Definitions and Classification of Obesity

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Definition and etymology

Obesity is a state of excess adipose tissue mass. This condition usually translates into excessive body weight. On the one hand obesity can develop even in the absence of excessive body weight, whereas on the other hand a person (eg, a body builder) can develop remarkable overweight without excessive body fatness.

Interestingly, the word “obesity” (from the Latin *obesitas)* , used today in a purely descriptive way, in its etimology, points to the most common behavioral condition leading to obesity, ie overeating. In fact, *obesitas* is the condition of the *obesus* (ie, plump), word that, in turn, is composed of *ob* (ie, over) and *esus* , ie the past participle of *edere* (ie, to eat).

Diagnosis

Given that an excess of body fat is the defining variable of obesity, a proper diagnosis of obesity would require the assessment of body fatness. This can be performed indirectly in many different ways. Most accurately nowadays by using the dual energy x-ray absorptiometry (DEXA), which quantifies total and regional body fat by assessing the differential attenuation of two x-ray beams with different intensities (ie, energy levels) as they travel through the body of an individual. However, this methodology requires a special machine which involves a minimal exposure to x-rays. For practical reasons, the measurement of body weight has been adopted as a valid proxy for body fat and it is used to calculate the so called body mass index (BMI), which is defined as weight/height2 (kg/m2) (Fig.1).

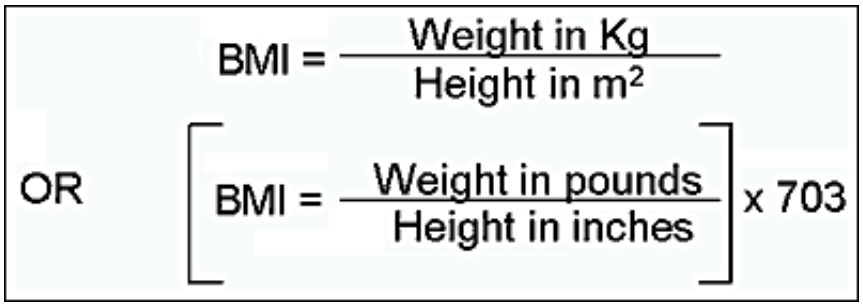


Figure. 1- Formulas for the calculation of BMI

The identification of the cut-off for obesity was based on large scale morbidity data, which pointed to a BMI =30 kg/m2 as the overall threshold for an increase in morbidity risk. Consequently, obesity is diagnosed in individuals with a BMI ≥30 kg/m2. In children, the diagnosis is based not on absolute parameters, but on the BMI percentiles of historical normal groups of the same age and gender, eg, the ones reported in the CDC growth charts (Figs.2-3). If the measured BMI is ≥ 95th percentile, obesity is diagnosed (1). A more severe obesity is staged at a BMI ≥ 97th percentile.

