The Global Epidemic of Obesity: An Overview

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Abbreviation: BMI, body mass index.

For centuries, the human race struggled to overcome food scarcity, disease, and a hostile environment. With the onset of the industrial revolution, the great powers understood that increasing the average body size of the population was an important social and political factor. The military and economic might of countries was critically dependent on the body size and strength of their young generations, from which soldiers and workers were drawn. Moving the body mass index (BMI) distribution of the population from the underweight range toward normality had an important impact on survival and productivity, playing a central role in the economic development of industrialized societies (1).

Historical records from developed countries indicate that height and weight increased progressively, particularly during the 19th century. During the 20th century, as populations from better-off countries began to approach their genetic potential for longitudinal growth, they began to gain proportionally more weight than height, with the resulting increase in average BMI. By the year 2000, the human race reached a sort of historical landmark, when for the first time in human evolution the number of adults with excess weight surpassed the number of those who were underweight (2). Excess adiposity/body weight is now widely recognized as one of today’s leading health threats in most countries around the world and as a major risk factor for type 2 diabetes, cardiovascular disease, and hypertension (3).

This overview provides an introduction to this issue of Epidemiologic Reviews, highlighting, in historical perspective, key scientific aspects of obesity that are addressed by the 11 articles that follow. This compilation of reviews underscores the multidisciplinary nature of obesity research and the need to expand even further our scope to fully understand and confront the obesity epidemic.

WHEN WAS THE LAST TIME WE WERE NOT OBESE?

Until the last decades of the 19th century, developed countries were still struggling with poverty, malnutrition, and communicable diseases. These health problems were considered a major cause of low industrial productivity (4). In the first decades of the 20th century, studies of poor children indicated that dietary energy supplementation (adding sugar and fat to the usual diet) improved growth, which became an important approach to reduce malnutrition and improve industrial productivity. An influential proponent of improving health and nutrition of the working class as a means to improve overall economic productivity was Boyd-Orr (5), who later became the founding director of the Food and Agriculture Organization. A major initial goal of this organization was to increase the availability of low-cost calorie sources, primarily edible fats and sugars. Over the following decades, these efforts indeed led to major increases in the availability of dietary energy. According to the Food and Agriculture Organization, global food production by 2002 reached about 2,600 kcal per capita and is projected to reach almost 3,000 kcal by 2030 (6). Major contributors to total calories continue to be refined sugars and vegetable oils. While extreme disparities in access to adequate food availability continue to affect millions of people, there is no question that our ability to ensure stable production of dietary energy is one of the major achievements in human evolution.

Although obesity did not attract the attention of the mass media until recent decades, its prevalence in industrialized countries began to increase progressively early in the last century. By the 1950s, life insurance companies were already using body weight data to determine premiums, having identified an association between excess weight and premature death. In the early 1950s, Breslow (7) proposed
a direct link between the increasing prevalence of obesity and the also-increasing rates of cardiovascular disease in the US population, a theme that was reemphasized by the US government in reports in the 1960s and 1970s (8). Clear evidence of the alarming trend in obesity rates was provided by the regular, nationally representative surveys performed from the 1960s on. These data showed the continuing rise in obesity prevalence over the past 30 years (9). By the year 2000, 65 percent of the adult population had a BMI (weight/height²) above 25, and 30 percent had a BMI above 30 (10).

DEFINING OBESITY

Obesity is defined as an excess of body adiposity. For practical reasons, body weight has been used as a surrogate for adiposity, which is not easy to measure in routine examinations. Until the 1970s, obesity was defined by reference to an “ideal body weight,” derived from actuarial tables compiled by the life insurance industry. A body weight within the ideal range carried a lower risk of premature death. In the 1980s, the ideal body weight approach was replaced by BMI, and the commonly used cutoffs for overweight (BMI 25–30) and obesity (BMI >30), for both men and women, were adopted to define obesity in adults. However, it is recognized that the BMI association with mortality and morbidity risk is a continuous one and that it may vary in different ethnic groups (11, 12). Some countries and regions have already adopted their own cutoff for risk assessment using BMI (e.g., a cutoff of 23 in some Asian countries). The limitations of BMI as a risk assessment tool are also recognized, and there is continuing interest in identifying alternative or complementary indices linking body adiposity and disease risk. For example, some studies suggest that abdominal circumference is better correlated than BMI with risk of type 2 diabetes (13). Although it is well established that visceral adiposity plays a central role in the metabolic disorders associated with obesity, the lack of a practical method to assess visceral fat in routine examinations precludes its use as a screening tool for the general population. Developing simple and reliable methods to assess body fat compartments should be an important priority of obesity research.

WEIGHT GAIN AND ENERGY BALANCE

In spite of extensive research over the past decades, the mechanisms by which people attain excessive body weight and adiposity are still only partially understood. We will now discuss briefly the components of energy balance, which encompass many of the metabolic, endocrine, and behavioral aspect of excess weight gain.

