

## Discovering the Full Spectrum of Cardiovascular Disease Minority Health Summit 2003

### Report of the Obesity, Metabolic Syndrome, and Hypertension Writing Group

Sidney C. Smith, Jr, MD, Chair; Luther T. Clark, MD; Richard S. Cooper, MD;  
Stephen R. Daniels, MD, PhD; Shiriki K. Kumanyika, PhD, MPH; Elizabeth Ofili, MD;  
Miguel A. Quinones, MD; Eduardo J. Sanchez, MD, MPH;  
Elijah Saunders, MD; Susan D. Tiukinhoy, MD

This article provides an overview of our current understanding of the epidemiology of obesity, the metabolic syndrome, and hypertension among racial/ethnic groups. Three presentations made at the conference by the present writing group are summarized and updated with other information on ethnic groups, and recommendations developed by the writing group for programs, public policy, and research are put forward.

#### Epidemiology of Obesity

- Obesity has been increasing across all US groups since 1980: children, adults, racial/ethnic groups, and socioeconomic status groups.
- Ethnic disparities are prevalent: Obesity prevalence is higher among black American women, Hispanic Americans (especially Mexican Americans and Puerto Ricans), American Indians/Alaska Natives, Pacific Islanders, and Native Hawaiians than among white Americans.
- Cardiovascular disease (CVD) risk for a given body mass index (BMI) may vary by race/ethnicity.

In the United States, obesity in adults is defined as a BMI  $\geq 30$  kg/m<sup>2</sup>, and individuals with a BMI  $\geq 25$  kg/m<sup>2</sup> are considered overweight. Abdominal obesity, defined as a waist circumference  $>88$  cm for women and  $>102$  cm for men, compounds the CVD risk associated with a given BMI level.<sup>1</sup> The prevalence of obesity (BMI  $\geq 30$  kg/m<sup>2</sup>) appears to be increasing in all US population segments, including both genders, children and adults of all ages, and diverse

racial/ethnic groups, across the spectrum of educational attainment and regardless of smoking status.<sup>2-4</sup> These statistics are actually a reflection of the global epidemic of obesity.<sup>5,6</sup>

The prevalence of BMI  $\geq 30$  kg/m<sup>2</sup> seems to be disproportionately higher in several racial/ethnic minority populations, specifically black American women, Mexican Americans, Puerto Ricans, several American Indian and Alaska Native populations, Native Hawaiians, and Pacific Islanders.<sup>1-3,7-11</sup> These longstanding disparities are aggravated by the current upward trend of increased obesity in the US population as a whole (Table), as clearly illustrated in the NHANES trend data for obesity among black and Mexican American women (Figure).<sup>2</sup> Obesity in children and adolescents is usually termed "overweight" and is defined according to age-specific BMI standards that take developmental changes in body size into account. Trends of increased overweight prevalence in children reflect an accelerated gradient in black and Mexican American children as compared with white children.<sup>3</sup>

Asian Americans and Pacific Islanders often are grouped in the same category; however, not only does the term "Asian American" refer to diverse ethnic subgroups but also Asian Americans in general have different BMI levels than do Pacific Islanders.<sup>14</sup> Asian Americans and Pacific Islanders have, respectively, lower than average and higher than average obesity prevalence. Hence, aggregate data on obesity prevalence for Asian Americans and Pacific Islanders can be misleading. Furthermore, the degree and pattern of body fatness at a given BMI level are different in Asians and Pacific Islanders as compared with whites (eg, more body fat

---

This paper represents a summary of a scientific conference sponsored by the American Heart Association. The opinions expressed in this paper are those of the authors and do not necessarily represent those of the editor or the American Heart Association. The publication of these proceedings was approved by the American Heart Association Science Advisory and Coordinating Committee on January 20, 2005. All writing group members were required to complete and submit shortly before the workshop a Faculty Disclosure Questionnaire. These disclosures are available as an appendix to the Executive Summary.

A single reprint is available by calling 800-242-8721 (US only) or by writing the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596. Ask for reprint no. 71-0316. To purchase additional reprints: up to 999 copies, call 800-611-6083 (US only) or fax 413-665-2671; 1000 or more copies, call 410-528-4121, fax 410-528-4264, or e-mail kgray@lww.com. To make photocopies for personal or educational use, call the Copyright Clearance Center, 978-750-8400.

