

Discovering the Full Spectrum of Cardiovascular Disease Minority Health Summit 2003 Executive Summary

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Of all the forms of inequality, injustice in health is the most shocking and inhumane. —Martin Luther King, Jr

The American Heart Association (AHA) has a stated goal of achieving a 25% reduction in coronary heart disease, stroke, and the risk for these diseases by 2010, with a specific emphasis on people who are at highest risk. To meet this goal, best practices in prevention, diagnosis, and treatment need to be applied broadly to the US population. That population is now described by a remarkably changing demographic profile. During the past several decades, the US population has become much more ethnically diverse than it once was. The current representation of non-Hispanic whites in this country has declined to 67% and is expected to be at 60% in another decade, and perhaps as low as 50% by the middle of the twenty-first century.¹ According to the US Census Bureau, the most populous state, California, no longer has a single majority population. Hispanics now represent 14% to 15% of the US population. The Hispanic segment is not only the largest minority population but also the fastest growing segment of the US population. People of predominantly African descent represent another 12% of the US population, and people of Asian descent and Native Americans constitute the remainder of the population.² If there is to be a meaningful impact on death and disability resulting from heart disease and stroke in the United States, then the diversity of the US demographic must be considered.

Unfortunately, an increasing database now firmly demonstrates that there are striking cardiovascular disparities as a function of race/ethnicity. *Disparities in health care* are defined as racial or ethnic differences in the quality of health care that are not due to access-related factors or clinical needs, preferences, and appropriateness of intervention.³ The most recent data from the AHA's 2005 Statistical Fact

Sheets⁴⁻⁷ indicate that the most striking disparity of all, mortality from heart disease and stroke, is highest in racial/ethnic groups (Figures 1 and 2). As detailed below, the genesis of disparities in health care is multifactorial and varies across health indicators and healthcare settings; it includes a complex milieu of genetic (genes and genetic diversity), physiological (differential burden of risk factors), cultural (bias in healthcare delivery systems, access to care, geography, and language), and socioeconomic (racial/ethnic bias, poverty, and education) factors. Regardless of the origin of disparities in health care, the elimination of disparate outcomes and the application of advances in heart disease and stroke therapies for the whole population represent necessary goals of the highest order for the AHA, its partner organizations, and its constituency.

Conference Participants and Process

In October 2003, the AHA convened the Minority Health Summit, consisting of a multidisciplinary group of social scientists, clinician scientists, population scientists, and basic scientists. These thought leaders in the field of cardiovascular disease (CVD) were charged with addressing the burgeoning awareness of disparate CVD outcomes as a function of race and ethnicity.

Representatives and members from key organizations that address disparities in health care were in attendance, including the American College of Cardiology (ACC); the Asian and Pacific Physicians Association; the Association of American Indian Physicians; the Association of Black Cardiologists (ABC); the Centers for Disease Control and Prevention (CDC); the International Society on Hypertension in Blacks (ISHIB); the National Heart, Lung, and Blood Institute (NHLBI); the National Institute of Neurological Disorders

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The reports of the Basic Science, the Outcomes, the Obesity, and the Advocacy Writing Groups are available online at <http://www.circulationaha.org> (*Circulation*. 2005;111:e120–e123; e124–e133; e134–e139; and e140–e149). (*Circulation*. 2005;111:1339-1349.)

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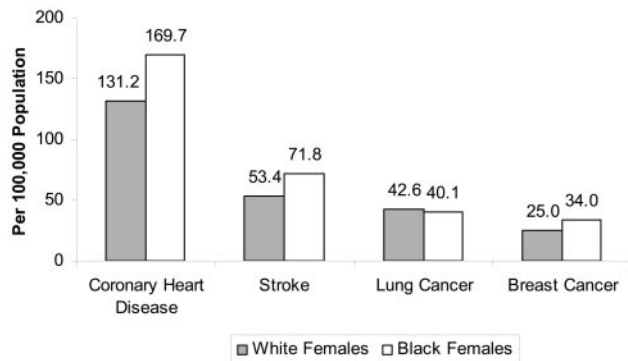


Figure 1. Age-adjusted death rates for coronary heart disease, stroke, and lung and breast cancers for US white and African American women, 2002. Adapted from *Heart Disease and Stroke Statistics*. Dallas, Tex: American Heart Association; 2005.

