

Childhood obesity, epidemiology, risk factors and consequences

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Introduction

During the past two decades, the prevalence of obesity in children has risen greatly worldwide. Obesity in children causes a wide range of serious complications and increases the risk of premature illness and death in later life.¹ Excess body weight is now the most common childhood disorder in Europe. It affects around one child in six, but in some parts it affects one child in three. Well-documented major increases in adult obesity levels are now mirrored in rapidly rising levels among children. Overweight children are more likely to become overweight adults, with a greater risk of cardiovascular disease, diabetes and other disorders.²

The prevalence of childhood obesity in the US has risen dramatically in the past several decades. About 25-30% of children are affected; this condition is underdiagnosed and undertreated. Hormonal and genetic factors are rarely the cause of childhood obesity; unnecessary diagnostic evaluation can be avoided with a careful history and physical examination. Obese children may suffer life-long physical and emotional consequences. Therefore, it is imperative to discuss prevention with parents during well-child examinations. All obese children should be screened for cardiac risk factors, as well as for possible orthopaedic, dermatological and psychiatric sequelae. Prevention is one of the hallmarks of paediatric practice. Documented trends in increasing prevalence of overweight and inactivity mean that paediatricians must focus on preventive efforts on childhood obesity, with its associated co-morbid conditions in childhood and likelihood of persistence into adulthood.³

Evaluation of obesity in childhood is important for several reasons. Firstly, it offers the best hope for preventing disease progression with its associated mortalities into adulthood. Secondly, while hormonal and genetic causes of obesity are rare, they do warrant consideration in obese children. In addition, obesity has a negative impact on the self-esteem of children and adolescents, which may have significant implications for long-term happiness and success in life. Finally, directed sessions that emphasise healthy eating and exercise habits for children and their families may have lasting effects on the lifestyle of these patients.³

Definition of obesity

Body mass index (BMI) is the ratio of weight in kilograms to the square of height in metres.⁴

- **At risk of overweight:** BMI for age 85th percentile to <95th percentile.
- **Overweight or obese:** BMI between the 85th and 95th percentile for age and sex.⁵
- **Overweight:** BMI for age >95th percentile.⁶
- **Underweight:** BMI for age <5th percentile.

Pathophysiology

A person gains weight when energy input exceeds energy output. Several studies have shown that, on average, obese children do not consume significantly more calories than their thin peers.⁷ Energy output comprises the basal metabolic rate, the thermal effect of food and activity. The thermal effect of food is the energy required to absorb and digest meals.⁸ By measure 3,500 calories is equivalent to 1lb; thus, an excess intake of 50-100 calories/day will lead to a 5-10lb weight gain over one year. As a result, a relatively small imbalance between energy input and output can lead to a significant weight gain over time. In fact, most obese children demonstrate a slow but consistent weight gain over several years.⁸

Epidemiology

The definition of overweight and obesity differs between epidemiological studies. The prevalence of childhood obesity is estimated to be 25-30%. The prevalence of obesity has increased by 54% in children aged 6-11 years and by 39% in adolescents aged 12-17 years. The prevalence of severe obesity has jumped 98% and 64% within these groups, respectively. Hispanic, native American and black patients tend to be more affected than other populations.

The prevalence of obesity has increased 2.3 to 3.3-fold over 25 years in the US, 20-fold over 10 years in England and 3.9-fold over 18 years in Egypt.^{3,9,10} The rate of childhood obesity in Canada has almost tripled over the past 20 years. The prevalence of obesity among 7-13 year olds rose from 15% in 1981 to 17% in 1996 for boys and 15% for girls. Canadian children are considerably more likely to

be overweight than their English, Scottish and Spanish peers. The probability of childhood obesity persisting into adulthood is estimated to increase from approximately 20%

at four years of age to approximately 80% by adolescence.¹¹

Tables 1 and 2 show International Obesity Task Force (IOTF) data for Europe.

Table 1. IOTF obesity in Europe childhood: collated data for children aged around 10 years.