The Executive Summary and reports of the Basic Science, the Outcomes, and the Advocacy Writing Groups are available online at <http://www.circulationaha.org> (*Circulation*. 2005;111:1339-1349; e120-e123; e124-e133; and e140-e148). (*Circulation*. 2005;111:e134-e139).

© 2005 American Heart Association, Inc.

*Circulation* is available at <http://www.circulationaha.org>

DOI: 10.1161/01.CIR.0000157743.54710.04

**Upward Trend of Increased Obesity in the US Population**

		Obesity (BMI ≥30)							
		NHANES III, 1988–1994		NHANES 1999–2000		Extreme Obesity (BMI ≥40)			
Sex	Racial/Ethnic Group	No.	% (SE)	No.	% (SE)	Change, % (95% CI)	NHANES III, 1988–1994, % (SE)	NHANES 1999–2000, % (SE)	Change, % (95% CI)
Both sexes	All*	16681	22.9 (0.68)	4115	30.5 (1.43)	7.6 (4.4–10.8)	2.9 (0.23)	4.7 (0.56)	1.8 (0.6–3.0)
Men	All*	7933	20.2 (0.72)	2043	27.5 (1.61)	7.3 (3.8–10.8)	1.7 (0.32)	3.1 (0.58)	1.4 (0.1–2.7)
	Non-Hispanic white	3285	20.3 (0.85)	946	27.3 (1.82)	7.0 (3.0–11.0)	1.8 (0.41)	3.0 (0.75)	1.2 (–0.5–2.9)
	Non-Hispanic black	2112	21.1 (1.02)	374	28.1 (2.27)	7.0 (2.0–12.0)	2.4 (0.38)	3.5 (1.24)	1.1 (–1.5–3.7)
Women	Mexican American	2250	23.9 (0.97)	538	28.9 (2.25)	5.0 (0.1–9.9)	1.1 (0.33)†	2.4 (0.74)	1.3 (–0.3–2.9)
	All*	8748	25.4 (0.95)	2072	33.4 (1.81)	8.0 (3.9–12.1)	4.0 (0.31)	6.3 (0.89)	2.3 (0.6–4.0)
	Non-Hispanic white	3755	22.9 (1.15)	885	30.1 (2.10)	7.2 (2.4–12.0)	3.4 (0.40)	4.9 (0.89)	1.5 (–0.5–3.5)
	Non-Hispanic black	2490	38.2 (1.37)	420	49.7 (2.79)	11.5 (5.3–17.7)	7.9 (0.51)	15.1 (2.05)	7.2 (3.0–11.4)
	Mexican American	2128	35.3 (1.36)	567	39.7 (3.65)	4.4 (–3.4–12.2)	4.8 (0.65)	5.5 (1.04)	0.7 (–1.8–3.2)

\*Includes racial/ethnic groups not shown separately.

† Does not meet the standard of statistical reliability and precision (relative SE >30%).

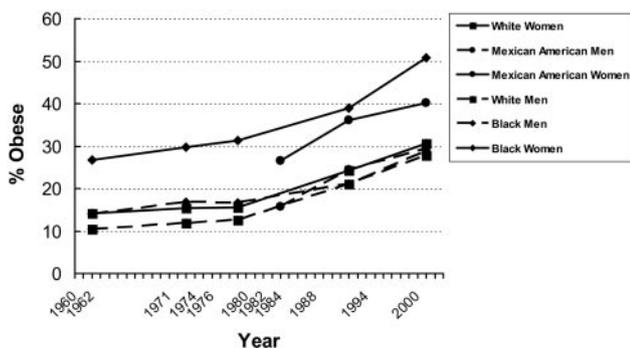
Reprinted with permission from *JAMA*.<sup>2</sup> Copyright 2002, American Medical Association. All Rights Reserved.