and Stroke (NINDS); the National Hispanic Medical Association; the National Medical Association (NMA); and the Robert Wood Johnson Foundation. This AHA-sponsored meeting was prompted by consistent research highlighting differences in CVD presentation, management, and outcomes along racial and ethnic lines and a lack of cohesive thought among clinicians, clinician scientists, basic investigators, and community leaders. The purpose of this summit was to explore the extent of known disparities in CVD, identify the behavioral and social underpinnings of poor outcomes in racial/ethnic groups, develop strategies to overcome these disparities, and serve as a catalyst for future AHA initiatives. Two parallel themes of discussion were established: advocacy and science. Each group reviewed the current literature, held extensive deliberations, and generated summary documents that can be found in the online version of this issue of *Circulation* (<http://circ.ahajournals.org/content/vol111/issue10>),^{8–11} The actual deliberations of the Summit are available as webcasts at <http://www.americanheart.org/presenter.jhtml?identifier=3019526>. This Executive Summary captures the themes of the meeting and proposes an agenda to address disparate outcomes in CVD.

Percent of Total Deaths

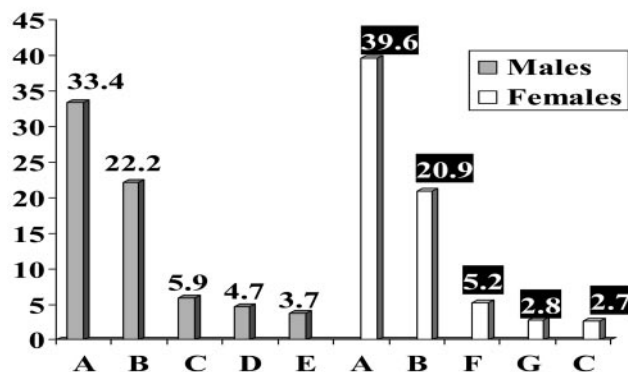


Figure 2. Leading causes of death for African American men and women, 2002. A indicates total CVD (preliminary); B, cancer; C, accidents; D, assault (homicide); E, HIV/AIDS; F, type 2 diabetes mellitus; G, nephritis, nephrotic syndrome, and nephrosis. Adapted from *Heart Disease and Stroke Statistics*. Dallas, Tex: American Heart Association; 2005.

Two seminal reports served as the framework for this conference: the Kaiser Family Foundation/American College of Cardiology Foundation report *Racial/Ethnic Differences in Cardiac Care: The Weight of the Evidence*¹² and the Institute of Medicine (IOM) report *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*.³ The Kaiser Family Foundation/American College of Cardiology Foundation report defined the disparate application of diagnostic and therapeutic procedures for CVD. The Kaiser report evaluated 81 studies published during an 8-year period, which were selected because they included previously underrepresented minority groups in a sufficient number that a reasonable statement about study outcomes could be made as a function of race.¹³ In 68 of 81 studies, there was demonstrable evidence that minorities received less than the standard of care. Only 11 of 81 studies found a similar application of standard therapeutic interventions.¹³ Notable among the many data points was the reduced likelihood that African American patients, compared with white patients, would undergo either percutaneous coronary intervention (odds ratio 0.20 to 0.87) or coronary artery bypass grafting (odds ratio 0.26 to 0.68).^{12,13} The application of thrombolytic therapy was applied 50% less often in African American patients despite similar disease presentation.^{12–14} Similar differences are noted with Hispanic/Latino Americans. Nine studies provided data on Hispanics/Latinos with half of the studies finding Latinos less likely than whites to undergo cardiac procedures.^{12,13} Five studies on Asian Americans showed a similar disparate use of cardiac procedures. Only one study included data on Native Americans.¹⁵ Of the strongest studies evaluated, 19 of 24 demonstrated that at least one racial/ethnic minority group was less likely to undergo cardiac catheterization even when age, insurance, comorbidities, and/or disease severity were considered. Twenty-six studies of coronary artery bypass grafting were believed to be methodologically strong. Of the 26 studies, 24 found at least one racial/ethnic group that was less likely than were whites to undergo surgical revascularization; again, this difference was apparent despite appropriate consideration of age, insurance status, comorbidities, and/or disease severity.^{12,13}

The IOM report³ undertook a similarly exhaustive review of the available literature and focused on both disparate therapeutic interventions and disparate care processes. No single factor appeared consistently among the described studies, suggesting that differential outcomes in health care are multifactorial in origin. Among the many factors identified by the IOM report, several were considered to be major contributions to disparities in health care: clinical appropriateness, patient choice, the healthcare delivery system, and bias. Given that disease presentation may vary considerably, clinical appropriateness dictates which interventions are indicated. Patients may choose to either embrace or reject a recommended treatment option. The healthcare delivery system may compromise patients because it lacks cultural sensitivity, allows for persistent communication barriers for non-English-speaking patients, or is simply too difficult to access because of insurance or financial limitations. Bias exhibited by practitioners, even if unrecognized, may preclude an objective decision-making process. These and many

