show that the prevalence of childhood obesity in Dhaka city is 7.6% among the 2-10 years age group. The prevalence rate indicates that the childhood obesity is an emerging health problem in Dhaka city. In a developing country like Bangladesh, the situation is an alarming one in Dhaka city where it is coexisted with the undernutrition.

Results show parental education level, occupation, family income and family expenditure on food are positively correlated with the obesity in the children. These positively associated factors are widely accepted components and collectively express as the composite index of socio-economic status.

Furthermore, the energy intake is higher and physical activity level is lower among the obese children. The energy balance in terms of energy intake & expenditure or energy intake & energy requirement, there is a significantly higher energy balance existed among the obese children. Dose response of energy balance on obesity shows the estimated relative risk of obesity increase with higher level of energy balance among the cases.

Hyperlipidemia usually indicates that there is intake of high level of fatty diet, which usually occurs among the affluence. Findings of the study show that most of the parameters of lipid profile of parents significantly correlated with that of the children.

It may be concluded here, on the basis of the findings of the study, that obesity in children are prevailing in the families those are passing a rapid socio-economic transition which led to an environment that promotes a sedentary life styles and consumption of high fat, energy dense diets. So, optimum energy intake and expenditure should be considered for a healthy and economically active life for a better tomorrow.

CHAPTER X

RECOMMENDATION

RECOMMENDATION

1. Childhood obesity is an emerging health problem in Dhaka city.

2. Childhood obesity issue should be incorporated in the public health policy of the country with special emphasize in Dhaka city.

3. Childhood obesity is co-existed with undernutrition in Dhaka city presenting a double burden in a developing country like Bangladesh. So, it is urgently needed to take necessary intervention strategies regarding unhealthy trend in diet and life styles.

4. School curriculum should be incorporated with day to day's life oriented health and nutrition topics.

5. Multi scetoral approach is needed for its prevention and management.

6. Regular serum lipid screening is needed for the vulnerable groups.

7. For prevention and management, further studies both cross-sectional and longitudinal are needed to explore, in which way the factors are responsible for development and maintenance of obesity in children.

CHAPTER XI

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CHAPTER XII

ANNEXURE

QUESTIONNAIRE

A study on epidemiology of childhood obesity in Dhaka city

ID No. ☐☐☐ [1] Case [2] Control Residence: Thana code ☐☐

	Child (2-10 yrs.)	**Father**	**Mother**
1. Name	.....	.....	.....
2. Age	☐☐☐☐ Yr Mo	☐☐ Yr	☐☐ Yr
3. Sex of child	[1] Male [2] Female		
4. Birth order	☐☐		
5. Education	☐☐	☐☐	☐☐
6. Occupation	☐☐	☐☐	☐☐
7. Working **Hrs.** of parents outside house-hold		☐☐	☐☐
8. Weight (Kg.)	☐☐☐	☐☐☐	☐☐☐
9. Height (Cm)	☐☐☐☐	☐☐☐☐	☐☐☐☐

10. Skinfold Thicknesses of Child (mm)

Biceps	**Triceps**	**Subscapular**	**Suprailiac**	**Total**

11. Clinical state

a. History of illness: [1] **No** [2] **Yes** (Specify) .....

b. H/o drug taking: [1] **No** [2] **Yes** (Specify) .....

c. Physical examination:

	Pulse	BP		Anaemia	Oedema	Heart	Lung	Kidney	Thyroid	Def.sign(s)	Others	Others
		S	D									
Child												
Father												
Mother												

12. Family structure: [1] Nuclear [2] Joint 13. Family size: ☐☐ 14. Religion ☐

15. Monthly family income (Tk.) : ☐☐☐☐☐☐

16. Monthly family expenditure (Tk.) : ☐☐☐☐☐☐

a. On food (Tk.)