at a given BMI for Asian populations when compared with white American or European populations, and comparatively less body fat and more muscle for Pacific Islander populations).<sup>7,14</sup> Asian Americans, at least in some subgroups, seem to gain abdominal fat preferentially,<sup>12–14</sup> and a substantial number of risk factors associated with the metabolic syndrome (eg, insulin resistance, hypertension, and diabetes) have been observed in Asian Americans, including, and perhaps particularly, in South Asians, with average BMI levels <25 kg/m<sup>2</sup>. This observation has led to a call for recommendations for lower BMI cutoffs for assessment and intervention for obesity in Asian American populations (eg, to 23 kg/m<sup>2</sup> for overweight and 25 kg/m<sup>2</sup> for obesity<sup>7</sup>) and, subsequently, to the suggestion that international comparisons of BMI be based on finer categories than the usual 5-BMI-unit thresholds used to define various classes of obesity.<sup>14</sup> These observations underscore that BMI, although easy to measure, is not the only criterion of interest when assessing the population burden of obesity in relation to

health, and that the risk of obesity needs to be considered in the context of CVD and the overall health profiles of the group in question; these profiles vary by race/ethnicity.

Substantial cultural heterogeneity also exists within each aggregate minority population. Within all populations, obesity prevalence varies by region, socioeconomic status, and other demographic variables. Among immigrants from societies with less obesity, weight levels generally increase with increasing duration of residence in the United States (eg, as observed in Asian Americans)<sup>15</sup>; however, variations in obesity prevalence do not always follow the same patterns in minority populations that they do in whites. For example, the inverse gradient of obesity prevalence that is observed among white women is not always observed in black women, or it is not as striking. Moreover, in populations with relatively low median income levels, obesity is at least initially less common among women with the lowest incomes and highest among those who are economically better off.<sup>16</sup> Attitudes about obesity also vary across cultures. Where thinness is associated with extreme poverty, deprivation, or wasting diseases, obesity may be viewed as a symbol of social stature, prosperity, and robustness.<sup>17</sup>

The role of obesity in predisposing individuals to diabetes, hypertension, and other aspects of CVD risk is well established,<sup>1</sup> and obesity contributes to racial/ethnic disparities in CVD risk. A gradient of increasing risk of diabetes, hypertension, and coronary heart disease with increasing BMI levels can be readily demonstrated in several available data sets for blacks, and some related data also are available for other racial/ethnic groups.<sup>18–20</sup> In particular, obesity has been identified as a contributor to the excess prevalence of diabetes in minority populations. The Diabetes Prevention Program, which deliberately enrolled ≈50% of participants from ethnic minority populations (blacks, Hispanic Americans, and American Indians) at high risk for diabetes, provides compelling evidence that lifestyle modification and weight reduction results in a substantial diabetes risk reduction in these



Age-adjusted prevalence of obesity (BMI ≥30 kg/m<sup>2</sup>) in successive NHANES by race/ethnicity and gender, US adults 20–74 years old. Data adapted from Pastor PN, Makuc DM, Reuben C, Xia H. *Chartbook on Trends in the Health of Americans: Health, United States, 2002*. Hyattsville, Md: National Center for Health Statistics; 2002. Available at: <http://www.cdc.gov/nchs/data/hus/02.pdf>.

populations as well as in whites.<sup>21</sup> Benefits of weight loss for reducing hypertension-related risks have been reported for black patients in several clinical trials.<sup>22</sup>

Traditionally, the association of obesity with mortality has been considered the “gold standard” for evaluating obesity-related health risks; however, for minority populations the strongest rationale for aggressive attention to the problem of obesity comes from morbidity data. Mortality rates for cardiovascular disease by ethnicity are not necessarily parallel to the prevalence of obesity at the population level. The within-population association of obesity with mortality in blacks has been inconsistent across data sets and also in comparison with data for white populations.<sup>23</sup> Mortality is influenced by many variables other than obesity such as other lifestyle-related variables, access to care, quality of care, and social context variables that determine overall survival. Thus, although obesity undoubtedly contributes to the CVD burden in blacks and other racial/ethnic minority populations, the effects of these other powerful determinants of mortality may make it difficult to attribute excess mortality to obesity as such.<sup>23</sup>