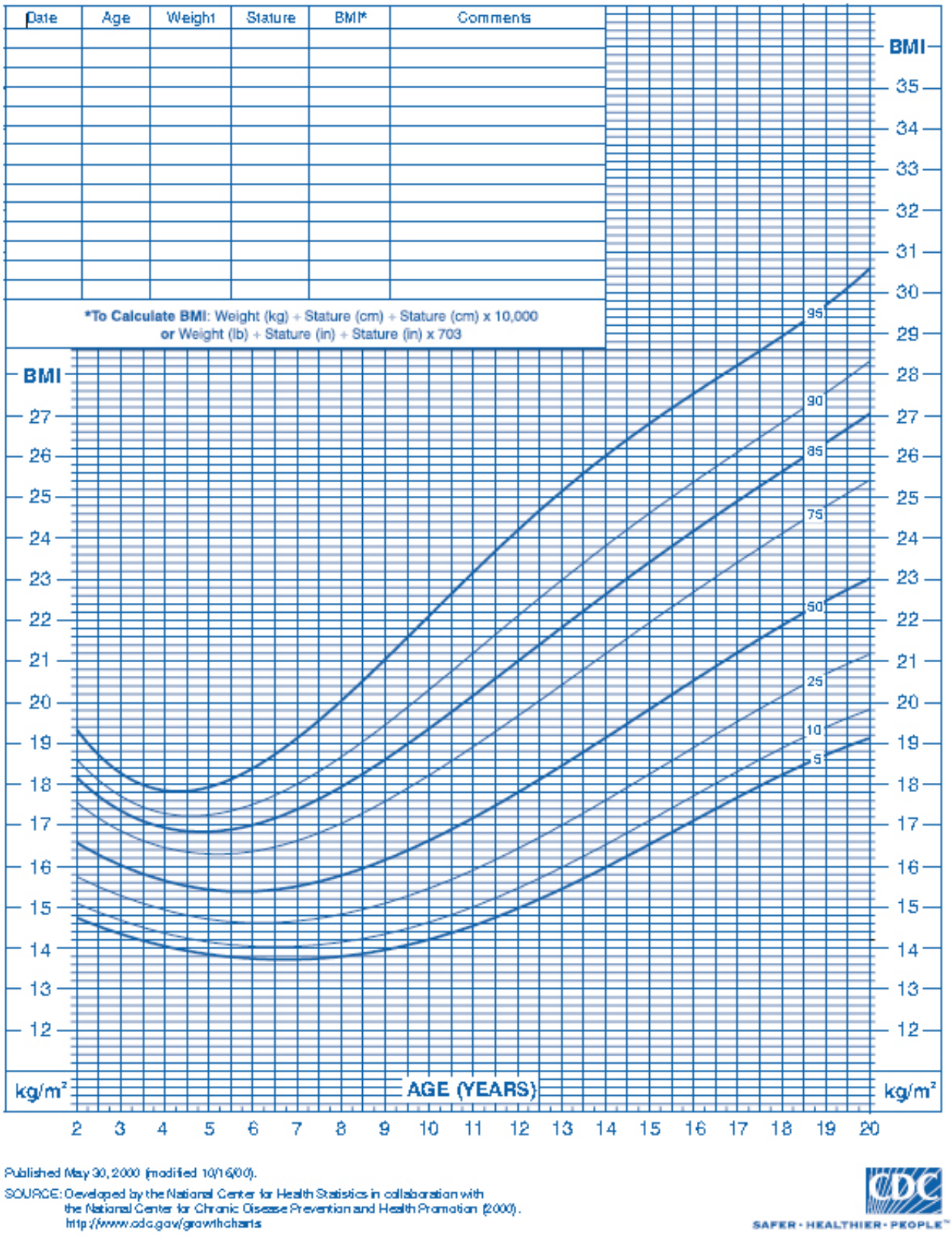
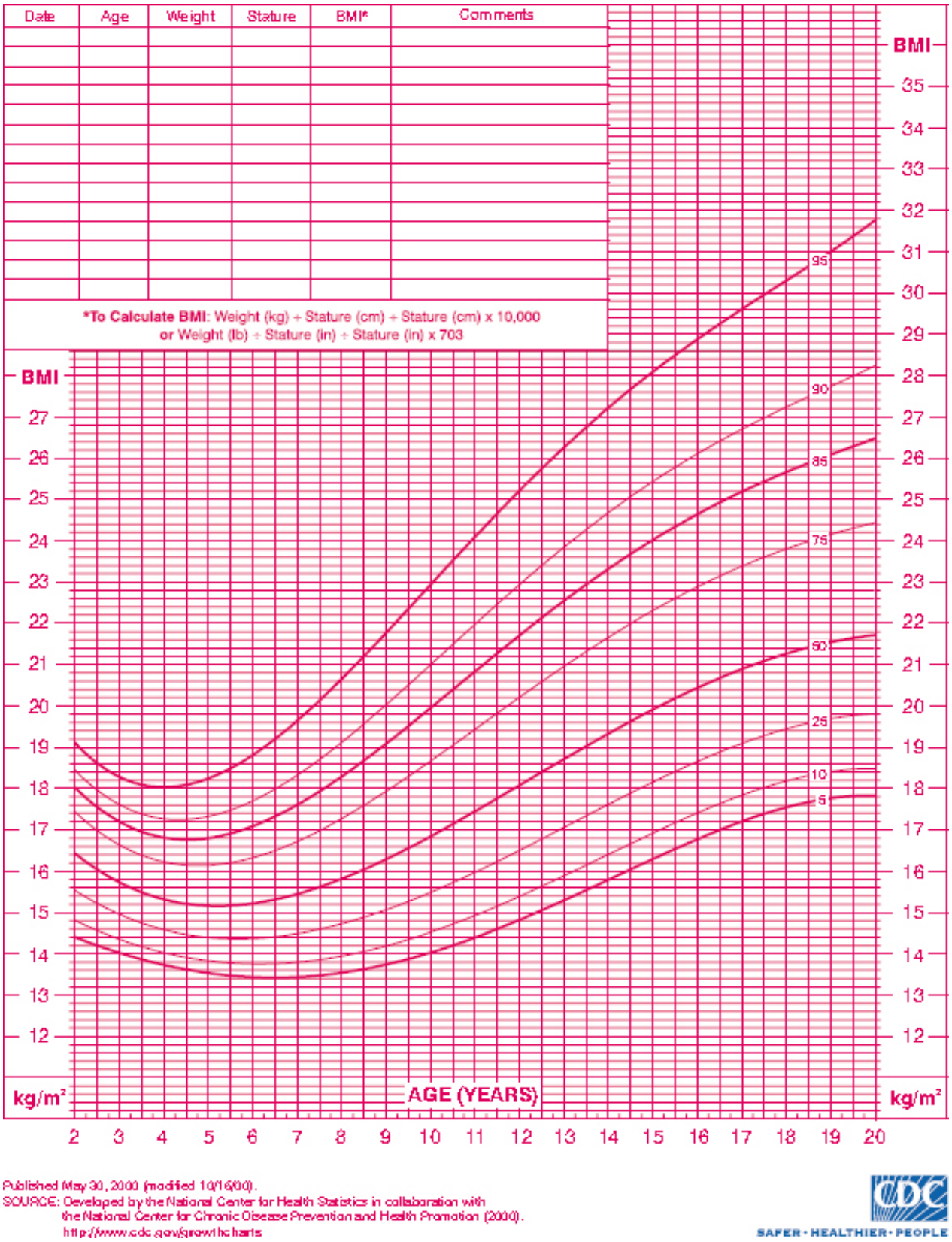


