Prevention of Overweight/Obesity as a Strategy to Optimize Cardiovascular Health

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The prevalence of obesity in the United States and the world has risen to epidemic/pandemic proportions. This increase has occurred despite efforts by healthcare providers and consumers alike to improve the health-related behaviors of the population and a tremendous push from the scientific community to better understand the pathophysiology of obesity. This epidemic is all the more concerning given the clear association between excess adiposity and adverse health consequences such as cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM). These risks associated with overweight/obesity are primarily related to the deposition of excess adiposity or body fatness. Weight loss, specifically loss of body fat, is associated with benefits in all of the obesity-related comorbidities, but, unfortunately, most weight loss interventions are associated with weight regain and are therefore not successful in the long term. It is for these reasons that efforts to prevent weight gain and overweight/obesity are necessary. This is especially important when one considers younger individuals, who have even more to lose as a consequence of a longer duration of excess adiposity.

After a brief review of the epidemiology of obesity, this statement will make the case for the importance of weight gain prevention. This argument will first include a review of the complications of overweight and obesity in both adults and children, including the future CVD risks of obesity in early life. Energy balance dysregulation and adaptations to the weight-reduced state, favoring weight regain, will then be reviewed as further argument for the need for obesity prevention. This will be followed by a discussion on the goals and strategies for accomplishing the difficult task of the prevention of weight gain and obesity.

Scope of the Problem

Classification of Overweight and Obesity

The body mass index (BMI) is the most widely used and accepted method for the assessment and classification of excess adiposity or body fatness. Overweight and obesity are classified according to BMI for adults. A BMI <18.5 kg/m² is considered underweight, between 18.5 and 24.9 kg/m² corresponds to a healthy weight, between 25.0 and 29.9 kg/m² is overweight, and ≥30.0 kg/m² is obese. Obesity is further classified as stage I or mild (BMI of 30.0 to 34.9 kg/m²), stage II or moderate (BMI of 35.0 to 39.9 kg/m²), and stage III or severe (BMI of ≥40.0 kg/m²). In children, BMI percentiles adjusted for age and sex and calculated on the basis of a compilation of national survey data collected over a 30-year period are used. In children aged 2 to 19 years, overweight is defined as a BMI between the 85th and 95th percentiles, and obesity is defined as a BMI ≥95th percentile.

Epidemiology of Overweight and Obesity

The prevalence of overweight and obesity in the United States and the world has risen dramatically over the last 4 to 5 decades and has been summarized. In the most recent US population estimates based on data from the 2007–2008 National Health and Nutrition Examination Survey (NHANES), 34% of adults in the United States were obese, and 68% were either overweight or obese. Although obesity prevalence appears to have stabilized to some degree over the past few years, the prevalence of severe obesity has continued to increase. The overall prevalence of overweight and obesity in children and adolescents has also increased dramatically over the last 4 decades, with rates of obesity now approaching 17%. Approximately 10% of preschool children, 20% of those aged 6 to 11 years, and 18% of adolescents are obese. As in adults, however, trends over the last decade have not shown a further increase in childhood obesity except in the heaviest boys.

Despite the apparent stabilization of obesity prevalence, there continue to be significant racial and ethnic, as well as geographic and socioeconomic, disparities. Hispanics and non-Hispanic blacks have a significantly higher prevalence of both overweight and obesity compared with non-Hispanic whites. Forty-four percent of non-Hispanic blacks are obese, and 80% of Hispanics are overweight or obese. Similar racial
and ethnic disparities are seen in children, especially among Hispanic boys and non-Hispanic black girls. The prevalence of obesity is 17% in non-Hispanic white boys, 20% in non-Hispanic black boys, and 27% in Hispanic boys. In girls, the prevalence is 14% in non-Hispanic whites, 29% in non-Hispanic blacks, and 17% in Hispanics. In addition, the prevalence of obesity is highest among low-income children.7 Geographic disparities are also evident, with the highest rates of obesity seen in the Southeast, Appalachia, and tribal lands in the West and Northern Plains.8

These changes in the prevalence of overweight and obesity have occurred over a relatively short period of time, suggesting that genetics, although important, are not the primary cause of this epidemic. Most agree that the epidemic is primarily due to changes in the environment that promote reduced energy expenditure and increased energy intake in "at-risk" individuals; however, environmental influences on gene expression or epigenetic effects remain possible.

