AHA Scientific Statement

Population-Based Prevention of Obesity

The Need for Comprehensive Promotion of Healthful Eating, Physical Activity, and Energy Balance

A Scientific Statement From American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (Formerly the Expert Panel on Population and Prevention Science)

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***Abstract*—**Obesity is a major influence on the development and course of cardiovascular diseases and affects physical and social functioning and quality of life. The importance of effective interventions to reduce obesity and related health risks has increased in recent decades because the number of adults and children who are obese has reached epidemic proportions. To prevent the development of overweight and obesity throughout the life course, population-based strategies that improve social and physical environmental contexts for healthful eating and physical activity are essential. Population-based approaches to obesity prevention are complementary to clinical preventive strategies and also to treatment programs for those who are already obese. This American Heart Association scientific statement aims: 1) to raise awareness of the importance of undertaking population-based initiatives specifically geared to the prevention of excess weight gain in adults and children; 2) to describe considerations for undertaking obesity prevention overall and in key risk subgroups; 3) to differentiate environmental and policy approaches to obesity prevention from those used in clinical prevention and obesity treatment; 4) to identify potential targets of environmental and policy change using an ecological model that includes multiple layers of influences on eating and physical activity across multiple societal sectors; and 5) to highlight the spectrum of potentially relevant interventions and the nature of evidence needed to inform population-based approaches. The evidence-based experience for population-wide approaches to obesity prevention is highlighted. **(*Circulation*. 2008;118:428-464.)**

Key Words: AHA Scientific Statement ■ obesity ■ overweight ■ prevention ■ population-based ■ policy strategies ■ environmental strategies

Obesity is a major influence on the development of cardiovascular disease (CVD) and affects physical and social functioning and quality of life.[1,2] The proportion of adults and children who are obese has reached epidemic proportions, moving steadily away from the Healthy People 2010 goals of 15% prevalence of obesity in adults and 5% prevalence in children.[3–5] These goals may be beyond our reach for several decades to come (Figures 1 and 2).

The obesity epidemic is a major concern for the health of populations in the United States and many other na-

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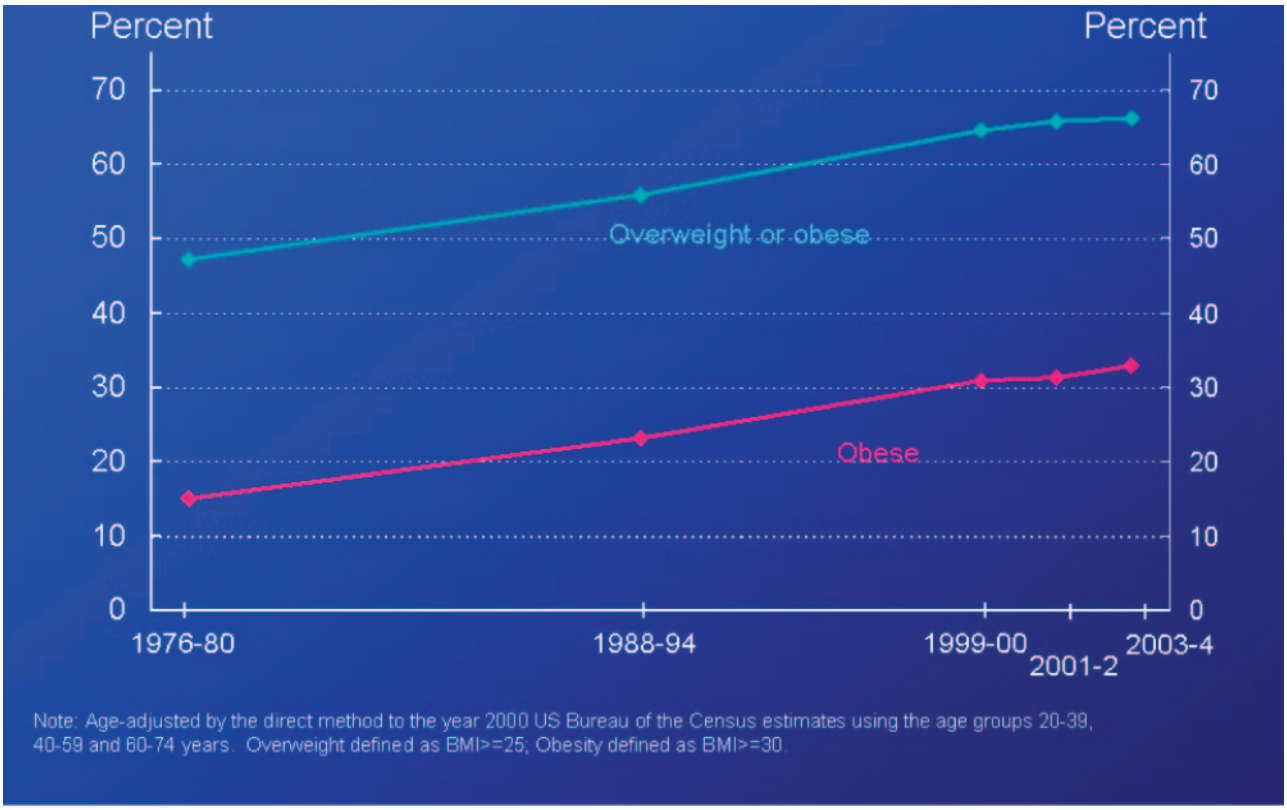

Figure 1. Trends in adult overweight and obesity ages 20 to 74 years. Source: National Center for Health Statistics.[4]

tions.[6–10] Based on data from the 2003–2004 US National Health and Nutrition Examination Survey (NHANES), approximately 66 million American adults (30 million men and 36 million women) are obese and an additional 74 million (42 million men and 32 million women) are overweight. Among American children 6 to 11 years of age, an estimated 4.2 million (2.3 million boys and 1.9 million girls) are overweight; among American adolescents 12 to 19 years of age, 5.7 million (3.1 million boys and 2.6 million girls) are overweight.[11] Assuming that the same trends continue, by 2015 2 in every 5 adults and 1 in every 4 children in the United States will be obese.[12] Obesity prevalence is also rising in countries throughout the world, reaching 20% to 30% in some European countries and 70% in Polynesia (International Obesity Task Force). According to the World Health Organization, the number of overweight and obese people worldwide will increase to 1.5 billion by 2015 if current trends continue.[13] Clearly, overweight and obesity place a large public health burden on society.

The prevalence of some obesity-related CVD risk factors (eg, elevated cholesterol and high blood pressure) decreased in the United States during the period from 1960 to 2000, despite increased obesity.[14] Nevertheless, the prevalence of these risk factors remained higher in overweight and obese than nonoverweight individuals, despite the concomitant trend of increased use of medications to treat these risk factors. Furthermore, the prevalence of diagnosed type 2 diabetes mellitus continued to increase concurrently with increases in obesity.[14] These trends underscore the importance of curbing the obesity epidemic. Control of type 2 diabetes requires a lifetime of medical care and usually drug therapy from the point of diagnosis, with the attendant financial costs and potential adverse effects on quality of life. Pharmacological control of high blood cholesterol and hypertension likewise requires lifelong medical therapy. Even with medical intervention, increased obesity may ultimately reverse gains made with respect to declines in related CVD risk factors. Thus, there is no room for complacency in dealing with this public health problem.

It is preferable to avoid, in the first place, the excess weight gain that leads to overweight and then obesity. Effective treatment of obese individuals can substantially reduce risk factors for CVD and improve disease management,[2,15] although some effects of long-standing obesity may not be reversible[16] or readily manageable. However, even those overweight people who are able to lose weight are often unable to maintain their weight at that level, and no clear guidance currently exists on definitive strategies to achieve long-term weight loss in the population at large.[15,17] The ability of weight loss to improve overall and CVD mortality has also not been clearly established, although a study to address this question is in progress.[18]

A major emphasis on obesity prevention is needed in the population at large[19,20] to prevent the development of obesity in those adults who are still in the normal weight range and in successive generations of children and adolescents during development. Treatment will continue to be of critical importance, but treatment alone cannot curb

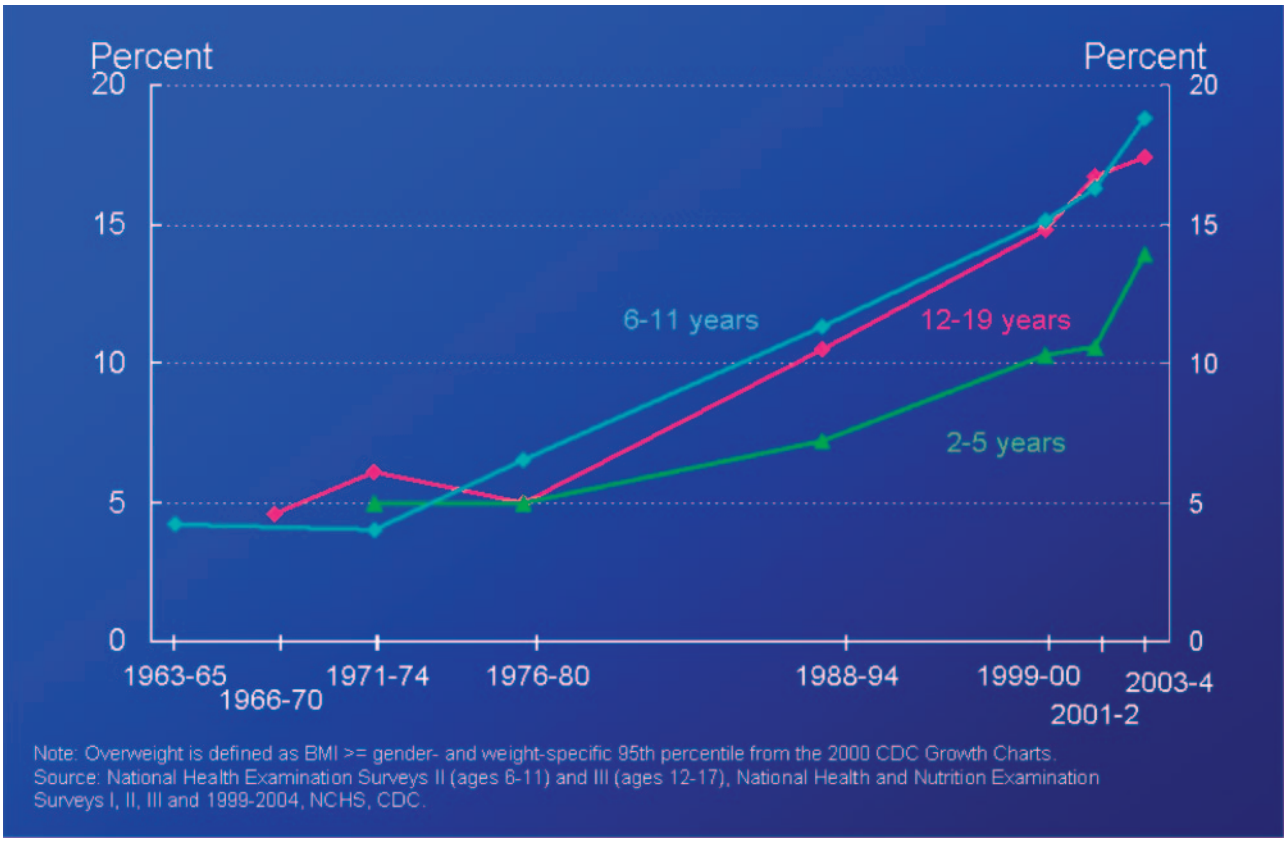

Figure 2. Trends in childhood overweight. Source: National Center for Health Statistics.[5]

the epidemic. Besides the limited long-term success of most obesity treatments, another factor is the limited ability to deliver enough treatment to enough people. We are already unable to deliver obesity treatment services to those who need such services, while the numbers needing treatment are rising. Health insurance seldom covers the cost of counseling for obesity, particularly the extended treatment of a year or more that is suggested to facilitate long-term weight loss.[17] The need for treatment is highest, relatively speaking, among low-income and ethnic minority populations,[21] who have a high burden of obesity, CVD, and stroke outcomes but less access to healthcare services.

This review provides a rationale for population-based obesity prevention efforts and research from a United States public health perspective. It is intended for a broad audience of health professionals, policy makers, and consumer advocates who may contribute to prevention efforts. As an overview of issues related to obesity prevention, this statement is complementary to published statements, workshop proceedings, and guidelines from the American Heart Association and other organizations that describe the effects of obesity and weight loss on CVD and its risk factors and provide guidance for obesity assessment and treatment and related lifestyle interventions[2,7,17,21–37] (Appendixes). This statement addresses the need to bring together, in one place, the various arguments for what needs to be done, and how, with respect to population-based initiatives to promote healthful eating, physical activity, and energy balance. A key goal is to motivate health professionals and others to contribute directly to broadly based obesity prevention efforts—"treating the community at large."[38] The relevance to clinicians is to describe population-based efforts needed to support and complement obesity prevention and treatment activities undertaken in day-to-day practice. Obesity prevention in the population at large is also highly relevant to obesity treatment in that it fosters social and environmental conditions that support healthful eating and active living. Such conditions are essential for all weight-control efforts.

The writing group objectives were as follows: 1) to raise awareness of the importance of undertaking population-based initiatives specifically geared to the prevention of excess weight gain in adults and children; 2) to describe considerations for undertaking obesity prevention overall and in key risk subgroups; 3) to differentiate environmental and policy approaches to obesity prevention from those used in clinical prevention and obesity treatment; 4) to identify potential targets of environmental and policy change using an ecological model that includes multiple layers of influences on eating and physical activity across multiple societal sectors; and 5) to highlight the spectrum of potentially relevant interventions and the nature of evidence needed to inform population-based approaches. The evidence reviewed includes primary sources, systematic reviews and expert reports, emphasizing articles published from June 1995 through May 2007. The population burden and health effects of obesity are described as background. Conceptual frameworks that can be used to describe and analyze prevention strategies are presented.

Background: Scope of the Problem

The Burden of Overweight and Obesity in the US Population

Adults

Overweight and obesity are generally defined using body mass index (BMI), a measure of weight relative to height that is closely correlated with total body fat content. BMI is calculated as weight in kilograms divided by height in meters squared or by dividing weight in pounds by height in inches squared and multiplying by a conversion factor of 703.[15] According to the National Heart, Lung, and Blood Institute, for adults, overweight is defined as a BMI of 25 to 29.9 kg/m^2; obesity, ≥30 kg/m^2; and extreme obesity, ≥40 kg/m^2.[15] Measures of waist circumference or waist-hip ratio are indicative of visceral adipose tissue, or intraabdominal fat, which may be more deleterious than overall overweight or obesity in some cases. Accordingly, the National Heart, Lung, and Blood Institute Clinical Guidelines recommend the use of waist circumference in addition to BMI in clinical screening of adults. High waist circumference, defined by cutoffs of >35 inches (>88 cm) for women and >40 inches (>102 cm) for men,[15] increases the level of risk associated with a given BMI level.

Data based on measured heights and weights, which are more reliable and valid than self-report, are available from NHANES.[9] In NHANES data for 2003–2004, an estimated 66.3% of US adults ≥20 years of age were either overweight or obese,[9] a relative increase of 18% from the previous estimate of 56% in NHANES III (1988–1994). The estimated prevalence of obesity alone was 32.3% in the 2003–2004 NHANES, a relative increase of 40.6% from the estimated 22.9% prevalence reported in NHANES III. The prevalence of extreme obesity (BMI ≥40) in the 2003–2004 NHANES was 4.8%. The prevalence of obesity generally increases across adult age groups. Previously observed gender differences in obesity prevalence, at the level of BMI 30 or above, have disappeared, with men "catching up" to women between 1999–2000 and 2003–2004. However, the prevalence of extreme obesity continues to be higher in women.

Long-term trends in overweight and obesity show notable increases (from 47.4% to 66.0%, or a relative increase of 39.2%) in the percent of persons who were either overweight or obese in the last quarter of the 20th century[39] (Figure 1). Most of the increase was attributable to increases in the prevalence of BMI ≥30 (obesity), whereas only minor increases occurred in the prevalence of BMI of 25 to 29.9 (overweight). The prevalence of obesity increased from 15.1% to 32.1% (a relative increase of 112.6%) for those aged 20 to 74 between the 1976–1980 NHANES and the 2001–2004 NHANES.

Children and Adolescents

For children and adolescents up to age 20 years, the term "overweight" rather than "obesity" is currently used by the Centers for Disease Control and Prevention (CDC) and generally defined as a BMI at or above the 95th percentile of sex-specific BMI-for-age values from the 2000 CDC growth charts.[40] In children and adolescents, the term "at risk of overweight" is the counterpart of overweight in adults, which the CDC defined as a BMI between the 85th and 95th percentiles. If recommendations of an expert panel convened by the American Medical Association, Health Services and Resources Administration and CDC[41] are implemented, the terminology for children will change to align with that for adults, ie, overweight and obesity. An estimated 17% of children and adolescents 2 to 19 years of age are overweight according to the 2003–2004 NHANES.[9] Among children 6 to 11 years of age, the percentage of those considered overweight increased from 4.2% to 18.8% (a 348% relative increase) between 1963–1965 and 2003–2004. Among adolescents 12 to 19 years of age, the percentage of those considered overweight increased from 4.6% to 17.4% (a 278% relative increase) between 1966–1970 (for adolescents 12 to 17 years of age) and 2003–2004[39] (Figure 2 for recent trends).

Waist circumference percentiles based on national data are available for white, African American, and Mexican American children.[42] However, whereas clinical guidelines for obesity assessment in adults include waist circumference, the above-referenced expert committee on child and adolescent overweight[41] did not find sufficient evidence or guidance to warrant a recommendation for routine clinical use of waist circumference in children at present.

Ethnic Disparities

In adults, NHANES data indicate consistent trends of higher obesity prevalence for non-Hispanic blacks and Mexican Americans compared with non-Hispanic whites but do not provide estimates for other ethnic minority populations. Drawing on other data sources—of which some rely on self-reported weight and height and, therefore, underestimate prevalence overall or in specific demographic groups[43,44]—obesity prevalence is also higher for American Indians and Alaska Natives, other Hispanic/Latino populations, Native Hawaiians, and Pacific Islanders in comparison with non-Hispanic whites, across the adult age spectrum.[15,25,45] Depending on the ethnic group, the prevalence of obesity is higher in females only or in both males and females.[9,46–52]

In the NHANES data, extreme obesity (BMI ≥40), which is associated with particularly high levels of CVD risk and total mortality,[53] affects approximately 15% of non-Hispanic black women compared with 6% and 8% of non-Hispanic white and Mexican American women and 2% to 5% or fewer in men in these ethnic groups.[9] Among immigrants in ethnic minority populations (Hispanic/Latino, Asian American, Pacific Islander, and possibly non-Hispanic blacks), obesity prevalence typically increases with a longer duration of US residence and, in some cases, approaches rates observed among US-born residents.[54–56]

Ethnic disparities in obesity prevalence apply to both BMI and waist circumference and are accompanied by disparities in obesity-related diseases.[21] However, there are ethnic differences in the interpretation of obesity indexes.[57–60] For example, the clinical consequences of obesity are higher for people of Asian descent at lower BMI and waist circumference cut points than for whites.[60,61] A report from the World Health Organization, Western Pacific Region,[62] suggested that overweight should be defined as a BMI of 23 kg/m^2 or

greater and obesity as a BMI of 25.0 kg/m^2 or greater in adults of Asian descent rather than using the respective cutoffs of BMIs of 25 and 30. A subsequent World Health Organization expert panel recognized that a range of plausible BMI cutoffs for overweight and obesity existed for these populations.[60] A more recent article[63] calls for revisions of BMI criteria for South Asians, Chinese, and Aboriginals.

Ethnic disparities in overweight prevalence are also observed in male or female children and adolescents, as in adults.[9,49,52,64–66] For example, in the NHANES data for 1999–2004, Mexican American male children and adolescents had a higher prevalence of overweight than non-Hispanic white male children and adolescents.[9] The prevalence of overweight among non-Hispanic black male children and adolescents was not materially different from that among non-Hispanic white male children and adolescents. The prevalence of overweight in Mexican American and non-Hispanic black female children and adolescents was higher than non-Hispanic while female children and adolescents. Rates of increase in overweight have been steepest in non-Hispanic black children compared with Mexican American and non-Hispanic white children and generally intermediate for Mexican American children.[67] Together with the higher prevalence of overweight in non-Hispanic black girls and Mexican American boys, these faster rates of increase indicate a particular need for preventive strategies addressed to these populations.

Socioeconomic Status and Geographic Variations

Population-based surveys show a higher prevalence of obesity in populations with lower socioeconomic status (SES), especially among white females,[68–71] although this relationship is less clear in more recent prevalence trends.[12] Patterns of SES differences in children and adolescents are complex and not consistent across age, gender, and ethnicity.[72–74] For example, in recent NHANES data, an inverse association of obesity prevalence with SES was observed in white girls, whereas higher SES was associated with higher obesity levels in African American girls.[74] Overall, SES differences in obesity are becoming less prominent in both adults and children.[12,74]

Geographic variation in obesity has been reported by state, as well as degree of urbanization. For example, interview data (ie, using self-reported height and weight data) from the 2005 Behavioral Risk Factor Surveillance System (BRFSS) survey indicate that the highest prevalence of obesity was seen in Louisiana, Mississippi, and West Virginia, whereas the lowest prevalence was seen in Colorado and Hawaii.[75] This may reflect socioeconomic differences among states.[76] Higher prevalence of obesity has been reported for rural populations compared with urban and suburban populations in the National Health Interview Survey.[77–79] For example, in 32 440 adult respondents to a 1998 National Health Interview Survey module,[78] obesity was more prevalent among adult residents of rural areas than residents of urban areas (20.4% versus 17.8%; $P=0.0002$) and this rural-urban difference was consistent across all ethnic groups. An analysis of 2000–2001 BRFSS data showed a similar pattern but with higher prevalence (23.0% and 20.5% in rural and urban areas, respectively). Rural-urban-suburban differences in obesity and health may also reflect socioeconomic differences, with rural areas being more characterized by local poverty and lack of resources,[77] at least in part.

Health Effects of Obesity

Adults

Obesity prevention in adults can potentially have a major impact in reducing morbidity and mortality that result from the chronic effects of excess body fatness.[8,80] The worldwide increase in obesity portends an increasing epidemic of diabetes and its serious consequences, including CVD. The American Heart Association (AHA) identified obesity as a major CVD risk factor in 1998.[81] As reviewed in a separate AHA scientific statement,[2] the impact of obesity in the pathophysiology of cardiovascular and pulmonary diseases and diabetes is well documented and has been recognized for decades.[2,82]

Weight gain after young adulthood is associated with an increased risk of CVD events and risk factors later in life independent of BMI levels. For example, in a cohort study of young adults, those who gained more than 5 lb over 15 years had unfavorable changes in CVD risk factors and higher incidence of metabolic syndrome and its components (waist, lipids, blood pressure), independent of initial BMI than those who had stable weight.[83,84] In a 20-year follow-up of middle-aged men, risks of major CVD events and type 2 diabetes mellitus were related to excess body weight at baseline (overweight and obesity) and to weight gain.[85] Also, in the Nurses Health Study, weight gain was associated with increased risk of all-cause, coronary heart disease, and CVD mortality at any level of initial BMI.[86] The association between obesity and several diseases begins when an individual is well within the "normal" weight range. For example, in a study of >7000 middle-aged men screened in British general practices and monitored for nearly 15 years, the lowest overall mortality rate was at a BMI of 22 to 27.9; however, for a combined end point of myocardial infarction, stroke, type 2 diabetes, and death, risk was lowest at a BMI of 20 to 23.9, and all major CVD risk factors increased progressively from a BMI of <20.[87]

As shown in Table 1, adverse health outcomes associated with obesity are not limited to CVD.[1,59,81–166] There is a large and growing body of evidence on the other myriad health effects of overweight and obesity, based on both animal and human studies, including mechanistic studies, epidemiological studies (eg, prospective cohort and case-control studies), and clinical trials. Of the adverse medical consequences of overweight in adults, diabetes is the most strongly linked with increasing BMI.[167] For example, insulin resistance, which is associated with obesity and is a risk factor for coronary heart disease, also appears to be related to liver disease and obstructive sleep apnea.[95] Obesity increases the risk for several types of cancer, including relatively common cancers, such as breast cancer in postmenopausal women[135] and prostate cancer.[96] As shown in Table 1, other relevant outcomes include osteoarthritis, gastroesophageal reflux disease, erectile dysfunction, and Alzheimer disease, as well as physical disability, employee absenteeism, impaired quality

Table 1. Adverse Outcomes for Which Obesity Increases Risk or Complications In Adulthood

Cardiovascular diseases, diabetes, and related conditions
Coronary heart disease (CHD)
Type 2 diabetes
CHD risk factors
Type 2 diabetes
Hypertension
Dyslipidemia
Inflammation
Hypercoagulability
Autonomic nervous system dysfunction
Heart failure
Stroke
Deep venous thrombosis
Pulmonary disease (including obesity hypoventilation syndrome, obstructive sleep apnea)
Other outcomes*
Absenteeism from work
Alzheimer's disease
Asthma
Cancer (including breast [postmenopausal], endometrial, esophageal, colorectal, kidney, and prostate)
Disability, physical
Erectile dysfunction
Fertility and pregnancy complications
Gallstones/cholecystitis
Gastroesophageal reflux disease
Gout
Healthcare costs
Impaired quality of life
Kidney stones
Liver (spectrum of nonalcoholic fatty liver disease)
Mortality
Obesity-related glomerulopathy
Osteoarthritis
Psychological disorders (eg, depression, aggressive behaviors)
Surgical complications

*Listed alphabetically. See text for relevant references.

of life, and increased healthcare costs. In older adults, obesity is associated with protection against hip fracture,[168] but this protective effect on bone status does not offset the extensive array of potential adverse effects on conditions that are common in the older population.

