

## **Review Article**

### **Childhood Obesity: Issues of the Burden, its Genesis and Prevention**

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**Abstract:** A variety of mechanisms participate in weight regulation and the development of obesity in children, including genetics, developmental influences (“metabolic programming”, or epigenetics), and environmental factors. The relative importance of each of these mechanisms is the subject of ongoing research and probably varies considerably between individuals and populations. Obesity has a cascade negative health, with grave social and economic consequences. Obesity in children and young adolescents is increasing world-wide, including India, in parallel with economic development. Childhood obesity is now acknowledged as a growing epidemic which demands a preventive management. It is difficult to assess obesity in the young due to a lack of consensus as to how to measure it, especially severe obesity. The standardized prevalence is difficult to compare amongst different countries due to differences in study design. In the last decade several theories on genesis of childhood obesity have emerged. It is established that the young obese are at increased risk of cardiovascular events in addition to other diseases, and a higher risk of all cause mortality during adulthood. The management is multi-disciplinary and complex, but preventive strategies should be the priority. In all cases efforts to incorporate individualized family counselling is preferred. There is a need to increase the awareness among governments, health care professionals and the community that childhood obesity is a major health problem with substantial economic costs.

**Keywords:** Childhood obesity, Obesity in young, BMI, Measurement of obesity.

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#### **INTRODUCTION**

The term "overweight" implies an excess of body weight, whereas "obesity" refers to an excess of fat. It is estimated that at present there are more than 300 million clinically obese people world-wide [1] out of which 43 million are children. Obesity is increasing rapidly, often associated with a large number of debilitating and life-threatening disorders [2] and is no longer considered a “condition”. But it is still unclear as to how to define obesity in the young [3]. Weight exceeding 125% of the median weight for height is taken as obesity alone is not a good index as it does not consider height. Under and over-nutritious school children are almost of equal proportions in India; hence there is an underestimation of obesity among children measured by an Indian or an International growth chart [4]. Moreover, the methods to directly measure body fat are unavailable in daily practice. Therefore, obesity is often assessed by indirect estimates of body fat (ie, anthropometrics).

Development of a consensus measure of childhood obesity has been limited by the lack of data on the validity of BMI as a measure of adiposity, the absence of a reference population for the assessment of obesity

in different populations, lack of agreement on cutoff points; and there are only few studies examining the sensitivity, specificity, and predictive value of the persistence of obesity and development of its complications [5]. The Indian Council of Medical Research (ICMR) growth chart of 1956-65, based largely on children from lower socio-economic status, was dismissed as it underestimated the prevalence of underweight.

A recent workshop on childhood obesity, by the International Task Force on Obesity (ITFO), suggested another approach to establish cutoff points to identify degrees of overweight among children and adolescents. It proposed to use adult indices of overweight to establish pediatric percentiles to use as cutoff points to identify different degrees of overweight. It was decided that the pediatric percentiles identified in late adolescence by a BMI of 25 and a BMI of 30 should constitute the cutoff points for the identification of childhood overweight and constitutes the consensus definitions [6-8] for children between 2 to 20 years (Table 1).

However, these values are comparable with Indian cut-off datas until 11 years for boys and 9 years for girls (Table 2) after which the difference increases suggesting there are still limitations in application of IOTF chart [9]. Globally, obese girls outnumber boys [10], the mean BMI of girls being significantly higher after 10 years of age. 80% of overweight 10-14 year old adolescents are at risk of becoming overweight adults compared to 25% of overweight children < 5 years and

50% of 6-9 year old overweight children [11]. About 5% of children and adolescents have severe obesity and has significantly more cardiovascular risk factors and a greater risk for having obesity in adulthood [8]. Therefore, this threshold defines a group with clinically significant and severe obesity in children and adolescents and need tertiary care intervention with a multidisciplinary pediatric weight management team, including bariatric surgery.

