

JUVENILE OBESITY



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Obesity is prevalent in our country and, in fact, the entire world today, surpassing malnutrition as the leading cause of nutrition-related morbidity and mortality. When googling “obesity definition,” one finds: “noun; the condition of being grossly fat or overweight,” with the example sentence being “the problem of obesity among children.” UpToDate, one of the most respected online and up-to-date medical references used today, states that the term “*obesity* refers to an excess of fat. However, the methods used to directly measure body fat are not available in daily practice.” The body mass index (BMI) is the accepted standard measure of overweight and obesity for children 2 years of age and older.

The origin of pediatric obesity is complex and has many contributing factors. Pediatric obesity has its basis in genetic susceptibilities influenced by a permissive environment starting in utero and extending through childhood and adolescence. Easy availability of calorie-dense, nutrient-poor foods is most certainly a contributing factor, as is the lack of physical activity in our progressively techno-savvy society. Studies have shown that children born from the same women before and after weight loss surgery have different risks of obesity. Those born to mothers before bariatric surgery have higher rates of obesity and extreme obesity than their siblings born to the same mothers after weight loss surgery. Much research is still needed on this fascinating yet impactful trend.

Do BMIs in children correlate to BMIs in adults?

Body mass index, or BMI, is calculated with the following formulas: $BMI = \text{mass in kg} / \text{height in m}^2$ or $\text{mass in lb} / \text{height in}^2 \times 703$.

For adults, BMI is neither age nor gender specific, and nutritional status is defined by fixed cutoff points (Table 1, next page).

Executive Summary *Obesity has been on the rise in both developed and developing countries for several years. Medical science and the insurance industry are just starting to understand the impact of juvenile obesity on both the morbidity and mortality of insured lives. Although one of the major causes of this epidemic is energy intake/energy expenditure mismatch, this is a multifactorial condition with many unknown variables. Regardless of the cause, modifiable risk factors for and comorbidities of juvenile obesity should be screened for and addressed. This article is an overview of the most recent research.*

A BMI chart is not currently recommended for use in babies aged newborn to 24 months. However, BMI charts for juveniles from ages 2-20 years have been developed and used by several different agencies, including the CDC and the WHO, to track healthy weight ranges and determine when additional screening or intervention should be obtained. A more accurate measure of adiposity would include skinfold thickness and densitometry measurements, among others, but these methods are onerous and impractical. BMI has been considered an alternative to more direct methods, as it is inexpensive and easy to perform.

In juveniles, the adult BMI ranges do not correlate to those in adults because of normal periods of growth in children. Standard norms have been calculated from CDC growth charts based on US national survey data collected between 1963-65 and 1988-94. As most are aware, there has been an increase in overweight and obese individuals in the past 40 years throughout the US and the world, and data containing only recent measurements may be skewed toward overweight and obese individuals. BMI levels among juveniles are expressed relative to other children of the same age and sex.

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.

The categories of BMIs for children are:

| Child and Adolescent Weight Status Classification | |
|---|---|
| Weight Status | BMI Category |
| Underweight | <5 th percentile |
| Normal Range | 5 th -84 th percentile |
| Overweight | 85 th -94 th percentile |
| Obese | ≥95 th percentile |

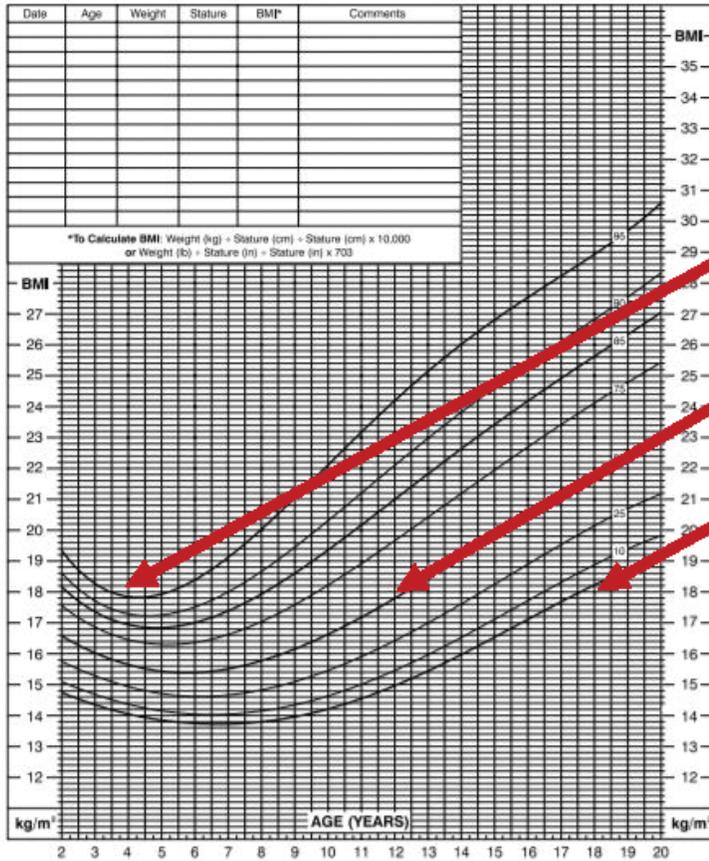
As age and sexual maturity are highly correlated with body fatness, the expression of healthy BMIs in juveniles is a J-shaped curve with a nadir between the ages of about 3 to 6 years of age (Figure 1, next page).

