Introduction
The new millennium has sharpened the focus of attention towards the beauty, balance, harmony and health. In a rare combination of factors mind and body have been identified to play a duet of symphony. When media is highlighting the beauty it is also fixing concern on growing Obesity.

The prevalence of obesity is increasing in adults and children alike. Many developing countries throughout the world report a steep rise in incidence of obesity. A report in Indian Express, 31 March, 2002 expressed concern at the rise of obesity in India especially among the affluent Indians. In India, in the last twenty years, the incidence of overweight and obesity in children has doubled. The number of overweight adolescents has almost tripled in this time. Overweight children have a higher likelihood of becoming overweight adults and developing medical complications.

In the US itself, about one third of American adults are obese. About 25% of US women are overweight and an additional 25% are obese. Same trend is reflected in Europe. But simply this may be the result of increasing sedentary life combined with high calorie foods in affluent societies. (Hales et al., 1999). This change in lifestyle has produced an increasing prevalence of obesity in all sections of society.

Sharma (2002), reported that one of the commonest problem for which parents are seeking help is regarding their obese children. In the US, obesity in children has alarmingly increased by 50% since 1976. According to research studies, about 80% of fat adolescents become obese adults. In Indian population, obesity in children as young as two years onwards has been reported. Children in preadolescent age group of 8 to 12 years, and adolescent age group 13 to 18 years are also facing this problem.
Obesity is a condition that clinicians have long preferred to ignore, too often perceiving it not as a medical but as a lifestyle choice. It is becoming clear, however, that obesity is the result of a complex interaction of genetic, environmental and psychological factors, and that it can and should be managed (Campbell, 2000).

Being overweight or obese is not only associated with a reduced life expectancy but also with an extensive range of medical, and psychological conditions which impinge upon the patient's quality of life. These include several illnesses in which governments usually set stringent targets for health improvement: cardiovascular diseases such as hypertension, stroke and coronary heart disease; metabolic disorders such as insulin resistance and type 2 diabetes; and a range of cancers (Campbell, 2000).

As the onset of obesity is insidious and successful treatment is known to be difficult it is generally agreed that the identification of potentially obese or overweight individuals before they become overweight is critically important. It has been agreed that childhood and adolescent obesity are important in their own right because of association with adult obesity and related disorders (Williams, 2001).

DEFINITIONS OF OBESITY

Obesity is a from of disordered nutrition. The word is derived from the Latin Obesus, meaning "eaten away," which shows that overeating may cause disease and even death. (International Encyclopedia of Psychology, 1996).

Defining the levels at which body fat and body weight cross threshold and become obesity and overweight is not easy and has generated considerable debate in the field. The precise point at which scientists and health officials believe increasing weight threatens health ranges from 5% to 30% above ideal weight, a
considerable spread. Furthermore, different tables of “ideal” weights have been embraced by different figures in the field. Obesity is to be distinguished from overweight, which refers to weight in excess of some standard (Brownell, 1995).

Obesity refers to an excessive amount of body fat. While obesity is a condition of most concern, overweight is usually measured in clinical settings. Overweight is simply weight that is above some standard of ideal weight (Kaplan et al., 1993).

The ideal weight for any adult is believed to correspond to his or her ideal weight from age 20 to 30. The most accurate method of determining body fat is the Body mass index nomogram, which has a correlation of 0.8 with body fat measured by more precise laboratory method. (Glass et al., 1999)

Body mass index, also called the Quetelet index (QI), is the ratio of weight in kilograms divided by height squared in metric units.

\[
\text{BMI} = \frac{\text{Weight in kg}}{(\text{height})^2 \text{ in metric units}}
\]

A typical nomogram is shown in Figure A.

BMI of 27 or more warrants treatment and attention. A BMI of 30 is roughly equivalent to 30% excess body weight. Overweight is defined as BMI of 25 to 29.9. Obesity is defined as BMI of 30 or more (Glass et al., 1999).

The body mass index (BMI, kg/m²) is also widely used as a measure of relative weight among adults, and its use among children and adolescents is rapidly gaining acceptance (Cole, 1991; Dietz and Bellizi, 1999). Mean BMI levels increase rapidly (by ~ 5 kg/m²) during the first year of life, but subsequently decrease and reach a nadir of ~ 15 kg/m² at 4-8 years of age. Levels subsequently increase, and reach values of 20-25 kg/m² by adulthood. The
beginning of this second rise in BMI has been termed the “adiposity rebound” (Cachera et al., 1984).