The Case for Prevention

It seems obvious that preventing a disorder or disease makes more sense than treating one; this concept holds especially true for obesity. As will be discussed in this section, the "case" for obesity prevention will be centered on 2 key premises. The first is that obesity is associated with many "downstream" complications and costs that can and need to be prevented. The second is that although treating obesity is associated with improvements in all obesity-related comorbidities (reviewed elsewhere),9 the long-term success of obesity treatment is fraught with difficulties, primarily because of the biological and behavioral impetus to regain weight.

Complications Associated With Overweight and Obesity

Comorbidities of Obesity in Adults

Obesity has been consistently shown to be associated with an increased risk of all-cause mortality.9,10–13 The data are conflicting, however, for the effects of overweight on mortality, with some studies showing no increased and even reduced mortality with overweight, which is known as the "obesity paradox."10,12 The effects of excess adiposity on mortality appear to be most apparent in adults during midlife compared with older adults and in healthy nonsmokers. The association between BMI and mortality has also been shown to vary by cause of death. Obesity, for example, has been shown to be associated with increased CVD and obesity-related cancer mortality11 but not with mortality due to other causes. Obesity has been shown to be independently associated with CVD, specifically coronary heart disease14–16 and stroke,17 which prompted the American Heart Association to adopt obesity as a major CVD risk factor in 1998.18 Weight gain independent of obesity status is also associated with increased risk of CVD.19 These associations appear to be especially true for those with central obesity, a core component of the metabolic syndrome (defined as ≥3 of the following: central obesity, elevated blood pressure, low high-density lipoprotein cholesterol, elevated triglycerides, impaired fasting glucose).20 Markers of central obesity such as waist circumference and waist-to-hip ratio have been shown to be independently associated with CVD risk.21–24 Furthermore, obesity has been shown to be associated with the development of T2DM, with excess adiposity being a key contributor to the development of insulin resistance.25,26 In fact, the significant rise in the prevalence of diabetes mellitus is thought to be primarily due to the obesity epidemic. Overweight and obesity are also associated with other CVD risk factors such as systemic hypertension, metabolic dyslipidemia, inflammation, and thrombosis. Finally, obesity has also been associated with many other non–CVD-related health complications, including osteoarthritis, obstructive sleep apnea, polycystic ovarian syndrome, depression, gastrointestinal diseases, and cancer, to name a few.27–38

Comorbidities of Obesity in Children and Adolescents

Obesity in childhood and adolescence is associated with a variety of concurrent metabolic and CVD risk factor derangements such as insulin resistance and T2DM, systemic hypertension, dyslipidemia, and inflammation. Worldwide, T2DM has increased in adolescents in the past few decades in parallel with the obesity pandemic,39 suggesting an impending earlier onset of diabetic vascular complications.40,41 Although numerous definitions of the metabolic syndrome exist in pediatrics,42 obesity is a key component, and the Princeton Lipids Clinic Follow-Up Study reported that for every 10-point increase in childhood BMI percentile, metabolic syndrome increased by 24%.43 Systemic hypertension,44 low high-density lipoprotein cholesterol, and elevated triglycerides45,46 are common CVD abnormalities seen with pediatric obesity. Childhood obesity is also associated with inflammation, as measured by elevated C-reactive protein, an important component of atherosclerosis.47

In addition to the metabolic derangements of obesity in children and adolescents, other associated comorbidities have been reviewed recently48 and include psychological (depression and decreased quality of life), pulmonary (asthma and obstructive sleep apnea), orthopedic (slipped capital femoral epiphysis and Blount’s disease), renal (increased proteinuria), hepatic (nonalcoholic fatty liver disease and steatohepatitis), and neurological (pseudotumor cerebri) disorders, among others. Finally, Franks et al49 recently found that obesity in childhood is strongly associated with increased rates of premature death from endogenous causes.