Developing strategies to ameliorate the problem of obesity in racial/ethnic minority populations requires attention to factors that drive the obesity epidemic as a whole in the general US population,<sup>24–26</sup> with special attention to factors that might predispose members of racial/ethnic minority populations to excess risk. The fact that the obesity excess observed among black women, Hispanic Americans, American Indians, Pacific Islanders, and Native Hawaiians predates the current epidemic suggests that many of these factors preexisted in these communities, at least among adults. Weight gain and retention in association with pregnancy have been identified as risk factors for obesity in black women.<sup>27</sup> Other potential causes can be found in macrosocietal changes that accompany economic and nutrition transitions, characteristics of local environments that constrain individual choices with respect to eating and physical activity, and sociocultural influences on lifestyle behaviors related to weight gain and weight control.<sup>28</sup> Some of these factors may exert their effects early in life, such as during gestation or in infancy,<sup>29,30</sup> through maternal weight status, or in parental feeding behaviors. Genes influence the individual predisposition to obesity, but the striking importance of environmental influences on population risk can be inferred from examples such as the marked gradient of obesity in the African Diaspora as people were removed from Africa through the Caribbean islands to the United States<sup>31</sup> or in the comparison of Pima Indians living in Arizona, who have high rates of obesity, with Pima Indians living a traditional lifestyle in Mexico, who do not.<sup>32</sup>

The epidemic pattern of obesity in children provides a strong mandate to make preventive strategies a priority, not only because obesity has harmful effects on health during childhood but also because obesity tends to continue into adulthood and, once established, is difficult to reverse.<sup>24</sup> The serious limitations of current approaches to obesity treatment and particularly the apparent less-than-average success of treatment approaches in racial/ethnic minority populations must be considered as additional arguments for preventing

the development of obesity among high-risk groups in particular.<sup>33</sup> Prevention requires both structural approaches (community-level changes and policies at local, state, and national levels) and individually oriented approaches.<sup>28</sup> These approaches must be carefully formulated in consideration of the ethnic, cultural, and social embedment of many of the factors that determine obesity and the possibility that efforts to address obesity in the mainstream will have inadvertent adverse effects on the least influential communities.<sup>34</sup>

### Pathophysiology of Hypertension in Racial/Ethnic Minorities

- Hypertension is common and poorly controlled among racial/ethnic minorities.
- Detection, awareness, and control are poor in the groups that have been assessed (eg, Hispanics, blacks, Asians, and Pacific Islanders).
- Detection and control of hypertension are notably low among Mexican Americans.
- Target-organ damage is a major cause of morbidity and mortality among blacks.

It is generally accepted that minorities (specifically blacks) are at higher risk for hypertension-related cardiovascular morbidity and mortality than are their white counterparts. The reasons for this observation are not clearly understood, but they are believed to be related to a greater number of concomitant risk factors (eg, diabetes, obesity, low socioeconomic status), as well as possible increased hereditary predisposition. Factors such as delay in diagnosis, dietary habits, and disparities in health care have been recognized as contributing significantly to these adverse outcomes.

Regardless of our understanding of the reasons for these phenomena, the following pathophysiological profiles have been observed or are speculated to occur in association with hypertension among US blacks:

- Earlier appearance of hypertension with higher prevalence in younger age groups than in whites
- More stage 2 hypertension than in comparable white groups
- More associated target-organ damage in the heart (left ventricular hypertrophy with diastolic dysfunction, often out of proportion to the level of blood pressure)
- More chronic kidney disease and end-stage renal disease, leading to an excessive need for renal replacement therapy
- High rates of thrombotic and hemorrhagic strokes resulting from intracerebral vascular disease
- More sudden cardiac death and out-of-hospital mortality resulting from ischemic heart disease with less demonstrable epicardial coronary artery disease
- Suspected differences in electrolyte and other transport and countertransport systems at the cellular membrane level that may exert an influence on blood pressure
- Higher urinary sodium/potassium ratios, contributing to more salt-sensitive, renin-independent hypertension
- Possible lower levels of vasodilator hormones (eg, bradykinin), resulting in higher vascular resistance