Country	% overweight + obese	Date of survey	Age (years)/number of children
Belgium	18*	1992	8-10/348
Bulgaria	20	2000	10/618
Croatia	27*	1998	10/581
Czech Republic	16	2000	7-11/3,345
Denmark	18	1998	10/319
Finland	13*	1999	1-12/33,000
France	18	2000	7-9.9/1,582
Germany	15	1995	10/2,960
Greece	31	200	6-10/1,226
Hungary	18	1993-1994	10/232
Italy	36	2001	9/41,149
Malta	35	1992	10/519
Netherlands	14*	1997	10/700
Norway	21	1996	10/174
Poland	19	1996-1999	10/250
Slovakia	10	1995-1999	11/635
Spain	30	1998-2000	6-13/970
Sweden	18	2000-2001	10/6,700
Switzerland	16	1999	9-10/90
UK	22*	1998	10/257
Yugoslavia	16	1998	9-10/6,288

*Estimate based on mean and SD of BMI.

Table 2. IOTF collated data for trends in childhood overweight.

Country	Date of survey	% overweight + obese
Czech Republic	1991	10
	2000	13
France	1963	3
	1977	6
	1990	10
	1995	13
	2000	16
Germany	1975	11
	1985	12
	1995	18
Netherlands	1980	10
	1997	18
Poland	1994	8
	2000	18
Spain	1980	12
	1995	19
Switzerland	1965	4
	1975	8
UK	1974	8
	1984	8
	1994	17
Yugoslavia	1989	12
	1998	16

Risk factors

Body weight is regulated by numerous physiological mechanisms that maintain balance between energy intake and energy expenditure. A positive energy balance of only 500kj (120kcal) per day would produce a 50kg increase in body mass over 10 years.¹²

Development of effective prevention strategies mandates that physicians recognise populations and individuals at risk. Interaction between genetics and biological, psychological, sociocultural and environmental factors are clearly evident in childhood obesity. Elucidation of the hormonal and neurochemical mechanisms that promote the energy imbalance that generates obesity has come from molecular genetics and neurochemistry. Knowledge of the genetic basis of differences in the complex of hormones and neurotransmitters (including growth hormone, leptin, neuropeptide Y, melanocortin and others) that are responsible for regulating satiety, hunger, lipogenesis and lipolysis, as well as growth and reproductive development, will eventually refine our understanding of the risk of childhood overweight and obesity and may lead to more effective treatment.

Genetic conditions known to be associated with propensity for obesity include Prader-Willi syndrome, Bardet-Biedl

syndrome, Cohen syndrome and Alstrom syndrome. Progress has been made in mapping the genetic loci for these syndromes, although the molecular causes of these obesity syndromes have not yet been identified.^{13,14} Only a small percentage of childhood obesity is associated with a hormonal or genetic defect, with the remainder being idiopathic in nature.

It has long been recognised that obesity runs in families. High birth weight, maternal diabetes and obesity in family members all are factors, however there are likely to be multiple genes and a strong interaction between genetics and environment that influence the degree of obesity. For young children, if one parent is obese, the odds ratio is approximately 3 for obesity in adulthood; however, if both parents are obese, the odds ratio increases to more than 10. Before three years of age, parental obesity is a stronger predictor of obesity in adulthood than the child's weight status.^{15,16}

There are critical periods of development for excessive weight gain. Extent and duration of breastfeeding have been found to be inversely associated with risk of obesity in later childhood. This is possibly mediated by physiological factors in human milk, as well as by the feeding and parenting pattern associated with nursing.¹⁷ A pattern of rapid weight gain during the first four months of life was associated with an increased risk of overweight status at age seven years, independent of weight and weight attained at age one year.¹⁸