Cardiovascular Disease Risk in Adulthood of Obese Children

As discussed above, increases in the prevalence and degree of obesity in childhood and adolescence have been well documented in the United States and worldwide over the past several decades. Numerous epidemiological studies worldwide demonstrate that childhood obesity is strongly correlated with or tracks into adulthood.50 Recently, data analyzed from the US National Longitudinal Study of Adolescent Health found that over a 13-year period between adolescence (1996) and adulthood (2007–2009), obese adolescents (>95th percentile) were 16 times more likely to develop severe obesity than normal-weight or overweight (85th to 95th percentile) adolescents.51 Studies performed in a longitudinal cohort in Minnesota have demonstrated that BMI at
13 years of age predicted BMI and insulin sensitivity at age 22 years and that adiposity and insulin sensitivity each independently predicted CVD risk factors, but they synergistically predicted increased CVD risk factors. The Bogalusa Heart Study reported that childhood BMI and age of obesity onset predict adult systemic hypertension, although not independent of adult obesity. Results from the Young Finns, Bogalusa Heart, and the Childhood Determinants of Adult Health studies concluded that adolescent lipid levels are more strongly associated with adult carotid intima-media thickness than change in lipid levels and that dyslipidemia combined with high BMI is associated more strongly with increased carotid intima-media thickness than either factor. Compelling data also exist on the association of obesity with atherosclerosis in autopsy specimens in adolescents and young adults in the Bogalusa Heart Study and the Pathobiologic Determinants of Atherosclerosis in Youth study. Currently, however, the precise mechanism for increased CVD in adulthood due to pediatric obesity is uncertain (Figure 1). Further data are needed on whether childhood obesity increases adulthood CVD risk simply because of the tracking of obesity from childhood to adulthood or via increases in CVD risk factors in childhood and young adulthood such as dysregulated glucose metabolism (insulin resistance/T2DM), systemic hypertension, dyslipidemia, and inflammation or via multiple pathways.

Acknowledgment of the need for surrogate markers detectable early in the natural history of CVD to study the effect of childhood obesity on adulthood CVD is another important consideration. Few cardiovascular events occur in young adulthood, and investigation of the association of childhood risk factors with adult disease requires following these cohorts for years to monitor change in surrogate markers and even longer, often many decades, for hard outcomes such as myocardial infarction or death. Although longitudinal cohorts have demonstrated that childhood obesity is associated with adulthood CVD as measured by surrogate markers, the Young Finns Study has also demonstrated that improvement of obesity from childhood to young adulthood can reverse the effect of childhood obesity on adult CVD risk factors and future atherosclerosis. This suggests that although primary prevention of obesity in childhood will reduce CVD risk factor burden over time, a window of opportunity exists in which efforts to reduce obesity in young adulthood can reduce adulthood CVD risk factors. Moreover, it has been suggested that prevention goals should be refocused from decreasing risk for future CVD events to preventing development of atherosclerotic plaques.

**Other Contributing Issues**

**Pregnancy**

Women who gain excessive weight during pregnancy have an increased risk of complications of pregnancy and babies with a greater chance of developing obesity. In addition, excessive weight gain increases the risk of postpartum obesity, with the failure to lose weight after gestation predictive of a higher likelihood of overweight or obesity to follow. At present, however, targeted interventions to reduce the amount of weight gain during pregnancy have been variably successful. To improve outcomes of pregnancy for both the mother and baby and reduce the risk of excessive weight gain to follow, revised gestational weight gain guidelines have been published recently by the Institute of Medicine. For women whose BMI is <18.5 kg/m² before pregnancy, a weight gain of 13 to 18 kg during pregnancy is recommended; for women whose BMI is 18.5 to 24.9 kg/m² prepregnancy, 11 to 16 kg of weight gain should ensue; women who are overweight with a BMI of 25 to 29.9 kg/m² should gain 7 to 11 kg; for obese women whose BMI is ³0 kg/m², only a weight gain of 5 to 9 kg is recommended. In the environment of increasing overweight/obesity that currently exists, however, it remains doubtful that these recommendations will be immediately successful.