The extensive data indicating that weight loss can reverse or arrest the harmful effects of obesity[26] are further evidence of the causal link between obesity and disease. Lifestyle intervention studies have shown the effectiveness of weight loss in improving cardiovascular risk factors, including blood pressure,[156,169] insulin resistance and type 2 diabetes,[100,159] lipid disorders, and the metabolic syndrome,[26,170] in some cases lowering the incidence of hypertension or diabetes in a population at high risk for such diagnoses. Data from surgically treated obese subjects with larger weight losses than those usually observed in lifestyle trials have further confirmed the marked improvements in systolic (2 years only) and diastolic blood pressure, pulse pressure, and glucose, insulin, uric acid, triglycerides, high-density lipoprotein cholesterol, and total cholesterol levels, associated with weight loss after up to 10 years of follow-up.[153]

With respect to mortality, although controversies continue, many studies show clear, statistically significant, positive associations of BMI with CVD mortality, suggesting that obesity prevention can improve longevity. Most studies show an association between BMI in the obese range (≥30) and mortality.[92,106,107,140] The Look AHEAD trial[18] was initiated to specifically clarify the potential benefits of intentional weight loss on mortality. However, mortality data—even if conclusive with respect to the presence or absence of an association of obesity or weight loss with longevity—do not reflect the full spectrum of obesity-related health or quality-of-life issues (Table 1).

Children and Adolescents

Events that occur at the earliest stages of human development—even before birth—may have a profound influence on risk for obesity, diabetes, CVD, and other common adult conditions and are, therefore, potentially important focal points for preventive efforts.[171] Excess weight during childhood is associated with chronic disease morbidity and adverse psychosocial effects from childhood onward and, therefore, the lifetime duration of these diseases. Obesity during childhood also increases the risk of being obese as an adult, with the attendant implications for the above-described morbidity during adulthood.

Prenatal Determinants of Obesity and Related Health Risks

Both higher and lower birth weight are correlated to later obesity-related consequences.[172] Higher birth weight is associated with larger amounts of gestational weight gain and with gestational diabetes, 2 factors also implicated in childhood obesity.[173,174] Lower birth weight is consistently associated with central fat distribution, insulin resistance, the metabolic syndrome, type 2 diabetes, and ischemic CVD.[172] Moreover, the phenotype of lower birth weight followed by higher BMI in childhood or adulthood appears to confer the highest risk of these outcomes. This pattern holds, for example, for insulin resistance among 8-year-olds in India,[175] blood pressure among Filipino adolescents,[176] the metabolic syndrome among white and Mexican American adults,[177] and coronary heart disease among Welsh men and American female nurses.[178,179] Recent studies have found that excess weight gain during childhood and adolescence appears to explain these observations.[180,181] Whether accelerated weight gain in infancy confers excess risk for these adult outcomes is controversial.[182]

Other prenatal determinants of obesity-related outcomes may span the entire "fetal supply line" from maternal dietary intake to alterations in uteroplacental blood flow, placental function, and fetal metabolism, and they may or may not have any influence on birth weight. Maternal smoking during

Table 2. Adverse Outcomes for Which Obesity Increases Risk During Childhood

Metabolic
Type 2 diabetes mellitus
Metabolic syndrome
Orthopedic
Slipped capital femoral epiphysis
Blount's disease
Cardiovascular
Dyslipidemia
Hypertension
Left ventricular hypertrophy
Atherosclerosis
Psychological
Depression
Poor quality of life
Neurological
Pseudotumor cerebri
Hepatic
Nonalcoholic fatty liver disease
Nonalcoholic steatohepatitis
Pulmonary
Obstructive sleep apnea
Asthma (exacerbation)
Renal
Proteinuria

Source: Reference 27.

pregnancy is one potentially modifiable factor that appears to increase the risk for obesity and elevated blood pressure levels in offspring.[183] Smoking among women is rising in developing countries; reversal of this trend has the potential to help curb the emergence of obesity as a public health threat around the world.

Consequences of Overweight During Childhood

CVD effects of obesity during childhood are reviewed in detail in other AHA statements.[23,27] In addition to CVD morbidity, obesity can also lead to a number of other adverse health outcomes in childhood, including sleep apnea, gastroesophageal reflux, fatty liver, and orthopedic problems[27] (Table 2). Evidence relating to type 2 diabetes, asthma, and psychosocial problems associated with childhood overweight is highlighted below.

Given the strong relationship of obesity and diabetes in adults, the increase in childhood obesity is likely driving the concomitant increase in rates of type 2 diabetes among children. Once considered rare in children and adolescents, referrals for type 2 diabetes now rival those for type 1 diabetes in some centers.[184] In a multiethnic, population-based study of diabetes in youth, type 2 diabetes was more common than type 1 diabetes among 10- to 17-year-olds who were black, Asian, or American Indian and almost as common among Hispanics.[185] Among 12- to 19-year-olds in the 1999–2000 NHANES, 32.1% of overweight adolescents met national criteria for the metabolic syndrome.[186] In a referral group of >400 obese children and adolescents studied in detail, elevated BMI was associated with prevalence of the metabolic syndrome, which reached 50% in the most severely obese.[187] Also, in a prospective study of nearly 2400 9- and 10-year-old girls, increased waist circumference was a robust predictor of metabolic syndrome at age 18.[188]

Although the definition of metabolic syndrome itself is controversial, childhood overweight is also related to its individual components. For example, in the Bogalusa Heart Study,[189] overweight children were 12 times more likely than their leaner peers to have high levels of fasting insulin; the relative risk was greater for whites than blacks. The race difference may reflect that independent of body fatness, blacks appear to have lower insulin sensitivity than whites.[190] Higher BMI is also associated with higher blood pressure and abnormal lipid (including higher triglyceride) levels in children and adolescents.[189,191] Girls who were overweight at age 9 were 10 times more likely than normal-weight girls to have elevated systolic blood pressure, 6 times more likely to have low high-density lipoprotein levels, and 2 to 3 times more likely to have elevated diastolic blood pressure, triglycerides, and total and low-density lipoprotein cholesterol.[192]

The association of obesity with asthma is noteworthy because asthma is the most common chronic disease of childhood. In the late 20th century, the increase in asthma incidence paralleled that of obesity.[193–196] In addition to the observation that asthmatic children can become overweight because asthma limits their physical activity, prospective studies support the hypothesis that overweight children are more likely than their peers to develop asthma. Among 3792 children and adolescents 7 to 18 years of age who were assessed annually between 1993 and 1998, those who were overweight or obese were nearly twice as likely as their leaner peers to develop asthma.[197] Data are sparse on the relation of weight status in infancy and subsequent risk for asthma but would be of interest given the recent increase in overweight among the youngest children and because the peak age incidence of asthma occurs in the preschool and school years. The results of a preliminary study suggest that increased weight for length at 6 months predicts more wheezing by age 3 years.[198] The mechanisms by which excess weight can increase the risk for asthma include the presence of inflammatory cytokines produced by adipocytes and mechanical disruption of respiration.[199]

Psychosocial problems associated with overweight in children relate to self-concept, discrimination, and excessive weight concern and overeating disorders. Even in children as young as 5 years, a weight for height exceeding the 85th percentile has been associated with impaired self-concept (eg, higher-weight 5-year-old girls having a lower perception of their cognitive ability compared with girls with lower weight status).[200] Overweight children are more likely to be teased or bullied.[201,202] Overweight adolescents are more likely than their lean counterparts to be socially isolated.[203,204] Overweight children and youth are also more likely to suffer decreased self-esteem[205,206] and more likely to be extremely concerned with their weight and engage in bulimic behaviors.[207–210] It is possible that binge eating leads to, rather than

results from, excess weight gain, however.[211,212] Overweight children appear to have lower physical functioning and overall psychosocial health,[213,214] and in 1 study,[215] their health-related quality of life was similar to that of children and adolescents diagnosed with cancer.

Consequences of Childhood Overweight for Later Morbidity and Mortality

Children and adolescents who are overweight tend to remain so over time, particularly for older compared with younger children and if 1 or both parents are overweight.[216–220] Overweight adolescents may be as much as 20 times more likely than their leaner peers to be obese in early adulthood.[220] In younger children, parental obesity is a more potent risk factor than the child's own weight status in predicting whether the child will become an obese adult, whereas the opposite is true for adolescents.[220] The elevated risk of adult obesity is not limited to children who are frankly overweight. Two studies have demonstrated that children with a BMI in the 50th to 74th percentiles are substantially more likely than children with a BMI below the 50th percentile to become overweight or obese adults.[221,222] Therefore, obesity prevention must not be limited to children in the highest weight status categories.

At least 4 studies[223–226] demonstrate that adolescent overweight is associated with higher overall mortality. In these studies, males who had a higher BMI had an approximately 1.5 to 2 times greater risk of overall mortality during follow-up periods of approximately 30 to 70 years. Curiously, in 2 studies that monitored both women and men, adolescent females with a higher BMI did not have a substantially elevated risk of dying.[223,224] However, the more recent study shows that adolescent obesity strongly predicts increased mortality among women at midlife.[226]

At least 1 study indicates that weight in late adolescence is strongly related to risk of developing type 2 diabetes in adulthood. After adjusting for subsequent weight gain, 18-year-old female adolescents with a BMI >30 kg/m^2 were about 10 times more likely to develop diabetes than those with a BMI <22 kg/m^2.[227] Both males and females with an elevated BMI in late adolescence appear more likely than their leaner peers to develop CVD. Morrison et al[228] report 25-year follow-up data that showed an association between the presence of metabolic syndrome in children, 77% of whom had BMI at or greater than the 90th percentile, and CVD during adulthood. In the Caerphilly Prospective Study, Yarnell et al[229] studied 2335 middle-aged men who provided recalled information on weight and height at age 18. Males who were obese at age 18 were >2 times more likely than their leaner peers to have a coronary event within 14 years of joining the prospective study. Among the 508 men and women in the Harvard Growth Study,[223] those who had been overweight as adolescents were more likely than their peers to have a coronary event in adulthood or to die from CVD. Greater weight in late childhood or adolescence is also associated with higher blood pressure in adulthood.[222,230,231] Excess weight in adolescence may also increase the adult risk of conditions such as polycystic ovarian syndrome[232] or its concomitant ovulatory infertility[233] and ovarian cancer.[234,235] It is unclear whether these long-term effects of obesity in childhood stem from its longer duration or from the presence of obesity at certain critical periods for risk development.

The Case for Prevention

Overall Goals and Objectives

The goals of obesity prevention, broadly defined, include avoidance of weight gain to levels defined as overweight or obese and stabilization of weight in those who may already be overweight or obese or after weight loss.[236] Obesity prevention in childhood also has the goal of preventing obesity during adolescence and adulthood. Treatment of obese children to promote weight loss and to avoid tracking of obesity into adulthood is also a goal of obesity prevention. A focus on obesity prevention in childhood may seem particularly intuitive, because, as noted in the previous section, the process of developing obesity may begin in early life,[27,171] and arresting development of obesity in childhood has the greatest long-term payoff in years of healthy life. Preventing or reducing obesity in adulthood may be cost effective, based on the potential immediate benefits of avoiding the otherwise high prevalence of obesity-related comorbidities that develop during adulthood,[80] although the best way to determine the overall cost-effectiveness of interventions in obesity is as yet unclear.[237]

Preventive strategies for adults may include the promotion of small changes in eating and physical activity[238] or small initial weight losses to counteract expected annual weight gains or both.[239] Implicit in all obesity prevention goals are the related objectives of promoting healthful eating and activity patterns, and for children, normal growth and development, avoidance of adverse psychosocial or quality-of-life effects, and improvement in obesity-related health risk factors and outcomes.[7,19]

Achieving Individual Energy Balance to Prevent Excess Weight Gain

Prevention of excess weight gain relies on the maintenance of energy balance, whereby energy intake equals energy expenditure (in growing children and adolescents, energy expenditure plus energy for healthy weight gain) over the long term. For children and youth, this means growth and development along an acceptable weight trajectory.[240] For adults, this means maintaining a relatively stable weight across life stages, including the reproductive years, in contrast to the average progressive gain of 0.5 to 1 kg per year commonly observed in US adults.[241] A positive imbalance will increase energy storage, deposited as body fat and observed as weight gain. Although the concept is beguilingly simple, the physiological systems that regulate body weight through energy intake and expenditure mechanisms are complex, interactive, homeostatic, and still poorly understood.[8,242] Furthermore, the components of energy balance are not measured easily or with sufficient precision to be practical as a guide to help individuals maintain energy balance. Theoretically, a small persistent energy imbalance of 50 kcal per day could result in a 5-lb weight gain in 1 year (18 250 kcal per year divided by 3500 kcal/lb weight gain), all other things being equal. This scenario is an oversimplification, however, because the en-

ergy cost of 1 lb of weight gain depends on the fat composition of the added weight,[243] and all things do not remain equal,[244] because, for example, energy expenditure increases with higher caloric intake and weight gain. Nevertheless, the accumulation of a constant positive energy imbalance over the long term causes weight gain, and the great ease with which this accumulation occurs in people in the United States and many other countries causes the high prevalence of obesity.[8,19]

Although prevention and treatment of obesity both rely on the same principles of energy balance, the application of the principles is quite different. For treatment of obesity, a large reduction in caloric intake of about 500 to 1000 kcal per day, along with increased physical activity, can produce a loss of approximately 8% to 10% of body weight over the relatively short period of about 6 months.[15] Although the types of low-calorie diets that best promote weight loss are the subject of current investigations,[244,245] behaviors for weight loss are focused on caloric reduction: decreasing overall food intake, reducing portion sizes, substituting lower-calorie for higher-calorie foods, and increasing physical activity. Weight loss is best accomplished by participation in a behavioral program using self-monitoring, goal-setting, and problem-solving techniques.[15,246] Motivation levels may be high for appearance reasons or if adverse health consequences and quality-of-life impairments associated with obesity are readily perceived. Apparently, behaviors learned for weight loss are not sustained, however, because weight regain after weight loss is common.[247] Motivations and strategies to maintain weight after weight loss may differ substantially from those used to initiate weight loss.[248,249]

The application of energy balance principles toward prevention of weight gain and obesity is more subtle, and the results are less evident and less reinforcing than those for treatment of obesity.[250] The goal is to prevent a persistent small positive energy imbalance. To prevent obesity and weight gain, permanent lifestyle changes must be achieved and maintained over the long term and perhaps even intensified, because aging and environmental influences continue to create the conditions for positive energy imbalance. On the energy intake side of the equation, a healthy, low-energy–dense diet, along the lines of Dietary Guidelines for Americans[251] and the AHA Dietary Guidelines[30] is recommended: rich in fruits, vegetables, and whole grains and limited in high-fat and sweetened foods with high-energy density and low nutritional value. Important strategies are reading the calorie and serving-size information on nutrition labels, requesting simply prepared foods at food establishments, and preparing and consuming appropriate portion sizes (at restaurants, a strategy is to order or consume only half-portions). Because it is unknown directly whether caloric balance is being maintained, frequent weighing helps determine whether weight is stable.[252,253] Physical activity also plays a critical role in the prevention of weight gain and obesity.[254] Current physical activity guidelines to prevent weight gain are 60 minutes per day of at least moderate-intensity physical activity,[255] which is more than the amount recommended for general health and cardiovascular function.[256] Motivation is a particularly important issue to address. There are no dramatic improvements on an individual level, because the results are no change in weight or health outcomes in contrast to the weight loss and decrease in risk factors associated with weight loss.

Achieving Energy Balance in Populations

"Population-based" obesity prevention approaches are designed to produce large-scale changes in eating behaviors and levels of physical activity to stabilize the distribution of BMI levels around a mean level that minimizes the percent who become overweight and obese, without increasing prevalence at the underweight end of the continuum.[19] Population-level obesity prevention can and should be approached not as the promotion of widespread "dieting" but rather from the perspective of promoting healthful eating and physical activity patterns and a balance between the two. This approach requires modifications of factors that shape individual choices, as well as individual habits and preferences. There is ample evidence that individual eating and physical activity behaviors are responsive to the surrounding social and physical environmental contexts both for adults and children and, thus, amenable to public health prevention interventions.[7,19,257–263] Population-based approaches are also compatible with a broad range of public health goals. For example, improvement of eating and physical activity behaviors promotes healthy growth and development in childhood and adolescence, independently of weight, protects against certain types of respiratory, musculoskeletal, and liver diseases, as well as cancer, and improves cardiopulmonary fitness and overall health and wellness. Population-based prevention approaches reach populations through a variety of routes that extend beyond clinics and traditional health services and, when prevalence is high, at a lower cost per person compared with treatment approaches.[264]

Intake-related behaviors that have been linked to obesity include frequent consumption of meals at fast-food and other eating establishments,[265–267] consumption of large portions at home and at restaurants,[268,269] consumption of energy-dense foods, such as high-fat, low-fiber foods,[270–272] and intake of sweetened beverages.[273–276] These behaviors occur in an environment in which energy-dense food is abundant, relatively inexpensive, easy to obtain, and easy to eat with minimal preparation.

Low levels of physical activity are widespread in the United States[277] and have been associated with obesity and weight gain.[278–280] In some reports, television viewing and other sedentary activities have also been related to increased body weight,[281,282] although more of the evidence relates to children.[283,284] Deficient expenditure of energy could occur not only from sedentary lifestyles, but also from physiological changes that occur with aging. With increasing age, decreases in muscle mass, resting metabolic rate, and aerobic capacity occur.[285] Also, sedentary lifestyles may indirectly result in higher energy intakes because of less ability to regulate energy balance, for example,[286] and more time and opportunity to eat. Low levels of physical activity occur in the context of an automated and automobile-oriented environment that is conducive to a sedentary lifestyle.[287]

Community design and infrastructure characteristics (sometimes referred to as the "built environment," as differentiated from naturally occurring environmental factors) have become increasingly prominent in efforts to identify population-level determinants of obesity.[288] Evidence related to several of the commonly used variables in this category is highlighted below.

Urban "sprawl" is a geographic concept that has recently been studied in relation to risk of obesity. There is some disagreement about how to define sprawl,[289] but regardless of how sprawl is defined, most agree that sprawl results in large areas of low-population density that encourage and usually require residents to drive from home to work, stores, school, and recreation facilities[290] rather than to walk or use public transportation. Several studies have examined the relation between urban sprawl and risk of obesity. For example, Ewing and associates[291] constructed a County Sprawl Index that included population density measures and block size; larger scores indicated less sprawl. Health status data, including BMI, were derived from the BRFSS and were self-reported. After adjusting for gender, age, race, education, and smoking status, residents of counties characterized by greater sprawl walked less, weighed more, were more likely to be obese, and were more likely to have hypertension. Similarly, Lopez[289] constructed a 100-point metropolitan sprawl index using the US Census and calculated it for 330 US metropolitan areas that could be linked with data from the 2000 BRFSS. After controlling for age, gender, race, individual income, and education, a significant relation was found between the sprawl index and risk for overweight and obesity. Sprawl at the state level also has been found to increase risk for obesity.[292]

Land use mix and street connectivity are other geographic concepts that also have been linked to obesity. Sprawl is characterized by less diverse land use mix and less street connectivity. Giles-Corti and colleagues[293] found that both overweight and obese adults were more likely to live in neighborhoods that lacked adequate sidewalks and proximal places for physical activity and that overweight people were more than 4 times more likely to live near a highway. Participants with poor access to recreational facilities were 1.68 times more likely to be obese.

Neighborhoods can also be described in terms of "walkability." Saelens et al[294] characterized residents as living in high-walkable (single- and multiple-family residences) and low-walkable (single-family residences) neighborhoods with comparable SES using census data. They collected data on physical activity using accelerometers, weight status, and self-reported neighborhood perceptions. Residents of highly walkable neighborhoods walked significantly more (eg, a difference of 63 minutes per week of moderate to vigorous physical activity) than residents of low-walkable neighborhoods. In addition, residents of low-walkable neighborhoods tended to report higher average BMIs and higher rates of overweight than residents living in highly walkable neighborhoods.[294]

Frank and associates[295] investigated the impact of community design and physical activity on obesity in the Atlanta metropolitan area, characterizing neighborhoods as connected or disconnected (ie, high- and low-walkable, respectively) by using land-use mix data from the county tax assessor and the 2000 census within a Geographic Information System framework. Participant data within each neighborhood were drawn from a transportation and air-quality survey, which measured individual-level factors. After adjusting for the effects of age, level of education, and individual income, a significant relation was found between land-use mix and the prevalence of obesity, although this relationship was mediated by physical activity (ie, distance walked during a 2-day period). For instance, for each single quartile increase in land-use mix, there was a concomitant 12.2% reduction (odds ratio, 0.878; 95% confidence interval, 0.839 to 0.919) in the probability of obesity.

As discussed in the next section, community characteristics that influence obesity risk in low-income and minority communities may differ from those just described. For example, communities in inner city urban areas may be very "walkable" in the sense of connectivity but offer limited opportunities for physical activity because of safety issues, a lack of affordable recreational facilities and programs, and limited access to healthy foods because of the types of food stores and restaurants that are available.

Considerations for Prevention in Key Risk Subgroups

Whereas clinical preventive services are often characterized in terms of the stage of disease when the intervention occurs (ie, primary, secondary, and tertiary prevention), comprehensive public health approaches can be characterized on the basis of the population segment of interest. In the World Health Organization's obesity prevention framework,[8] whole-population approaches that target the entire community without prior screening of risk (although those at high risk are included) are termed "universal prevention." As will be discussed, whole-population approaches that are "passive" (ie, have their effects through environmental and policy changes) improve opportunities for healthful eating and physical activity without requiring deliberate actions by individuals and can be particularly useful in addressing inequities. Universal prevention approaches that rely only on changing individual behaviors directly through social marketing campaigns or community education may actually worsen disparities if they are only feasible for or attractive to relatively advantaged individuals. A combination of these types of approaches is needed.

"High-risk" approaches focus specifically on groups or individuals who are identified as being at high risk. When the focus is on groups at high risk, defined by demographic, health characteristics, or life stage, the term "selective prevention" is used in the World Health Organization framework. Focusing on specific individuals at high risk, including individuals with existing weight problems, is termed "targeted prevention." As will be discussed in a subsequent section, population approaches draw on tools and strategies from health promotion and public health to reach whole communities with educational or motivational messages or to foster environmental and policy changes that render physical and social contexts more conducive to weight control,

whereas high-risk approaches often resemble treatment programs because they involve screening and follow-up at the individual level and may occur in primary care or specialized treatment settings.

Obesity prevention is important throughout the life course and for both sexes, although prevention approaches and issues may differ according to gender. Body composition (higher percent body fat) and fat distribution (generally more gynoid and less abdominal fat distribution) among females may influence the health effects of a given BMI.[296,297] Men are an important population of interest because of their higher absolute risk for obesity-related diseases and lower likelihood of seeking treatment for obesity compared with women.[17] As discussed in this section, people with mental and physical disabilities are important subpopulations for focused efforts to prevent excess weight gain.

Childbearing-Age Women

Women of childbearing age in general and particularly women who are pregnant or postpartum are of particular interest for obesity prevention during adulthood. Excess pregnancy weight gain is particularly common among women who were overweight before pregnancy and having their first child.[298] Maternal prepregnancy BMI is a strong risk factor for gestational diabetes and is a reminder that the rise in rates of obesity among girls and women of childbearing age is producing a concomitant increase in rates of gestational diabetes, which in turn will likely lead to more obesity—and thus gestational diabetes—in the next generation. This vicious cycle may well fuel the obesity epidemic for decades to come, both in the developed and the developing worlds, particularly given that perpetuation of obesity in girls may ultimately affect the gestational environments of future generations. In addition, the potential for retention of excess weight gained during pregnancy greatly increases a woman's risk of later obesity-related diseases.