Figure2- BMI for age: percentiles – Boys, 2-20 years of age



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).
http://www.cdc.gov/growthcharts

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Figure 3- BMI for age: percentiles – Girls, 2-20 years of age

In their simplicity, which favored their wide use, these diagnostic criteria are very approximate. Besides the nosological inconsistence with the definition of obesity as excess body fat, a body weight based universal criterion lacks in sensitivity, for example, to the gender and ethnic related differences in relative body fatness. In fact, for the same BMI, women are, on average, fatter than men, and Asians are, on average, fatter than Caucasians. As a result, in Japan, obesity is diagnosed at a BMI ≥25 kg/m2 (2), in China at a BMI ≥28 kg/m2 (3), while for Caucasians, a BMI in the interval 25-30 kg/m2 is diagnosed as overweight. Diagnostic thresholds for obesity based on percent body fat have also been proposed: > 25% for men and > 33% for women (4), but there is no uniform consensus on these values (also because technique-dependent) and no general access to the gold standard technique for the assessment of body fat, ie DEXA, as discussed above.

Classification

Obesity can be classified in several different ways: for example, by BMI intervals and related aggregate risk of mortality, by anatomic phenotypes or by etiologic criteria.

According to the World Health Organization (WHO), obesity is classified as class I for a BMI between 30 and 34.9 kg/m2, class II for a BMI between 35 and 39.9 kg/m2, and class III for a BMI ≥ 40 kg/m2 (5). In turn, class I obesity is associated with (hence, labeled as) a “moderate risk”, class II with a “high risk”, and class III with a “very high risk” of mortality (4).

The most common anatomical characterization refers to a prevalently visceral or a prevalently subcutaneous deposition of fat. The ratio of waist circumference to hip circumference (WHR) has served the purpose of defining the degree of central (ie visceral) vs. peripheral (ie subcutaneous) obesity. It is known that visceral adiposity is a major risk factor for metabolic complications of obesity, while subcutaneous fat seems to be much more benign, and in some cases even protective against the development of metabolic complications (6).

From an etiologic standpoint obesity can be fundamentally classified as primary or secondary. Obesity, in fact, can be iatrogenic, ie secondary to pharmacologic treatments, including some antipsychotics, some antidepressants, some antiepileptics, and steroids. An obesity phenotype is also characteristic of some diseases or conditions, including polycystic ovary syndrome, Cushing’s syndrome, hypothyroidism, hypothalamic defects, and growth hormone deficiency. On the other hand, as a primary disorder, obesity still has an elusive etiology. While its pathogenesis can be expressed in relatively simple thermodynamic terms, ie the excess of body fat storage as a result of a chronic positive energy balance (ie, surplus of energy intake vs. expenditure), the identification of the primary causes of the chronic energy imbalance remains challenging, while the metabolic, psychological, and behavioral phenotypes leading to “garden variety” obesity are still controversial. In fact, excessive energy intake (or hyperphagia) is considered an obvious phenotype of obesity (7). However, linking hyperphagia to actual weight gain has proved exceptionally difficult (8;9), most likely because it is inherently challenging to measure energy intake in free-living individuals. Other aspects of food intake and their relationship to obesity, such as diet composition (10-16), energy density of food (17-19), rate of meal consumption (20), taste preferences (21-24), eating behavioral style (25;26), and sub-

phenotypes (27-31), have also been explored with somewhat contradictory results.