**Aging**

In general, aging is associated with a plateau in body weight or even modest weight loss in the elderly. This loss in part relates to unintentional weight loss as an indicator of undiagnosed disease. Even if weight is stabilized, however, body composition changes unfavorably with an increase in fat mass, including intra-abdominal fat, and loss in lean tissue. These changes in body composition result in limitations of BMI as a surrogate marker of adiposity. With the decrease in lean body mass with aging, a reduction in basal/resting metabolic rate is expected. However, with aging there appears to be a protective effect of higher body weight on survival. This obesity paradox in the overweight/obese elderly is noteworthy in that despite an increased risk of CVD, there is decreased mortality from related diseases. Thus, unless obesity-related comorbidities that are known to affect the quality of life and/or limit life expectancy exist, weight loss is not recommended. Nevertheless, there is no evidence that overfeeding in the elderly will increase survival.

**Cost**

The costs, both direct and indirect, of overweight and obesity are substantial and have been reviewed recently elsewhere. The direct cost of medical care for overweight/obesity is 5% to 10% of healthcare spending in the United States. Obese individuals have direct medical costs ≈30% greater than normal-weight individuals. Overweight and obesity in younger adults are associated with significantly greater healthcare expenditures later in life. This is especially concerning for obese children, who already at a young age have greater direct medical costs. Waist circumference, a surrogate marker of abdominal obesity, further predicts healthcare costs beyond BMI. Obesity is also associated with significant increases in indirect costs such as...
Adaptations to Weight Loss

Obesity is a metabolic condition best described as a defense of an expanded adipose tissue mass. In a recent report from the Coronary Artery Risk Development in Young Adults (CARDIA) study, among the 1869 overweight/obese individuals without major disease in 1995, only 29% lost ≥5% between 1995 and 2000, and among those who lost weight, only 34% maintained at least 75% of their weight loss between 2000 and 2005. NHANES also examined weight data from 14,306 participants (aged 20 to 84 years) in the interval 1999–2006. Weight loss maintenance was defined, however, as weight loss maintained for a minimum of 1 year. Among US adults who had ever been overweight or obese, 36.6%, 17.3%, 8.5%, and 4.4% reported weight loss maintenance of only 5%, 10%, 15%, and 20% of their initial weight loss, respectively. This effect was more evident among adults aged 75 to 84 years versus those aged 20 to 34 years, in non-Hispanic whites versus Hispanics, and in women versus men. However, when NHANES and CARDIA are compared, the interval of 5 years versus 1 year is critically important. Overall, <10% of obese subjects are successful at maintaining weight loss for ≥5 years, and the percentage is perhaps far lower because studies in which weight loss was not maintained may be less likely to be reported. Thus, the case for prevention can be more than made and needs to be the focus of everyone, including patients, their families, friends, workplace, primary care physician, subspecialist, third-party payers, school systems, local communities, and beyond.

Brief Review of Energy Balance Regulation

Energy balance is defined as a homeostatic yet dynamic environment wherein energy intake (food) matches energy expenditure. Energy expenditure has 3 basic components: basal/resting metabolic rate, energy expended in the form of physical activity, and thermogenesis (Figure 2). All components of energy expenditure can be measured in humans with a whole room calorimeter (almost always indirect calorimetry) and/or doubly labeled water. In general, the majority of energy expenditure is accounted for by the basal/resting metabolic rate (55% to 70%), with the smallest contribution from thermogenesis (5% to 10%) and the most variable form from physical activity (20% to 40%).

Adaptations to Weight-Reduced State

Changes in Energy Expenditure

In general, weight loss is associated with a reduction in total energy expenditure, including reductions in basal/resting metabolic rate and nonresting energy expenditure (Table 1). Reductions in resting metabolic rate best reflect the expected

Table 1. Potential Adaptations to the Weight-Reduced State

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<td>Resting energy expenditure</td>
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<td>Nonexercise and planned activity, changes in efficiency</td>
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<td>Thermogenesis (digestion)</td>
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Changes in substrate metabolism

| Fat oxidation |

Homeostatic signals: changes in appetite-related hormones

Nonhomeostatic signals: motivation, reward, attention, behavior
absence of increases in physical activity. This is reflected by an increase in the respiratory quotient (RQ), a measure of relative carbohydrate versus fat oxidation. Moreover, a significant correlation has been found between RQ after weight reduction and the spontaneous rate of body weight gain after cessation of the period of low energy intake. Thus, if carbohydrate is preferentially oxidized and fat stored, the metabolic response to sustained weight reduction appears to compensate for the diminished energy storage to return body composition to the previous energy-replete state.