Gender-Related Differences in Obesity Prevention

Compared with men, women on average are more interested in food and nutrition, eat healthier diets than men, are more likely to do the household food shopping and preparation, and are more concerned about weight and familiar with dieting.[299–302] Nevertheless, obesity prevention may be more difficult for women than men. Women have lower caloric requirements than men on average and must, therefore, consume less food than men if they are to remain in energy balance.[255] This may be particularly disadvantageous for women when eating out, given that restaurant and take-home portion sizes have increased and are the same whether the customer is male or female or large or small. It appears that appetite controls in humans are more effective for avoiding hunger than preventing overeating. Experimental studies have demonstrated that the more food people are given, the more they are likely to eat.[271] Unwitting consumption of a few hundred extra calories is more detrimental to energy balance for women than men. Offsetting excess caloric intake by extra expenditure through physical activity is difficult because of the time it takes. For example, moderate activity, such as 15 minutes of walking, burns only 100 calories for an average size adult, whereas it is quite easy to consume an extra 200 or 300 calories in a much shorter time. In addition, the amount of calories expended is proportional to body size and the amount of lean tissue. A potential female advantage with respect to controlling food shopping and preparation may be offset by factors related to food preparation. Both women who work outside the home and busy homemakers may rely on convenience foods, prepared foods, or eating take out or restaurant foods, all of which are associated with higher calorie content. Depression, which is more common in women than in men,[303] has been associated with overeating and weight gain, both with respect to using food for comfort and because many antidepressants cause weight gain.[304,305] Also, stress has been associated with increased food intake, which could contribute to obesity.[306] In a recent survey, more women than men reported overeating under stress.[307]

In addition to the lower metabolism and energy expenditure associated with having higher percent body fat or a smaller body size, women are also at a disadvantage with respect to energy expenditure from a social and behavioral perspective. Leisure time or recreational activity levels are lower for females than males,[39] declining markedly in adolescence and particularly among African American girls.[308] Occupational activity levels are also lower for females.[309] Moreover, opportunities for physical activity in women are constrained by greater caregiving responsibilities and safety concerns that affect times and places available for physical activity.[310,311] Socially acceptable forms of physical activity may be fewer for women than men, particularly in some ethnic groups. Social concerns may include how exercise affects one's hairstyle or image of femininity,[312] as well as the possible displeasure of spouses or other household members, because exercise may be perceived as taking a woman away from family responsibilities.[311]

The greater concern about weight and dieting among women compared with men is well recognized and is apparently reflected in the tendency of women to participate in weight-loss programs.[17] The literature on treatment of obesity is dominated by studies in women to a much greater extent than can be explained by any gender differences in the prevalence of the problem. At any given time, nearly half of women, compared with about one third of men, are trying to lose weight, and women attempt weight loss at a lesser degree of overweight than men.[313] But dieting as such does not appear to be associated with success at preventing weight gain or obesity, perhaps because those who diet have the greatest difficulty controlling their weight or because dieting periods are interspersed with periods of overeating.

Social norms and attitudes about attractiveness differ for men and women. Slenderness has a much stronger importance for women, which appears to increase with upward mobility or high social position.[314] Social disapproval of obesity and excess weight in men is less strong, and the inverse gradient of obesity with SES, observed in women in many ethnic groups, is less predictable in men and is sometimes absent or reversed (eg, obesity may increase with increasing social position). Another reason for the higher

weight concern in women is retention of weight gained during pregnancy. This may be a major contributor to lifetime weight gain among women, particularly in ethnic groups such as African American women, for whom pregnancy-associated weight gain is more marked.[315,316]

The advantages and disadvantages for men in relation to obesity prevention are the opposite of those in women. Men may be less knowledgeable about or interested in healthful diets or calorie counting, and men's lower participation in weight-control programs than women may reflect and reinforce social norms that weight-control issues are not relevant to men and not important or as important for men. Health risks for which men are more susceptible (eg, risk of heart attack) or an interest in physical fitness may attract men to weight control. Physical activity expenditure among men may also be facilitated by their greater participation in sports or higher level of occupational activity.[309] Nevertheless, sedentary pastimes, such as watching television, are popular among men, as well as among women.[317]

Adults With Mental and Physical Disabilities

People with disabilities are included in the Healthy People 2010 focus on elimination of health disparities,[3] and those with either mental or physical disabilities constitute an important audience for obesity prevention. This diverse population has higher rates of overweight, obesity, and extreme obesity than those found in the general population.[318,319] A wide variety of disabilities have an impact on diet and physical activity, with the result that many different issues must be considered when designing obesity prevention strategies. Issues affecting overweight and obesity in the disabled vary greatly with the type of disability, including effects on physical condition and appetite, physical limitations that affect the ability to participate in regular physical activity, issues regarding responsibility for food decisions, and effects of prescription drugs on intake and activity. Because the issues are different for each type of disability, only a few examples are included here.

Physical limitations have obvious effects on the ability to perform physical activity,[320] which is important in the prevention of weight gain. Physical limitations can be part of a vicious cycle in which obesity contributes to the physical limitation (eg, low-back pain, osteoarthritis of the knee, foot injuries in diabetics), which in turn affects the person's ability or willingness to perform physical activity. Depression can also be a factor.

Adults with Down syndrome have a higher prevalence of overweight and obesity than adults in the general population.[321–323] Adults with Down syndrome who live at home have higher rates of overweight and obesity than those who live in group homes.[321–323] Hypotonia (weak muscle tone) may lead to reduced physical activity and may thus contribute to the high prevalence of overweight. Overweight and obesity are also common in persons with schizophrenia and schizoaffective disorder.[324] There is some evidence that the disability itself may contribute to overweight and obesity, and it is well known that several antipsychotic drugs cause substantial weight gain.[304] Limited attempts have been made at achieving weight loss among persons with mental disabilities. When cognitive impairment is present, interventions to change behavior can raise ethical issues, such as in Prader-Willi syndrome, in which the appetite is increased and the ability to understand health consequences is decreased.[325]

Children and Adolescents

General Issues

Fetal life, infancy, childhood, and adolescence are periods of tremendous physiological changes, which may explain why some periods may be critical in the establishment of not only behaviors, but also physiological processes. As stated previously, the possibility of physiological imprinting or programming early in life suggests that there may be sensitive or critical periods in childhood when an intervention will affect lifelong physiological processes that would be more difficult to change at a later age. Reduced fetal growth is thought to be associated with central fat distribution,[326] whereas weight gain in early infancy[327] and excessive weight gain in adolescence are associated with obesity in adulthood.[216]

Eating and physical activity behaviors learned during childhood may persist into adulthood,[328–332] and food and taste preferences may be established early in life.[333,334] Thus, interventions aimed at changing behavior during this period have the potential of establishing healthy behaviors that will continue over the individual's life span. Addressing gestational determinants of childhood obesity requires prevention of obesity in women of childbearing age. Apart from associations of lower birth weight with adverse cardiovascular outcomes that have garnered much recent attention, the well-established association of higher birth weight with higher BMI in childhood and adulthood should be emphasized.[172,335] Gestational diabetes, which leads to fetal hyperinsulinemia and increased fetal growth, may cause obesity and impaired glucose tolerance as the child becomes an adult.[173] Excessive weight gain by the mother during pregnancy is also associated with a higher BMI in the child at age 3.[174] Because women are increasingly beginning pregnancy at greater weights and because excessive weight gain during pregnancy has also probably increased during the past 1 to 2 decades,[336] avoiding excess pregnancy weight gain is another potential strategy to reduce the burden of obesity-related consequences in the next generation.

Obesity prevention in the pediatric ages involves specific circumstances and considerations. Interventions aimed at this population should be adapted to the neurodevelopmental characteristics of the target age and will require expertise in child development. Because developmental changes are rapid, most behavioral interventions likely need to be targeted at relatively narrow target age groups. Because children and adolescents are generally more sensitive than adults to outside influences (parents, media, and peers), prevention interventions based on changes in the child/adolescent's environment are particularly attractive for changing behavior in this age group to achieve population-based prevention of obesity.[7]

Another aspect of obesity prevention in children and adolescents is the potential setting of the interventions. Most children attend school or go to daycare centers, where they spend a large part of their waking time, have opportunities for physical activity, and eat 1 or 2 meals. Schools and daycare

centers are, therefore, ideal settings for interventions for obesity prevention in children. Schools have been used extensively[337–339] for such interventions, and there are some interventions in preschool, head start, or daycare settings.[340–343] Schools are also increasingly the setting for battles over politically charged decisions, such as exclusive contracts with beverage companies, regulation of advertising on school grounds, and community pressure on time and funding for physical education.[7] Child-specific settings, such as youth and recreational centers, have also been used for community-based interventions. Well-child visits to the primary care physician offer opportunities for pediatric obesity prevention. However, despite their dedication to preventive care, pediatric care providers are insufficiently trained to feel comfortable about implementing obesity prevention in the office[344] and are not appropriately compensated to implement obesity treatment.[345]

Children and Adolescents With Mental and Physical Disabilities

As in adults, children and adolescents with mental and physical disabilities are an important subpopulation of children who require special attention in relation to obesity prevention. Participation of children with disabilities in school and other social activities is lower than in the general population of children, and children with disabilities are more likely to be institutionalized. Such children are, therefore, less likely to be exposed to population-based obesity prevention strategies based in schools or community organizations.

Children with disabilities constitute a large but very heterogeneous population group with a variety of functional disabilities and medical impairments. In 1994, it was estimated that 12% of noninstitutionalized children and adolescents in the United States 5 to 17 years of age had some type of functional limitation, a percentage that corresponds to >6 million individuals.[346] These numbers have likely increased since 1994. Children with disabilities are overrepresented among US populations at increased risk for obesity, such as minorities and the poor.[3,346] Children with developmental disorders have a prevalence of overweight as high or higher than that of other children.[347] Many of these children with disabilities use medications that increase the risk of excessive weight gain, such as antiepileptics, antipsychotics, antidepressants, and steroids. However, although children with some types of disabilities and medical impairments are at increased risk for obesity (Down syndrome, brain cancer survivors), others are at decreased risk for obesity because of undernutrition (sickle cell anemia, cystic fibrosis). Even within the same medical impairment, for example, cerebral palsy, some patients can present with undernutrition, whereas others present with overnutrition.[348,349]

The disabilities affecting children and adolescents are heterogeneous in nature and severity, making it difficult to design a strategy that fits all children with disabilities. Because of limited mobility, communication, or learning abilities, many children and adolescents with disabilities will not be able to participate optimally in obesity prevention programs designed for the general population, and adapting obesity prevention strategies to a wide range of types and severity of disability will be a significant challenge. Existing initiatives, such as the Special Olympics, however, have been successful at increasing physical activity levels in children, adolescents, and adults with a wide range of disabilities and overcoming physical and societal barriers to sports. This could provide a model for prevention of obesity in this population.[350,351]

Ethnic Minority and Low-Income Populations

Several factors are thought to contribute to the ethnic disparities in obesity in ways that potentially influence the nature of preventive interventions that will be effective.[352–354] Historical and current exposure to social inequities may lead to adverse eating and physical activity patterns through various mechanisms,[355] including the possibility that overeating is used as a mechanism to cope with stress or that children are overfed as "insurance" against hunger.[356] Studies have indicated ethnic differences in consumption of calories and fat,[357–359] which to some extent is associated with high levels of consumption of fast foods[360–362] and in levels of sedentary behaviors.[358,362] A number of studies have shown that African American women are more likely to accept a larger ideal body image[357,363–367] than are women from other ethnic groups, although the ways in which body image influences weight control are uncertain. Also, the nature and impact of body image variables for ethnic groups other than African Americans are unclear. The diversity of ethnic subgroups within the major categories of Hispanics/Latinos, American Indians, and Pacific Islanders makes it inappropriate to state generalities for these groups as a whole. The issue of body image is relevant, at least theoretically, to motivation for weight control and prevention of obesity. Survey data suggest that African American women who are overweight are less likely than Hispanic or non-Hispanic white women to try to lose weight[313] and may not perceive themselves to be overweight.[368] Ethnic minority populations in general are underrepresented in the weight-control literature, although this may reflect the access (both location and eligibility requirements) of minority populations to the studies that have been conducted. Studies comparing weight loss in African Americans and whites in the same program indicate lower average weight loss among African Americans than whites, within sex.[369–371] This lesser level of success in weight-control programs could reflect social/environmental context issues, motivation, cultural appropriateness of the program, or other factors not yet identified. Again, whether this applies to other ethnic minority populations and also whether the results of treatment studies are informative for designing prevention strategies are unknown.

Recent attention has focused on aspects of the social contexts for obesity development that are less favorable for African Americans and other ethnic minority populations, including types of foods and retail food outlets available, range and accessibility of healthy food availability, opportunities for physical activity, and exposure to targeted marketing of less healthful foods.[352,372–375] Acculturation may play a significant role in the association of obesity with increased duration of US residence. In some studies conducted among Asian and Hispanic adolescents, acculturation to a US life-

style was shown to be associated with adoption of unhealthy behaviors in those born outside of the United States, such as sedentary behavior and poor dietary habits.[376,377] However, culture of origin and circumstances after immigration are important variables to consider. There may also be instances in which less acculturation is associated with a higher occurrence of overweight, as suggested in a study of Chinese American children.[378]

Access to supermarkets, which increases access to healthy foods, has been associated with better dietary quality[379] (eg, greater consumption of fruits and vegetables). Supermarket access is relatively lower in census tracts with a high proportion of African American residents. For example, Morland and colleagues[380] reported that 4 times as many supermarkets were located in non-Hispanic white neighborhoods than in African American neighborhoods. In addition, the ratio of supermarkets:residents was substantially higher in predominantly non-Hispanic white neighborhoods (1:3816 residents) than in African American neighborhoods (1:23 582 residents). Zenk et al[381] found that the most impoverished neighborhoods in Detroit with high proportions of African Americans were farther away (1.1 miles on average) from the nearest supermarket than neighborhoods that were less impoverished and had low proportions of African American residents. In contrast, access to fast-food restaurants may be greater in black or low-SES neighborhoods. Block and colleagues[360] showed that the density of fast-food restaurants was greatest in neighborhoods in which residents were predominantly African American and low income. Neighborhoods in which 80% of the residents were African American had 2.4 fast-food restaurants per square mile, whereas neighborhoods in which 80% of the residents were non-Hispanic white had only 1.5 fast-food restaurants per square mile.

In addition to issues related to types of available food stores, the relative costs of low- versus high-calorie foods is another potentially critical influence on efforts to prevent obesity in low-SES communities. As reviewed by Drewnowski,[382] several lines of evidence converge to suggest that the likelihood of being able to consume a healthful diet with calories appropriate to energy needs decreases with decreasing income. Limited income means limited money to spend on food and less flexibility in food spending as a percentage of available funds. The current price structure of foods is such that products high in fat and sugar and low in other nutrients are the least expensive, whereas fruits, vegetables, and whole-grain products, which are both lower in calories per unit weight and higher in essential nutrients, are relatively more expensive. Therefore, even where supermarkets are available, people with low incomes may purchase a relatively higher-calorie diet of less expensive, higher-calorie foods. High-fat and high-sugar foods are "energy dense" (eg, have more calories per unit weight) and are often highly palatable, making them relatively easy to overconsume. The perception that people with low incomes can afford a healthful, calorically appropriate diet is perpetuated by federal policy—specifically the "Thrifty Food Plan" that is used to calculate the Food Stamp Program benefits—that assumes a base diet of raw foods that will be cooked "from scratch."[383] However, from a practical perspective, few people, including recipients of federal nutrition assistance or income support, are spending sufficient time in food preparation to consume such a diet.[383]

Studies that suggest that low-SES areas negatively influence physical activity include 1 study by Yen and Kaplan[384] based on data from the Alameda County Study, a population-based longitudinal cohort study that began in 1965. Overall physical activity decreased between 1965 and 1974 but decreased significantly more in areas of poverty than in nonpoverty areas. Even after adjustment for numerous potential confounds, including age, gender, baseline physical activity score, smoking, individual income, education, BMI, alcohol consumption, and perceived health status, living in an area of poverty was significantly associated with a greater decrease in physical activity. Observed interactions indicated differences in effects according to race/ethnicity and individual income. There were no racial/ethnic differences (comparing blacks with all others) in the pattern of changes within poverty areas but a greater decrease in physical activity among blacks versus others in the nonpoverty areas, adjusting for potential confounders. A similar interaction was seen with individual income (ie, similar patterns within poverty areas but greater decreases among those with inadequate incomes in nonpoverty areas). This reduction in physical activity in poorer areas may be owing to the possibility that physical activity–friendly environments (ie, safe, affordable, well maintained, and appealing) are less common in low-SES areas. For example, Powell et al[373] studied 409 communities and found high-poverty areas had significantly fewer sports areas, parks, greenways, and bike paths than areas characterized by higher median household income and lower poverty rates.

Considerations for Taking Action

The motivational and behavioral issues that people encounter in achieving and maintaining energy balance combined with the fact that the many environmental context factors that influence energy balance are beyond the individual's control provide a compelling rationale for taking a public health, or population-wide, approach to prevention of obesity. This type of approach is comprehensive, including educational and motivational messages aimed at the entire population, as well as societal, worksite, government, public health, and health-care organizations promoting health consciousness, providing opportunities for physical activity, and making healthy foods accessible.[19,20,236] Such efforts make healthy eating and physically active lifestyles easier to adopt and more socially acceptable and self-reinforcing. The pillar of the rationale for a public health approach to obesity prevention lies in the overall strategy for preventive medicine as outlined by the late Geoffrey Rose.[264]

Determining Where to Intervene: Targets for Action in an Ecological Framework

An Institute of Medicine[385] committee concluded that approaches informed by an ecological model are critical for effectively addressing major public health challenges gener-

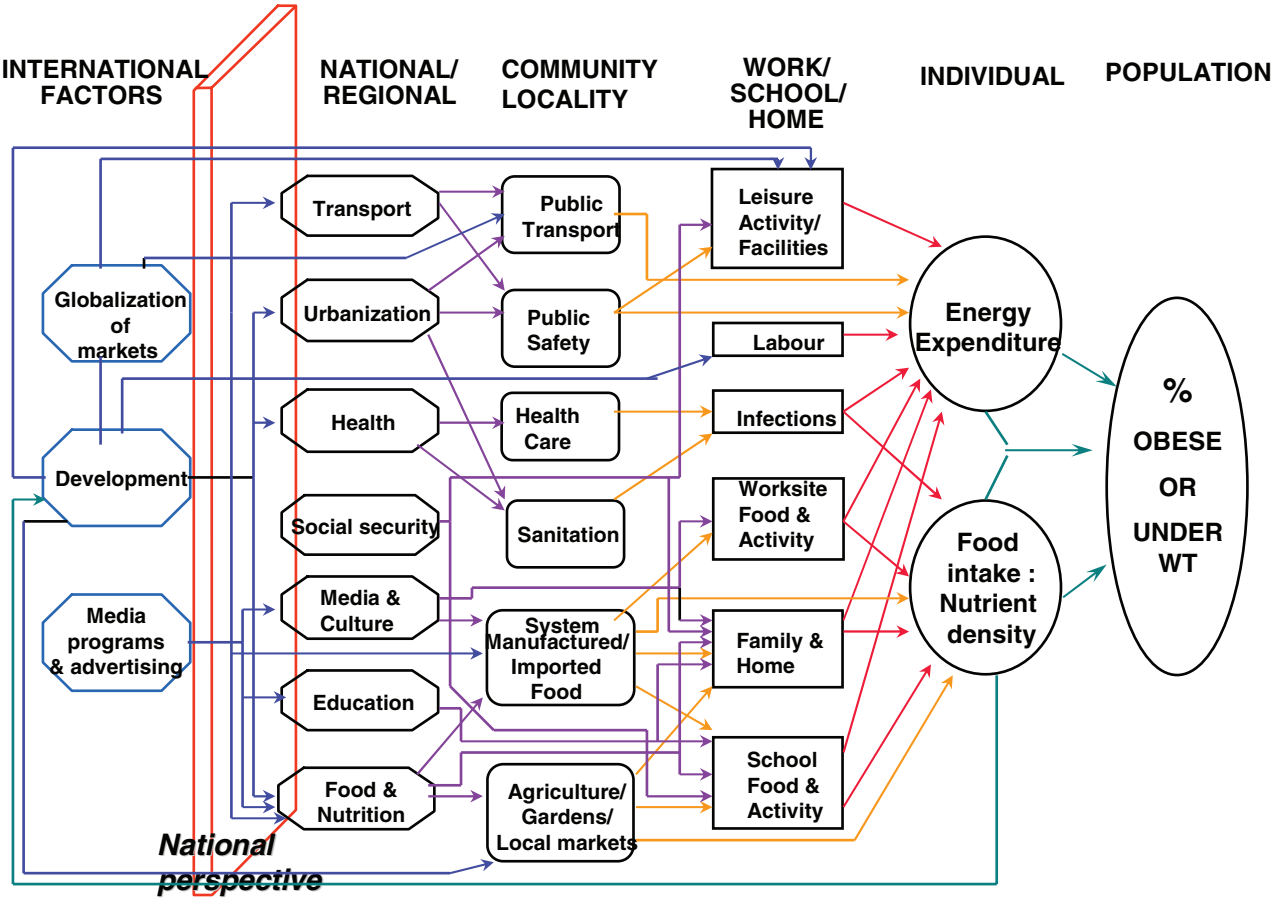

Figure 3. Societal policies and processes influencing the population prevalence of obesity.[19]

ally, and a subsequent Institute of Medicine committee used an ecological framework as the basis for a comprehensive national action plan to address the epidemic of obesity in children and youth.[7] Ecological frameworks emphasize the importance of social, environment, and policy contexts as influences on individual behavior and the interactions and interdependence of influences across different levels extending from the individual to the society at large.

The need for a multilevel, multisectoral approach to population-based obesity prevention has been emphasized[8,19] and is illustrated in Figure 3. This "causal web" of societal-level influences on obesity provides a framework for conceptualizing the different sectors or processes from which they arise and act (eg, transportation, urbanization, commerce, social welfare, media and marketing, education, agriculture, food and nutrition, and health) and the different levels at which these factors operate to influence the contexts for food choices and activity patterns in the population at large (global, national, regional, and local, as well as immediate environments such as work, school, and home).[19] The arrows in Figure 3 indicate the complexity and interrelationships among processes and pathways emanating from different sectors.

