

Racism and Cardiovascular Disease in African Americans

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ABSTRACT: This article provides an overview of the evidence on the ways racism can affect the disproportionate rates of cardiovascular disease (CVD) in African Americans. It describes the significant health disparities in CVD for blacks and whites and suggests that racial disparities should be understood within the context of persistent inequities in societal institutions and relations. Evidence and potential pathways for exploring effects of 3 levels of racism on cardiovascular health risk factors and outcomes are reviewed. First, *institutional racism* can lead to limited opportunities for socioeconomic mobility, differential access to goods and resources, and poor living conditions that can adversely affect cardiovascular health. Second, *perceived/personally mediated racism* acts as a

stressor and can induce psychophysiological reactions that negatively affect cardiovascular health. Third, in race-conscious societies, such as the United States, the negative self-evaluations of accepting negative cultural stereotypes as true (*internalized racism*) can have deleterious effects on cardiovascular health. Few population-based studies have examined the relationship between racism and CVD. The findings, though suggestive of a positive association, are neither consistent nor clear. The research agenda of the Jackson Heart Study in addressing the role of racism in CVD is presented. Race; Racism; Stress; Discrimination; Cardiovascular disease; Hypertension; Behavioral risk factors; Ethnicity; Jackson Heart Study. [*Am J Med Sci* 2003;325(6):315–331.]

Despite decreasing overall levels of cardiovascular disease (CVD) over the past several decades, significant ethnic disparities in morbidity and mortality remain. For example, during the 1980s and 1990s, African American (or black) men and women had rates of decline in coronary heart disease mortality that were 30 and 18% lower than their European American (or white) counterparts. The CVD mortality rate for African Americans in 1995 was 1.6 times that of European Americans. Even more striking is that this rate is identical to that of 1950.¹ Racial discrepancy in heart disease and diabetes, a related risk factor, was larger in 1995 than in 1950. Most impressive is the fact that

CVD deaths, both coronary heart disease and strokes, occur much earlier in blacks than whites.²

CVD mortality rates in the southeastern part of the United States have been higher than in the rest of the nation. In particular, Mississippi, the site of the Jackson Heart Study (JHS), continues to have the highest age-adjusted mortality rates for all race-gender groups. The rates are approximately 25% higher than the national rates. The rates for African American women were 75% higher than in European American women and they were 47% higher in African American men than in European American men.³ Although CVD mortality rates have decreased nationally for African Americans, rates have increased in Mississippi, further widening the health disparity gap between African Americans and European Americans.² Of the potential explanations hypothesized for these inequalities, racism and its effects are among the most disturbing.⁴ Current evidence is scant and difficult to interpret. Emerging theoretical and methodological developments provide new possibilities for addressing the issues of racism and CVD in population studies of African Americans. The JHS, the largest prospective epidemiological study of African Americans in the United States, provides a unique opportunity to unravel the complex relationships among racism and CVD outcomes.

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Race, Racism and Discrimination in the United States

Race: What Is It?

What race actually represents is still subject to debate. Reflecting the enduring theme of race in this country, the United States is the only major industrial country that routinely reports health status variations among its population on the basis of race rather than social class.⁵ Across our history, race has been viewed alternately as a biological and a social construct, fueling the polemic between innate versus acquired bases for health differences between ethnic groups. Early research attempted to document that blacks were biologically inferior, making them more susceptible to a myriad of illnesses.⁶ Racial taxonomic approaches to classifying genetic differences assume that race is a valid biological category and that genes determining race likewise determine the health problems that manifest. Countermanding this early biological reductionist explanation was evidence that apparent differences in health outcomes were not the consequence of innate factors but of inherited, socially created environments. Social taxonomic approaches assume that race is a social classification, based on phenotype, that governs the distribution of risks and opportunities in our race-conscious society. As such, it reflects a cultural heritage and measures a societally imposed identity and consequent exposure to the societal constraints associated with that identity. From this perspective, race is thought to measure some combination of social class, culture and genes.⁷ Jones,⁸ in a recent commentary on the use of race in epidemiological research, further suggests that the race an investigator notes or a subject self-reports is a measure, at least in part, of exposure to racism. She notes, "it is perhaps this aspect of race that profoundly impacts health and results in race-associated differences in health outcomes that are large in magnitude, occur across the life span, and involve many different organ systems."

Racism and Discrimination

Some argue that racism no longer exists in the United States, and a recent review of available national survey data reveals increasing egalitarian attitudes among the white population.⁹ Yet these improving attitudes continue to coexist with a desire to maintain at least some social distance from blacks and a less than resounding commitment to social policies that aim to eradicate entrenched inequalities.¹⁰ Likewise, endorsement of negative stereotypes of blacks among whites persists. Considerable cultural support for racist attitudes, institutions, and policies is suggested by the persistence of negative stereotypes of African Americans. Likewise, analyses of the prevalence of lifetime, recent, and

everyday racism experienced by blacks and whites in Detroit reconfirms that racism remains a significant obstacle for blacks, including the most successful.¹¹

Racism is viewed as a multifaceted social category that captures differential access to power and desirable resources in society.⁴ Racism refers to an organized system, rooted in an ideology of inferiority, that categorizes, ranks, and differentially allocates societal resources to human population groups.¹² It may or may not be accompanied by individual prejudice providing the context within which stereotypes (overgeneralized labels of persons or groups), prejudice (negative judgments and attitudes), and discrimination (unfair treatment) are developed and sustained.¹³

Jones⁸ argues that further specificity of the characteristics and mechanisms of racism is needed if we are to discern whether racism is indeed a root cause of observed race-associated health disparities. Toward that end, she delineates 3 levels of racism: institutionalized, personally mediated, and internalized. For each level, she proposes a pathway through which racism may act to produce deleterious health outcomes. The simultaneous exposure to racism at all levels composes the total experience of racism.¹⁴ Research examining the effects of racism on health outcomes is in its infancy; few studies explicitly examine the impact of racism on various health outcomes. Table 1 provides a chronologically ordered synopsis of research using community samples to directly examine the relationship of racism with CVD risk factors or outcomes. The remainder of this article will review the pathways and overview the extant evidence for disparate cardiovascular health consequences in African Americans for each level of racism.

Institutionalized Racism and CVD

Carmichael and Hamilton coined the term *institutional racism* in 1967 as a way of describing the systematic, more subtle forms of racism whereby historically mediated societal ideologies, practices, and policies concerning race are sustained in organizations and systems through customs, standards, and regulations.¹⁵ Institutional racism takes on a life of its own, separate from the persons within organizations, who may harbor no racial prejudice. Jones,¹⁶ defining institutional racism, states: . . . it is normative, sometimes legalized, and often manifests as inherited disadvantage. It is structural, having been codified in our institutions of custom, practice and law, so there need not be an identifiable perpetrator. Indeed, institutional racism is often evident as inaction in the face of need. Institutional racism manifests itself both in material conditions and in access to power. . . has its origins in discrete historical events but persists because of structural factors

Table 1. Chronological Summary of Research Linking Racism with CVD Risk Factor/Outcome

Study	Sample	Measure of Racism/Discrimination	Measure of Risk Factor/CVD Outcome	Findings/Discussion
James et al, 1984	112 black men in North Carolina	Race as hindrance to job success, unfair wages	Self-report hypertension (Ever told by health professional had high blood pressure of hypertension)	Discrimination + associated with hypertension, but not in dose-response relationship
Krieger, 1990	51 black and 50 white women in Oakland, CA	Ever discriminated against at school, getting a job, at work, getting housing, getting medical care, from police or in the courts Response to unfair treatment (accept vs do something, talk to others vs keep it to self) Four questions on discrimination at work, regarding pay raises, promotion, job responsibilities, overall pay as part of items in scale on long-term stress	Self-report hypertension (Ever told by health professional had high blood pressure of hypertension)	Black women who report few or no experiences of discrimination and respond by keeping quiet at greatest risk for hypertension
Dressler, 1990	186 African American adults in Alabama	16-item organizational prejudice-discrimination scale developed by authors	Self-report hypertension (Ever told by health professional had high blood pressure of hypertension)	No effect on BP
James et al, 1994	89 minority workers; 18% African American	Variation of Krieger (1990): Past 3 years experience of discrimination, prevented from doing something or made to feel inferior in getting a job, at work, at home, shopping, and from police Two items: Perception of whites' intentions (keep blacks down, blacks get a better break, don't care); they or family member treated badly because of race in past 30 days	Self-report of HTN within past 2 years BP measured by digital finger-cuff or mercury sphygmomanometer	Discrimination + related to BP; negatively mediated by collective esteem
Broman, 1996	Cross-sectional sample of 312 African American adults (18 years or older) in Detroit from Wave 2 of random digit dial- telephone panel survey (Detroit Area Survey). Sample over represents middle class African Americans.	Two items: Perception of whites' intentions (keep blacks down, blacks get a better break, don't care); they or family member treated badly because of race in past 30 days	Self-report hypertension (Ever told by health professional had high blood pressure of hypertension)	No effect of experience of racism on self-reported BP or CVD If responded with anger, non-significant trend in direction of > BP/CVD
Jackson et al, 1996	623 African American adults participating in four waves, national panel data from National Survey of Black Americans	Krieger, 1990 plus ever discriminated against on the street or in a public place	Count of doctor-reported serious health conditions (including CVD) Health disability	Not related to health outcomes, but if report > 2 experiences in 30 days tend to have lower levels of health problems and disability
Krieger & Sidney, 1996	4,086 black and white adults in multi-center CARDIA study.	McNeilly Perceived Racism Scale (51 items) with frequency of exposure in past year and lifetime on the job, in academic settings, in public settings, racist statements	Random zero sphygmomanometer measured systolic and diastolic blood pressure	Elevated BP in blacks who denied discrimination and accepted unfair treatment; Decreased BP in blacks who typically challenge unfair treatment and denied discrimination
McNeilly et al, 1996	165 African American college students and 25 adults in North Carolina	See Krieger & Sidney, 1996	None	None-developed for use with public health and CVD research
Murrel, 1996	165 African American women in northern California	Perceptions of Racism Scale (Green,1995) (20 Item) medical, life experiences of racism	Stress Birth weight	Increased stress No association with low birth weight
Yen, 1999	883 Multiethnic participants in San Francisco Muni Health and Safety Study. 476 were African American	See Krieger & Sidney, 1996	Drinks/month calculated from self-report of times/mo and drinks/occasion 4-item CAGE for ETOH dependence	+ association with > number of domains of discrimination and drinks/mo, likelihood of heavy drinking, ETOH dependence
Collins et al, 2000	25 case and 60 control subjects African American low income women giving birth at Cook County Hospital, 1994-1996	See Krieger, 1990. Ever experienced racism in major domains and active/passive coping response	Birth weight in grams (very low birth weight for cases < 1500 g; non-low birth weight controls > 2500 gm)	+ relationship with low birth weight and discrimination, not entirely independent of other variables related to institutional discrimination
Landrine & Klonoff, 2000	Report of two studies: Study 1: 153 African American adults in educational setting Study 2: 195 African American adults in public settings	Schedule of Racist Events [Landrine, 1995 #1608] 18 item includes frequency (lifetime and past year) of discrimination in salary, housing, by store clerks, called names. Also includes measure of respondent appraisal of stressfulness of event	Study 1: Self-reported smoking (yes/no) Study 2: Have you smoked at least 100 cigarettes in your entire lifetime? Do you smoke cigarettes now? Stress—Hopkins symptom Checklist, including measures of anxiety and depression	Increased smoking with high discrimination and more stress Smoking not associated with > discrimination, but smokers found discrimination more stressful
Liebkind et al, 2000	1146 multi-ethnic immigrants in Finland	18 items covering application for job, atmosphere at work, career advancement, dismissal from work, renting/buying home, racism in public places, racist violence, deterring entry, obtaining credit/loans, treatment by federal & municipal authorities, health care system and police	Stress—Hopkins symptom Checklist, including measures of anxiety and depression	Limited by low response rate (37.3%) to mailed questionnaire. Greater the perceived discrimination and racism, the less trust in authorities, and the greater stress. Of interest, groups experiencing the greatest discrimination reported the least stress symptoms. External attribution of negative outcomes to racism was hypothesized as protective of psychological well-being