The age at which the minimum BMI occurs \( \text{age}_{\text{min}} \) is inversely related to adult levels of relative weight and an early adiposity rebound (e.g. before 5.5 year of age) increases the risk of adult obesity (Cachera et al., 1984; 1987; Williams et al., 1999). It has been suggested that early childhood may be a critical period for the development of obesity (Dietz, 1997), with an early adiposity rebound indicative of hyperplastic obesity (Cachera, 1984) or allowing for a longer period of adipose tissue accumulation (Siervogal et al., 1991).

It is uncertain, however if \( \text{age}_{\text{min}} \) is predictive of adult obesity if childhood levels of BMI are known. Although Whitaker et al. (1998) found the relation of adiposity rebound to adult obesity to be independent of the minimum BMI in childhood, it has been suggested by Williams et al. (1999) that the BMI levels at age 7 years could be substituted for \( \text{age}_{\text{min}} \).

Barlow and Dietz (1998) reported that the Maternal and Child Health Bureau, Health Resources and Services Administration, the Department of Health and Human Services convened a committee of pediatric obesity experts to develop the recommendations for treatment of obesity in the US. The committee recommended that children with a body mass index (BMI) greater than or equal to the 85th percentile with complications of obesity or with a BMI greater than or equal to the 95th percentile, with or without complications should undergo evaluation and possible treatment. Clinicians should be aware of signs of the rare exogenous causes of obesity, including genetic syndromes, endocrinologic diseases and psychological disorders. Clinicians should screen for complications
of obesity including hypertension, dyslipidemia, orthopedic disorders, sleep disorders, gall bladder disease, and insulin resistance (Refer to Table A).

Usually obesity is due to positive energy balance. That is the intake of calories is more than the expenditure of calories (Sri Lakshmi, 1993).

Obesity may also be defined as maladaptive increase in amount of energy stored as fat. There is currently no method to accurately determine the optimal amount of body fat stores or the ideal body weight for a given individual. The method most commonly used in an office setting to determine the degrees of overweight is the nomogram that relates weight to age, sex and height. The shortcomings of this method include a failure to take into account ethnic differences and variations in body frame an inability to distinguish weight due to muscle from that due to fat, and the inclusions of obese children in the sample of children and adolescents studied to create the nomogram (Fink, 2000).

HISTORICAL PERSPECTIVE OF MEASUREMENT OF OBESITY

Quetelet

Lambert Adolf-Jacques Quetelet is credited with the concept of the Body Mass Index (BMI). The proposal was made in a monograph in 1835 on the development of the human body. It was Quetelet who introduced the concept of quantification in measurement of the human being, thus providing a framework for progress in epidemiology and statistics.
Table A RECOMMENDED CUT OFF VALUES FOR BODY MASS INDEX (BMI, IN KG/M²) FOR ADOLESCENTS WHO ARE OVERWEIGHT OR AT RISK OF OVERWEIGHT DURING ADOLESCENCE.

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<th>Age (year)</th>
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Figure Nomogram for Body Mass Index.
Dananaberg and Caro (2001)
LIFE INSURANCE DATA

As reported in Handbook of Nutrition and Food (Brary, 2001), nearly 70 years after Quetelet the Life Insurance industry in the United States began to weigh in on the importance of excess weight as a risk for early death. It was also noted that a central distribution of weight was important. The 1922 statistical Bulletin of the Metropolitan Life Insurance Company reported:

*It is generally recognized that weight of the human body in relation to its height plays a part in determining the health and longevity of the individual. It is only recently, however, that the long experience of the insurance companies has made possible the crystallization of this impression into a series of definite propositions. It is known now, for example, that overweight is a serious impairment among insured lives, the gravity increasing with the excess in weight over the average for the height and age. But even this statement has its exceptions because, at younger ages, a limited amount of overweight is apparently an advantage. Such persons have uniformly lower death rates from tuberculosis. It is after the age of 35 that overweight, even in relatively small amounts, begins to be dangerous. The seriousness increases with advancing age and with the amount of overweight.*

From this point forward until the last decade of the 20th century, there were “weight tables” of appropriate desirable, or ideal weight proposed by the life insurance industry. The Framingham study, which was the first American effort at a long-term population based evaluation of health risks, used the Metropolitan Life Insurance Table of 1959 as the basis for comparing the weights of people living in Framingham with some standard. The term came to be called the Metropolitan Relative Weight, which was the weight for
height of an individual to the expected weight for height from the Metropolitan Life Insurance Table median frame grouping.