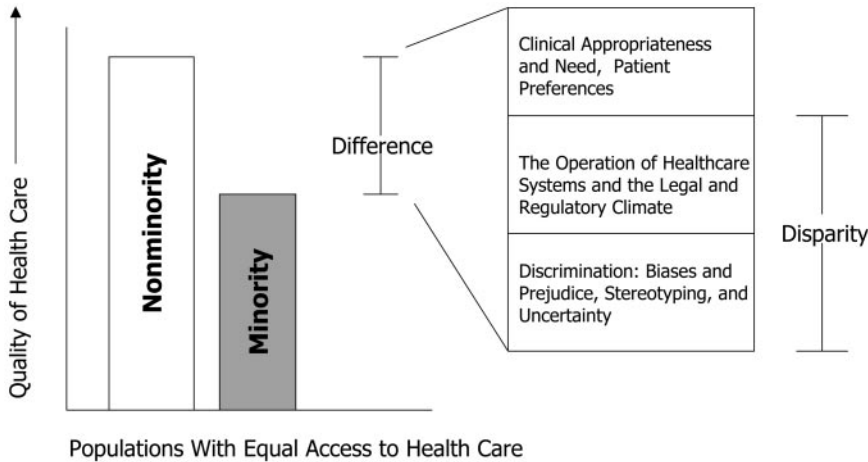


Figure 3. Disparities, racial, or ethnic differences in health care that are not the result of access-related factors, clinical needs, patient preferences, or the appropriateness of the intervention. Adapted with permission from Smedley et al.³

other factors are presumed to be responsible for observed disparities in CVD presentation, management, and outcomes (Figure 3).³

Taken together, these two compelling data sources establish both the existence and the complexity of disparities in health care. We opted to address the broad issue of disparate CVD health care from several perspectives: basic science, epidemiological science, risk factors, clinical trials, and the sociological and cultural basis of clinical disease.

Basic Science

Taking a basic science perspective on disparities in health care necessarily raises the argument that genetic differences may be at the core of differential disease expression and may be integral to altered responses to standard medical therapies. It must be emphasized that race is a poor, perhaps even unacceptable, proxy for genetics. Knowledge culled from the Human Genome Project and research on human genome variation increasingly challenges the applicability of the term “race” to human population groups and raises questions about the validity of inferences made about race in the biomedical and scientific literature.¹⁶ If genetic variation exists among racial groups, it is likely due to the presence of DNA sequence variations called single nucleotide polymorphisms, which result in altered protein function. At least 15 million genetic polymorphisms exist within the human genome¹⁷; however, certain at-risk polymorphisms may be more common in certain racial groups. Classic examples include factor V Leiden mutation (recurrent thrombosis), β -globulin gene mutations (sickle cell anemia), and renal sodium channels in the kidney (ENaC, leading to salt-sensitive hypertension).^{18,19} New data reveal a growing list of candidate polymorphisms that may be especially pertinent in people of African descent and could be plausibly implicated in the genesis of excessive CVD.²⁰ Even in this regard, no evidence exists that any genetic polymorphism achieves 100% distribution within a racial group. Importantly, the influence of genetics must be considered to be contextual: Gene–gene interactions, gene–drug interactions, and gene–environment interactions all may lead to differential disease expression.^{21,22}

In this careful context, several genetic variables emerge as plausible factors that contribute to differential disease expres-

sion. The HyperGEN Network (Hypertension Genetic Epidemiology Network)²³ suggests that early-onset hypertension in African American patients might be linked to chromosomal loci, including the mineralocorticoid receptor on chromosome 4. Yet another candidate gene is the C825T polymorphism of the gene encoding for the G protein β_3 subunit (*GNB3*).²⁴ Both the TT allele and the CT allele for this gene confer a greater risk of left ventricular hypertrophy than does the CC homozygote. This C285T polymorphism is seen in Americans of African descent, Africans, and Aborigines, but its penetration is highly variable.²⁵ A genetic basis also may be pertinent in understanding the response to medical therapy for CVD. Variants in the cytochrome P450 enzymes have been implicated in the response to β -blocker therapy.²¹ Most recently, loss-of-gain β_1 receptor polymorphisms, focused on position 389, have been suggested as plausible explanations for suboptimal responses to β -blockers in African American patients.²⁶ This loss-of-gain function may explain the sub par response to β -blocker therapy that has been observed in the African American cohort in the Beta-Blocker Evaluation of Survival Trial (BEST)²⁷ and is the source of ongoing investigation.

