REVIEW OF LITERATURE

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One of the major topics of discussion around the globe today, is the problem of overweight and obesity. It is not restricted to being only a clinical problem, but it has now attained a public health dimension. It is discussed unanimously among scientists, academics, medical health practitioners and various public health policy planners. It has emerged as one of the most serious public health problems found in all age groups.

Overnutrition is associated with a number of consequences ranging from life style diseases such as diabetes, cardiovascular disease, hypertension and some forms of cancer and social and psychological impact on afflicted children and adults. Obesity is due to the interaction of genetics, behaviour and environment. However controversies do exist in certain areas of its assessment and prevention and management at the individual and public health levels.

The review of literature is discussed hereunder to facilitate in-depth understanding of the various aspects of childhood obesity.

- Obesity : An overview
- Magnitude of the problem of childhood obesity

   Global scenario

   Indian scenario
- Childhood obesity : Etiology and risk factors

   Genetics and Environmental factors

   Lifestyle related factors

   Dietary factors

   Hormonal factors – Role of leptin in obesity
- Concerns related to childhood obesity
- Metabolic aberration in childhood obesity
- Assessment of obesity in children

   Indicators used for the assessment

   Defining overweight and obesity in children
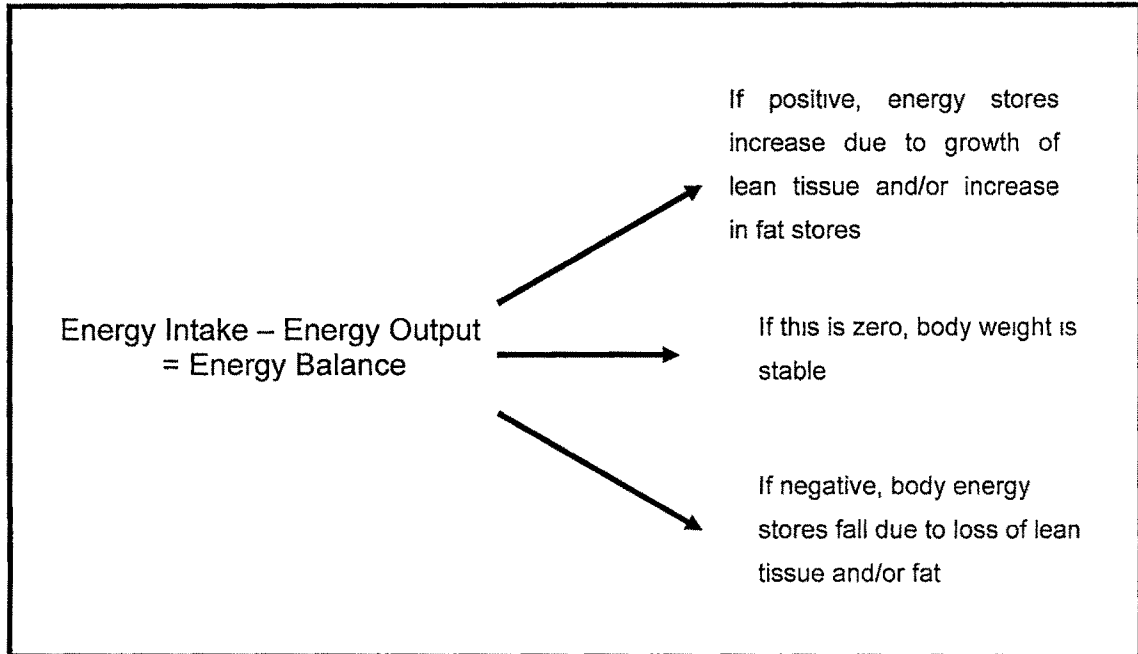
OBESITY : AN OVERVIEW

Till recently, weight gain and accumulation of fat in human body was considered as one of the signs of health and prosperity. However with the changing times the people at large have started realising that it is one of a growing threat to the human health. Obesity is a chronic disease found in children as well as adults ; in developed as well as in developing countries. Obesity is now considered to be a major factor contributing to ill health and is given more significance than undernutrition and other infectious diseases. It is a key risk factor in the development of various chronic mainly diets related non-communicable diseases.

Evidences of obesity have been found as far as Greek-Roman times. Bluemenkrantz (2002) opined that obesity is probably the oldest metabolic disturbance as an obese Stone Age statue has been uncaved. Similar evidences have also been found in Egyptian mummies and in Greek sculpture. People tend to become obese when an inconsistency arises between their energy intake and energy expenditure due to consumption of more food and excess leisure time (Figure 2.1).

The prevalence of obesity had never been such widespread before as it is in the recent times. Over the years a series of changes have occurred in diet, nutritional status and physical activity of the human race due to major demographic and socio-economic changes in their life style.
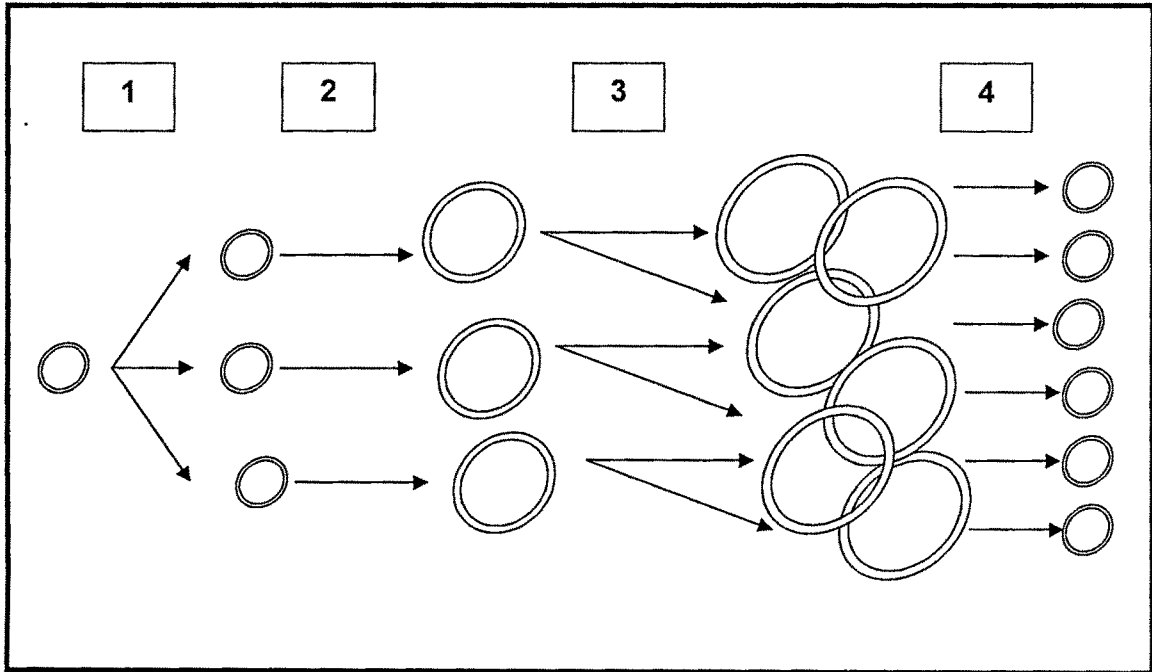
The nineteenth century saw the works of Lavoiser and others, which indicated that the process of metabolism was similar to combustion and both obese and lean humans obeyed the laws of thermodynamics. The discovery that fat is stored in 'cells' led to the idea that obesity arose due to too many fat cells (Figure 2.2). Further statistics of various insurance companies led to conclusion that obesity was associated with an increased mortality rate. A more familial basis for obesity was suggested along with descriptions of genetic disease such as Cushing's disease, hypothalamic obesity etc. Later the introduction of thyroid

FIGURE 2.1

THE ENERGY BALANCE EQUATION

Energy Intake – Energy Output = Energy Balance

If positive, energy stores increase due to growth of lean tissue and/or increase in fat stores

If this is zero, body weight is stable

If negative, body energy stores fall due to loss of lean tissue and/or fat

Source : Webb 2002

FIGURE 2.2

HYPERTROPHY AND HYPERPLASIA OF FAT CELLS

1- During growth fat cells increase in number.
2- When energy intake exceeds energy expenditure, fat cells increase in size.
3- If energy intake still continues to exceed energy expenditure, fat cells increase in number again.
4- When fat loss, the size of the fat cells shrinks, but not the number.

Source : Webb 2002

hormone, dinitrophenol and amphetamine as one of the pharmacological treatments of obesity opened the doors to the use of drugs and the field of genetics and established various forms of obesity resulting from genetic defects.

Omran (1971) conceptualised the Theory of Nutrition Transition stating that the epidemioiogical transition moves from a pattern of high prevalence of infectious diseases and malnutrition to one in which chronic and degenerative diseases predominate. Popkin (1998) supported this theory and gave the current stages in the nutrition transition and its health implications (Figure 2.3). The period after the industrial and second agricultural revolution was marked by a shift to the high fat refined Carbohydrate, low-fibre diet with an increase in obesity, non insulin dependent diabetes mellitus and other related chronic diseases. Thus, the theory indicates that these stages relate to the complex interplay of changes in patterns of agriculture, health and socio-economic factors.

There exist two theories of obesity - Push theory and Pull theory.

Push Theory

According to Push theory, obesity results due to voluntary pushing of excess nutrients in a body. It is assumed that obesity is primarily an eating disorder. Hence the line of remedy is on behavioural and dietary changes.

Pull Theory

According to Pull theory, obesity results due to inborne metabolic predispositions. This leads to false homeostatic signals resulting in condition maintaining excess fat deposition. This theory further proposes that overfeeding behaviours are caused by metabolic factors and are not causative factors themselves. The treatment of obesity is not that easy, as one needs to comprehend the metabolic and genetic modifications responsible for obesity.

Childhood obesity results from various factors such as genes, environment, lifestyle and their interactions. Some of the causative factors of obesity are enlisted in Table 2.1.

FIGURE 2.3

NUTRITION TRANSITION AND ITS HEALTH IMPLICATIONS

OMRAN CONCEPT (1971)	POPKIN CONCEPT (1998)
The age of collecting food ⇩	• Hunters perform high physical activities • Diet low in fat & high in fibre • Taller robust with fewer nutritional deficiencies
The age of famine (After 10-12,000 years) ⇩	• Early agriculturalist • Simpler diet with fluctuations
The age of receding famine ⇩	• Industrial & second agricultural revolution • Reduced problems of famine • Large shifts in diet
The age of degenerative disease ⇩	• Marked shift to a high fat refined CHO and low fibre diet • Increase in obesity & all CDDs
The age of behavioural change ⇩	A new stage as a reaction to DNCDs

Source : Popkin 1998

TABLE 2.1

CAUSATIVE FACTORS OF OBESITY

Factors	Determinants
Biological factors	• Early nutrition injury • Metabolic programming • Gene medicated adaptation
Ecological factors Demographic changes	• Reduction in infant mortality • Increase in life expectancy • Urbanisation
Lifestyle changes	• Lower energy expenditure • Labour mechanisation • Automation • Television viewing • Other secondary activities
Dietary changes	• Fast-food culture • Increase in energy density of diets (> % fat) • More meals consumed outside home.

Source : Caballero 2001

During the last two decades obesity has manifested itself as an epidemic in developed countries. It is a matter of alarm alike for both wealthy and middle income people in developed as well as developing countries. The prevalence of obesity is not restricted to any one region, country, or ethnic grouping. The highest levels of obesity have been observed in Middle East, Western Pacific and Latin America among middle and lower income countries. In Asia it is less common but the prevalence is on an increase. The problem is increasing rapidly in children also as studies indicate that obesity in school children is approaching nearly 10%, not only in industrialised countries such as US, Canada and Japan but also in industrialising countries such as Argentina, Egypt, Chile, Indonesia, Peru and Thailand (Yadav 2002).

Obesity is defined as a condition of abnormal or excessive fat accumulation in adipose tissue to the extent that the health may be impaired. The individuals with obesity differ not only according to degree of excess fat, but also in the regional distribution of the fat within the body. The distribution of fat induced weight gain affects the risks associated with obesity and the kinds of resulting diseases. The excess abdominal fat is a great risk factor for various diseases. Hence there is a need for a method differentiating people with android obesity in which there is abdominal fat distribution and those with gynoid fat distribution in which fat is more evenly and peripherally distributed around the body.

Highlights - Overview

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

- Visceral upper body fat carries more risk than lower body fat. Obesity is now considered to be one of the major factors contributing to ill health.

> - An increasing prevalence of obesity is a matter of concern alike for both wealthy and middle income people, in developed as well as developing countries.

MAGNITUDE OF THE PROBLEM OF CHILDHOOD OBESITY

Global Scenario

Obesity is now widely prevalent in several developing countries, particularly those in rapid transition and is affecting both children and adults. It is now competing with other traditional public health concerns such as undernutrition and infectious diseases responsible for ill health of the population. The increasing prevalence of obesity in a population, particularly among children and adolescents is an early indicator of emerging health burden resulting in non-communicable diseases in developing societies.

In the US since the 1960's, large nationally representative surveys have assessed the prevalence of obesity in children. The National Health Examination Surveys (NHES) I, II and III have reported the prevalence to be ranging from 25%-30%. The surveys found that the Hispanics, Native Americans and Black patients were more affected. The estimates from Canada revealed childhood obesity from 7%- 43%. The condition is more common among native Canadians. A national survey in Britain in 1974 showed that 7.3% of the children and adolescents between 7-16 years of age were obese. In Japan between 1974 and 1993, the frequency of obese school children increased from 5% to 10%. In Thailand also it rose from 12.7% to 15.6% during 1991 to 1993. In Saudi Arabia it was 15.8% among males from 6 to 18 years of age (Yadav 2002).

In the US the prevalence of overweight increased from 15% in 1963 to 22% in 1991 in children and adolescents (> 85th centile of US reference population used to assess overweight). In the UK the triceps skin fold

thickness of 7 year old children increased by about 10% from 1972 to 1994. Recent data revealed that in the age group of 6-11 year old children and in adolescents (12-17 years) the number of obese children was greater than 22%. The prevalence of obesity in both these groups has more than doubled in the last 20 years (Dimarco 2001).

There has also been a rapid increase in childhood obesity in England, US and certain other countries of the world. Bundred et al (2001) reported that among 3 to 4 year old English children, there was a 60% increase in the prevalence of overweight (having a BMI > 85th centile) and a 70% increase in the prevalence of obesity (BMI >95th centile) between 1989 and 1998. Blossner (1999) analysed 160 nationally representative cross sectional surveys from 94 countries and reported the global prevalence of overweight as 3.3%.

In Tunisia 9.1% of adolescent girls are at a risk for being overweight based on criterion BMI > 85th percentile (Cabellaro 2001). The risk of overweight among adolescent girls increases with age. In contrast to girls the risk of obesity in boys decreases with age. The risk of obesity at 19 years of age is 9.5% in girls as compared to 5.1% in boys (Mokhtar et al 2001). Kotani et al (1997) reported that in Japan the number of obese children aged 6–14 years increased from 5% to 10% between 1974 to 1993 (> 120% standard body weight used to measure obesity).

Chinn and Rona (2001) applied Cole et al's BMI standards to data on British children that were collected in 1974, 1984 and 1994. No change was found in prevalence of overweight and obesity between 1974 and 1984 but the prevalence increased by 44% and 67% in the next decade for overweight and obesity respectively. Flegal (1998) reported trends in overweight prevalence for youths 6-17 years of age by nationally representative surveys of NHES and National Health and Nutrition Examination Survey (NHANES). The prevalence of overweight increased primarily since NHANES in second half of 1970.

Pediatric obesity is now common worldwide. Investigators reviewed National Health Surveys on more than 6000 US children aged 6 to 18 years between 1988 to 1994 and more than 3000 children in china and nearly 7000 children in Russia from the early 1990s. In the US about 11% children were obese and more than 14% were overweight. In comparison 6% of kids in Russia were obese and 10% were overweight, while in China 3.6% kids were obese and 3.4% were overweight. It was also stated that in Brazil the prevalence of overweight increased from 4% to 14% for the years 1975 to 1997 and in China the prevalence moved from 6.4% to 7.7% in kids from 1991 to 1997. The data from a nationally representative sample of 2630 English children showed that the frequency of overweight ranged from 22% at age of 6 years to 17% at age 15 years (Reilly and Dorosty 1999).

A study on a large sample size to examine the trends of overweight and obesity in young persons aged 6 to 18 years using international reference revealed that the prevalence of overweight increased in Brazil from 4.1% to 13.9% (1975-1977), China 6.4% to 7.7% (1992-1998), United States 15.4% to 25.6% (1971-1974, 1988-1994). The annual rates of increase in prevalence of overweight were 0.5% in Brazil, 0.2% in China, 1.1% in Russia and 0.6% in US. The burden of nutritional problem is shifting from energy imbalance deficiency to excess among older children and adolescents in Brazil and China. The variation across countries may relate to changes and difference in key environmental factors (Wang et al 2002).

