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Prevention Conference VII
Obesity, a Worldwide Epidemic Related to Heart Disease and Stroke
Executive Summary

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On April 27 and 28, 2002, the American Heart Association (AHA) sponsored a scientific conference, "Obesity, a Worldwide Epidemic Related to Heart Disease and Stroke," in Honolulu, Hawaii. The purpose of the conference was to develop a plan to reduce cardiovascular diseases (CVDs) associated with overweight and obesity. This report discusses the activities of the 4 working groups held before the conference, presentations at the conference, and extensive discussions among working group members after the conference. The primary objectives of this meeting were to

- Identify the worldwide demographics and comorbidities of obesity.
- Identify the age-dependent risk factors for the development of obesity.
- Identify the clinical, behavioral, and community strategies and policies for the prevention/treatment of obesity.
- Develop networks to encourage new directions in research and collaborations for the prevention of obesity worldwide.

The major findings of each working group are presented in this Executive Summary of the conference proceedings. The complete conference report with references is available online at http://www.circulationaha.org in the November 2, 2004, issue of *Circulation*.

Writing Group I: Worldwide Demographics of Obesity

The prevalence of obesity is increasing in virtually all populations and age groups worldwide. Although this increase is most evident in the United States, it is not limited to the more developed, affluent nations of the world. The escalation in obesity rates reflects the upward shift in body weights of individual populations in response to environmental changes. BMI, or weight in kilograms per square meter of height (kg/m^2), generally is accepted as a convenient measurement that provides a crude indication of body fat. The classifications of normal weight (BMI 18.5 to 24.9), overweight (BMI 25.0 to 29.9), and obesity (BMI >30.0) are somewhat arbitrary but are based on international analyses of the health impact of different BMIs.

By using approaches similar to those used for birth cohort trends in cholesterol and blood pressure levels, Howard and colleagues (see Writing Group II report) examined changes in the distribution of BMI across US birth cohorts as sampled in the National Health Examination Survey and the National Health and Nutrition Examination Surveys I, II, and III. Data were analyzed for 50 091 participants born between 1887 and 1975, who were examined beginning at ages 18 through 74 years between 1959 and 1994 (Figure 1). Steady and significant increases were demonstrated for each percentile of BMI, which suggests that this rise in BMI has been occurring over many decades and is greater at higher percentiles. Specifically, at age 50 the 10th, 25th, 50th, 75th, and 90th percentiles of BMI (adjusted for age, race, and sex) were estimated to be greater by 0.31, 0.51, 0.69, 1.00, and 1.37 kg/m^2, respectively, for successive 10-year increments in date of birth. These data suggest that in the United States the dual effects of a general upward shift in BMI distribution coupled with an accelerated shift at the highest levels of BMI may be associated with larger public health implications than previously predicted.

Nearly 30% of the US population is composed of diverse ethnic groups that are broadly categorized as black or African American, Hispanic or Latino (including primarily Mexican Americans, Puerto Ricans, Cuban Americans, and Central Americans), Asians (including persons born in any part of the Asian continent or with ancestry from the Asian continent),

Pacific Islanders, American Indians or Alaska Natives, and Native Hawaiians. This proportion will increase to nearly 40% over the next 2 decades. The prevalence of obesity varies considerably among these ethnic populations; therefore, the overall US population data provide an incomplete picture. However, the relation of BMI to hard clinical events or deaths is significantly different across various sex and ethnic/racial groups.

In Europe, as in many other parts of the world, obesity obviously is a heterogeneous condition. The definition of "European country" has changed in the aftermath of developments in eastern and central Europe and conflicts in central and southeast Europe. The creation of several new countries complicates opportunities to obtain accurate data on prevalence and incidence in all age groups, varying socioeconomic settings, and both sexes.

In Australia the prevalence of obesity has increased, particularly over the past 20 years. In 1980 the prevalence of obesity was 8.0% in women; in 2000 it had increased to 21.8%. In 1980 prevalence was 9.3% in men; in 2000 it was 19.1%. Currently in Australian adults the prevalence of obesity is 20.5%. Among men, 67.4% have a BMI >25; among women, the corresponding amount is 52%. The major increase in obesity has occurred since 1989.

In China overweight is defined by a BMI of 24.0 to 27.9 and obesity by a BMI >28. In the 1990s among Chinese adults aged 20 to 70 years, the prevalence of overweight was 22.4% and the prevalence of obesity was 3.01%. For men, the prevalence of overweight was 20.4% and the prevalence of obesity was 2.1%. For women, the prevalence of overweight was 22.4% and the prevalence of obesity was 3.9%. In 1992 urban-rural differences were observed; in the urban population mean BMI was 23.6, compared with a BMI of 21.9 in the rural population. Because China is a country in economic transition, dietary fat intake increased during the 1980s and has remained stable since 1998. The overweight/obesity trend increased rapidly in the past 17 years. In the southern part of China (Guangzhou, Shanghai) the prevalence of overweight once was low but has increased at a more rapid rate than in northern China. In 1998, even in the poor rural area of Shanxi, the prevalence of overweight reached 10%.