**Table 1: IOTF consensus to measure obesity in children**

Degree of obesity & overweight	BMI in relation to percentile
<b>Underweight</b>	BMI <5 <sup>th</sup> percentile for age and sex
<b>Normal weight</b>	BMI between the 5 <sup>th</sup> and 85 <sup>th</sup> percentile for age and sex
<b>Overweight</b>	BMI between the 85 <sup>th</sup> and 95 <sup>th</sup> percentile for age and sex
<b>Obese</b>	BMI ≥95 <sup>th</sup> percentile for age and sex
<b>Severe obesity</b>	BMI ≥120% of the 95 <sup>th</sup> percentile values, or a BMI ≥35 kg/m <sup>2</sup> (whichever is lower). This corresponds to ~ 99 <sup>th</sup> percentile, or BMI Z-score ≥2.33 (2.33 SD above mean).

**Table 2: Comparison of 95<sup>th</sup> percentile of BMI among boys and girls between Indian standard and the IOTF standard**

Age (yrs)	Indian Standard		IOTF Standard	
	Boys	Girls	Boys	Girls
5	17.0	18.3	19.30	19.17
6	17.8	18.8	19.78	19.65
7	18.8	19.7	20.63	20.51
8	19.7	21.4	21.6	21.57
9	21.0	21.7	22.77	22.81
10	22.1	23.2	24.0	24.11
11	23.4	24.5	25.1	25.42
12	23.8	25.7	26.02	26.67
13	25.3	27.1	26.84	27.76
14	25.3	27.4	27.63	28.57
15	27.3	27.7	28.3	29.11
16	27.6	27.4	28.88	29.43
17	26.8	25.9	29.41	29.69
18	28.0	---	30	30

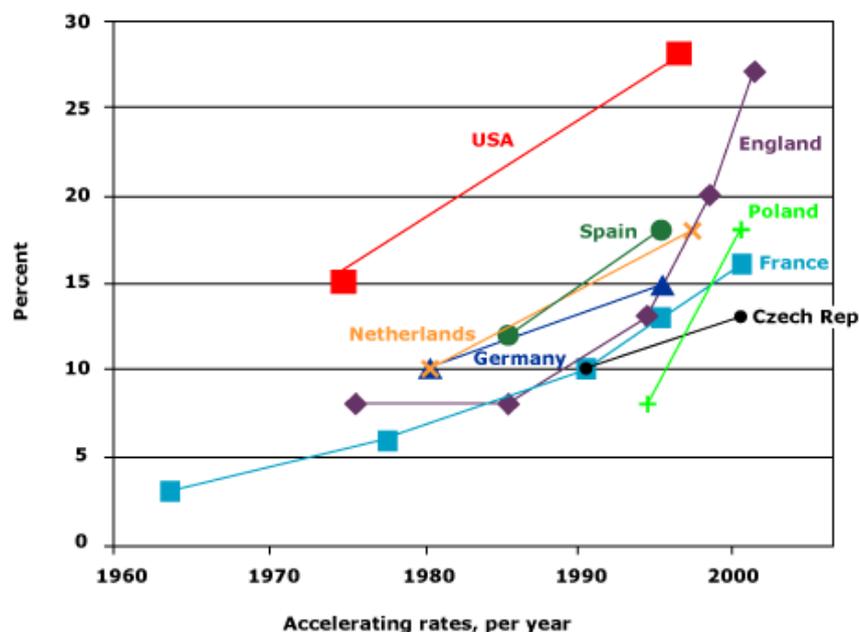
**GLOBAL BURDEN AND TRENDS OF CHILDHOOD OBESITY**

Although the prevalence of childhood obesity is high in developed countries, this is "unequally distributed"[12] The calculated global prevalence of overweight/obesity in children aged 5-17 years is estimated by the WHO and IOTF to be ~10% and will continue to rise as the number of severely overweight adults will be twice that of underweight during 1995-2025 [13]. It has been projected that by the 2030, levels of obesity will be 50-80% in the USA, 30-40% in Australia, England and Mauritius, and > 20% in some developing countries [14].