Table 3, which is complementary to Figure 3, was adapted from an ecological framework developed by the Partnership to Promote Healthy Eating and Active Living.[257] The listings in columns 1 to 3 give examples of specific categories of factors that might provide leverage points and settings for interventions in various sectors and settings with a goal of shifting influences in a direction less conducive to chronic positive energy imbalance. Together with Figure 3, these listings illustrate that some influences that relate to obesity may require action through national and international channels (eg, those related to the food industry), whereas others can be influenced by policies and practices that are controlled by state or regional authorities, at the city or neighborhood level, or in schools and workplaces. The other 4 columns in Table 3 reflect the societal and individual response variables that will affect the feasibility and effectiveness of obesity prevention initiatives. Many of these variables are reflected in the earlier described considerations for prevention in key risk subgroups. A longitudinal analysis of patterns of weight gain among members of social networks, including unrelated individuals and spouses, as well as family members who were genetically related, underscores the potentially powerful influence of social relationships in transmission of environmental risks of obesity.[386]

Figure 3 illustrates the complexity of the social and environmental contexts that produce the greatest challenge for obesity prevention. Implicit in the causal web (Figure 3) are processes and pathways that are fundamental to the social fabric and to day-to-day lifestyles. The number and types of potential stakeholders and vested interests potentially af-

Table 3. Influences on Physical Activity and Eating Behavior in Sectors and Settings: Ecological Layers From Macrosocietal to Individual Level

Focal Points and Settings for Interventions			Practical, Social, and Personal Influences on Intervention Effectiveness			
Distal Leverage Points	Proximal Leverage Points	Behavioral Settings	Enablers of Choice	Social	Ethnic/ Cultural	Individual
Architecture and building codes	Community	Community activity providers	Accessibility	Educational attainment	Beliefs	Genetics
Education system	Developers	Day care	Convenience	Interpersonal relationships	Ethnic identities	Hierarchy of needs
Entertainment industry	Employer	Food stores	Cost	Life stage	Habits	Physiology
Exercise, physical activity, and sports industry	Family	Health club	Knowledge	Social roles	Life experience	Pleasure
Food industry	Food stores	Home	Safety	Socioeconomic status	Values	Self identities
Government	Healthcare providers	Local school	Seasonality			
Healthcare industry	Local government	Neighborhood	Situation or context physical and social			
Information industry	Nongovernmental organizations	Parks, recreation centers, senior centers	Social trends			
Labor-saving device industry	Nonprofit providers	Religious, community, and nongovernmental	Source of information			
Political advocacy/lobbying	Property owners	Restaurants	Time			
Recreation industry	Recreation facilities	Shopping malls				
Transportation system	Restaurants and food outlets	Vehicle of transport				
	School boards/ districts	Workplace				
	Shopping mall					

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fected by interventions in these sectors and channels are vast. Policy makers, industries, and consumers may not support making changes in these factors, even when they recognize the need for action on obesity, because of the structural nature of these factors and the perceived negative consequences for other outcomes, both commercial and personal. Also evident in this understanding of what is required for obesity prevention is that influences controlled by health professionals or health policy makers are only 1 type of influence and are not involved in many important pathways. Creating the multisectoral, multilevel, and interdisciplinary partnerships and initiatives that are needed to influence the many other sectors is one of the major challenges of obesity prevention.[20]

Increasing the emphasis on population approaches that go "upstream" to focus on environmental and policy change requires a shift in thinking for those trained in clinical or individually oriented interventions. It is difficult to know when one is being effective when taking action so far removed from the ultimate behavioral outcome of interest. Prevailing attitudes of health professionals and others may also argue against reducing the focus on individuals to change their behaviors. The "upstream-downstream" argument is often made by analogy to a situation in which a continuing number of people are struggling in the water downstream, about to drown. Going upstream to find out why people keep falling into the river (eg, a bridge might have collapsed) is as critical as working downstream to pull the people out of the river one at a time. This is not a dichotomy; the goal is both to save "those who are drowning" and to stop others from "falling in." The analogy is used to make the point that the clinical approaches in which we are so well trained and perhaps confident can never be sufficient to solve widespread population health problems unless broad-based population strategies are also applied. Moreover, upstream approaches are the most cost effective when problems are widespread because individualized screening and counseling are, by comparison, much more costly on a per capita basis. As noted previously, upstream approaches are also the most likely to level the playing field for socially disadvantaged populations whose options for healthy eating and physical activity patterns are the most limited, for a variety of reasons,[352,372] and who, because of limited resources and limited social capital or power, are more constrained by the available options than those with more advantages, who may be able to find ways to

work around constraints and create new options for themselves.[387,388]

How to Intervene: Determining What to Do and Whether It Works

Figure 3 and Table 3 describe potential targets for action—covering many different sectors, levels, and specific potential focal points. How to actually have an impact on these targets requires a more process-oriented perspective related to the design of specific intervention programs or community action initiatives. Useful insights for how to take action can be drawn from general public health and prevention models. For example, the "Spectrum of Prevention"[389] is useful for characterizing and differentiating interventions at all of the levels that may be needed to address obesity at the population level and how these levels interrelate. This framework, described in Table 4 and discussed below in relation to obesity, has 7 bands or levels that indicate different types of strategies for environmental and policy changes, as well as community mobilization and individual education directed to selected combinations of the intervention targets outlined above. Consistent with an ecological model, the complementarity of these different strategies should be emphasized. In particular, the more upstream strategies at the upper levels of the spectrum (influencing policy and legislation, mobilizing communities and neighborhoods, changing organizational practices, and fostering coalitions and networks) are important for enabling the effectiveness of those oriented to individuals. Table 4 also includes examples of activities at each level of the spectrum to promote increased physical activity, based on an initiative in California.[390] The following narrative, which is organized according to the 4 top bands in the spectrum (influencing policy and legislation, mobilizing neighborhoods and communities, changing organizational practices, and fostering coalitions and networks), provides further highlights of how obesity prevention might be approached at these more upstream levels. Guidance relevant to providers and individual education and counseling is referenced in Appendix 2.

Influencing Policy and Legislation

Initiatives to foster changes in policy and legislation may be undertaken at local, state, and federal levels with a focus on the relative availability or cost of high- versus low-calorie foods or on opportunities to be physically active. Formal or informal policy changes are core to upstream interventions in that they change behavioral options and can reach large numbers of people, regardless of individual health motivations. Food-related policy targets might include snack foods and sweetened beverages, for which the goal would be to decrease consumption, or fruits and vegetables or water, for which the goal would be increase consumption. Activity-related targets might include aspects of community design that are more or less conducive to traveling on foot or by bicycle, availability and cost of recreational facilities, automobile use and availability of public transportation, and factors related to safety (eg, rates of street crime, condition of playgrounds, traffic-related measures to create safe routes for children to walk or bike to school).[7]

Options for types of policies include taxation of snack foods, subsidy of fruits and vegetables, regulations requiring foods served or sold in schools to meet specified nutritional standards, restrictions on advertising high-calorie foods to children, nutrition labeling regulations, financial incentives to industry (eg, to encourage siting of supermarkets in inner city areas with limited food access), or requiring school physical education classes and health education. Taxation mechanisms may be targeted to raising funds to support prevention programs directly. Worksite policies might include providing time off or facilities and equipment for exercise, providing bike racks and showers for people who cycle to work, providing weight-control programs or covering the cost of such programs or of gym memberships. Policies can also address monitoring and surveillance of weight levels (eg, of school children). Receptivity to various types of policy solutions varies among individuals and communities. There may be concerns that some policies will disadvantage commercial interests, limit individual freedom of choice, or create or aggravate social inequities. For example, taxation to raise the price of certain high-calorie foods could be problematic for people with very low incomes who depend on having cheap sources of calories.

The Institute of Medicine committee to evaluate progress to prevent childhood obesity[355] identified 717 bills (of which 123 were passed) and 134 resolutions (of which 53% were passed) relevant to childhood obesity that had been introduced in the United States between 2003 and 2005. Bills with a high rate of passage were related to farmers' markets, walking and biking paths, establishing task forces or study groups, and model school policies and safe routes to school. None of the 74 bills related to taxes on sodas and snacks passed. Policies to protect children specifically may garner more support than those directed to the general population because the potential vulnerability to environmental factors is relatively easier to argue with respect to children than for adults.

Mobilizing Communities and Neighborhoods

This level of the Spectrum of Prevention emphasizes the importance of community engagement, contrasting the traditional medical model, with the provider expert at the center (which also characterizes many public health activities), with the additional need to involve communities directly in assessing needs and planning and taking actions to address identified problems.[389] Such engagement with community members helps to align priorities as viewed by community members with those identified by public health workers and increases the likelihood that resulting initiatives will generate community interest and follow through. Public health workers and academic research partners can support community-generated initiatives through technical assistance. Some obesity prevention research involves community-based participatory research.[391–393] There has been increasing rec-

Table 4. Components of the Comprehensive "Spectrum of Prevention" as Applied to Obesity Prevention

Prevention Strategy	Rationale	Examples Related to Increasing Physical Activity*
Influencing policy and legislation	Both formal and informal policies have the ability to affect large numbers of people by improving the environments in which they live and work, encouraging people to lead healthy lifestyles, and providing for consumer protections	Land use policy established for community gardens Stable funding for Indian Health Service clinics to promote physical activity and nutrition
Mobilizing neighborhoods and communities	Particularly in low-income communities confronting more urgent concerns of violence, drug use, unemployment, and the struggle to keep families together, engaging community members in developing agendas and priorities is essential	Mapping community assets related to physical activity options Assisting community residents in setting priorities relevant to physical activity Providing technical assistance to help community residents implement action plans related to physical activity
Changing organizational practices	Modifying the internal policies and practices of agencies and institutions can result in improved health and safety for staff of the organization, better services for clients, and a healthier community environment; advocacy for such changes can result in a broad impact on community health	Protocols for physician assessment, sliding fees, counseling, and referral Bilingual staff at YMCA Work site policies Walking trail signage Improve safety in parks Provide fitness programs in public housing
Fostering coalitions and networks	Coalitions and networks, composed of community organizations, policy makers, businesses, health providers, and community residents working together, can be powerful advocates for legislation and organizational change and provide an opportunity for joint planning, system-wide problem solving and collaborative policy development	Local project coalitions and advisory committees Local park and recreation departments Healthy Cities coalitions American College of Sports Medicine volunteers Local Governor's Council on Physical Fitness and Sports
Educating providers	Service providers within and outside the health system can encourage adoption of healthy behaviors, screen for health risks, contribute to community education, and advocate for policies and legislation	Training for physician screening and referrals Park and recreation staff training Community exercise leader training Curriculum at university
Promoting community education	Community education can reach the greatest number of individuals possible with health education messages and also build a critical mass of people who will become involved in improving community health. This includes the use of mass media to shape the public's understanding of health issues—termed "media advocacy"	Community walkathon Media campaign Work site programs Interdenominational sports leagues Community fitness event Community advocate training Community gardens Church and community bulletins
Strengthening individual knowledge and skills	This strategy involves working directly with clients in the home, community settings, or in clinics, providing health information to promote well-being among children, families, senior citizens, and other population groups. It also includes working with both youth and adults to build their capacity in areas such as media advocacy, community mobilizing, and working with policy makers to make positive changes in the health of their communities	Walking club orientation Exercise classes Education classes Field trips Handouts Outreach contacts Home visits/instructions Exercise demonstrations

Adapted from Rattray et al,[389] with permission.

*Examples, except for those related to "mobilizing communities," were taken directly from Reference 390.

ognition of the importance of community-based participatory research, particularly with respect to research to address health disparities. REACH 2010 (Racial and Ethnic Approaches to Community Health) projects funded by the CDC are examples of such efforts that have specifically mobilized community members through participatory research related to food access and broader issues related to obesity.[394] Resource inventories or assets mapping are useful tools in this approach.[395–397]

Changing Organizational Practices

Schools and child care facilities, workplaces, and primary care are important settings for implementation of policies and programmatic initiatives. Relevant policy or programs may involve specifying the nutrition composition or cost of foods served or sold in cafeterias, instituting requirements for physical education in schools, increasing the availability of physical activity options or the time available to take advantage of these options, implementing training programs to enable school teachers to provide nutrition or physical education, and providing financial support for programs and services related to weight control. The appeal of setting-based approaches of this type includes the ability to work with a "captive audience" and to also influence social norms within the setting, with possible transfer to behavior outside of the setting. For example, policies that foster integration of 10-minute physical activity breaks into the regular work day or school day appear to be feasible, well received, and associated with meaningful increases in physical activity and possibly improved performance. This approach may be sustainable given that the activity breaks can be led by regular staff or teachers.[398–400]

Of the possible setting-based interventions, the Task Force on Community Preventive Services has found sufficient evidence to recommend "multicomponent interventions aimed at diet, physical activity, and cognitive change" in worksite settings.[35] In this report, evidence was deemed insufficient to determine effectiveness of single component interventions in worksites or of school-based programs for children and adolescents, and reviews of evidence to support various types of healthcare system interventions and community-wide interventions were still pending. Key issues for intervening in specific settings relate to perceived or actual competition of the interventions with the mission or other priorities of the setting, fear of liability, resource issues, privacy issues, the potential for increasing discrimination against those with existing weight problems, or consumer dissatisfaction. For example, efforts to increase time spent in physical activity may compete with time needed for academic work. Efforts to change school food options may compete with the use of food sales to raise funds for other school activities, as well as be unpopular with students and parents, leading to other problems for school officials. Screening children for BMI levels is controversial owing to the potential for adverse psychosocial effects of identifying children as overweight or obese and also because it is meaningless without the ability to implement ameliorative interventions.[401] Workplace issues with respect to productivity, consumer acceptance, and the potential for discrimination are similar. Injuries associated with increased physical activity may be a liability concern. In workplaces, there is potential competition with time spent working, where productivity is at issue. Competition with time to address more pressing medical or social issues can be a deterrent to adding weight-related counseling to primary care settings. Although many primary care physicians and their patients may be very motivated to provide or receive such counseling, the length of typical visits is too short to allow this, and reimbursement for obesity-related counseling—if available at all—may be limited to people with established obesity-related comorbidities.

Table 5. Components of the American Heart Association–Clinton Foundation Alliance for a Healthier Generation Initiatives to Foster Childhood Obesity Prevention

Founders	American Heart Association and William J. Clinton Foundation
Co-Lead	Governor Arnold Schwarzenegger of California
Mission	To eliminate childhood obesity and to inspire all young people in the United States to develop lifelong, healthy habits
Goals	To stop the nationwide increase in childhood obesity by 2010 and to empower kids nationwide to make healthy lifestyle choices
	The Alliance is positively affecting the places that make a difference to a child's health: homes, schools, restaurants, doctor's offices, and the community
Programs	*Healthy Schools Program*
	Industry Initiative
	Kids' Movement
	Healthcare Initiative

Source: Reference 402.

The Alliance for a Healthier Generation, a joint initiative of the American Heart Association and the William J. Clinton Foundation,[402] is an increasingly prominent example of a comprehensive national-level obesity-prevention strategy that focuses on school settings. Components of this initiative are listed in Table 5. Indicative of the importance of this program in the national obesity prevention effort, the Robert Wood Johnson Foundation, which has a major commitment to reversing the epidemic of child and adolescent obesity, initially awarded $8 million in 2006 to support the first phase of the Healthy Schools Program. A year later, it announced the award of an additional $20 million to support expansion of the program that will focus on states with the highest obesity rates, as well as expand on-line support for schools nationwide. As shown in Table 5, the Alliance initiatives go beyond a school focus and address several other levels of the Spectrum of Prevention.

Fostering Coalitions and Networks

Community organizations or coalitions of community organizations or members who have a stake in obesity prevention

may undertake community action to raise awareness of a problem, identify potential solutions, and seek to implement these solutions through changes in policy and practice. Some coalitions have a single focus, while others take on a broader set of community priorities. Community members may mobilize spontaneously (eg, in response to a perceived crisis or intolerable situation). Community mobilization may also be initiated as a health-promotion strategy (eg, through efforts of a state or local public health agency, other health services provider, or a community-based organization with a relevant mandate). Researchers who can provide technical assistance and advice are often partners in these efforts. Broadly based, multisectoral efforts may be particularly effective. For example, the Consortium to Lower Childhood Obesity in Chicago Children (CLOCC) provides a rubric for pooling the efforts of hundreds of organizations representing a variety of entities with relevant interests.[403] CLOCC activities include training public school teachers in strategies to improve student nutrition and physical activity, community-wide health-promotion events, Web site development, and an initiative to foster walk-to-school programs. A School Nutrition Task Force in Philadelphia mobilized a successful effort to create healthier vending options in schools.[404]

Evidence-Based Experience

In contrast to the extensive database available on obesity treatment, research to identify specific interventions to prevent obesity is still at a relatively early stage.[7,20,236,405] Elements of promising strategies for obesity prevention can be identified, and there are many relevant efforts under way. These efforts include programs generated spontaneously in communities, as well as formal research or demonstration projects undertaken based on program logic and combinations of strategies that appear to be effective. However, it is difficult to identify what set of interventions will be effective in shifting the BMI distribution for a whole community (also see Research Challenges). Effective interventions will, separately, improve dietary intake and the level of physical activity, but in combination, they must not only improve dietary quality and energy output or fitness but must also result in the avoidance of positive energy imbalance. Effects must also be sustainable over time in that the risk of excess weight gain is ever present. As explained previously, the applicability to obesity prevention of the literature on obesity treatment may be limited, because the challenges of achieving energy balance are different for prevention from those for treatment at the individual level. In addition, social and environmental changes, although relevant to both prevention and treatment, are fundamental to obesity prevention.

Numerous systematic reviews have assessed available scientific evidence on obesity prevention. Results of selected reviews published during the last decade are shown in Table 6.[405–417] Two reviews focus on adults in primary care settings,[414,415] 2 focus on environmental and policy interventions,[412,413] and 2 cover all ages.[417,418] The remaining articles focus on children or school settings.[405–411] Almost all include studies both in the United States and abroad. Perhaps the most striking finding in Table 6 is the relatively small numbers of eligible studies for these reviews of obesity prevention, although the number is increasing. Searches sometimes identify thousands of possible articles, but the number ultimately reviewed and included is relatively small. This is owing in part to the inclusion and exclusion criteria applicable to many reviews (not all of these criteria are included in the Table 6 entries, for brevity; eg, requirements for controlled trials—either randomized or nonrandomized—inclusion of only completed trials, exclusions on the basis of a rating of poor quality, or exclusion of studies that did not provide a measurement of weight status or fatness). With respect to the findings on weight outcomes, the findings are encouraging in identifying many studies that were successful, although evidence of the ability of interventions to change average BMI levels is limited. The relatively limited breadth of studies identified, mainly school based and mainly individually oriented, indicates an urgent need to explore preventive interventions in other settings and at multiple levels upstream. Ongoing research may broaden the evidence base to some extent, but there is an overall impression that this critical area of research has far too little focus.

Research on how to implement effective environmental and policy change is a relatively new aspect to the field of obesity research, and appropriate measures and evaluation designs are still being developed. These measures and designs are needed not only for deliberate experimentation that involves environmental and policy changes, but also for the many spontaneous changes that are occurring in legislatures and communities on a day-to-day basis. Changes in school food and beverage vending policies are a prominent example of spontaneous changes that are being implemented with a limited empirical basis (ie, natural experiments). In addition, as noted above, evaluation of specific interventions is complicated by the fact that additive or synergistic effects of multiple interventions across different levels and sectors may be necessary to have an impact on behaviors related to energy balance and to see effects on weight.[411,418] This can be addressed in part by multilevel interventions or combinations of studies, but to date, these studies are few in number.[409,411] Another challenging and strongly debated issue is how study designs with the highest level of internal validity, randomized, controlled trials apply in that they may impose limitations on both the feasibility and relevance of testing obesity prevention approaches in naturalistic settings.[418,419]

An example of a promising multilevel intervention, evaluated with a nonrandomized, controlled trial design is the "Shape Up Somerville" study.[420] This study compared the effects of a comprehensive intervention, conducted in partnership with entities in the study communities, on physical activity and food options during the child's entire day on BMI z-scores 1 year after the initiation of the interventions. Participants were 1178 elementary school-aged children in all 30 schools in 3 participating communities: 10 schools in the intervention community; 10 and 5 schools in the 2 control communities, which received no intervention. An extra control group was used to ensure against the spontaneous

Table 6. Highlights of Selected Systematic Reviews of Intervention Studies Related to Obesity Prevention (Listed Alphabetically by First Author Within Year of Publication, Most Recent First)

Reference	Focus, Scope, and Key Inclusion Criteria	Eligible Studies Identified	Main Findings
Bluford et al[406]	**Preschool children** United States and international, published in 1966 through March 2005 Interventions to prevent or treat obesity in preschool children (ages 2 to <6 years) of at least 3 months' duration	7 studies Settings included schools, day care/Head Start programs, clinics, and home settings	Significant reductions in weight status or body fat were identified in 4 of the 7 studies, of which 3 sustained reductions 1 to 2 years after the program began 2 studies reported no change; the other study found no change in Latino or black children but an increase in weight status in white children
De Mattia et al[407]	**Children and adolescents** United States and international, published in 1966 to February 2005 Interventions to limit sedentary behaviors (recreational screen time but not homework or reading) in children or adolescents in natural settings (eg, at school or home or in a primary care setting	12 studies	All of the studies, including 6 that targeted clinic-based populations and 6 that were population based reduced sedentary behaviors (self-reported) and improved weight outcomes (measured)
Sharma[408]	**Children and adolescents** Only studies from countries outside of the United States, published in 1999–2005 School based interventions for obesity prevention in children; not all studies included measured weight outcomes; and not all had been completed	21 interventions, of which 17 were from elementary schools	Most studies focused on individual level approaches; 16 of the 21 interventions were delivered by existing teachers, often with additional training Measured weight or fatness variables were available in 11 studies, of which 6 showed improvements; all 3 completed studies that included parents improved measured weight outcomes
Doak et al[409]	**Children and adolescents** United States and international, published through August 2005 Interventions and programs to prevent obesity in children and adolescents, with measured weight or fatness outcomes	25 interventions	17 of the 25 interventions reported statistically significant improvements in obesity measures; estimation of effectiveness differed according to whether skinfold or BMI measures were used 5 studies found gender differences in effects and 1 study found differences by ethnicity No ideal age for intervention could be identified from these studies Physical education and reduction of television viewing were highlighted as examples of effective approaches One of the effective interventions was also associated with an increase in underweight prevalence
Flodmark et al[410]	**Children and adolescents** United States and international, published until 2004 Setting or population-based interventions (ie, in groups of children not specifically selected for being overweight or obese to prevent obesity of at least 12 months' duration; with measured weight or fatness outcomes Articles published until 2004 were added to a prior 2002 review; results of 5 other systematic reviews were also evaluated	24 studies in this review 39 total studies when including other reviews	8 studies reported significant positive results on measures of obesity, and 16 were neutral; none had negative results Considering these results together with those of 5 other systematic reviews yielded 39 studies of which 15 had positive results and the other 24 were neutral; no studies reported harmful effects on children Effective programs were relatively limited school-based programs that promoted a combination of healthful eating and increased physical activity

(*Continued*)

Table 6. Continued

Reference	Focus, Scope, and Key Inclusion Criteria	Eligible Studies Identified	Main Findings
Flynn et al[411]	**Children and adolescents** United States and international, including government reports and other published or unpublished sources identified apart from databases of published articles, 1982–2003 Accounts of programs that could shed light on best practices related to reduction of obesity and related chronic disease risk in children	147 programs were analyzed	No single program emerged as a model of best practice, although promising elements applicable to various populations and settings were identified More upstream and population-focused interventions are needed to balance the emphasis on individually oriented strategies There is a particular need for programs tailored to ethnic minority and new immigrant children and based in community or home settings
Health et al[412]	**Policy and environmental changes** United States and international, published through 2003 Studies of the effectiveness of urban design and land use and transport policies and practices for increasing physical activity; reviewed for the Guide to Community Preventive Services	12 studies on community-scale urban design, 6 studies on street-scale urban design; and 3 studies on transportation and travel policies and practices	Both community-scale and street-scale urban design and land use policies and practices were found effective in promoting physical activity, with evidence rated as "sufficient." The evidence to evaluate the effectiveness of travel and transport policies is insufficient. Also reported are the following additional findings of the Guide to Community Preventive Services with respect to physical activity promotion, based on prior systematic reviews: strong evidence for community-wide campaigns, individually adapted health behavior change, school based physical education, social support in community settings, and the enhancement of access to physical activity options combined with informational outreach activities There is sufficient evidence for point-of-decision prompts
Summerbell et al[405]	**Children and adolescents** United States and international, published in 1990 through February 2005 Interventions to prevent obesity, of at least 12 weeks' duration, in randomized controlled or controlled trials	10 long-term (at least 12 months) and 12 short-term (12 weeks to 12 months)	Of the long-term studies that focused on both diet and physical activity, 5 studies found improvements in weight or fatness outcomes for both boys and girls and 1 found improvements for girls only; a long-term study of a multimedia intervention to improve physical activity was effective; studies that focused on nutrition education only were not effective Two of the short-term studies were effective in improving weight or fatness outcomes; both focused on physical activity; 2 others that focused on physical activity and 10 that focused on both physical activity and diet were not effective
Matson-Koffman et al[413]	**Policy and environmental changes** United States and international, published in 1970–2003 Policy or environmental interventions to promote physical activity or good nutrition, excluding studies of the built environment and media-only campaigns; included studies in whole communities, schools, worksites and restaurants, and healthcare settings	65 studies before 1990 and 64 studies between 1990 and 2003	Strongest evidence was found for - Promoting stair use - Improving access to place and options for physical activity - Improving school physical education - Implementing comprehensive worksite approaches - Increasing availability of nutritious foods - Information at point of food purchase - Systems for reminding healthcare providers to provide nutrition counseling

(*Continued*)

Table 6. Continued

Reference	Focus, Scope, and Key Inclusion Criteria	Eligible Studies Identified	Main Findings
Pignone et al[414]	**Adults** United States and international, published in 1966–2001 Trials of counseling of adults in primary care settings to promote a healthy diet, of at least 3 months' duration, with behavioral outcomes reported, excluding trials in people selected on the basis of overweight or obesity or a chronic disease; reviewed for the US Preventive Services Task Force	21 trials	Relatively modest improvements in self-reported dietary intakes of saturated fat, fruits and vegetables, and possibly dietary fiber in response to brief interventions using a variety of modalities; greater intensity was associated with better results but had less potential feasibility in these settings
Eden et al[415]	**Adults** United States and international, published in 1994 through March 2002 Trials in which counseling to improve physical activity was provided and some part of the intervention was performed by a primary care clinician (physician, nurse practitioner, nurse, or physician's assistant); reviewed for the US Preventive Services Task Force	8 trials; 5 other trials judged to be of poor quality were excluded	Limited support was found for the effectiveness of these interventions; 3 of the trials that included a usual care control group reported a significant improvement associated with the intervention; in 1 study, a written prescription was more effective than advice alone; another suggested that women may need more intensive counseling than men In the 1 study that reported harm, about 60% of all patients reported some type of musculoskeletal injury and some reported cardiovascular events that required hospitalization; however, no comparison data were available to estimate background rates
Hardeman et al[416]	**All ages** United States and International, published in 1966–1999 Published studies using any type of study design involving testing of an intervention to prevent weight gain among people not preselected on the basis of weight or age; studies in subpopulations such as those stopping smoking and studies of multifactorial interventions targeting specific diseases and studies targeting weight loss were not included	11 articles describing 9 distinct interventions 5 were in schools and 4 were in the community at large; 2 were in adults	Effectiveness seemed to be greater among older participants, men, nonsmokers, and those with high income Of 5 randomized, controlled trials, only 1 reported a significant effect on weight
Glenny et al[417]	**All ages** US and international, published through 1995 Randomized trials of treatment and both randomized and nonrandomized studies evaluating interventions for obesity prevention, at least 12 months' duration	Among 97 eligible trials of obesity treatment or prevention, only 4 were of prevention, 1 in children and 3 in adults	In the study in children, a 12-month family therapy intervention was initially successful compared with conventional treatment or no-treatment control, but effect was not present at 1-year follow-up 2 of the 2 studies involved comprehensive community-wide cardiovascular disease risk reduction programs; 1 reported a significantly smaller BMI increment over time in the intervention compared with control communities; the other study, based on mailed newsletters, optional group contact, and a financial incentive, reported a significant advantage for the intervention group after 1 year of follow-up

development of a nonstudy-related intervention in 1 of the control communities. The numerous activities targeted the home, school, and community environments and included environmental changes and policy development related to food availability and physical activity options, newsletters, training of teachers and medical professionals, and implementation of a restaurant certification program. Children in the intervention community had a more favorable BMI trajectory than those in the comparison arm.