A study to assess the emerging problem of overweight and obesity on 1208 school children (8-10 years) of private and public school of Manila, Philippines revealed that private school children were taller and heavier and had high BMI values than public school children resulting in a much lower prevalence of undernutrition and a much higher prevalence of overnutrition (Figure 2.4). The prevalence of overweight and obesity in public school children was found to be 5.8% and 3.3% and that of private school children was 24.9% and 12.0% respectively (Florentino et al 2002).

FIGURE 2.4

PREVALENCE OF UNDERNUTRITION AND OVERNUTRITION IN 8-10 YEAR OLD SCHOOL CHILDREN OF MANILA
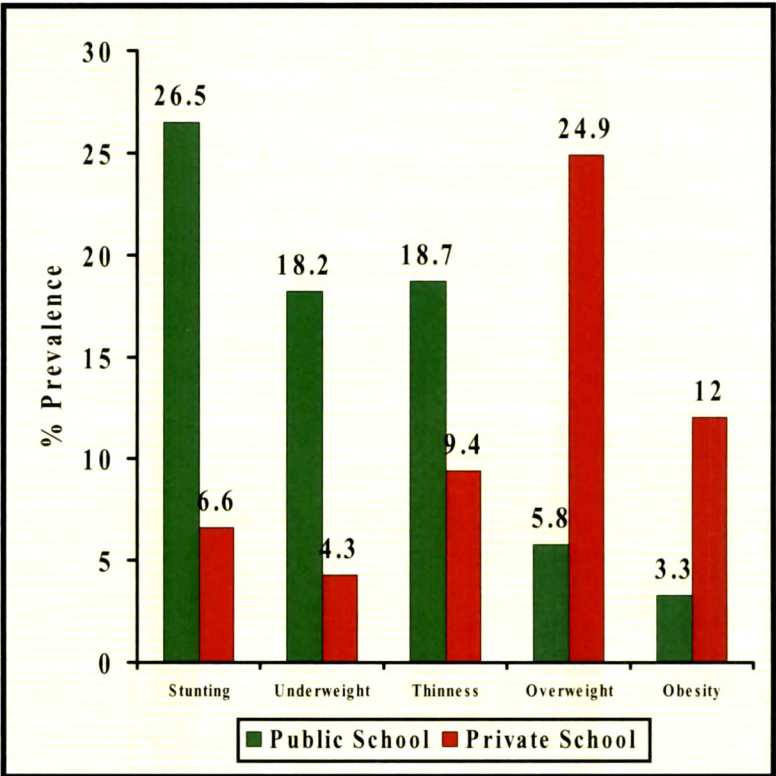
Source : Florentino et al 2002

Soekirman et al (2002) mapped the prevalence of overweight on 1367 school children aged 8 to 10 years in West Jakarta and Indonesia. The criterion used to classify children in overweight was BMI greater than 85th percentile for age. He reported prevalence of overweight to be 15.3% in girls and 17.8% in boys. More overweight children were identified in private schools than the public schools. In Malaysia, E-Siong Tee et al (2002) studied the prevalence of overweight or obesity on 5995 children (7-10 year old) from 160 schools. The prevalence of overweight at or above 95th percentile of BMI for age was 9.7% and 7.1% for boys and girls respectively. The overall prevalence was found to be 8.4%.

Al-Nuaim (1996) studied the prevalence of overweight and obesity among 9061 male school children (6 to 18 years) in Saudi Arabia. The 50th percentile of the National Centre for Health Statistics (NCHS) / Centre for Disease Prevention and Control (CDC) reference population was used as the expected standard population values for defining children as overweight or obesity. He found that the overall prevalence of overweight was 11.7% and that of obesity was 15.8%. Al-Shammari (2001) reported that out of 1848 children in Riyadh, Saudi Arabia 10.5% were overweight and 8.7% were obese based on age and sex specific percentile criterion. Among children a multivariate logistic regression analysis showed that 6 to 10 years and non Saudi children were associated with childhood obesity.

In the US, the prevalence of overweight of 5-24 year old has increased by almost two fold between 1973 and 1994. A similar trend was observed in Japan where obesity increased by 5%-10% among 6-14 year old in almost the same time span. It was observed that one third of these obese children grew into obese adults (WHO 1997). The prevalence of obesity in Italy among 10 year old children was found to be much higher, 2.2- 5.2 times as high as in other western countries (Esposito-Del et al 1996). In another study in United States it was observed that prevalence of obesity among 5-11 year old children was found to be significantly higher in black than in white children and was also significantly greater in girls than boys. It was reported that obesity increased from 13%-23% to 29%-47% in

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case of black children and 6%-10% to 22%-27% in case of white children (Figueroa-Colon et al 1997).

In a Canadian study the prevalence of obesity in 9-10 year children in low income multi ethnic group was found to be 15.1% in boys and 13.3% in girls (O'Loughlin et al 1998). In a Mexican study, physical activity and television viewing in 9-16 year old children were related to obesity prevalence, which was found to be 24% (Hernandez et al 1999). In yet another study obesity based on elevated BMI was highly prevalent (18%-32%) among American Indian school children aged 5-17 year (Zephier et al 1999). A few studies have also reported that obesity is often acquired during childhood and adolescence. One of the study carried out between 1971-1981 in Iowa city, US revealed that 48% to 75% of children in the upper quintile of BMI were found to be in upper quintile as adults also and 49% to 70% children in upper quintile of weight were found in the upper quintile as adults. These measures, track from childhood into young adult life and majority of obese children became obese (Clarke et al 1993).

In US, it was found that the prevalence of obesity increased considerably as students enter adolescence. The overall prevalence of obesity for grade 4, 5 and 6 versus grade 9, 10 and 11 was 9.1% and 14.8% respectively (Huse et al 1982). The alarming increase in obesity among early childhood is also reported in US. It was found that the proportion of obese (% ideal body weight for height > 120%) and severely obese (% ideal body weight for height > 140%) increased from 0% to 0.6% between ages 1 to 7 years (Unger et al 1990).

Ho (1983) studied the prevalence of obesity in Singaporean primary school children during 1976 to 1980 based on criterion as body weight above 120% of Harvard standard weight for height and reported the prevalence to be 3.51% with a significantly higher rate in boys (3.98%) than in girls (3.06%). Furthermore, primary 6th standard children had a higher prevalence rate (4.29%) compared to the primary 1st standard children (2.75%). The prevalence rates rose with a rate of 5.33% in 1980 compared to 1.8% in 1976. The tendency to become obese increased with age and boys were more prone to obesity. Lohman

et al (1999) assessed the body composition in 81 boys and 75 girls (11 years old) from height, weight, and triceps and sub scapular skin fold thickness and bioelectrical resistance. The study confirmed the high prevalence of excess body fat in school age American Indian children.

The findings from the Child and Adolescent Trial for Cardiovascular Health (CATCH) study in a multi ethnic pediatric population suggest that children in CATCH were markedly heavy and fat. The prevalence of obesity based on BMI and triceps skin fold (> 95th percentile) among CATCH children was higher in boys than in girls at both base line (9.1% Vs 8.6%) and follow up (11.7% Vs 7.2%). It was higher among African Americans and Hispanics than Whites for both sexes (Dwyer et al 2000). The prevalence of overweight and obesity among 5514 school children (4.5-17.4 years) in Seychelles was 12.6% and 3.8% respectively based on IOTF recommended Cole et al standards (Stettler et al 2002).

In a study carried out by Pakpcanikitvatna et al (2002) in Thailand on 4339 adolescents aged 10 to 16 years showed that 7.2% and 2.3% of male and female children were obese according to weight greater than 85th percentile for age and sex specific reference according to NCHS. When obesity was defined as BMI > 25, it was found that 11.0 % and 6.0% of males and females respectively had BMI > 25. Koon at al (2002) studied the prevalence on 1426 boys and 1326 girls (9.5 to 12.5 years) of Malay, Chinese and Indian ethnicity from 6 schools in Kula Lumpur, Malaysia (Figure 2.5). He reported that using WHO (1995) cut offs the prevalence of overweight was highest in Chinese (16.6%), followed by Indian (12.8%) and Malays (11.2%). However the prevalence of obesity was highest among Indians (10.3%) followed by Chinese (10.1%) and Malays (8.7%). In a retrospective study carried out in Singapore by Johnston et al (2002) the prevalence of overweight was found to be 41.6% in the age group of 6 to 12 year based on ideal weight for height charts for Singapore children.

The study carried out by Praenglampoo et al (2002) in Muang district of Thailand reported the prevalence of overweight, preobesity and obesity as

FIGURE 2.5

PREVALENCE OF OVERWEIGHT AND OBESITY IN SCHOOL CHILDREN OF DIFFERENT ETHNICITY
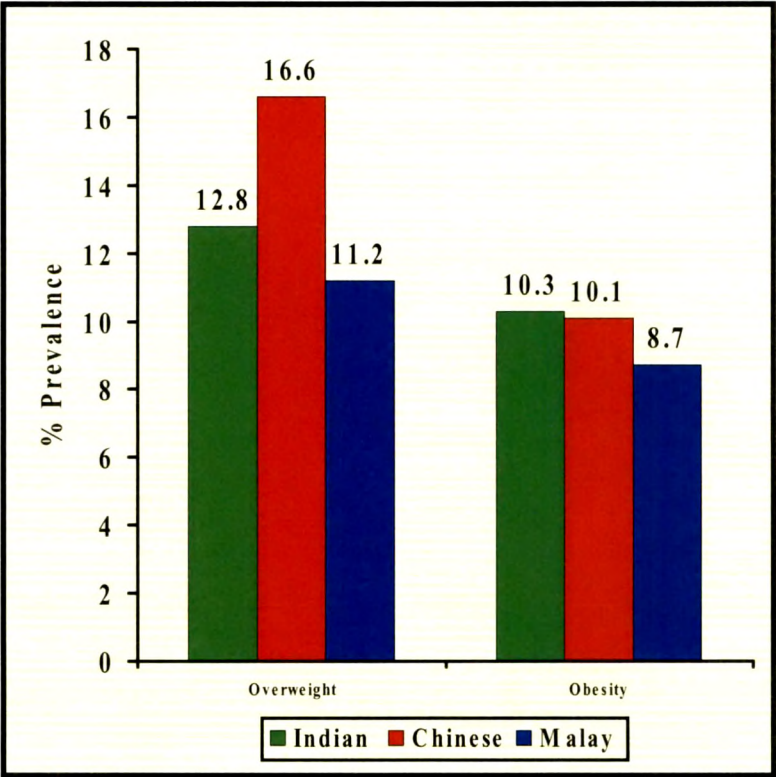
Source : Koon et al 2002

3.82%, 6.07% and 5.83% respectively. The study enrolled 2537 children in the age group of 6 to 15 years from public and private schools including primary to intermediate classes. The nutritional status of the children was assessed using the standard growth reference (weight to height) developed by Department of Health, Thailand (1994). The prevalence of overnutrition in boys was higher than that in the girls (18.19% Vs 13.46%). In private schools the prevalence of overnutrition among the students at the primary level was higher than that of intermediate students (19.18% Vs 14.72%). A recent study carried out by Frige and Heinrich (2003) also reported the increasing trend for overweight and obesity in 11 to 14 year and 8 to 10 year old children.

From the overview on global prevalence of childhood obesity, it is evident that the prevalence of obesity is increasing world wide including some developing countries, which earlier had very low prevalence rates for overnutrition.

Indian Scenario

Few studies, which are available on the childhood obesity, report an increase in prevalence of overnutrition in children and adolescent in India also.

A study carried out by Gupta et al (1990) amongst 3861 school children (5 to 15 years) of Delhi reported the prevalence of obesity as 7.5% using weight/height (kg/cm^2) > 2.26 as cut off point to classify children. In the study carried out by Jayshree (2001) in Dharwad, Karnataka reported the prevalence of overweight as 16.28% in children and 2.80% in adolescent based on WHO classification (1995). Vijayalakshmi et al (2002) assessed the prevalence of obesity in 1000 adolescent school children of different socio-economic group in Andhra Pradesh. In her study, overnutrition was assessed by 85th BMI percentile value whereas criteria for obesity was obtained by adding the BMI > 85th percentile value to the triceps and sub scapular > 90th percentile value according to WHO (1995). She reported that overweight was observed in 17.5% adolescent boys and 16.0% of the adolescent girls in the high income group whereas in low income group it was only 0.4% and 1.3% respectively. Obesity was present in 11.9% in the boys and 5.9% in adolescent girls of high income group. None of the

boys and girls of low income group showed obesity. Table 2.2 gives the comparison of clinical and anthropometrical variations seen in high income group and low income group children.

In a cross sectional study carried out by Kapil et al (2002) in Delhi on prevalence of obesity among 870 affluent adolescent school children (10 to 16 years), the overall prevalence of obesity was observed to be 7.4% based on international BMI cut off points. About 8% of boys and 6% of the girls were obese. The maximum prevalence of obesity was found during the pre pubertal period between 10 to 12 years.

Parikh (2002) and Akolkar (2003) from the studies carried out in school children of urban Vadodara reported that the prevalence of overweight and obesity based on Cole et al standards was higher in younger children (6 to 12 years) than older children (12 to 17 years). The prevalence of overweight and obesity was 20.3% in younger children and 7.7% in older children. Mani et al (2004) observed the prevalence of overweight and obesity to be 16% in adolescent children (10-18 years) in urban school children of Vadodara. Venugopal (2004) observed a similar trend in school children aged 11-15 years.

Thus the magnitude of the problem of overweight and obesity in children is of great concern.

Highlights - Magnitude

- Globally, though the affluent countries contribute more towards the proportion of children with overweight and obesity, developing countries like India are also not far behind.

- The increasing prevalence of obesity in a population particularly among children and adolescents is an early indicator of emerging

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TABLE 2.2

COMPARATIVE CLINICAL AND ANTHROPOMETRIC PROFILES OF HIGH AND LOW INCOME GROUP ADOLESCENTS

| Parameters	Boys		Girls	
	LIG (n = 246)	HIG (n = 303)	LIG (n = 244)	HIG (n = 238)
Prevalence (%)				
Overweight	0.4	17.5	1.29	16.0
Obesity	-	11.9	-	5.9
Clinical profile (mm/Hg)				
Systolic	103.8 ± 0.7	112.7 ± 0.6	102.1 ± 0.6	110.7 ± 0.6
Diastolic	71.5 ± 0.5	74.1 ± 0.4	68.3 ± 0.5	78.4 ± 0.4
Anthropometric profile				
Body weight (kg)	41.2 ± 0.4	53.18 ± 0.71	41.5 ± 0.3	51.89 ± 0.66
BMI	16.6 ± 0.1	19.84 ± 0.2	17.3 ± 0.1	20.69 ± 0.23
WHR	0.83 ± 0.003	0.80 ± 0.003	0.78 ± 0.004	0.78 ± 0.004
% of body fat	13.4 ± 0.2	21.66 ± 0.37	19.4 ± 0.3	30.15 ± 0.45

Source : Vijayalakshmi et al 2003

health burden resulting in non-communicable diseases in developing societies.

- Global epidemic of overweight and obesity is at our doorstep also.

CHILDHOOD OBESITY – ETIOLOGY AND RISK FACTORS

Hirsch (1997) stated that in the recent times the pathogenesis of obesity has shifted from simple analysis of psychological factors to in-depth knowledge of the mechanism of fat storage and energy metabolism. Obesity and its amelioration is the result of shift in dietary practices as well as physical activity.

On the basis of explanation regarding the control of energy intake by the dual center hypothesis, there has been a massive influence on assessment regarding control of energy balance and body weight. It has been verified that the satiety area of the dual center hypothesis - the ventromedial region of the hypothalamus plays a key role in the control of feeding and energy expenditure. Webb (2002) reported that though the dual centre hypothesis is regarded as quite basic, it does have the following three basic elements, a system requires for controlling food intake.