In Latin America epidemiological transition is well under way (ie, the main causes of morbidity and mortality are shifting from malnutrition and infection to chronic diseases). This is a diverse region, both across and within nations, ranging from poor countries, such as Haiti and Honduras, to middle-income countries such as Argentina and Chile. Brazil and Mexico are 2 examples of countries with marked disparities in health and economic conditions among social classes and geographic regions. Overweight and obesity are common in women aged 15 to 49 years. Levels of overweight and obesity range from ≈30% to 50% in Bolivia, Brazil, Colombia, the Dominican Republic, Guatemala, Honduras, Mexico, Nicaragua, and Peru. In some countries the prevalence of overweight and obesity is approaching US levels. With greater economic development, the prevalence of obesity rises and begins to affect rich and poor alike.

Developing populations in Africa are still battling poverty, food insecurity, undernutrition, and infectious diseases, con-

ditions exacerbated in sub-Saharan Africa by the HIV/AIDS epidemic. Overweight/obesity levels are rising in more developed parts of Africa, however, causing undernutrition and overnutrition to coexist in many countries. Data from northern and southern Africa suggest that obesity is characterized by a gender difference, with the rate of obesity in women 3 to 5 times that in men. When rates of overweight are compared, however, the difference is smaller, as indicated by the Mauritius data presented above. In Tunisia and Morocco, the prevalence rates of overweight in men from 1997 to 1999 were 23.3% and 28.0%, respectively, compared with 28.2% and 33.0%, respectively, in women. The same pattern was observed in Africans living in Cape Town, South Africa, where the prevalence of overweight was 22.0% in men and 36.9% in women. These data suggest that overweight and obesity emerge "earlier" in African women.

In all societies, the risk of comorbidities tends to rise with a BMI of ≈20, and clear evidence exists that specific ethnic groups are particularly vulnerable to obesity. However, considerable variation exists across ethnic groups in the proportion of fat and lean tissue at equivalent BMIs. Together with other environmental-genetic interactions, this variation may explain why the association between comorbidities and higher BMIs differs among various ethnic groups. Education and socioeconomic status affect the prevalence of obesity, but the effects may be diametrically opposite in different populations around the world. Urbanization, reduced level of physical activity, increased dietary fat, and the westernization of diets all have been implicated as significant environmental influences that contribute to the rapid increase in obesity worldwide.

Overweight/obesity in children also is increasing with a prevalence that is 2 to 3 times that of 2 decades ago. In the United States in children and adolescents aged 6 to 19 years, overweight prevalence (95th percentile of the Centers for Disease Control and Prevention standard) is 13% to 14%, or essentially 3 times the 4% to 5% prevalence rate observed in the 1960s. Another 10% of children and adolescents in the United States are at risk of being overweight (ie, with a BMI between the 85th and 95th percentiles). Preschool-age children, particularly those aged 4 to 6 years, also are affected by the trend of increased prevalence of overweight/obesity. Definition issues complicate the interpretation of weight trends in children aged <2 years.

European researchers are greatly concerned that obesity is rapidly increasing in young people. In England and Ireland, the prevalence of obesity is 14% and 31%, respectively, in those aged 15 to 24 years. More than 30% of 10-year-old boys and girls in Malta, Germany, and northern Italy had a BMI above the 85th percentile, which corresponds to an adult BMI >25. A similar increase in overweight/obesity has been observed among children in Australia. In Latin America, trends of increasing obesity are present among women of reproductive age and children aged <5 years.