Conclusions

A main objective of this scientific statement is to provide an overview of the types of strategies needed to prevent obesity

using a comprehensive, population-based approach rather than relying only on clinic-based or individually oriented strategies. Given that the ultimate determinants of obesity are individual eating and physical activity behaviors, the perception that one can solve the problem by refining the ability to help individuals to change their behaviors will persist. Central themes here are that what it will take for individuals on average to change their behaviors to the point of avoiding excess weight gain throughout the life course is affected by environmental factors that are not under their personal control. Research recommendations and programmatic initiatives for obesity prevention call for a broad range of strategies, many of which go beyond the knowledge, skill, and experience base of health professionals.

Investigators involved in pilot studies of obesity prevention identified a number of challenges to the design and conduct of research on obesity prevention in various organizational settings and study populations.[234,421] Foremost among these were the difficulty of motivating people to make the amount of effort needed for prevention of weight gain, the difficulty of measuring energy balance, the need to differentiate adverse weight gain from an increase in weight because of leaner body composition, and the large sample sizes needed to detect statistically significant differences when the primary outcome is no change in weight as opposed to the substantial weight losses obtained in treatment studies.[236] Perhaps partly for these reasons, the evidence to date includes many examples of obesity prevention interventions that have not shown significant differences in weight favoring the intervention group, making it especially important to identify examples of programs that might work.

Although the picture of how to intervene is far from complete, guidance and research recommendations developed by various expert panels,[7,355,422] working groups,[236] and systematic reviews (Table 6) have led to an increase in obesity prevention research. One of these expert reports, developed by the Institute of Medicine, provides a national action plan for childhood obesity prevention and includes more than 50 recommendations for actions applicable to governments, industries, communities at large, schools, and homes.[7] A subsequent Institute of Medicine report provides a framework for evaluating progress and an update on progress in implementing elements of the plan.[355] Targeted funding from the National Institutes of Health,[423] from the CDC (www.cdc.gov), and, for childhood obesity prevention, from the Robert Wood Johnson Foundation (www.rwjf.org), for example, is a major incentive to conduct population-based obesity prevention research. This research includes community-partnered and community action research and research on the effectiveness of policies implemented in various sectors and at various levels. Unproven efforts will continue as an important part of the community response to this pressing health problem, but the mandate to ground these efforts with some type of mechanism for evaluation is increasingly emphasized and funded.[355]

Ongoing activities, such as the CDC Guide to Community Preventive Services[424] and Cochrane evidence reviews,[405] policy tracking, report cards,[425–428] and web sites that serve as clearinghouses for sharing information about available resources and extant community programs[429,430] are creating an increasingly strong platform for action. Several initiatives specifically designed to generate policy and environmental changes and identify effective approaches in this respect have been funded by the Robert Wood Johnson Foundation as part of their commitment to reversing the childhood obesity epidemic by 2015 (see www.rwjf.org). A study of 9 countries in Europe has set the precedent for comprehensive study of how various policy options for obesity prevention are viewed by a broad range of stakeholders.[431] With respect to direct physician involvement, a model of potential interest is the Physicians for Healthy Communities Initiative[432] developed by the California Medical Association Foundation in partnership with the California Nutrition Network for Healthy, Active Families and Kaiser Permanente. This initiative will promote policy and environmental changes in schools and communities and will also assist physicians with training in community collaboration, nutrition messages, and advocacy techniques to enable them to become champions to promote healthy eating and active living throughout California.

Finally, some aspects of the scenario with respect to obesity prevention should sound very familiar to those experienced with CVD prevention. Strategies across the spectrum have been applied to promotion of changes in food intake and physical activity and the needs for upstream interventions clearly articulated, both in the United States and globally.[433] The North Karelia project, in which policy level interventions were implemented to generate population-wide reductions in intake of saturated fat, with benefits for reductions in CVD mortality, is perhaps the best known example of the success of policy changes for CVD risk reduction.[434] The concept of policy level interventions to change contexts for individual behavior is also well known from the experience with tobacco,[435] although the differences between food, which is essential to life and inherently good for health, and tobacco, which is nonessential and inherently bad for health, limit the direct transfer of some concepts and strategies. Many lessons from both tobacco and CVD prevention generally are applicable to obesity prevention. The most overarching lesson is that there is, indeed, the potential for success in combating such a far-reaching and deeply embedded societal pandemic.[435–437] Obesity treatment and prevention have always been a part of CVD prevention but, especially for prevention, have not been the primary focus. The rapid rise in obesity on a population level—associated with changes in the quantities of food available, marketed, and consumed, along with the very low level of obligatory physical activity for most people—makes obesity prevention efforts as a primary focus truly daunting. Furthermore, the inability to specify—at a population or individual level—the exact behaviors expected to result in energy balance considerably adds to the challenge. Avoiding unhealthy weight gain goes beyond the success of individual efforts to achieve good dietary quality and adequate physical fitness. It requires a broad range of strategies that include environmental and societal efforts.

Appendixes

Appendix 1. AHA Statements and Workshop Proceedings Related to Obesity Etiology, Complications, Prevention, and Treatment, 2004–2006

Reference		Description
Williams et al[22]	Children and adolescents	Provides practical guidelines to clinicians to decrease CVD risk factors in youth, including low physical activity, obesity, insulin resistance and type 2 diabetes, high blood pressure, hypercholesterolemia, and cigarette smoking
Steinberger et al[23]	Children and adolescents	Summarizes evidence to provide a rationale for lifestyle modification and weight control in childhood to reduce risks of developing insulin resistance, type 2 diabetes, and CVD; oriented to clinical practitioners
Hayman et al[24]	Children and adolescents	Provides guidance about how to optimize school environments in population-based strategies to promote cardiovascular health for US children and adolescents; intended for health and education professionals, child health advocates, policy makers, and community leaders; includes recommendations for school curricula, policies, and linkages to community resources and infrastructures
Mullis et al[25]	Adults, children, and families	Explains the complementarity of population-based and high-risk approaches to obesity prevention and treatment; describes important settings for instituting interventions to influence energy balance and the need for creative approaches to developing and evaluating broad policy approaches; makes research recommendations
Klein et al[26]	Adults	Reviews evidence on the clinical effects of weight loss on a variety of cardiovascular risk factors and outcomes and the clinical efficacy of treatments for obesity, including dietary and physical activity change, behavioral modification, pharmacotherapy, and surgery; summarizes guidelines for clinical evaluation and treatment of obese adults
Smith et al[21]	Adults in racial/ethnic minority populations	Highlights the higher-than-average risk of some or all metabolic syndrome components in African Americans, Hispanic Americans, American Indians/Alaska Natives, Asian Americans and Pacific Islanders; makes recommendation to the AHA for initiatives to reduce the related health disparities through professional/lay programs, public policy/advocacy, and research
Daniels et al[27]	Children and adolescents	Summarizes information on the pathophysiology and epidemiology of overweight in children and adolescents; provides an update on adverse health effects of childhood overweight and discusses approaches to prevention and treatment of overweight in children and adolescents
Grundy et al[28]	Adults	Reviews and provides updated information in support of the AHA recommendations for clinical diagnosis, therapeutic goals and management of the metabolic syndrome; identifies related areas of needed research
Gidding et al[29]	Children and adolescents	Summarizes current available information on cardiovascular nutrition in children and makes recommendations for both primordial and primary prevention of cardiovascular disease beginning at a young age; emphasizes the importance of nutrition early in life, including the fetal milieu; includes brief overview of public health issues related to nutrition
Poirier et al[2]	Adults	Updates the evidence for the impact of obesity on CVD, including cardiac structure and function and summarizes the benefits of weight loss on the cardiopulmonary system; discusses potential CVD risks associated with certain clinical weight loss approaches
American Heart Association Nutrition Committee et al[30]	Adults primarily, although applicable to children	Updates the AHA public health and clinical recommendations for diet and other lifestyle behaviors to prevent and manage CVD, including the guideline to "aim for a healthy weight"; includes practical tips for individuals to achieve these guidelines; provides recommendations for practitioners, restaurants, the food industry, schools, and local governments to promote a more supportive environment for achieving goals
Pate et al[31]	Children and adolescents	Highlights physical activity to be a key determinant of weight status, summarizes the evidence supporting schools' potential for effectively improving and promoting physical activity, and recommends several key changes in school policy and practice
Kavey et al[32]	Children and adolescents	Provides guidelines for CVD prevention in children and adolescents who are at high risk for early coronary disease; these guidelines recommend more aggressive treatment of CVD risk factors, including obesity, than in the general population for children and adolescents with conditions such as familial hypercholesterolemia, diabetes, chronic kidney disease, heart transplantation, Kawasaki disease, systemic lupus erythematosus, rheumatoid arthritis, congenital heart disease, and past history of cancer treatment
Hayman et al[33]	Children and adolescents	Reviews rationale for primary prevention of CVD in youth and reports interventions at the population level and in high-risk individuals; provides guidelines with particular emphasis on nursing practice

Appendix 2. Selected Evidence-Based Recommendations and Guidelines for Obesity Prevention and Treatment in Adults and Child/Adolescent Populations

Source	Relevance*
National Institutes of Health[34]	Adults
McTigue et al[17]	Adults
Katz et al[35]	Children/adolescents and adults
Koplan et al[7]	Children/adolescents
American Heart Association Nutrition Committee et al[30]	Children/adolescents and adults
Lau et al[36]	Children/adolescents and adults
National Initiative for Children's Healthcare Quality et al[37]	Children/adolescents

*Children under age 2 years are not targeted in any of the guidelines listed.

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*Modest.

†Significant.

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References

1. Visscher TL, Seidell JC. The public health impact of obesity. *Annu Rev Public Health*. 2001;22:355–375.
2. Poirier P, Giles TD, Bray GA, Hong Y, Stern JS, Pi-Sunyer FX, Eckel RH; American Heart Association; Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation.* 2006;113:898–918.
3. US Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*. Boston, MA: Jones and Bartlett Publishers; 2001.
4. National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overweight/overwght_adult_03.htm. Accessed May 8, 2008.
5. National Center for Health Statistics. Available at: http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overweight/overwght_child_03.htm. Accessed May 8, 2008.
6. US Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity*. Rockville, MD: US Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001. Available at: http://www.surgeongeneral.gov/topics/obesity/. Accessed May 8, 2008.
7. Koplan J, Liverman CT, Kraak VI. *Preventing Childhood Obesity: Health in the Balance*. Washington, DC: National Academies Press; 2005.
8. *Obesity: Preventing and Managing the Global Epidemic*. WHO Technical Report Series No. 894. Geneva, Switzerland: World Health Organization; 2000.
9. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295:1549–1555.
10. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. *Int J Pediatr Obes*. 2006;1:11–25.
11. Rosamond W, Flegal K, Friday G, Furie K, Go A, Greenlund K, Haase N, Ho M, Howard V, Kissela B, Kittner S, Lloyd-Jones D, McDermott M, Meigs J, Moy C, Nichol G, O'Donnell CJ, Roger V, Rumsfeld J, Sorlie P, Steinberger J, Thom T, Wasserthiel-Smoller S, Hong Y; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics–2007 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2007;115:e69–e171.
12. Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev*. 2007;29:6–28.
13. World Health Organization. *Risk Factor Projects. Overweight and Obesity*. 2005. Available at: http://www.who.int/chp/chronic_disease_report/part2_ch1/en/index16.html. Accessed September 11, 2007.
14. Gregg EW, Cheng YJ, Cadwell BL, Imperatore G, Williams DE, Flegal KM, Narayan V, Williamson DF. Secular trends in cardiovascular disease risk factors according to body mass index in US adults. *JAMA*. 2005;293:1868–1874.
15. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. *Obes Res*. 1998;6(suppl 2):51S–209S.
16. Harris TB, Savage PJ, Tell GS, Haan M, Kumanyika S, Lynch JC. Carrying the burden of cardiovascular risk in old age: associations of weight and weight change with prevalent cardiovascular disease, risk factors, and health status in the Cardiovascular Health Study. *Am J Clin Nutr*. 1997;66:837–844.
17. McTigue KM, Harris R, Hemphill B, Lux L, Sutton S, Bunton AJ, Lohr KN. Screening and interventions for obesity in adults: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2003;139:933–949.
18. Ryan DH, Espeland MA, Foster GD, Haffner SM, Hubbard VS, Johnson KC, Kahn SE, Knowler WC, Yanovski SZ; Look AHEAD Research Group. Look AHEAD (Action for Health in Diabetes). Design and methods for a clinical trial of weight loss for the prevention of cardiovascular disease in type 2 diabetes. *Control Clin Trials*. 2003;24:610–628.
19. Kumanyika S, Jeffery RW, Morabia A, Ritenbaugh C, Antipatis VJ; Public Health Approaches to the Prevention of Obesity (PHAPO) Working Group of the International Obesity Task Force (IOTF). Obesity prevention: the case for action. *Int J Obes Relat Metab Disord*. 2002;26:425–436.
20. Kumanyika S, Brownson RC, eds. *Handbook of Obesity Prevention: A Resource for Health Professionals*. New York, NY: Springer; 2007.
21. Smith SC Jr, Clark LT, Cooper RS, Daniels SR, Kumanyika SK, Ofili E, Quinones MA, Sanchez EJ, Saunders E, Tiukinhoy SD; American Heart Association Obesity, Metabolic Syndrome, and Hypertension Writing Group. Discovering the full spectrum of cardiovascular disease: Minority Health Summit 2003: report of the Obesity, Metabolic Syndrome, and Hypertension Writing Group. *Circulation*. 2005;111:e134–e139.
22. Williams CL, Hayman LL, Daniels SR, Robinson TN, Steinberger J, Paridon S, Bazzarre T. Cardiovascular health in childhood: a statement for health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation*. 2002;106:143–160.
23. Steinberger J, Daniels SR; American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young); American Heart Association Diabetes Committee (Council on Nutrition, Physical Activity, and Metabolism). Obesity, insulin resistance, diabetes, and cardiovascular risk in children: an American Heart Association scientific statement from the Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young) and the Diabetes Committee (Council on Nutrition, Physical Activity, and Metabolism). *Circulation*. 2003;107:1448–1453.
24. Hayman LL, Williams CL, Daniels SR, Steinberger J, Paridon S, Dennison BA, McCrindle BW; Committee on Atherosclerosis, Hypertension, and Obesity in Youth (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. Cardiovascular health promotion in the schools: a statement for health and education professionals and child health advocates from the Committee on Atherosclerosis, Hypertension, and Obesity in Youth (AHOY) of the

Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation.* 2004;110:2266–2275.
25. Mullis RM, Blair SN, Aronne LJ, Bier DM, Denke MA, Dietz W, Donato KA, Drewnowski A, French SA, Howard BV, Robinson TN, Swinburn B, Wechsler H; American Heart Association. Prevention conference VII: obesity, a worldwide epidemic related to heart disease and stroke: group IV: prevention/treatment. *Circulation*. 2004;110: e484–e488.
26. Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH; American Heart Association Council on Nutrition, Physical Activity, and Metabolism. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2004;110:2952–2967.
27. Daniels SR, Arnett DK, Eckel RH, Gidding SS, Hayman LL, Kumanyika S, Robinson TN, Scott BJ, St Jeor S, Williams CL. Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation*. 2005;111:1999–2012.
28. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith SC Jr, Spertus JA, Costa F; American Heart Association; National Heart, Lung, and Blood Institute. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*. 2005;112:2735–2752.
29. Gidding SS, Dennison BA, Birch LL, Daniels SR, Gillman MW, Lichtenstein AH, Rattay KT, Steinberger J, Stettler N, Van Horn L; American Heart Association; American Academy of Pediatrics. Dietary recommendations for children and adolescents: a guide for practitioners: consensus statement from the American Heart Association. *Circulation*. 2005;112:2061–2075.
30. American Heart Association Nutrition Committee, Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, Franklin B, Kris-Etherton P, Harris WS, Howard B, Karanja N, Lefevre M, Rudel L, Sacks F, Van Horn L, Winston M, Wylie-Rosett J. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006; 114:82–96.
31. Pate RR, Davis MG, Robinson TN, Stone EJ, McKenzie TL, Young JC; American Heart Association Council on Nutrition, Physical Activity, and Metabolism (Physical Activity Committee); Council on Cardiovascular Disease in the Young; Council on Cardiovascular Nursing. Promoting physical activity in children and youth: a leadership role for schools: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism (Physical Activity Committee) in collaboration with the Councils on Cardiovascular Disease in the Young and Cardiovascular Nursing. *Circulation*. 2006;114:1214–1224.
32. Kavey RE, Allada V, Daniels SR, Hayman LL, McCrindle BW, Newburger JW, Parekh RS, Steinberger J; American Heart Association Expert Panel on Population and Prevention Science; American Heart Association Council on Cardiovascular Disease in the Young; American Heart Association Council on Epidemiology and Prevention; American Heart Association Council on Nutrition, Physical Activity and Metabolism; American Heart Association Council on High Blood Pressure Research; American Heart Association Council on Cardiovascular Nursing; American Heart Association Council on the Kidney in Heart Disease; Interdisciplinary Working Group on Quality of Care and Outcomes Research. Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American Heart Association Expert Panel on Population and Prevention Science; the Councils on Cardiovascular Disease in the Young, Epidemiology and Prevention, Nutrition, Physical Activity and Metabolism, High Blood Pressure Research, Cardiovascular Nursing, and the Kidney in Heart Disease; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research. *Circulation*. 2006;114:2710–2738.
33. Hayman LL, Meininger JC, Daniels SR, McCrindle BW, Helden L, Ross J, Dennison BA, Steinberger J, Williams CL; American Heart Association Committee on Atherosclerosis, Hypertension, and Obesity in Youth of the Council on Cardiovascular Disease in the Young; American Heart Association Council on Cardiovascular Nursing; American Heart Association Council on Epidemiology and Prevention; American Heart Association Council on Nutrition, Physical Activity, and Metabolism. Primary prevention of cardiovascular disease in nursing practice: focus on children and youth: a scientific statement from the American Heart Association Committee on Atherosclerosis, Hypertension, and Obesity in Youth of the Council on Cardiovascular Disease in the Young, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2007;116:344–357.
34. National Institutes of Health, National Heart, Lung and Blood Institute, North American Association for the Study of Obesity. *The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults*. NIH Publication Number 00–4084. October 2000. Available at: http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_c.pdf. Accessed May 8, 2008.
35. Katz DL, O'Connell M, Yeh MC, Nawaz H, Njike V, Anderson LM, Cory S, Dietz W; Task Force on Community Preventive Services. Public health strategies for preventing and controlling overweight and obesity in school and worksite settings. *MMWR Recomm Rep*. 2005;54:1–12.
36. Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E; Obesity Canada Clinical Practice Guidelines Expert Panel. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children [summary]. *CMAJ*. 2007;176:S1–S13.
37. National Initiative for Children's Healthcare Quality. *Expert Committee Recommendations on the Assessment, Prevention, and Treatment of Child and Adolescent Overweight and Obesity 2007*. An Implementation Guide from the Childhood Obesity Action Network. Available at: http://www.ihi.org/IHI/Topics/ChronicConditions/AllConditions/Resources/NICHQChildhoodObesityImplementationGuide.htm. Accessed September 2, 2007.
38. Lavizzo-Mourey R. Childhood obesity: what it means for physicians. *JAMA*. 2007;298:920–922.
39. National Center for Health Statistics. Hyattsville, MD: Chartbook on Trends in the Health of Americans; 2006. Available at: http://www.cdc.gov/nchs/data/hus/hus06.pdf. Accessed May 8, 2008.
40. Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, Mei Z, Curtin LR, Roche AF, Johnson CL. CDC growth charts: United States. *Adv Data.* 2000;314:1–27.
41. Barlow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity [summary report]. *Pediatrics* 2007;120(suppl 4): S164–S192.
42. Fernández JR, Redden DT, Pietrobelli A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *J Pediatr*. 2004;145:439–444.
43. Yun S, Zhu BP, Black W, Brownson RC. A comparison of national estimates of obesity prevalence from the behavioral risk factor surveillance system and the National Health and Nutrition Examination Survey. *Int J Obes* (*Lond*). 2006;30:164–170.
44. Gillum RF, Sempos CT. Ethnic variation in validity of classification of overweight and obesity using self-reported weight and height in American women and men: the Third National Health and Nutrition Examination Survey. *Nutr J*. 2005;4:27.
45. Adams PF, Schoenborn CA. Health behaviors of adults: United States, 2002–04. *Vital Health Stat 10.* 2006;230:1–140.
46. Denny CH, Holtzman D, Goins RT, Croft JB. Disparities in chronic disease risk factors and health status between American Indian/Alaska Native and White elders: findings from a telephone survey, 2001 and 2002. *Am J Public Health*. 2005;95:825–827.
47. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005;111:1233–1241
48. Das SR, Kinsinger LS, Yancy WS Jr, Wang A, Ciesco E, Burdick M, Yevich SJ. Obesity prevalence among veterans at Veterans Affairs medical facilities. *Am J Prev Med*. 2005;28:291–294.
49. Baskin ML, Ard J, Franklin F, Allison DB. Prevalence of obesity in the United States. *Obes Rev*. 2005;6:5–7.
50. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA*. 2002;288:1723–1727.
51. Denney JT, Krueger PM, Rogers RG, Boardman JD. Race/ethnic and sex differentials in body mass among US adults. *Ethn Dis*. 2004;14: 389–398.
52. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA*. 2004;291:2847–2850.
53. McTigue K, Larson JC, Valoski A, Burke G, Kotchen J, Lewis CE, Stefanick ML, Van Horn L, Kuller L. Mortality and cardiac and vascular outcomes in extremely obese women. *JAMA*. 2006;296:79–86.