Appestat

| Satiety signals | Hunger drive | Energy expenditure |
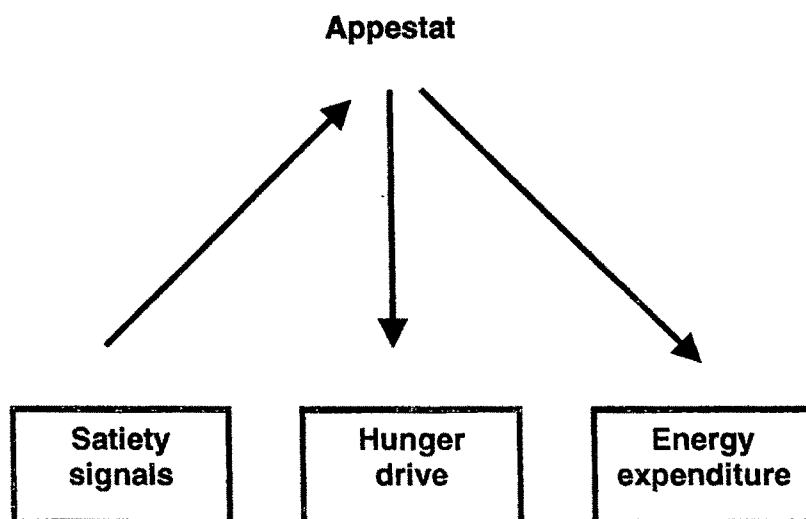
- Satiety signals - a series of physiological inputs to the appestat providing information about the feeding status and the level of body energy stores.

- The appestat - the areas within the brain that integrate information on feeding status and level of body energy stores.

- A hunger drive that can be modified by the appestat in response to the information received from the satiety inputs (also mechanisms for controlling energy expenditure.)

The satiety signals fall into three major categories.

Gut-Fill Cues

According to this theory the signals emanate from the alimentary tract and are transmitted via sensory nerves or hormones released from the gut and provide information about the amount and the nature of food in the gut. Smith et al (1981) reported that the hormone Cholecystokinin (CCK) is released from the intestine when food is present and it induces satiety and reduces feeding. It is said to act by activating sensory branches of the vagus nerve in the gut and these relay the information to the appestat.

The Glucostat Theory

After eating, the blood concentration of substrates - glucose and amino acid rises, as they are absorbed from the gut. These concentrations fall in the post absorptive state. If three meals are taken per day and it takes around four hours for its digestion and absorption, the food is being absorbed for most of the waking hours. The concept of blood glucose concentration or the rate of glucose utilisation is a major satiety signal.

The Lipostat Theory

These are the signals that emanate from adipose tissue and indicate the level of body fat stores (fat). Gordon Kennedy (1950) proposed this theory and has used to explain how body energy stores could be regulated in the long term by regulation of apparently short term phenomena like hunger and satiety.

The decline in physical activity is clearly implicated as an important cause of rapid increase in the number of overweight and obese people in Europe, US and in other countries (Prentice and Jebb 1995). However, even strong and consistent association between obesity and inactivity in a variety of studies has not proved that inactivity causes obesity. Webb (2002) reported that apart from the difficulty in measuring activity level, it is also likely that excess weight gain reduces fitness and exercise tolerance, which in turn discourages activity - an effect and cause relationship as shown below.

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┌─────────────────────┐         ┌─────────────────────┐
│                     │         │                     │
│     Inactivity      │  ───►   │     Weight gain     │
│                     │         │                     │
└─────────────────────┘         └─────────────────────┘
          ▲                                │
          │                                ▼
┌─────────────────────┐         ┌─────────────────────┐
│                     │         │                     │
│ Reduced exercise    │  ◄───   │  Decreasing fitness │
│ tolerance           │         │                     │
└─────────────────────┘         └─────────────────────┘
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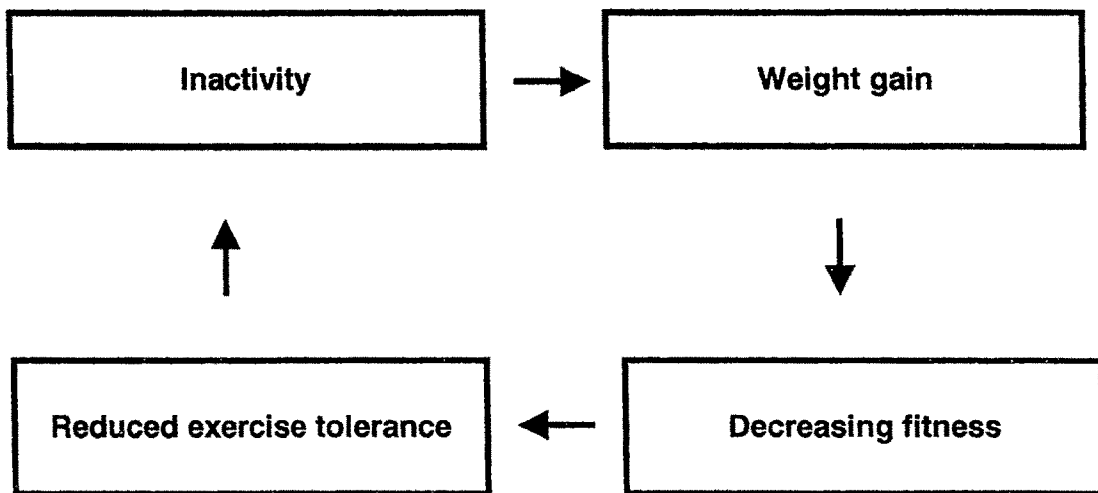
The cellular and metabolic features play an important role on the various behavioural elements and their study have now gained momentum. The studies on adipose tissue metabolism and cellularity that were carried out in the last two to three decades have gained much significance with the discovery of certain peptides secreted by adiposities that affecting energy balance. Certain new methods for the study of energy metabolism have led to the analysis of the role of factors responsible for the pathogenesis of obesity. The determinants and correlates of excess body weight or fat are depicted in Table 2.3.

Genetics and Environmental Factors

Childhood obesity has its origin in a variety of social factors most of which can be found in the family environment. Obesity is said to be inversely related to the number of family members. The birth order seems to be one of the predisposing factors and is said to have direct relation to obesity. The oldest and

TABLE 2.3

DETERMINANTS AND CORRELATES OF

EXCESS BODY WEIGHT OR FAT

Determinants	Correlation
Age	More prevalent in adults and middle-aged individuals
Gender	Females have more fat
Positive energy balance	An absolute requirement over a relatively long period
Amount of energy intake	Overfeeding leads to gain in weight and fat mass
Composition of intake	High fat intake may be a contributing factor
Physical activity level	Low or decreasing level of activity
Resting metabolic rate	A low value with respect to body mass and fat free mass is correlated with weight gain
Thermic effect of food	Low for energy intake in some obesity cases
Lipid oxidation	A high respiratory quotient is correlated with body fat and weight gain
Ratio of fat to lean tissue	A high fat mass to fat free mass ratio is correlated with excess weight or weight gain
Adipose tissue lipoprotein lipase activity	High in obese individuals and remains high (perhaps even increases) with weight loss
Variety of social and behavioural factors	Obesity is associated with socioeconomic status, familial conditions, network of friends, pattern of leisure activities, television time, smoking habits, alcohol intake etc.
Undetermined genetic characteristics	These affect energy balance particularly via the energy expenditure components, the deposition of the energy surplus as fat or as lean tissue, and the relative proportion of lipids and carbohydrates oxidised.

Source : Bouchard 1991

single children were reported to have higher prevalence of obesity. The genetic disposition and constitutional factors have great importance for the onset of some forms of obesities. The strength of the genetic component was said to be greater in older children. More prevalence of obesity was reported in girls than boys.

Some significant environmental factors like culture, diet, type of activity, education of the parents and socio-economic status of the family were observed to have profound influence in the causation of obesity (Okasha et al 2003, Anderson et al 1998, Errikson et al 2001 and Cavadini et al 2000). Obesity was also observed in some low income multi ethnic school children, which was attributed to their high consumption of carbohydrates.

The effect of urbanisation, enhanced amenities and availability of ready to eat instant food mixes added to sedentary life style in most of the high socio-economic families have been identified as causative factors. Several studies have shown a greater association of obesity with television viewing. Moreover, reduced energy expenditure, increased calorie intake and taking snacks while TV viewing was reported to be directly related to the prevalence of obesity. The more time a child spends on TV viewing, more the child is tempted to consume the calorie dense foods during TV viewing and also tempted to consume sugarated breakfast cereals, candy and other fast foods sold in food joints advertised on TV.

Economic growth and social changes in almost every country around the globe in the last few decades has also led to an increase in the prevalence of overweight and obesity. This unintended consequence is found in both children and adults alike (Kumanyika et al 2002).

Obesigenic environment

Various studies have identified numerous genetic factors influencing obesity susceptibility. However genetic factors per se cannot be held responsible for obesity. There has been no significant change in the human gene pool in the past century whereas secular obesity trends have increased at an alarming rate.

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In the US for example obesity prevalence has increased by 50% over the past decade such that 64% of adults have a BMI exceeding 25 and 31% of adult Americans have BMIs exceeding 30.

It has become evident that the rising rate of obesity is driven not by our genes but by our changing environment. The manners in which this environment encourages and rewards individuals to make their life style choices promote obesity (Peters et al 2002). Such an environment promotes a high level of food and energy consumption but a very low level of physical activity causing positive energy balance and weight gain. A number of risk factors and health consequences have been associated with increased level of body fatness in youth (Ball and Mccargar 2003). There are hardly any scientific evidence authenticating various obesiogenic environmental influences on individual behaviours and their relative contributions to obesity risk.

The etiology of obesity is multi factorial ; both genetic and environmental are clearly determinants. Childhood obesity has a complex etiology consequential of the combined effect of genes, environment, life style and their interactions. Omran (1971) who put forward the Theory of Nutrition Transition stated that the epidemiological transition moves from a pattern of high prevalence of infectious diseases and malnutrition to one in which chronic and degenerative diseases predominate. Popkin (1998) supported this theory and gave the stages in the nutrition transition and its health implications. The period after the industrial and second agricultural revolution was marked by a swing to a high fat, refined carbohydrate (CHO), low-fibre diet resulting in an increase in obesity, and other diet related non-communicable diseases. The complex relationship of various changes in agriculture, health and socio-economic factors contributed to obesity risk.

Biological factors

Obesity is associated with various demographic and behavioural factors. The three critical periods for development of obesity are prenatal period, the period of adiposity rebound and adolescence (Dietz 1994). An

in-utero nutritional exposure effect is that appetite regulation and adipocyte numbers are entrained during the period. Undernutrition during late in-utero exposure reduces adipocyte replication whereas overnutrition during late in-utero exposure may bring about adipocyte hyperplasia. Therefore early undernutrition may improve the regulation of food intake and predispose to later obesity.

Whitaker and Dietz (1998) stated that maternal obesity increases transfer of nutrients across the placenta. This induces permanent changes in appetite, neuro endocrine functioning and energy metabolism of the infant. Pettit et al (1983) observed that those infants born to diabetic mothers were found to be heavier as compared to that of non diabetic mothers. He stated that the prevalence of obesity among children of diabetic mothers appeared to be independent of the mother's obesity status at the time of child's birth.

Emma Rose (1999) observed that longer the duration the babies were breastfed exclusively, less were their chances of starting with school overweight or obesity. Those infants who were breastfed till the age of 3–5 months had more than one third less possibility of being obese by the age of 5 to 6 years, than babies given only formula milk. Those breastfed exclusively for 6 months were 43% less likely to be obese. BMI normally decreases till the age of 5 to 6 years. It then increases during adolescence. The age at which the BMI increases is termed as the adiposity rebound (Rolland et al 1984).

Rolland et al (1984 and 1987) and Siervogel et al (1991) reported that BMI and sub scapular skin fold thickness were significantly greater among children whose adiposity rebound began early (before 5.5 years age) compared to those whose adiposity rebound was average (6–6.5 years) or late (after 7 years). The causes of obesity are complex but genetics play a major role and amount for about 25%-40% of its etiology. With one of the parents being obese, there is 50% risk of the child being

obese. When both the parents are obese, the risk is 80%. Obese children themselves have an 85% risk of becoming obese adults (Silberstein 1997). Catherine et al (2001) reported that children who inherit a paternal polymorphism associated with altered expression of the insulin genes have an increased risk of developing early onset obesity.

Adolescence represents the final proposed critical period for the development of obesity. Both the risk of onset and persistence of obesity appear greater for females than males. A long term follow up study of adolescents suggest that approximately 30% of all obese adult women were obese early in their adolescence where as only 10% of obese adult males had onset of their obesity as teenagers (Braddon 1986). Kapil et al (2002) reported that the maximum prevalence of obesity was during pubertal period (10-12 years) of children on account of increase in adipose tissue and weight gain during their pubertal growth spurt.

Lenthe et al (1996) in the Amsterdam Growth and Health Study investigated the effects of rapid and slow biological maturation in adolescence on the development of obesity in subjects between 13 to 27 years of age. He reported that individuals who mature rapidly in adolescence seemed to have a long term consequence for obesity and hence this should be considered a risk indicator for the development of obesity. Whitaker et al (1997) carried out study on 854 subjects aged 1 to 17 years and reported that the probability of being obese as an young adult increased with the age of obese child and was higher at all ages for the group of very obese children. After 6 years of age, the probability of obesity in adulthood exceeded 50% for obese children as compared with about 10% for non obese children. In a pair matched case control study that included 460 obese Kuwaiti children from 20 different schools, Moussa et al (1999) reported that familiar and environmental factors are also associated with childhood obesity. The conditional logistic regression analysis showed that the family history of obesity, diabetes mellitus was significantly

associated with childhood obesity after adjusting for social and behavioural factors.

It has been speculated that increasing rates of diabetes during pregnancy may account for some proportional increase in childhood obesity. Sugarman (1989) reported that the prevalence of diabetes during pregnancy in Navajo women was about twice that in the general US population. The longitudinal studies of diabetes in the Pima community revealed that the offspring of women who had diabetes during pregnancy on an average were obese, had higher glucose concentration and had more cases of diabetes at an earlier age, than the offspring of women who developed diabetes after pregnancy or who remained non diabetic at follow up. The pre natal exposure to the diabetic intrauterine environment appears to have lasting effect on the anthromorphic and metabolic development of the offspring. It is postulated that a hyperglycemic intrauterine environment may cause fatal adaptations to an excess of fuels or nutrients supplied during gestation, thus mediating obesity (Ravussin et al 1994).

In many longitudinal studies of childhood obesity, parental obesity is a consistent demographic variable and identified as a risk factor in large number of subjects (Salbe et al 2002). In a study carried out by Savval et al (2004) on 357 young children of Caucasian origins in Cyprus to identify short term predictors of overweight in early adolescence, it was observed that parental obesity was one of the factors for obesity. Triglyceride and high density lipoprotein cholesterol levels have been proven predictors for overweight in early adolescents for the first time.

A study carried out on 1581 school children aged 7 to 15 years from national nutrition survey revealed that having parents especially mothers who were overweight/obesity may increase the risk of children being overweight/obesity (Wang et al 2002). Skinner et al (2004) from the longitudinal study on 70 white children showed that factors in early life are associated with children's BMI at 8 years of age. Hui et al (2003) carried out

a study to identify risk factors for overweight in Hongkong children aged 6-7 years. Logistic regression analysis showed that childhood overweight was significantly associated with parental obesity (BMI > 25 Asian reference) but not parental overweight (BMI- 23 to 25). Also birth weight > 3 kg was one of the risk factors for childhood overweight. It was also significantly associated with higher energy consumption and having a father who was a current smoker.

A recent study by Delany et al (2004) determined the relation between race, sex, tanner stage and energy expenditure in a two year follow up study in 114 African-American and White girls and boys aged 12.7 ± 0.1 year. He concluded that average total daily expenditure did not change over two years but Resting Metabolic Rate (RMR) increased significantly and activity related energy expenditure decreased significantly. The difference in trunk and limb lean mass of White and African-American children may explain some of the ethnic differences in energy expenditure. The decrease in physical activity over two years may contribute to the risk of obesity.

Ecological factors
Demographic changes
The shifts associated with demographic change are attributed to rapid urbanisation. Some of the reasons for this are considerable increase in number of persons residing in urban areas, urban agglomeration and the shift of poor towards slum in urban areas (Popkin 1998). An urban dwelling seems to improve growth patterns of the children reducing their percentage with low weight for age. An urban dwelling provides a more positive energy balance to the children giving them an opportunity to grow at a better rate than rural children.

A longitudinal study to evaluate the associations between BMI at 31 year of age and family social class during early childhood, maternal BMI, BMI at birth and age at menarche in Northern Finland on 2876 males and

3404 females, found that the mean BMI at birth was highest in offspring from the highest social class. However BMI was inversely related to social class at 1 year of age. BMI, WHR and the proportion of obese subjects were inversely related to social class at 31 year. The heavier the mother, the heavier the offspring from birth to 31 years. Linear regression analysis indicated that the most important predictors of BMI at 31 year of age were BMI at 14 years (Laitinen 2001).