OTHER INDICES

Several indices relating height to weight were proposed in the middle of the 20th century. The BMI or what might be appropriately called the Quetelet Index (QI), was compared against several other indices by Keys et al. (1972). They evaluated three other indices of weight and height and found QI to be the best.

CURRENT ASSESSMENT OF OBESITY

Measuring weight is easy and inexpensive, while measuring body fat is not. Consequently overweight is often used as a proxy for obesity. Because body weight and body fat are highly correlated in most cases an individual classified as overweight would also meet the criterion for obesity. There are exceptions however, so the choice of measuring weight or body fat must be made in the context of specific research (Brownell, 1995).

It is very important to identify and accurately estimate obesity. BMI the anthropometric index (BMI = kg/m²) are recommended to identify overweight children (Dietz et al. 1999; Wang and Adair 2001; Freedman et al. 2001). Also the WHO and International Obesity Task Force advocate BMI reference for international uses. WHO defined overweight as (BMI > age-sex specific) BMI> 85th percentile.

The WHO guidelines define adults with a BMI of 25 kg/m² or more are overweight and those with a BMI of 30 or more as obese as shown in Table-A. Being overweight in childhood is thought to be an important determinant of adult obesity (Williams, 2001).
QI or BMI has been in use since the last hundred years and is one of the most frequent methods used to measure obesity.

**GENDER DIFFERENCES IN IDEAL BODY WEIGHT**

As reported by Crawford and Campbell (1999), the BMI at which women considered themselves to be at their ideal weight was significantly lower than that for men. While 46% of men described their ideal weight at a BMI of more than 25 kg/m² only 17.2% of women did so. One in seven women (14%) defined their ideal weight at a BMI less than that currently used to define underweight, compared to only 1% of men.

Ideal body weight is calculated by following formula:

\[
\text{IBW} = \frac{\text{Actual weight}}{\text{Ideal weight}}
\]

**OVERWEIGHT AND GENDER**

According to Crawford and Campbell (1999), the BMI at which women considered themselves to be overweight was significantly lower than that for men, and was well within the acceptable BMI range. The average weight at which women considered themselves overweight translated to a mean BMI of 23.7 kg/m², whereas the average weight that men defined as overweight translated to a mean BMI of 26.1 kg/m².

**EPIDEMIOLOGY OF OVERWEIGHT AND OBESITY**

The diseases of overweight and obesity are global in scope. For every developed country in the world in which data are available, the incidence and prevalence of excessive weight have increased over time (Keil and Kuulasmaa, 1989). Epidemiologic analysis of obesity is complicated in that the criteria by which overweight and obesity are defined have shifted over time. Currently
it is recommended that body mass index [BMI, weight, divided by
height squared (kg/m²)] be used for establishing diagnoses of
overweight (BMI of 25.0 - 29.9kg/m2) and obesity (BMI> 30.0 kg/m²)
Danato et al., (1998). These cutoffs were chosen as predictors of
morbidity and mortality. Based on these criteria it is currently
estimated that 64 million Americans are overweight and another 44
million are obese (Kuczmarski et al., 1997), and these figures reflect
an increase in the prevalence of obesity over time. The prevalence
of both overweight and obesity has increased dramatically in both
men and women in the United States between 1960 and 1994. The
overall prevalence of overweight has increased from 43.3% of
individuals aged 20 to 74 during the period 1960 to 1962 (Stamler et
al., 1978) to 54.9% of all individuals 20 years and older during the
period 1988 to 1994 (Flegal et al., 1998). In parallel, costs for weight
related health care have sky rocked.

According to a survey published in Indian Express dated,
31st March, 2002 obesity among children and adolescents in
India also has assumed an epidemic proportion.

A study was reported by Misra et al. (2001) on prevalence of
diabetes and obesity in urban slum population in northern India,
Based on Body mass Index (BMI), Obesity was more prevalent in
females (15.6%) than in males (13.3%). On the other hand,
classifying obesity based on percentage body fat, 10.6% of males
and 40.2% of females were obese. High waist-to-hip ratio (WHR)
was observed in 9.4% of males and 51.1%, of the females. In both
males and females above 30 year of age, there was a steep
increase in the prevalence of high waist hip ratio and in females,
body fat percentage was very high (particularly in % BF quartile >
30%). In a guide to obesity management printed by Pharmaceutical
company Cipla (2001) desirable waist to hip ratio for females is <.8, and for males <1.0.