Dietary energy intake

According to the laws of thermodynamics, the only way to accumulate excess body weight is through a positive energy balance, that is, when the input into the system exceeds the output. The relative contribution of excess energy intake versus reduced energy expenditure to the obesity epidemic in the United States and in other countries has been the subject of much study and debate. There are still methodological limitations in our ability to accurately measure dietary energy intake and energy expenditure in free-living populations. Therefore, estimates of energy balance in populations are based on self-reported dietary intake and physical activity and on food production and disappearance data. More accurate methods to measure energy output, such as gas-exchange calorimetry and doubly labeled water, can usually be applied to small groups only, studied under controlled conditions. Both approaches are useful—one to understand homeostatic mechanisms and regulatory factors, the other to assess the impact of those factors on body weight and disease risk in populations.

Data on dietary intake in the US population, which has one of the highest rates of obesity in the world, show a clear trend toward increased dietary energy intake. Dietary surveys and food disappearance data are consistent in indicating an increase in caloric intake in the US population of about 200 kcal/day over the past 20 years (14). A large proportion of this increase corresponds to the increased consumption of sweetened beverages, which now accounts for almost 25 percent of daily calories in young adults (15, 16). These “empty” calories have displaced healthier ones, particularly from fresh fruits and vegetables, whose consumption continues to be below recommended levels (17). Other factors cited as favoring excess dietary intake include low cost of energy-dense foods (18), increased consumption of prepared meals, and ample opportunities to eat throughout the day. Taken together, these data suggest that an increase in daily caloric intake is a contributing factor to the US obesity epidemic. As discussed below, increased availability of low-cost, energy-dense foods is also playing a role in the increasing rates of obesity seen in urban areas of developing countries.

Energy output: physical activity

The sedentary lifestyle of the US population was already a concern in the 1950s, when President Eisenhower created the Council on Fitness and Health to promote physical activity in the population. While secular data to assess trends are limited, in 2000 the Centers for Disease Control and Prevention estimated that less than 30 percent of the US population has an adequate level of physical activity, another 30 percent is active but not sufficiently, and the remainder is sedentary (19). A longitudinal study of girls aged 9–18 years documented the dramatic decline in physical activity during adolescence, particularly among Black girls (20). A number of factors may result in limited physical activity at schools, such as budget constraints and pressure to meet academic performance targets. Out of school, physical activity is also frequently limited. The Centers for Disease Control and Prevention reported a dramatic decline in the proportion of children who walk or bike to school, from close to 42 percent in 1969 to 16 percent in 2001 (21). At home, the average US teenager spends over 30 hours per week watching television (22). This activity is not only sedentary but also associated with reduced consumption of fresh fruits and vegetables (23), possibly related to
consumption of snack foods while watching television and to the influence of food commercials, most of which advertise low-nutrient-density foods (24).

The relative contribution of increased energy intake and decreased energy expenditure to the obesity epidemic is not easy to quantify. In countries such as the United States, the data show a dramatically low level of physical activity, particularly among children and adolescents, so one would conclude that this is a major factor in causing a positive energy balance in the US population. In turn, energy balance at such a low level of energy output could be maintained only by major reductions in food intake, perhaps to the point of jeopardizing intake of essential nutrients. Conversely, even minor increases in energy intake will result in a positive balance and weight gain. Both terms of the energy balance equation must change in order to put weight stability within the reach of most of the population.

**EARLY GROWTH AND LATER OBESITY**

The epidemiologist David Barker (25, 26) is credited with finding a link between early (fetal) growth patterns and risk of several chronic diseases in adulthood. In a retrospective review of medical records in Southampton, United Kingdom, Barker found that a high percentage of middle-age adults with cardiovascular disease were born at a low birth weight (27). This finding was somewhat counterintuitive, because the population studied was predominantly of low socioeconomic level and cardiovascular disease was traditionally associated with the better-off segments of the population. Still, Barker’s findings were consistent with observations among survivors of the Dutch famine during World War II, where intrauterine growth retardation was found to be associated with a high incidence of cardiovascular disease and diabetes in adulthood (28, 29). The concept of the “fetal origins” of adult diseases emerged from those observations, and it evolved into “developmental origins” to encompass pre- and postnatal events associated with accelerated growth and altered development of metabolic systems (30). Studies in animal models have shown that pups born undernourished exhibit hyperphagia, resulting in accelerated weight gain and increased body adiposity (31, 32). Pups gaining weight more gradually reach similar adult weight but have a normal body composition.

Low birth weight and excess weight gain in adulthood are additive risk factors for the comorbidities of obesity, particularly insulin resistance and type 2 diabetes (33). This association of earlier undernutrition with adult obesity has critical implications for developing countries, where intrauterine growth retardation and stunting during early childhood are common (34). As discussed below, the nutrition transition in developing countries results in increasing rates of adult obesity, leading to the emerging problem of chronic noncommunicable diseases in those countries (35).