Type 2 diabetes mellitus is a recent phenomenon in children, particularly in the United States, with variances in ethnicity, socioeconomic group, and urbanization similar to those seen in adults. In several parts of the world, malnutrition and obesity coexist in the same community, and child-
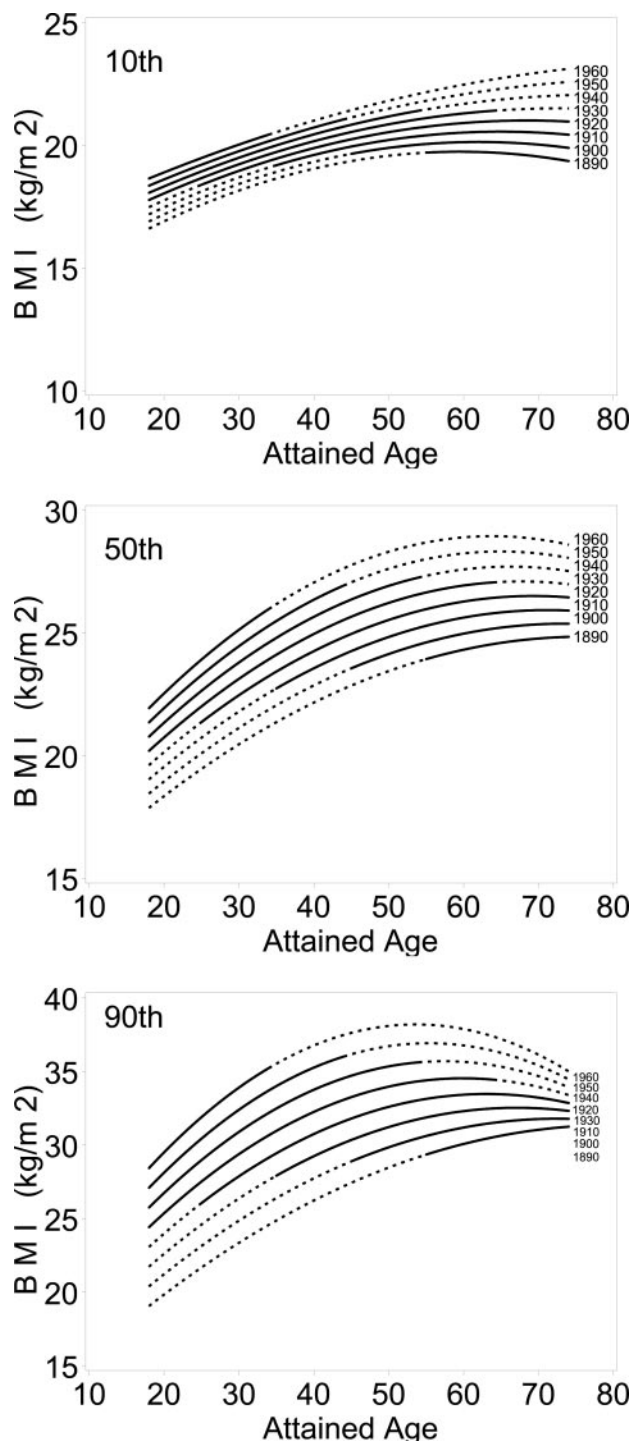
Figure 1. The 10th (A), 50th (B), and 90th (C) predicted or "smoothed" percentiles of BMI are presented as a function of age (horizontal axis) and birth cohort (individual lines, with birth year shown to the right of each line). The solid portion of each line indicates the ages for which data were available from the NHANES Surveys. The dashed portion represents extrapolated values beyond the range of observed data.

edly. Consequently, they produce heavier babies who are themselves prone to early childhood obesity and adolescent type 2 diabetes mellitus. A vicious intergenerational amplification of higher birth weight, childhood obesity, and early type 2 diabetes mellitus seems to be under way.

Future population studies should focus on the need for accurate information for all nations, more insight into the prevalence of childhood obesity, identification of groups that are especially susceptible to obesity, and better understanding of the impact of environmental factors on the increasing prevalence of obesity.

Writing Group II: Age-Dependent Risk Factors for Obesity and Comorbidities

A life-course approach to chronic disease classifies determinants in several different ways. A "critical period" refers to a specific period of development when an insult has lasting effects on the structure or function of organs, tissues, and body systems. When the effects of the insult are not long lasting, these periods may be referred to as "sensitive" rather than as "critical." During critical periods the effects of an insult may act alone or be modified by later insults. Alternatively, some insults may act at any period of development, may accumulate over time independently, or may be correlated with one another through clustering. One important insult after birth is physical inactivity. These factors are useful for helping determine which early life events are important in assessment of later disease. Although age-dependent risk factors may vary worldwide, identification of common critical or sensitive periods and plausible determinants will help improve overall treatment and prevention of overweight/obesity over the life span.

Dynamic processes that occur over the life span determine the development of obesity and associated chronic diseases. Perturbations, or "insults," that determine excessive weight gain leading to obesity may occur at any time from before conception through embryonic, fetal, infant, child, adolescent, and adult life. These insults can affect both somatic growth and maturation of metabolic systems and may include a range of determinants, including societal, lifestyle, biological, and genetic factors that often act in concert with one another.

Higher birth weight is associated with a higher body mass index (BMI) and increased prevalence of obesity in adult life, although the effect is relatively small. Small birth weight is associated with increased truncal fat or a higher waist-to-hip ratio once body mass has been taken into account. These general patterns have been found in developed and developing countries. The importance of intrauterine life in later obesity may be in determining vulnerability to increased body mass in childhood or adulthood.