Table 1. (Continued)

Study	Sample	Measure of Racism/Discrimination	Measure of Risk Factor/CVD Outcome	Findings/Discussion
Poston et al, 2001	91 African American and 86 African-born RNs or pharmacists living in Houston	McNeilly's Perceived Racism Scale	Blood pressure (mercury manometer using HDFFP guidelines)	No relationship
Stancil et al, 2000	Subset of first 94 African American women aged 18–39 enrolled in longitudinal study of pregnancy and exposure to environmental lead; Pittsburgh PA	See Kreiger, 1990. Ever experienced racism in major life domains and active/passive coping response	Stress—urinary cortisol adjusted for creatinine; Life Events Scale; Perceived Stress Scale, neighborhood stress Blood pressure (using ARIC protocol)	Younger age, higher income, lower education and experiences of racism were related to > perceived stress. High perceived stress score associated with elevated SBP @ wk 32–36; cortisol predictive of BP after week 36. Racial discrimination associated with > perceived stress, active coping with lower stress. > DBP reactivity in black women attributing unfair treatment to race
Guyll et al, 2001	SWAN study-Pittsburgh 363 midlife women; 101 African American, 262 European American	Williams Everyday Discrimination Scale including 10 items, attribution of cause (race, ethnicity, gender, age, income, language, physical appearance, sexual orientation, other) and frequency of mistreatment (sometimes, often, rarely, never) Interpersonal racism (tone, verbal abuse, physical attack) Institutional racism (belief British employers would refuse job for race)	Cardiovascular reactivity (SBP, DBP, HR) to mirror tracing (nonsocial stressor) and speech task (social stressor)	60% increased risk of HTN among those with + perception of institutional racism Positive association between interpersonal racism and CVD
Karlson & Nazroo, 2002	Fourth National Survey of Ethnic Minorities-United Kingdom. Nationally representative sample of 5196 people of Caribbean, South Asian and Chinese origin and a comparison sample of 2867 whites	Ethnic identity Interpersonal racism-Racial harassment (victim of racially motivated verbal or physical abuse to person or property attack in past 12 months) Institutional racism (belief British employers would refuse job for race)	Self-report diagnosed blood pressure, possible ischemic heart attack (severe chest pain, diagnosed heart attack, diagnosed angina)	Independent of social position, 60% increase in report of poor/fair health with + racial harassment. Independent of social position, 50–66% increase in report of fair/poor health with + perception of institutional racism. Ethnic identity—broad similarity in experience of racism across groups and does not appear to influence health. Rather ethnicity as structure (e.g. racialisation and class experience) is closely associated with health. African Americans had significantly higher composite stress scores, experienced significantly more Unfair Treatment, and were more likely to report Economic Hardship. African Americans had significantly higher average IMT, + linear relationship between stress and IMT in African Americans, but not Caucasians. Unfair Treatment showed significant + relationship for African Americans only .Biological mediators associated with insulin resistance account for substantial proportion of variance Suggestion that chronic stress may lead to increased disease risk through association with known CVD risk factors; however accumulated stress exerts independent effect as well.
Karlson & Nazroo, 2002	Fourth National Survey of Ethnic Minorities-United Kingdom. Nationally representative sample (see above description). Analysis for this study included data from 2507, including 591 Caribbean	Composite index of chronic stress (measures of life events, ongoing stressors, discrimination, economic hardship). Racism was measured by affirmative answer to at least one of 10 item from the Williams Everyday Discrimination Scale (Response categories included race, ethnicity, gender, age, income, language, physical appearance, sexual orientation, other)	Self-report of health status, heart disease (diagnosed angina, heart attack or severe chest pain), diagnosed diabetes, diagnosed hypertension	Subclinical carotid disease using carotid intima-medial thickness (IMT) measured by carotid ultrasound. Biological variables (fasting glucose, insulin, lipids, BP, BMI, smoking, physical activity)
Troxel et al, in press	109 African American and 225 Caucasian premenopausal women participating in Pittsburgh site of Study of Women's Health Across the Nation (SWAN)			

that perpetuate those historical injustices. . . [I]t is because of institutional racism that there is an association between socioeconomic status and health in this country.

Socioeconomic status (SES) and access to health care are posited as pathways through which institutionalized racism affects health. Williams and Williams-Morris,¹⁰ in their recent overview of experiences of racism and mental health, provide a dismal portrait of the process and consequences of institutional racism for African Americans. Driven by the dominant mechanism of continuing residential segregation, limited and unequal access to services and resources, education, and employment opportunities, the socioeconomic mobility of African Americans has been seriously truncated. Not only is there more poverty (African Americans are 3 times more likely to be poor, and more than half of black households are in an economically vulnerable category), but blacks are twice as likely to be unemployed. Furthermore, most socioeconomic indicators are not equivalent across race. A given level of education does not necessarily reflect equivalent skills and preparation. At each level of education, blacks earn substantially less than whites at comparable years of schooling.¹⁷ At a given level of income, the purchasing power is reduced for blacks,¹⁸ with significant racial differences in wealth at every level of income.¹⁹ Let us turn to the evidence regarding the relationship of socioeconomic differences, access, and neighborhood variables to CVD risk factors and outcomes.

Socioeconomic Differences and CVD

Differences in SES have a profound and well-documented impact on cardiovascular health outcomes. Whether measured by education, income, occupation, or other factors (neighborhood, indicators of wealth, occupational prestige), SES is among the most potent predictors of adverse changes in cardiovascular health. For nearly a decade, it has been argued that SES is an independent risk factor for CVD.²⁰ The lower the SES, the greater the risk of CVD. The moderating effects of SES on CVD are clearly seen in relation to ethnicity. SES accounts for much of the observed racial disparities in health, although racial differences often persist after statistical adjustment even at "equivalent" levels of SES. These findings suggest that there may be a very different experience of SES among ethnic groups; thus, even when statistical controls for SES "explain" differences, the processes by which these differences occur are not uniform among or within ethnic groups. More than a confounder, race is both an antecedent and determinant of SES that reflects, in part, successful implementation of racist policies. Williams¹ provides an extensive review of the ways in which race, racism, and SES affect health outcomes. That these differences may persist from birth

is suggested by cumulating work in childhood and life course SES.^{21,22} At least 1 study has linked racism and low birth weight,²³ an increasingly recognized correlate for adult CVD.²⁴ The reader is directed to comprehensive reviews of SES-health-CVD findings offered by several authors for a more complete discussion^{18,25,26} of the inverse relationship of SES and CVD outcomes and risk factors.