Since 1971, childhood obesity has been increasing consistently in developed countries (Fig 1) and recently also in developing countries. In Scandinavian countries the prevalence of childhood obesity is lower as compared to Mediterranean countries, but is rising in both regions. The prevalence is higher in the Middle East, Central and Eastern Europe. Iran is one of the seven countries with the highest prevalence. In a recent report from developed countries, prevalence of overweight youth (10-16 years) was > 15% in North America, Great Britain and some South Western European countries. The prevalence among school-aged children (6-11 years) and adolescents (12-19 years) in the United States dramatically increased between 1976-1980 and 2009-2010 (6.5-18.0% in children, and 5.0-

18.4% in adolescents). However, plateauing of prevalence of childhood obesity is reported in population studies from Australia [15] and France [16] and decreasing rates from Switzerland [17]. Increased

awareness, school-based programmes to increase physical activity and healthy nutrition may have helped to control this problem.



**Fig. 1: Rising prevalence of overweight (BMI > 85 percentile as per IOTF) children, aged 5 to 11 years**

Datas from developing countries is limited especially for children > 5 years, and adolescents. In Egypt 14% of adolescents and in Cyprus 25% of 6-11 year old children were reportedly overweight/obese. Obesity is increasing in countries that have acquired affluence in the recent times e.g. Taiwan and Saudi Arabia. In Saudi Arabia, one out of six 6-18 year old children is obese. The incidence of obesity across all ages is rising in the developing countries including India. India is undergoing a rapid epidemiological, nutritional transition and demographic transition, and is facing a “double threat” of under and over nutrition, particularly in urban settings. Obesity prevalence in Indian school children is between 5.74% and 8.82% [18]. According to a report from urban South India, 21.4 % of boys and 18.5% of girls aged 13-18 years were overweight/obese. Cross-sectional studies from various parts of India in the last decade among school children report the prevalence of overweight between 2.3%- 25.1% and of obesity between 0.3%-11.3%, more in affluent classes [19-34].

It is difficult to directly compare prevalence rates between countries because of differences in measurement definitions and datas. ITFO standards typically results in lower prevalence estimates than other standards [35, 36]. Urban poor in developed countries and urban rich in developing countries are both at risk. Comparable statistics show that rates are particularly high (> 30%) in most countries in North and South America, Great Britain, Greece, Italy, Malta, Portugal, and Spain [37]. In China, the prevalence of overweight children is  $\sim 1/3^{\text{rd}}$  of that in the US, but a

larger proportion of pre-school children are affected [38]. Prevalence is lower (0-15%) in the Nordic countries (Denmark, Finland, Norway, Sweden), and the central Western Europe. In Russia and Eastern Europe the prevalence of overweight is low (<10%), but is increasing. Overall, across the world, studies generally show a rising prevalence of obesity in children in the last 50 years. However, the obesity prevalence plateaued around 2000 and the percentage of children and adolescents in each weight category remained stable between 2000-2010 [39]. In developing countries, a number of beliefs passed down over generations are important determinants [40] where most physicians are unaware of or non-adherent to national recommendations regarding childhood obesity.

#### FACTORS INFLUENCING THE DEVELOPMENT OF OBESITY

Weight gain is the result of an imbalance between energy intake and energy output. The pathogenesis is multi-factorial and is interplay between genetic predisposition and environmental factors. The combination of our genetic propensity to store fat, ready availability of calorie-dense foods, and sedentary lifestyle promotes weight-gain. Childhood obesity results from a several factors which usually act in combination [41]. The child's food environment at home and parental obesity are strong determinants. Long- term follow up reveal that childhood obesity persists into adulthood, particularly for children with an obese parent. The severity of obesity during adolescence is another important predictor of adulthood obesity [5]. Whether gender affects the progression of

obesity into adulthood varies markedly in different studies. A vast majority (90%) of obese adolescents remain obese into their 30's (94% females and 88% males). Annual obesity incidence is highest among black females and lowest in Asian females and black and Hispanic females seem particularly at risk [42].

