

Review Article

ADOLESCENT OBESITY: A SILENT EPIDEMIC

***Ravi Rohilla¹, Jyoti Rohilla², Madhur Verma¹**

¹Department of Community Medicine, Pt. B. D. Sharma PGIMS, Rohtak (Haryana)

²Department of Anatomy, Pt. B. D. Sharma PGIMS, Rohtak (Haryana)

**Author for Correspondence*

ABSTRACT

The World Health Organization (WHO) describes overweight and obesity as one of today's most important public health problem, which is escalating as a global epidemic. Obesity is associated with the onset of major chronic diseases leading to complications and also psychosocial problems in children and adults. The greater concern is that the risks of obesity during childhood will persist into adolescence and adulthood. Tackling the problems of the growing numbers of overweight individuals is a major challenge for most countries. The fundamental cause of childhood overweight and obesity is an energy imbalance between calories consumed and calories expended. The problem of obesity demands a population-based multisectoral, multi-disciplinary, and culturally relevant approach which can prevent from the huge expenses involved treating this epidemic.

Key Words: Obesity, Overweight, Adolescence, Nutrition, Body Mass Index, Life Style

INTRODUCTION

The World Health Organization (WHO) describes overweight and obesity as one of today's most important public health problem, which is escalating as a global epidemic (WHO, 2003). It is also increasingly recognized as a significant problem in developing countries and countries undergoing economic transition (Popkin, 2001). The problem of overweight and obesity is confined not only to adults but also being reported among the children and adolescents of developed as well as developing countries. The World is being home to 1200 million adolescents and 90% of them live in developing countries. These young people constitute 18% and 20% of the population in the World and India respectively (The State of the World's Children Report, 2011).

Traditionally, adolescence is defined as the period from the onset of puberty to the termination of physical growth and attainment of final adult height and characteristics. Adolescence is considered as a period of transition from childhood to adulthood. WHO has defined adolescence as 'a period of life from 10-19 years (The State of the World's Children Report, 2011). Healthy development of adolescents is dependent on several complex factors: their socioeconomic circumstances, the quality of relationships with their families, communities and peer groups and the opportunities for education and employment. Thus this is a period of challenges and opportunities.

During this crucial period, food intake patterns and dietary patterns are set in place and these patterns can have vital impact on lifetime nutritional status and health of the individuals. In the longer term, food consumed in childhood, particularly adolescence can set the pattern for future food preferences and eating behaviour in adult life. Young people's eating behaviour determines adult practice and thus strategies to promote healthy eating at this stage will have long term health benefits (Story *et al.*, 2002).

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Prevalence of Adolescent Obesity

Global trends of childhood obesity show huge shifts in recent times. Surveys from 144 countries (in 2010) suggest that 43 million preschool children (35 million in developing countries) are overweight and obese and 92 million are at risk of overweight. The worldwide prevalence of childhood overweight and obesity increased from 4.2% in 1990 to 6.7% in 2010. This trend is likely to continue and the prevalence is

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expected to reach 9.1%, or 60 million, in 2020. The estimated prevalence of childhood overweight and obesity in Africa in 2010 was 8.5% and is expected to reach 12.7% in 2020. The prevalence is lower in Asia (4.9% in 2010) than in Africa, but the number of affected children (18 million) is higher in Asia (De Onis and Blossner, 2000).

Asian countries are not immune to this phenomenon. For example, in China, the prevalence of overweight and obesity among children aged 7-9 year increased from 1-2 per cent in 1985 to 17 per cent among girls and 25 per cent among boys in 2000 (Wang *et al.*, 2005). Indian data regarding current trends in childhood obesity are emerging. A recent study conducted among 24,000 school children in south India showed that the proportion of overweight children increased from 4.94 per cent of the total students in 2003 to 6.57 per cent in 2005 demonstrating the time trend of this rapidly growing epidemic (Raj *et al.*, 2007).