Merlin et al (2002) carried out a study to assess the factors responsible for percent body fat on 618 Malaysian adolescent school children and observed that results of stepwise multiple regression analysis revealed that percent body fat among male adolescent can be predicted from demographic factors (age and ethnicity) and energy expenditure. For female adolescents ethnicity and energy expenditure were the only factors responsible for percent body fat. Storey et al (2003) reported that in the children of 6 to 11 years and 12 to 19 years, the statistically significant demographic predictors of BMI were age, race, gender and family income. They accounted for almost half the explained variance for both children and adolescence.

Life Style Related Factors

Environment strongly influences obesity. This includes life style behaviours such as what and how much a person eats as well as his level of physical activity. Physical inactivity along with other risk factors is a significant contributor to the global burden of the disease (Figure 2.6). Lack of physical activity along with excess energy intake causes obesity in children. Trost et al (2003) from a cross sectional study suggested that obese children in south Carolina spent less time in moderate and vigorous physical activity than their non obese counterparts. He concluded that a significant proportion of children might be at increased risk for further gain in adiposity because of low levels of physical activity during preschool days.

FIGURE 2.6

PHYSICAL ACTIVITY & DIETARY EFFECTS ON OBESITY AND
RELATED CHRONIC DISEASES
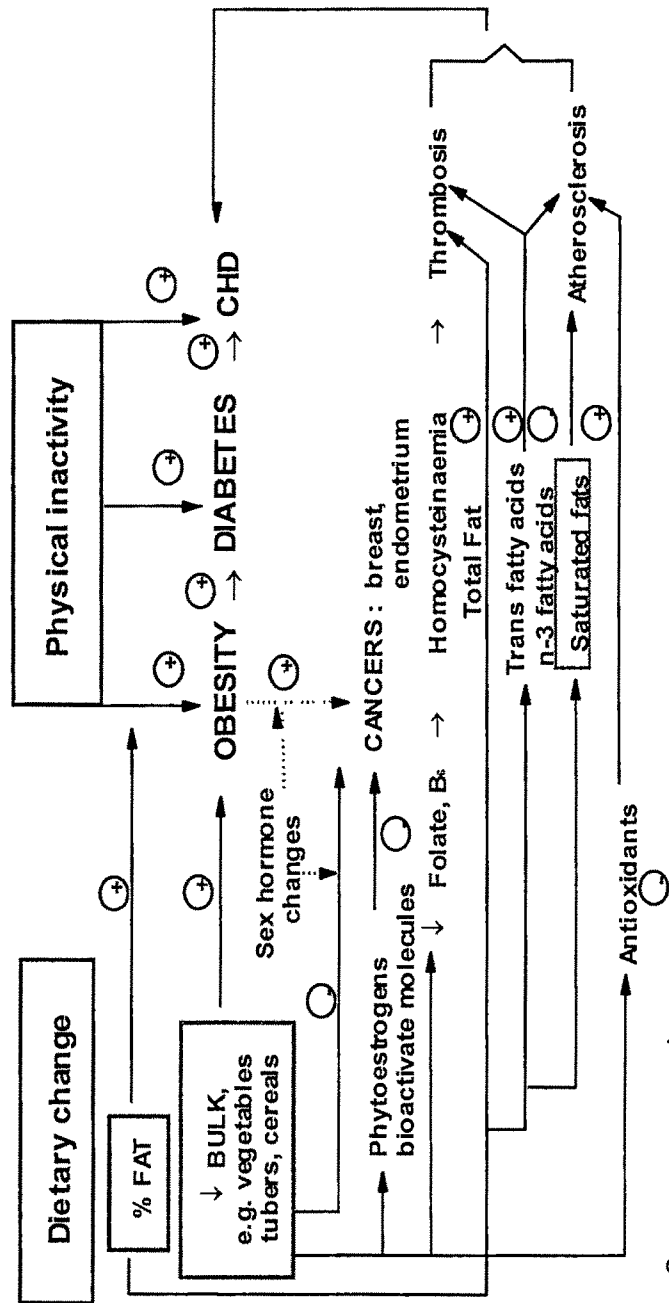
Source : www.google.com

A study by researchers at John Hopkins University School of Medicine along with experts of CDC and the National Institutes of Health concluded that a child's weight increases with number of hours he spends watching TV each day. Klesger et al (1993) reported that the metabolic rate declines during TV viewing. Taras et al (1989) suggested that TV viewing might promote intake of calorie dense foods. Goldman et al (1990) reported that girls, who usually consumed food while viewing TV, had higher energy intake than those who did not. The British and US children view approximately ten food commercials screened per hour of TV time, most for foods, soft drinks, sweets and sugar sweetened breakfast cereals (Kotz et al 1994, Lewis et al 1998 and Taras et al 1995) which increases the likelihood of 3-5 year old, in selecting an advertised food when presented with options (Robinson 2001).

The National Heart, Lung and Blood Institute Growth and Health study on 2379 Black and White girls aged 9 to 10 years established TV and video viewing as a strong marker for sedentary behaviour that strongly influences energy balance. The best statistical model for white girls included age, number of hours of TV and video watching and percent energy from total fat which indicated that body fatness is related to energy intake and expenditure in both Black and White girls (Obarzanek et al 1994). Shannon et al (1991) showed TV viewing as an explanatory variable for variations in BMI and triceps skin fold thickness in children from Pennsylvania (6th grade). A study by Storey et al (2003) on 6 to 19 year old children suggested that among the life style variables in both children and adolescent, television viewing was markedly associated with BMI and participation in team sports was negatively associated with BMI. Television viewing, diet and family income accounted for roughly half of the explained variance for both children and adolescence.

Kijboonchoo et al (2002) studied the physical performance and physical activity in Thai schoolboys aged 8 to 9 years. He concluded that the normal boys who were physically active performed better in health related fitness using fitnessgram than overweight and obese boys. Apparently a less active life style might contribute to a lowered level of physical fitness and increased blood pressure. Fontivieille et al (1993) assessed the physical activity in Pima Indians

and White children in Arizona. He found that the Pima children spent more time viewing television and had less involvement in sports than White children. Proctor et al (2003) in a longitudinal study examined the relation between television viewing and body fat change in 106 children from preschool to early adolescence (4 to 11 years). He reported that television viewing was an independent predictor of the change in children's BMI, triceps and sum of 5 skin folds throughout childhood. By eleven years of age children who viewed TV for 3 hours or more per day had a higher sums of skin fold compared to those who viewed less than 1.75 hours per day. Also the adverse effect of TV viewing was worse in those children who had also sedentary life style or had a higher fat diet.

Trembly and Williams (2003) in their epidemiological study examined the link between physical inactivity and obesity in Canadian children aged 7 to 11 years and concluded that TV viewing and video game were risk factors for being overweight (17% to 44% increased risk) or obesity (10% to 61% increased risk). Hernandez et al (1999) assessed the association between physical activity, TV viewing and other forms of video viewing with prevalence of obesity on 461 Mexican school children aged 9 to 16 years and concluded that physical activity and TV viewing but not video games were related to obesity prevalence. Gortmarker et al (1996) in the study on US children estimated that more than 60% of overweight could be attributed to excessive television viewing. Epstein et al (1995) also reported that if persons continuously ate while TV viewing, it might lead to a conditioned stimulus for eating. Gortmaker (1990) observed that television viewing has been associated with the onset of obesity, a decrease in the remission of obesity, a decrease in activity levels and may also possibly influence diet. Recent observations in children suggest increased television viewing and playing computer games, indicating less physical activity than in the past (Strong et al, 1992).

Various studies indicate a positive correlation between urbanisation and BMI of population and have pointed out that with the advancement of urbanisation, the BMI distribution curve of the population shifts to the right

(INCLEN 1996). Further studies indicate that there is a decrease in physical activities of the population at large including children, and an increase in sedentary lifestyle. Nowadays, hardly 5% of children walk or bicycle to school as compared to that of 80% some twenty years ago. The time allotted to physical education in school has also been reduced but the time for TV viewing has increased. Sriram (2001) reported that there is a 2% increase in the prevalence of obesity for each additional hour of TV viewing.

A cohort study on 115 White and Black children to consider the relationship between aerobic fitness and weight gain reported that the initial fat mass was the main predictor of increasing adiposity in children. A negative correlation was found between aerobic fitness and the rate of increase in adiposity. None of the measures of energy expenditure (resting, total and activity related) significantly predicted increasing adiposity in white and black children (Johnson et al 2000). Gutin et al (2002) determined the effects of physical training intensity on the cardiovascular fitness, percentage of body fat and visceral adipose tissue of 80 obese adolescent (13-16 years). He reported that cardiovascular fitness of obese adolescents was significantly improved by physical training, especially high intensity physical training. It also reduced both visceral and total body adiposity, but there was no clear effect of the intensity of physical training.

Dietary Factors

One of the most important causes of obesity is an increase in consumption of fast foods. It includes almost all the adverse dietary factors such as various saturated and trans fat, high glycemic index, high density, and an increasingly large portion size. The relation between fast food consumption and total energy intake and body weight in adolescents as well as adults has long been established. McNutt et al (1997) reported higher prevalence of obesity among girls (9-10 years) consuming fast foods four times or more a week due to consumption of 185-260 calories more per day than those who did not consume fast foods. Ludwig et al (1999 and 2002)

reported that high glycemic index foods like breads, cakes, biscuits, ready to eat cereals, and soft drinks brought about a series of hormonal events stimulating hunger and resultantly causing over eating in adolescents.

The compensation for calories consumed in a liquid form is less complete than for calories consumed in solid form. Hence sugar sweetened soft drinks promote energy intake and excessive weight gain. Harnack (1999) reported a 10% increase in total energy intake among children who consumed soft drinks than those who did not. Ludwing et al (2001) reported that the risk of developing obesity increases by 60% for every additional daily serving of any sugar sweetened drink. Zoumas et al (2001) reported that children consume more energy when they take meals in a restaurant as they serve larger portions of energy dense food.

Rolls et al (2000) reported that younger children (3.6 years) ate same amount of macroni and cheese irrespective of portion size where as older children (5 years) consumed more energy when given a large versus a small portion which suggests that with increase in age, children become less responsive to internal hunger and satiety and are more reactive to environmental stimuli.

All the above mentioned studies show a direct association with the raised economic status of people. Income is one of the important elements in this nutrition evolution as it controls the flow of goods and services. Popkin (1998) reported that income permits one to purchase goods or services affecting diet, activity and nutritional status.

Patton et al (1999) from the study of the predictors of new eating disorders in adolescents of age 14–15 years reported that dieting is one of the most important predictor of new eating disorder. The variation in the incidences of eating disorders between the sexes was accounted for by the high rates of earlier dieting and psychiatric morbidity in the female subjects. He concluded that adolescent females who diet at a severe level are 18 times more likely to develop an eating disorder than those who do

not diet, and those who diet at a moderate level are five times more likely to develop an eating disorder. A press release from NASSO in 2000 reported that frequent dieting among youths might increase future obesity risk.

Popkin et al (2000) studied adolescent food consumption trends in the US from 1965 to 1996 between age group of 11–18 years. He observed a considerable shift in adolescent diet. The total energy intake decreased, as did the proportion of energy from total fat (39% to 32%) and saturated fat (15% to 12%). There was a simultaneous increase in the consumption of higher fat potatoes and mixed dishes such as pizza, macaroni, and cheese. The low fat milk replaced high fat milk but the total milk consumption decreased by 30%. This decrease was accompanied by an increase in consumption of soft drinks and non citrus juices. Muluihill (2001) reported that obesity risk appears to jump 60% for every soft drink or sugar sweetened beverage consumed.

Ortega et al (1998) investigated the breakfast habits of 200 overweight or obese Spanish school children and compared them with those of normal weight aged 9–13 years. He observed that obese children especially girls omitted breakfast more frequently and had smaller amounts of grain products at breakfast. The breakfast habits of obese children were less satisfactory than normal weight children resulting in poorer food choices rest of the day and promoting obesity.

The findings from animal models indicate dietary fat as one of the potentially important component in the etiology of human obesity. The experiments carried out on animals have shown that the percentage of energy derived from fat in the diet is positively correlated with body fat with few exceptions. Obesity was induced by high fat diet in animals such as monkeys, dogs, pigs, hamsters, squirrels, rats and mice. (David and Barbara 1998).

Seidell (1998) reported that dietary fat has an effect on weight gain and development of obesity to an extent larger than is expected on the basis of energy value of fat and is mainly experimental. Prentice and Poppit (1996) opined that obesity resulted from an imbalance between energy intake and energy expenditure. The relative excessive energy intake results generally from an excess fat intake, as the diets high in fat are energy dense and also palatable.

Berkey et al (2000) examined the role of physical activity, inactivity and dietary pattern on annual weight changes on a cohort of 6149 girls and 4620 boys of US aged 9-14 year on the basis of growth and development. He reported that the larger increase in BMI from 1996 to 1997 were among those girls who reported higher calorie intake, less physical activity and spending more time viewing TV/Video. The BMI increased among those boys who spent more time viewing TV/Video. For both boys and girls, a larger rise in calorie intake from 1996 to 1997 predicted larger BMI increase per increase of 100 kcal/day. Some of the major food items whose wide use may contribute to obesity can be identified as butter, lard, whole milk, bread, meat and generous use of fats in the preparation of meals. In some groups, sweets and snacks may also contribute to increased energy intake.

The correlation between the diet of an adolescent and probability of chronic disease is predicted on the hypothesis that the eating habits are inculcated during childhood and adolescence that are than maintained in adulthood.

Gilbert et al (1992) reported that Navajo adolescents consumed sweetened soft drinks at more than twice the national average. Boston (2001) observed that for each additional daily serving of sugar sweetened soft drink the incidence of obesity significantly increased. The likelihood of becoming obese increased 1.6 times for each additional can or glass of sugar sweetened soft drink consumed above the daily average. Lytle (2002) reported that the weight gain was a result of positive energy balance or consumption of energy in a proportion greater than was required. The obesity in adolescents appears to be

affected by energy intake and expenditure. Between NHANES II and NHANES III, mean energy expenditure increased by approximately 100 to 300 kcal for adolescents.

Gazzanija and Burns (1993) observed a significant correlation between percent body fat and dietary fat in terms of percent energy intake in preadolescent children aged 9-11 year. Doucet et al (1998) carried out a study in 128 adult men to investigate relationship between dietary fat compositions and adiposity and confirmed the conception that the high fat diet might lead to excess body fat deposition over a period of time.

A cross sectional study carried out by Mc Gloin et al (2002) on 114 prepubertal children (5-8 years) of Northern Ireland revealed that energy intake was 98% of total energy intake (TEE) in low risk children, 95% in high risk children and 86% in obese children. Although energy intake was similar in each group, obese children consumed more fat in absolute and relative terms (% energy) than low risk children. There was a significant linear trend towards increasing fat intake (% energy) with increasing risk of obesity. Dietary fat intake (% energy) was weakly but significantly related to body fatness by stepwise regression. There was a significant trend for increasing fatness as fat intake increased.

Few studies have also been carried out on childhood obesity and its risk factors on urban school children of Vadodara.

Parikh (2002) observed that the heredity factor contributed significantly to prevalence of obesity. Forty two percent of the children in the age group 12-17 year were overweight if either or both the parents were obese. The type of diet did not show any influence on prevalence of overweight and obesity. However, the mean calorie intake of overweight and obese children was higher compared to their energy expenditure. The fat intake accounted for more than 30% of the energy intake for all the children. Atherogenic lipoprotein (LDL-C and TG) and atherogenic indices (TC/H and L/H) were also significantly elevated in overweight

and obese children. The TG and the LDL-C were significantly higher and HDL-C lower in subjects consuming > 60 g fat per day.

Akolkar (2003) observed that the heredity factor and the income of the parents had influenced prevalence of overweight and obesity among school children. The relative risk for the children in the age group of 6-12 year to be overweight or obese with either or both their parents being overweight or obese were 2.19 and 1.91 respectively. The type of diet did not have any influence on the prevalence of obesity. The mean calorie intake was higher in obese and overweight children as compared to the normal children. Fat intake accounted for about 33.9% of the energy intake for all the children. The lipid profile values and the atherogenic indices did not show any variation.

Mani et al (2004) from the study on school children in the age group 10-18 years reported that the per capita income of Rs. 5,000/- and above showed a higher prevalence of overweight and obesity. The type of diet did not show any influence on the prevalence. A considerably high intake of fat was observed in both overweight and obese children. The percent prevalence of overweight/obesity was found to be higher when the mothers were overweight/obese as compared to the fathers. Atherogenic lipids showed an appreciable increase in overweight and obese children.

Though the effects seem to be trivial, the collective effect during adolescence would create a considerable increase in body weight. Figure 2.7 shows the critical factors responsible for increase in obesity among Indian children.