Not surprisingly, the molecular biology of obesity is also only partially understood. This is likely due to the heterogeneity of “garden variety” obesity and the fact that it is caused, like other complex diseases, not by a single genetic mutation but by multiple allelic defects, which determine susceptibility to environmental factors (32). Individuals who carry only one or some of these alleles may still not develop the disease, because they either lack another allele (gene-gene interaction) or are not exposed to the precipitating environment (gene-environment interaction). Furthermore, there is controversial evidence for a direct association between genotypes and lifestyle (33) or anatomical (34) phenotypes of obesity.

Epidemiology, mortality

Obesity affects a large proportion of the population, worldwide. However, estimates of its prevalence are not available for all countries, and the available data are not uniformly accurate or comparable (35;36). In the United States, the National Health and Nutrition Examination Survey (NHANES) in the last 50 years has provided a continuous monitoring of prevalence and incidence of obesity in a nationally representative sample of individuals. These data show that the prevalence of obesity among adults (age ≥ 20 years) started to increase markedly after 1980. In 2007-08 (the most recent set of data available) it reached an age-adjusted prevalence of 33.8% overall, correspondent to 32.2% in men and 35.5% in women (36) (Fig.4). However, over the last decade (1999-2008), no significant changes have been observed in the prevalence of adult obesity among women and only an overall linear trend for men has been observed, indicating that the prevalence of obesity among adults is not growing at the same rate recorded for the previous two decades (36). The highest prevalence is currently observed among non-Hispanic Blacks (44.1%, 37.3% for men, 49.6% or women), followed by Hispanics (38.7%, 34.3%, 43.0%), and non-Hispanic Whites (32.4%, 31.9%, 33.0%) (36) (Fig.5). By severity, grade 2 obesity has an overall prevalence of 14.3% (10.7% among men; 17.8% among women), while grade 3 reaches a prevalence of 5.7% (4.2% among men, 7.2% among women). The racial distribution follows the same pattern as for overall obesity, except for grade 3 which is as prevalent among Hispanics (5.5%) as among non-Hispanic Whites (5.2%) (36).

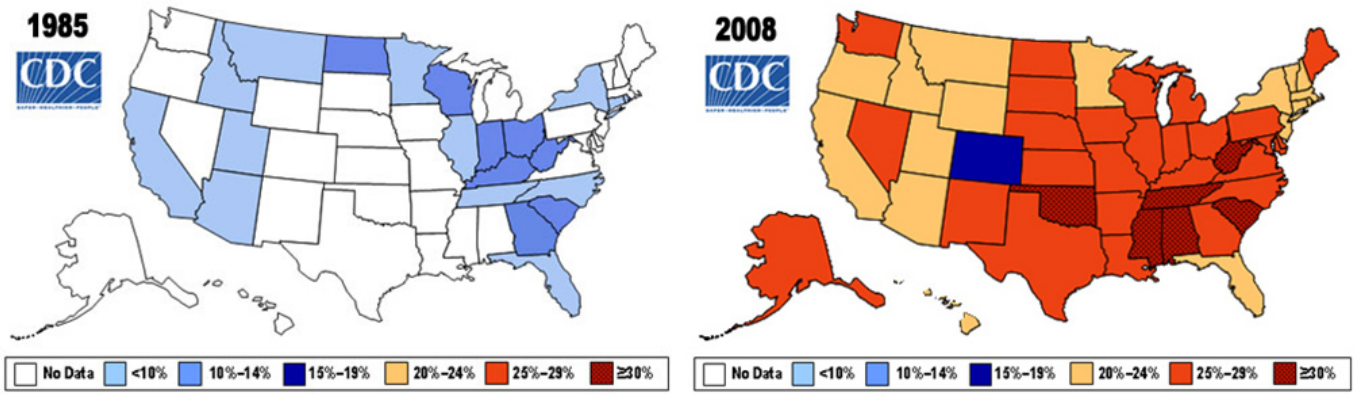

Figure 4- Prevalence of obesity (BMI ≥30 kg/m2) among U.S. adults in 1985 and 2008 (Source:

CDC Behavioral Risk Factor Surveillance System)

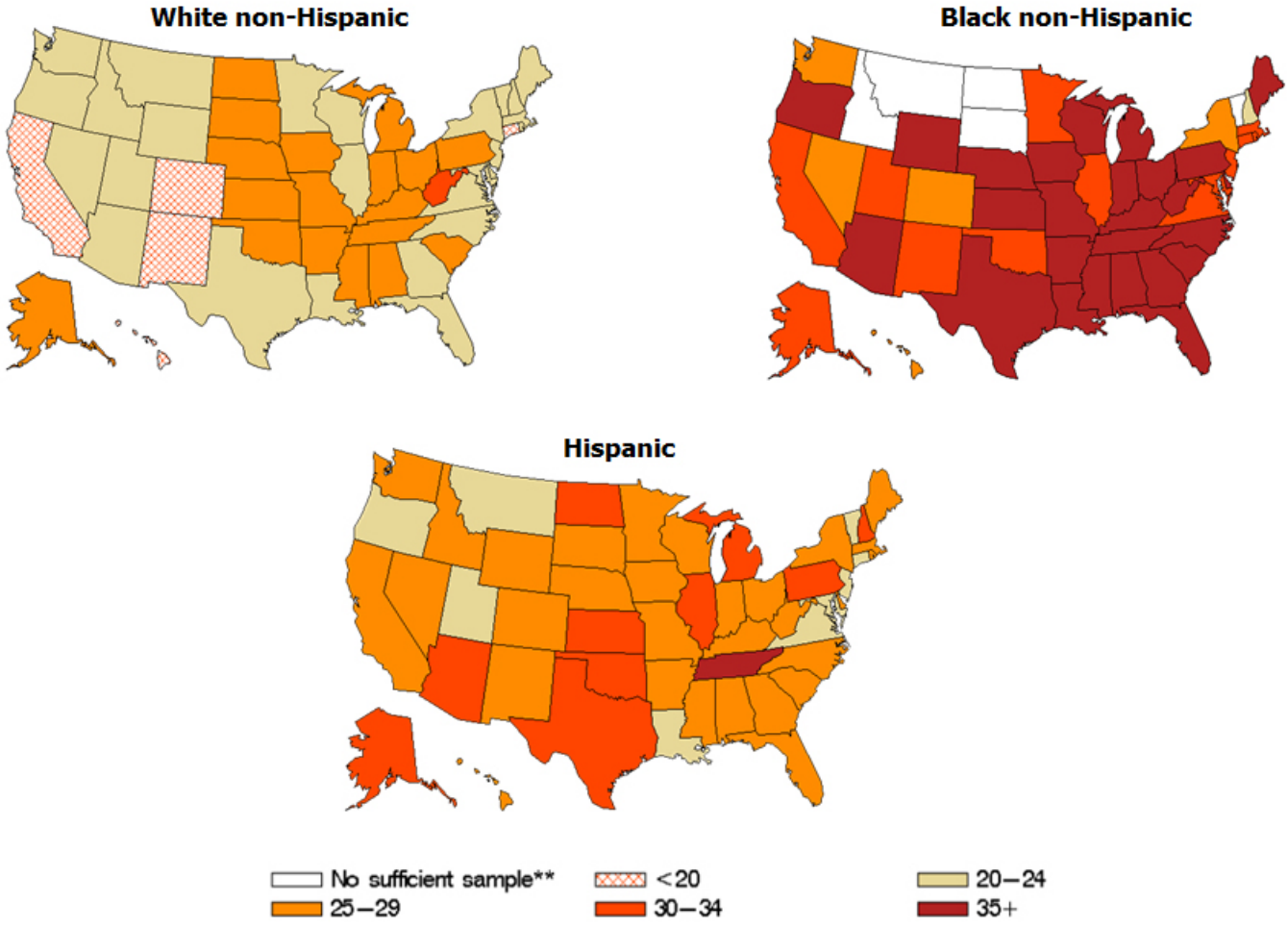

Figure 5- Prevalence (%) of obesity (BMI ≥30 kg/m2) among U.S. adults by Race/Ethnicity, 2006-2008 (Source: CDC Behavioral Risk Factor Surveillance System)