Beyond these described variables, a growing awareness of alterations in nitric oxide bioactivity is emerging, and recent clinical trial data strongly assert that nitric oxide enhancement is a beneficial treatment option for African American patients with advanced heart failure.²⁸ Polymorphisms in the endothelial nitric oxide synthase gene (*NOS3*; previously called eNOS), specifically the 4a variant in intron 4 in African Americans, are associated with a decrease in the expression of *NOS3* and a reduction in nitric oxide production.²⁹ In addition, glucose-6-phosphate dehydrogenase is important in the production of NADPH, which is necessary for the production of nitric oxide, yet glucose-6-phosphate dehydrogenase deficiency occurs in up to 15% of African Americans.³⁰ Healthy African American women have been shown to have lower circulating levels of nitric oxide and higher levels of superoxide and peroxynitrate than healthy white women of similar age.³¹ This combination of impaired nitric oxide bioactivity and increased oxidant stress may participate in the genesis of premature or aggressive (or both) CVD in African American patients.

Any discussion of genetics and race yields troublesome questions that eclipse science and involve the sociopolitical framework. An understanding of racial differences in genetic variation is a reasonable target of research to better characterize clinical phenotypes now crudely represented by race. Nevertheless, any such discoveries need to be interpreted carefully and within the appropriate clinical, socioeconomic, and environmental context.

Epidemiology

The epidemiology of CVD has been instrumental in our understanding risk, targeting of interventions, and generating plausible hypotheses to drive clinical investigation. The unfortunate paradox has been that we have the least amount of epidemiological data for the groups that appear to be at higher risk for CVD. Many of the major epidemiological studies have predominantly involved whites and have lacked sufficient diversity to be reliably and consistently generalized to other racial/ethnic groups. Several new data sources have emerged, however, that either are more inclusive of the broad population or have focused on previously underrepresented at-risk groups.

National Health and Nutrition Examination Survey

During a >30-year period, data from the NHANES (National Health and Nutrition Examination Survey) study have emerged that uniquely characterize CVD and risk for racial/ethnic minorities. NHANES III oversampled African American, Mexican American, and white non-Hispanic women from 1988 to 1994.³² The aggregate data from NHANES have alerted the community to the striking prevalence of obesity in both African Americans and Mexican Americans and have suggested that 80% of African American women >40 years old are overweight.³³ Mexican American women bear a disproportionate burden of obesity as well, and differences in body mass index for both Mexican American and African American children become apparent in the first 2 decades of life.³⁴ Both African American and Mexican American men have a higher prevalence of dangerous risk factors including diabetes, systolic hypertension, and physical inactivity. NHANES is incapable of assigning mechanisms to this preponderance of risk, but plausible variables include socioeconomic stress, cultural pressures, and lifestyle choices.

Honolulu Heart Program

Since 1965, the Honolulu Heart Program has studied ≈8000 Japanese men living in Oahu to determine the reasons for the differences in the rate of heart disease and stroke among Japanese men living in Japan, Hawaii, or the US mainland.^{35,36} The Honolulu Heart Program has identified the adverse risk of impaired glucose tolerance in this population and demonstrated its striking correlation with thromboembolic stroke, hemorrhagic stroke, coronary artery disease, sudden cardiac death, and all-cause mortality. Of note, despite having a “normal” body mass index (<25 kg/m²), many Japanese men, especially older men, had diabetes.^{35,36} Both the threshold fasting blood glucose for the diagnosis of

diabetes and the definition of normal weight by anthropometric standards may need to be redefined for Japanese men.