The rate of CVD has been increasing in American Indians overall.<sup>20</sup> Hypertension prevalence is higher than average in some groups of American Indians but varies substantially across populations<sup>35</sup>; however, awareness and control of it may be improving.<sup>36</sup> Obtaining a representative picture of disease prevalence rates among Asian Americans and Pacific Islanders often is difficult because of the aforementioned differences in the BMI distributions of Asians and Pacific Islanders and also because of the diversity of the populations that are included within these broad categories. For several broad minority population categories, poor health status in some groups can be hidden in aggregate data. Hypertension is highly prevalent among Filipino Americans, Japanese Americans, and Southeast Asian populations, with prevalence rates similar to those of black Americans.<sup>37,38</sup> Awareness of hypertension is low and control of blood pressure is poor in these communities, partly related to the multilingual necessity of any screening programs and educational materials used. Lack of health insurance also disproportionately affects these fast-growing racial/ethnic groups.<sup>39</sup> Improvement in the quality and accessibility of linguistically and culturally appropriate community-based health care for these populations is necessary.

### The Metabolic Syndrome in Racial/Ethnic Minorities

- Prevalence is highest among Hispanic men and women, black women, and older individuals.
- Susceptibility to risk factors for the metabolic syndrome varies (eg, dyslipidemia in whites of European descent; hypertension in blacks and Asians; diabetes in Hispanics, Pacific Islanders, and Native Americans).

The metabolic syndrome—also known as the insulin-resistance syndrome, metabolic syndrome X, and dysmetabolic syndrome—refers to a specific clustering of cardiovascular risk factors in the same individual: abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance, a prothrombotic state, and a proinflammatory state. Patients with the metabolic syndrome are at increased risk for developing diabetes and CVD. Although the metabolic syndrome has been variably defined in the past, the National Cholesterol Education Program Adult Treatment Panel III guidelines provide a working definition of the metabolic syndrome.<sup>40</sup> According to the Adult Treatment Panel III definition, the metabolic syndrome is present in individuals with any 3 of the following 5 abnormalities: (1) waist circumference >102 cm (40 in) in men or >88 cm (35 in) in women, (2) serum triglyceride levels >150 mg/dL, (3) HDL-cholesterol level <40 mg/dL in men or <50 mg/dL in women, (4) blood pressure >130/85 mm Hg, and (5) fasting serum glucose >110 mg/dL.

According to a recent analysis of data from the Third National Health and Nutrition Examination Survey (NHANES III),<sup>41</sup> ≈47 million Americans (23.7% of the population) have the metabolic syndrome. The highest rates were observed in Mexican American women and men. Black women had an ≈57% higher prevalence for the syndrome

than did black men, who had the lowest prevalence in this study. The prevalence of the metabolic syndrome also increases with age, with a prevalence of >30% in adults >40 years old, and >40% for adults >60 years old. The Strong Heart Study investigators have estimated the prevalence of the metabolic syndrome in American Indians to be more than twice as high as in the NHANES population: 43.6% in men 45 to 49 years old as compared with 20.0% among all men in NHANES III; in the same age group, the prevalence of metabolic syndrome was 56.7% as compared with 23.1% among NHANES III women.<sup>42</sup> As noted previously, people of Asian descent may have a higher than average predisposition to develop the metabolic syndrome and at relatively low BMI levels.<sup>14</sup>

Although no comparisons have been made of susceptibility and patterns of the metabolic syndrome in different populations, racial and ethnic variability in susceptibility to the specific risk factors of the metabolic syndrome exists. US whites of European origin appear to be more predisposed to atherogenic dyslipidemia than are other groups. US blacks of African origin are more prone to hypertension, type 2 diabetes mellitus, and obesity. Hispanics and Native Americans appear to be especially susceptible to type 2 diabetes mellitus but develop hypertension less often than do blacks. Although a high degree of association between individual components of the metabolic syndrome and CVD risk exists, several recent analyses have confirmed that the cluster of risk factors in the metabolic syndrome is associated with an increased risk of cardiovascular morbidity and mortality. Because of the strong relationship of obesity to the metabolic syndrome, the rising prevalence of obesity in the United States is cause for particular concern. The metabolic syndrome is closely associated with insulin resistance, although the mechanisms of the association between insulin resistance and metabolic risk factors have not been fully elucidated.