Hence, a BMI of 18 would be considered obese for a 4-yr-old boy, about 50th percentile for a 12-yr-old boy, and underweight for an 18-yr-old boy.

Figure 1

2 to 20 years: Boys
Body mass index-for-age percentiles

NAME _____
RECORD # _____



4 yo
OVERWEIGHT

12 yo
STANDARD

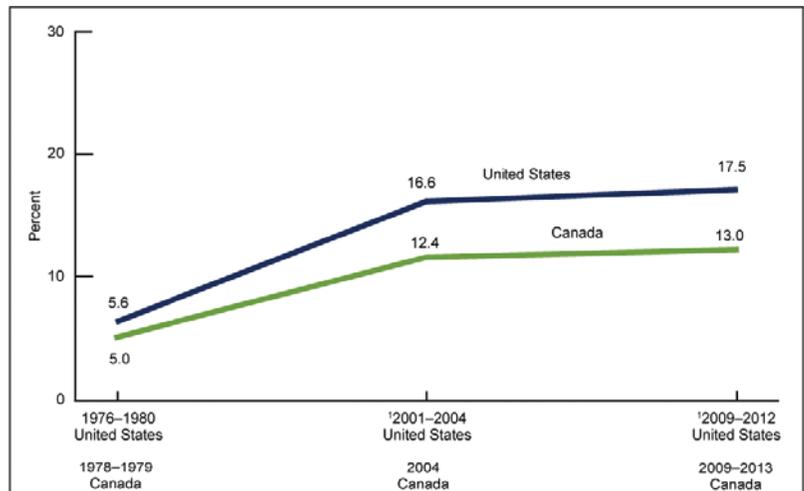
18 yo
UNDERWEIGHT

Published May 30, 2000 (modified 10/16/00). Source: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
www.cdc.gov/growthcharts

Figure 2: Trends in childhood obesity among children and adolescents aged 3-19 in Canada and the US

How prevalent of a problem is juvenile obesity?

The prevalence of overweight and obese children and adults has risen substantially in the last several decades, initially in developed nations and now in developing countries (Figure 2). The rates of severe obesity >99th percentile in children in the US in 2007 has risen 300% since the National Health and Nutrition Examination Survey (NHANES) in 1976, and has increased over 70% since NHANES in 1994 (Figure 3, next page). Obesity in the developed world appears to have leveled off, while it is still rising in developing countries. It appears to be of increasing concern to those of lower socioeconomic means, as nutrient-poor, caloric-dense foods are readily available and low in cost. Hence, throughout the world, the emphasis has been shifted from under-nutrition in most places to overnutrition.