Adolescence is another critical period for development of obesity. The normal tendency during early puberty for insulin resistance may be a natural co-factor for excessive weight gain, as well as various morbidities of obesity. Early menarche is clearly associated with degree of overweight, with a two-fold increase in rate of early menarche associated with a BMI greater than the 85th percentile.¹⁹ The risk of obesity persisting into adulthood is higher among obese adolescents than among young children. Data suggest that adolescents who engage in high risk behaviour, such as smoking, ethanol use and early sexual experimentation, may be at greater risk of poor dietary and exercise habits.²⁰

Environmental risk factors for overweight and obesity, including family and parental dynamics, are enormous and complicated. Food insecurity may contribute to the inverse relation of obesity prevalence with socio-economic status, but the relationship is a complex one. Low income families, low cognitive stimulus in the house and maternal obesity all predict development of obesity.²¹

Leisure activity is increasingly sedentary, with wide availability of entertainment such as television, videos and computer games. In addition, with increasing urbanisation, there has been a decrease in the frequency and duration of physical activities of daily living for children, such as walking to school and doing household chores.

Furthermore, having a television in the bedroom has been reported to be a strong predictor of being overweight, even in pre-school-aged children. All these factors play a potential part in the epidemic of overweight.²² National survey data indicate that 20% of US children aged 8-16 years reported two or fewer bouts of vigorous physical activity per week and more than 25% watched at least four hours of television per day. Children who watched four or more hours of television per day had significantly greater BMI compared with those who watched fewer than two hours per day.²³

Television advertisements could adversely affect dietary pattern at other times throughout the day. British and US children are exposed to about 10 food commercials per hour of television time, most for fast food, soft drinks, sweets and sugar-sweetened breakfast cereals. Exposure to 30-second commercials increases the likelihood that three to five year olds would later select an advertised food when presented with options.²⁴

Children are exposed from the foetal stage onwards to a range of influences which increase their risk of becoming overweight. Those influences, which are embedded in the child's social surroundings, may be described collectively as the child's 'obesogenic environment' — the influences that lead a child to gain excess weight.

Health consequences of childhood obesity

Short-term consequences

1. Psychological consequences — Obese children are more likely to experience psychological and psychiatric problems than non-obese children. Girls are at greater risk than boys and the risk of psychological morbidity increases with age. Low self-esteem and behavioural problems were commonly associated with obesity. Strauss²⁵ found that 34% of obese white 13-14 year old girls had low self-esteem compared to 8% of non-obese white girls. However, there was also some evidence that parental, psychological or psychiatric problems might have a greater influence on the child's psychological morbidity than obesity or gender.

In addition, obese children with decreased levels of self-esteem demonstrate significantly high rates of sadness, loneliness and nervousness and are more likely to engage in high risk behaviour such as smoking or consuming alcohol.²⁵

2. Cardiovascular problems — Freedman et al²⁶ (results from US children aged 5-10 years) reported significant odds ratios (OR) for raised diastolic blood pressure (OR 4.5), raised systolic blood pressure (OR 4.5), raised LDL cholesterol (OR 3.0), low HDL cholesterol (OR 3.4), raised triglycerides (OR 7.1) and high fasting insulin concentration (OR 12.1).

The same study demonstrated that 58% of obese 5-10 year



olds had at least one of these five cardiovascular risk factors, 255 had two or more and the ORs for having two and three of the five risk factors (relative to non-obese 5-10 year old children in cohort) were 9.7 and 43.5, respectively.

3. Other clinical consequences — Becoming obese significantly increased risk of developing asthma symptoms in girls who were apparently non-asthmatic at base line.²⁷ Hypponen et al²⁸ found that paediatric obesity was associated with a more than two-fold risk of developing type 2 diabetes. Visser et al²⁹ found that of 3,512 children aged 8-16 years old participating in the third US National Health and Nutrition Examination Survey, overweight was significantly and independently associated with increased serum C reactive protein concentration. The adjusted OR reported relative to children with BMI<85th centile were 3.74 in boys and 3.17 in girls.