Because the root causes of the metabolic syndrome (overweight/obesity and physical inactivity) are reversible and the individual components of the metabolic syndrome are modifiable, recognition of the metabolic syndrome provides a great opportunity for risk reduction. Management of the metabolic syndrome consists primarily of 2 strategies: modification or reversal of the root causes, including weight reduction and increased physical activity, and direct treatment of the metabolic risk factors, including atherogenic dyslipidemia, elevated blood pressure, the prothrombotic state, and underlying insulin resistance. All of the components of the metabolic syndrome may be improved with weight reduction and increased physical activity. Treatment of several of the individual risk factors associated with the metabolic syndrome has been shown to decrease CVD risk, although no randomized clinical trials are yet available to show a decrease in clinical events or increased survival following treatment of the metabolic syndrome per se. In an analysis of the benefits of treating elevated blood pressure and dyslipidemia in individuals with the metabolic syndrome, however, Wong et al<sup>43</sup> found that aggressive treatment of risk factors and control to optimal levels could, at least theoretically, result in the prevention of >80% of cardiovascular events.

Recognition, diagnosis, and treatment of the metabolic syndrome have the potential to contribute importantly to the reduction of health disparities. Although data on other racial/ethnic minorities are limited, the high morbidity and mortality rates from CVD in US blacks can be explained in part by the high prevalence and severity of modifiable risk factors. Thus, the opportunities for risk reduction and prevention of CVD by treating the metabolic syndrome are great. Patients who are at high risk for the metabolic syndrome, such as those with multiple risk factors, should be targeted for intensive risk-reduction measures. The clinical approaches to CVD identification, evaluation, and treatment strategies may need to be modified to take these findings into consideration. Reduction in racial/ethnic disparities in CVD mortality requires conscious targeting of these populations for vigorous prevention and risk-reduction measures.

## Recommendations

### Professional/Lay Programs

- Update the AHA scientific statements related to obesity, the metabolic syndrome, and hypertension so that they contain the appropriate multiethnic emphasis
- Update and widely disseminate information on physical activity and nutrition to healthcare providers and the public, especially racial/ethnic minorities. Coordinate with other groups (eg, community-based organizations, the American Diabetes Association, the Centers for Disease Control and Prevention, the US Department of Agriculture, the US Public Health Service, the American Medical Association, the American Public Health Association, and the National Heart, Lung, and Blood Institute) to disseminate information.
- Partner with organizations in racial/ethnic minority communities on obesity, the metabolic syndrome, and hypertension prevention and control initiatives.
- Launch a special campaign to prevent and control childhood obesity.
- Launch educational programs targeted toward healthcare providers in selected high-risk communities to learn to recognize and treat obesity, the metabolic syndrome, and hypertension in their patients.
- Create racially/ethnically focused materials to support healthy eating and active living in partnership with community organizations in minority population areas.

### Public Policy/Advocacy

- Advocate research funding to train obesity researchers recruited from racial/ethnic minority populations.
- Lobby the food and entertainment industries for standards of conduct that limit the aggressive targeting of advertising and marketing of high-calorie, low-nutrient-density products to young children or people of color.
- Lobby for research funding from the Centers for Disease Control and Prevention to study ways to stimulate grass-roots advocacy of policy and environmental changes that will reduce the “obesogenicity” of daily living environments.

- Advocate the expansion or development of programs that encourage racial/ethnic minority high school and college students to consider careers in the sciences and health-related professions and deliver services to racial/ethnic minorities.
- Advocate the treatment of obesity as a reimbursable service in health insurance policies.