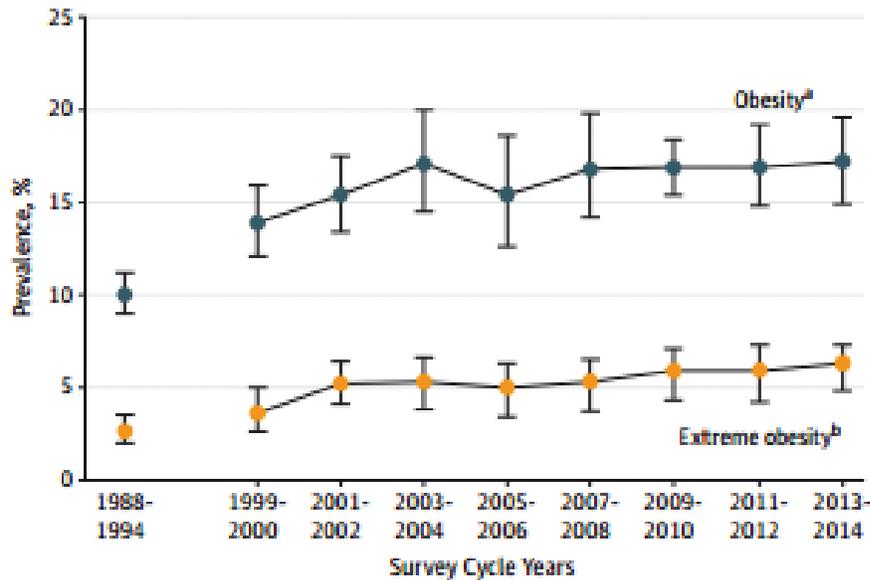


¹Statistically significant difference compared with Canada, p<0.001.

Note: Pregnant girls are excluded.

Sources: CDC/NCHS, National Health and Nutrition Examination Surveys, 1976-1980, 2001-2004, and 2009-2012; Canada Health, 1978-1979; Canadian Community Health Survey-Nutrition 2004; and Canadian Health Measures Survey, 2009-2013.
www.cdc.gov/nchs/products/databriefs/db211.htm

Figure 3: Prevalence of obesity and extreme obesity in US children and adolescents aged 2 to 19 years from 1988-1994 through 2013-2014



Data are from the National Health and Nutrition Examination Surveys. The error bars indicate 95% confidence intervals. The prevalence estimates are weighted.

^a Defined as at or above the sex-specific 95th percentile on the US Centers for Disease Control and Prevention (CDC) BMI-for-age growth charts.

^b Defined as at or above 120% of the sex-specific 95th percentile on the CDC BMI-for-age growth charts.

Ogden, Cynthia L., et al. "Trends in obesity prevalence among children and adolescents in the US, 1988-1994 through 2013-2014." *JAMA* 315.21 (2016): 2292-2299.

Differential diagnosis and American Academy of Pediatrics recommendations.

Differential diagnosis of childhood obesity

Although rare, the evaluation of a child with obesity must include the possibility of endocrine diseases, congenital and acquired hypothalamic defects, genetic syndromes, or the use of medications affecting weight and/or appetite. If signs or symptoms are noted, e.g., decreased growth velocity despite continued weight gain, appropriate investigations should be taken, such as TSH and free T4 measurement, or referral to an endocrinologist. The vast majority of children, however, will fall into the category of energy intake/expenditure mismatch. Regardless of the cause of the obesity, modifiable lifestyle risk factors should be addressed and comorbidities and complications of obesity should be screened for.

American Academy of Pediatrics recommendations

It is recommended by the American Academy of Pediatrics in its 2007 Child and Adolescent Overweight and Obesity Summary Report that all children with BMIs greater than the 85th percentile should:

- Have a blood pressure taken (with the correct cuff size).
- Have an initial fasting lipid profile (if over the age of 10 years).

- Have biannual fasting glucose and AST and ALT.
- Have screening by history and physical for many of the morbidities of childhood obesity.
- Be offered comprehensive multidisciplinary intervention.

The Endocrine Society, the European Society of Endocrinology and the Pediatric Endocrine Society have co-sponsored similar guidelines.

What are the morbidity and mortality concerns for elevated BMIs in juveniles?

Lifetime implications of childhood obesity

There are many morbidity and mortality implications to childhood obesity. Quite frequently an obese child becomes an obese adult, although about one half of currently obese adults were not obese as children. Approximately two-thirds of children in the highest BMI quartile become adults in the highest BMI quartile.

Obesity in children at 5 years and 9 years of age does not appear to be strongly correlated with adult mortality. Adult obesity, when compared to childhood obesity at these young ages, appears to be correlated to the parent's BMI, rather than the child's BMI. The risk of adult obesity is stronger if a child has ≥ 1 obese

parents. At this point in history, however, these children are quite likely to have obese parents.