Of particular interest to the JHS, data from the ARIC study confirmed multiple risk factor differences between blacks and whites. Investigators found striking differences in obesity between black and white women (66% for blacks versus 35% for whites); higher fasting glucose levels and greater prevalence of diabetes in black women and men; and higher blood pressure in black participants, with poorer rates of control in those with hypertension and a greater propensity for clustering of multiple risk factors.²⁷ SES differentials were imputed as possible explanations for some of these differences; however, no comparisons were made with family history. The hypothesis that higher risk factor prevalence in ethnic minorities would be attenuated by adjusting for SES was not supported in analyses of the National Health and Nutrition Survey (NHANES) III²⁸ and several smaller studies.^{29,30} These findings lead some to speculate that genetic predisposition plays a greater role than psychosocial or environmental factors. Low-income African American women participating in focus group discussions, however, attribute much of their increased CVD risk to the effects of low SES and stress, including the stress of racism.³¹ Few studies have focused on racism-related stress and CVD risk factors. Relationships have been found between racism-related stress and cigarette smoking,³² alcohol consumption,³³ substance abuse,³⁴ and eating problems³⁵ (see Table 1). Additional data is needed to determine whether racism is related to CVD risk factors, although such relationships are conceptually plausible within the stress process, where we might hypothesize an increased proclivity to negative health behaviors with excess chronic stress. More research is needed to adequately elucidate the specific psychological, physiological, and biochemical mechanisms by which low SES gives rise to stressors and other risk factors that compromise the health of African Americans. Studies of the additive and interactive effects of SES with a variety of physiological, psychological, behavioral, and demographic variables, including ethnicity, are needed.

Differential Access to Health Care Resources

The existence of ethnic and racial disparities in access to care and health outcomes has been the subject of research and discussions within the health services literature for more than 30 years.³⁶ Cumulative research evidence demonstrates that persons who are members of ethnic and racial minority groups are more likely to experience barriers to receiving health care and are less likely to have

health insurance. This disparity is strongest and most consistent in studies of cardiovascular care. The recently released report of the Institute of Medicine's Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care³⁷ concluded that ethnic and racial disparities in the provision of cardiac care persist even after adjustment for confounding factors such as disease severity, disease prevalence, comorbidity, clinical characteristics, refusal rates, SES, site of care, and overuse of services by whites.

SES greatly influences access to health services. Persons who are poor, are unemployed, have less education, or live in rural or inner-city communities are more likely to experience difficulties obtaining and/or affording needed care, to report that they have gone without needed care, and to have not visited a health care provider in the past 2 years.³⁸⁻⁴¹ Because African Americans are disproportionately represented among persons of low SES, they are also more likely to experience problems accessing the health care system.

Health insurance provides individuals and families with a means of reducing the financial risk and burden associated with financing health care.⁴² Racial and ethnic disparities in health insurance status can explain some but not all of the racial disparities in access to health care. Compared with whites, African Americans and Hispanics are more likely to report having no insurance or to be covered by public insurance programs such as Medicaid or Medicare.⁴³ Researchers have consistently found that persons who have private insurance also have access to a wider range and volume of health care services.^{39,40,44} The same discriminatory factors that contribute to socioeconomic disparities also influence the types of health insurance that are available to ethnic and racial minorities. Most Americans obtain private health insurance through employer-based health insurance programs as a benefit of employment⁴⁵; therefore, factors that influence the likelihood of being employed and the type of jobs held also influence the availability of private health insurance. These same factors subsequently influence the type and amount of health services that ethnic minorities receive.⁴⁶

Considering the increased incidence and prevalence of CVD in African Americans, one would expect to see a corresponding increase in the use of cardiac care compared with European Americans. However, blacks are less likely to receive drug therapy, diagnostic angiography and catheterization, thrombolytic therapy, percutaneous transluminal coronary angioplasty, coronary artery bypass graft surgery, or carotid endarterectomy than whites who have similar clinical disease.^{37,38,47} These disparities in the utilization of cardiac care are diminished among the privately insured compared with the uninsured and Medicaid populations.³⁸ In addition,

differentials in care are significantly diminished, but not eliminated, in various situations and populations in which access to care is assumed to be equitably distributed, such as among Medicare recipients, military veterans receiving care in Department of Veterans Affairs hospitals, and persons whose care is covered by nationalized health plans (eg, Canada and Great Britain). Furthermore, less disparity is observed when care is received in hospitals that provide a full range of cardiac services compared with hospitals that do not provide these services.³⁷

It is apparent that differentials in access to care exist. Reasons behind these differentials are not as easy to identify. The persistence of racial and ethnic disparities in the use of care after controlling for socioeconomic and clinical factors is often attributed to discrimination. The American health care system is still struggling to overcome the legacy of racial segregation in which racial and ethnic minority groups were "denied all but the most basic level of care other than whatever was available in a segregated facility."⁴⁸ Biases on the part of health providers have been shown to adversely affect their decisions to offer cardiovascular services to ethnic minorities and women.^{41,49,50} Legal challenges aimed at overt health care discrimination have produced changes in admission procedures, ambulance routes, staff privileges rules, and the elimination of preadmission deposit and prepayment requirements. However, cases involving more covert discriminatory practices, such as the closure or relocation of hospital and transfer of services away from minority communities and into more economically generous markets, have not been successful⁴⁸

Neighborhood/Living Conditions and CVD

Racial segregation and economic isolation are the structural manifestations of racism most often affecting the daily lives of African Americans. It is highly likely that residence in the segregated, economically impoverished neighborhoods created by institutionalized racism adversely affects cardiovascular health outcomes.⁴⁹ Suggested pathways include absolute deprivation of resources and facilities, differential exposure to health risks in the physical environment because of economically disadvantaged neighborhoods and poor quality housing, higher costs of goods and services in deprived areas, as well as the mediating roles of social networks and social capital.^{51,52} The effects of patterned sets of exposures, opportunities, and resources, creating varying levels of neighborhood social disorganization and social obligations necessary for survival, take a disproportionate toll in poor health and early death of African Americans. The cumulative burden of social stress potentially accelerates the decline in heart disease mortality. The degree of disadvantage is further accelerated among black

women in neighborhoods with a high density of households headed by women.⁵³ Environmental impoverishment was hypothesized to account for much of the 50% excess risk of age-adjusted all-cause mortality among African Americans living in Harlem.⁵⁴

Ecological research reveals that several characteristics of these neighborhoods can have a negative effect on the functioning of adults and children. Important characteristics may include high levels of population turnover, crime, violence, fear of crime, noise, and crowding, not to mention the absence of goods and services, including parks and recreational/exercise facilities.⁵⁵ LaVeist and Wallace⁵⁶ found the distribution of liquor stores to favor low SES (SES) and African American communities with disproportionate alcohol-related health problems in these neighborhoods. Neighborhood characteristics have generally been found to relate to health outcomes independent of individual indicators of SES.⁵⁷

Since the early 1970s, the importance of the 'socioecological niche,' or combinations of gender, race, and residence that reflect social stress in a neighborhood, has been related to blood pressure. Harburg and others^{58,59} found that blacks in high socioecological stress areas had significantly elevated blood pressures. Neighborhood characteristics have also been related to congestive heart disease (CHD) mortality, although most studies could not determine whether the relationships held after controlling for individual level variables. Wing and colleagues⁶⁰ and Tyroler et al⁶¹ show the predictive relationship between changes in neighborhood characteristics (eg, economic profile, average education) and ischemic heart disease mortality. Relationships between neighborhood environments and various risk factors for coronary heart disease (CHD), including blood pressure, smoking habits, and diet, have been identified by several studies.⁶²⁻⁶⁴ At least 1 study has found little evidence of area effects on smoking behavior.⁶⁵ There is, however, some suggestion of independence of individual and neighborhood indicators.^{66,67}

Diez-Roux et al⁶⁸ conducted a multilevel analysis of the prevalence of CHD and several major CHD risk factors: blood pressure, smoking, and systolic blood pressure to ascertain the relative contributions of neighborhood and individual variables. Individual level data on social class indicators, CHD prevalence, and risk factors were obtained from the baseline visit (1987-1989) of the Atherosclerosis Risk in Community (ARIC) study; neighborhood (census block) data on education, income, house value, and occupation were obtained from the 1990 Census. ARIC data were collected from 4 US communities (Forsyth County, NC; Jackson, MS; northwest suburbs of Minneapolis, MN; and Washington County, MD) with the Jackson, MS, site providing an all-African American sample. Findings of this

study confirm that neighborhood context, independent of individual level variables, may be important in shaping CHD prevalence and risk factors. In particular, increased neighborhood disadvantage was associated with increased prevalence of risk factors and increasing CHD odds after adjustment for individual variables. Patterns were somewhat different in Jackson, with decreasing prevalence of CHD in the poorest neighborhoods, whereas associations for cholesterol were more notable at the middle 50% of the distribution of neighborhood characteristics, especially for men. This pattern suggests parallels between the distribution of CHD in more "rich" and "poor" countries and "developing" countries,^{69,70} where the shift in social class distribution of CHD may have occurred later in African Americans than European Americans, especially in the southeastern United States. Notably, these same patterns did not hold for the small sample of African Americans in Forsyth County, North Carolina, where the neighborhood indicators were slightly more favorable than those in Jackson. Thus, neighborhood context may be 1 of many links between social structure and CHD; further study is needed to clarify differing patterns of linkage among ethnic groups.

Institutional discrimination takes its toll through subtle mechanisms that are hard to recognize and disentangle. Kaplan,⁷¹ for example, notes that considerably more work is needed to understand the variable characteristics of where people live. He notes that "progress will require more data collection on the daily experiences of individuals, on the material and symbolic demands that challenge them, on the personal and community resources available to meet these challenges and on the macroeconomic forces that affect both the individual and the community." In-depth interviews by Essed⁷² and Cattel⁵² with selected communities provide insight on how African American women actively implement a variety of strategies to deal with the multiple vulnerabilities of gender and racial discrimination. These strategies were revealed as both a source of social capital to buffer its harsh effects or increased stress from excess obligations contributing to demoralization or heightened vigilance. Thus the negative aspects of social ties may be more strongly linked to health outcomes than are the positive, supportive ones.⁴ For example, hopelessness has been linked with heart disease.⁷³ Likewise, responses to the pressure of racism and environmental stressors may show up in persistent elevations of blood pressure during sleep. Several studies have identified that African Americans maintain a higher level of nocturnal blood pressure than do European Americans.^{74,75} Further research is needed to specify the mechanisms by which the various manifestations of institutionalized racism "gets under the skin,"⁷⁶ creating a "biology of inequality"⁷⁷ or allo-

static load⁷⁸ that enhances CVD morbidity and mortality.