Items	Rice	Atu	Dal	Vegetables	Meat	Fish	Egg	Oils/Ghee/Butter	Spices	Milk	Snacks	Fruits	Others	Total
Price (Tk)														

b. Others (Tk.) ☐☐☐☐☐☐

ID No. [ ][ ][ ] [1] Case [2] Control

Age: [ ][ ] Yrs. [ ][ ] Months Sex: [1] Male [2] Female

17. 24-Hr. Dietary Recall

Food Items		Serving Size	**Breakfast**	Pre-lunch	**Lunch**	Evening	**Dinner**	Bedtime	Code No.	Total	Comments
ভাত Rice									002		
রুটি Bread									006		
পরোটা/লুচি Porota / Lucy									020		
খিচুড়ি Mixed rice											
মুড়ি " /খই " / চিড়া " Fried Rice											
বিস্কুট/টোষ্ট/ওয়েফার Biscuit / Toast Wafers											
পায়েস/পুডিং Rice puding / Puding											
আলু Potato									031		
ডাল Dal	পাতলা								059		
	ঘন										
শাক Shoak											
সব্জী Vegetables											
মাছ Fish	বড় Large								319		
	ছোট Small								336		
মাংস Meat	গরু Beaf								251		
	খাসী Mutton								255		
	মুরগী Chicken								254		

Food Items		Serving Size	**Breakfast**	Pre-lunch	**Lunch**	Evening	**Dinner**	Bedtime	Code No.	Total	Comments
ডিম	মুরগী Hen								347		
	হাঁস Duck								346		
দুধ	গরু Cow's								351		
	পাউডার (দুধ ক্রিম) Powder										
	হাফক্রিম Powder milk (Half cream)								366		
	মায়ের দুধ Breast milk								354		
	ছানা/মাখন Butter/whey								358		
	মিষ্টি Sweet meat								047		
	আইসক্রিম Icecream										
	দই Curd										
ফল Fruits	আপেল Apple								199		
	আঙ্গুর Grape								200		
	কলা Banana								194		
	কমলা Orange								204		
	আম Mango										
	জাম Rose-apple										
	কাঁঠাল Jackfruit										
	লিচু Lichi										
	পেঁপে Papaya										
	টমেটো (সালা) Tomato								240		
	শশা/কাঁকরা Cucumber								213		
চিনি "/ গুড় " Sugar / Molasses											
চকলেট/ লজেন্স Chocolet / Lozens									041		
তৈল Oil	সোয়াবিন Soyabean								375		
	বাটার/ঘি Butter/Ghee								372		
বুট"/ বাদাম"/চানাচুর	Nut / chanachur										
চিপস্ Chips											
জুস Juice									046		
ডাবের পানি Coconut (Green)											

18. **24-hr. Activity recalls**

Code	Activity	Duration		Code	Activity	Duration	
		Hrs.	Min.			Hrs.	Min.
01	Sleep at night			18	Sleep (day time)		
02	Washing, Dressing			19	Eating snack		
03	Eating Breakfast				Playing		
04	Walking			20	Group discussion, sitting &talking		
05	PT			21	Watching TV, Video		
06	Sitting class room			22	Computer, V.games, Calculator		
07	Walking to and fro			23	Reading & Writing		
08	Eating snacks			24	Others (Specify)		
09	Playing Cricket			25			
10	Playing Football			26			
11	Playing Badminton			27			
12	Playing Carom			28			
	Playing						
13	Walking			29			
14	Climbing stairs			30			
15	Undressing, showering						
16	Walking home						
17	Lying						

19. **Weekly TV-Video- viewing recall**

Day Time	**Day-1**	**Day-2**	**Day-3**	**Day-4**	**Day-5**	**Day-6**	**Day-7**	**Total Hrs.**
06 am-12am								
12 am-6pm								
06pm-12pm								
12pm-06am								
Total Hrs.								

A study on epidemiology of childhood obesity in Dhaka city

ID No. [ ][ ][ ] [1] Case [2] Control

Child (2-10 yrs.)

2. Age [ ][ ][ ][ ]

Yr Mo

3. Sex of child [1] Male [2] Female

8. Weight (Kg.) [ ][ ][ ]

9. Height (Cm) [ ][ ][ ][ ]

10. Dehydration status No Yes (Specify) .....

2) Resistance [ ][ ][ ] Ohm.

3) Reactance [ ][ ][ ] Ohm.

20. **Lipid Profile**

	TG (mg/dl)	Cholesterol(mg/dl)	HDL(mg/dl)	LDL(mg/dl)
Child (2-10 yrs)				
Father				
Mother				