Khadilkar *et al.*, (2010) conducted a multi-centric study in eleven affluent urban schools from five geographical zones of India recruiting 20,243 children in the age group of 2–17 years. Height and weight were measured and Body Mass Index (BMI) was calculated (kg/m²). International Obesity Task Force (IOTF) and WHO cut-offs were used to calculate the percentage prevalence of overweight and obesity. The overall prevalence of overweight and obesity was 18.2% by the IOTF classification and 23.9% by the WHO standards. The prevalence of overweight and obesity was higher in boys than girls (Khadilkar *et al.*, 2010).

Kaur *et al.*, (2012) carried out a cross-sectional study among 10-16 years girls in Ludhiana, Punjab taking subjects both from government and private schools. A total sample of 1,049 children were taken to assess overweight and underweight with Body Mass Index (BMI) utilizing age and sex specific cut off points. According to WHO criteria the percentage of underweight, overweight and obesity was found as 6.42%, 28.59% and 7.78% in private school girls and 23.17%, 5.42 % and 0.93% in government school girls. A positive correlation of parental BMI was found on BMI of children suggesting that overweight parents are likely to have overweight children (Kaur and Mehta, 2012).

Pathophysiology of Overweight and Obesity

The fundamental cause of childhood overweight and obesity is an energy imbalance between calories consumed and calories expended. Global increases in childhood overweight and obesity are attributable to a number of factors including:

- A global shift in diet towards increased intake of energy-dense foods that are high in fat and sugars but low in vitamins, minerals and other healthy micronutrients;
- A trend towards decreased physical activity levels due to the increasingly sedentary nature of many forms of recreation time, changing modes of transportation and increasing urbanization (Mohan *et al.*, 1986).

Understanding the regulation of energy intake requires differentiating the short-term signals that control hunger, food intake, and satiety, as well as the long-term signals that relate to the defence of energy stores, lean tissue, or both. In short-term regulation, gastrointestinal signals provide important input to the brain. For the most part, hormones released from the stomach and intestine that affect food ingestion are inhibitory (Woods, 2004). One example is the recently identified polypeptide YY3-36, which is produced by the L cells of the small intestine. When YY3-36 is infused into lean or obese subjects, reductions in food intake by 30% are seen (Batterham *et al.*, 2003). Ghrelin is an exception. This peptide is produced by the stomach and proximal small intestine, and its release stimulates food intake. Ghrelin declines after a meal and rises before the next meal. Ghrelin is elevated in Prader-Willi syndrome (Cummings *et al.*, 2002) which is a genetic form of obesity accompanied by marked hyperphagia. Ghrelin is dramatically reduced after gastric bypass surgery (Cummings *et al.*, 2002); this fall in ghrelin is a potential explanation for the sustained anorexia and long-term benefit of this operation.

Adipose tissue is critically involved in feedback regulation of energy balance by the production of a number of peptide hormones, and leptin and adiponectin are 2 of the most important. The absence of leptin produces massive obesity, and treatment of leptin-deficient individuals reduces food intake and

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body weight (Farooqi *et al.*, 1999). In most obese people, however, leptin has little effect on food intake or body weight (Heymsfield *et al.*, 1999). Adiponectin is the most abundant hormone from fat cells, increases insulin sensitivity, and appears to be a cytokine that is anti-inflammatory (Ouchi *et al.*, 2003). To a large extent, the signals directed by leptin and other adipose tissue– derived peptides are integrated in the hindbrain and mid-brain through various signals (monoamines, neuropeptide-Y, agouti-related peptide, melanocyte–stimulating hormone), which in turn send efferent signals for food seeking and modulation of function of various organs, including the pancreas and muscle (glycerol3-phosphate dehydrogenase), and in rodents, brown adipose tissue (uncoupling proteins) (McMinn *et al.*, 2000). Metabolism of the adrenal steroid in adipose tissue may provide a mechanism for the increase in visceral fat. When the enzyme 11-hydroxysteroid dehydrogenase type-1, which converts cortisol to the inactive cortisone in fat cells, is genetically disrupted, mice develop visceral obesity (Masuzaki *et al.*, 2001). This pathophysiology may also apply to humans.

Overweight and Obesity- Definitions and Classification

Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2).