Increases in insulin sensitivity are expected after weight reduction and in part explain the increase in RQ. This increase in insulin sensitivity, however, is variably predictive of weight regain. Of added interest, in studies of reduced obese subjects, not only did insulin sensitivity improve but so did the ability of insulin to suppress fat oxidation; however, fasting leg RQ or skeletal muscle macronutrient oxidation did not change. Moreover, changes in the tissue-specific regulation of lipoprotein lipase, the lipid-partitioning triglyceride-rich lipoprotein hydrolytic enzyme, are also seen after weight loss. In reduced obese subjects, increases in upregulation of lipoprotein lipase by meals and insulin are seen in adipose tissue, with opposite effects in skeletal muscle.

Overall, these findings suggest that increases in lipid partitioning and the reduced capacity for fat oxidation contribute to the increase in RQ in the reduced obese state and the weight gain that so often follows. Importantly, changes in insulin sensitivity, substrate partitioning, and/or oxidative metabolism do not change the energetic value of the calorie. To regain the lost weight, energy intake must still exceed energy expenditure.

**Changes in Energy Intake**

Not only is the weight-reduced state associated with reduced energy expenditure, but it is also associated with an increased drive to eat and resultant increased energy intake. In fact, all aspects of the regulation of energy intake are affected by weight loss (Table 1). It is clear that appetite is enhanced with calorie-restricted weight loss, the mechanisms of which are likely due to a complex interaction between physiological or homeostatic signals and reward/motivation/behavior-related signals. Adiposity-related signals, such as leptin and insulin, are reduced with weight loss, resulting in enhanced hunger and reduced satiety signaling in the brain as well as reduced energy expenditure. Of note, changes in leptin with weight loss have been shown to be predictive of weight regain. Short-term gut-related peptides such as ghrelin, PYY, and GLP-1, which play important roles in the meal-to-meal signaling of appetite, are altered by weight loss, favoring increased food intake. Recently, neuroimaging studies have shown that the weight-reduced state is associated with enhanced signaling in the motivation, reward, attention, and behavioral centers of the brain and that these brain responses correspond to physiological signals such as leptin.

In summary, although caloric restriction results in short-term weight loss, it is extremely difficult for individuals to maintain this weight loss over the long term. Significant advances over the last couple of decades have been made toward understanding the complex mechanisms responsible for the regulation of energy balance. The weight-reduced state is clearly associated with a dysregulation of these mechanisms, resulting in a milieu promoting weight regain and thus being one of the major obstacles of “treating” obesity and reducing its comorbidities.

**Prevention of Overweight/Obesity**

Biological mechanisms that favor weight regain in the weight-reduced state, limited success with interventions to maintain weight loss, adverse effects of overweight that are amplified when overweight occurs during critical developmental periods, and adverse effects that increase with duration of overweight all argue for identifying strategies to achieve and maintain a healthy body weight beginning in utero and early childhood and continuing throughout the life course.

**How Much Behavior Change Is Needed to Achieve Healthier Weights?**

Permanent voluntary changes in body weight require permanent changes in behavior. The types of behavior change needed are similar regardless of whether the goal is to maintain weight loss or prevent excessive weight gain. However, the magnitude of behavior changes needed is greater if the goal is to achieve and maintain a significant weight loss versus to prevent the gradual weight gain experienced by many people today. The reasons for this have been explained in detail but relate to the manner in which energy balance is regulated. The overweight or obese state appears to be defended by both biological and behavioral compensatory mechanisms, so that negative energy balance and weight loss are opposed by a lowering of energy expenditure and an increased desire to eat. The greater the negative energy balance and weight loss, the greater is the compensation. Thus, maintaining the reduced obese state requires substantial and permanent behavior changes from the overweight or obese state. Lesser weight losses would require lesser behavior change to maintain. Prevention of weight gain would require the least permanent change in behavior because there does not need to be either substantial negative energy balance or weight loss, so that compensation would be small or nonexistent. Hill and colleagues have estimated that maintaining a 10% weight loss would require permanent changes in behavior (a combination of reduction in energy intake and increase in energy expenditure) equivalent to 190 to 200 kcal/d and that maintaining a 15% weight loss would require behavior change equaling >280 to 300 kcal/d. Although this would be the equivalent of eating 1 fewer chocolate or candy bar (200 to 300 kcal), drinking 16 to 25 fewer ounces of a soft drink, or taking an additional 4000 to 6000 steps per day, these types of changes have been shown to be difficult to sustain over long periods of time. Alternatively, it has been estimated that prevention of weight gain can be accomplished with much smaller changes in behavior such that most adults could prevent weight gain with behavior changes of as little as only 100 kcal/d. It has also been estimated that excessive weight gain in children can be prevented by small behavior changes. Even these small
changes are difficult to sustain over time, and substantial effort will be needed to use this as a population strategy. Prevention of excessive weight gain could be an effective long-term strategy to reduce obesity rates. This strategy could be applied to people of all ages and with all ranges of BMI. It is likely, for example, that preventing further weight gain in those who are already overweight or obese could help to prevent acceleration or development of many chronic diseases.