54. Goel MS, McCarthy EP, Phillips RS, Wee CC. Obesity among US immigrant subgroups by duration of residence. *JAMA*. 2004;292:2860–2867.
55. Kaplan MS, Huguet N, Newsom JT, McFarland BH. The association between length of residence and obesity among Hispanic immigrants. *Am J Prev Med*. 2004;27:323–326.
56. Himmelgreen DA, Pérez-Escamilla R, Martinez D, Bretnall A, Eells B, Peng Y, Bermúdez A. The longer you stay, the bigger you get: length of time and language use in the U.S. are associated with obesity in Puerto Rican women. *Am J Phys Anthropol*. 2004;125:90–96.
57. Seidell JC, Kahn HS, Williamson DF, Lissner L, Valdez R. Report from a Centers for Disease Control and Prevention Workshop on use of adult anthropometry for public health and primary health care. *Am J Clin Nutr*. 2001;73:123–126.
58. Zhu S, Heymsfield SB, Toyoshima H, Wang Z, Pietrobelli A, Heshka S. Race-ethnicity-specific waist circumference cutoffs for identifying cardiovascular disease risk factors. *Am J Clin Nutr*. 2005;81:409–415.
59. Pan SY, Johnson KC, Ugnat AM, Wen SW, Mao Y; Canadian Cancer Registries Epidemiology Research Group. Association of obesity and cancer risk in Canada. *Am J Epidemiol*. 2004;159:259–268.
60. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363:157–163.
61. Misra A, Wasir JS, Vikram NK. Waist circumference criteria for the diagnosis of abdominal obesity are not applicable uniformly to all populations and ethnic groups. *Nutrition*. 2005;21:969–976.
62. World Health Organization and International Obesity Task Force. The Asian-Pacific Perspective: Redefining Obesity and Its Treatment. Melbourne, Australia: Health Communications Australia; 2000. Available at: http://www.diabetes.com.au/pdf/obesity_report.pdf. Accessed May 8, 2008.
63. Razak F, Anand SS, Shannon H, Vuksan V, Davis B, Jacobs R, Teo KK, McQueen M, Yusuf S. Defining obesity cut points in a multiethnic population. *Circulation*. 2007;115:2111–2118.
64. Slusser WM, Cumberland WG, Browdy BL, Winham DM, Neumann CG. Overweight in urban, low-income, African American and Hispanic children attending Los Angeles elementary schools: research stimulating action. *Public Health Nutr*. 2005;8:141–148.
65. Thorpe LE, List DG, Marx T, May L, Helgerson SD, Frieden TR. Childhood obesity in New York City elementary school students. *Am J Public Health*. 2004;94:1496–1500.
66. Story M, Stevens J, Himes J, Stone E, Rock BH, Ethelbah B, Davis S. Obesity in American-Indian children: prevalence, consequences, and prevention. *Prev Med*. 2003;37:S3–S12.
67. Freedman DS, Khan LK, Serdula MK, Ogden CL, Dietz WH. Racial and ethnic differences in secular trends for childhood BMI, weight, and height. *Obesity* (*Silver Spring*). 2006;14:301–308.
68. Zhang Q, Wang Y. Trends in the association between obesity and socioeconomic status in U.S. adults: 1971 to 2000. *Obes Res*. 2004;12:1622-1632.
69. Roberts SA, Reither EN. A multilevel analysis of race, community disadvantage, and body mass index among adults in the US. *Soc Sci Med*. 2004;59:2421–2434.
70. Goodman E. The role of socioeconomic status gradients in explaining differences in US adolescents' health. *Am J Public Health*. 1999;89:1522–1528.
71. Kimm SY, Obarzanek E, Barton BA, Aston CE, Similo SL, Morrison JA, Sabry ZI, Schreiber GE, McMahan RP. Race, socioeconomic status, and obesity in 9- to 10-year-old girls: the NHLBI Growth and Health Study. *Ann Epidemiol*. 1996;6:266–275.
72. Gordon-Larsen P, Adair LS, Popkin BM. The relationship of ethnicity, socioeconomic factors, and overweight in US adolescents. *Obes Res*. 2003;11:121–129.
73. Miech RA, Kumanyika SK, Stettler N, Link BG, Phelan JC, Chang VW. Trends in the association of poverty with overweight among US adolescents, 1971–2004. *JAMA*. 2006;295:2385–2393.
74. Wang Y, Zhang Q. Are American children and adolescents of low socioeconomic status at increased risk of obesity? Changes in the association between overweight and family income between 1971 and 2002. *Am J Clin Nutr*. 2006;84:707–716.
75. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. Behavioral Risk Factor Surveillance System (BRFSS). 2006. Available at: http://apps.nccd.cdc.gov/brfss/index.asp. Accessed May 8, 2008.
76. Kahn HS, Tatham LM, Pamuk ER, Heath CW. Are geographic regions with high income inequality associated with risk of abdominal weight gain? *Soc Sci Med*. 1998;47:1–6.
77. Ramsey PW, Glenn L. Obesity and health status in rural, urban, and suburban Southern women. *South Med J*. 2002;95:666–671.
78. Patterson PD, Moore CG, Probst JC, Shinogle JA. Obesity and physical activity in rural America. *J Rural Health*. 2004;20:151–159.
79. Jackson JE, Doescher MP, Jerant AF, Hart LG. A national study of obesity prevalence and trends by type of rural county. *J Rural Health*. 2005;21:140–148.
80. Seidell JC, Nooyens AJ, Visscher TL. Cost-effective measures to prevent obesity: epidemiological basis and appropriate target groups. *Proc Nutr Soc*. 2005;64:1–5.
81. Eckel RH, Krauss RM. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. AHA Nutrition Committee. *Circulation* 1998;97:2099–2100.
82. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation*. 1983;67:968–977.
83. Truesdale KP, Stevens J, Lewis CE, Schreiner PJ, Loria CM, Cai J. Changes in risk factors for cardiovascular disease by baseline weight status in young adults who maintain or gain weight over 15 years: the CARDIA study. *Int J Obesity* (*Lond*). 2006;30:1397–1407.
84. Lloyd-Jones DM, Liu K, Colangelo LA, Yan LL, Klein L, Loria CM, Lewis CE, Savage P. Consistently stable or decreased body mass index in young adulthood and longitudinal changes in metabolic syndrome components. The Coronary Artery Risk Development in Young Adults Study. *Circulation*. 2007;115:1004–1011.
85. Wannamethee SG, Shaper AG, Walker M. Overweight and obesity and weight change in middle aged men: impact on cardiovascular disease and diabetes. *J Epidemiol Community Health*. 2005;59:134–139.
86. Manson JE, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, Hennekens CH, Speizer FE. Body weight and mortality among women. *N Engl J Med*. 1995;333:677–685.
87. Shaper AG, Wannamethee SG, Walker M. Body weight: implications for the prevention of coronary heart disease, stroke, and diabetes mellitus in a cohort study of middle aged men. *BMJ*. 1997;314:1311–1317.
88. Abdollahi M, Cushman M, Rosendaal FR. Obesity: risk of venous thrombosis and the interaction with coagulation factor levels and oral contraceptive use. *Thromb Haemost*. 2003;89:493–498.
89. Abu S, Shamsuzzaman M, Gersh BJ, Somers VK. Obstructive sleep apnea: implications for cardiac and vascular disease. *JAMA*. 2003;290:1906–1914.
90. Acalovschi MV, Blendea D, Pascu M, Georoceanu A, Badea RI, Prelipceanu M. Risk of asymptomatic and symptomatic gallstones in moderately obese women: a longitudinal follow-up study. *Am J Gastroenterol*. 1997;92:127–131.
91. Ajani UA, Lotufo PA, Gaziano JM, Lee IM, Spelsberg A, Buring JE, Willett WC, Manson JE. Body mass index and mortality among US male physicians. *Ann Epidemiol*. 2004;14:731–739.
92. Allison DB, Fontaine KR, Manson JE, Stevens U, VanItallie TB. Annual deaths attributable to obesity in the United States. *JAMA*. 1999;282:1530–1538.
93. Aucott L, Poobalan A, Smith WCS, Avenell A, Jung R, Broom J. Effects of weight loss in overweight/obese individuals and long-term hypertension outcomes: a systematic review. *Hypertension*. 2005;45:1035–1041.
94. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *N Engl J Med*. 2003;348:1625–1638.
95. Caterson ID, Hubbard V, Bray GA, Grunstein R, Hansen BC, Hong Y, Labarthe D, Seidell JC, Smith SC Jr; American Heart Association. Prevention Conference VII: obesity, a worldwide epidemic related to heart disease and stroke: group III: worldwide comorbidities of obesity. *Circulation*. 2004;110:e476–e483.
96. Cerhan JR, Torner JC, Lynch CF, Rubenstein LM, Lemke JH, Cohen MB, Lubaroff DM, Wallace RB. Association of smoking, body mass, and physical activity with risk of prostate cancer in the Iowa 65+ Rural Health Study (United States). *Cancer Causes Control*. 1997;8:229–238.
97. Choi HK, Atkinson K, Karlson EW, Curhan G. Obesity, weight change, hypertension, diuretic use, and risk of gout in men: the health professionals follow-up study. *Arch Intern Med*. 2005;165:742–748.

98. Daviglus ML, Liu K, Yan LL, Pirzada A, Manheim L, Manning W, Garside DB, Wang R, Dyer AR, Greenland P, Stamler J. Relation of body mass index in young adulthood and middle age to Medicare expenditures in older age. *JAMA*. 2004;292:2743–2749.
99. Derby CA, Mohr BA, Goldstein I, Feldman HA, Johannes CB, McKinlay JB. Modifiable risk factors and erectile dysfunction: can lifestyle changes modify risk? *Urology*. 2000;56:302–306.
100. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.* 2002;346:393–403.
101. Dyer AR, Stamler J, Garside DB, Greenland P. Long-term consequences of body mass index for cardiovascular mortality: the Chicago Heart Association Detection Project in Industry study. *Ann Epidemiol*. 2004; 14:101–108.
102. Engström G, Hedblad B, Stavenow L, Jonsson S, Lind P, Janzon L, Lindgärde F. Incidence of obesity-associated cardiovascular disease is related to inflammation-sensitive plasma proteins: a population-based cohort study. *Arterioscler Thromb Vasc Biol*. 2004;24:1498–1502.
103. Esposito K, Giugliano F, DiPalo C, Giugliano G, Marfella R, D'Andrea FD, D'Armiento M, Giugliano D. Effect of lifestyle changes on erectile dysfunction in obese men: a randomized controlled trial. *JAMA*. 2004; 291:2978–2984.
104. Esposito K, Pontillo A, DiPalo C, Giugliano G, Masella M, Marfella R, Giugliano D. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. *JAMA*. 2003;289:1799–1804.
105. Field AE, Coakley EH, Must A, Spadano JL, Laird N, Dietz WH, Rimm E, Golditz GA. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. *Arch Intern Med*. 2001;161: 1581–1586.
106. Flegal KM, Graubard BI, Williamson DF. Methods of calculating deaths attributable to obesity. *Am J Epidemiol*. 2004;160:331–338.
107. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA*. 2005;293: 1861–1867.
108. Gillum R, Mussolino ME, Madans JH. Body fat distribution, obesity, overweight and stroke incidence in women and men—the NHANES I Epidemiologic Follow-up Study. *Int J Obes Relat Metab Disord*. 2001; 25:628–638.
109. Gurm HS, Whitlow PL, Kip KE; BARI Investigators. The impact of body mass index on short- and long-term outcomes in patients undergoing coronary revascularization: insights from the bypass angioplasty revascularization investigation (BARI). *J Am Coll Cardiol* 2002; 39:834–840.
110. Gustafson D, Rothenberg E, Blennow K, Steen B, Skoog I. An 18-year follow-up of overweight and risk of Alzheimer disease. *Arch Intern Med*. 2003;163:1524–1528.
111. Hamaguchi M, Kojima T, Takeda N, Nakagawa T, Taniguchi H, Fuiji K, Omatsu T, Nakajima T, Sarui H, Shimazaki M, Kato T, Okuda J, Ida K. The metabolic syndrome as a predictor of nonalcoholic fatty liver disease. *Ann Intern Med*. 2005;143:722–728.
112. Hampel H, Abraham NS, El-Serag HB. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. *Ann Intern Med*. 2005;143:199–211.
113. Hasler G, Pine DS, Gamma A, Milos G, Ajdacic V, Eich D, Rössler W, Angst J. The associations between psychopathology and being overweight: a 20-year prospective study. *Psychol Med*. 2004;34:1047–1057.
114. Hu FB, Willett WC, Li T, Stampfer MJ, Colditz GA, Manson JA. Adiposity as compared with physical activity in predicting mortality among women. *N Engl J Med*. 2004;351:2694–2703.
115. Hu G, Tuomilehto J, Silventoinen K, Barengo N, Jousilahti P. Joint effects of physical activity, body mass index, waist circumference and waist-to-hip ratio with the risk of cardiovascular disease among middle-aged Finnish men and women. *Eur Heart J*. 2004;25:2212–2219.
116. Ioannou GN, Weiss NS, Boyko EJ, Kowdley KV, Kahn SE, Carithers RL, Tsai EC, Dominitz JA. Is central obesity associated with cirrhosis-related death or hospitalization? A population-based, cohort study. *Clin Gastroenterol Hepatol*. 2005;3:67–74.
117. Jin R, Grunkemeier GL, Furnary AP, Handy JR Jr. Is obesity a risk factor for mortality in coronary artery bypass surgery? *Circulation* 2005;111:3359–3365.
118. Jonsson S, Hedblad B, Engström G, Nilsson P, Berglund G, Janzon L. Influence of obesity on cardiovascular risk. Twenty-three-year follow-up of 22,025 men from an urban Swedish population. *Int J Obes Relat Metab Disord*. 2002;26:1046–1053.
119. Jood K, Jern C, Wilhelmsen L, Rosengren A. Body mass index in mid-life is associated with a first stroke in men: a prospective population study over 28 years. *Stroke*. 2004;35:2764–2769.
120. Jousilahti P, Tuomilehto J, Vartiainen E, Pekkanen J, Puska P. Body weight, cardiovascular risk factors, and coronary mortality: 15-year follow-up of middle-aged men and women in eastern Finland. *Circulation*. 1996;93:1372–1379.
121. Jubber AS. Respiratory complications of obesity. *Int J Clin Practice*. 2004;58:573–580.
122. Kambham N, Markowitz GS, Valeri AM, Lin J, D'Agati VD. Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int*. 2001;59: 1498–1509.
123. Kenchaiah S, Evans JC, Levy D, Wilson PWF, Benjamin EJ, Larson MG, Kannel WB, Vasan RS. Obesity and the risk of heart failure. *N Engl J Med*. 2002;347:305–313.
124. Kristensen J, Vestergaard M, Wisborg K, Kesmodel U, Secher NJ. Pre-pregnancy weight and the risk of stillbirth and neonatal death. *BJOG* 2005;112:403–408.
125. Kurth R, Gaziano JM, Rexrode KM, Kase CS, Cook NR, Manson JE, Buring JE. Prospective study of body mass index and risk of stroke in apparently healthy women. *Circulation*. 2005;111:1992–1998.
126. Lakka HM, Lakka TA, Tuomilehto J, Salonen JT. Abdominal obesity is associated with increased risk of acute coronary events in men. *Eur Heart J*. 2002;23:706–713.
127. Lashen H, Fear K, Sturdee DW. Obesity is associated with increased risk of first trimester and recurrent miscarriage: matched case-control study. *Hum Reprod*. 2004;19:1644–1646.
128. Linné Y. Effects of obesity on women's reproduction and complications during pregnancy. *Obes Rev*. 2004;5:137–143.
129. Luder E, Ehrlich RI, Lou WY, Melnik TA, Kattan M. Body mass index and the risk of asthma in adults. *Respir Med*. 2004;98:29–37.
130. Mao Y, Pan S, Wen SW, Johnson KC; Canadian Cancer Registries Epidemiology Research Group. Physical inactivity, energy intake, obesity and the risk of rectal cancer in Canada. *Int J Cancer*. 2003;105: 831–837.
131. Mertens I, VanGaal LF. Obesity, haemostasis and the fibrinolytic system. *Obes Rev*. 2002;3:85–101.
132. Moore LL, Bradlee ML, Singer MR, Splansky GL, Proctor MH, Ellison RC, Kreger BE. BMI and waist circumference as predictors of lifetime colon cancer risk in Framingham Study adults. *Int J Obes Relat Metab Disord*. 2004;28:559–567.
133. Morales E, Valero M, León M, Hernández E, Praga M. Beneficial effects of weight loss in overweight patients with chronic proteinuric nephropathies. *Am J Kidney Dis*. 2003;41:319–327.
134. Moreau M, Valente F, Mak R, Pelfrene E, de Smet P, De Backer G, Kornitzer M. Obesity, body fat distribution and incidence of sick leave in the Belgian workforce: the Belstress study. *Int J Obes Relat Metab Disord*. 2004;28:574–582.
135. Morimoto LM, White E, Chen Z, Chlebowski RT, Hays J, Kuller L, Lopez AM, Manson J, Margolis KL, Muti PC, Stefanick ML, McTiernan A. Obesity, body size, and risk of postmenopausal breast cancer: the Women's Health Initiative (United States). *Cancer Causes Control*. 2002;13:741–751.
136. National Task Force on the Prevention and Treatment of Obesity. Overweight, obesity, and health risk. *Arch Intern Med*. 2000;160:898–904.
137. Nicklas BJ, Penninx BW, Cesari M, Kritchevsky SB, Newman AB, Kanaya AM, Pahor M, Jingzhong D, Harris TB; Health, Aging and Body Composition Study. Association of visceral adipose tissue with incident myocardial infarction in older men and women: the Health, Aging and Body Composition Study. *Am J Epidemiol*. 2004;160: 741–749.
138. Normal JE, Bild D, Lewis CE, Liu K, West DS; CARDIA Study. The impact of weight change on cardiovascular disease risk factors in young black and white adults: the CARDIA study. *Int J Obes Relat Metab Disord*. 2003;27:369–376.
139. Nowbar S, Burkart KM, Gonzales R, Fedorowicz A, Gozansky WS, Gaudio JC, Taylor MRG, Zwillich CW. Obesity-associated hypoventilation in hospitalized patients: prevalence, effects, and outcome. *Am J Med*. 2004;116:1–7.
140. Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, Hayflick L, Butler RN, Allison DB, Ludwig DS. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med*. 2005;352:1138–1145.

141. Oppert JM, Charles MA, Thibult N, Guy-Grand B, Eschwège E, Ducimetière P. Anthropometric estimates of muscle and fat mass in relation to cardiac and cancer mortality in men: the Paris Prospective Study. *Am J Clin Nutr*. 2002;75:1107–1113.
142. Peeters A, Bonneux L, Nusselder WJ, De Laet C, Barendregt JJ. Adult obesity and the burden of disability throughout life. *Obes Res*. 2004;12:1145–1151.
143. Prabhakar G, Haan CK, Peterson ED, Coombs LP, Cruzzavala JL, Murray GF. The risks of moderate and extreme obesity for coronary artery bypass grafting outcomes: a study from the Society of Thoracic Surgeons' database. *Ann Thorac Surg*. 2002;74:1125–1130.
144. Ranne D, Medalie JH, Goldbourt U. Body fat distribution and long-term risk of stroke mortality. *Stroke*. 2005;36:1021–1025.
145. Rexrode KM, Buring JE, Manson JE. Abdominal and total adiposity and risk of coronary heart disease in men. *Int J Obes Relat Metab Disord*. 2001;25:1047–1056.
146. Rexrode KM, Garey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfler MJ, Willett WC, Manson JE. Abdominal adiposity and coronary heart disease in women. *JAMA*. 1998;280:1843–1848.
147. Rosen RC, Wing R, Schneider S, Gendrano N 3rd. Epidemiology of erectile dysfunction: the role of medical comorbidities and lifestyle factors. *Urol Clin North Am*. 2005;32:403–417.
148. Rosengren A, Wedel H, Wilhelmsen L. Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality: a prospective population study. *Eur Heart J*. 1999;20:269–277.
149. Ross JA, Parker E, Blair CK, Cerhan JR, Folsom AR. Body mass index and risk of leukemia in older women. *Cancer Epidemiol Biomarkers Prev*. 2004;13:1810–1813.
150. Sahi T, Paffenbarger RS Jr, Hsieh CC, Lee IM. Body mass index, cigarette smoking, and other characteristics as predictors of self-reported, physician-diagnosed gallbladder disease in male college alumni. *Am J Epidemiol*. 1998;147:644–651.
151. Sahyoun NR, Hochberg MC, Helmick CG, Harris T, Pamuk ER. Body mass index, weight change, and incidence of self-reported physician-diagnosed arthritis among women. *Am J Public Health*. 1999;89:391–394.
152. Santillan AA, Camargo CA. Body mass index and asthma among Mexican adults: the effect of using self-reported vs measured weight and height. *Int J Obes Relat Metab Disord*. 2003;27:1430–1433.
153. Sjöström L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjöström CD, Sullivan M, Wedel H; Swedish Obese Subjects Study Scientific Group. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med.* 2004;351:2683–2693.
154. Song YM, Sung J, Davey Smith G, Ebrahim S. Body mass index and ischemic and hemorrhagic stroke: a prospective study in Korean men. *Stroke*. 2004;35:831–836.
155. Stevens J, Cai J, Evenson KR, Thomas R. Fitness and fatness as predictors of mortality from all causes and from cardiovascular disease in men and women in the lipid research clinics study. *Am J Epidemiol*. 2002;156:832–841.
156. Stevens VJ, Obarzanek E, Cook NR, Lee IM, Appel LJ, West DS, Milas NC, Mattfeldt-Beman M, Belden L, Bragg C, Millstone M, Raczynski J, Brewer A, Singh B, Cohen J; Trials for the Hypertension Prevention Research Group. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med*. 2001;134:1–11.
157. Strandberg TE, Strandberg A, Salomaa VV, Pitkälä K, Miettinen TA. Impact of midlife weight change on mortality and quality of life in old age. Prospective cohort study. *Int J Obes Relat Metab Disord*. 2003;27:950–954.
158. Taylor EN, Stampfer MJ, Curhan GC. Obesity, weight gain, and the risk of kidney stones. *JAMA*. 2005;293:455–462.
159. Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, Keinänen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M; Finnish Diabetes Prevention Study Group. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343–1350.
160. Vgontzas AN, Bixler EO, Chrousos GP. Metabolic disturbances in obesity versus sleep apnoea: the importance of visceral obesity and insulin resistance. *J Intern Med*. 2003;254:32–44.
161. Visscher TL, Rissanen A, Seidell JC, Heliövaara M, Knekt P, Reunanen A, Aromaa A. Obesity and unhealthy life-years in adult Finns: an empirical approach. *Arch Intern Med*. 2004;164:1413–1420.
162. Walker SP, Rimm EB, Ascherio A, Kawachi I, Stampfer MJ, Willett WC. Body size and fat distribution as predictors of stroke among US men. *Am J Epidemiol*. 1996;144:1143–1150.
163. Weiss JL, Malone FD, Emig D, Ball RH, Nyberg DA, Comstock CH, Saade G, Eddleman K, Carter SM, Craigo SD, Carr SR, D'Alton ME; FASTER Research Consortium. Obesity, obstetric complications and cesarean delivery rate—a population-based screening study. *Am J Obstet Gynecol*. 2004;190:1091–1097.
164. Wolk R, Shamsuzzaman M, Somers VK. Obesity, sleep apnea, and hypertension. *Hypertension*. 2003;42:1067–1074.
165. He XZ, Baker DB. Body mass index, physical activity, and the risk of decline in overall health and physical functioning in late middle age. *Am J Public Health*. 2004;94:1567–1573.
166. Vainio H, Bianchini F, eds. *IARC Handbooks of Cancer Prevention: Weight Control and Physical Activity*. Lyon, France: IARC Press; 2002.
167. Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. *N Engl J Med*. 1999;341:427–434.
168. McTigue KM, Hess R, Ziouras J. Obesity in older adults: a systematic review of the evidence for diagnosis and treatment. *Obesity*. 2006;14:1485–1497.
169. Elmer PJ, Obarzanek E, Vollmer WM, Simons-Morton D, Rohm-Young D, Lin PH, Champagne C, Harsha DW, Svetkey LP, Ard J, Brantley PJ, Proschan MA, Erlinger TP, Stevens VJ, Appel LJ; PREMIER Collaborative Research Group. Effects of comprehensive lifestyle modification on diet, weight, physical fitness, and blood pressure control: 18-month results of a randomized trial. *Ann Intern Med*. 2006;144:485–495.
170. Orchard TJ, Temprosa M, Goldberg R, Haffner S, Ratner R, Marcovina S, Fowler S; Diabetes Prevention Program Research Group. The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: the Diabetes Prevention Program randomized trial. *Ann Intern Med.* 2005;142:611–619.
171. Barker DJP. *Mothers, Babies, and Disease in Later Life*. 2nd ed. London, UK: Harcourt Brace & Co, Ltd; 1998.
172. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11:496–506.
173. Gillman MW, Rifas-Shiman SL, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight, and adolescent obesity. *Pediatrics*. 2003;111:e221–e226.
174. Oken E, Taveras EM, Kleinman K, Rich-Edwards JW, Gillman MW. Gestational weight gain and child adiposity at age 3 years. *Am J Obstet Gynecol*. 2007;196:322.e1–e8.
175. Bavdekar A, Yajnik CS, Fall CH, Bapat S, Pandit AN, Deshpande V, Bhave S, Kellingray SD, Joglekar C. Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? *Diabetes*. 1999;48:2422–2429.
176. Adair LS, Cole TJ. Rapid child growth raises blood pressure in adolescent boys who were thin at birth. *Hypertension*. 2003;41:451–456.
177. Valdez R, Athens MA, Thompson GH, Bradshaw BS, Stern MP. Birthweight and adult health outcomes in a biethnic population in the USA. *Diabetologia*. 1994;37:624–631.
178. Frankel S, Elwood P, Sweetnam P, Yarnell J, Davey Smith G. Birthweight, body-mass index in middle age, and incident coronary heart disease. *Lancet*. 1996;348:1478–1480.
179. Rich-Edwards JW, Kleinman K, Michels KB, Stampfer MJ, Manson JE, Rexrode KM, Hibert EN, Willett WC. Longitudinal study of birth weight and adult body mass index in predicting risk of coronary heart disease and stroke in women. *BMJ*. 2005;330:1115.
180. Barker DJP, Osmond C, Forsen TJ, Kajantie E, Eriksson JG. Trajectories of growth among children who later have coronary events. *N Engl J Med*. 2005;353:1802–1809.
181. Bhargava SK, Sachdev HS, Fall CH, Osmond C, Lakshmy R, Barker DJ, Biswas SK, Ramji S, Prabhakaran D, Reddy KS. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. *N Engl J Med*. 2004;350:865–875.
182. Gillman MW. Developmental origins of health and disease. *N Engl J Med*. 2005;353:1848–1850.
183. Oken E, Huh SY, Taveras EM, Rich-Edwards JW, Gillman MW. Associations of maternal prenatal smoking with child adiposity and blood pressure. *Obes Res*. 2005;13:2021–2028.
184. Daniels SR. The consequences of childhood overweight and obesity. *Future Child*. 2006;16:47–67.