#### **“Obesogenic” social and environmental factors**

Changes in environmental and social factors are mostly responsible for doubling of severe childhood obesity over the last 30 years, because obese children are strongly influenced by environmental factors. While environmental factors explain only part of obesity risk, they are important targets for treatment as they are potentially modifiable. "Obesogenic environmental conditions" that promote overeating or sedentary life increases obesity. Consumers respond positively to changes in their environment. "Passive Over-Consumption" due to changes taking place in food marketing, consumer behavior and targeted marketing of high calorie-dense foods with low nutrients is increasing [43]. Several environmental factors that facilitate or limit physical activities have been identified. Urban housing design and land use influence the physical activity of the residents. Studies have shown that increased access to physical activity in an organized, structured and supervised manner is effectively beneficial for children [44]. Results of studies testing interventions to prevent childhood obesity are variable. It is difficult to draw definite conclusions from them as interventional measures vary in form and intensity, non-homogenous study population and selection bias, with other potential unmeasured confounders that could affect weight outcomes. It appears that effects of lifestyle interventions on BMI are small with substantial variation, and thus are not detectable without very large sample sizes.

Despite this, meta-analyses conclude that preventive interventions in childhood obesity are generally effective. One meta-analysis reported that children in intervention groups had a mean reduction in adiposity compared to control groups (mean difference in BMI of  $-0.15 \text{ kg/m}^2$  [95% CI  $-0.21$  to  $-0.09$ ]) [45]. While the effect size is small, this represents a clinically important difference across a large population. The best interventional strategies were school-based programs that enhance physical activity, nutrition education, quality of food served at school, and parent-targeted interventions to encourage children to be more active and reduce television viewing. In another meta-analysis, the strongest evidence was for school-based interventions emphasizing physical activity (with/without dietary intervention), but with a home component [46]. Studies involving younger children (6 - 12 years) showed most effectiveness. Because the intervention strategies and results varied widely among these studies, the effect of each intervention component is unclear.

#### **Diet**

The modulation of food intake from multiple meals is normally integrated to maintain a constant weight status, and defective integration produces obesity or leanness. An increase in both visceral and nonvisceral/subcutaneous fat requires that energy intake be increased over energy expenditure over an extended period of time. Over the last decades, food has become more affordable to larger population as the price of food has decreased substantially relative to income. The concept of 'food' has shifted from a means of nourishment to a marker of lifestyle and a source of pleasure. Clearly, increases in physical activity are not likely to offset an energy rich, poor nutritive diet. It takes between 1-2 hours of vigorous activity to counteract a single large-sized ( $\geq 780$  kcal) children's fast food. Frequent consumption of such a diet can hardly be offset by an average child [44].

#### **Calorie intake**

Total calorie intake is difficult to measure accurately at a population level. However, a small caloric imbalance over a long period of time is sufficient to lead to obesity. Surveys carried out during the past few decades in the UK suggested that average energy intakes in all age groups are lower than before [47]. Some small studies also found similar energy intake among obese children and their lean counterparts which suggests that physical inactivity is more important cause of obesity than increased calorie consumption [48]. Consumption of popular sugar-sweetened beverages (including fruit juice) too is an important contributor to the development of obesity. In a survey of children in the United States, sugar-sweetened beverages supplied an average of 270 kcal/day, representing 10 to 15% of total caloric intake [49].

#### **Fat & Protein intake**

Although excess fat intake leads to obesity, there is doubtful evidence that it is the chief reason for the ascending trend of childhood obesity. Many studies have demonstrated rise in the prevalence of obesity in spite of the decrease in mean dietary fat consumption in both sexes [50]. In fact the macronutrient composition of food only affects energy expenditure, but not fat storage. A randomized trial designed to determine whether the level of dietary protein affects body composition, weight gain, and energy expenditure, 28 individuals were randomly assigned to overfeeding with diets containing 5%, 15%, or 25% of energy from protein. Overconsumption of a diet with normal (15% of energy) or high (25%) compared with low (5%) protein increased resting energy expenditure and lean body mass. Although overall weight gain was less with a low protein diet ( $\sim 3$  versus 6 kg), body fat accumulation was similar and was related to excess calories consumed rather than the protein composition of the diet [51].