Factors in early childhood may lead to obesity through one or more pathways, including metabolic programming, establishment and tracking of lifestyle behaviors, and early pathological changes. Breastfeeding may protect infants against the development of excess weight during later childhood. Most but not all epidemiological studies demonstrate this protective effect, which could be mediated by behavioral and/or physiological mechanisms. However, residual confounding

hood undernutrition and adult obesity coexist in the same family. As children become more overweight and obese, the likelihood increases that overweight girls will become overweight women. With pregnancy, the risk of developing glucose intolerance and gestational diabetes increases mark-

by cultural factors associated with both the decision to breastfeed and later obesity also is possible. Recent data also suggest that rapid weight gain during infancy is associated with obesity later in childhood, perhaps reflecting a combination of genetically determined catch-up growth and postnatal environmental factors.

Adiposity rebound is most commonly measured as the age at which BMI is at its nadir and generally occurs between ages 4 and 8 years. Several studies have shown that earlier age at adiposity rebound is associated with the development of obesity. Clinical utility is limited by several factors, however. BMI is not a direct measure of adiposity, and other measures of fatness do not show the same associations. In addition, early age at adiposity rebound is often a byproduct of excess weight gain in the first years of life, itself probably a more important etiologic factor. Moreover, BMI at age 7 or 8 years is as good a predictor of obesity as age at adiposity rebound and is easier to measure.

Adolescence is universally recognized as a time of transition from childhood to adulthood. From the standpoint of obesity, it is important both for the timing of obesity development and because it is the period during which the comorbidities related to obesity begin to appear. Obesity during adolescence is directly associated with obesity in adulthood. This phenomenon of tracking has important implications for early efforts to prevent the development of obesity and to treat it once it has developed. Furthermore, obesity in adolescence is associated with the metabolic syndrome and clustering of risk factors for the development of CVD, including elevated blood pressure, dyslipidemia, and increased prevalence of type 2 diabetes mellitus.

Although it is important to determine who is most at risk for developing obesity in adulthood, the determination of risk factors for obesity associated with comorbidities is of even greater importance. Observations of differences in the expression of obesity-related comorbidities indicate that some characteristics may be important in determining such risk. These include such phenotypic characteristics as ethnicity, distribution of body fat, fat deposition in muscles and organs, behavioral characteristics such as physical activity, and genetic factors and interactions of genetic characteristics with the environment in which they are expressed.

The relationship of overweight/obesity to CVD risk factors in older adults remains enigmatic. The number of overweight and obese older adults is increasing. In addition, aging is associated with a loss of fat-free mass (including muscle mass) and related consequences (eg, strength, glucose disposal). Despite this change in body composition, some evidence indicates that overweight or obesity may predict a better survival rate, particularly in African-American women. Some overweight or obese older persons survive to an older age, which suggests a possible protective effect of being overweight or obese from a metabolic or CVD standpoint. It is unclear whether overweight/obesity carries the same health risks in older persons as it does in younger persons. Obesity and its effect on CVD may be different in elderly persons, but this has not been systematically examined.

The direction of research should focus on understanding the effects of various critical periods of growth and development on overweight/obesity and comorbidities and how these influences may differ around the globe, when to intervene in modifying additional weight gain or treatment of comorbidities, and perhaps when not to intervene in elderly persons.

Writing Group III: Worldwide Comorbidities of Obesity

Obesity predisposes patients to a large number of comorbidities and increased mortality rates. Mortality associated with excess weight increases as the degree of obesity and overweight increases. One study estimated that from 280 000 to 325 000 deaths annually in the United States could be attributed to obesity. The increase in number of deaths from obesity is not unique to the United States; it has been documented in a number of studies from around the world.

Coronary heart disease (CHD) and stroke are foremost in the global burden of obesity and CVD. The Global Burden of Disease Study ranked these conditions first and second among all categories of death in worldwide mortality for 1990 and projected that these conditions would continue to rank first and second among causes of death up to 2020. Heart failure due to atherosclerosis and hypertension is a closely related condition clinically but was not analyzed separately. Other conditions (eg, rheumatic heart disease, Chagas disease) add to the burden of heart failure in many countries.

The World Heart Federation has compiled extensive data on the burden of CVD in major geopolitical regions of the world (more information is available at www.world-heart.org). By 1996 the proportion of deaths attributable to circulatory conditions was estimated to be 29.0% worldwide, 45.6% in developed countries, and 24.5% in developing countries, the latter increasing from 16%— or by one half—as estimated by the World Health Organization (WHO) in 1980. From 1990 to 2010 the corresponding increases in industrial market economies, economies in transition, and developing countries were projected to be 1.8%, 19.4%, and 28.2%, respectively. Thus, the global burden of these cardiovascular conditions is great and growing.