185. Writing Group for the SEARCH for Diabetes in Youth Study Group, Dabelea D, Bell RA, D'Agostino RB Jr, Imperatore G, Johansen JM, Linder B, Liu LL, Loots B, Marcovina S, Mayer-Davis EJ, Pettitt DJ, Waitzfelder B. Incidence of diabetes in youth in the United States. *JAMA*. 2007;297:2716–2724.
186. Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among U.S. adolescents, 1999–2000. *Diabetes Care*. 2004;27:2438–2443.
187. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoye M, Morrison J, Sherwin RS, Caprio S. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med*. 2004;350:2362–2374.
188. Morrison JA, Friedman LA, Harlan WR, Harlan LC, Barton BA, Schreiber GB, Klein DJ. Development of the metabolic syndrome in black and white adolescent girls: a longitudinal assessment. *Pediatrics*. 2005;116:1178–1182.
189. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics*. 1999;103:1175–1182.
190. Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity, and lipids in prepubertal children. *Diabetes*. 1999;48:1515–1521.
191. Morrison JA, Sprecher DL, Barton BA, Waclawiw MA, Daniels SR. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr*. 1999;135:458–464.
192. Thompson DR, Obarzanek E, Franko DL, Barton BA, Morrison J, Biro FM, Daniels SR, Striegel-Moore RH. Childhood overweight and cardiovascular disease risk factors: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr*. 2007;150:18–25.
193. Gold DR, Damokosh AI, Dockery DW, Berkey CS. Body-mass index as a predictor of incident asthma in a prospective cohort of children. *Pediatr Pulmonol*. 2003;36:514–521.
194. Akinbami L. *The State of Childhood Asthma, United States, 1980–2005*. Advance data from Vital and Health Statistics (No. 381). Hyattsville, MD: National Center for Health Statistics; 2006.
195. Lozano P, Sullivan SD, Smith DH, Weiss KB. The economic burden of asthma in US children: estimates from the National Medical Expenditure Survey. *J Allergy Clin Immunol*. 1999;104:957–963.
196. Wang G, Dietz WH. Economic burden of obesity in youths aged 6 to 17 years: 1979–1999. *Pediatrics*. 2002;109:e81.
197. Gilliland FD, Berhane K, Islam T, McConnell R, Gauderman WJ, Gilliland SS, Avol E, Peters JM. Obesity and the risk of newly diagnosed asthma in school-age children. *Am J Epidemiol*. 2003;158:406–415.
198. Taveras EM, Rifas-Shiman SL, Gold DR, Camargo CA Jr, Oken E, Weiss ST, Gillman MW. Infant weight-for-length as a predictor of wheeze in early childhood. *Pediatr Res*. 2005;58:1029.
199. Tantisira KG, Weiss ST. Complex interactions in complex traits: obesity and asthma. *Thorax*. 2001;56(suppl 2):ii64–ii73.
200. Davison KK, Birch LL. Weight status, parent reaction, and self-concept in five-year-old girls. *Pediatrics*. 2001;107:46–53.
201. Neumark-Sztainer D, Falkner N, Story M, Perry C, Hannan PJ, Mulert S. Weight-teasing among adolescents: correlations with weight status and disordered eating behaviors. *Int J Obes Relat Metab Disord*. 2002;26:123–131.
202. Hayden-Wade H, Stein R, Ghaderi A, Saelens B, Zabinski M, Wilfley D. Prevalence, characteristics, and correlates of teasing experiences among overweight children vs. non-overweight peers. *Obes Res*. 2005;13:1381–1392.
203. Falkner NH, Neumark-Sztainer D, Story M, Jeffery RW, Beuhring T, Resnick MD. Social, educational, and psychological correlates of weight status in adolescents. *Obes Res*. 2001;9:32–42.
204. Strauss RS, Pollack HA. Social marginalization of overweight children. *Arch Pediatr Adolesc Med*. 2003;157:746–752.
205. Strauss RS. Childhood obesity and self-esteem. *Pediatrics*. 2000;105:e15.
206. Biro F, Striegel-Moore R, Franko D, Padgett J, Bean J. Self-esteem in adolescent females. *J Adolesc Health*. 2006;39:501–507.
207. Field AE, Camargo CA Jr, Taylor CB, Berkey CS, Frazier AL, Gillman MW, Colditz GA. Overweight, weight concerns, and bulimic behaviors among girls and boys. *J Am Acad Child Adolesc Psychiatry*. 1999;38:754–760.
208. Ackard DM, Neumark-Sztainer D, Story M, Perry C. Overeating among adolescents: prevalence and associations with weight-related characteristics and psychological health. *Pediatrics*. 2003;111:67–74.
209. Boutelle K, Neumark-Sztainer D, Story M, Resnick M. Weight control behaviors among obese, overweight, and nonoverweight adolescents. *J Pediatr Psychol*. 2002;27:531–540.
210. Neumark-Sztainer D, Story M, Hannan PJ, Perry CL, Irving LM. Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders. *Arch Pediatr Adolesc Med*. 2002;156:171–178.
211. Field AE, Austin SB, Taylor CB, Malspeis S, Rosner B, Rockett HR, Gillman MW, Colditz GA. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics*. 2003;112:900–906.
212. Stunkard AJ, Allison KC. Two forms of disordered eating in obesity: binge eating and night eating. *Int J Obes Relat Metab Disord*. 2003;27:1–12.
213. Friedlander SL, Larkin EK, Rosen CL, Palermo TM, Redline S. Decreased quality of life associated with obesity in school-aged children. *Arch Pediatr Adolesc Med*. 2003;157:1206–1211.
214. Swallen K, Reither E, Haas S, Meier A. Overweight, obesity, and health-related quality of life among adolescents: the National Longitudinal Study of Adolescent Health. *Pediatrics*. 2005;115:340–347.
215. Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. *JAMA*. 2003;289:1813–1819.
216. Guo SS, Huang C, Maynard LM, Demerath E, Towne B, Chumlea WC, Siervogel RM. Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord*. 2000;24:1628–1635.
217. Lauer RM, Clarke WR. Childhood risk factors for high adult blood pressure: the Muscatine Study. *Pediatrics*. 1989;84:633–641.
218. Magarey AM, Daniels LA, Boulton TJ, Cockington RA. Predicting obesity in early adulthood from childhood and parental obesity. *Int J Obes Relat Metab Disord*. 2003;27:505–513.
219. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med*. 1993;22:167–177.
220. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337:869–873.
221. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics*. 2001;108:712–718.
222. Field AE, Cook NR, Gillman MW. Weight status in childhood as a predictor of becoming overweight or hypertensive in early adulthood. *Obes Res*. 2005;13:163–169.
223. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med*. 1992;327:1350–1355.
224. Gunnell DJ, Frankel SJ, Nanchahal K, Peters TJ, Davey Smith G. Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd Orr cohort. *Am J Clin Nutr*. 1998;67:1111–1118.
225. Hoffmans MD, Kromhout D, Coulander CD. Body mass index at the age of 18 and its effects on 32-year-mortality from coronary heart disease and cancer: a nested case-control study among the entire 1932 Dutch male birth cohort. *J Clin Epidemiol*. 1989;42:513–520.
226. van Dam RM, Willett WC, Manson JE, Hu FB. The relationship between overweight in adolescence and premature death in women. *Ann Intern Med*. 2006;145:91–97.
227. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med*. 1995;122:481–486.
228. Morrison JA, Friedman LA, Gray-McGuire C. Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: the Princeton Lipid Research Clinics Follow-up Study. *Pediatrics*. 2007;120:340–345.
229. Yarnell JW, Patterson CC, Thomas HF, Sweetnam PM. Comparison of weight in middle age, weight at 18 years, and weight change between, in predicting subsequent 14 year mortality and coronary events: Caerphilly Prospective Study. *J Epidemiol Community Health*. 2000;54:344–348.
230. Huang Z, Willett WC, Manson JE, Rosner B, Stampfer MJ, Speizer FE, Colditz GA. Body weight, weight change, and risk for hypertension in women. *Ann Intern Med*. 1998;128:81–88.

231. Srinivasan SR, Bao W, Wattigney WA, Berenson GS. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism*. 1996;45:235–240.
232. Laitinen J, Taponen S, Martikainen H, Pouta A, Millwood I, Hartikainen AL, Ruokonen A, Sovio U, McCarthy MI, Franks S, Järvelin MR. Body size from birth to adulthood as a predictor of self-reported polycystic ovary syndrome symptoms. *Int J Obes Relat Metab Disord*. 2003;27:710–715.
233. Rich-Edwards JW, Goldman MB, Willett WC, Hunter DJ, Stampfer MJ, Colditz GA, Manson JE. Adolescent body mass index and infertility caused by ovulatory disorder. *Am J Obstet Gynecol*. 1994;171:171–177.
234. Lubin F, Chetrit A, Freedman LS, Alfandary E, Fishler Y, Nitzan H, Zultan A, Modan B. Body mass index at age 18 years and during adult life and ovarian cancer risk. *Am J Epidemiol*. 2003;157:113–120.
235. Fairfield KM, Willett WC, Rosner BA, Manson JE, Speizer FE, Hankinson SE. Obesity, weight gain, and ovarian cancer. *Obstet Gynecol*. 2002;100:288–296.
236. Kumanyika SK, Obarzanek E. Pathways to obesity prevention: report of a National Institutes of Health workshop. *Obes Res*. 2003;11:1263–1274.
237. Cawley J. The cost-effectiveness of programs to prevent or reduce obesity: the state of the literature and a future research agenda. *Arch Pediatr Adolesc Med*. 2007;161:611–614.
238. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science*. 2003;299:853–855.
239. Simkin-Silverman LR, Wing RR, Boraz MA, Kuller LH. Lifestyle intervention can prevent weight gain during menopause: results from a 5-year randomized clinical trial. *Ann Behav Med*. 2003;26:212–220.
240. Dietz WH, Gortmaker SL. Preventing obesity in children and adolescents. *Annu Rev Public Health*. 2001;22:337–353.
241. Lewis CE, Jacobs DR Jr, McCreath H, Kiefe CI, Schreiner PJ, Smith DE, Williams OD. Weight gain continues in the 1990s: 10-year trends in weight and overweight from the CARDIA study: coronary artery risk development in young adults. *Am J Epidemiol*. 2000;151:1172–1181.
242. Woods SC, Seeley RJ, Porte D Jr, Schwartz MW. Signals that regulate food intake and energy homeostasis. *Science*. 1998;280:1378–1383.
243. Forbes GB. Body fat content influences the body composition response to nutrition and exercise. *Ann N Y Acad Sci*. 2000;904:359–365.
244. Schoeller DA, Buccholz AC. Energetics of obesity and weight control: does diet composition matter? *J Am Diet Assoc*. 2005;105(suppl 1):S24-S28.
245. Gardner CD, Kiazand A, Alhassan S, Kim S, Stafford RS, Balise RR, Kraemer HC, King AC. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA*. 2007;297:969–997.
246. Foster GD, Makris AP, Bailer BA. Behavioral treatment of obesity. *Am J Clin Nutr*. 2005;82(suppl 1):230S–235S.
247. Jeffery RW, Drewnowski A, Epstein LH, Stunkard AJ, Wilson GT, Wing RR, Hill R. Long-term maintenance of weight loss: current status. *Health Psychol*. 2000;19(suppl 1):5–16.
248. Rothman AJ. Toward a theory-based analysis of behavioral maintenance. *Health Psychol*. 2000;19(suppl 1):64–69.
249. Wing RR, Phelan S. Long-term weight loss maintenance. *Am J Clin Nutr*. 2005;82(suppl 1):222S–225S.
250. Grundy SM. Multifactorial causation of obesity: implications for prevention. *Am J Clin Nutr*. 1998;67(suppl 3):563S–572S.
251. US Department of Health and Human Services, US Department of Agriculture. *Dietary Guidelines for Americans, 2005*. Available at: http://www.health.gov/dietaryguidelines/dga2005/recommendations.htm. Accessed May 8, 2008.
252. Wing RR, Tate DF, Gorin AA, Raynor HA, Fava JL. A self-regulation program for maintenance of weight loss. *N Engl J Med*. 2006;355:1563–1571.
253. Linde JA, Jeffery RW, French SA, Pronk NP, Boyle RG. Self-weighing in weight gain prevention and weight loss trials. *Ann Behav Med*. 2005;30:210–216.
254. Jakicic JM. The role of physical activity in prevention and treatment. *J Nutr*. 2002;132:3826S–3829S.
255. Institute of Medicine. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients)*. Washington, DC: The National Academies Press; 2005.
256. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Med Sci Sports Exerc*. 2007;39:1423–1434.
257. Booth SL, Sallis JF, Ritenbaugh C, Hill JO, Birch LL, Frank LD, Glanz K, Himmelgreen DA, Mudd M, Popkin BM, Rickard KA, St Jeor S, Hays NP. Environmental and societal factors affect food choice and physical activity: rationale, influences, and leverage points. *Nutr Rev*. 2001;59:S21–S39; discussion S57–S65.
258. French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health*. 2001;22:309–335.
259. Diez Roux AV, Jacobs DR, Kiefe CI; Coronary Artery Risk Development in Young Adults (CARDIA) Study. Neighborhood characteristics and components of the insulin resistance syndrome in young adults: the coronary artery risk development in young adults (CARDIA) study. *Diabetes Care*. 2002;25:1976–1982.
260. Handy SL, Boarnet MG, Ewing R, Killingsworth RE. How the built environment affects physical activity: views from urban planning. *Am J Prev Med*. 2002;23(suppl 2):64–73.
261. Wadden TA, Brownell KD, Foster GD. Obesity: responding to the global epidemic. *J Consult Clin Psychol*. 2002;70:510–525.
262. Sallis JF, Glanz K. The role of built environments in physical activity, eating, and obesity in childhood. *Future Child*. 2006;16:89–108.
263. McGinnis JM, Gootman JA, Kraak VI, eds; Committee on Food Marketing and the Diets of Children and Youth, Food Marketing to Children and Youth; Institute of Medicine. *Threat or Opportunity*? Washington, DC: National Academies Press; 2006.
264. Rose G. *The Strategy of Preventive Medicine*. Oxford, UK: Oxford University Press; 1992.
265. Lin BH, Guthrie J, Frazao E, eds. *Nutrient Contribution of Food Away from Home: America's Eating Habits: Changes and Consequences*. Economic Research Service Report, Agriculture Information Bulletin No. 750; 1999:213–239.
266. French S, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. *Int J Obes Relat Metab Disord*. 2000;24:1353–1359.
267. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. *Lancet*. 2005;365:36–42.
268. Smiciklas-Wright H, Mitchell DC, Mickle SJ, Goldman JD, Cook A. Foods commonly eaten in the United States, 1989–1991 and 1994–1996: are portion sizes changing? *J Am Diet Assoc*. 2003;103:41–47.
269. Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health*. 2002;92:246–294.
270. Poppitt SD, Prentice AM. Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite*. 1996;26:153–174.
271. Kral TV, Rolls BJ. Energy density and portion size: their independent and combined effects on energy intake. *Physiol Behav*. 2004;82:131–138.
272. Ledikwe JH, Rolls BJ, Smiciklas-Wright H, Mitchell DC, Ard JD, Champagne C, Karanja N, Lin PH, Stevens VJ, Appel LJ. Reductions in dietary energy density are associated with weight loss in overweight and obese participants in the PREMIER trial. *Am J Clin Nutr*. 2007;85:1212–1221.
273. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet*. 2001;357:505–508.
274. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr*. 2004;79:537–543.
275. Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willet WC, Hu FB. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA*. 2004;292:927–934.
276. Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics*. 2006;117:673–680.
277. Centers for Disease Control and Prevention (CDC). Adult participation in recommended levels of physical activity—United States, 2001 and 2003. *MMWR Morb Mortal Wkly Rep*. 2005;54:1208–1212.

278. DiPietro L. Physical activity, body weight, and adiposity: an epidemiologic perspective. *Exerc Sports Sci Rev*. 1995;23:275–303.
279. Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain—a systematic review. *Obes Rev*. 2000;1:95–111.
280. Littman AJ, Kristal AR, White E. Effects of obligatory physical activity intensity, frequency, and activity type on 10-y weight change in middle-aged men and women. *Int J Obes Relat Metab Disord*. 2005;29:524–533.
281. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA*. 2003;289:1785–1791.
282. Tucker LA, Friedman GM. Television viewing and obesity in adult males. *Am J Public Health*. 1989;79:516–518.
283. Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986–1990. *Arch Pediatr Adolesc Med*. 1996;150:356–362.
284. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA*. 1999;282:1561–1567.
285. Taylor AH, Cable NT, Faulkner G, Hillsdon M, Narici M, van der Biz AK. Physical activity and older adults: a review of health benefits and the effectiveness of interventions. *J Sports Sci*. 2004;22:703–725.
286. Prentice A, Jebb S. Energy intake/physical activity interactions in the homeostasis of body weight regulation. *Nutr Rev*. 2004;62:S98–S104.
287. Epstein LH, Saelens BE. Behavioral economics of obesity: food intake and energy expenditure. In: Bickel WK, Vuchinich RE, eds. *Reframing Health Behavior Change with Behavioral Economics*. Mahwah, NJ: Lawrence Erlbaum Associates; 2000.
288. Yancey AK, Fielding JE, Flores GR, Sallis JF, McCarthy WJ, Breslow L. Creating a robust public health infrastructure for physical activity promotion. *Am J Prev Med*. 2007;32:68–78.
289. Lopez R. Urban sprawl and risk for being overweight or obese. *Am J Public Health*. 2004;94:1574–1579.
290. Hayne CL, Moran PA, Ford MM. Regulating environments to reduce obesity. *J Public Health Policy*. 2004;25:391–407.
291. Ewing R, Schmid T, Killingsworth R, Zlot A, Raudenbush S. Relationship between urban sprawl and physical activity, obesity, and morbidity. *Am J Health Promot*. 2003;18:47–57.
292. Vandegrift D, Yoked T. Obesity rates, income, and suburban sprawl: an analysis of US states. *Health Place*. 2004;10:221–229.
293. Giles-Corti B, Macintyre S, Clarkson JP, Pikora T, Donovan RJ. Environmental and lifestyle factors associated with overweight and obesity in Perth, Australia. *Am J Health Promot*. 2003;18:93–102.
294. Saelens BE, Sallis JF, Black JB, Chen D. Neighborhood-based differences in physical activity: an environment scale evaluation. *Am J Public Health*. 2003;93:1552–1558.
295. Frank LD, Andresen MA, Schmid TL. Obesity relationships with community design, physical activity, and time spent in cars. *Am J Prev Med*. 2004;27:87–96.
296. Legato MJ. Gender-specific aspects of obesity. *Int J Fertil Womens Med*. 1997;42:184–197.
297. Lovejoy JC. The influence of sex hormones on obesity across the female life span. *J Womens Health*. 1998;7:1247–1256.
298. Gunderson EP, Murtaugh MA, Lewis CE, Quesenberry CP, West DS, Sidney S. Excess gains in weight and waist circumference associated with childbearing: The Coronary Artery Risk Development in Young Adults Study (CARDIA). *Int J Obes Relat Metab Disord*. 2004;28:525–535.
299. Glanz K, Basil M, Maibach E, Goldberg J, Snyder D. Why Americans eat what they do: taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. *J Am Diet Assoc*. 1998;98:1118–1126.
300. Girois SB, Kumanyika SK, Morabia A, Mauger E. A comparison of knowledge and attitudes about diet and health among 35- to 75-year-old adults in the United States and Geneva, Switzerland. *Am J Public Health*. 2001;91:418–424.
301. Bowman SA. Food shoppers' nutrition attitudes and relationship to dietary and lifestyle practices. *Nutr Res*. 2005;25:281–293.
302. Satia JA, Galanko JA, Neuhouser ML. Food nutrition label use is associated with demographic, behavioral, and psychosocial factors and dietary intake among African Americans in North Carolina. *J Am Diet Assoc*. 2005;105:392–402; discussion 402–413.
303. Gorman JM. Gender differences in depression and response to psychotropic medication. *Gend Med*. 2006;3:93–109.
304. Daumit GL, Clark JM, Steinwachs DM, Graham CM, Lehman A, Ford DE. Prevalence and correlates of obesity in a community sample of individuals with severe and persistent mental illness *J Nerv Ment Dis*. 2003;191:799–805.
305. Gentile S. Long-term treatment with atypical antipsychotics and the risk of weight gain: a literature analysis. *Drug Saf*. 2006;29:303–319.
306. Adam T, Epel ES. Stress, eating and the reward system. *Physiol Behav*. 2007;91:449–458.
307. Zellner DA, Loaiza S, Gonzalez Z, Pita J, Morales J, Pecora D, Wolf A. Food selection changes under stress. *Physiol Behav*. 2006;87:789–793.
308. Kimm SY, Glynn NW, Kriska AM, Barton BA, Kronsberg SS, Daniels SR, Crawford PB, Sabry ZI, Liu K. Decline in physical activity in black girls and white girls during adolescence. *N Engl J Med*. 2002;347:709–715.
309. Centers for Disease Control and Prevention (CDC). Prevalence of leisure-time and occupational physical activity among employed adults—United States, 1990. *MMWR Morb Mortal Wkly Rep*. 2000;49:420–424.
310. King AC, Castro C, Wilcox S, Eyler AA, Sallis JF, Brownson RC. Personal and environmental factors associated with physical inactivity among different racial-ethnic groups of U.S. middle-aged and older-aged women. *Health Psychol*. 2000;19:354–364.
311. Eyler AA, Matson-Koffman D, Vest JR, Evenson KR, Sanderson B, Thompson JL, Wilbur J, Wilcox S, Young DR. Environmental, policy, and cultural factors related to physical activity in a diverse sample of women: the Women's Cardiovascular Health Network Project—summary and discussion. *Women Health*. 2002;36:123–134.
312. Barnes AS, Goodrick GK, Pavlik V, Markesino J, Laws DY, Taylor WC. Weight loss maintenance in African-American women: focus group results and questionnaire development. *J Gen Intern Med*. 2007;22:915–922.
313. Bish CL, Blanck HM, Serdula MK, Marcus M, Kohl HW 3rd, Khan LK. Diet and physical activity behaviors among Americans trying to lose weight: 2000 Behavioral Risk Factor Surveillance System. *Obes Res*. 2005;13:596–607.
314. Rodin J, Silberstein L, Striegel-Moore R. Women and weight: a normative discontent. *Nebr Symp Motiv*. 1984;32:267–307.
315. Smith DE, Lewis CE, Caveny JL, Perkins LL, Burke GL, Bild DE. Longitudinal changes in adiposity associated with pregnancy: The Coronary Artery Risk Development in Young Adults Study. *JAMA*. 1994;271:1747–1751.
316. Rosenberg L, Palmer JR, Wise LA, Horton NJ, Kumanyika SK, Adams-Campbell LL. A prospective study of the effect of childbearing on weight gain in African-American women. *Obes Res*. 2003;11:1526–1535.
317. Sidney S, Sternfeld B, Haskell WL, Jacobs DR Jr, Chesney MA, Hulley SB. Television viewing and cardiovascular risk factors in young adults: the CARDIA study. *Ann Epidemiol*. 1996;6:154–159.
318. Rimmer JH, Wang E. Obesity prevalence among a group of Chicago residents with disabilities. *Arch Phys Med Rehabil*. 2005;86:1461–1464.
319. Yamaki K. Body weight status among adults with intellectual disability in the community. *Ment Retard*. 2005;43:1–10.
320. Finch C, Owen N, Price R. Current injury or disability as a barrier to being more physically active. *Med Sci Sports Exerc*. 2001;33:778–782.
321. Prasher VP. Overweight and obesity amongst Down's syndrome adults. *J Intellect Disabil Res*. 1995;39:437–441.
322. Fonseca CT, Amaral DM, Ribeiro MG, Beserra IC, Guimarães MM. Insulin resistance in adolescents with Down syndrome: a cross-sectional study. *BMC Endocr Disord*. 2005;5:6.
323. Rubin SS, Rimmer JH, Chicoine B, Braddock D, McGuire DE. Overweight prevalence in persons with Down syndrome. *Ment Retard*. 1998;36:175–181.
324. Coodin S. Body mass index in persons with schizophrenia. *Can J Psychiatry*. 2001;46:549–555.
325. Holland AJ, Wong J. Genetically determined obesity in Prader-Willi syndrome: the ethics and legality of treatment. *J Med Ethics*. 1999;25:230–236.
326. Barker M, Robinson S, Osmond C, Barker DJ. Birth weight and body fat distribution in adolescent girls. *Arch Dis Child*. 1997;77:381–383.
327. Stettler N, Stallings VA, Troxel AB, Zhao J, Schinnar R, Nelson SE, Ziegler EE, Strom BL. Weight gain in the first week of life and overweight in adulthood: a cohort study of European American subjects fed infant formula. *Circulation*. 2005;111:1897–1903.
328. Cusatis DC, Chinchilli VM, Johnson-Rollings N, Kieselhorst K, Stallings VA, Lloyd T. Longitudinal nutrient intake patterns of US