The WHO definition is:

- BMI greater than or equal to 25 is overweight
- BMI greater than or equal to 30 is obesity.

BMI provides the most useful population-level measures of overweight and obesity as it is same for both sexes and all ages of adults (Mohan *et al.*, 1986).

Table 1: The International Classification of adult underweight, overweight and obesity according to BMI

Classification	BMI (kg/m^2)	
	Principal cut-off points	Additional cut-off points
UNDERWEIGHT	<18.50	<18.50
Severe thinness	<16.00	<16.00
Moderate thinness	16.00-16.99	16.00-16.99
Mild thinness	17.00-18.49	17.00-18.49
NORMAL RANGE	18.50-24.99	18.50-22.99
		23.00-24.99
OVERWEIGHT	> 25.00	> 25.00
Pre Obese	25.00-29.99	25.00-27.49
		27.50-29.99
OBESE	>30.00	> 30.00
Obese class I	30.00-34.99	30.00-32.49
		32.50-34.99
Obese class II	35.00-39.99	35.00-37.49
		37.50-39.99
Obese class III	>40.00	> 40.00

Source: Adapted from WHO, 1995, WHO, 2000 and WHO 2004

BMI is the parameter most frequently used for the screening of excess body fat because it is easy to determine and it tends to correlate well with body fat.

In children and adolescents, based on BMI centile curves, International Obesity Task Force (IOTF) BMI cut off points have been recently proposed for each half-year of age, which correspond to the adult BMI

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values of 25 and 30 kg/m² at the age of 18 y (Cole *et al.*, 2000). This standard international definition allows the screening of adolescent overweight and obesity worldwide under the same criterion.

Implications of being Obese

Overweight and obesity are the fifth leading risk for global deaths. At least 2.8 million adults die each year as a result of being overweight or obese. In addition, 44% of the diabetes burden, 23% of the ischaemic heart disease burden and between 7-41% of certain cancer burdens are attributable to overweight and obesity. Research has shown that as weight increases, the risk for the following conditions also increases (National Heart Lung and Blood Institute, 1998):

- a) Coronary heart disease
- b) Type 2 diabetes
- c) Cancers (endometrial, breast, and colon)
- d) Hypertension (high blood pressure)
- e) Dyslipidemia (for example, high total cholesterol or high levels of triglycerides)
- f) Stroke
- g) Liver and Gallbladder disease
- h) Sleep apnea and respiratory problems
- i) Osteoarthritis (a degeneration of cartilage and its underlying bone within a joint)
- j) Gynecological problems (abnormal menses, infertility)

Strategies for Prevention

It has been recognized that the increasing prevalence of childhood obesity results from changes in society. Childhood obesity is mainly associated with unhealthy eating and low levels of physical activity, but the problem is linked not only to children's behaviour but also, increasingly, to social and economic development and policies in the areas of agriculture, transport, urban planning, the environment, food processing, distribution and marketing, as well as education.

The problem is societal and therefore it demands a population-based multisectoral, multi-disciplinary, and culturally relevant approach. Unlike most adults, children and adolescents cannot choose the environment in which they live or the food they eat. They also have a limited ability to understand the long-term consequences of their behaviour. They therefore require special attention when fighting the obesity epidemic.

Approaches pertaining to life style and community based needs to be implemented in order to address this silent epidemic.

- A. Life style approach
 - a) Healthy eating patterns
 - b) Increasing physical activity levels
 - c) Behaviour change communication
- B. Community based programmes
 - a) Training and orientation of teachers in lifestyle, nutrition and activity.
 - b) School health check-ups on a regular basis
 - c) Incorporating BMI charts in routine medical records.

Hence close monitoring of obesity prevalence in children and adolescents and taking timely preventive measures will be an effective approach in dealing with the problem of obesity.

REFERENCES

Batterham RL, Cohen MA, Ellis SM, Le Roux CW, Withers DJ, Frost GS, Ghatei MA and Bloom SR (2003). Inhibition of food intake in obese subjects by peptide YY3–36. *New England Journal of Medicine* **349** 941–48.