Variations among countries in the occurrence of these conditions were first investigated systematically in the Seven Countries Study, beginning in the late 1950s and 1960s. A >10-fold range in incidence of CHD was found in the Seven Countries Study among 16 cohorts of middle-aged men in Europe, Japan, and the United States. These differences were explained primarily by differences in dietary fat consumption, blood cholesterol concentration, and blood pressure among the cohorts, but not by differences in BMI. This finding possibly was the result of the narrow range and mainly "normal" mean values of BMI at baseline among the cohorts. In addition, because of the wartime dietary conditions of most of these populations at the time of baseline examinations, BMI may have measured relatively greater amounts of lean mass and less fat mass than would be the case for today's populations in the same areas.

More recently a similarly wide range of incidence and mortality rates from CHD was observed in the WHO MONICA Project, which was conducted in 31 countries from the mid-1980s through the mid-1990s. The majority of study populations were from Europe, although Australia, China,

TABLE 1.   Estimates of Regional Prevalence of Diabetes in 2000 and 2010 in Millions of Cases

Region	2000	2010	Increase, %
North America	14.2	17.5	23
Europe	26.5	32.0	24
Australia	1.0	1.3	33
South America	15.6	22.5	44
Africa	9.4	14.1	50
Asia	84.5	132.3	57
World average	151	221	46

Data from Zimmet et al, 2001.

New Zealand, and the United States were included. Ten-year trends in fatal and nonfatal CHD events were compared with trends in BMI as measured in population samples in the same study areas. The results depended on inclusion or exclusion of anomalous findings from 5 populations in the Commonwealth of Independent States that experienced decreases in BMI. Only after these populations were excluded from analysis did change in BMI contribute importantly to the observed trends in coronary event rates, explaining 31% and 10% for men and for women, respectively, of the variation.

On a population-wide basis, the contribution of BMI in the overweight and obese ranges to the pandemic of CHD and stroke is at least partly independent of the major risk factors: elevated blood cholesterol concentration, high blood pressure, physical inactivity, and cigarette smoking. However, complex interrelationships exist between obesity and all of these risk factors, some of which are in the causal pathway, and others may be effect modifiers of the obesity and health association. More important, as the prevalence of overweight has increased in pediatric age groups, so has the frequency of diagnosis of complications of obesity typically seen in the adult population. Risk factors for the development of CHD coexist in obese adolescents, and studies have demonstrated that BMI and abdominal panniculus are related to early atherosclerosis in children and young adults who died from accidents, homicides, or suicides.

Type 2 diabetes mellitus is strongly associated with obesity in all ethnic groups. Projections by the WHO show an alarming rate of increase in the prevalence of diabetes over the next decade (Table), due in large part to the increasing prevalence of obesity and physical inactivity. Important regional differences in the rate of increase in diabetes have been recognized, with the lowest rates of increase in North America, Europe, and Australia and the highest rate of increase in Asia.

The risk of type 2 diabetes mellitus increases with the degree and duration of obesity and with the central distribution of body fat. A study of 51 000 male health professionals in the United States found a strong positive association between overall obesity as measured by BMI and risk of diabetes. The relative risk for diabetes in men with a BMI of 35 kg/m^2 was 40 times higher than that for men with a BMI of 23. A similarly strong curvilinear relationship between BMI and risk of type 2 diabetes mellitus was found in women. The lowest risk was associated with a BMI <22. At

a BMI >35 the relative risk for diabetes adjusted for age increased to 61. Risk may be further increased by sedentary lifestyle or decreased by exercise. Weight gain after age 18 in women and age 20 in men also increased the risk of type 2 diabetes mellitus. Among the Pima Indians (a group with a particularly high incidence of type 2 diabetes mellitus), for example, body weight gradually increased 30 kg (from 60 to 90 kg) in the years preceding the diagnosis of diabetes. In long-term follow-up studies a strong relationship also was demonstrated between the duration of obesity and change in plasma glucose concentrations during an oral glucose tolerance test.

Insulin resistance with hyperinsulinemia is characteristic of obesity and physical inactivity and is present before the onset of hyperglycemia. With obesity, early demonstrable changes are alterations in insulin-mediated antilipolysis, impairment in glucose removal, and increased glucose production, which result in hyperinsulinemia. Hyperinsulinemia also is associated with increases in hepatic very-low-density lipoprotein triglyceride synthesis, plasminogen activator inhibitor-1 synthesis, sympathetic nervous system activity, and sodium reabsorption. These changes contribute to dyslipidemia and hypertension in obese subjects. The insulin resistance characteristic of type 2 diabetes mellitus probably results from a combination of obesity and genetic factors. The mechanism by which obesity induces insulin resistance is poorly understood. Many factors, including free fatty acids, tumor necrosis factor-α, pattern of fat distribution, and multiple genetic abnormalities, may be important and may be interactive.