Perceived/Personally Mediated Racism and CVD

Jones⁸ defines *personally mediated racism* as the interpersonal experience of prejudice and discrimination. This aspect of discrimination is what comes to mind for most persons when they hear the word "racism." Measured at the individual level, prejudice involves negative attitudes and beliefs about the motivations, abilities, and intentions toward racial outgroups, whereas discrimination is a behavioral manifestation of that prejudice.⁷⁹ As Krieger⁸⁰ emphasizes, "interpersonal discrimination refers to directly perceived discriminatory interactions between individuals. . . . In all cases perpetrators of discrimination act unfairly toward members of socially defined subordinate groups to reinforce relations of dominance and subordination, thereby bolstering privileges conferred to them as members of a dominant group." Personally mediated racism is perceived in the subjective experience of prejudice or discrimination.⁸¹ Important to this argument is the assumption that perceptions of racism do not have to be objectively "real" to affect health outcomes. As such, both the short- and long-term experiences of everyday racism⁷² provide a potential source of stress, becoming a part of the daily fabric of life for most African Americans. Nearly one half of non-Hispanic black respondents to a recent national survey reported experiencing at least 1 episode of major discrimination whereas more than 70% reported the occurrence of day-to-day perceived discrimination either often or sometimes. Nearly 90% reported race as the most common reason for discrimination.⁸²

Jones⁸ further posits that the stresses of everyday racism provide the pathway by which personally mediated racism impacts health. Clark and colleagues⁸¹ synthesized a biopsychosocial model for perceived racism as a guide for future research. This model postulates that personally mediated racism behaves as an environmental stimulus perceived as a stressor, triggering exaggerated psychological and physiological responses that are influenced by constitutional, sociodemographic, psychological, and behavioral factors. These responses are hypothesized to act much as the "fight or flight" response to mobilize lipids and glucose to increase energy supplies and sensory vigilance, produce transient elevations in blood pressure, and trigger those pathways, leading to sustained hypertension. The physiological responses after exposure to stress most notably involve immune, neuroendocrine, and cardiovascular functioning, including vascular reactivity and endothelial function. Likewise, numerous psychological responses, such as denial, anger, hostility, hopelessness, depression, fear, and so forth, may follow perceptions of racial stressors. These responses may, in

turn, invoke differential coping responses that may help explain the 2-decade-old call to consider the effects of racism as a possible explanation for why the differential exposure to stress impacts health in lower SES and African American groups.⁸³

Anderson and colleagues⁸⁴ conducted a comprehensive review of autonomic reactivity and blood pressure in blacks as a clarion call to laboratory and field research to test the relevance of the stress model. The following section will review the evidence to date, examining progress toward linking racism and psychophysiological mediators, coping, and CVD risk factors and outcomes. The majority of the available evidence addresses blood pressure. Hypertension is considered a major health problem of African Americans⁸⁵ and accounts for 40 to 60% of the coronary heart disease mortality risk,⁸⁶ highlighting the importance of this evidence in subsequent CVD outcomes.

Racism and Physiological Functioning

Anderson and colleagues⁸⁴ show that cardiovascular changes, as well as kidney function and neurochemical mechanisms involved in blood pressure and endothelial function, are affected by long-term stress, including the stress of racism.

Cardiovascular Reactivity. There is considerable evidence that autonomic reactivity is correlated with elevations in blood pressure. Situations of long-term elevated stress are thought to potentiate heightened cardiovascular reactivity,⁸⁷ potentially with an increased likelihood of atherosclerotic disease. African Americans have generally shown greater cardiovascular reactivity at rest or to a variety of standard laboratory stressors (eg, cold pressor, arithmetic) compared with European Americans,^{84,88} although these responses are not universal. Although family history of hypertension was important in predicting vascular reactivity for whites, no similar relationship has been found in black adults. Blacks exhibited greater α -adrenergic patterned constriction of the peripheral vascular bed, leading to greater increases in blood pressure than whites.

Although these initial studies shed light on the differences in reactivity between blacks and whites, they did not provide information about the generalizability of reactivity to real-life situations of racial stress. A second wave of research addressed the issue of racism and reactivity, mostly in all-African American samples, using analogues of racist events in the laboratory. In general, this line of research confirmed increased cardiovascular reactivity with exposure to racist versus neutral events.⁸⁸⁻⁹⁰ One recent study using a biracial sample found that both black and white men demonstrated increased reactivity to both anger-provoking and racist events compared with neutral scenarios.⁹¹ Unlike previous studies, the authors found no increased reactivity to racial events in African Americans. However, the

authors conclude that even if the response to individual events does not differ by race, the cumulative burden of events does differ for African Americans, exposing them to a higher load of reaction-producing events.

A third body of research has addressed the limitation of assuming reactivity as a stable trait without considering the ways in which individual predispositions and varying social situations interact to produce heightened reactivity. This research has examined the interactive effects of a complex array of undesirable social situations, personality factors, or potentially buffering psychosocial factors such as SES, anger, Afrocentrism, John Henryism (self-perception of environmental mastery through hard work and determination), and social support with cardiovascular reactivity. Some studies also included the combined assessment of perceptions of actual racism experiences with laboratory reactivity. In general, the direction of this research is as expected. That is, low SES and hyperreactivity interact to increase the 4-year progression of atherosclerosis⁹²; anger and hostility were positively associated with higher reactivity⁹³; and higher Afrocentrism scores were associated with more negative reactions to racist events.⁸⁹ Combining perceptions of real-life racism with laboratory stress reveals a positive association between reactivity and higher perceived racism scores in African Americans but not European Americans. Several studies suggest the type of reactivity is predictive of future hypertension.^{94,95} A recent study of African American and European American women specifically links subtle but not blatant mistreatment attributed to racism with increased diastolic blood pressure reactivity in the African American women.⁹⁶

Taken together, the bulk of cardiovascular reactivity research provides indirect support for the association between the exposure to racism, heightened vascular reactivity, and subsequent long-term elevation of blood pressure, especially in persons with certain predispositions and exposed to stressful situations.⁹⁷ One recent study found a positive association between long-term everyday discrimination and subclinical CVD with increased carotid intimal/medial wall thickness for black but not white women.⁹⁸

Sodium Excretion. One of the major ways the body regulates blood volume is by varying rates of sodium excretion. Sympathetic nervous system activation of the kidney during long-term stress can lead to reductions in sodium excretion, thus contributing to blood pressure elevation through the associated volume overload. A significant body of evidence suggests that African Americans excrete less sodium than do European Americans and are more salt-sensitive.⁹⁹ High levels of sodium are known to potentiate the release and action of norepinephrine, further contributing to constriction of peripheral

arteries and subsequent elevated blood pressure.⁸⁴ The effects of stress on sodium excretion are well documented in animals¹⁰⁰ and humans. This work is nicely summarized by Light,¹⁰¹ demonstrating, for example, that stress induces sodium retention in the children of black hypertensive parents.¹⁰²

Low Renin Levels. Of interest, blacks have lower renin levels apparent at an early age¹⁰³ associated with higher blood pressure levels. The classic form of low-renin hypertension is primary aldosteronism with excess aldosterone suppressing renin and contributing to sodium retention and hypokalemia. Notably, autopsy,¹⁰⁴ and surgery specimen¹⁰⁶ studies have shown an increased prevalence of adrenal hyperplasia in African Americans. These findings led some to postulate a “subtle” form of aldosteronism based on observations that despite reduced renin levels, essentially normal levels of aldosterone production are maintained. Mechanisms are unclear; however, animal studies have shown that psychosocial stress leads to adrenal hyperplasia and can be associated with low-renin hypertension.¹⁰⁷ Recent ARIC data suggests that African Americans do have a lower potassium level¹⁰⁸; however, no linkages between hypokalemia and social stressors, including racism, have been attempted.

Neurochemical. In addition to the actions of the sympathetic-adrenal system imputed in cardiovascular reactivity discussed above, myriad neurochemical hormones are carried throughout the body over a more prolonged period by activation of the hypothalamic-pituitary-adrenal system. Triggering the release of adrenal corticotrophic hormone and cortisol, immune function is inhibited, preventing the body from destroying pathogens and increasing vulnerability to an array of negative health outcomes, resulting in increased likelihood of infection. Both animal¹⁰⁹ and human studies¹¹⁰ have found an association with infection (*Chlamydia pneumoniae*, *Helicobacter pylori*), elevated C-reactive protein levels,¹¹¹ and atherosclerosis, leading to the hypothesis that the body’s inflammatory response may play an important role in the development and progression of atherosclerosis.¹¹²

Thus far, the leading biological exegesis offered to explain how exposure to long-term stress, including racism, may “get under the skin”⁷⁶ includes cardiovascular reactivity with alteration in autonomic tone as manifested by heart rate variability and changes in the sympathetic nervous system, the corticotropin-cortisol axis, and vascular reactivity, particularly endothelial function. Alteration in blood pressure is a common manifestation.

Blood Pressure. Twelve studies have focused on racism and blood pressure as the CVD outcome. Findings have been mixed and complex^{70,96,113–121} (Table 1). Initial studies^{114,122} indicated a positive relationship between blood pressure and discrimination, whereas the findings of subsequent studies^{70,117} were more equivocal. Discrimination was

unrelated to hypertension in 2 studies.^{116,119} Important to the relationship between racism and blood pressure is the way the person responds to or copes with the discrimination experienced.^{113,114} For example, blood pressure was higher in those who reported acceptance of unfair treatment and no experience of discrimination, whereas the blood pressures of those who respond to unfair treatment with action are comparatively lower. This finding was associated with gender and class differences¹¹⁷ and is in keeping with a body of evidence that persons who experience discrimination are more likely to attribute racist experience to their group, but not to themselves, perhaps finding it painful to admit personal experience. Thus, internalized responses to racism may play an important role in the development of hypertension.