Strong Heart Study

The Native American population likely has the least-well-understood profile of CVD. Striking heterogeneity among the >500 tribes in the United States along with geographic dispersion and both cultural and language barriers have made the accumulation of data on this group challenging. The National Heart, Lung, and Blood Institute has supported an epidemiological survey of American Indians known as the Strong Heart Study since 1988.³⁷ The Strong Heart Study has demonstrated that mortality due to CVD is at least equivalent to, if not higher than, that observed in the general population. Diabetes is a particularly prevalent risk factor, and outcomes of people affected with CVD are less than ideal.³⁸ A more in-depth discussion of misclassification of CVD disease in Native Americans can be examined in this issue of *Circulation*.³⁹

Multi-Ethnic Study of Atherosclerosis

Another National Heart, Lung, and Blood Institute-supported effort, the Multi-Ethnic Study of Atherosclerosis (MESA), is designed to study the prevalence, correlates, and progression of subclinical CVD in multiple ethnicities. Nearly 7000 patients are involved and emanate from 4 distinct groups: white (38%), African American (28%), Hispanic (22%), and Chinese (12%).⁴⁰ Basing their research on the premise that subclinical markers of disease may be present in varying proportions among ethnic groups and that the progression of CVD may likewise vary, MESA investigators are studying a number of new markers of disease, including coronary artery calcium scores from electron beam and multislice computed tomography, carotid intimal-medial thickness, and ankle-brachial index. Early data from MESA demonstrated that African Americans have thicker vascular walls than do whites and that the prevalence of coronary artery calcification also may differ.⁴¹ This issue of *Circulation* features one of the first published results from the MESA study,⁴² which documents racial/ethnic differences in CVD risk factors and coronary artery calcification. Ongoing surveys including the Dallas Heart Disease Prevention Project and the Jackson Heart Study⁴³ will provide additional multiethnic data that should further illuminate disparities in CVD.

Observational studies confront many challenges in investigating racial/ethnic disparities in CVD. The requirement for self-identification of ethnicity and race complicate the assessment of disparities, and an increasing percentage of the US population now declares a multiethnic or biracial heritage. In addition, the amalgamation of different ethnicities and races within US society challenges the very existence of race as a definite entity. Much of what is currently identified as race will need to be reclassified along more definitive demographic, physiological, or cultural lines. The CVD burden attributable to lower socioeconomic status is likely a core component of disparate health care and undoubtedly permeates all racial and ethnic boundaries; it may actually supplant both. Socioeconomic status is difficult to objectify, however, and is poorly characterized in most databases, which further obscures identification of the real population at risk.

Risk Factors

The evaluation of risk factors allows a further understanding of disparities in CVD in racial/ethnic groups. An extraordinary burden of risk is borne by the confluence of obesity, hypertension, and the metabolic syndrome. Obesity, defined as a body mass index of ≥ 30 kg/m², has become epidemic in the US, with a disproportionately higher burden in African American women, Mexican Americans, Puerto Ricans, American Indians, Alaska Natives, and Pacific Islanders.^{44,45} Obesity varies by region, socioeconomic status, and other demographic variables. Patterns of obesity for racial/ethnic minorities may differ substantially from those for whites. In whites, obesity prevalence is inversely related to socioeconomic status, whereas in some racial/ethnic groups an opposite gradient is noted. Nevertheless, obesity is consistently associated with CVD risk and the development of diabetes,⁴⁴ though mortality rates for CVD do not reliably track the prevalence of obesity in racial/ethnic groups.⁴⁶ This is especially pertinent for Mexican Americans but less so for African Americans. Interventions to alter the rates of obesity, especially in children of at-risk racial/ethnic groups, are challenged in part by socioeconomic constraints and by sociocultural influences on lifestyle and behavior.

Hypertension is more prevalent in racial/ethnic minority groups than in whites and the consequences of hypertension-related illnesses are greater in these groups, especially in African Americans. Target-organ damage occurs with an alarming frequency in African Americans with hypertension: End-stage renal disease is as much as 20-fold more likely than in whites⁴⁷; the incidences of both thrombotic and hemorrhagic stroke are higher, left ventricular hypertrophy is as much as 3-fold higher,⁴⁸ and heart failure is twice as likely.⁴⁹ In Hispanic Americans, the detection, awareness, and treatment of hypertension are notably lower. The treatment of hypertension, especially in African American patients, appears to be sensitive to the racial/ethnic grouping. More intensive therapy (ie, multi-drug regimens) typically is required in African American patients,⁵⁰ and the response to usual blood pressure-lowering agents may vary slightly as a function of race. It is imperative, however, that control of blood pressure be achieved at targeted levels as suggested by both the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure-7 guidelines⁵¹ and the International Society on Hypertension in Blacks treatment algorithm.⁵²