Our success in long-term obesity treatment is poor. We must devote efforts to improving treatment, and we should especially focus our research on weight loss maintenance more than weight loss. However, currently we have a much better chance of success with a strategy of preventing excessive weight gain. At worst, this will help our obesity rates to keep from rising, and at best, this could help us to reduce those rates gradually over time.

What Kind of Behavior Change Is Needed?
Any changes in body weight and body composition must be the result of an imbalance between energy intake and energy expenditure. One question that is often asked is whether changes in diet are more important than changes in physical activity for either producing weight loss or preventing weight gain. Because energy intake and energy expenditure are interrelated and because changes in either can affect changes in the other, this is probably not a useful question to ask. Furthermore, both diet and physical activity are important, independent of each other, for preventing CVD risk through numerous mechanisms such as insulin sensitivity, effects on lipids, and inflammation. For example, reductions in energy intake can produce substantial weight loss, even without any physical activity. However, increased physical activity seems to be critical for maintenance of weight loss, and energy restriction is difficult to maintain over the long-term. When it comes to prevention, the good news is that it is necessary neither to achieve dramatic energy restriction nor to become a marathon runner. The problem with both of these dramatic strategies is that they are extremely difficult to maintain over time. Prevention of weight gain can be achieved with physical activity behaviors such as walking, which can be incorporated into most daily routines and can be monitored with a step counter, and strategies for eating smarter, which produce small reductions in energy intake without producing the increased hunger that more dramatic food restriction produces.

How and Where Should Prevention of Overweight and Obesity Occur?
The rise of overweight and obesity over the last 4 decades has occurred too rapidly to be accounted for by changes in genetic susceptibility, and it appears to be a sentinel indicator of a larger phenomenon. Concurrent with the rise in childhood obesity has been an increase in other childhood conditions including asthma, attention deficit hyperactivity disorder, dental caries, and mental health issues. With this temporal correlation in emerging childhood chronic conditions, common etiologies have been identified. For example, increased time spent indoors is associated with both decreased physical activity and increased exposure to allergens and pollutants. The relatively rapid increase in chronic health conditions in childhood and the similar temporal trend across many communities, especially disadvantaged communities, point to the importance of societal-level influences that affect families, communities, and the life experience of children.

Reversing the obesity epidemic will therefore require an ecological approach that not only includes individual and population-based behavioral interventions but also addresses root causes at the societal level, such as widening economic disparities; parents working away from home who are stressed, with less time and social support to nurture children; and limited access to healthy food and activity-promoting environments. These root causes reduce the effectiveness of individual and population-based behavioral interventions to treat overweight and obesity because healthy lifestyle choices, the targets of most behavioral interventions, are difficult to achieve in many of the social and physical environments of families and communities today. These same root causes have direct effects on the onset of overweight and obesity and will have to be addressed to have an impact on primary prevention and the future incidence of overweight and obesity, especially in socially disadvantaged populations.