Nonalcoholic fatty liver disease (NAFLD) is one of the most common abnormalities observed in obese persons and refers to a wide spectrum of liver disease. Classically, suspicion of NAFLD is raised by an elevation of serum transaminases in the absence of excessive alcohol intake, negative serology for viral hepatitis, no autoimmune disease, and lack of other known causes of liver disease. Imaging studies are consistent with steatosis. A liver biopsy shows steatosis; inflammatory cell infiltration with portal predominance, often surrounding ballooned hepatocytes; and fibrosis that will eventually progress to severe cirrhosis. Nonalcoholic steatohepatitis, a term used in earlier reports of liver disease associated with obesity, is only one stage in the spectrum of NAFLD. It is not clear why simple steatosis develops only in some persons, whereas others progress to severe cirrhosis. It has been estimated, however, that severe fibrosis occurs in up to 50% of persons with NAFLD, and cirrhosis develops in 7% to 16%. Dyslipidemia and insulin resistance both are strongly associated with the presence of NAFLD, and limited data suggest a correlation between the severity of the metabolic disorder and the severity of liver disease.

Obstructive sleep apnea is highly associated with obesity. When obstructive sleep apnea is unrecognized and untreated, substantial comorbidities (eg, pulmonary hypertension, right heart failure, cardiac arrhythmias, sudden death) can occur. It has been suggested that obstructive sleep apnea contributes to a substantial proportion of excess morbidity and mortality in obese persons.

Overall, health risks and mortality increase as the degree of overweight increases. Many of these health risks, such as
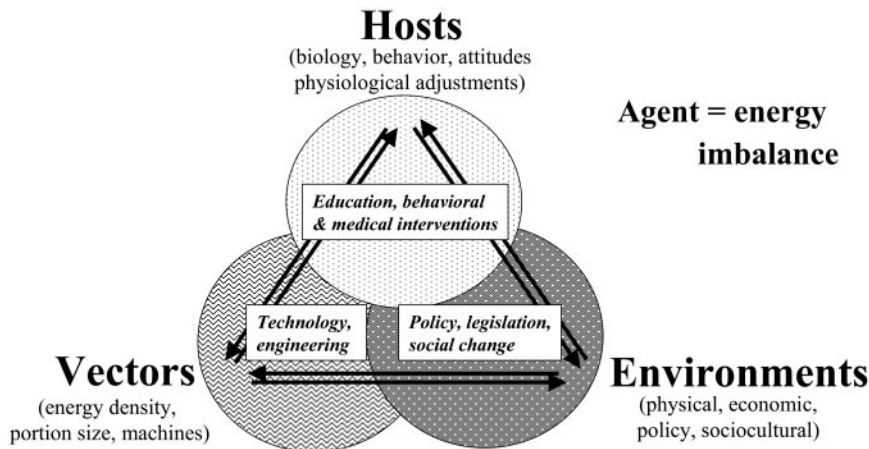
Figure 2. The epidemiological triad as it applies to obesity. The determinants are at each corner, and the circles refer to the dominant strategies available to address them. Swinburn B, Egger G. Preventive strategies against weight gain and obesity. Reproduced with permission from *Obes Rev.* 2002;3:289–301.

CVD, diabetes, liver and gallbladder diseases, and some forms of cancer, are associated with central or abdominal obesity. Certain musculoskeletal disorders, varicose veins, and skin ulcerations, however, are examples of health conditions that do not require central or abdominal obesity. Evidence also exists that physical activity patterns, age, gender, race, and ethnicity all affect the development of comorbidities in obese persons and the BMI at which they occur. For instance, in the United States the association of obesity with all-cause mortality is greater for white men than it is for African-American women. In Asian populations diabetes develops at a lower BMI. Small birth size is associated with higher mortality from CVD and higher levels of risk factors, including type 2 diabetes and hypertension. These relationships suggest that part of the risk for CVD may be "programmed" in utero. Additional insight into these differences is strongly needed.

The research agenda should include long-term studies to determine why overweight/obesity and relationship to CVD and other complications differ between populations and ethnic groups, what predisposes patients with excess abdominal adipose tissue to comorbidities, and whether weight reduction can favorably modify the incidence/prevalence of CVD and other comorbidities.

Writing Group IV: Prevention/Treatment

The prevalence of obesity is increasing in most of the world, but issues in both prevention and treatment differ among countries, on the basis of a variety of factors. In countries in which the prevalence of obesity is relatively low, true primordial prevention may be possible. For many countries, however, the response to the obesity epidemic must include both primary and secondary prevention of obesity.