Hormonal Factors – Role of Leptin in Obesity

The discovery of a fat melting hormone named 'Leptin' (Greek root – Leptose meaning thin) by Friedman led to the hope of a simple solution to cure the obesity epidemic (Caro et al 1996). Leptin - the protein product of the obesity genes ever since its discovery in 1994 has caused

FIGURE 2.7

FACTORS RESPONSIBLE FOR OBESITY AMONG INDIAN CHILDREN

Food or eating out
- Eating by teenagers
- Fast Foods
- Ice cream/sugar dense

Inappropriate recreation
- TV/Video
- Computer
- Internet surfing
- Telephone conversations
- Reading comics

Childhood Obesity in India

Sedentary life style
- Less working in high income group
- Very few household responsibilities
- Mode of transport to school
- Lac of traditional physical activities

School Environment
- Reduced physical activities
- No playground
- More class room activities
- Too competitive environment

Source : Mayuri K 2002

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considerable attention in the understanding of obesity and metabolic research. Leptin is synthesised and secreted by adipocytes and serum concentrations reflect the amount of energy stored in adipose tissue. The assimilation, storage and use of energy from nutrients constitute a homeostatic system that is essential for life.

The vertebrates owe their existence during the intervals of food scarcity encountered during evolution to their ability to store requisite energy dense triglyceride in their adipose tissues. Nevertheless the presence of excess adipose tissue can be maladaptive. Hence a complex physiological system has developed to regulate fuel stores and energy balance at an optimum level. Leptin is an integral component of this system. It also indicates nutritional status to numerous other physiological systems and also modulates their functions (Friedman and Halaas 1996).

The role of leptin in pathogenesis of the obesity can be inferred by measurement of plasma leptin (Figure 2.8). There are three general ways in which alterations of the leptin regulatory loop could lead to obesity.

- Failure to produce leptin as seen in ob/ob mice, would result in obesity.
- Inappropriately low leptin secretion for a given fat mass.
- From relative or absolute insensitivity to leptin at its site of action.

 Leptin binds to receptors in the hypothalamus and influences the expression of several neuropeptides that regulate energy intake, energy expenditure and neuroendocrine function. Leptin maintains energy homeostasis. When the body gains fat, the increase in leptin shifts energy balance towards the negative (Less energy intake but more expenditure). This means that all fat gains are followed by losses. However in reality it is not so. Though most of the obese people have high levels of leptin, their energy balance does not automatically shift to the negative. This suggests that there exists a resistance to leptin's action in obesity. When the body loses fat, the increase in leptin shifts energy balance towards the positive

FIGURE 2.8
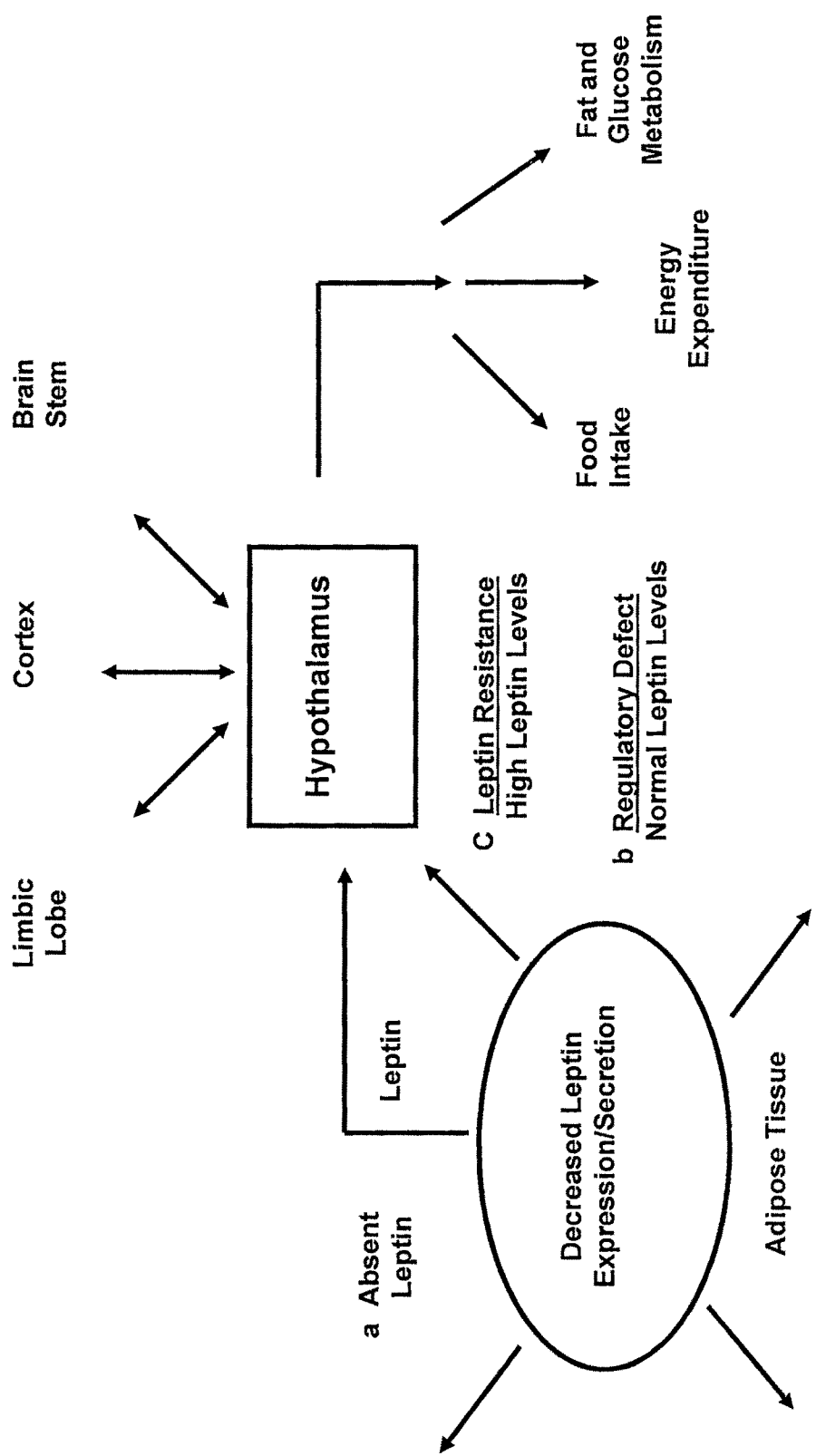
PATHOGENESIS OF OBESITY

Source : Considine et al 1996

(More energy intake but less expenditure). Figure 2.9 explains the leptin's action in the body.

Considine et al (1996) reported that though few extreme obese persons were leptin deficient, most of the obese persons had hyperleptinemia that was proportionate to their body fat and appeared to be leptin resistant. Serum leptin concentration reflects the amount of adipose tissue in the body.
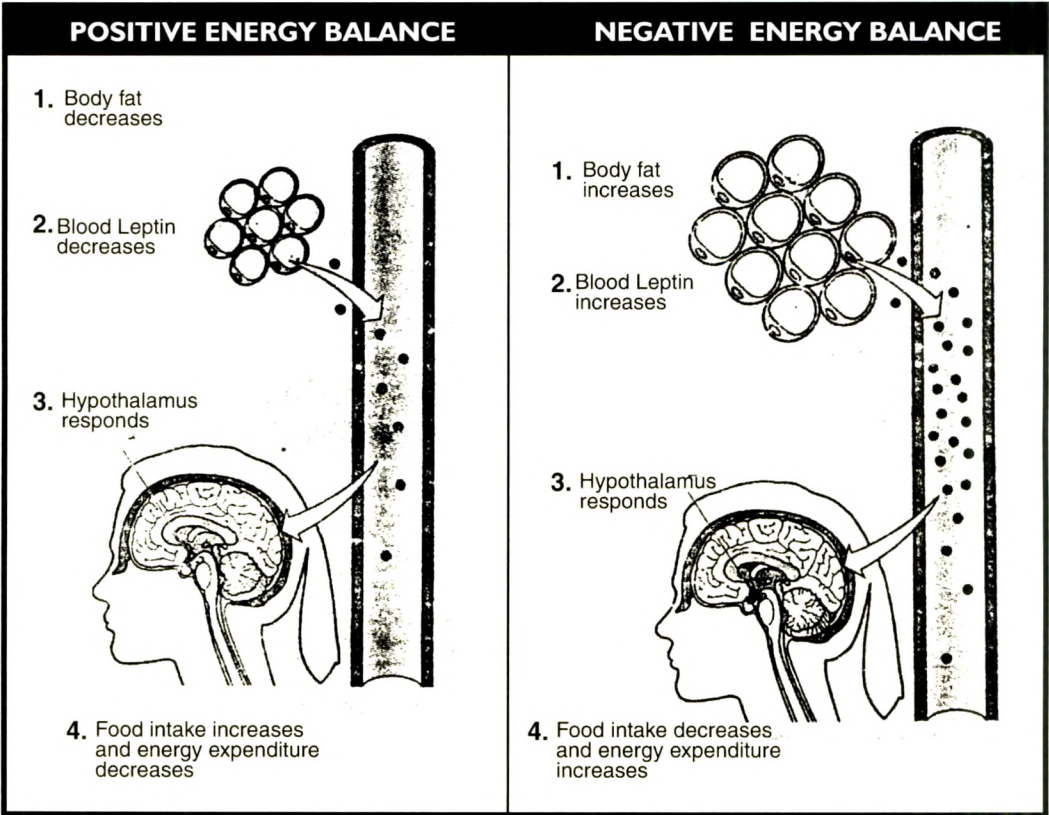
Banks et al (1996) reported that majority of obese subjects do not have a defect in the production of leptin. However, there may exist intravascular defects such as leptin antibodies, leptin antagonists, or increased production of leptin binding proteins to limit the concentration of free leptin that reaches the brain. Since the circulating leptin is a 1-4-6 amino acid protein, it will be excluded from the blood brain barrier and the blood cerebrospinal fluid barrier unless a transporter facilitates this. Ruhl and Everhart (2001) stated that the physiologic function of leptin is still not fully understood but it may be regarded as one of the determinants of obesity and its related consequences.

Little attention has been paid to leptin concentrations with anthropometric measurements relation in population based studies. Ruhl et al (2001) examined the leptin concentration in relation to the demographic and anthropometric measures on 3603 subjects of different ethnicity in US. On the basis of multivariate analysis, he reported that leptin concentrations were associated with the sum of 4 skin fold thickness, waist and hip circumference, ethnicity and age of the people. He concluded that the mean serum leptin concentration was much higher in women than in men.

Various studies have been conducted in other populations to examine the relation between leptin concentrations and the measures of body fat distribution. Haffner et al (1996) from a study on Mexican

FIGURE 2.9

LEPTIN'S ACTION IN THE BODY

POSITIVE ENERGY BALANCE	NEGATIVE ENERGY BALANCE
1. Body fat decreases	1. Body fat increases
2. Blood Leptin decreases	2. Blood Leptin increases
3. Hypothalamus responds	3. Hypothalamus responds
4. Food intake increases and energy expenditure decreases	4. Food intake decreases and energy expenditure increases

Source : Whitney and Cataldo 1983

Americans reported higher correlations of leptin concentrations with BMI and waist and hip circumferences and concluded that leptin concentrations were associated with overall adiposity and not with any specific fat deposition site. However, some other authors found an association of leptin concentration with waist circumference that was independent of BMI or percentage body fat, and they concluded that body fat distribution may also be an important determinant of leptin concentrations (Hanley et al 1997). Bennett et al (1997) reported that leptin concentrations were not related to waist circumference after adjustment for fat mass but were associated with hip circumference in women only.

Till today, there are very few studies available on the children with regard to leptin concentration. Lahlou et al (1997) reported that leptin concentrations were found to be higher in obese children than lean children. These high leptin concentrations should have reduced energy intake in such children. But it was observed that the obese children had high energy intake than the lean children, which suggested that they might have some form of leptin resistance. These results suggest that in children leptin is simply a marker of adiposity. It does not suppress energy intake or arrest fat deposition among them. The causes of leptin resistance can be attributed to either physiologic in nature or resulting from environmental factors such as easily accessible energy rich foods.

Gutin et al (1991) from the study on 34 obese children aged 7 to 11 years with respect to their physical training concluded that in obese children the leptin concentration decreased during the four months of physical training and increased during the subsequent 4 months without physical training. The fat mass was found to be highly correlated with baseline leptin. The reduction in leptin was greater among children having 4 months of physical training who had higher leptin levels before training and in those whose total mass increased least.

Caprio et al (1996) in a cross sectional study reported that leptin concentration was elevated in obese children as compared with other non obese, adolescent children and young adults which suggests that hyperleptinemia is an early indication of juvenile obesity. Salbe et al (2002) reported that leptin deficiency or disruption in the melanocortin pathway resulted in severe early onset of obesity among few children. Friedman and Halaas (1996) stated that various clinical trials are being carried out to identify possible therapeutic benefit of leptin treatment in humans. If leptin is introduced as one of the anti obesity drugs, the use of modern methods to identify and target the components of the leptin signaling path way will lay the foundation for a new pharmacological approach to the treatment of obesity and other nutritional disorders.

Highlights - Etiology

- Childhood obesity has a complex etiology, consequential of the combined effect of genes, environment, life style and their interactions.

- Parental obesity is a consistent demographic variable and identified as a risk factor in a large number of subjects.

- Family income is emerging as one of the contributing factors for childhood obesity.

- Inactivity is major factor in recent increases in the prevalence of overweight and obesity because it encourages weight gain and weight gain probably discourages activity, leading to an inactivity obesity cycle.

- Lack of physical activity along with excess energy intake causes obesity in children.

- In both children and adolescents, television viewing is markedly associated with increased BMI.

- Satiety signals relay information to the appestat about the amount and nature of food in the gut, the level of usage of glucose and the amount of fat stored in adipose tissue.

- The increase in consumption of fast foods is one of the important causes of obesity in children.

- Leptin - the protein product of obese gene was discovered in 1994 and has caused considerable attention in the understanding of obesity and metabolic research.

- The role of leptin in pathogenesis of obesity can be inferred by measurement of plasma leptin.

- The possible therapeutic benefit of leptin treatment in human is under clinical trials.

CONCERNS RELATED TO CHILDHOOD OBESITY

In a country where millions of people can't even manage to get one square meal per day, it is surprising that the problem of obesity among children has also to be discussed. The gross inequalities of socio-economic conditions among people have resulted in problems of both under privileged people and those of affluent societies simultaneously. Obesity is now considered not only as one of the appearance problems but also as one of the health hazards. Obesity as a risk factor for various health consequences has been highlighted in Table 2.4 (AOA 2003). The adult obesity has been recognised as a threat to life but the

TABLE 2.4

OBESITY AS A RISK FACTOR FOR VARIOUS HEALTH CONSEQUENCES FOR MEN AND WOMEN

Disease	Prevalence Ratio (%)							
	BMI 18.5-24.9		BMI 25.0-29.9		BMI 30.0-34.9		BMI $\geq$ 40	
	M	F	M	F	M	F	M	F
Type 2 Diabetes	2.03	2.38	4.93	7.12	10.10	7.24	10.65	19.89
CHD	8.84	6.87	9.60	11.13	16.01	12.56	13.97	19.22
High blood pressure	23.47	23.26	34.16	38.77	48.95	47.95	64.53	63.16
Osteoarthritis	2.59	5.22	4.55	8.51	4.66	9.94	10.04	17.19

Source : NHANES III

problem lies with childhood obesity. Hardly anybody recognise it as a threat to the child's health though it is one of the important predictor of adult obesity. Various studies have indicated that obese children are at risk for becoming obese adults also with the predisposition for all health hazards that accompany obesity, such as hypertension, diabetes, cardiac problems, arthritis, respiratory complications etc. (Fuentes et al 2002, Eriksson et al 2002 and Wright et al 2001). Statistics indicate that day by day more and more urban middle and higher socio-economic status children are prone to increased risk of obesity across the world. The vicious cycle of childhood obesity has been depicted in Figure 2.10. Some of the consequences of childhood obesity are enumerated below.

- Reduced work capacity
- Less School work
- Low Play activity
- Less house hold work
- Social problems
- Personality problems

Three critical periods have been identified for the development of obesity and its consequences namely gestation, early infancy, the period of adiposity rebound (5 to 7 years) and adolescence. Dietz (1994) reported that the mechanisms responsible for an increased risk associated with obesity still remain unclear. Braddon et al (1986) suggested from long term follow up studies of adiposity that nearly 30% of all obese adult women were obese early in adolescence where as 10% of obese adult males had onset of their obesity as teenagers. Must et al (1992) reported that overweight in children was related to morbidity and mortality rates in adulthood. Abram et al (1994) reported that the body weight of the children was an important determinant of adulthood overweight.