### Physical activity

The major component of energy expenditure is activity and exercise, called activity thermogenesis, and includes both exercise and nonexercise activity thermogenesis (NEAT). An often overlooked component is spontaneous physical activity (fidgeting), which can expend 100 to 800 kcal/day and, along with genetic factors, is responsible for much of the variance in energy expenditure among individuals which is not explained by differences in fat-free mass. It is hypothesized that a steady decline in physical activity among all age groups has heavily contributed to rising rates of obesity globally. Low participation rates in sports and physical education, particularly among adolescent girls, are also associated with increased obesity prevalence. In urban areas parents prefer having their children watch television at home rather than play outside unattended. Watching television and playing computer games are associated with increased prevalence of obesity [52]. In two longitudinal studies, television viewing at  $\geq 5$  years was independently associated with increased BMI at age 26 to 30 years [53, 54]. In fact, television viewing now may be the most important established environmental influence on the development of obesity during childhood by leading to decreased physical activity, depression of metabolic rate and adverse diet quality.

To sum up, increasing trends in glycemic index of foods, sugar-containing beverages, portion sizes for prepared foods, fast food services, diminishing family presence at meals, decreasing structured physical activity, increasing use of computer-oriented play activity, school meal nutrition content, and elements of the built environment (eg, availability of sidewalks and playgrounds) are all being considered as causal influences on the rise in obesity [55]. A number of well-designed studies have particularly shown associations between intake of sugar-containing beverages or low physical activity and obesity and metabolic abnormalities [56-58]. Though causal associations seem likely but are difficult to prove because of different study designs.

### Genetics

Polymorphisms in various genes controlling appetite and metabolism can predispose individuals to develop obesity, but are yet to be isolated. Five single-gene defects causing obesity in rodents have been identified with corresponding defects in several of these genes in humans. Obesity is a major feature of some rare genetic conditions that present in childhood [59]. Obesity is also a feature of at least 24 genetic disorders [60]. Thus, genetic contributions to obesity is strong, but the molecular mechanisms for them are yet to be determined. Childhood obesity is often the result of interplay between many genetic and environmental factors. A variety of specific syndromes and single-gene defects linked to obesity in childhood are known but

accounting for <1% of childhood obesity. *Prader-Willi syndrome* characterized by hyperphagia and food preoccupations leading to rapid weight gain, *Bardet-Biedl syndrome*, *MOMO syndrome*, *Leptin receptor mutations*, *Congenital leptin deficiency*, *Melanocortin receptor mutations* are some genetic disorders characterized by obesity [61, 62]. Out of children with early-onset obesity (onset <10 years and BMI > 3 SD above normal), 7% harbor a single locus mutation. Offsprings of two obese parents are likely to become obese in contrast to the offspring of two parents of normal weight. The percentage of obesity that can be attributed to genetics as a whole varies from 6% to 85% depending on the population examined [63] taking these factors into consideration. Interestingly, a large observational study in adults, the association between obesity and sugar-sweetened beverages and appeared to be mediated partly by genetic factors, quantified by a “genetic-predisposition” score, based on 32 BMI-associated loci [64].

### Home environment

Children's food choices are also influenced by content of family meals. Families with sedentary life style and non-vegetarian, calorie dense, high fat foods are more likely to have obese children [65].