Obese children are at increased risk for orthopaedic problems. These include tibial torsion and bowed legs, slipped capital femoral epiphysis (especially in boys) and symptoms of weight stress in the joints of the lower extremities. Obese children are much more prone to skin disorders than non-obese children. These disorders include heat rash, intertrigo, monilial dermatitis and acanthosis nigricans (a condition that may be a marker for type 2 diabetes).³⁰ There is also a strong association between obesity and benign intracranial hypertension.

Long-term consequences of paediatric obesity

1. Social and economic effects — Obesity in adolescence has adverse effects on social and economic outcomes in young adulthood (e.g. income, educational attainment), controlled for a number of other variables, including intelligence quotient. There was some evidence that these effects might be more marked in women than in men. For example, British girls born in 1958 who had BMI>90th centile when studied at age 16 years had significantly lower income than girls with BMI<90th centile (by 7% on average) at age 23 years, controlled for social class and intelligence quotient.³¹

2. Persistence of obesity from childhood — Persistence of childhood obesity into adulthood was substantially more likely where children had at least one obese parent, where obesity was more severe (e.g. defined as BMI>95th centile compared to BMI>85th centile) and present at older ages. Adolescent obesity is probably even more likely to persist into adulthood than childhood obesity. Whitaker et al found that 69% of obese (defined as BMI>95th centile) 6-9 year olds in the US were obese as adults. In the same cohort, 83% of obese (BMI>95th centile) 10-14 year olds became obese adults.³² Estimates of obesity persistence based on cohort studies suggest that around 40-70% of obese pre-pubertal children will become obese

adults. These studies will underestimate the magnitude of the effect for modern children in more 'obesogenic' environments. For example, Freedman et al found that 77% of obese (BMI>95th centile) children were obese as adults.³³

Impact of childhood obesity on adult morbidity and risk of premature mortality

One cohort study of high methodological quality found that a BMI of >25kg/m² at age 18 years was associated with significantly increased mortality within 20 years of follow up. By the 32 year follow up, a BMI of ≥26kg/m² at age 18 years was associated with significantly increased mortality risk. All of the evidence appraised on long-term morbidity/mortality risk suggests adverse effects of childhood or adolescent obesity. Evidence on cardiovascular risk factors predicts increased adult cardiovascular morbidity and mortality arising from childhood obesity; this conclusion was also reached by all expert opinions.³⁴

Cardiovascular risk factors in adulthood

Most of these were reports from the Bogalusa Heart Study. Most of the publications reported significant associations between atherogenic profiles associated with obesity in childhood and those in adulthood. We can conclude that obesity-mediated cardiovascular morbidity in adulthood can have its origins in childhood obesity and that the magnitude of this problem is likely to be much greater now than in the past, following the epidemic of childhood obesity. Expert opinion has consistently reached similar conclusions.³⁵

Economic burden of obesity in youths aged 6-17 years

Using a multiyear data file of the National Hospital Discharge Survey (1979-1999), obesity-associated diseases and economic costs in youths (aged 6-17 years), it can be seen that the changes emerged over time. Diabetes, obesity, sleep apnoea and gallbladder disease were examined to explore the trend of the disease burden. Obesity-associated hospital costs were estimated from the discharges with obesity listed as a principal or secondary diagnosis.

From 1979 to 1981 and from 1997 to 1999, the percentage of discharges with obesity-associated diseases increased. The discharges of diabetes almost doubled (from 1.43% to 2.36%), obesity and gallbladder diseases tripled (0.36% to 1.07% and 0.18% to 0.59%, respectively) and sleep apnoea increased five-fold (from 0.14% to 0.75%). Ninety-six per cent of discharges with a diagnosis of obesity listed obesity as a secondary diagnosis. Asthma and some mental disorders were the most common principal diagnoses when obesity was listed as a secondary diagnosis. Obesity-associated annual hospital costs (based on 2001 constant US dollar value) increased more than three-fold from \$35 million (0.43% of total hospital costs) during 1979-1981 to \$127

million (1.70% of total hospital costs) during 1997-1999.³⁶

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