Data from NHANES III demonstrate that the metabolic syndrome affects 47 million people in the United States, with the highest rates observed in Hispanic American women and the lowest rates observed in African American men.⁵³ The individual features of the metabolic syndrome also may vary as a function of ethnicity/race. Whites of European origin appear to be more susceptible to atherogenic dyslipidemia, whereas African Americans are more at risk for hypertension, obesity, and type 2 diabetes mellitus. Hispanic Americans and Native Americans are more vulnerable to diabetes but develop hypertension less frequently than do African Americans. The origin of the metabolic syndrome is presumed to be obesity, which is theoretically prevent-

able, and the consequences of the metabolic syndrome are modifiable with medical therapy. Thus, the burden of CVD explained by the metabolic syndrome appears to be a suitable target for prevention and intervention strategies. It has been estimated that aggressive management of the metabolic syndrome would result in an $\geq 80\%$ reduction in cardiovascular events,⁵⁴ many of which occur in racial/ethnic groups. Specific therapy for the metabolic syndrome per se does not exist, and any future research in this area must necessarily reflect the population at risk and include high-risk racial/ethnic groups.

Clinical Trials

The most reliable repository of data on the treatment of CVD emanates from clinical trials. Much like the experience with epidemiological data, many of the reference clinical trials (from which guidelines that direct care evolve) have had striking variability in the inclusion of racial/ethnic groups. Extrapolation of the findings from these large trials, which typically include white and male patients, to the general population requires considerable latitude in clinical trial interpretation and ignores the observed dissimilarity in outcomes of CVD as it affects racial/ethnic groups. The landmark HOPE (Heart Outcomes Prevention Evaluation) trial involved so few African American subjects that no meaningful statement can be made about the benefit of angiotensin-converting enzyme (ACE)-inhibitor therapy in patients at risk for CVD who happen to be African American.⁵⁵ Conversely, the LIFE (Losartan Intervention for Endpoint Reduction) trial included a modest-size group of African American patients ($\approx 6\%$), but a retrospective post hoc analysis of this underpowered subgroup demonstrated that the benefit of losartan on the primary end point was inferior to that of atenolol—a finding opposite of that observed in white patients.⁵⁶ A statement that now appears in the product label for losartan notes that the benefits of losartan seen in LIFE may not apply to African American patients. Given the striking mortality benefit provided by losartan to patients with hypertension, diabetes, and left ventricular hypertrophy and the reduction in the progression to stroke in all participants, it is problematic that a large at-risk segment of the population is dissuaded from being treated with this effective regimen.

Few clinical trials have prospectively acquired a meaningful representation of racial/ethnic groups and virtually none has done so in a statistically rigorous way. Thus, most of the data from clinical trials pertinent to outcomes in these groups have been the result of compromised post hoc underpowered analyses. The risks of such an exercise involve both the exaggeration of positive effects and the potential overstatement of apparent risks as noted in the foregoing example. Only recently have prospective trials been done, either of sufficient size to incorporate meaningful subgroup data (eg, Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial [ALLHAT]⁵⁷) or in a single at-risk group [eg, African American Study of Kidney Disease and Hypertension [AASK]⁵⁰ and the African American Heart Failure Trial [A-HeFT]²⁸], to make meaningful statements about targeted groups. The lessons learned from these experiences have been illuminating.

As a group, African American patients with hypertension respond well to thiazide diuretics (eg, chlorthali-

done),⁵⁷ but in groups at higher risk, such as African American patients with hypertensive nephrosclerosis, a multidrug regimen that includes ACE inhibitors (previously believed to be ineffective in African Americans) retards the progression of chronic kidney disease.⁵⁰ Much debate has surrounded the use of β -blockers for heart failure in African Americans, with disparate reports in the literature^{27,58}; however, the recently completed prospective A-HeFT trial conducted exclusively in African American subjects demonstrated a control population (ie, treated with diuretics, β -blockers, and ACE inhibitors or angiotensin-receptor blockers) and an event rate (10-month mortality rate of 10% for class III heart failure with impaired left ventricular ejection fraction on evidence-based medical therapy) that were entirely consistent with published results for patients with similar heart failure–disease severity.²⁸

New data emerging from the ADHERE (Acute Decompensated Heart Failure National Registry) database⁵⁹ define the incidence of heart failure in Hispanic Americans. Only 3% of those patients affected by acute decompensated heart failure are Hispanic American despite a high prevalence of known risk factors, especially diabetes. Outcomes of acute decompensated heart failure for Hispanic Americans with heart failure are worse than in whites but better than that observed in African Americans.⁶⁰ Reasons for the difference in disease expression and the differential outcomes are not readily apparent but emphasize the growing issue of cardiovascular health disparity as a function of racial/ethnic designation.