Guo et al (1994) suggested that larger BMI values were associated with increased morbidity and mortality in adulthood. Significant correlations between BMI values in childhood and in adulthood had been observed. The analysis of data for 555 White children indicated that overweight at 35 years could be

FIGURE 2.10

THE VICIOUS CYCLE OF CHILDHOOD OBESITY
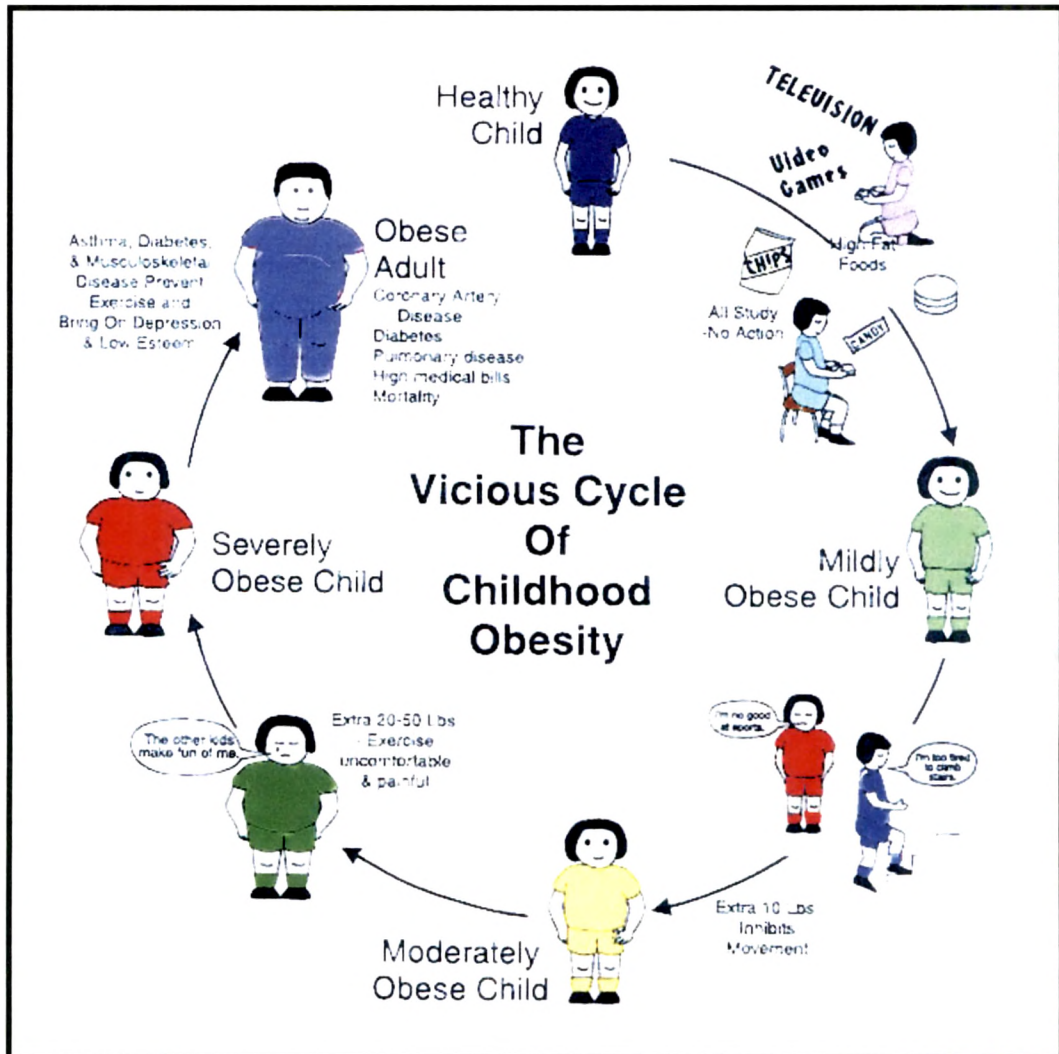
predicted from BMI at a younger age. The prediction was found to be excellent at 18 years of age, good at 13 but only moderate at ages younger than 13 years. For an 18 year old with a BMI value exceeding the 60th percentile, the odd of overweight at 35 years was 34% for men and 37% for women. The study used the predicted value of children BMI for overweight at 35 ± 7 years defined as BMI > 28 for men and > 26 for women. In 2002 he reported that a child or adolescent with high BMI percentile on CDC BMI for age growth charts had a high risk of being overweight or obese at 35 years of age and this risk increased with age. The probability of adult obesity at the 85th percentile for young males was < 20% upto 17 years of age and 20%-54% afterwards. The corresponding probability for young females was 20.0%-31.9% upto to 18 years of age and 40.0%-59.9% later. The logistic models were fitted to relate adult overweight and obesity in children and adolescent. The BMI values at each age for 166 males and 181 females in the Fels Longitudinal Study (Guo 2002) were applied to predict adult overweight and obesity at 75th, 85th and 95th percentile on the CDC charts for children.

Whitaker et al (1998) reported that an early Adiposity Rebound (AR) is associated with an increased risk of adult obesity that is independent of parent obesity and the BMI at adiposity rebound. The time of AR may be a critical period in children in development of obesity. An early AR is associated with higher BMI in adolescent and in early adulthood. Parasons et al (2001) from a longitudinal study in 1958 British cohort on 10683 subjects studied the BMI at 7, 11, 16, 23, and 33 years in relation to birth weight and other potential confounding factors and found that maternal weight (or BMI) chiefly explained the association between birth weight and adult BMI. Maternal weight might be a more important risk factor for obesity in the child rather than its birth weight. Intergenerational association between mother and offspring's BMI seem to underlie the well known association between birth weight and BMI.

Clarke and Lauer (1993) examined the childhood obesity track into adulthood on 2631 school children aged 9-18 years in Muscatine coronary risk factors project. He observed that nearly ½ to ⅔ of children were in the upper

quintile of weight. Their BMI were again in the upper quintile as adults where as ¼ to ½ of children in the upper quintile of triceps skin fold thickness were in the upper quintile as adults. Magarey et al (2003) from the longitudinal observational study on 155 Australian boys and girls born in 1975-1976 revealed that the prevalence of overweight and obesity increased with age and was higher than that reported in international reference population. BMI from 6th year age of a child is a good predictor of later BMI. The tracking of BMI was stronger for shorter intervals and for those children with both parents overweight compared with those with only one or either parent overweight.

Serdula et al (1993) from a review of literature (1970-1992) on tracking of childhood obesity into adulthood revealed that about 26%-41% of obese pre school children were obese as adults and about 42%-63% of obese school age children were obese as adults. It was found that for all studies and across all ages the risk of adult obesity was at least twice as high for obese children as for non obese children. A longitudinal study carried out by Skinner (2004) on 70 White children showed that children's BMI at 8 years was positively predicted by their BMI at age 2. Unger et al (1990) reviewed 175 growth charts of children aged 1-14 years and reported that the proportion of obese (% IBWH $\geq$ 120%) and severely obese (% IBWH $\geq$ 140%) increased between ages 1-7 years.

Wang et al (2002) examined the trend of obesity associated disease in youth (6-17 years) and their related economic costs and observed that the percent of discharge with obesity associated diseases had increased. Obesity associated annual hospital cost increased more that 3 fold during the period 1979 to 1991. Johnson et al (2002) from a retrospective study on 398 children aged 1 to 18 years observed that among obese and overweight children 10.8% had acanthosis nigricans, 5.8% had sleep apnoea and 5% had impaired glucose tolerance test. Reich et al (2003) investigated the relationship between different indices of body fat and blood pressure in 2365 children and adolescents aged 8 to 16 years. He observed a positive correlation between body fat and hypertension in children above 10 years of age. Among all the other indices of body fat the BMI had the strongest association with blood pressure. The

correlation between obesity and various health hazards such as non insulin dependent diabetes mellitus, cardiovascular diseases, hypertension and gall bladder diseases and certain types of cancer is well established.

Dyslipidemia

Obesity during adolescence is associated with a series of cardiovascular risk factors. Among sexually mature adolescents, changes in serum lipids and androgens seems to correlate more strongly with body fat distribution than with absolute weight. The obese adolescents with central obesity are more likely to manifest these cardiovascular risk factors than those individuals with peripheral obesity (Freemark 2001). Low HDL-C, increased LDL-C, and triglycerides have been observed in obese children. Gupta (1990) examined 237 children in Jaipur and determined the prevalence of risk factors. Borderline hypercholesterolemia (170–199 mg/dl) was observed in 32.9% children whereas definite hypercholesterolemia (> 200 mg/dl) was observed in 6.8% children. The overall prevalence of obesity was found to be 10.1%. Sriram (2001) reported that the Central adiposity was associated with increased VLDL-C and LDL-C synthesis on account of hyperinsulinemia. Grieger (1999) studied the association of location of body fat and CVD risk factors on 127 children aged 9–17 years and observed children with an apple shaped body to be having higher triglycerides and lower HDL-C and higher incidence of left ventricular mass, all of which indicated an increased risk of CVD.

Diabetes Mellitus/Glucose Intolerance

Obesity has been found to be a strong risk factor for an early onset of type 2 diabetes in childhood. From the data that is available about the frequency of glucose intolerance among obese children and adolescents, recent observation revealed that NIDDM accounted for one third of the new cases of diabetes in Cincinnati in 1994. Dietz (1998) reported that the prevalence of NIDDM among adolescents in Cincinnati seemed to have increased almost 10 fold since 1982.

Hypertension

It is found that persistent elevated blood pressure is said to occur 9 times more frequently in obese people as compared to the non obese people. A study carried out by Aligarh Muslim University in India, 292 (7.56%) of the 3861 school children in the age group 5–15 years were identified as obese. The mean blood pressure level in the obese children was found to be significantly higher as compared to non obese children. Nearly 3.4% of the obese children were found to have persistent hypertension compared to 0.16% of the non obese children. About 1/3rd of the hypertensive children were also found to have dyslipidemia. Dietz (1998) observed that hypertension seems to be an outcome of hyperinsulinemia. It produces a significant decrease in renal sodium retention in both obese and non obese adolescents.

Orthopedic Complications

Sriram (2001) reported that the 'bowing of tibia and overgrowth of the medial tibial metaphysis' also known as Blounts disease has been found to be more common in obese children. Also slipped capital femoral epiphysis is more often seen in 30%–50% of obese children.

Polycystic Ovary Syndrome

Dietz (1998) observed that obesity was frequently associated with polycystic ovary syndrome. The menstrual abnormalities are more likely to begin at adolescence. Among adult women who considered themselves normal and who had not undertaken treatment for menstrual irregularities, infertility or hirsutism, 14% had polycystic ovaries diagonised by Ultrasonography. Upto 30% of women with polycystic ovary syndrome are likely to be obese.

Respiratory Complications

Sriram (2000) observed sleep apnoea in 7% of obese children. The daytime somnolence, snoring and neurocognitive defects have also been reported in obese children.

Table 2.5 gives the prevalence of various complications of obesity in children.

Highlights - Concerns

- Obesity is not only one of the appearance problems but also has several consequences.

- Obese children are at greater risk for becoming obese adults.

- Childhood obesity is associated with various complications such as hypertension, diabetes and heart problem in adulthood.

METABOLIC ABERRATIONS IN CHILDHOOD OBESITY

Obesity is found to have a negative impact on lipid risk factors-particularly TG and HDL cholesterol. Various studies have shown a correlation between visceral fat accumulation and pathological changes and the lipoprotein profile particularly at the LDL level. TG is also positively correlated with visceral fat accumulation and TG has a strong impact on lipoprotein profile. The central obesity could influence lipoprotein metabolism directly or through the raised TG levels. Chan-Cua and Regidor (2002) determined the lipid levels of 75 obese Philippino children and adolescents and reported the occurrence of hyperlipidemia in obese children and adolescents. About 1/3 of the obese children had elevated serum cholesterol and TG.

Valle (2003) from a cross sectional study on 41 obese prepubertal children aged 6-9 years and 41 age and sex matched non obese children revealed that the serum leptin was significantly higher in obese children. In the obese group leptin showed a positive correlation with BMI, insulin, TG, plasminogen activator inhibitor 1 (PAI 1) and correlated negatively with sex hormone binding globulin, apolipoprotein A1, HDL cholesterol. The multivariate regression analysis showed

TABLE 2.5

HEALTH CONSEQUENCES OF CHILDHOOD OBESITY

Conditions	Prevalence
Hypothyroidism	1-2/1000 school children
Sleep apnoea	1/100 pediatric obesity
NIDDM	1/1400 children aged 10-19 years
Polycystic ovary syndrome	1-3/4 adolescent
Hypertension BP > 90th percentile	¼ obese children (5-11 years)
Dyslipidemias	1-2/5 children (TSF > 85th percentile)

Source : Barlow and Dietz 1998

that only insulin and BMI were independent predictive factors for leptin. Moussa et al (1999) from a pair matched case control study on 460 obese children in Kuwait stated that biochemical variables and blood pressure were adversely affected in obese children.

Asayama et al (1995) verified the anthropometric indices associated with the biochemical risk factors for atherosclerosis in Japanese obese elementary school children aged 6 to 12 years. He reported that in obese boys the body fat distribution was linked to certain biochemical complications of childhood obesity, and that androgyny in fat pattern causes metabolic aberrations in children. Invitti et al (2003) from a most recent study carried out on 710 grossly obese Italian children aged 6 to 18 years reported that in grossly obese children both insulin resistance and impaired insulin secretion contributed to the elevation of glycemia. The degree of obesity was related to cardiovascular risk factors independently of insulin resistance. He also observed that 25% of the boys and 29% of the girls had elevated value of systolic blood pressure and 15% showed elevated diastolic blood pressure. Anand et al (2002) from a study on 100 children (5-15 years) reported that mean serum TC, HDL-C and LDL-C was significantly higher (p < 0.05) in the children with parental history of ischaemic heart disease, hypertension and diabetes mellitus as compared to the controls.

Sinha et al (2002) determined the prevalence of impaired glucose tolerance in a multi ethnic cohort of 55 obese children (4-10 years) and 112 obese adolescents (11-18 years) in US. He reported that impaired glucose tolerance was detected in 25% children and 21% adolescents irrespective of ethnic group. Silent type 2 diabetes was identified in 4% of obese adolescents. The fasting lipid and lipoprotein profiles were similar in all groups except that fasting TG levels were higher among the adolescents with impaired glucose tolerance than among those with normal glucose tolerance. No difference in systolic and diastolic blood pressure was observed between children or adolescent with normal glucose tolerance and those with impaired glucose tolerance.

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│   Highlights – Metabolic aberrations                        │
│                                                             │
│                                                             │
│   •  Obesity has been found to have negative impact on lipid metabolism │
│      parameters like TG and HDL-C.                          │
│                                                             │
│                                                             │
│   •  Insulin resistance is seen in grossly obese children thereby │
│      predisposing them to early onset of diabetes mellitus and │
│      cardiovascular diseases.                               │
│                                                             │
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ASSESSMENT OF OBESITY IN CHILDREN

Evaluation of body fat and its distribution can be done by accurate measurements of body fat which requires special techniques (Table 2.6). However some of these techniques are often expensive and not useful for children. Careful measurement of height and weight is currently the initial step in the clinical assessment or in school setting to screen overweight/obese subjects, as it is cost effective, feasible and with high accuracy.

Common Indicators used for Assessment

The body fat and its distribution can be measured using the following indices.

- Weight/Height percentiles
- Waist/Hip ratio
- Body Mass Index
- Skin fold thickness

Weight/Height percentiles

The weight/height percentile tables are based on weight associated with the lowest mortality rate. These tables usually have a range of acceptable weights for a person of a given height. It helps to classify the

TABLE 2.6

TECHNIQUES FOR ESTIMATING BODY FAT

Technique	Cost*	Ease of use	Accuracy	Regional fat
Height and weight	$	Easy	High	No
Skin fold thickness	$	Easy	Low	Yes
Circumferences	$	Easy	Moderate	Yes
Density – water immersion	$$	Moderate	High	No
Petlysmograph	$$$	Difficult	High	No
H2O	$$	Moderate	High	No
D_2O or 18 OH_2	$$	Moderate	High	No
Potassium (40 K)	$$$	Difficult	High	No
Conductivity (TOBEC)	$$$	Moderate	Moderate	No
Impedance	$$	Easy	Moderate	No
Fat soluble gas	$$	Difficult	High	No
Neutron activation	$$$$	Difficult	High	No
Computed tomography	$$$$	Difficult	High	Yes
Ultra sound	$$$	Moderate	Moderate	Yes
Magnetic resonance	$$$$	Difficult	High	Yes
Dual photon absorptiometry	$$$	Moderate	High	No

* $ low cost
  $$ moderate cost
  $$$ high cost
  $$$$ very high cost

Source : Bray 1992

person into overweight/obese category and identify degree of risk involved. It also facilitates guidelines for treatment. However these tables have certain limitations (NIH consensus statement 1985). They do not distinguish excess fat from muscles, do not provide information on degree of obesity, are population specific, and does not take age into account.