OBESITY IN ADOLESCENCE

Obesity is among the most easy to recognize and the most difficult to treat of medical conditions. Obesity is the commonest problem among adolescents and children also, because of its association with other disease. Obesity in children occurs as a result of a complex interaction between genetic and environmental factors (Franzese et al., 1998). The financial burden of Childhood obesity for industrialized societies can only be estimated. The annual economic costs due to medical expenses and loss of income as a result of complications of adult obesity is approximately 70 billion dollars in United States. At least another 30 billion dollars are thought to be spent on diet, foods products and programs to lose weight. Obesity in childhood and adolescence has already become a major factor in healthcare planning systems and within the health care industry as such (Kiess et al., 2001), and needs attention.

Adolescence is a period of transition from childhood to adulthood, marked by interlocking changes in the body, the mind and in social relationships. The body develops in size and reproductive capability and becomes more sexually defined. The mind becomes more capable of abstract thinking, future orientation, internal control and a wider awareness of the environment; and the close relationships and dependence on parents and older family members begins to give way to more intense relationships with peers and adults outside the family. (Friedman, 1989)

Adolescence has been defined chronologically by WHO (World Health Organization) as being between the age of 10 to 20 years recognizing that this is a definition of statistical convenience
rather than one which identifies the precise timing of biological, social and psychological changes that characterize it. Most cultures relate the beginning of adolescence to the onset of puberty. Regardless of culture, however, some changes are universal (Friedman, 1989).

The onset of puberty is triggered by a complex interplay between the endocrine glands and the brain stimulating the secretion of sex hormones, which not only have various effects on body tissues but are also related to changes in sexual and emotional behavior. The timing of these events however shows wide variability from one individual to another, e.g. in normal boys there is roughly a five year range (from about 11 to 16) for the age at which puberty is reached. In girls, puberty begins on an average some two years earlier and extends over slightly shorter period. This is often the source of anxiety in adolescents who are highly sensitive to differences between themselves and their peers, especially in appearance. (Friedman, 1989).

Adolescence or Puberty is a high-risk period for the development of obesity, especially in girls. In boys, puberty is associated with large gains in fat free mass; in girls, there is a significant deposition in body fat along with a much smaller increase in fat free mass. In both sexes, there is an increase in central fat and decrease in peripheral fat. The influence and nature of changes in energy expenditure during puberty remains an important area for future investigation. (Fink, 2000).

According to (Fink, 2000), Unfortunately, 75% to 80% of obese adolescents become obese adults. Longitudinal studies indicate that overweight and fatness during adolescence, even if individuals are of normal weight as adults, result in increase adult
morbidity and mortality. Medical complications can begin before puberty if the individual is extremely obese. The social, psychological and economic consequences of obesity, although less often stressed, are of equal or greater magnitude.

ETIOLOGICAL AND THEORETICAL PERSPECTIVE OF OBESITY

According to Kiess et al., (2001) Multiple factors are related to high incidence of childhood obesity. Both genetic/endogenous and environmental/exogenous factors contribute to development of high degree of body fatness early in life. They further reported that at least 50 percent of tendency towards obesity is inherited. There is also increasing evidence that responsiveness to dietary intervention is genetically determined.

GENETIC AND ENDOGENOUS FACTORS

As reported in International (Kazdin, 1996), Obesity is not a new condition. As far back as the Stone Age, some humans had large amounts of fat stores, as is proved by primitive sculptures, carvings, and paintings found in caves and archaeological excavations. Even in historical times obese women were considered desirable. The reason for this may be the fact that nomadic human groups experienced periods of famine in which only those with adequate energy reserves could survive. The continuing existence of the group depended on females who could resist starvation and still breast-feed their babies. Therefore, males learned to select women that would be considered obese as mates, perpetuating the genetic trait of a tendency toward obesity.

This genetic trait determined a particular type of metabolism that responded to food deprivation by lowering its basal metabolic rate, thus burning less fuel, allowing longer survival during
starvation. When times of plenty returned, that low metabolism, paired with a large energy intake, induced obesity, which ensured a better survival during the next famine. This type of metabolism is still present in members of Western societies. Therefore, the tendency toward obesity is innate in many people. This statement is supported by an interesting study done by Stunkard (1986) and his collaborators, in which they examined the roles played by genetics and environment in the development of overweight children.