Cole TJ, Bellizzi MC, Flegal M and Dietz WH (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey. *British Medical Journal* **320** 1240–43.

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Cummings DE, Clement K, Purnell JQ, Vaisse C, Foster KE, Frayo RS, Schwartz MW, Basdevant A and Weigle DS (2002). Elevated plasma ghrelin levels in Prader Willi syndrome. *Nature Medicine* **8** 643–44.

Cummings DE, Weigle DS, Frayo RS, Breen PA, Ma MK, Dellinger EP and Purnell JQ (2002). Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. *New England Journal of Medicine* **346** 1623–30.

De Onis M and Blossner M (2000). Prevalence and trends of overweight among preschool children in developing countries. *American Journal of Clinical Nutrition* **72** 1032–1039.

Farooqi IS, Jebb SA, Langmack G, Lawrence E, Cheetham CH, Prentice AM, Hughes IA, McCamish MA and O’Rahilly S (1999). Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *New England Journal of Medicine* **341** 879–84.

Heysfield SB, Greenberg AS, Fujioka K, Dixon RM, Kushner R, Hunt T, Lubina JA, Patane J, Self B, Hunt P and McCamish M (1992). Recombinant leptin for weight loss in obese and lean adults: a randomized, controlled, dose-escalation trial. *Journal of American Medical Association* **282** 1568–75.

Kaur and Mehta (2012). Overweight and underweight among girls in Ludhiana (Punjab). *Human Biology Review* **1**(2) 197-206

Khadilkar VV, Khadilkar AV, Cole TJ, Chiplonkar SA and Pandit D (2010). Overweight and obesity prevalence and body mass index trends in Indian children. *International Journal of Pediatric Obesity* (Early Online) **1** - 9.

Masuzaki H, Paterson J, Shinyama H, Morton NM, Mullins JJ, Seckl JR and Flier JS (2001). A transgenic model of visceral obesity and the metabolic syndrome. *Science* **294** 2166–70.

McMinn JE, Baskin DG and Schwartz MW (2000). Neuroendocrine mechanisms regulating food intake and body weight. *Obesity Reviews* **1** 37–46.

Mohan V, Sharp PS, Cloke HR, Burrin JM, Schemer B and Kohner EM (1986). Serum immunoreactive insulin responses to a glucose load in Asian Indian and European Type 2 (non-insulin dependent) diabetic patients and control subjects. *Diabetologia* **29** 235-237.

National Heart Lung and Blood Institute (1998). Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. Available online: http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf [Accessed 17 August 2013].

Ouchi N, Kihara S, Funahashi T, Matsuzawa Y and Walsh K (2003). Obesity, adiponectin and vascular inflammatory disease. *Current Opinion in Lipidology* **14** 561–566.

Popkin BM (2001). The nutrition transition and obesity in the developing world. *Journal of Nutrition* **131**(S) 871-73.

Raj M, Sundaram KR, Paul M, Deepa AS and Kumar RK (2007). Obesity in Indian children: time trends and relationship with hypertension. *National Medical Journal of India* **20** 288-293.

Story M, Neumark-Sztainer D and French S (2002). Individual and environmental influences on adolescent eating behaviors. *Journal of the American Dietetic Association* **102** S40-51.

United Nations Children’s Fund (UNICEF) (2011). The State of the World’s Children Report. New York: UNICEF, Available from: http://www.unicef.org/sowc2011/pdfs/SOWC-2011-Main-Report_EN_02092011.pdf [Accessed 15 August 2013].

Wang L, Kong L, Wu F, Bai Y and Burton R (2005). Preventing chronic diseases in China. *Lancet* **366** 1821-1824.

Woods SC (2004). Gastrointestinal satiety signals I. An overview of gastrointestinal signals that influence food intake, *American Journal of Physiology - Gastrointestinal and Liver Physiology* **286** G7–G13.

World Health Organization (2003). Nutrition: Controlling the global obesity epidemic. Geneva, Available from: <https://apps.who.int/nut/obs.htm> [Accessed 15 August 2013].