A study conducted on well to do school children in Haryana in age group of 5-15 years reported that the weight of boys was more than girls up to 9 years, after which the girls overtook and weighed more than the boys till the age of 12 years. The boys were taller than girls upto 9 years after which due to adolescent growth spurt in girls they overtook boys. By the age of 13 years boys were again taller than girls (Bhasin et al 1990).

Waist/Hip ratio

Waist/Hip ratio identifies subjects with abdominal fat accumulation. It is a ratio of measurement of waist at its narrowest point and hips at the widest point. The normal ratio of adult women is < 0.85 and adult men < 1.0. Individuals with waist to hip ratio above these are said to be obese and are at increased health risk because of their fat distribution. However there are no set standards available for children. Boys and to a lesser extent girls appear to deposit fat centrally and lose fat peripherally as they mature (Mueller 1982). Amongst waist circumference, waist/hip ratio and BMI, waist circumference can be used as an independent measure to identify those at risk from either increased body weight or central fat distribution or both (Dasgupta and Hazra 1999). Waist circumference measurements are useful since height contributes little to the variance, accurately predicts obesity and high waist/hip ratio and predicts traditional coronary artery disease risk factors (Tivan et al 2001).

Body mass index

BMI is calculated by the formula BMI= Body Weight (kg)/Height (m)^2. It is a simple measurement and highly correlates with other estimates of fatness (NIN consensus statements 1985). It minimises the effect of height,

has an advantage of permitting comparison of populations, correlates with markers of secondary complications of obesity and can be calculated easily from height and weight of the subjects. However, It does not distinguish between weight associated with muscle and weight associated with fat.

Cole et al (2000) developed international cut off points for BMI for overweight and obesity by sex between 2-18 years, defined to pass through BMI of 25 and 30 kg/m^2 at an age of 18 years by averaging data from Brazil, Great Britain, Hong Kong, Netherlands, Singapore and US. Cole et al standards are recommended by International Obesity Task Force (IOTF) and are internationally used for classification of overweight and obesity in children. The Centres for Disease Control and Prevention (CDC) at Atlanta introduced the clinical use of BMI in new and revised BMI for age charts for young males and females aged 2 to 20 years using data from National Health Examination Survey cycle II and III, data from National Heart and Nutrition Examination Survey I and II and data for children for 2-6 years of age from NHANES III (Kuezmarski et al 2000). Agarwal et al (2003) assessed the anthropometric data on affluent school children from various parts of India and developed the percentiles for BMI, height, weight and skin fold thickness in relation to age and sexual maturity.

Skin fold thickness

Body weight and height provide information about total body mass and linear growth, while SFT has proved to be a useful indicator for body fat and hence of calorie reserves. Calipers like Harpender, Lange and USA MRNL calipers have been used to measure total body subcutaneous fat at the abdomen, triceps and subscapular subcoastal sites. Harpender Caliper is widely used to measure fat at the triceps. It measures skin fold thickness at the midpoint between the acromion and olecranon process on the posterior surface of the right arm. However measurements from such calipers varies with the user and needs different equations to transform data to body composition. A tricep skin fold thickness higher than 95th percentile provides evidence that child has excess fat rather than increased lean body mass. Thus skin fold

measures are more accurate determinants of fatness (Dietz 1983 and Lohman 1987). Bhasin et al (1990) conducted a study on well nourished children from well to do families in Haryana of age group 5-15 years and reported that children belonging to low income groups had smaller fat folds at all ages and SFT was uniformly higher in girls as compared to boys at all ages.

Defining Overweight and Obesity in Children

Anthropometry based on weight and height measurements is one of the commonly used methods of identifying obese people and can detect children at higher risk for nutritional and health diseases (WHO 1997). The likelihood of nutritional disorders can be diagnosed by comparing an anthropometric index with reference values (Robera et al 2001). The body mass index is widely accepted and used in adult anthropometry with highest and lowest limits of normality based on statistical criterion relating the higher mortality of people having higher or lower BMI than these values (Bellazi et al 1999).

The use of BMI for nutritional evaluation of children and adolescents has become more common after Must et al (1991) published the percentile values according to age and sex which are considered by WHO as reference values to classify adolescents as overweight and obese. Since then alternative percentile curves for children have been proposed by several authors (Table 2.7). The reference used and the type of obesity indicators used influence the prevalence of overweight/obesity rates. A rigorous scientific definition of obesity in children is yet not available. In fact, there is no agreement among researchers on the adiposity index to use and on the best cut off to define overweight and obesity in children (Luciano et al 2001 and 2003). WHO (1997) also pointed out that though BMI has often been used in nutritional evaluation there is no consensus about cut off values to classify children as overweight or obese.

Cole et al (2000) were the first authors to generate internationally applicable age and sex specific BMI cut off points for children and adolescents (2-18 years) related to adult BMI based on data sets from western population

TABLE 2.7

BMI PERCENTILES DEVELOPED BY VARIOUS AUTHORS

Author	Year	Sample Size	Age (y)	Calculated percentile
Hammer et al	1991	5679	1-19	5-10-25-50-75-90-95
Must et al	1991	20839	6-74	5-15-50-85-95
Cole et al[a]	1995	30535	0-23	0.4-2-9-25-50-75-91-98-99.6
Lindgren et al[a,b]	1995	3633	6-16[c]	3-10-25-50-75-90-97
Sichiere and Allamab	1996	11419	10-17	5-10-15-25-50-75-85-90-95
Luciano et al[a,b]	1997	41869	3-19	3-10-25-50-75-90-97
Rosner et al	1998	66772	5-17	5-15-50-75-85-95
Anjos et al	1998	16641	0-25	3-5-15-25-50-75-85-95-97
Cole et al[d]–IOTF	2000	192727	2-18	2-9-25-50-75-91-98
Kuczmarski et al–CDC	2000	11096	2-20	3-5-10-25-50-75-85-90-95-97
Agarwal et al	2003	19557	5-18	5-10-25-50-75-85-95

a - Studies recommended by WHO (1997)

b - percentile and Z scores

c - for males

d - percentiles related to BMI of 25 and 30 at 18 years of age

Source: Abrantes et al 2002

from six countries. However in puberty the sensitivity of their curves was affected (Agarwal et al 2001). In the year 2002 the Centre for Disease Control also developed the similar cut off values for 2-20 years of children and adolescents based on western population. The difficulty of establishing consensual criterion for superior limit of normality in children and adolescents has been limited by the lack of validity of BMI as a measure of obesity due to the absence of a worldwide reference population, disagreement about what criterion are used for the cut off points, and too few studies of sensitivity, specificity and predictive value of obesity persistence and its complications (Dietz et al 1998). Rosner et al (1998) have documented significant ethnic variation in BMI. James (2004) mentioned that health experts have suggested a lower BMI scale for Asians because of evidence showing their risk of obesity related diseases such as high blood pressure, abnormal cholesterol and development of diabetes risk if their BMI > 23. As the global standard for measuring overweight and obesity is based on western criterion the BMI cut off needs to be adjusted for Asians.

Therefore the need was felt to establish normal reference for BMI for diagnostic purpose of obesity and thinness for Indian children. Ethnic group specific standards are more appropriate for comparing health compromised children especially in country like India where the problem is more pronounced for undernutrition than overnutrition. There were no standards available for BMI and ponderal index percentiles for Indian affluent children on an acceptable number of subjects. So the data on affluent school children (5-18 years) of 23 public schools from 12 different cities in India was assessed and percentiles for BMI, height, weight and SFT were calculated in relation to age and sexual maturity also (Agarwal et al 2003). Khadgwat et al (1998) had evaluated the data and recommended it for use as reference for Indian school children. Table 2.8 provides the summary of various commonly used standards to classify overweight and obesity in children.

Various studies on children and adolescents have been carried out to establish the definition of childhood obesity as WHO (1995) supported the concept that the definition of obesity in children and adolescents should be

TABLE 2.8

SUMMARY OF COMMON STANDARDS TO CLASSIFY

OVERWEIGHT AND OBESITY IN CHILDREN

Standard	Age group	Sample size	Subjects	Criterion used to develop cut off points
Must et al 1991 (WHO)	6-74 years with 1 year interval	20839	US NHANES I	> 85th percentile of BMI for overweight > 95th percentile of BMI for obesity
Cole et al 2000 (IOTF)	2-18 years with 6 months interval	192727	Six large national survey on children from Brazil, Great Britain, Hong Kong, Netherlands, Singapore and US	Age and sex specific BMI cut offs related to adult cut off of BMI 25 for overweight and BMI 30 for obesity at the age of 18
CDC 2000	2-20 years with 6 months interval	11096	US NHESII, III and NHANES I, II, III	Age and sex specific BMI growth charts > 85th percentile of BMI for overweight > 95th percentile of BMI for obesity
Agarwal et al 2003	5-18 years with 1 year interval	19557	Private school children from 23 schools of 12 cities from India	> 85th percentile of BMI for overweight > 95th percentile of BMI for obesity

related to the adult definition 'as there may not be frank diseases during adolescence and it is particularly important to consider the degree to which adolescent anthropometry may predict risk factor of diseases in adulthood'.

Moussa et al (1994) mentioned from a study carried out on 220 obese and 220 non obese Saudi Arabian school children aged 7-18 years, that BMI was significantly related to blood pressure but WHR was not, indicating that WHR may not be a reliable indicator of body fat distribution in children. Gei et al (2001) found that in 838 pre pubescent children, weight to height indices such as BMI (overweight defined by 90th age and sex specific percentile) or ponderal index predict cardio vascular risk factors better than obesity parameters using skin fold measure. The BMI may be superior to the PI as the association between BMI and cardiovascular risk factors was less affected by gender. Daniels et al (2000) from a study on 201 children and adolescents (7-17 years) concluded that waist circumference was the best simple measure of fat distribution for children since it was least affected by gender, race and overall adiposity.

Florentino et al (2002) suggested that there appears to be a need to validate the anthropometric reference standards and cut off points in children and adolescents to define nutritional status better and ascertain the influence of ethnicity. Mei et al (2002) carried out a study to validate the performance of age and sex specific BMI compared with the Rohrer index and weight for height in screening for both undernutrition and overnutrition in children aged 2-19 years. The data from the National Health and Nutrition Examination Survey III and a pulled data set from 3 studies that used dual energy x-ray absorptiometry (n=920) were examined. It revealed that for children (2-19 years) BMI for age was significantly better than weight to height and Rohrer index for age in detecting overweight when average skin fold thickness was used as the standard. No differences were found in detecting overweight, when percent body fat or total fatness was used as the standard. BMI for age was significantly better than Rohrer index for age in detecting overweight in children aged 3-19 years.

Reich et al (2003) suggested that BMI should be the preferred index to assess body fat in children (8-16 years). The BMI had the strongest association with blood pressure among the other indicators of body fat used. Flegal (2000) in a study to compare the overweight prevalence for age grouping Vs month specific BMI percentile values concluded that it is important to understand how age is handled in the construction of the reference population and to select age categories consistent with these for the reference population. Maffeis et al (2001) in their cross sectional study to explore relationship between anthropometric measurements, lipid concentrations and blood pressure in a sample of 818 prepubescent (3-11 years) and to assess the clinical relevance of waist circumference in identifying the children with higher cardiovascular risk reported that waist circumference had a higher concordance with blood pressure than triceps and subscapular skin fold thickness and relative body weight. Multivariate linear model analysis showed that Apo A1/Apo B, HDL-C, TC/HDL and systolic as well as diastolic blood pressure were significantly associated with waist circumference. The children with waist circumference > 90th percentiles were more likely to have multiple risk factors than children with waist circumference < 90th percentile.

Chu and Savva (2000) also suggested that waist circumference had a consistent association with cardiovascular risk factors in children. Taylor et al (2000) found recently that waist circumference correctly identified high proportion of children and adolescents with high trunk fat mass as measured by a state of the art measurement. It was concluded that waist circumference is a simple technique that could be used to screen high central obesity in children. Pan et al (2004) suggested the need to set lower BMI cut off for Asians based on variations found between BMI and metabolic co-morbidities among Asians and US whites and blacks. Koon et al (2002) from the study on Malaysian school children (1426 boys and 1326 girls) compared the prevalence of overweight and obesity using various standards like WHO (1995), CDC (2002) and Cole et al (2000). It was mentioned that each of these reference standards varies in its assessments and there is a need for suitable indicators with acceptable cut off values to compare nutritional status among adolescents worldwide.

Kappa index of measurement

Abrantes et al (2002) reported good concordance between the values proposed by Cole et al (2000) and Must et al (1991) for defining overweight and obesity in children and adolescents (Table 2.9). He recommended that population studies of prevalence based on these references can be compared.

Highlights -Assessment

- Careful measurement of height and weight is currently the initial step in the clinical assessment or in school setting to screen overweight/obese children.

- A rigorous scientific definition of obesity in childhood is not yet available. However age and sex specific BMI cut off points related to adult obesity are recommended to identify overweight and obesity in children.

HIGHLIGHTS OF SOME IMPORTANT STUDIES

Magnitude of the Problem of Childhood Obesity

Child and Adolescent Trial for Cardiovascular Health (CATCH)

Aim : To determine the prevalence of marked overweight and obesity among children in CATCH study.

Duration : 1991 to 1994

Participants and Methods : A total number of 5106 school children aged 9 years enrolled for CATCH in 1991 were followed up for anthropometric measurement and skin fold thickness in 1994.

Highlights :The prevalence of obesity based on BMI and triceps skin fold among CATCH children increased from 9.1% to 11.0% in case of boys. It was higher among African Americans and Hispanics than whites for both the sexes.

Source : Johanna et al 2000

TABLE 2.9

COMPARISON OF NUTRITIONAL STATUS CLASSIFICATION ACCORDING TO COLE et al AND MUST et al STANDARDS FOR CHILDREN AND ADOLESCENTS

Must et al	Cole et al			Total
	Normal weight	Overweight	Obesity	
Normal weight	4921	92	0	5,013 (87%)
Overweight	57	403	2	462 (8%)
Obesity	0	78	183	261 (5%)
Total	4978 (87%)	573 (10%)	185 (3%)	5736

Kappa = 0.828 (0.804-0.848)

Nationally Representative Surveys from US, Brazil, China and Russia

Aim : To predict the trends of overweight and underweight in young persons aged 6-18 years using IOTF recommended Cole et al standards.

Duration : US 1971-1974 (NHANES I), 1988-1994 (NHANES III), Brazil 1975-1997, China 1991-1997 China Health and Nutrition Survey, Russia 1992-1998 Russian Longitudinal Monitoring Survey

Participants and Methods : The main variable studied was the subjects' height, weight, sex, residence and socio-economic profile. The body composition was estimated using BMI. Cole et al standards were used to classify children into overweight and obesity. According to Must et al standards children with BMI < 5th percentile were defined as underweight.

Highlights : The burden of nutrition problem is shifting from energy imbalance deficiency to excess among older children in US, Brazil and China. The prevalence figures for overweight and underweight is summarised below.

Country	Sample Size n	Overweight %	Underweight %	Annual rate of increase in overweight %
US 1971-1974 1988-1994	4472 6108	15.4 25.6	5.1 3.3	0.6
Brazil 1974 1997	56295 4875	4.1 13.9	14.8 8.6	0.5
China 1991 1997	3014 2688	6.4 7.7	14.4 13.1	0.2
Russia 1992 1998	6883 2182	15.6 9.0	6.9 8.1	-1.1

Source : Wang et al 2002

Wirral Health Authority Survey

Aim : To determine trends in weight, height, and body mass index in children

Duration : 1989-1998

Participation and Methods : The study population consisted of 35662 infants (1-3 months) and 28768 children (2.9-4.0 years). The height, weight and BMI of the children were examined over the years. The overweight and obesity was defined as BMI > 85th percentile and > 95th percentile respectively.

Highlights : There was significant increase in weight and BMI in children under 4 years of age. The prevalence of overweight in creased from 14.7% to 23.6% and that of obesity from 5.4% to 9.2 % in children.

Source : Bundred et al 2001

Bitterfeld Study

Aim : To analyse the trends in overweight and obesity in East German children.

Duration : 1992-1999

Participation and Methods : The data regarding parental education, living conditions, breast feeding and children's health was obtained by self administered questionnaire from three consecutive surveys performed in 1992-1993, 1995-1996 and 1998-1999 in East Germany. The total sample size was 6650 children including 5-7 year old school entrants, 8-10 years old third grade and 11-14 years sixth grade children.