Also important to understanding the relationship between discrimination and CVD outcomes is the period of exposure to an event(s) in relation to the etiologic period for a given outcome. Most studies to date have employed short-term measures of racism without reference to the time of disease diagnosis. In these studies, the onset of the outcome variable (hypertension) may have occurred before the period in which the measured exposure to discrimination occurred, contributing to a negative relationship. Some evidence for the importance of attending to the cumulative impact of living in a racialized society is provided by more recent national data from the United Kingdom. Karlsen and Nazroo^{120,121} found a positive association between perceived institutional discrimination, blood pressure, and risk of CVD independent of socioeconomic position. Their findings speak to deleterious CVD effects when discrimination is viewed as pervasive and stable; eg, the effects of living in a racist society.¹²³ Taken together, the studies on racism and blood pressure suggest that both individual and institutional factors influence variations in blood pressure and lend credence to Krieger and Sidney's¹¹⁷ conclusion that "racial discrimination shapes patterns of blood pressure among the US black population and black-white differences in blood pressure." Our current limited understanding of the way in which discrimination shapes blood pressure responses and other CVD outcomes is a function of both the limited conceptualization and measurement of racism and the absence of needed models that link exposure to the stress of discrimination with the development of hypertension using a plausible window of exposure.

Racism, and Psychological/Behavioral Functioning: Predispositions, Appraisal and Coping Responses

There is a long history of studying psychosocial factors and stress responses in disease causation.

Mental health correlates of racism are the most studied health outcome.^{10,80} Both psychosocial predispositions or personality traits as well as stress appraisal and coping processes are considered important in mediating the potential effects of racism on CVD outcomes. In the 1960s, Grier and Cobbs¹²⁴ noted suspiciousness, anger, and distrust as adaptive responses to racism among African Americans. A wide range of psychological and behavioral factors is postulated to affect the stress process, cardiovascular functioning, and the immune process. Among them are type A behavior, cynical hostility, neuroticism, self-esteem, hardiness, perceived control, and anger expression-suppression.¹²⁵⁻¹²⁸

Appraising a stressful experience as racist or not is a subjective process that can serve as an active coping response or a predisposition to vulnerability. Heightened environmental vigilance and surveillance among African Americans is a legacy of the practices of oppression enacted over the decades of racialized mistreatment in this country. Listen as a participant in an in-depth interview regarding potential research participation¹²⁹ describes the safekeeping practice of vigilance: "It's a lot. . . see I'm an open-minded person, but at the same time I'm open-minded, I'm also guarded. Yes ma'am. I'm guarded because I've learned to live my life that way. I didn't grow up with the-the house and the picket fence and the dog."

Being guarded or on guard keeps a person safe. It's a kind of listening with one's whole being, a speaking that interprets the intentions of the speaker and gives back what I think you want to hear, and as such keeps me at a distance and assures my safety. The danger lies in seeing even the most innocent, trivial comments or gestures as profoundly racial⁸¹ placing one at risk for increased reactivity and hypertension. On the other hand, denial of racism as a source of stress may also increase hypertension. Taylor and colleagues¹³⁰ have distinguished between personal and group experiences of racism, noting strong evidence that African Americans perceive racism toward their ethnic group far more commonly than they identify individual experiences of racism.¹³¹ This differential appraisal of group-individual racism is hypothesized as serving a protective role, thus minimizing the distress of acknowledging and coping with racism¹³² while maintaining a sense of personal control. Krieger¹¹⁴ found that African American women who denied or reported no experiences of racism were almost 3 times as likely to report hypertension than those who reported 1 or more such experiences. She noted that those with lower SES were more likely to underreport or deny experiences with racism, especially in situations of attributional ambiguity,¹²³ increasing their vulnerability to CVD sequelae. Irrespective of the ultimate appraisal in a given

situation, it is clear that African Americans spend considerable emotional and cognitive energy on racism^{133,134} and the continual replaying of a situation can be stressful again, above and beyond the actual experience.¹³⁵

Some African Americans may choose denial and self-degradation, others may embrace vigilance and resistance, and others may accept the status quo in the face of racist experiences. Prior research suggests that the nature and type of response to racism is a key determinant of the physiological impact of discrimination. Few studies have examined the relationship between racism-specific coping responses and health outcomes, particularly CVD outcomes. Kreiger,¹¹⁷ McNeilly,¹³⁶ and Harrell¹⁴ have recently conceptualized a broad range of coping behavior typologies and developed methods for measuring them. These typologies generally include behavior on 3 continuums: active/passive, individual/collective, and inner-directed/outer-directed. In general, the more passive or internalized the coping style, the higher the blood pressure.^{114,117,137}

Internalized Racism and CVD

Another significant manifestation of racism is its attack on the ego integrity of its victims. *Internalized racism* results from acceptance of cultural stereotypes of inferiority by a marginalized social group.¹⁶ Negative images of blacks are pervasive in American culture, with blatant and subtle racist stereotypes in common use.¹³⁸ Internalization of negative cultural stereotypes attacks individual self-worth and ethnic identity, undermining the importance of their very existence. In addition to the adverse affect the stigma of inferiority plays in the medical treatment of black patients (see prior discussion), potential relationships with depression or other negative emotions (anger, hostility) and engaging in risky health behaviors are the most likely pathway through which internalized racism may affect CVD.

Internalization of negative stereotypes has been positively related to psychological distress,¹³⁹ lower self-esteem and ego integrity,¹⁴⁰ and depression.¹⁴¹ Linked with excessive glucocorticoid production¹⁴² and high plasma norepinephrine levels,¹⁴³ depression predicts future coronary heart disease.¹⁴⁴ Furthermore, both the NHANES^{145,146} and the Coronary Artery Risk Development in Young Adults¹⁴⁷ study found a greater impact of depression on cardiovascular outcomes in African Americans. When anger is used as a coping response to discriminatory laboratory events, the magnitude of cardiovascular reactivity is greater.^{81,148} Moreover, hostility in response to racial stressors has been associated with higher blood pressure levels but only during the recovery period. Such physiological response during

long recovery is potentially indicative of allostatic load, or “daily wear and tear,”⁷⁸ and its increased risk of CVD.⁹⁵

Direct evidence relating internalized racism, risky health behaviors, and CVD outcomes are sparse. Most studies conducted to date employ an intermediate measure of acculturation or racial identity. Using a measure of the extent to which blacks feel uncomfortable around other blacks and believe in their innate inferiority, Taylor and Jackson¹³⁰ found a positive association between internalized racism and alcohol consumption. Klonoff and Landrine¹⁴⁹ found that, irrespective of social class and education, African American smokers tend to have a more ‘traditional’ racial identity, having little contact with whites, holding a variety of superstitious health beliefs, and a tendency to consume an unhealthy diet. Although tentative, they suggest that the stress of discrimination may be positively related to smoking behavior. Tull and colleagues¹⁵⁰ found that internalized racism was related to increased risk of obesity and abdominal adiposity among Afro-Caribbean women. A study of urban African American adults found that healthy lifestyle practices (eg, lower fat diet, more leisure time activity, not smoking, and, in women, moderate alcohol consumption) were positively associated with an expressed identification with the African American culture and self-assessed success in moving between black and white ways of life.¹⁵¹ The authors emphasized the need for future studies with methodological approaches that differentiate positive affirmation of African American culture from negative experiences associated with discrimination and social disadvantage. Two recent studies have explored the relationship between racial identity and blood pressure. The data of Torres and Bowen¹⁵² on college students suggest that as racial identity accumulates, systolic blood pressure increases. A more ‘transitional’ (bicultural) racial identity was found to be associated with higher resting diastolic blood pressure and ambulatory systolic and diastolic blood pressure in a sample of normotensive and minimally treated hypertensive adults.¹⁵³ Both studies suggest the need for additional research to unravel the mechanisms by which racial identity attitudes may increase hypertensive risk.

Opportunities for the Jackson Heart Study

In the final analysis, although the evidence is compelling and growing that racism is pathogenic, we know far too little about why some African Americans are affected by racism and others fare far better. Early work focused heavily on the dysfunctional psychosocial and behavioral aspects of racism’s influence. There may be many things inherent in the culture and traditions of African Americans that may counteract the negative effects of long-

term stress. Although not covered in this overview, there is ample evidence of the role of social support, extended family network, and religion in buffering general stress responses. For example, it has been shown among whites that regular church attendance is associated with lower resting blood pressure.¹⁵⁴ It seems reasonable that such factors could buffer the CVD response to racism as well. Full understanding of the impact of racism on cardiovascular health requires similar attention to the protective and functional aspects of responding to racism.⁷⁸ As Krieger⁸⁰ so cogently notes, “. . .the epidemiology of health consequences of discrimination is, at heart, the investigation of intimate connections between our social and biological existence. . . [and]. . .we accordingly must draw on not only a nuanced understanding of the likely biological pathways of embodying discrimination, from conception to death, but also a finely tuned, historical, social, and political sensibility, situating both the people we study and ourselves in the larger context of our times.”

Although studies of the indirect measures of institutional discrimination (SES, neighborhood, health care access) provide powerful evidence of the role social institutions and policies play in shaping the disparate CVD outcomes for African Americans, they are indeed indirect measures and do not yet provide sufficient data for imputing mechanisms of embodiment. Likewise, studies employing direct measures of personally mediated and internalized racism are limited by the lack of comprehensive, well-validated measures and the subjective nature of self-report, including the likely under-reporting of experiences of discrimination, attributional ambiguity, and self-blame. Accordingly, the findings, although suggestive of a positive relationship between discrimination and CVD risk factors and outcomes, are neither consistent nor clear.