The many associated comorbidities with childhood obesity include: asthma, Type 2 diabetes mellitus (T2DM), metabolic syndrome, nonalcoholic fatty liver disease (NAFLD), polycystic ovary syndrome (PCOS), obstructive sleep apnea (OSA), hypertension, premature puberty, slipped capital femoral epiphysis (SCFE) and acanthosis nigricans, among others. Adolescents with a higher BMI experience higher mortality rates as young and middle-aged adults, although the mortality is mostly associated with the higher adult BMI. This suggests that an obese adolescent can decrease his risk by moving into a healthier weight range. However, being an obese adolescent is still associated with a higher risk of multiple comorbidities in adulthood even if the obesity does not persist. In 1992, *New England Journal of Medicine* published "Long-Term Morbidity and Mortality of Overweight Adolescents, A Follow-Up of the Harvard Growth Study of 1922 to 1935," which followed a group of elementary school children for 55 years, and found that adolescent overweight (between 13-18 years old) was a more powerful indicator than adult overweight (at the age of 53 years) for all-cause mortality, coronary artery disease, angina and atherosclerosis for both men and women. The risk of death from colorectal cancer was increased in men, and the risk of morbidity, but not mortality, for osteoarthritis was increased in women. Increased risk of diabetes was present only for those with adult overweight.

It has been well documented that adult obesity portends increased mortality risk from hypertension, coronary artery disease, NAFLD, Type 2 DM, cerebral vascular disease, chronic kidney disease, respiratory disease and cancer. Several studies suggest that adolescents with higher percentile BMIs in the teen years continue on to be in the higher percentile BMIs in their adult years. That correlation has not been as strong with younger children, in which case the likelihood of obesity in adulthood of younger children has been more correlated to the parents' obesity status.

Nonalcoholic fatty liver disease (NAFLD)

NAFLD in children has been increasing, and has now become the most common liver abnormality in children aged 2-19 years. NAFLD is defined as hepatic fat infiltration in >5% hepatocytes in the absence of excessive alcohol intake, or the evidence of viral, autoimmune or drug-induced liver disease. Childhood NAFLD, as in the adult variety, is associated with metabolic impairments such as insulin resistance increasing the risk of T2DM, hypertension, metabolic syndrome, abdominal obesity, dyslipidemia and car-

diovascular disease. Both genetic and environmental factors appear to be involved. A few populations have a higher incidence of NAFLD, such as Hispanics and Asians, while African Americans have the lowest incidence. The prevalence is hard to determine, as definitive diagnosis requires a liver biopsy. The liver function tests AST and ALT are often slightly elevated in children with NAFLD; therefore, their measurements are a valuable and noninvasive screen for the disease. Ultrasonography has also been used as a screening tool, although fibrosis is undetectable with either of these methods. Although not widespread, by any means, fibroscan is being used increasingly to evaluate fibrosis.

An autopsy study in San Diego County from 1993-2003, published in *Pediatrics*, examined 742 children who died from unnatural causes. The prevalence of fatty liver, adjusted for age, gender, race and ethnicity, was 9.6%. A study in Taiwan in 2009 evaluated 748 school children aged 6-12 years by ultrasound, of which 219 were found to have steatosis. The rates of NAFLD were 3% in normal weight, 25% in overweight and 76% in obese children. A 20-year study of 66 adolescents with NAFLD (but not T2DM) reported that 6% died or required a liver transplant. Those not requiring transplantation had a standardized mortality ratio of 13.6. Several other studies done by examining liver biopsies obtained during bariatric surgery in adolescents have found an increasing prevalence of NAFLD with increasing BMI (incidence of 59-83% with BMIs > 50). Treatment to help prevent the severe progression to cirrhosis and liver transplant includes lifestyle modifications of diet and exercise, and often medical therapies (metformin, statins) to target the metabolic syndrome or the hepatoprotective response.

