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Obesity

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DEFINITION AND EPIDEMIOLOGY

Obesity is a disease that is usually defined as a body mass index (BMI) greater than or equal to 30 kg/m^2 (weight [kg]/(height [m])^2). A BMI of 30 to 34.9 is considered class 1 obesity, 35 to 39.9 is class 2 obesity, and 40 or higher is class 3 or severe obesity. The term "morbid obesity" previously was applied to individuals weighing at least 45 kg (100 lb) more than, or typically about 60% more than, desirable body weight; the term also has been applied to any individual with a BMI greater than or equal to 40 kg/m^2.

There is increasing recognition of limitations to defining obesity based on BMI resulting from the variable correlation between BMI and amount of body fat in different ethnic (genetic) populations or in individuals with different degrees of muscularity. Many investigators and clinicians are moving toward a definition that defines obesity as an excess of body fat sufficient to confer risk. Linking obesity to cardiometabolic risk, body fat distribution, and waist circumference is more important than measuring percentage body fat or BMI alone. People who accumulate visceral fat and clinically have higher waist circumference (metabolic obesity) are at much higher risk for cardiovascular disease and diabetes than those with the same BMI or the same percentage of body fat but a lower waist circumference. The National Cholesterol Education Program Adult Treatment Panel III (ATP III) considered a waist circumference greater than 40 inches (102 cm) in American men or 35 inches (88 cm) in American women to be among the five criteria that define the cardiometabolic syndrome. In spite of its limitations, BMI remains a simple measurement with utility in estimating a person's health risks and comparing outcomes between trials.

During the last 30 years, there has been a dramatic increase in the percentage of both adults and children in the United States who are overweight (defined as BMI of 25 to 30) or obese. According to the 2009-2010 National Health and Nutrition Examination Survey (NHANES) conducted by the Centers for Disease Control and Prevention (CDC), more than one third of U.S. adults (35.7%) were obese. This was more than double the prevalence in the 1976-1980 NHANES data (15.0%). Non-Hispanic blacks had the highest age-adjusted rates of obesity (49.5%), followed by Mexican Americans (40.4%), all Hispanics (39.1%), and non-Hispanic whites (34.3%). More recently, there appears to have been a slowing of the rate of increase or even a leveling off. Obesity prevalence varies significantly across states, from a low of 20.5% in Colorado to a high of 34.7% in Louisiana in 2012. In general, higher prevalence of adult obesity was found in the midwest (29.5%) and the south (29.4%) and lower prevalence in the northeast (25.3%) and the west (25.1%).

The percentage of children and adolescents who are overweight or obese has almost tripled since 1980. Currently, 17% of children and adolescents aged 2 to 19 years (12.5 million individuals) are obese. NHANES data from 1976-1980 and from 2009-2010 show the prevalence of obesity increasing from 5.0% to 12.1%, respectively, for children aged 2 to 5 years and from 5.0% to 18.4% for those aged 12 to 19 years. Among low-income preschool children, the prevalence of obesity increased between 1998 and 2003 from 13.0% to 15.2%, and severe obesity from 1.8% to 2.2%. These rates decreased slightly between 2003 and 2010: obesity from 15.2% to 14.9%, and severe obesity from 2.2% to 2.1%.

Overweight and obesity and their associated health problems have a significant economic impact on the U.S. health care system through direct medical expenses and indirect costs (e.g., loss of work time and productivity). Medical costs of obesity account for an estimated 10% of total U.S. medical expenditures. The estimated total annual medical costs of obesity in the United States was $147 billion in 2008, with medical costs on average $1429 higher per year for obese compared with normal-weight individuals. Approximately half of these costs were paid by Medicaid and Medicare.

PATHOLOGY OF OBESITY

Obesity develops as a consequence of genetic-environmental interactions, such that genetically prone individuals who lead a sedentary lifestyle and consume larger amounts of dietary calories are at higher risk. Children of obese parents are 80% more likely to become obese, and it is believed that this results from a combination of genetic and environmental influences.

The genetic contributions to obesity are most commonly considered to reflect the combined effects of variations in multiple genes and only rarely appear to result from a defect in a single powerful gene. Single-gene defects identified in experimental animals have been useful to demonstrate appetite and satiety mechanisms, and mutations in some of these same genes have subsequently been identified in rare human forms of genetic obesity. For example, loss-of-function mutations in the leptin gene and in the cellular receptor for leptin were first identified as a cause of obesity in laboratory mice (*ob/ob* and *db/db* mice, respectively). Leptin is a hormone that is produced in fat cells, mostly in subcutaneous fat. It is a potent satiety factor that acts in the arcuate nucleus of the hypothalamus to reduce the production of neuropeptide Y, a stimulator of food intake. After its discovery in mice, leptin gene mutations were identified as a cause