### Developmental factors and the role of metabolic programming

There is increasing evidence that environmental and nutritional influences during critical periods in development before and after birth can have permanent effects on a child's predisposition to obesity and metabolic diseases. The precise mediators or mechanisms for these effects have not been established, and are the subject of ongoing investigations [66]. This is called “Metabolic Programming” which starts *in utero* and maternal nutrition or endocrine profile during gestation being probably important determinants. Prepregnancy weight and weight gain during pregnancy are important predictors of the child's birthweight, after accounting for genetic and other prenatal environmental factors [67] although a large longitudinal study involving 4654 parent-child pairs failed to demonstrate intergenerational acceleration mechanisms (maternal-child transmission) from maternal weight status during pregnancy [68]. The father-offspring and mother-offspring associations for BMI were found to be equally strong. Parental height and weight were self-reported during the pregnancy, and the child's BMI was measured at approximately 7.5 years of age. No effects of maternal obesity transmitted to the child through the intrauterine environment were detected. Thus, if metabolic programming is a mechanism for transmission of obesity, the effect is either minimal, mediators are more complex than maternal BMI or there may be other influencing factors. However, this study systematically under-estimated parental BMIs because they were self-reported.

Several population-based studies confirmed an association between birthweight (reflecting fetal nutrition) and later development of diabetes, heart disease, insulin resistance, and obesity [69]. Maternal preeclampsia, with or without prematurity, is associated with higher BMI in the offspring by adolescence [70]. Infancy and early childhood are also critical periods for metabolic programming. Studies in a variety of populations have shown consistent associations between rates of weight gain during infancy or early childhood and subsequent obesity or metabolic syndrome during early childhood adolescence, or adulthood [71, 72]. With evidences favouring metabolic programming, these observations suggest that early intervention might be an important tool in preventing obesity.

Various other developmental factors can also affect metabolic programming and obesity development. Babies fed on formula feeds are likely to have higher BMI as children and adolescents [73]. A child's body growth pattern may influence the tendency to gain weight. Fat babies at four months are 1.38 times more likely to be overweight at seven years old compared to normal weight babies. Fat babies at the age of one are 1.17 times more likely to be overweight at age seven compared to normal weight babies [74]. But majority, but not all, obese children will become obese adults.

The likelihood of persistence of childhood obesity into adulthood is related to age of obesity development [75]. Longitudinal studies reveal that a major component of adolescent obesity is established before five years of age. In a large study from the US, children who were overweight at entry into kindergarten were four times likely to become obese by 8th grade compared to normal-weight children [76] which supports the importance of early interventions to prevent obesity by efforts to optimize nutrition during gestation, infancy, and early childhood. Goals to optimize glycemic control in pregnant women and target moderate rates of weight gain in infants and young children are reasonable. Nutritional goals are less clear for low-birthweight infants, for whom catch-up growth is associated with improved neurodevelopment, but with future risks of metabolic diseases.

#### **Medical/Endocrine diseases**

Cushing's syndrome, hyperinsulinism, Hypothyroidism and craniopharyngioma with hypothalamic involvement are the established hormonal causes of obesity.

#### **Psychological factors**

A positive correlation between obesity and low self-esteem is established. Decreased self esteem led to 19% of obese children feeling sad, 48% feeling bored, and 21% feeling nervous. In comparison, 8% of normal weight children felt sad, 42% felt bored, and 12% felt

nervous. Stress and depression can influence a child's eating habits including overeating [77, 78].

#### **Virus**

Preliminary evidence suggests that obesity can be triggered /exacerbated by exposure to a virus like Adenovirus 36, as it increases body fat in several animal models [79]. Human studies, including a small study in twins and a multicentre study, have shown an association between adenovirus 36 antibodies and obesity status in adults as well as children and adolescents [80, 81]. Possible explanations include a true causal association, vulnerability to adenovirus infection or persistence among individuals with obesity, or the presence of unmeasured confounders but need further prospective validation.

#### **Gut microbiota**

Studies have also suggested a relationship between the normal intestinal bacteria and the propensity for weight gain [82]. The hypothesis is that certain combinations of gut bacteria have a greater potential for salvaging calories from the indigestible portion of the diet through fermentation. However, it is unlikely to be an important factor in the obesity epidemic.