Among children and adolescents (age < 20 years), based on the 2007-08 NHANES, obesity reached a prevalence of 9.5% in boys and girls 0-2 years of age (10.0% for boys, 9.0% for girls), while the prevalence for children 2-19 years of age was 16.9% overall (17.8% for boys, 15.9% for girls)(37). Although the prevalence of obesity has tripled among school-age children and adolescents since 1980(37), no significant trend was observed in the last decade examined (1999-2008), except at the highest BMI cut point (BMI ≥ 97 th percentile) among 6-19 year-old boys (37). The prevalence of obesity by race-ethnicity in children is highest among Hispanics: 12.5% for 0-2 year-old children and 20.9% for 2-19 year-old children, followed by non-Hispanic Blacks: 10.3% for 0-2 year-old children and 20.0% for 2-19 year-old children, and non-Hispanic Whites: 8.7% for 0-2 year-old children and 15.3% for 2-19 year-old children (37).

Interestingly, obesity is also emerging as a pandemic in pets (Fig.6), particularly in dogs, with an estimated 20% to 40% of domestic dogs being obese (weight ≥ 115% ideal weight) (38). The importance of this growing problem is underscored by the availability of a specific drug (dirlotapide, Slentrol ®) recently approved by the FDA for treatment of canine obesity.

Figure 6-Una Familia[A Family] (a painting by F. Botero)

Although morbidity and mortality risks are generally higher for obese people, the relationship with BMI, in the obesity range, is not necessarily linear or uniform for disease (eg cancer) (39;40) subtypes , gender (41), or race/ethnic groups (41). In some cases, data do not support an increased risk in obese people (39;40). For hip fractures, the risk is reported to be lower for a BMI around 30 kg/m2 (39), while in obese patients undergoing hemodialysis (42), or with heart failure and peripheral artery disease (43), health outcomes seem to be better than for patients with normal weight. These observations have generated the so called “obesity survival paradox” (42). According to some authors, this paradox is explained by the fact that patients lose weight as the underlying illness progresses (44); another confounder could be that more aggressive treatment is administered to obese patients compared to normal weight patients (45). On the other hand, this paradox has recently been suggested as a potential explanation for two well established epidemiological observations: (1) the U-shaped (ie, concave) relationship between BMI and mortality rate, such that people with intermediate BMI (25-30 kg/m2) tend to live longer than people with lower or higher BMIs; and (2) the nadirs of these curves tend to increase

monotonically with age (46).

Historical references

Although it has exploded into pandemic proportions in the last two decades, obesity has been known since prehistoric times, as epitomized by the famous Venus of Willendorf (Fig. 7). The first to point out health risks associated with obesity and the pathogenetic role of excessive food intake was, however, Hippocrates, the great ancient Greek physician (47) around 400 BC. A few centuries later (AD160) Galen, a great ancient Roman physician, documented the first known successful weight loss program by increasing physical exercise and controlling food intake (47). Through the Schola Medica Salernitana and other medieval medical schools in Europe this knowledge was passed on to a growing number of practitioners and gained solid scientific bases by accruing clinical observations and post-mortem examinations. In fact, the first great pathologist, Morgagni, revealed in 1765 the existence of different fat depots and discussed the pathological relevance of the intra-abdominal fat and its correlated phenotypes (47). The research on the pathophysiology and phenotypization of obesity was then launched, and 245 years later remains a fertile field of scientific discoveries and controversies.

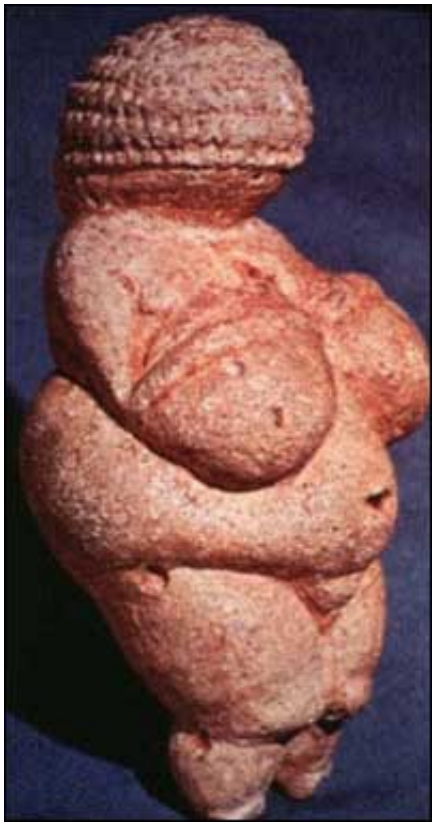

Figure 7- Venus of Willendorf. c.24,000-22,000 BCE (Naturhistorisches Museum, Vienna)

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