### Research

- Support studies that define the genetic contributions to CVD, especially those associated with obesity, the metabolic syndrome, and hypertension and their potential relationship to racial/ethnic disparities in CVD.
- Study the environmental contributors to CVD, especially those associated with obesity, the metabolic syndrome, and hypertension.
- Study how racial/ethnic designation contributes positively or negatively to the treatment of obesity, the metabolic syndrome, and hypertension.
- Fund research to better understand the patterns of obesity and its relationship to CVD. Such research should address questions of
  - Gender-specific differences in obesity across racial/ethnic groups
  - Regional, socioeconomic, and cohort variables that determine obesity in racial/ethnic minorities
  - Varied effectiveness of conventional weight loss programs in different racial/ethnic groups
  - The relationship of level of obesity to CVD outcomes among different racial/ethnic groups
- Support studies on the patterns of CVD and the metabolic syndrome in different racial/ethnic groups
- Endorse the concept that clinical trials of new therapies should include an appropriate number of minorities, not only as research subjects but at all levels of the research team
- Study effective approaches to educate physicians about recognizing and treating obesity, the metabolic syndrome, and hypertension

### References

1. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults—The Evidence Report. National Institutes of Health. *Obes Res.* 1998;6:51S–209S.
2. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA.* 2002;288:1723–1727.
3. Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999–2000. *JAMA.* 2002;288:1728–1732.
4. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion. 1991–2001 Prevalence of Obesity Among U.S. Adults, by Characteristics. Available at: [http://www.cdc.gov/nccdphp/dnpa/obesity/trend/prev\\_char.htm](http://www.cdc.gov/nccdphp/dnpa/obesity/trend/prev_char.htm). Accessed February 4, 2005.
5. *Obesity: Preventing and Managing the Global Epidemic.* WHO Technical Report Series No. 894. Geneva, Switzerland: World Health Organization; 2000.
6. James PT, Leach R, Kalamara E, Shayeghi M. The worldwide obesity epidemic. *Obes Res.* 2001;9:228S–233S.
7. WHO Regional Office for the Western Pacific/International Association for the Study of Obesity/International Obesity Task Force. *The Asia-Pacific Perspective: Redefining Obesity and Its Treatment.* Sydney, Australia: Health Communications Australia; 2000.