Their subjects were adopted children, their biological parents, and their adoptive parents. Based on body-mass index (the weight in kilograms divided by the square of the height in meters), Stunkard divided the children and the parents into four classes: thin, medium, overweight, and obese. The results of the study showed that there was a strong correlation between the body-mass index of the biological parents and that of their children; in contrast, no correlation was found between the children and the adoptive parents.

ENVIRONMENTAL/EXOGENOUS FACTORS

Exogenous factors such as over consumption of fat rich diets, the excessive use of modern media (TV) and lack of physical activity (sedentary life styles) always contributes to the development of obesity in childhood and adolescence as well as in adults. Nutrition and diet early in infancy is thought to influence growth rate and body fatness beyond infancy. (Kiess et al., 2001; Sharma, 2002).

Dietz (2001) reported that, in world over in past 30 years important changes have occurred in family eating patterns and in the consumption of fast food, pre-prepared meals, and fizzy drinks. Likewise the amount of physical activity that children engage in has
been reduced by an increase in the use of cars, and increase in the amount of time spent watching TV and a decrease in the opportunities in many communities for physical activities on the way to school or in school. Although television viewing seems to cause obesity in children, it is yet not clear how many of these other factors promote obesity in young children. Both food intake and activity in young children are strongly influenced by parents. In early childhood the more parents encourage children to eat certain foods the less likely they are to do so thus the foods that have been forbidden may have been overconsumed when children finally have access to them. Children of mothers who exert a high level of control over their food intake become less able to regulate their own intake although a mother’s reaction may occur secondary to their children inability to control their own food intake. Children who eat meals with their family consume more fruits and vegetables, fewer fizzy drinks and less fat and food both at home and away from home. Television advertising of food directed at young children helps, explaining why reduced Television viewing reduced risk of weight gain.

Josefson and Francisco (2001) reported that obesity and lack of exercise contribute to up to a third of cancers of the colon, breast, kidney and digestive tract, says a new report from WHO. The Centre for Disease Control and Prevention (CDC) estimate that obesity causes 300,000 deaths in the US annually, a number exceeded only by deaths related to tobacco. Half of the European adults and 61% of Americans are overweight. Moreover, the proliferation of Western Diets and Sedentary Lifestyles in developing countries poses a threat to people who were previously at low risk of obesity (Sharma, 2002).
THEORETICAL PERSPECTIVE

Two theories of the physiology of obesity are currently supported by research and are receiving the most attention in the literature (Brownell, 1982; Sri Lakshmi, 1993).

**Fat Cell Theory:** The fat cell theory states that there are at least two types of obesity. Hyperplastic obesity is an excess number of fat cells, and hypertrophic obesity refers to fat cells of excess size. Obesity that begins in childhood is usually due to hyperplastic cells; obese children may have 5 times the number of fat cells of normal weight children. Obesity with adult onset tends to be due to hypertrophic cells. Weight loss is typically accomplished by reducing the size of fat cells, but it is extremely difficult to reduce the number of fat cells. There are limits to reducing the size of fat cells, and Bjorntorp and colleagues (1975) showed that when fat cell size reached normal levels, women stopped losing weight and tended to drop out of the weight loss program. Thus, both fat cell number and fat cell size may have biologic limits to weight loss.

It appears that the health risks of obesity may be more related to fat cell size than fat cell number (Sjostrom, 1980). If this is confirmed then a hyperplastic obese individual could reduce risk of disease by decreasing fat cell size, although they would remain obese due to excess fat cell number. Such an outcome would satisfy health professionals, but it may not satisfy the obese individual because they would still be obese. In order to make the best use of the growing knowledge of fat cells, it is necessary to measure fat cell size and number. However, this is rarely done in practice because it requires a painful biopsy of fat tissue (Brownell, 1984).
In addition of the type of fat cell, the distribution of fat is a very important determinant of the health risks of obesity. It has been obvious for some time that men and women have different characteristic patterns of fat distribution, but it has only been recently learned that different levels of risk were associated with each pattern. Women tend to accumulate fat on the hips and thighs (pear shape), and this pattern does not increase Cardiovascular Disease risk very much whether it is found on men or women. Men characteristically accumulate fat in the abdomen (apple shape), and this pattern of fat increases Cardiovascular Disease risk substantially as well as risk of diabetes in both men and women (Krotkiewski et al., 1983). Gillum (1987) studied fat distribution in the 1960 National Health Examination Survey of 2669 adults. Excessive abdominal fat was associated with blood pressure, total serum cholesterol, and diagnosed coronary artery disease. The best index of Cardiovascular disease risk from obesity now appears to be the ratio of waist to hip circumference, and this measure is being used increasingly in obesity and Cardiovascular disease research.