Stroke is the third leading cause of death in the United States and the leading cause of serious long-term disability in adults. African Americans carry an excess burden of stroke incidence and mortality rates.⁴ In fact, the disparities seen in stroke in African Americans are even higher than those observed in African Americans with heart disease.⁴ The incidence of stroke among African Americans is about twice that of US whites.⁴ Even after adjusting for established vascular risk factors such as diabetes, hypertension, and established coronary heart disease, African Americans still have a 38% greater risk of incident ischemic stroke as compared with whites.⁶¹ The excess mortality rate due to stroke is greater in the younger age groups of African Americans, and those who are between 35 and 44 years old are 4 times more likely to die from stroke as compared with white Americans of the same age. The relative risk of stroke mortality in the 45- to 54-year and 55- to 64-year age groups is 3.9 and 1.9, respectively.⁴ Substantial geographic variation in stroke mortality exists in the United States for both African Americans and whites and the causes of this variation are largely unknown. The so-called Stroke Belt defines a region of the United States that includes contiguous states in the southeast with the highest incidence of and mortality from stroke in the United States. Within the Stroke Belt, the highest rates of mortality are seen in Georgia, North Carolina, and South Carolina.⁶² The reasons for ethnic differences in stroke incidence and mortality rates are not well understood and few studies have examined racial/ethnic differences in

stroke risk factors. Some studies have shown that among patients who are hospitalized for ischemic stroke, a higher prevalence of risk factors exists for recurrent stroke among African American or Hispanic patients,⁶³ and a more recent study showed that African American and Hispanic stroke survivors were more likely than other racial/ethnic groups to report diabetes, obesity, and inadequate levels of physical activity. African Americans were more likely to report hypertension.⁶⁴

The National Institute of Neurological Disorders and Stroke supports the major multicenter randomized, controlled clinical trials in the prevention and treatment of stroke, including AAASPS (African American Antiplatelet Stroke Prevention Study) and the WASID (Warfarin versus Aspirin for Symptomatic Intracranial Disease) trial, which investigated the antithrombotic management of intracranial stenosis, especially common in African Americans.^{65,66} To date, it is not clear whether racial/ethnic differences exist with regard to the efficacy of evidence-based treatment strategies for stroke.

Sociological and Cultural Basis of Clinical Disease

Among the varied ways to address the question of disparities in CVD, a better understanding of the cultural landscape of the United States is preferable. Significant cultural diversity exists in this country, and cultural awareness, more precisely termed “cultural competence,” is at the core of any effort to fully address disparities in CVD.⁶⁷ Cultural competence has a broad definition and encompasses clarity in communication with those for whom English is not the primary language, an imperative to incorporate a more attuned healthcare delivery system for the unique needs of the population served, a necessity to mesh resources (local, state, federal, and private) to effect greater change in disparities in health care, and a need to build more trust between providers and the communities served.

An especially challenging issue is the definition of “minority.” Important cultural variations are evident both between and within minority groups. Neither uniformity in CVD risk nor homogeneity in disease expression exists as a function of race/ethnicity. Assessing the mean behavior of a group is inherently risky, and discussions about disparities in CVD in racial/ethnic groups must be conducted with extreme caution. The risk of generalization of disease to one group to the exclusion of patient-specific assessments is a worrisome consequence of this dialogue, and the practitioner is reminded of the need to avoid such generalities.

Communication issues are a special concern. The majority of non-English-speaking Latinos do not have a regular physician and among those who have visited a physician, significant communication barriers have been identified. This issue is magnified for patients of Asian descent because lack of inclusiveness of these targeted groups in the healthcare system is also identified as a component contributor to disparities in CVD.⁶⁸ Furthermore, the lack of a professional workforce from targeted racial/ethnic groups in the acquisition of data, interpretation of data, dissemination of information, treatment, and administration of health care also may affect CVD outcomes. For many of the targeted groups, the community infrastructure

either facilitates or impedes appropriate health care. Substantial obstacles must be addressed to embrace communities, and among racial/ethnic groups the community support and challenges vary. Clearly, the lack of trust between healthcare providers and patients is a major hurdle that must be addressed. The trust factor is enhanced by conscience (ie, appropriate cultural awareness) and professional competence while being limited by access to health care and bias (Figure 4). The provider issue is further complicated by a higher representation of non-board-certified practitioners who lack additional subspecialty training in the care of at-risk racial/ethnic groups.