8. *African Americans and Cardiovascular Diseases*. Statistics Fact Sheet. Dallas, Tex: American Heart Association; 2004.
9. *American Indians/Alaska Natives and Cardiovascular Diseases*. Statistics Fact Sheet. Dallas, Tex: American Heart Association; 2003.
10. *Asian/Pacific Islanders and Cardiovascular Diseases*. Statistics Fact Sheet. Dallas, Tex: American Heart Association; 2004.
11. *Hispanics and Cardiovascular Diseases*. Statistics Fact Sheet. Dallas, Tex: American Heart Association; 2004.
12. Ko GT, Chan JC, Cockram CS, Woo J. Prediction of hypertension, diabetes, dyslipidaemia or albuminuria using simple anthropometric indexes in Hong Kong Chinese. *Int J Obes Relat Metab Disord*. 1999; 23:1136–1142.
13. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis among different ethnic groups. *Int J Obes Relat Metab Disord*. 1998;22:1164–1171.
14. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363:157–163.
15. Lauderdale DS, Rathouz PJ. Body mass index in a US national sample of Asian Americans: effects of nativity, years since immigration and socio-economic status. *Int J Obes Relat Metab Disord*. 2000;24:1188–1194.
16. Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull*. 1989;105:260–275.
17. Brown PJ, Konner M. An anthropological perspective on obesity. *Ann N Y Acad Sci*. 1987;499:29–46. Review.
18. Lipton R, Keenan H, Onyemere KU, Freels S. Incidence and onset features of diabetes in African-American and Latino children in Chicago, 1985–1994. *Diabetes Metab Res Rev*. 2002;18:135–142.
19. Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA*. 1999;282: 1523–1529.
20. Howard BV, Lee ET, Cowan LD, Devereux RB, Galloway JM, Go OT, Howard WJ, Rhoades ER, Robbins DC, Sievers ML, Welty TK. Rising tide of cardiovascular disease in American Indians. The Strong Heart Study. *Circulation*. 1999;99:2389–2395.
21. 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.
22. Kumanyika SK. The impact of obesity on hypertension management in African Americans. *J Health Care Poor Underserved*. 1997;8:352–365.
23. Stevens J. Obesity and mortality in Africans-Americans. *Nutr Rev*. 2000; 58:346–353.
24. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *Lancet*. 2002;360:473–482.
25. Wadden TA, Brownell KD, Foster GD. Obesity: responding to the global epidemic. *J Consult Clin Psychol*. 2002;70:510–525.
26. Kumanyika S, Jeffrey 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.
27. Smith DE, Lewis CE, Caveny JL, Perkins LL, Burke GL, Bild DE. Longitudinal changes in adiposity associated with pregnancy. The CARDIA Study. Coronary Artery Risk Development in Young Adults Study. *JAMA*. 1994;271:1747–1751.
28. Kumanyika SK. Minisymposium on obesity: overview and some strategic considerations. *Annu Rev Public Health*. 2001;22:293–308.
29. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11: 496–506.
30. Stettler N, Kumanyika SK, Katz SH, Zemel BS, Stallings VA. Rapid weight gain during infancy and obesity in young adulthood in a cohort of African Americans. *Am J Clin Nutr*. 2003;77:1374–1378.
31. Luke A, Cooper RS, Prewitt TE, Adeyemo AA, Forrester TE. Nutritional consequences of the African diaspora. *Annu Rev Nutr*. 2001;21:47–71.
32. Ravussin E, Valencia ME, Esparza J, Bennett PH, Schulz LO. Effects of a traditional lifestyle on obesity in Pima Indians. *Diabetes Care*. 1994; 17:1067–1074.
33. Kumanyika SK. Obesity treatment in minorities. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment*. New York, NY: Guilford Publications; 2002:416–446.
34. Kumanyika S. The minority factor in the obesity epidemic. *Ethn Dis*. 2002;12:316–319.
35. Levin S, Welch VL, Bell RA, Casper ML. Geographic variation in cardiovascular disease risk factors among American Indians and comparisons with the corresponding state populations. *Ethn Health*. 2002;7: 57–67.
36. Welty TK, Rhoades DA, Yeh F, Lee ET, Cowan LD, Fabsitz RR, Robbins DC, Devereux RB, Henderson JA, Howard BV. Changes in cardiovascular disease risk factors among American Indians. The Strong Heart Study. *Ann Epidemiol*. 2002;12:97–106.
37. US Department of Health and Human Services. *Cardiovascular Risk in the Filipino Community: Formative Research from Dale City and San Francisco, California*. Rockville, Md: US Department of Health and Human Services; 2003.
38. Diabetes Prevention Program Research Group. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension*. 2002;40:679–686.
39. Mills R. *Health Insurance Coverage: 2001*. Washington, DC: US Census Bureau; 2002.
40. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143–3421.
41. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA*. 2002;287:356–359.
42. Resnick HE, Jones K, Ruotolo G, Jain AK, Henderson J, Lu W, Howard BV; Strong Heart Study. Insulin resistance, the metabolic syndrome, and risk of incident cardiovascular disease in nondiabetic American Indians: the Strong Heart Study. *Diabetes Care*. 2003;26:861–867.
43. Wong ND, Pio JR, Franklin SS, L'Italiani GJ, Kamath TV, Williams GR. Preventing coronary events by optimal control of blood pressure and lipids in patients with the metabolic syndrome. *Am J Cardiol*. 2003;91: 1421–1426.

KEY WORDS: AHA Conference Proceedings ■ obesity ■ metabolic syndrome ■ hypertension ■ trials, clinical

**Discovering the Full Spectrum of Cardiovascular Disease: Minority Health Summit 2003:  
Report of the Obesity, Metabolic Syndrome, and Hypertension Writing Group**

Sidney C. Smith, Jr, Luther T. Clark, Richard S. Cooper, Stephen R. Daniels, Shiriki K. Kumanyika, Elizabeth Ofili, Miguel A. Quinones, Eduardo J. Sanchez, Elijah Saunders and Susan D. Tiukinhoy

*Circulation*. 2005;111:e134-e139

doi: 10.1161/01.CIR.0000157743.54710.04

*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2005 American Heart Association, Inc. All rights reserved.

Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the  
World Wide Web at:

<http://circ.ahajournals.org/content/111/10/e134>

**Permissions:** Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

**Reprints:** Information about reprints can be found online at:  
<http://www.lww.com/reprints>

**Subscriptions:** Information about subscribing to *Circulation* is online at:  
<http://circ.ahajournals.org/subscriptions/>