**Set Point Theory:** A second physiological theory of obesity is called the set point theory. The set point is thought of as the body’s ideal weight, and the body will work to stay as close to that ideal weight as possible. The set point acts like a thermostat that attempts to keep temperature in an ideal range. While a set point may be “ideal” to an individual’s body, it may be well above the cultural ideal and it may be at a level that increases health risks. Two classic studies suggest that the set point theory may be valid. Keys et al. (1950) and colleagues studied 36 men who were maintained for an extended period on a starvation diet of half their usual calories. The men became preoccupied with food and had
numerous psychological problems. However, they also conserved calories so as to lose as little weight as possible. They became listless and ceased most activities. Their resting metabolic rates also decreased dramatically so that they were burning very few calories. The same phenomenon has been documented with dieters. The more that food is restricted, the more the body resists losing weight by reducing caloric expenditure. Sims and Horton (1968) studied prison inmates who volunteered to gain 20 to 25 percent of their body weight. Many of the men had difficulty gaining weight even though they ate large amounts of food. They lost interest in food and required more calories than expected to maintain their weight gains. After the experiment the men returned to their starting weights quickly. It seems that the body has a preferred weight, and it is difficult to change that weight very much because metabolism changes to preserve that weight.

A landmark animal study by Brownell (1986) and colleagues, indicates that repeated dieting may create physiological changes but does not change the set point. The Control rats were fed regular chow. The Obese Controls were fed high-fat chow consistently. The Obese cycling rats were put on a cyclic diet. They were fed high-fat chow until they became obese. Then they were subjected to two cycles of food restriction and re-feeding. Weight loss was rapid during the first restriction period, but it was twice as slow during the second restriction. Weight gain was 3 times faster during the second re-feeding than during the first one. The dieting pattern was designed to be like the “yo-yo” dieting of many obese humans, who diet and regain weight repeatedly. This study shows that frequent dieting may make it more difficult to lose weight later. The yo-yo effect implies that it is important to maintain weight that is lost,
because repeated cycles of loss and regain can lead to increases in fat stores.

All these studies indicate that the body makes adjustments to maintain a body weight. The resting metabolic rate increases or decreases, and there are changes in the desire for food. While these studies and others support the set point theory, it is far from proven. There is no way to know what an individual’s set point is. If it is true, set point theory suggests that losing weight is not as simple as decreasing food intake.

MEDICAL COMPLICATIONS OF OBESITY

The percentage of obese adults may be increasing slowly. Not only is 15 to 30 percent of the population obese but also obesity is associated with a long list of serious health problems. Most important, there is a strong relationship between obesity and mortality. (Kaplan, 1993).

Obesity is a very common condition. Using the definition of 20 percent overweight, the prevalence of obesity increases from about 7 percent of 20 to 24 years old men to 17 percent of 35 to 44 years old men. The prevalence is higher among women, increasing from about 10 percent in 20 to 24 years old women to about 35 percent in 55 to 64 year old women. (Bray, 1983)

Obese children have a tendency towards even more excessive weight. It has become clear that childhood obesity has reached epidemic proportion in all industrialized countries. The current age adjusted prevalence may be as high as 20-30 percent. The prevalence of massive obesity as defined as a BMI of ≥ 25 kg/m² in French infants was found to approximately 2% in 1996. (Kiess et al., 2001).
Many epidemiological studies, prospective, cross sectional and retrospective studies have shown that the risk of developing certain health problems and a shortened life span is higher among overweight individuals than among the non-overweight of the same sex, race, age and socioeconomic status (Van Itallie et al., 1990). The risk increases as the degree of obesity increases. They also increase depending on the patterns of fat distribution, with the upper segment obesity pattern ("abdominal", "apple" or "android") associated with a higher risk of developing physiologic complications than the lower segment ("Femoral-gluteal", "pear" or "gynoids"). The pattern of fat distribution can be determined either visually or by measuring the circumferences of waist and hip. A waist to hip ratio (WHR) > 0.8 in females and WHR > 1.0 in males is high enough to provide that risk. (Bjorntorp, 1987)