There is a pressing need to identify intervening and interacting processes and structures that link racialized social structures and personally mediated and internalized experiences of racism to cardiovascular and other health outcomes. The experience and outcome of racism is not monotonic. As Pierce¹³⁵ pointed out, we know too little about why some people are devastated by racism and others are not. We need to illuminate pathways to health as well as disorder to fully understand the impact of racism on CVD. Qualitative inquiry into daily practices of managing racism and health can uncover ways in which families transmit patterns of dealing with the stress of racism across generational lines. Conceptual models of the biopsychosocial factors have been advanced that suggest proximal mechanisms through which societal factors and personal experiences with racism affect health.^{10,81,155} Adequate testing of these models requires a longitudinal data

set that includes a range of physiologic (eg, neurochemical, autonomic reactivity, renal, CVD risk factors and outcomes), behavioral (eg, health behaviors and risk factors, negative emotions, coping resources such as religion and social support), and social (eg, multilevel SES, geocontextual factors) measures as well as measures of the various levels of racism and other forms of oppression (eg, multiple discriminatory bind of racism, sexism, ageism, classism)¹⁴ to allow full elucidation of the proposed biologic and behavioral mechanisms by which discrimination may “get under the skin.”

Measurement of experiences of discrimination remains a major challenge to elucidating the relationship between racism and CVD. As outlined in this article, discrimination is a multidimensional construct with institutional, individual, and internalized levels. Clearly articulated measures are needed that conceptualize discrimination for each level that elicit a saturated expression of the experience. Population-level measures providing information regarding the often invisible patterns of institutional discrimination, in combination with individual-level measures of exposures and responses to interpersonal discrimination (perceived/personally mediated discrimination), including internalization of racial stereotypes and beliefs (internalized discrimination), are needed to fully understand the relationships. The development of both population- and individual-level measures is in its infancy; however, there is increasing clarity concerning the parameters for adequate assessment of the full impact of racism. For detailed information on the issues and current status of measurement at each level, readers are referred to an excellent review by Krieger.⁸⁰ Despite their psychometric limitations,¹⁵⁶ taken together, these measures provide the basis for rigorous scientific evaluation of the relationship between racism and CVD.

The JHS is poised to further the state of the science on racism and CVD. The overall and socio-cultural design for the JHS is described in a published abstract¹⁵⁸ and a forthcoming article by the JHS investigators. Data in collection for the baseline examination addresses institutional and perceived/personally mediated racism. Measures of SES (individual, family, neighborhood/community, wealth, self-assessment), access to care, and diagnostic and treatment procedures are included and will be supplemented with contextual analyses using census-based geo-coding of the data set. Perceived/personally mediated racism includes measures of major episodic and everyday minor experiences of racism in multiple life domains incorporating assessment of most recent and life-time exposure, attribution of source, and active versus passive response. Measures of internalized racism are slated for the second exam cycle. Combining far-reaching measures of stress (eg, global long-term, major life events, minor

life events), psychosocial vulnerabilities and resources, and extensive multilevel assessment of SES, coupled with broad-based medical history and physical assessment, including 24-hour blood pressure monitoring and urine measurement, the JHS will provide the most robust data base currently available to tease out the complex additive and interactive relationships that are likely to account for the relationship of various dimensions of racism and CVD health outcomes in African Americans.

Conclusion

According to Lenfant,¹⁵⁹ the scarcity of comprehensive data on heart disease in blacks limits attempts to further explain the disparities in disease risk factors and outcomes. We do not yet know to what degree racial and ethnic inequalities in health and health care are caused and sustained by racism. We are similarly unaware of how multiple aspects of racism may combine in additive and interactive ways with other psychosocial risk factors and resources to affect CVD risk. Despite hypothesized links between perceptions of racism, perceived discrimination, and CVD, few population-based studies have examined these associations. Findings, though inconsistent, do suggest the potential for such a relationship, particularly with hypertension. The evidence reviewed suggests the compelling need for greater acknowledgment and further study of racism and its effects on CVD risk factors and outcomes. Attention to the nature of racism as well as the possible mediating and moderating factors contributing to the link between racism and CVD is needed. Promising directions for such research were identified, including the need for longitudinal prospective study to enhance our understanding of the temporal and causal relationships. The JHS, 1 of the largest epidemiological cohort studies of the risk factors and causes of CVD in African Americans, offers a unique opportunity for examining the association between racism, perceived discrimination, and CVD outcomes over time.

References

1. **Williams DR.** Race, socioeconomic status, and health: the added effects of racism and discrimination. *Ann NY Acad Sci* 1999;896:173–88.
2. **Jones D, Sempos C, Thom T, et al.** Rising levels of cardiovascular mortality in Mississippi, 1979–1995. *Am J Med Sci* 2000;319:131–7.
3. **Sempos CT, Bild DE, Manolio TA.** Overview of the Jackson Heart Study: a study of cardiovascular diseases in African American men and women. *Am J Med Sci* 1999;317:142–6.
4. **Williams DR.** Race and health: basic questions, emerging directions. *Ann Epidemiol* 1997;7:322–33.
5. **Navarro V.** Race or class versus race and class: mortality differentials in the United States. *Lancet* 1990;36:1238–40.
6. **Krieger N.** Shades of difference: theoretical underpinnings of the medical controversy on black/white differences in the United States, 1830–1870. *Int J Health Serv* 1987;17:259–78.
7. **Lin S, Kelsey J.** Use of race and ethnicity in epidemiologic research: concepts, methodologic issues, and suggestions for research. *Epidemiol Rev* 2000;22:187–202.
8. **Jones CP.** Invited commentary: “race,” racism, and the practice of epidemiology. *Am J Epidemiol* 2001;154:299–304.
9. **Schuman H, Steeh C, Bobo L, et al.** Racial attitudes in America: trends and interpretations. Cambridge (MA): Harvard University Press; 1997.
10. **Williams D, Williams-Morris R.** Racism and mental health: the African American experience. *Ethn Health* 2000;5:243–69.
11. **Forman TA, Williams DR, Jackson JS.** Race, place and discrimination. *Perspect Soc Prob* 1997;9:231–61.
12. **Bonilla-Silva E.** Rethinking racism: toward a structural interpretation. *Am Sociol Rev* 1996;62:465–80.
13. **Lott B, Maluso D.** Introduction: framing the questions. In: Lott B, Maluso D, editors. *The social psychology of interpersonal discrimination*. New York: Guilford Press; 1995. p. 1–11.
14. **Harrell SP.** A multidimensional conceptualization of racism-related stress: implications for the well-being of people of color. *Am J Orthopsychiatry* 2000;70:42–57.
15. **King G.** Institutional racism and the medical/health complex: a conceptual analysis. *Ethn Dis* 1996;6:30–46.
16. **Jones CP.** Levels of racism: a theoretic framework and a gardener’s tale. *Am J Public Health* 2000;90:1212–5.
17. **Pamuk E, Makuc D, Heck K, et al.** Socioeconomic status and health chartbook. Health, United States, 1998. Hyattsville (MD): National Center for Health Statistics; 1998.
18. **Williams DR, Collins C.** US Socioeconomic and racial differences in health: patterns and explanations. *Annu Rev Sociol* 1995;21:349–86.
19. **Eller T.** Household wealth and asset ownership: 1991. Current population report P70-34. Washington DC: US Bureau of the Census; 1994.
20. **Kaplan GA.** Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* 1993;88:1973–98.
21. **Kaplan GA, Salonin JT.** Socioeconomic conditions in childhood and ischemic heart disease during middle age. *BMJ* 1990;301:1121–3.
22. **Barker DJ, Martyn CN.** The maternal and fetal origins of cardiovascular disease. *J Epidemiol Community Health* 1992;46:8–11.
23. **Collins JW, David RJ, Symons R, et al.** Low income African-Americans mothers’ perception of exposure to racial discrimination and infant birth weight. *Epidemiology* 2000;11:242–8.
24. **Fuller KE.** Low birth-weight infants: the continuing ethnic disparity and the interaction of biology and environment. *Ethn Dis* 2000;10:432–45.
25. **Adler NE, Marmot M, McEwen BS.** Socioeconomic status and health in industrial nations: social, psychological and biological pathways. *Ann N Y Acad Sci* 1999;896:1–501.
26. **Ren XS, Amick BC, Williams DR.** Racial/ethnic disparities in health: the interplay between discrimination and socioeconomic status. *Ethn Dis* 1999;9:151–65.
27. **Hutchinson RG, Watson RL, Davis CE, et al.** Racial differences in risk factors for atherosclerosis. *Angiology* 1997;48:279–89.
28. **Winkleby MA, Kraemer HC, Ahn DK, et al.** Ethnic and socioeconomic differences in cardiovascular disease risk factors: findings for women from the Third National Health