Highlights : Overweight and obesity showed a significant increasing trend for 11-14 years and 8-10 years children but not for school entrants. After adjustment for age, sex, season and area, the risk of being overweight in 1998-1999 compared to 1992-1993 was 1.6 (CI 1.4-1.9) and that of obesity was 1.9 (CI 1.4-2.5). Low birth weight and higher parental education were protective factors for overweight and obesity. Breast feeding was protective with regard to obesity.

Source : Frye et al 2003

Australian Population Survey

Aim : To determine changes in the population prevalence of overweight and obesity among young Australians aged 7-15 years.

Duration : 1969-1997

Participation and Methods : The data from 5 independent population surveys (n=26449) were analysed for change in prevalence of overweight and obesity using Cole et al standards.

Highlights : The data showed that in 1985-1987, the prevalence of overweight and obesity combined doubled and that of obesity trebled among young Australians, but the increase over the previous 16 years was far smaller. For 1969-1985, there was no change in the prevalence of overweight and obesity among girls but among boys the prevalence of overweight increased by 35%, the prevalence of obesity trebled and the prevalence of overweight and obesity combined increased by 60%.

Source : Booth et al 2003

NHANES 1999-2000

Aim : To assess the change in the trends of overweight among children and adults surveyed in various NHANES surveys.

Duration : 1963-2000

Participation and Methods : The prevalence estimates for participants in the NHANES 1999-2000 were compared with estimates of those who had participated in earlier NHANES surveys.

Highlights : Fifteen percent of children and adolescents aged 6-19 years were overweight representing a 4% increase from the overweight estimates of 11% in NHANES III. The prevalence of overweight among children and adolescents aged 6-19 years for selected years 1963-1965 through 1999-2000 is summarised below.

Age (y)	% Prevalence of Overweight				
	1963-65, 1966-70	1971-74	1976-80	1988-94	1999-2000
6-11	4	4	7	11	15
12-19	5	6	5	11	15

Source : WWW.google.com

British Cross Sectional Surveys

Aim : To compare changes over time in waist circumference and BMI in British youth (11-16 years).

Duration : 1977-1997

Participants and Methods : BMI and WC measurements were taken at the interval of 10 years in case of British girls and 20 years in case of boys aged 11-16 years respectively. The total sample size at the final follow up was 776 for both the sexes. Overweight and obesity was defined as BMI > 91st percentile and 98th percentile respectively.

Highlights : Large increases in both the indices substantially more for waist circumference was observed. This suggests a steeper rise in abdominal obesity than whole body obesity based on weight and height.

Source : Mc Carthy et al 2003

Risk Factors Associated with Childhood Obesity

Glasgow Alumni Cohort

Aim : To explore the relation between childhood socioeconomic position and weight in early and later adulthood.

Duration : 1948-1968

Participants and Methods : The height and weight measurements of the 15322 subjects who attended university health service were taken initially in 1948-1950 and were followed up again during 1963–1966. The childhood social class was determined from the father's occupation. The likelihood ratio tests based on linear and logistic regression models were used to calculate p values for trend across social class.

Highlights : The childhood social origin may have a long term impact on obesity.

Source : Okasha et al 2003

1958 British Cohort : Longitudinal Study

Aim : To determine the influence of birth weight on body mass index at different stages of later life.

Duration : 1958-1991

Participants and Methods : Children (17414) born in England during 3rd to 9th March 1958 were followed up at ages 7, 11, 16, 23 and 33 years. At the time of final follow up the total sample size were 10683. The main outcome measures were maternal weight, birth weight of children and BMI of the children at ages 7, 11, 16, 23 and 33 years.

Highlights : Maternal weight or BMI largely explained the association between birth weight and adult BMI, and it may be more important risk factor than birth weight for obesity in the child. The birth weight and maternal weight seem to modify the effect of childhood linear growth on adult obesity in men. Inter generational association between the mother's and her off spring's BMI seem to underlie well documented association between birth weight and BMI.

Source : Parasons et al 2001

United States Department of Agriculture Surveys

Aim : To examine adolescent food consumption trends in US with important chronic diseases implications.

Duration : 1965-1996

Participants and Methods : The analysis of dietary intake data (24 hour dietary recall method) from 4 nationally representative United States Department of Agriculture Surveys (USDA) surveys of subjects (n=12448) aged 11-18 years was carried out.

Highlights : There was a considerable shift in the adolescents diet from 1965 to 1996. The total energy intake decreased, as did the proportion of energy from total fat (39% to 32%) and saturated fat (15% to 12%). Concurrent increases were noticed in the consumption of high fat potatoes and mixed dishes like pizza, macaroni and cheese. An increase in consumption of soft drink and non citrus juices was also reported.

Source : Cavadini et al 2000

Menarche Age And Obesity : The Bogalusa Heart Study

Aim : To study the relation of Menarche age and obesity in childhood and adulthood.

Duration : 1973-1996

Participants and Methods : In the Bogalusa heart study, the interrelationship of age at menarche and obesity among 1179 girls who were examined as child at the age 9, adolescent and adult (mean age 26 years) was studied.

Highlights : Both white and black women who reported that they underwent menarche before age 12 years had an average higher adult levels of weight (+ 10 kg), BMI (+ 4 kg/m^2) and SFT (+ 6 mm) than those women who underwent menarche at the age 13.5 years. However, relatively fat girls tended to undergo menarche earlier than thinner girls. Stratified and regression analysis indicated that adult obesity was strongly associated with childhood obesity than with menarche age. About 60% to 75% of the apparent effect of the menarche age was due to the influence of childhood obesity on both menarche age and adult obesity.

Source : Freedman et al 2003

National Health and Nutrition Examination Survey III

Aim : To assess participation of US children in activity level and TV viewing and their relationship to body weight and fatness.

Duration : 1988-1994

Participants and Methods : Episodes of weekly activity level and daily hours of TV viewed were examined for 4063 children aged 8-16 years enrolled for NHANES III.

Highlights : US children viewed great deal of TV and were inadequately active. Activity levels were lowest among girls, non-Hispanic blacks and Mexican Americans. Among US children intervention strategies to promote life long physical activity to stem the adverse health consequences of inactivity were advocated.

Source : Anderson et al 1998

The Northern Finland Birth Cohort for 1966 : Longitudinal Study

Aim : To evaluate the association between BMI at 31 years of age and family social class during early childhood, maternal BMI before pregnancy, BMI at birth and at 1 year and 14 years of age and age at menarche.

Duration : 1966-1999

Participants and Methods : Children born in 1966 in Finland's two northern most provinces were measured at birth, at 1 year, 14 year and 31 years of age. The final sample size at age 31 years was 6280.

Highlights : The mean BMI at birth was highest in children from the highest social classes. The BMI was inversely related to social class at age 1. BMI, waist to hip ratio and proportion of obese subjects were inversely related to social class at 31 years. BMI at 14 years was the most important predictor of BMI at 31 years. Low social class of the children's family, a high maternal BMI before pregnancy, a high BMI during adolescence and early menarche were predictors of obesity n adulthood.

Source : Laitinen et al 2001

National Heart Lung and Blood Institute Growth and Health Survey

Aim : To investigate the relationship between energy intake, physical activity and body fat in girls aged 9-10 years enrolled for NHLBI study.

Duration : Baseline of NHLBI study

Participants and Methods : Three field centres recruited 2379 girls and 3-day food records, 3-day physical activity diaries, physical activity pattern questionnaire and assessment of number of hours of TV and video viewing were obtained. BMI and skin fold measures at triceps, subscapular and super iliac sites were also obtained.

Highlights : Analysis showed that body fatness was related to energy intake and expenditure in both black and white girls. Longitudinal studies are required to assess the value of these variables in predicting changes in body fat.

Source : Obarzanek et al 1994

Concerns Related to Childhood Obesity

Childhood to Teenage Years : Finland Project

Aim : To investigate BMI during childhood and effect of birth weight and family history of obesity on BMI developed during childhood.

Duration : 1981-1997

Participants and Methods : All children born during 1981-1982 in a rural community of eastern Finland were followed up at ages 6 months, 7 and 15 years. Out of 205 children, 138 completed the full follow up period. The data on birth weight, family history of obesity and BMI at different time intervals were obtained.

Highlights : The study confirmed the tracking of BMI during childhood. Neither birth weight nor family history of obesity was found a good predictor of BMI during childhood. The risk of obesity in adolescence can be determined during middle childhood. Obese children should be targeted for life style advice to reverse this trend.

Source : Fuentes et al 2002

Helsinki University General Hospital Project

Aim : To examine the relation of obesity in adult life in relation to growth and living conditions during childhood.

Duration : 1934-2000

Participants and Methods : A total of 4515 people (2135 men and 2380 women) born at Helsinki university general hospital between 1934-1944 and who attended child welfare clinics and were still residents of Finland in the year 2000 were included for the study. The main explanatory measurements were size at birth, childhood growth and socio economic status in childhood and in adult life.

Highlights : The cumulative indices of obesity was 33.8% in men and 32.4% in women. Childhood BMI was a strong predictor of adult obesity than body size at birth. A higher maternal BMI in pregnancy was associated with a more rapid childhood growth and an increased risk of becoming obese in adult life. High socio-economic status and better education attainment were associated with a lower prevalence of obesity. There was no association between the duration of breast feeding and later obesity.

Source : Eriksoon et al 2003

The New Castle 1000 Family Prospective Survey

Aim : To determine whether childhood obesity increases adult obesity and risk of diseases.

Duration : 1947-1998

Participants and Methods : 932 members of 1000 families born in 1947 were followed up till their age of 50. The final follow up at the age 50 included 412 subjects and their blood pressure, carotid artery intima-media thickness, fibrinogen concentration, total LDL-C, HDL-C, TG, fasting insulin, 2 hours glucose concentration, BMI and percent body fat were measured.

Highlights : Body mass index at the age 9 was significantly correlated with BMI at age 50 ($r=0.24$ $p< 0.0001$) but not with percent body fat at age 50 ($r=0.10$ $p < 0.07$). Only those children who were obese at the age 13 showed an increased risk of obesity as adults. No excess adult health risk from childhood to teenage was found. The thin children had higher risk of being obese as adults.

Source : Wright et al 2001

Fels Longitudinal Study

Aim : To predict adult obesity at the age of 35 ± 5 years from the value of childhood BMI

Duration : 1929-1991 and 2002

Participants and Methods : The data on 277 males and 278 females participants born between 1929-1960 enrolled for Fels longitudinal study were analysed for prediction of adult overweight at the age 35 ± 5 years. In 1994 analysis, overweight was defined as BMI > 28 for men and > 26 for women. Whereas in 2002 analysis was carried out using CDC BMI charts to identify overweight or obese subjects.

Highlights : Analysis of data for 555 white children in 1994 indicated that overweight at the age 35 years could be predicted from BMI at younger age. The prediction was excellent at age 18 years, good at 13 years but only moderate at ages younger than 13 years. Analysis carried out on 166 males and 181 females in 2002 showed that a child or adolescent with a high BMI percentile on the CDC BMI for age growth charts had a high risk of being overweight or obese at the age 35 years which increased with the increase in age.

Source : Guo et al 1994 and 2002

The Bogalusa Heart Study

Aim : To understand the early natural history of coronary artery disease and essential hypertension.

Duration : 1972-2005

Participants and Methods : It is the only major program studying a well defined, biracial (black-white) population of children in a total semi-rural community. The community represents the southeastern United States. Over 160 sub studies have been conducted over the years. These sub studies include special studies on socioeconomic evaluations, blood pressure studies, lipids study, genetics studies, exercise, heart murmurs studies, newborn cohort, diabetes, and pathology, to mention just a few. Currently there is the post high school study, which carries the study of children up to the age of 38, as well as a precursor study of children with/without parental history of myocardial infarction. Blood samples have been sent to Boston, Johns Hopkins in Baltimore, Sweden and Finland for special analyses.

Highlights : More than 700 publications, three textbooks and numerous monographs have been produced which describe cross sectional and longitudinal observations on more than 14,000 children and young adults in Bogalusa, Louisiana. Results from the Bogalusa heart study give a clear picture of the early natural history of CV risk factors, early coronary artery disease, and essential hypertension in a total biracial population.

Some of the notable accomplishments of the Bogalusa heart study are

- The observations clearly show that the major etiologies of adult heart disease, atherosclerosis, coronary heart disease, and essential hypertension begin in childhood.
- Cardiovascular risk factors can be identified in early life. Methods to study cardiovascular risk factors are now developed, and normative values from a large biracial (black-white) population (approximately 10,000 individuals) are available for comparison.
- The levels of risk factors in childhood are different than those in the adult years. Levels change with growth phases, in the first year of life, during puberty and adolescence, and in the transition to young adulthood.

- Autopsy studies show lesions in the aorta, coronary vessels, and kidney, which relate strongly to clinical cardiovascular risk factors, clearly indicating atherosclerosis and hypertension begin early in life.
- Environmental factors are significant and influence dyslipidemia, hypertension, and obesity. Those that are controllable include diet, exercise, and cigarette smoking.
- Lifestyle and behaviours that influence cardiovascular risk are learned and began early in life. Healthy lifestyles should be adopted in childhood, because they are critical to modulation of risk factors later in life. Primary care physicians and pediatricians can play a major leadership role in the prevention of adult heart diseases beginning in childhood. Physicians are encouraged to obtain risk factor profiles on children, along with a family history of heart disease.

Source : www.obesity.org

Assessment of Obesity

National Health and Nutrition Examination Survey I

Aim : To generate race specific and population based 85th and 95th percentiles of BMI and triceps skin fold thickness for people aged 6 months to 74 years.

Duration : 1971-1974, 1991

Participants and Methods : Anthropometric data on 20839 subjects (6 months to 74 years) were analysed from NHANES I survey. The 5th, 15th, 50th, 85th, and 95th percentiles were calculated for each age, sex, and race by using frequency procedures.

Highlights : Race specific and population based cut off points using smoothed 85th and 95th percentile curves for BMI and TSF were generated for 6 months to 74 years for classification of overweight and obesity.

Source : Must et al 1991

Cole et al Standards

Aim : To develop an internationally acceptable definition of child overweight and obesity and age and sex specific points.

Participants and Methods : Data on body mass index 97876 males and 94851 females from birth to 25 years of age from 6 large nationally representative cross sectional growth studies from Brazil, Great Britain, Hong Kong, The Netherlands, Singapore and The United States were analysed. Centile curves for BMI were constructed using the LMS method.

Highlights : For each of the surveys centile curves were drawn at age 18 years which passed through the widely used cut off points of 25 kg/m^2 and 30 kg/m^2 for adult overweight and obesity. The resulting curves were averaged to provide age and sex specific cut off points from 2-18 years, which were less arbitrary and more internationally accepted to provide internationally comparable rates of overweight and obesity in children.

Source : Cole et al 2000

Centre for Disease Prevention and Control Standards

Aim : To develop age and sex specific BMI growth charts.

Participants and Methods : The data on BMI on 11096 children aged 2-20 years from NHES II and III and NHANES I, II and III were analysed.

Highlights : Age and sex specific BMI growth charts to classify overweight and obesity in children aged 2-20 years were developed.

Source : Kuczmarski et al 2000

Agarwal et al Standards

Aim : To develop growth standards based on BMI, ponderal index and skin fold thickness for affluent Indian school going adolescents.

Participants and Methods : Anthropometric measurements were recorded in healthy affluent school going adolescents from 23 public schools of 12 cities of India. The total sample size was 19557 school children (863 boys, 7694 girls). The means and percentiles of BMI, ponderal index and skin fold thickness was derived for each sex at yearly intervals, which was related to sexual maturity.

Highlights : BMI, PI and SFT were higher in girls. There was lower variability of these parameters with sexual maturity ratings as compared to age, suggesting use of these indices in relation to sexual maturity for assessment of thinness and obesity in adolescent Indian children

Source : Agarwal et al 2001
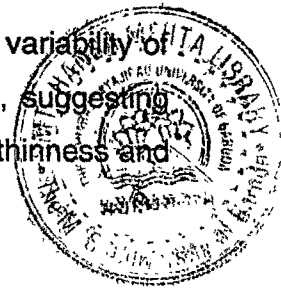
National Health and Nutrition Examination Survey III

Aim : To validate the performance of age and sex specific body mass index compared to Rohrer index and weight for height in screening of overweight and obesity in children aged 2-19 years.

Duration : Base line data NHANES III

Participants and Methods : The data from NHANES III on 11096 children and a pool data set from 3 studies that used DXA for 920 subjects were analysed.

Highlights : For children and adolescents aged 2-19 years, the performance of BMI for age is better than Rohrer index for age in prediction of underweight and overweight but is similar to that of weight for height.

Source : Mei et al 2002

Thus it is evident that pediatric obesity is of growing concern and efforts need to be developed to tackle/arrest this growing herd.