Obesity affects a great variety of physiological functions. Blood circulation may be overloaded as body weight increases. Prevalence of carbohydrate intolerance in grossly obese subjects is nearly 50%

**DISEASES ASSOCIATED WITH OBESITY**

**Mortality**

According to Dananberg and Caro (2001), Obesity and overweight themselves independently confer an increased risk of mortality. Although the link between obesity and increased rates of morbidity and mortality has been questioned in older population, many other studies have established such a direct linkage. The increase in risk begins to rise at a BMI greater than 20kg/m². The risk rises slowly at levels over 25 kg/m² and then rises steeply at levels greater than 30 kg/m². Individuals with a BMI of 30 kg/m² or greater have a 1.5 to 2 fold excess independent risk of mortality.
than do individuals with a BMI less than 25 kg/m². Paradoxically, a BMI lower than 20 kg/m² is associated with a modest increase in mortality, even after adjusting for confounding variables. The data relating obesity to mortality risk were drawn from epidemiologic studies of primarily white populations. In other groups, the inflection point at which mortality risk increase with increased BMI may be shifted. For example, in black American populations mortality risk appears to risk at BMI levels of 27 kg/m² and greater.

**Cardiovascular And Cerebrovascular Disease**

According to Dananberg and Caro, (2001) overweight, obesity and abdominal fat increase the risk of the both cardiovascular and cerebrovascular disease. The reasons for the increased risk for cardiovascular and cerebrovascular diseases may include elevations of blood pressure, low density lipoprotein cholesterol (LDL-C), triglycerides, small dense LDL-C, total cholesterol, librinogen, plasminogen activator inhibitor-1, and insulin and decreases in high density lipoprotein cholesterol.

In 1983, the Framingham study published a 26-year follow-up of participants showing a 26% to 46% increase in the relative risk for cardiovascular disease in individuals who were 30% over ideal body weight versus individuals at ideal body weight. In the Nurses Health Study of more than 100,000 women monitored for 14 years, the risk of coronary heart disease (CHD) was nearly two times greater in subjects with a BMI between 25 and 28.9 kg/m² and nearly three times greater in subjects with a BMI of 29 kg/m² or greater than in subject with BMI of 21 kg/m² or lower. In a cohort study of middle-aged British men, the incidence of coronary events increased 10% for each-1kg/m² rise in the BMI over 22. Although in some studies an association has been well established in all studies that
controlled for other risk factors such as smoking, age, family history, upper body adiposity, and menopausal and hormone replacement status.

Hypertension

According to Dananberg and Caro, (2001) the INTERSALT study involving more than 10,000 men and women reported that a 10 kg increase in weight was associated with a 3.0 mm Hg rise in systolic blood pressure and a 2.3 mm Hg rise in diastolic blood pressure. This degree of blood pressure elevations has been associated with a 12% increase in CHD and a 24% increase in stroke. The precise mechanism by which changes in weight alter blood pressure has not been established.

Congestive Heart Failure

According to Dananberg and Caro, (2001) both overweight and obesity have been shown to be independent risk factors for the development of congestive heart failure. Furthermore, because both hypertension and diabetes are also associated with congestive heart failure, the overall risk when these dependent factors are taken into account is proportionally increased.

Stroke

According to Dananberg and Caro, (2001) fewer studies have carefully examined the association of cerebrovascular disease and weight versus cardiovascular disease and weight. An association has been established in the evaluation of both fatal and nonfatal strokes, particularly when a subset of patients with ischemic stroke is evaluated. The risk of stroke is nearly twofold higher in women with a BMI greater than 32 kg/m² than in women with a BMI less than 31 kg/m².
Diabetes Mellitus

According to Dananberg and Caro, (2001) numerous studies have shown an association between increases in weight and the development of type 2 diabetes mellitus. In fact, the risk for diabetes increases at BMI levels below that established for the diagnosis of overweight. In the Nurses’ Health Study, BMI values above 22 kg/m² were associated with an increased risk of diabetes. It has been estimated that the relative risk for diabetes increases by 25% for each unit of BMI above 22 kg/m². It has also been estimated that more than a quarter of all newly diagnosed cases of diabetes in the United States were due to weight gain of more than 5 kg.