#### **Drugs and Toxins**

A number of drugs can cause weight gain, including many psychoactive drugs, antiepileptic drugs, and glucocorticoids. Preliminary evidence suggests the possibility that obesity could be triggered or exacerbated by exposure to environmental endocrine disrupting chemicals, such as bisphenol A (BPA), a compound used to manufacture polycarbonate resin, which is a common contaminant of foods sold in cans and plastic packaging. Epidemiologic studies demonstrate an association between urinary BPA concentrations and obesity or obesity-related diseases, including diabetes and cardiovascular disease [83]. BPA is a selective modulator of estrogen receptors, and accelerates adipogenesis and postnatal somatic growth.

### **MEDICO-SOCIAL IMPLICATIONS OF CHILDHOOD OBESITY**

Obesity has unending negative health, social and economic consequences. Mortality and morbidity are higher among overweight and obese individuals than in lean people. The comorbidities of obesity in childhood and adolescence include abnormalities in the endocrine, cardiovascular, hepatic, gastrointestinal, pulmonary, orthopedic, neurologic, dermatologic, and psychosocial systems. The first problems to occur in obese children are usually emotional or psychological including low and negative self esteem. Obese children often suffer from teasing by their peers, some are harassed or discriminated against by their own family. Other problems include withdrawal from interaction with peers, depression, anxiety, and the feeling of chronic rejection which are often underestimated.

Certain comorbidities, such as type 2 diabetes mellitus and steatohepatitis that used to be considered "adult diseases" are now regularly seen in obese children. Moreover, obesity during adolescence increases the risk for disease and premature death during adulthood, independent of their obesity status after reaching adulthood. In a longitudinal study, overweight children had an increased risk of death from all causes in adulthood [84]. Males who had been overweight during childhood had an increased risk of death from ischemic heart disease while females showed an increased risk of breast carcinoma.

Atherosclerosis is documented more frequently among overweight adolescents. Childhood obesity is also associated with several markers of atherosclerosis like endothelial dysfunction, carotid intima-media thickening, development of early aortic and coronary arterial fatty streaks and fibrous plaques, and increased arterial stiffness. These observations lend additional support that atherosclerotic processes begin at an early age and are associated with obesity, inflammation, hypertension, and abnormal lipid profiles. They have their carotid arteries prematurely aged by as much as thirty years in addition to an abnormal level of cholesterol. A prospective study in over 35,000 young Israeli men found that BMI at 17 years of age was associated with angiography-proven coronary heart disease during adulthood, and the association persisted even after adjusting for adult BMI [85]. Overweight children have 2.4-4.5 times higher risk for developing hypertension and 2.4-8.0 fold rise in prevalence of dyslipidemia as adults aged 27-31 years, compared to children with normal BMI as well as type-2 diabetes. Obesity has been recently identified as a major independent risk factor for coronary heart disease (CHD) by the American Heart Association (1997). A predictive model estimated that the prevalence of CHD in the United States will increase 5 to 16 percent by 2035, with more than 100,000 excess cases of attributable to the increase in childhood obesity [86]. A population-based study demonstrated a significant effect of childhood BMI on the critical outcome of all cardiovascular events [87].

Metabolic syndrome, hyperinsulinemia and Type 2 diabetes has been documented among Asian adolescents at lower BMI compared to the Caucasians, probably due to higher body fat and greater central obesity. The long-term implications of the metabolic syndrome in children and adolescents are unknown. Modest weight reduction can significantly reduce the risk of these serious health conditions. In addition to the physical consequences on health, obesity creates a massive socio-economic burden and potentially it may overwhelm health systems world-wide [88]. Obesity has been described as the "last remaining socially acceptable form of prejudice" which seriously impedes the treatment of overweight and obese patients. Often overshadowed by the health and social consequences of

obesity is the economic cost to society and to the individual. In several developed countries obesity has been estimated to account for 2-7% of the total health care costs. The deleterious effects of nutrient deficiencies are rapidly replaced by the unhealthy patterns of excess nutrition in present times in developing countries like India, and are at crossroads facing the burden of both these extremes [89]. Costs in terms of impaired school performance are also adding to the direct costs of obesity. The logical way to combat the obesity epidemic is to lessen its burden in children by early intervention, not only for the individual, but for the country as well.