Summary and Recommendations

The issues of disparities in CVD, when viewed through the prism of ethnicity and especially race, are both polarizing and paralyzing. Few issues are more volatile in US society, but even fewer are more important with regard to cardiovascular health and outcomes. The inexorable momentum toward the amelioration of CVD and stroke in the United States has been laudable, but the underrepresentation of all segments of the population is regrettable and keeps the level of CVD burden elevated. The commensurate consequences of this dissimilarity in the application of state-of-the-art prevention, diagnosis, and treatment of CVD have far-reaching effects on the economic, political, and social constructs of our society. It is appropriate to have these sensitive discourses, not because it is politically correct but because it is medically justified given the high CVD burden observed in racial/ethnic groups. If the discussions about CVD disparities result only in the healthcare provider and the healthcare delivery system's achieving a heightened sense of awareness of the acuity and severity of the CVD burden in targeted racial/ethnic groups, then much will have been accomplished. Much remains unknown about racial/ethnic disparities in CVD, however, and as such, participants in the Cardiovascular Science and Health Care Disparities Minority Health Summit sought to develop recommendations, broad based in nature, to invite dialogue and action on the issue. Racial/ethnic disparities in CVD are indeed complex and multifactorial, and they occur at all levels of the medical care system. Any truly effective amelioration of disparities requires a partnership among governmental, public, private, and individual stakeholders.

The recommendations of the Cardiovascular Science and Health Care Disparities Minority Health Summit follow.

I. Research

1. Genetic

Examine key polymorphisms believed to be responsible for cardiovascular disease and its risk factors; determine whether these polymorphisms are enriched in certain racial/ethnic groups and whether such polymorphisms contribute significantly to racial/ethnic disparities in CVD risk.

Study whether these polymorphisms can be used to guide prevention and treatment in the general population.

2. Environmental

Encourage additional research on racial/ethnic disparities in CVD risk factors, outcomes, and healthcare use and effective risk factor modification.

3. Participation in research

Support efforts to increase minority participation in the conduct of scientific studies, broaden the racial/ethnic groups included, and increase the validity of studies.

Encourage the development of more racial/ethnic-aligned investigators; diversity is critical at all levels of the investigative team.

4. Workforce issues

Improve the number of qualified minority investigators and practitioners, but do not impede the development of others. Such efforts require enlarging the "pipeline" of investigators by providing outreach and training programs to college, high school, and secondary school students.

II. Advocacy

5. Discrimination

Advocate against genetic discrimination in insurance, health care, and employment.

6. Federal funding

Identify gaps in access to high-quality healthcare services.

Fund systems and policy-level research to improve the quality of care.

Focus particularly on obesity and the metabolic syndrome.

7. Increase incentives/reimbursement and implementation of primary prevention; this is critical for children (similar initiatives should be available for adults at risk).

Use healthcare teams.

Encourage community outreach programs in schools, workplaces, and faith-based settings.

Focus on the prevention and treatment of obesity and diabetes by emphasizing attendant risk factors, prevention, and community influences.

8. Improve the availability of care for all Americans regardless of race/ethnicity, socioeconomic status, and geographical barriers.

Increase access to continuity in primary and secondary care in urban, suburban, and rural settings.

Increase economic incentives to disseminate and implement evidence-based guidelines in primary and secondary prevention.

III. Education

9. Continue to highlight CVD racial/ethnic risk factors and outcomes.

Statistical updates should continue to contain racial/ethnic-specific data.

Scientific conferences should integrate information on racial/ethnic disparities in the program.

Update AHA scientific statements and practice guidelines to include appropriate multiracial/ethnic sections.

10. Cultural and linguistic competence

Make sure healthcare providers are trained in cultural competence.

Make sure the public and patients receive linguistically and culturally appropriate educational materials and health care.

Trust Formula
Trust = 2 x Conscience + Competence



Figure 4. Adapted from: Shore DA. *The Trust Prescription for Healthcare: Building Your Reputation With Customers*. Chicago, Ill: Health Administration Press; 2005.

Appendix

Discovering the Full Spectrum of Cardiovascular Disease: Minority Health Summit 2003 Participants

Cardiovascular Science and Healthcare Disparities Program Subcommittee

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Outcomes—Impact From Observational Studies Writing Group

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Clinical Science Writing Group

Domenic A. Sica, MD, Chair; Edgar J. Kenton, III, MD; Jackson T. Wright, Jr, MD, PhD; Martha N. Hill, RN, PhD; Clyde W. Yancy, MD

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Ivor J. Benjamin	University of Utah	None	None	None	None	None
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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 groups were required to complete and submit shortly before the conference.

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