Cancer

According to Dananberg and Caro, (2001) cancer of the colon, particularly the distal end of the colon, has been shown in a number of studies to be strongly associated with obesity in men, as well as obesity in women, but to a lesser extent. In some cases, the incidence of colon cancer is nearly twofold greater in individuals with a BMI greater than 29 kg/m² than in those with a BMI less than 21 kg/m².

Although obesity has been shown to be inversely associated with the incidence of breast cancer in premenopausal women, that “protection” diminishes approximately 10 years after menopause. Increase of 8 kg over adulthood can lead to a twofold increase in the risk of breast cancer, an association that is particularly evident for women who have not received postmenopausal estrogen replacement therapy.

Female Reproductive Health

According to Dananberg and Caro, (2001) polycystic ovarian syndrome, a disorder that includes hirsutism, obesity, ovulatory and
menstrual dysfunction, and insulin resistance, is among the most common causes of altered reproductive function in women who are overweight. Even modest increases in weight in young women can adversely affect fertility, and women with polycystic ovarian syndrome and obesity have an increased risk of infertility.

Obesity during pregnancy is also associated with excessive morbidity. Pregnant women with obesity have a nearly a 10-fold excess risk of hypertension and a significant increase in the risk of gestational diabetes. Furthermore, the risk of congenital malformations, primarily neural tube defects, is increased in the pregnancy of obese women. Finally, increased weight before pregnancy has been shown to result in an increased risk of adverse fetal outcomes.

**Other Diseases**

According to Dananberg and Caro, (2001) for women with a BMI greater than 40 kg/m², the risk of gallstones is nearly seven times higher than for women with a BMI less than 24 kg/m². A twin study estimated that for every 1-kg rise in body weight, the risk of osteoarthritis increases by approximately 10%. Conversely, weight loss is associated with an increase in pain-free range of motion and a decrease in analgesic use. Sleep apnea is another morbidity associated directly with weight gain. Diagnosis and treatment of sleep apnea in obese patients are particularly important because of the sequelae of hypoxia, hypertension, myocardial infarction, and cardiac arrhythmia.

McCarthy (2000) in a study found that much of the blame for the diabetic problems seems to lie with rapid rise in obesity. It was further found that on an average for every added kg in weight, a person's risk of diabetes rose by about 9 percent. Such a rise may
indicate that many are already dangerously overweight, making even a small gain enough to push them over the diabetic threshold.

There is no doubt that obesity is an undesirable state of existence for children. It is even more undesirable for adolescents for whom even mild degrees of overweight may act as a damaging barrier on society obsessed with slimness.

Obesity has become an epidemic and prevalence of adolescents/child obesity is a recognized public health priority. Since obesity in adolescents predicts adult obesity and is associated with chronic disease, it is critical to understand current trends in obesity among adolescents (Kiess et al., 2001).

**NEED AND FOCUS OF THE STUDY**

Among most common sequel of primary childhood obesity are hypertension, dyslipidemia, and psychosocial problems. An increase in risk of death from cardiovascular disease in adults has been in subjects whose BMI had been greater than the 75th percentile as adolescents childhood obesity seems to increase the risk of subsequent morbidity whether or not obesity persists into adulthood (Kiess et al., 2001).

Obesity is a multifactorial condition with wide ranging causes. Both genetic, psychological and life style factors are involved.

Psychological factors have also been identified as key causative factors in obesity and weight gain. The Psychodynamic explanation of obesity centered on two hypotheses. First there were unconscious conflicts arising from childhood that led to overeating. Second, overeating was seen as a means of coping with emotional distress such as depression and anxiety. Increase in emotional
distress during dieting is evidence for psychodynamic view (Kaplan et al., 1993).

There is also some evidence about the role of personality, self concept and body image in obesity. Diet and physical activity also been known to play a major role in maintaining weight status.

Adolescence is a period when weight gain parameters may play a precise role. Hence the focus of the study was to compare obese and non-obese adolescents on Personality and its dimensions, Perceived Stress and Strain, Perceived Family Environment and its dimensions, Perceived Parental Acceptance Rejection and its dimensions, and Attitude towards Body Image and its dimensions. Indices of Negative Affect (viz., Irritability and Depression), Optimism and Psychological Well-Being and Eating habits.