- and Nutrition Examination Survey, 1988–1994. *JAMA* 1998;280:356–62.
29. **Shea S, Stein AD, Basch CE, et al.** Independent associations of educational attainment and ethnicity with behavioral risk factors for cardiovascular disease. *Am J Epidemiol* 1991;134:567–82.
 30. **Gaillard TR, Schuster DP, Bossetti BM, et al.** The impact of socioeconomic status on cardiovascular risk factors in African-Americans at high risk for type II diabetes. *Diabetes Care* 1997;20:745–52.
 31. **Behera SK, Winkleby MA, Collins R.** Low awareness of cardiovascular disease risk among low-income African-American Women. *Am J Health Promot* 2000;14:301–5.
 32. **Landrine H, Klonoff EA.** Racial discrimination and cigarette smoking among Blacks: findings from two studies. *Ethn Dis* 2000;10:195–202.
 33. **Yen I, Ragland D, Greiner B, et al.** Racial discrimination and alcohol-related behavior in urban transit operators: findings from the San Francisco Muni Health and Safety Study. *Pub Health Rep* 1999;114:448–58.
 34. **Neuspiel DR.** Racism and perinatal addiction. *Ethn Dis* 1996;6:47–55.
 35. **Thompson B.** A way outa no way: eating problems among African American, Latina, and white women. *Gender Society* 1992;6:546–61.
 36. **Davis K.** Inequality and access to health care. *Milbank Q* 1991;69:253–73.
 37. **Smedley BD, Stith AY, Nelson AR.** Cardiovascular care. Unequal treatment: confronting racial and ethnic disparities in health care. Washington DC: National Academy Press; 2002. p. 35–42.
 38. **Mayberry RM, Mili F, Ofili E.** Racial and ethnic differences in access to medical care. *Med Care Res Rev* 2000;57 Suppl 1:108–45.
 39. **Waidmann TA, Rajan S.** Race and ethnic disparities in health care access and utilization: an examination of state variation. *Med Care Res Rev* 2000;57 Suppl 1:55–84.
 40. **Weinick RM, Zuvekas SH, Cohen JW.** Racial and ethnic differences in access to and use of health care services, 1977 to 1996. *Med Care Res Rev* 2000;57 Suppl 1:36–54.
 41. **Fiscella K, Franks P, Doescher MP, et al.** Disparities in health care by race, ethnicity, and language among the insured: findings from a national sample. *Med Care* 2002; 40:52–9.
 42. **Taylor AK, Cohen JW, Machlin SR.** Being uninsured in 1996 compared to 1987: how has the experience of the uninsured changed over time? *Health Serv Res* 2001;36:16–31.
 43. **Mills RJ.** Health insurance coverage 2000. Current population report P60-215. Washington DC: US Bureau of the Census; 2001. Available at: URL: <http://www.census.gov/prod/2001pubs/p60-215.pdf>
 44. **Ross CE, Mirowsky J.** Does medical insurance contribute to socioeconomic differentials in health? *Milbank Q* 2000;78: 291–321, 151–2.
 45. **Cunningham PJ.** Declining employer-sponsored coverage: the role of public programs and implications for access to care. *Med Care Res Rev* 2002;59:79–103.
 46. **Committee on the Consequences of Uninsurance.** Coverage matters: insurance and health care. Washington DC: Board on Health Care Services, Institute of Medicine; 2001.
 47. **Sheifer SE, Escarce JJ, Schulman KA.** Race and sex differences in the management of coronary artery disease. *Am Heart J* 2000;139:848–57.
 48. **Rosenbaum S, Markus A, Darnell J.** U. S. civil rights policy and access to health care by minority Americans: implications for a changing health care system. *Med Care Res Rev* 2000;57 Suppl 1:236–59.
 49. **Anderson RT, Sorlie P, Backlund E, et al.** Mortality effects of community socioeconomic status. *Epidemiology* 1996;8:42–7.
 50. **Kressin NR, Petersen LA.** Racial differences in the use of invasive cardiovascular procedures: review of the literature and prescription for future research. *Ann Intern Med* 2001; 135:352–66.
 51. **Williams DR, Collins C.** Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep* 2001;116:404–16.
 52. **Cattell V.** Poor people, poor places, and poor health: the mediating role of social networks and social capital. *Soc Sci Med* 2001;52:1501–16.
 53. **LeClere FB, Rogers RG, Peters K.** Neighborhood social context and racial differences in women's heart disease mortality. *J Health Soc Behav* 1998;39:91–107.
 54. **McCord C, Freeman HP.** Excess mortality in Harlem. *N Engl J Med* 1990;322:173–7.
 55. **Wandersman A, Nation M.** Urban neighborhoods and mental health: psychological contributions to understanding toxicity, resilience, and interventions. *Am Psychol* 1998;53: 647–56.
 56. **LaVeist TA, Wallace JM Jr.** Health risk and inequitable distribution of liquor stores in African American neighborhoods. *Soc Sci Med* 2000;51:613–7.
 57. **Pickett KE, Pearl M.** Multilevel analysis of neighbourhood socioeconomic context and health outcomes: a critical review. *J Epidemiol Community Health* 2001;55:111–22.
 58. **Harburg E, Chape C, Hauenstein LS, et al.** Socioecological stressor areas and black-white blood pressure: Detroit. *J Chronic Dis* 1973;26:595–611.
 59. **James SA, Kleinman DG.** Socioecologic stress and hypertension related mortality rates in North Carolina. *Am J Public Health* 1976;66:354–8.
 60. **Wing S, Casper M, Davis W, et al.** Trends in geographic inequality of cardiovascular disease mortality in the United States, 1962–1982. *Soc Sci Med* 1990;30:261–6.
 61. **Tyroler H, Wing S, Knowles M.** Increasing inequality in coronary heart disease mortality in relation to educational achievement: profile of places of residence, United States 1962–1987. *Ann Epidemiol* 1993;3(Suppl):S51–4, .
 62. **Harburg E, Erfurt JC, Hastein LS, et al.** Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosom Med* 1973;35:276–96.
 63. **Manfredi C, Lacey L, Warnecke R, et al.** Smoking-related behavior, beliefs, and social environment of young black women in subsidized public housing in Chicago. *Am J Public Health* 1992;82:267–72.
 64. **Diehr P, Koepsell T, Cheadell A, et al.** Do communities differ in health behaviors? *J Clin Epidemiol* 1993;46:1141–9.
 65. **Duncan C, Jones K, Moon G.** Do places matter? A multi-level analysis of regional variations of health-related behaviour in Britain. *J Epidemiol Community Health* 1993;37: 276–84.
 66. **Krieger N.** Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am J Public Health* 1992;82:703–10.
 67. **Kleinschmidt I, Hills M, Elliott P.** Smoking behaviour can be predicted by neighbourhood deprivation measures. *J Epidemiol Community Health* 1995;49 Suppl 2:S72–7.
 68. **Diez-Roux AV, Nieto FJ, Muntaner C, et al.** Neighborhood environments and coronary heart disease: a multilevel analysis. *Am J Epidemiol* 1997;146:48–63.

69. **Marmot MG.** Socio-economic and cultural factors in ischaemic heart disease. *Adv Cardiol* 1982;29:68–76.
70. **Dressler WW.** Lifestyle, stress, and blood pressure in a Southern black community. *Psychosom Med* 1990;52:182–98.
71. **Kaplan GA.** People and places: contrasting perspectives on the association between social class and health. *Int J Health Serv* 1996;26:507–19.
72. **Essed P.** Everyday racism: reports from women of two cultures. Claremont (CA): Hunter House Inc., 1990.
73. **Kaplan GA.** Biological and methodological approaches to the association between socio economics and cardiovascular disease. Presented at Report of the Conference on Socioeconomic Status and Cardiovascular Health and Diseases, 1995; Bethesda, MD.
74. **Murphy M, Nelson K, Elliott W.** Racial differences in diurnal blood pressure profile. *Am J Hypertens* 1988;1:55.
75. **James G.** Race and perceived stress independently affect the diurnal variation of blood pressure in women. *Am J Hypertens* 1991;4:382–4.
76. **Taylor SE, Repetti RL, Seeman T.** Health psychology: what is an unhealthy environment and how does it get under the skin? *Annu Rev Psychol* 1997;48:411–47.
77. **Brunner E.** Socioeconomic determinants of health: stress and the biology of inequality. *BMJ* 1997;314:1472–6.
78. **McEwen BS, Seeman T.** Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci* 1999;896:30–47.
79. **Williams DR, Neighbors H.** Racism, discrimination and hypertension: evidence and needed research. *Ethn Dis* 2001; 11:800–16.
80. **Krieger N.** Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int J Health Serv* 1999;29:295–352.
81. **Clark R, Anderson NB, Clark VR, et al.** Racism as a stressor for African Americans: a biopsychosocial model. *Am Psychol* 1999;54:805–16.
82. **Kessler RC, Mickelson KD, Williams DR.** The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. *J Health Soc Behav* 1999;40:208–230.
83. **Thoits P.** Dimensions of life events that influence psychological distress: an evaluation and synthesis of the literature. In: Kaplan H, editor. *Psychological research: trends in theory and research.* New York: Academic Press; 1983. p. 33–103.
84. **Anderson N, McNeilly M, Myers H.** Toward understanding race differences in autonomic reactivity. In: Turner JR, editor. *Individual differences in cardiovascular response to stress.* New York: Plenum Press; 1992. p. 125–45.
85. **Saunders E, Brest AN.** *Cardiovascular diseases in blacks.* 3rd ed. Philadelphia: FA Davis Co.; 1991.
86. **Deubner D, Tyroler H, Cassel J, et al.** Attributable risk, population attributable risk and populations attributable fraction of death associated with hypertension in a biracial population. *Circulation* 1975;52:901–8.
87. **Manuck S, Kasprowiez A, Muldoon M.** Behaviorally-evoked cardiovascular reactivity and hypertension: conceptual issues and potential associations. *Ann Behav Med* 1990; 12:17–29.
88. **Harrell JP, Merritt MM, Kalu J.** Racism, stress and disease. In: Jones R, ed. *African American mental health.* Hampton (VA): Cobb & Henry Publishers 1998;247–80.
89. **Jones DR, Harrell JP, Morris-Prather CE, et al.** Affective and physiological responses to racism: the roles of Afrocentrism and mode of presentation. *Ethn Dis* 1996;6:109–22.
90. **Morris-Prather C, Harrell J, Collins R, et al.** Gender differences in mood and cardiovascular responses to socially stressful stimuli. *Ethn Dis* 1996;6:123–31.
91. **Fang CY, Myers HF.** The effects of racial stressors and hostility on cardiovascular reactivity in African American and Caucasian men. *Health Psychol* 2001;20:64–70.
92. **Lynch JW, Everson SA, Kaplan GA, et al.** Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? *Am J Public Health* 1998;88:389–94.
93. **Gump B, Matthews KA, Raikkonen K.** Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity and left ventricular mass in African-American and white children. *Health Psychol* 1999;18:140–50.
94. **Light K, Obrist P, Sherwood A, et al.** Effects of race and marginally elevated blood pressure responses to stress. *J Hypertens* 1987;10:555–63.
95. **Manuck S.** Cardiovascular reactivity in cardiovascular disease: “Once more unto the breach.” *Int J Behav Med* 1994; 124–31.
96. **Guyl M, Matthews KA, Bromberger JT.** Discrimination and unfair treatment: relationship to cardiovascular reactivity among African American and European American women. *Health Psychol* 2001;20:315–25.
97. **Gerin W, Pickering TG, Glynn L, et al.** An historical context for behavioral models of hypertension. *J Psychosom Res* 2000;48:369–77.
98. **Troxel W, Matthews K, Bromberger J, et al.** Chronic stress burden, discrimination and subclinical cardiovascular disease in African American and Caucasian women. *Hypertension.* In press.
99. **Luft F, Grim C, Weinberger M.** Electrolyte and volume homeostasis in Blacks. In: Hall WD, Saunders E, Shulman N, editors. *Hypertension in Blacks: epidemiology, pathophysiology and treatment.* Chicago (IL): Yearbook Medical; 1985. p. 131–51.
100. **Koepke J, Light K, Grignolo A, et al.** Neural control of renal excretory function during behavioral stress in dogs. *Am J Physiol* 1983;245:251–8.
101. **Light K.** Differential responses to salt intake-stress interactions: relevance to hypertension. In: Turner J, Sherwood A, Light K, editors. *Individual differences in cardiovascular response to stress.* New York: Plenum Press; 1992. p. 245–64.
102. **Light KC, Koepke JP, Obrist PA, et al.** Psychological stress induces sodium and fluid retention in men at high risk for hypertension. *Science* 1983;220:429–31.
103. **Berenson G, Voors A, Dalferes E, et al.** Creatinine clearance, electrolytes, and plasma renin activity related to the blood pressure of black and white children—The Bogalooosa Heart Study. *J Lab Clin Med* 1979;95:535–48.
104. **Russell RP, Masi AT.** Significant association of adrenal cortical abnormalities with “essential” hypertension. *Am J Med* 1973;54:44–51.
105. **Deleted in proof.**
106. **Longo DL, Esterly JA, Grim CE, et al.** Pathology of the adrenal gland in refractory low renin hypertension. *Arch Pathol Lab Med* 1978;102:322–7.
107. **Henry J, Kross M, Stephens P, et al.** Evidence that differing psychosocial stimuli lead to adrenal cortical stimulation by autonomic or endocrine pathways. In: Usdin E, Kvetnansky R, Kopin I, editors. *Catecholamines and stress.* Oxford: Pergamon Press; 1976. p. 457–68.