adolescent women: the Penn State Young Women's Health Study. *J Adolesc Health*. 2000;26:194–204.
329. Rajeshwari R, Nicklas TA, Yang SJ, Berenson GS. Longitudinal changes in intake and food sources of calcium from childhood to young adulthood: the bogalusa heart study. *J Am Coll Nutr*. 2004;23:341–350.
330. Kvaavik E, Andersen LF, Klepp KI. The stability of soft drinks intake from adolescence to adult age and the association between long-term consumption of soft drinks and lifestyle factors and body weight. *Public Health Nutr*. 2005;8:149–157.
331. Nelson MC, Gordon-Larsen P, Adair LS, Popkin BM. Adolescent physical activity and sedentary behavior: patterning and long-term maintenance. *Am J Prev Med*. 2005;28:259–266.
332. Raitakari OT, Porkka KV, Taimela S, Telama R, Räsänen L, Viikari JS. Effects of persistent physical activity and inactivity on coronary risk factors in children and young adults: the Cardiovascular Risk in Young Finns Study. *Am J Epidemiol*. 1994;140:195–205.
333. Stein LJ, Cowart BJ, Epstein AN, Pilot LJ, Laskin CR, Beauchamp GK. Increased liking for salty foods in adolescents exposed during infancy to a chloride-deficient feeding formula. *Appetite*. 1996;27:65–77.
334. Birch LL. Development of food preferences. *Annu Rev Nutr*. 1999;19:41–62.
335. Gillman MW. Lifecourse approach to obesity. In: Kuh D, Ben-Schlomo Y, eds. *A Life Course Approach to Chronic Disease Epidemiology 2*. London, UK: Oxford University Press; 2004:189–217.
336. Institute of Medicine. *Nutrition During Pregnancy*. Washington, DC: National Academy Press; 1990.
337. Gortmaker SL, Peterson K, Wiecha J, Sobol AM, Dixit S, Fox MK, Laird N. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Arch Pediatr Adolesc Med*. 1999;153:409–418.
338. Sallis JF, McKenzie TL, Alcaraz JE, Kolody B, Hovell MF, Nader PR. Project SPARK. Effects of physical education on adiposity in children. *Ann N Y Acad Sci*. 1993;691:127–136.
339. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ*. 2004;328:1237.
340. Reilly JJ, Kelly L, Montgomery C, Williamson A, Fisher A, McColl JH, Lo Conte R, Paton JY, Grant S. Physical activity to prevent obesity in young children: cluster randomised controlled trial. *BMJ*. 2006;333:1041–1045.
341. Story M, Kaphingst KM, French S. The role of child care settings in obesity prevention. *Future Child*. 2006;16:143–168.
342. Dennison BA, Russo TJ, Burdick PA, Jenkins PL. An intervention to reduce television viewing by preschool children. *Arch Pediatr Adolesc Med*. 2004;158:170–176.
343. Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, Kaufer Christoffel K, Dyer A. Two-year follow-up results for Hip-Hop to Health Jr: a randomized controlled trial for overweight prevention in preschool minority children. *J Pediatr*. 2005;146:618–625.
344. Story MT, Neumark-Stzainer DR, Sherwood NE, Holt K, Sofka D, Trowbridge FL, Barlow SE. Management of child and adolescent obesity: attitudes, barriers, skills, and training needs among health care professionals. *Pediatrics*. 2002;110:210–214.
345. Tershakovec AM, Watson MH, Wenner WJ Jr, Marx AL. Insurance reimbursement for the treatment of obesity in children. *J Pediatr*. 1999;134:573–578.
346. Hogan DP, Msall ME, Rogers ML, Avery RC. Improved disability population estimates of functional limitation among American children aged 5–17. *Matern Child Health J*. 1997;1:203–216.
347. Bandini LG, Curtin C, Hamad C, Tybor DJ, Must A. Prevalence of overweight in children with developmental disorders in the continuous national health and nutrition examination survey (NHANES) 1999–2002. *J Pediatr*. 2005;146:738–743.
348. Sullivan PB, Alder N, Bachlet AM, Grant H, Juszczak E, Henry J, Vernon-Roberts A, Warner J, Wells J. Gastrostomy feeding in cerebral palsy: too much of a good thing? *Dev Med Child Neurol*. 2006;48:877–882.
349. Day SM, Strauss DJ, Vachon PJ, Rosenbloom L, Shavelle RM, Wu YW. Growth patterns in a population of children and adolescents with cerebral palsy. *Dev Med Child Neurol*. 2007;49:167–171.
350. Stanish HI, Temple VA, Frey GC. Health-promoting physical activity of adults with mental retardation. *Ment Retard Dev Disabil Res Rev*. 2006;12:13–21.
351. Dykens EM, Rosner BA, Butterbaugh G. Exercise and sports in children and adolescents with developmental disabilities: positive physical and psychosocial effects. *Child Adolesc Psychiatr Clin N Am*. 1998;7:757–771.
352. Taylor WC, Poston WSC, Jones L, Kraft MK. Environmental justice: obesity, physical activity, and healthy eating. *J Phys Activity Health*. 2006;3(suppl 1):s30–s54.
353. Day K. Active living and social justice: planning for physical activity in low-income, black, and Latino communities. *J Am Planning Assoc*. 2006;72:88–99.
354. Yancey AK, Ory M, Davis SM. Dissemination of physical activity promotion interventions in underserved populations. *Am J Prev Med*. 2006;31:S82–S91.
355. Koplan JP, Liverman CT, Kraak VI, Wisham SL, eds. *Progress in Preventing Childhood Obesity, How Do We Measure Up*? Washington, DC: The National Academies Press; 2006.
356. Kumanyika S. Environmental influences on childhood obesity: ethnic and cultural influences in context. *Physiol Behav*. 2008;94:61–70.
357. Sánchez-Johnsen LA, Fitzgibbon ML, Martinovich Z, Stolley MR, Dyer AR, Van Horn L. Ethnic differences in correlates of obesity between Latin-American and black women. *Obes Res*. 2004;12:652–660.
358. Patt MR, Yanek LR, Moy TF, Becker DM. Sociodemographic, behavioral, and psychological correlates of current overweight and obesity in older, urban African American women. *Health Educ Behav*. 2004;31(suppl 4):57S–68S.
359. Satia JA, Galanko JA, Siega-Riz AM. Eating at fast-food restaurants is associated with dietary intake, demographic, psychosocial and behavioural factors among African Americans in North Carolina. *Public Health Nutr*. 2004;7:1089–1096.
360. Block JP, Scribner RA, DeSalvo KB. Fast food, race/ethnicity, and income: a geographic analysis. *Am J Prev Med*. 2004;27:211–217.
361. Schmidt M, Affenito SG, Striegel-Moore R, Khoury PR, Barton B, Crawford P, Kronsberg S, Schreiber G, Obarzanek E, Daniels S. Fast-food intake and diet quality in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *Arch Pediatr Adolesc Med*. 2005;159:626–631.
362. Lowry R, Wechsler H, Galuska DA, Fulton JE, Kann L. Television viewing and its associations with overweight, sedentary lifestyle, and insufficient consumption of fruits and vegetables among US high school students: differences by race, ethnicity, and gender. *J Sch Health*. 2002;72:413–421.
363. Katz ML, Gordon-Larsen P, Bentley ME, Kelsey K, Shields K, Ammerman A. 'Does skinny mean healthy?' Perceived ideal, current, and healthy body sizes among African-American girls and their female caregivers. *Ethn Dis*. 2004;14:533–541.
364. Gipson GW, Reese S, Vieweg WV, Anum EA, Pandurangi AK, Olbrisch ME, Sood B, Silverman JJ. Body image and attitude toward obesity in an historically black university. *J Natl Med Assoc*. 2005;97:225–236.
365. Perry AC, Rosenblatt EB, Wang X. Physical, behavioral, and body image characteristics in a tri-racial group of adolescent girls. *Obes Res*. 2004;12:1670–1679.
366. Welch C, Gross SM, Bronner Y, Dewberry-Moore N, Paige DM. Discrepancies in body image perception among fourth-grade public school children from urban, suburban, and rural Maryland. *J Am Diet Assoc*. 2004;104:1080–1085.
367. Fitzgibbon ML, Blackman LR, Avellone ME. The relationship between body image discrepancy and body mass index across ethnic groups. *Obes Res*. 2000;8:582–589.
368. Yancey AK, Simon PA, McCarthy WJ, Lightstone AS, Fielding JE. Ethnic and sex variations in overweight self-perception: relationship to sedentariness. *Obesity* (*Silver Spring*). 2006;14:980–988.
369. Kumanyika SK. Obesity treatment in minorities. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment.* 3rd ed. New York, NY: Guilford Publications, Inc; 2002:416–446.
370. Wing RR, Hamman RF, Bray GA, Delahanty L, Edelstein SL, Hill JO, Horton ES, Hoskin MA, Kriska A, Lachin J, Mayer-Davis EJ, Pi-Sunyer X, Regensteiner JG, Venditti B, Wylie-Rosett J; Diabetes Prevention Program Research Group. Achieving weight and activity goals among diabetes prevention program lifestyle participants. *Obes Res*. 2004;12:1426–1434.
371. Svetkey LP, Erlinger TP, Vollmer WM, Feldstein A, Cooper LS, Appel LJ, Ard JD, Elmer PJ, Harsha D, Stevens VJ. Effect of lifestyle modifications on blood pressure by race, sex, hypertension status, and age. *J Hum Hypertens*. 2005;19:21–31.
372. Kumanyika S, Grier S. Targeting interventions for ethnic minority and low-income populations. *Future Child*. 2006;16:187–207.

373. Powell LM, Slater S, Chaloupka FJ. The relationship between community physical activity settings and race, ethnicity and socioeconomic status. *Evidence-Based Prev Med*. 2004;1:135–144.
374. Henderson VR, Kelly B. Food advertising in the age of obesity: content analysis of food advertising on general market and African American television. *J Nutr Educ Behav*. 2005;37:191–196.
375. Gordon-Larsen P, Nelson MC, Page P, Popkin BM. Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics*. 2006;117:417–424.
376. Unger JB, Reynolds K, Shakib S, Spruijt-Metz D, Sun P, Johnson CA. Acculturation, physical activity, and fast-food consumption among Asian-American and Hispanic adolescents. *J Community Health*. 2004; 29:467–481.
377. Gordon-Larsen P, Harris KM, Ward DS, Popkin BM; National Longitudinal Study of Adolescent Health. Acculturation and overweight-related behaviors among Hispanic immigrants to the US: the National Longitudinal Study of Adolescent Health. *Soc Sci Med*. 2003;57: 2023–2034.
378. Chen JL, Kennedy C. Factors associated with obesity in Chinese-American children. *Pediatr Nurs*. 2005;31:110–115.
379. Morland K, Wing S, Diez Roux A. The contextual effect of the local food environment on residents' diets: the atherosclerosis risk in communities study. *Am J Public Health*. 2002;92:1761–1767.
380. Morland K, Wing S, Diez Roux A, Poole C. Neighborhood characteristics associated with the location of food stores and food service places. *Am J Prev Med*. 2002;22:23–29.
381. Zenk AN, Schulz AJ, Israel BA, James SA, Bao S, Wilson ML. Neighborhood racial composition, neighborhood poverty, and spatial accessibility of supermarkets in metropolitan Detroit. *Am J Public Health*. 2005;95:660–667.
382. Drewnowski A. The real contribution of added sugars and fats to obesity. *Epidemiol Rev*. 2007;29:160–171.
383. Rose D. Food stamps, the Thrifty Food Plan, and meal preparation: the importance of the time dimension for US nutrition policy. *J Nutr Educ Behav*. 2007;39:226–232.
384. Yen IH, Kaplan GA. Poverty area residence and changes in physical activity level: evidence from the Alameda County Study. *Am J Public Health*. 1998;88:1709–1712.
385. Institute of Medicine. *The Future of the Public's Health in the 21st Century*. Washington, DC: National Academies Press; 2003.
386. Christakis NA, Fowler JH. The spread of obesity in a large social network over 32 years. *N Engl J Med*. 2007;357:370–379.
387. Power EM. Determinants of healthy eating among low-income Canadians. *Can J Public Health*. 2005;96(suppl 3):S37–S42, S42–S48.
388. Cockerham WC, Rütten A, Abel T. Conceptualizing contemporary health lifestyles: moving beyond Weber. *Sociol Q*. 1997;38:321–341.
389. Rattray T, Brunner W, Freestone J. *The New Spectrum of Prevention: A Model for Public Health Practice*. Contra Costa Health Services. Public Health Division, 597 Center Avenue, Martinez CA 94553. 2002.
390. Cassady D, Jang V, Tanjasiri S, Morrison C. California gets "ON THE MOVE!" *J Health Education*. 1999;30:S6–S12.
391. Viswanathan M, Ammerman A, Eng E, Garlehner G, Lohr KN, Griffith D, Rhodes S, Samuel-Hodge C, Maty S, Lux L, Webb L, Sutton SF, Swinson T, Jackman A, Whitener L. Community-based participatory research: assessing the evidence. *Evid Rep Technol Assess* (*Summ*). 2004;99:1–8.
392. Esperat MC, Feng D, Owen DC, Green AE. Transformation for health: a framework for health disparities research. *Nurs Outlook*. 2005;53: 113–120.
393. Higgins DL, Metzler M. Implementing community-based participatory research centers in diverse urban settings. *J Urban Health*. 2001;78: 488–494.
394. Ma'at I, Owens M, Hughes M. Observations from the CDC. REACH 2010 coalitions: reaching for ways to prevent cardiovascular disease and diabetes. *J Womens Health* (*Larchmt*). 2002;11:829–839.
395. Sloane D, Nascimento L, Flynn G, Lewis L, Guinyard JJ, Galloway-Gilliam L, Diamant A, Yancey AK. Assessing resource environments to target prevention interventions in community chronic disease control. *J Health Care Poor Underserved*. 2006;17(suppl 2):146–158.
396. Baker IR, Dennison BA, Boyer PS, Sellers KF, Russo TJ, Sherwood NA. An asset-based community initiative to reduce television viewing in New York state. *Prev Med*. 2007;44:437–441.
397. Aronson RE, Wallis AB, O'Campo PJ, Schafer P. Neighborhood mapping and evaluation: a methodology for participatory community health initiatives. *Matern Child Health J*. 2007;11:373–383.
398. Pronk SJ, Pronk NP, Sisco A, Ingalls DS, Ochoa C. Impact of a daily 10-minute strength and flexibility program in a manufacturing plant. *Am J Health Promot*. 1995;9:175–178.
399. Yancey AK, McCarthy WJ, Taylor WC, Merlo A, Gewa C, Weber MD, Fielding JE. The Los Angeles Lift Off: a sociocultural environmental change intervention to integrate physical activity into the workplace. *Prev Med*. 2004;38:848–856.
400. Mahar MT, Murphy SK, Rowe DA, Golden J, Shields AT, Raedeke TD. Effects of a classroom-based program on physical activity and on-task behavior. *Med Sci Sports Exerc*. 2006;38:2086–2094.
401. Ikeda JP, Crawford PB, Woodward-Lopez G. BMI screening in schools: helpful or harmful. *Health Educ Res*. 2006;21:761–769.
402. American Heart Association, William J. Clinton Foundation. *Alliance for a Healthier Generation*. Available at: http://www.healthiergeneration.org/about.aspx. Accessed May 8, 2008.
403. Chicago Consortium to Lower Obesity in Chicago Chldren. Available at: http://www.clocc.org. Accessed June 13, 2008.
404. The Food Trust. Available at: http://www.thefoodtrust.org. Accessed May 8, 2008.
405. Summerbell C, Waters E, Edmunds L, Kelly S, Brown T, Campbell K. Interventions for preventing obesity in children. *Cochrane Database Syst Rev*. 2005:CD001871.
406. Bluford DA, Sherry B, Scanlon KS. Interventions to prevent or treat obesity in preschool children: a review of evaluated programs. *Obesity* (*Silver Spring*). 2007;15:1356–1372.
407. DeMattia L, Lemont L, Meurer L. Do interventions to limit sedentary behaviours change behaviour and reduce childhood obesity? A critical review of the literature. *Obes Rev*. 2007;8:69–81.
408. Sharma M. International school-based interventions for preventing obesity in children. *Obes Rev*. 2007;8:155–167.
409. Doak CM, Visscher TL, Renders CM, Seidell JC. The prevention of overweight and obesity in children and adolescents: a review of interventions and programmes. *Obes Rev*. 2006;7:111–136.
410. Flodmark CE, Marcus C, Britton M. Interventions to prevent obesity in children and adolescents: a systematic literature review. *Int J Obes* (*Lond*). 2006;30:579–589.
411. Flynn MA, McNeil DA, Maloff B, Mutasingwa D, Wu M, Ford C, Tough SC. Reducing obesity and related chronic disease risk in children and youth: a synthesis of evidence with 'best practice' recommendations. *Obes Rev*. 2006;7(suppl 1):7–66.
412. Heath GW, Brownson RC, Kruger J, Miles R, Powell KE. Ramsey LT; Task Force on Community Preventive Services. The effectiveness of urban design and land use and transport policies and practices to increase physical activity: a systematic review. *J Phys Activity Health*. 2006;3(suppl 1):S55–S76.
413. Matson-Koffman DM, Brownstein JN, Neiner JA, Greaney ML. A site-specific literature review of policy and environmental interventions that promote physical activity and nutrition for cardiovascular health: what works? *Am J Health Promot*. 2005;19:167–193.
414. Pignone MP, Ammerman A, Fernandez L, Orleans CT, Pender N, Woolf S, Lohr KN, Sutton S. Counseling to promote a healthy diet in adults: a summary of the evidence for the U.S. Preventive Services Task Force. *Am J Prev Med*. 2003;24:75–92.
415. Eden KB, Orleans CT, Mulrow CD, Pender NJ, Teutsch SM. Does counseling by clinicians improve physical activity? A summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2002;137:208–215.
416. Hardeman W, Griffin S, Johnston M, Kinmonth AL, Wareham NJ. Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *Int J Obes Relat Metab Disord*. 2000;24:131–143.
417. Glenny AM, O'Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes Relat Metab Disord*. 1997;21:715–737.
418. Swinburn B, Gill T, Kumanyika S. Obesity prevention: a proposed framework for translating evidence into action. *Obes Rev*. 2005;6:23–33.
419. Rosen L, Manor O, Engelhard D, Zucker D. In defense of the randomized controlled trial for health promotion research. *Am J Public Health*. 2006;96:1181–1186.
420. Economos CD, Hyatt RR, Goldberg JP, Must A, Naumova EN, Collins JJ, Nelson ME. A community intervention reduces BMI z-score in children: Shape Up Somerville first year results. *Obesity* (*Silver Spring*). 2007;15:1325–1336.
421. Kuczmarski R. *Second Investigators Workshop on Innovative Approaches to Prevention of Obesity*. Workshop Report. National

Institute of Diabetes and Digestive and Kidney Diseases. National Institutes of Health. 2002. Available at: http://www.niddk.nih.gov/fund/other/archived-conferences/2002/obesity_report.pdf. Accessed May 8, 2008.
422. National Heart, Lung, and Blood Institute. *Think Tank on Enhancing Obesity Research at the National Heart Lung and Blood Institute*. US DHHS. National Institutes of Health National Heart, Lung, and Blood Institute NIH Publication No. 04–5249. August 2004.
423. US Department of Health and Human Services. *Strategic Plan for NIH Obesity Research: A Report of the NIH Obesity Research Task Force*. Available at: http://obesityresearch.nih.gov/About/Obesity_EntireDocument.pdf. Accessed May 8, 2008.
424. *Centers for Disease Control and Prevention Guide to Community Preventive Services*. Obesity. Available at: http://www.thecommunityguide.org/obese/default.htm. Accessed July 15, 2007.
425. National Conference of State Legislators. *Childhood Obesity—2006 Update and Overview of Policy Options*. Available at: http://www.ncsl.org/programs/health/ChildhoodObesity-2006.htm. Accessed May 8, 2008.
426. Health Policy Tracking Service. *Balance: A Report on State Action to Promote Nutrition, Increase Physical Activity and Prevent Obesity, October 2006*. Available at: http://www.rwjf.org/pr/product.jsp?id=15950. Accessed May 8, 2008.
427. University of Baltimore. *The UB Obesity Report Card: An Overview*. Available at: http://www.ubalt.edu/experts/obesity/index.html. Accessed May 8, 2008.
428. Trust for America's Health. *F as in Fat: How Obesity Policies Are Failing in America, 2007*. Available at: http://healthyamericans.org/reports/obesity2007/. Accessed September 11, 2007.
429. *Shaping America's Youth*. Available at: http://www.shapingamericasyouth.org. Accessed May 8, 2008.
430. *Action for Healthy Kids*. Available at: http://www.actionforhealthykids.org. Accessed May 8, 2008.
431. Millstone E, Lobstein T. The PorGrow project: overall cross-national results, comparisons and implications. *Obes Rev*. 2007;8(suppl 2):29–36.
432. California Medical Association Foundation. *Physicians for Healthy Communities Initiative: Obesity Prevention Project*. Available at: http://www.calmedfoundation.org/projects/phyChampion.aspx. Accessed May 8, 2008.
433. The Osaka Declaration. *Health, Economics and Political Action: Stemming the Global Tide of Cardiovascular Disease*. Declaration of the Fourth International Heart Health Conference, Osaka, Japan, May 2001.
434. Pietinen P, Lahti-Koski M, Vartiainen E, Puska P. Nutrition and cardiovascular disease in Finland since the early 1970s: a success story. *J Nutr Health Aging*. 2001;5:150–154.
435. Kersh R, Morone J. The politics of obesity: seven steps to government action. *Health Aff* (*Millwood*). 2002;21:142–153.
436. Eriksen M. Lessons learned from public health efforts and their relevance to preventing childhood obesity. In: Koplan JP, Liverman CT, Kraak VI, eds. *Preventing Childhood Obesity: Health in the Balance*. Washington, DC: IOM, National Academies Press; 2005:343–376.
437. Economos CD, Brownson RC, DeAngelis MA, Novelli P, Foerster SB, Foreman CT, Gregson J, Kumanyika SK, Pate RR. What lessons have been learned from other attempts to guide social change? *Nutr Rev*. 2001;59:S40–S56, discussion S57–S65.

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