**THE “OBESOGENIC” ENVIRONMENT: ENERGY BALANCE IN AN UNBALANCED WORLD**

Historically, human obesity was commonly associated with gluttony and lack of self-control at the table. Thus, treatment and prevention approaches were largely focused on individual behavior. Over the past decades, however, as the obesity epidemic continued to advance in the United States, there has been increasing focus on the external determinants of energy balance. The “built environment” represents the working and living conditions collectively created by societies and is a key determinant of opportunities and restriction to food consumption and physical activity. One dramatic example of how the built environment affects energy balance is mechanization and automation, which have sharply reduced the amount of energy we need to spend in basic survival activities and at work. Until recently, the obesity field was largely unfamiliar with the quantification of environmental variables such as air pollution, traffic patterns, and urban density, which have been widely used in environmental and occupational health. There are now incipient efforts to identify major factors in the built environment associated with excess weight gain (36). Factors in the built environment likely to have a significant impact on the average BMI of populations include 1) urban planning that promotes car use, necessitates long commutes, and restricts opportunities for walking (37); 2) limited and/or unsafe public spaces for recreational physical activity and for children to walk to school; 3) the pervasive presence of food outlets and opportunities to eat, usually fast, energy-dense foods; and 4) increasing dependency on prepared foods, usually consumed away from home.

**A GLOBAL EPIDEMIC**

Until relatively recently, obesity was considered a condition associated with high socioeconomic status. Indeed, early in the 20th century, most populations in which obesity became a public health problem were in the developed world, primarily the United States and Europe. In more recent decades, available data show that the most dramatic increases in obesity are in developing countries such as Mexico, China, and Thailand (38). The global nature of the obesity epidemic was formally recognized by a World Health Organization consultation in 1997 (39). Although few developing countries have nationally representative longitudinal data to assess trends, global estimates using both longitudinal and cross-sectional data indicate that obesity prevalence in countries in intermediate development has increased from 30 percent to 100 percent over the past decade (40).

The emergence of obesity in developing countries initially affected primarily the higher socioeconomic strata of the population. But more recent trends show a shift in prevalence from the higher to the lower socioeconomic level. For example, national surveys in Brazil found that while in 1989 obesity in adults was more prevalent in the higher socioeconomic status, 10 years later the higher prevalence was observed among the lower socioeconomic status (41). This change increasingly results in the existence of households with an undernourished child and an overweight adult, a situation called the “dual burden” of disease (42, 43).

Of the multiple causal factors associated with the rise in obesity in developing countries, perhaps the two most
important are urbanization and globalization of food production and marketing. Urban dwelling has a profound effect on energy balance, particularly on energy expenditure. On the energy output side, urban living is usually associated with lower energy demands compared with rural life. The energy-intense manual labor typical of rural areas may be replaced by a sedentary desk or sidewalk job. Long walks to work or to procure wood or water are replaced by mechanized transportation and public utilities. The global nature of modern commerce, sustained by the technical advances in food production and transportation, has permitted the introduction of low-cost, energy-dense foods in the domestic food market of many developing countries. Marketing campaigns and price incentives have an important impact on food purchasing patterns in developing countries, where as much as 60 percent of household income is spent on food. Consumption of energy-dense foods coupled with reduced energy expenditure facilitates weight gain in adults. In children, the low nutrient content of these foods may not be adequate to sustain normal growth because children require far more nutrients per calorie than adults do. As economic development brings some characteristics of urban lifestyle to rural communities, these populations also begin to show increasing rates of obesity, particularly among women (44).

The economic transition that brings urbanization is supposed to elevate socioeconomic level as well. However, several studies suggest that economic development may not reduce urban poverty but in fact may increase economic disparities, particularly in the case of children (45, 46). The result is households in which underweight children coexist with overweight adults, particularly women, a phenomenon termed the “dual burden” household (42, 43).

How can we swing the pendulum back to a healthier BMI level? During the past decades, numerous scientists and organizations around the world have worked tirelessly to answer those questions, so far with only modest success. But there is no question that we have made enormous progress in understanding obesity, from the genetics of energy metabolism and adipocyte regulation to social and individual behaviors and the role of the built environment. Continuing efforts are improving and standardizing our diagnostic and management tools, while there is an increasing emphasis on reducing risks as early in life as possible.

As discussed in this overview, there is an increasing consensus among obesity experts that changing the “obesogenic” environment is a critical step toward reducing obesity. Reversing the factors described above that lead to increased caloric consumption and reduced physical activity would require major changes in urban planning, transportation, public safety, and food production and marketing. Possible options to effect these changes have already been proposed by expert panels from the Institute of Medicine (47, 48). Several states and counties around the country have moved forward with obesity awareness and prevention initiatives in schools, the workplace, and the community, and even a few food corporations have responded by changing product composition and marketing practices. The next challenge will be to integrate all these efforts into a sustainable and coherent prevention plan that truly changes the unhealthy aspects of our living environment. Globally, there is also solid consensus on the steps needed to stop the obesity epidemic, which were outlined in the Food and Agriculture Organization/World Health Organization global strategy for the prevention of diet-related chronic diseases (49). But political leaders still tend to regard obesity as a disorder of individual behavior, rather than highly conditioned by the socioeconomic environment. This perception must change in order to recognize that the threat of obesity and its comorbidities is already affecting the future of young generations throughout the world.

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REFERENCES