Type 2 diabetes mellitus (T2DM)

Type 2 diabetes has increased in prevalence over the years, while both Type 1 and Type 2 diabetes have increased in juveniles. The requirements for the diagnosis of diabetes mellitus in youth are the same as those for adults. Once the diagnosis of diabetes is made, one must then attempt to differentiate which type of diabetes the patient is exhibiting. In Cincinnati, OH, from 1982-1994, Type 2 DM diagnosis in adolescents rose 10-fold, with an average BMI at the time of diagnosis of 37.7. Type 2 DM has been continuing to increase in frequency, and now accounts for 20-50% of new onset diabetics in youth aged 10-19, depending on the population examined. It has continued to increase, as the overall prevalence of T2DM in youth has increased from 2001-2009 in five geographic areas in the US by 30.5% as noted by Dabelea et al. in *JAMA* in 2014. Currently, T2DM

is extremely rare under the age of 10 years. Obesity appears to be the driver in the increase in diagnosis, and acanthosis nigricans and PCOS are frequently associated. The presence of acanthosis nigricans, a hyperpigmented velvety rash most frequently present on the neck or at other skinfolds of adolescents, doubles the risk of T2DM over other youth at a similar BMI without acanthosis nigricans. Children with T2DM have a higher risk of long-term complications compared to adults with the same disease. They also have a higher risk of developing early diabetes-related aggressive complications than youth with T1DM with the same duration of illness, such as cardiovascular disease, peripheral vascular disease and neuropathy.

Children do not respond in the same fashion to the same degree that adults with T2DM do with the familiar medications. Depending on the severity of the hyperglycemia, the American Academy of Pediatrics recommends lifestyle changes along with insulin or metformin, the only medications currently approved for pediatric use.

Hypertension

Hypertension in children is defined as >95th percentile of BP according to age, sex and height. As in adults, blood pressure measurements must be taken at three separate visits for the diagnosis of hypertension, unless obvious morbidities of hypertension are present. It is extremely important to use the proper cuff size to obtain accurate measurements. Secondary hypertension is quite common in pediatrics, especially under the age of 10 years, with renal disease, coarctation of the aorta and adrenal disorders being three of the largest causes. Prepubertal, a secondary cause is most likely to be found, whereas post-pubertal, a primary cause is more likely, yet still a secondary cause must be excluded, as rates in some studies are as high as 50% prevalence of secondary hypertension, even in teens. Primary hypertension is growing in prevalence, and is highly associated with heredity and obesity. Hypertensive children, like adults, often are asymptomatic. Twenty percent of obese children have primary hypertension, while an even higher percentage of obese children fall in the prehypertensive range. Obese children have a three-fold higher risk for hypertension than nonobese children. Lifestyle changes and antihypertensive medications are the mainstays of treatment for primary hypertension, with the goal of limiting the long-term results of left ventricular hypertrophy and myocardial infarctions.

Implications in underwriting

When assessing the chart of an overweight or obese juvenile, care must be taken to evaluate for some of the target obesity-related diseases, which if present

we would appropriately rate or decline. Prudently, then, the remaining risk of early mortality must be assessed. A 2011 Pediatric Review in the *International Journal of Obesity* systematically reviewed several recent studies, and came to the conclusion that adolescent obesity increases the risk of all-cause mortality, with increasing MR (mortality rate) with increasing BMI levels, especially > 99th percentile. In *Annals of Internal Medicine* in 2006, vanDam published the conclusion of the Nurses Health Study II, which compared BMI at age 18 of over 100,000 female nurses who were cancer-free at baseline. They were enrolled at age 24-44 years, and were followed for 12 years. The overweight women had an SMR (standardized mortality rate) of 1.52, and the obese women had an SMR of 1.99 in that short period of time. Twig published in the *New England Journal of Medicine* in 2016 a study of 2.3 million Israeli 17-year-olds (more males than females) who were examined in 1967-2010, 1 year before entering military service. They were followed for 1-44 years. Those with a BMI of 85th-94th percentile were found to have an SMR of 1.3, while those with a BMI of 95th percentile or higher had an SMR of 1.68. Must published a follow-up of the Harvard Growth Study of 1922-1935 in the *New England Journal of Medicine* in 1992. There were 508 elementary school children followed for 55 years. BMIs between 13-18 years were used as baseline. They found that the all-cause mortality for overweight compared to lean had a relative risk of 1.8 for men, but was 1.0 for women.

Evaluating the above-mentioned studies, along with other research in the literature, leads to the conclusion that increasing BMI in adolescents predicts increased mortality risk as adults. In general, a BMI in the 85th-94th percentile has an SMR of about 1.5; a BMI above the 95th percentile has an SMR of about 1.75- 2; and at BMIs greater than the 99th percentile, the SMR is even higher starting at 2.5, and rising with increasing BMI. As with adults, individual factors should be taken into consideration, such as family history, degree of physical activity, and screening labs and medical tests, to most accurately assess the risk of any individual case.

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