High-risk and population-based approaches are complementary strategies that provide a continuum of interventions. Supportive environments will increase the availability of healthy choices in foods and activities and promote the adoption of these behaviors by overweight persons. Consumer pressure for those goods, services, and environments will increase, ensuring that these choices will continue to be available.

The epidemiological triad (Figure 2) approach is a plausible way to address the problem of obesity. This approach requires the identification of host factors (biology, behavior, physiological adjustments), vectors (energy density of food, portion size, labor-saving devices), and environments (physical, economic, policy, sociocultural) to develop and implement coherent strategies.

The components of the epidemiological triad are obviously interconnected, but the strategies for intervention differ somewhat for each. Host-related strategies tend to be educational (one-on-one and public education, behavioral interventions) or medical (pharmaceutical, surgical). Vector-related solutions often are based on technology or engineering or otherwise modifying the carrier of the agent. Environment-related solutions can be physical (changing the structures and availability of goods, services, and amenities), economic (influencing cost of goods and services and income to pay for them), policymaking (changing rules), or sociocultural (influencing attitudes, beliefs, and perceptions). Historically, physical activity and nutrition intervention strategies have been dominated by the education-based approach or individual/small behaviorally based interventions; the use of environmental options has been more limited.

Treatment of Obesity

The treatment of obesity typically occurs in a healthcare setting, although not all physicians give high priority to the identification and treatment of obesity. Several factors contribute to the relative lack of obesity treatment. Many physicians are discouraged by the lack of success, which is partly due to the lack of efficacious tools. Unfortunately, obese patients often are stigmatized in healthcare settings, and an atmosphere of "blame the victim" exists.

At a minimum, all healthcare providers should measure each patient's height and weight, determine BMI and waist girth, and refer to the National Institutes of Health guidelines for the treatment of patients who are overweight or obese. Such patients should be counseled about the importance of a healthy lifestyle, including a healthy diet and regular physical activity. Some patients may need pharmacological or surgical intervention. It is also important to advocate policy change so that Medicare and Medicaid classify obesity as a disease and provide reimbursement for its treatment. In addition to healthcare settings, obesity treatment programs should be

expanded into other settings (eg, home, school, work site, community) to reinforce treatment efforts.

Prevention of Obesity

The prevention of overweight and obesity will require extensive changes in many aspects of society. Much can be done to restructure physical, social, and overall community environments to promote healthier lifestyles and reduce the risk associated with a positive energy balance.

- *Family/home setting*. Families need to be encouraged to become aware of more healthy food choices and implement better menu planning. The return of the family meal will be useful in many instances. Families should look for opportunities for physical recreational activities and focus on integrating more physical activity into their daily routines.
- *School setting*. Schools should make more intensive efforts to provide meals that offer a variety of foods with high nutritional value and controlled-energy density. School lunch programs also should provide opportunities for enhancing nutrition education. School health and physical education programs should be structured to promote healthy eating and regular physical activity.
- *Work site setting*. Work sites can provide a variety of programs that promote healthy eating and regular physical activity. These programs can range from formal classes and interventions to efforts to improve the physical environment to increase the frequency of routine physical activity.
- *Community setting*. Communities can become more proactive in promoting healthy lifestyles (eg, greater access to parks, trails, and other recreational opportunities). Communities also could expand programs to communicate healthy eating patterns; some examples of successful programs of this type are available (see Writing Group IV report).

Policies to Facilitate/Enable Recommendations

Although little research exists on the effectiveness of broad-based policies in the prevention of obesity, it seems clear that such programs should be developed and extensively evaluated. Policies to be considered should include physical and social environmental changes, financial incentives and tax policies, factors related to delivery of health care, and school and work site policies.

Several creative approaches to broad policy recommendations must be evaluated:

- Host factors, vectors, and environments must be addressed (currently, environmental approaches are the least well promulgated and evaluated).
- Healthcare professionals, the government, and the public must treat the obesity epidemic seriously (too often obesity is regarded as a cosmetic issue rather than as a health issue).
- Serious investments are needed in interventions, programs, monitoring, and evaluation of the obesity epidemic.
- Because knowledge alone is not enough to influence behavior, messages and programs to counteract the obesity epidemic must be strong and unambiguous, with a focus on mass behavioral change.
- Large numbers of people must be encouraged to expend higher levels of energy to blunt the obesity epidemic. In many countries, physical activity has been largely engineered out of life, with many people leading sedentary lives. Environmental changes are central to a broad approach to address this epidemic.