#### MANAGEMENT AND TREATMENT PLAN

Because of widespread cultural bias, many families with obesity are sensitive about discussing the issue. The clinician has a duty in educating the family about the health risks associated with obesity and importance of early intervention. It is important for health providers to understand the role of genetics and epigenetics in the development of obesity, even though their assessment and intervention must give priority to modifiable environmental factors. Family involvement and counselling is important alongwith a) behavioural therapy for healthy behaviors rather than weight goals and b) stimulus control to reduce environmental cues which encourage unhealthy behaviors. Self-monitoring of target behaviors (logs of food, activity, or other behaviors, recorded by patient/family) can help to recognize which behaviors may be contributing to their weight gain. Clinician feedback throughout the self-monitoring process is essential. A patient's food log may also identify other contributors to eating behaviors, such as meal-time environment, boredom, and level of hunger, all of which can be valuable in the evaluation of stimulus control. Activity and food habits should be assessed. Counseling to improve physical activity should focus on reducing sedentary activities and increasing physical activity. Few clinical trials have evaluated structured dietary interventions for children with obesity, and even fewer have attempted to control for complex nutritional behaviors and the family/home environment. Therefore, attempt should be made to focus on changing the eating behaviour rather than prescribing a structured diet.

*Clinical Assessment (Body fat estimation and Risk assessment):*

- Direct methods: Dual-energy X-ray absorptiometry (DXA).
- Indirect methods: Anthropometry (Weight, Height, BMI, Waist circumference, Waist-Hip ratio, Skin fold thickness),
- Clinical parameters: Blood pressure,
- Biochemical parameters: Lipid profile, Lipoprotein, Apo-lipoprotein; Blood glucose, HbA1C, Insulin levels; FFA levels, hepatic transaminases. The triglycerides, HDL, fasting hyperglycemia and hypertension do not seem to have association with metabolic syndrome in children unlike in adults. Hence, it is practical to redefine the diagnostic parameters of metabolic syndrome, in children with population-based values of these biochemical

parameters [35]. Central adiposity has increased to a higher degree than general adiposity in children and adolescents in recent decades. Waist circumference (WC) in adults is correlated with the volume of metabolically active abdominal fat and is associated with cardio-metabolic risk factors and adverse outcomes, WC can not be a routine measurement in clinical practice as it is found to be a poor indicator of obesity in children [90].

Obesity demands a preventive management. Single most important way to control the onset and development of obesity is by limiting the dietary intake. In June 1997 the WHO, alongwith IOTF, held an expert consultation on obesity, that resulted in the publication of an interim report: "Obesity - preventing and managing the global epidemic" (WHO 1998) and the subsequent WHO Technical Report Series 894 [91]. IOTF was established in May 1996 to tackle the emerging global epidemic of obesity.

The IOTF initiative on the prevention and management of obesity has four main goals [91]:

- To increase the awareness among governments, health care professionals and the community that childhood obesity is a serious medical condition and a major health problem with substantial economic costs.
- To provide evidence and guidance for the development of better prevention and management strategies.
- To secure the commitment of policy makers to action.
- To foster the development of national, regional and international structures to enable and support the implementation of action on overweight and obesity.

The IOTF aims to achieve action on the prevention and management of overweight and obesity and endeavours to create an environment that encourages and supports the development of appropriate public and health policies and programmes for prevention and management of obesity. Their principal strategy is prevention through changing the environment to make it less 'obesogenic'. The focus is on how to tackle childhood obesity [91].

#### Pharmacotherapy

No medications are currently approved for the treatment of obesity in children. Orlistat and sibutramine may however be helpful in managing moderate obesity in adolescence. Sibutramine, an anorectic, is approved for adolescents older than 16. Orlistat is approved for adolescents >12 years. It prevents fat absorption in the intestines. Adolescents with BMI>40 with associated complications are to be considered for bariatric surgery.

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