In 2008, the American Heart Association published an in-depth review of the literature and a rationale outlining the need for a comprehensive population-based approach to healthful eating, physical activity, and energy balance for obesity prevention. Environmental and policy approaches were differentiated from clinical prevention and obesity treatment, and the ecological model was used to highlight the multiple layers of influence on eating and physical activity (eg, individual, interpersonal, organizational, environmental, policy) across multiple societal sectors (eg, government, industry, communities, schools, home). In 2009, the Centers for Disease Control and Prevention and the Institute of Medicine issued recommendations for obesity prevention strategies for communities and local governments, respectively. In the absence of definitive evidence, available data and explicit criteria were used to prioritize community strategies. Over the last decade, increased understanding of the complex etiology of chronic conditions like obesity and the diverse contextual factors that influence behavior and intervention effectiveness in community settings has highlighted the need to look beyond traditional research paradigms to establish generalizable evidence and to increase the capacity of community decision makers to employ evidence-based processes using the best available evidence while factoring in local evidence on needs and context.

To address the need to act in an informed way even when there are gaps in the evidence, an Institute of Medicine committee recently published a framework, the L.E.A.D. Framework (Locate evidence, Evaluate evidence, Assemble evidence, and inform Decisions), to guide evidence-informed decision making on obesity prevention strategies by a broad community of decision makers (eg, policy makers, public health professionals), as well as researchers working to fill the evidence gaps on questions that will have program and policy relevance. L.E.A.D. is designed to help to identify the type of evidence that is needed and to clarify needed changes in current approaches for creating and using evi-
The Health Professionals’ Role

The important role of the healthcare provider, especially the physician, in the prevention of obesity is a hypothesis yet to be formally tested. The hypothesis is that if physicians had the necessary time, expertise, and reimbursement to practice effective preventive medicine, overweight- and obesity-related outcomes would be favorably influenced. Talking to patients about their lifestyle is worthwhile for 3 reasons. First, the patient will understand that you, as their physician, believe that a healthy lifestyle is important. Second, an interview about current lifestyle habits opens the door to a productive discussion about ways to modify these habits. Third, patients may be more responsive to lifestyle modifications when the advice comes from their physician or healthcare provider. In fact, obese patients want their primary care physicians to address their weight problems, yet among patients gaining >3 pounds per year, physicians documented weight gain as a problem in only 10%. As stated in an American Heart Association presidential address, a 3-minute interview by the physician may be an important approach to demonstrate interest in and the importance of lifestyle in CVD prevention, including weight loss if needed and/or prevention of undesired weight gain. Of note, the American College of Preventive Medicine recently addressed the need to guide physicians in counseling children and adolescents to prevent overweight and obesity. Although data have been sufficiently lacking to permit the development of formal recommendations by the US Preventive Services Task Force in this area, recommendations have been provided by numerous public health and physician organizations. Finally, the lack of education of even young physicians mandates that training programs need to step up to the task of promoting effective clinical obesity prevention.

Behavior Change at Individual and Population Levels

Prevention of obesity can occur at individual or population levels. Although targeted behavioral changes may be similar at these 2 levels, the way in which these changes are achieved may be difficult. Achieving individual goals for behavioral change will require learning how to manage better within an environment and culture where overeating and being sedentary are easy and acceptable. It will require the patient to make good choices when these choices are difficult and are not being chosen by many people around the patient. Alternatively, a population strategy to preventing weight gain might address how to change the environment to one in which the healthier option is an easier option and how to change the culture to that in which healthy eating and active living are the socially desirable choices. Neither strategy will be easy, and the 2 strategies are not mutually exclusive. We can certainly begin to teach people how to manage better within the current environment and culture while we are making efforts to change it.

Summary and Conclusions

The majority of US adults and a significant number of children are overweight or obese. This epidemic of obesity is
associated with serious comorbidities, increased mortality, reduced quality of life, and a significant economic burden. Although short-term weight loss is achievable in most and is associated with improvements in comorbidities, there is limited success with interventions to maintain the long-term weight loss that is necessary. This limited success is primarily due to biological mechanisms that clearly favor weight regain in the weight-reduced state. For these reasons, strategies to achieve and maintain a healthy body weight throughout life to prevent overweight and obesity are critical. Prevention of weight gain is also more realistic from a behavioral change perspective, requiring fewer changes than those needed for weight loss and weight loss maintenance. Even these smaller changes are difficult to sustain over time, and substantial effort will be needed. The prevention of overweight/obesity will therefore require approaches that not only include individual and population-based behavioral interventions, but also address root causes at the societal level.

Disclosures

None.

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