It is important for medical and public health groups to engage industry in efforts to develop and evaluate policies to prevent obesity. Mass media, food companies, and sporting goods companies are obvious industry partners. In addition to promoting behaviors to maintain energy balance, industry, especially the mass media, can help by promoting realistic expectations about body image. Messages and images should emphasize that diversity in body sizes and shapes is normal. The focus should be health, not body size. Policies promoting healthy dietary patterns must take into account consumer factors such as taste, variety, convenience, and enjoyment. Diets that are lower in fat and energy can be more costly than high-fat, high-energy diets; therefore, people in lower socioeconomic groups are often at a dietary disadvantage. All consumers want to minimize cost and maximize taste and convenience. Advertising for healthy foods should focus on these factors. Sporting goods companies and those providing physical activity and fitness products and services should help to develop policies that promote moderate-intensity physical activity by all persons rather than the current emphasis on competitive athletics.

Overall, primary prevention and successful treatment of obesity will require the extensive involvement of many sectors of society. Many of the suggestions above are unproved, and benefits need to be documented. Research studies need to be longitudinal and lengthy; the cost will be expensive. In the interim and without data, however, a concerted and sustained effort is needed to focus on broad environmental changes and community support for healthy behaviors in schools, work sites, churches, and other venues; the commitment of families to healthy behaviors is critical. The need is great and the time to address the problem of prevention and treatment of obesity is *now,* even before the intervention is evidence based.

Disclosures

Writing Group Member Name	Research Grant	Speakers Bureau/Honoraria	Stock Ownership	Consultant/Science Advisory Board	Other
Dr Louis J. Aronne	Regeneron; Sanofi	Ortho McNeil	None	Roche	None
Dr Dennis M. Bier	Food and Infant Formula Industries			Gerber Inc.; Mars Inc.; ILSI International; Glutamate Technical Committee; Eli Lilly; Genentech; Archer Daniels Midland; Ajiomoto	
Dr Steven N. Blair	Knoll Pharmaceuticals; Human Kinetics	M&M Mars Inc.; The Sugar Association Inc.		Life Fitness International; Jenny Craig; Bally Total Fitness Sports; Sherbrooke Capital; Miavita; International Life Sciences Institute Center for Health Promotion; Healthetech; Westport Realty	None
Dr George A. Bray	None	None	None	None	None
Dr Ian Caterson	Servier Laboratories; 3M; Roche Products; Sanofi; Metabolic Pharmaceuticals	Servier Laboratories; 3M Pharmaceuticals; Roche Products; Pfizer; Abbott Laboratories	None	Pfizer; Abbot Laboratories	None
Dr C.M. Chen	None	None	None	International Life Science Institute Center for Health Promotion	None
Dr Stephen R. Daniels	None	None	None	None	None
Dr Margo A. Denke	None	Merck; Roche; Merck-Schering Plough	None	None	None
Dr William Dietz	None	None	None	None	None
Karen Donato	None	None	None	None	None
Dr Adam Drewnoski	None	None	None	None	None
Dr Robert H. Eckel	Merck; Slimfast; Abbott	Merck; Pfizer; Abbott; Kos	None	Merck; Pfizer; GSK	None
Dr Simone A. French	None	None	None	None	None
Dr Matthew W. Gillman	None	None	None	None	None
Dr Ron Grunstein	None	None	None	None	None
Dr Barbara C. Hansen	None	None	None	None	None
Dr Laura L. Hayman	None	None	None	None	None
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Dr George Howard	None	None	None	None	None
Dr Van S. Hubbard	None	None	None	None	None
Dr W.P.T. James	None	None	None	None	None
Dr Shiriki Kumanyika	None	None	None	Weight Watchers International	None
Dr Darwin Labarthe	None	None	None	None	None
Dr Catherine M. Law	None	None	None	None	None
Dr Cora Elizabeth Lewis	None	None	None	Roche	None
Dr Reynaldo Martorell	None	None	None	None	None
Dr Rebecca M. Mullis	None	None	None	None	Nutrition Expert, The Egg Nutrition Center
Dr Eric Poehlman	None	None	None	None	None
Dr Thomas N. Robinson	None	Nestle	None	None	None
Dr Stephan Rössner	None	None	None	None	None
Dr Jacob C. Seidell	None	None	None	None	None
Dr Sidney C. Smith	None	None	None	None	None
Dr Sachiko T. St. Jeor	None	None	None	Healthetech; National Cattlemens Beef Association	None
Dr Boyd Swinburn	None	None	None	None	None
Dr H.H. Vorster	SA Dry Bean Producers Organisation; Sugar Association	None	None	None	None
Dr Howell Wechsler	None	None	None	None	None
Dr David York	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Faculty Disclosure Questionnaire, which all members of the writing group were required to complete and submit shortly before the conference.