108. **Andrew M, Jones D, Wofford M, et al.** Ethnicity and unprovoked hypokalemia in the Atherosclerosis Risk in Communities study. *Am J Hypertens* 2002;15:594–9.
109. **Blessing E, Mellon T-M, Campbell LA, et al.** Chlamydia pneumoniae induces inflammatory changes in the heart and aorta of normocholesterolemic C57BL/6J mice. *Infect Immunol* 2000;68:4765–8.
110. **Ramirez J.** Isolation of *Chlamydia pneumoniae* from the coronary artery of a patient with coronary atherosclerosis. The *Chlamydia pneumoniae*/Atherosclerosis Study Group. *Ann Intern Med* 1996;125:979–82.
111. **Mendall M, Patel P, Ballam L, et al.** C reactive protein and its relation to cardiovascular risk factors: a population based cross sectional study. *BMJ* 1996;312:1061–5.
112. **Campbell L, Kuo C-C, Grayston JT.** Chlamydiae pneumonia and cardiovascular disease. *Emerg Infect Dis* 1998;4.
113. **James S, LaCroix A, Kleinbaum D, et al.** John Henryism and blood pressure differences among black men. II. The role of occupational stressors. *J Behav Med* 1984;7:259–75.
114. **Krieger N.** Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med* 1990;30:1273–81.
115. **James K, Lovato C, Khoo G.** Social identity correlates of minority workers' health. *Acad Management J* 1994;37:383–96.
116. **Broman CL.** The health consequences of racial discrimination: a study of African Americans. *Ethn Dis* 1996;6:148–53.
117. **Krieger N, Sidney S.** Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health* 1996;86:1370–8.
118. **Liebkind K, Jasinskaja-Lahti I.** The influence of experiences of discrimination on psychological stress: a comparison of seven immigrant groups. *J Community Appl Psychol* 2000;10:1–16.
119. **Poston W, Pavlik V, Hyman D, et al.** Genetic bottlenecks, perceived racism, and hypertension risk among African Americans and first-generation African immigrants. *J Hum Hypertens* 2001;15:341–51.
120. **Karlsen S, Nazroo JY.** Agency and structure: the impact of ethnic identity and racism on the health of ethnic minority people. *Sociol Health Illn* 2002;24:1–20.
121. **Karlsen S, Nazroo JY.** Relation between racial discrimination, social class, and health among ethnic minority groups. *Am J Public Health* 2002;92:624–31.
122. **James SA, Strogatz DS, Wing SB, et al.** Socioeconomic status, John Henryism, and hypertension in blacks and whites. *Am J Epidemiol* 1987;126:59–67.
123. **Branscombe NR, Schmitt MT.** Perceiving pervasive discrimination among African Americans: implications for group identification and well-being. *J Pers Soc Psychol* 1999;77:135–49.
124. **Grier WH, Cobbs PH.** *Black rage*. New York: Basic Books; 1968.
125. **Adams J, Dressler W.** Perceptions of injustice in a black community: dimensions and variations. *Hum Relations* 1988;41:753–67.
126. **Pearlin L.** The sociological study of stress. *J Health Soc Behav* 1989;30:241–56.
127. **Wiebe D, Williams P.** Hardiness and health: a social psychophysiological perspective on stress and adaptation. *J Soc Clin Psychol* 1992;11:238–62.
128. **Everson SA, Goldberg DE, Kaplan GA, et al.** Anger expression and incident hypertension. *Psychosom Med* 1998;60:730–35.
129. **Wyatt S, AlFadhli H, Andrew M, et al.** Community-driven model of recruitment: the Jackson Heart Study. A final report of the Jackson Heart Study participant recruitment and retention study. Washington DC: National Heart Lung and Blood Institute; 1999.
130. **Taylor D, Wright S, Moghaddam F, et al.** The personal/group discrimination discrepancy: perceiving my group, but not myself to be a target for discrimination. *Pers Soc Psychol Bull* 1990;16:254–62.
131. **Crosby F.** The denial of personal discrimination. *Am Behav Sci* 1984;27:371–86.
132. **Lalonde R, Cameron J.** Behavioral responses to discrimination: a focus on action. In: Zanna M, Olson J, editors. *The psychology of prejudice: the Ontario symposium*. Vol. 7. Hillsdale (NJ): Lawrence Erlbaum; 1994. p. 257–88.
133. **Essed P.** *Understanding everyday racism: an interdisciplinary theory*. Newbury Park (CA): Sage Publications; 1991.
134. **Feagin J, Sikes M.** *Living with racism: the black middle class experience*. Boston: Beacon Press; 1994.
135. **Pierce C.** Stress analogs of racism and sexism: terrorism, torture and disaster. In: Willie C, Reiker P, Kramer B, Brown B, editors. *Mental health, racism and sexism*. Pittsburgh: University of Pittsburgh Press; 1995. p. 277–93.
136. **McNeilly MD, Anderson NB, Armstead CA, et al.** The Perceived Racism Scale: a multidimensional assessment of the experience of white racism among African Americans. *Ethn Dis* 1996;6:154–66.
137. **Clark V, Harrell J.** The relationship among Type A behavior, styles used in coping with racism, and blood pressure. *J Black Psychol* 1982;8:89–99.
138. **Moore RB.** Racism in the English language. In: Rothenberg PS, editor. *Race, class and gender in the United States: an integrated study*. 4th ed. New York: St Martins' Press; 1988. p. 465–75.
139. **Taylor J, Grundy C.** Measuring black internalization of white stereotypes about African Americans: the Nadanolitization Scale. In: Jones R, editor. *Handbook of tests and measurements for Black populations*. Vol. 2. Hampton (VA): Cobb & Henry; 1996. p. 217–26.
140. **McCorkle KC.** Effects of maternal internalized racism, socioeconomic status, depression, and parenting quality upon child maladjustment [dissertation]. Pittsburgh: University of Pittsburgh; 1991.
141. **Taylor J, Henderson D, Jackson B.** A holistic model for understanding and predicting depression in African American women. *J Commun Psychol* 1991;19:306–20.
142. **Checkley S.** The neuroendocrinology of depression and chronic stress. *Br Med Bull* 1996;53:597–617.
143. **Lake CR, Pickar D, Dixon K, et al.** High plasma norepinephrine levels in patients with major affective disorder. *Am J Psychiatry* 1982;139:1315–8.
144. **Hemingway H, Marmot M.** Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. *BMJ* 1999;318:1460–7.
145. **Jonas BS, Franks P, Ingram DD.** Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination I Epidemiologic Follow-up Study. *Arch Fam Med* 1997;6:43–49.
146. **Ferketich AK, Schwartzbaum JA, Frid DJ, et al.** Depression as an antecedent to coronary heart disease among women and men in the NHANES I study. *Arch Intern Med* 2000;160:1261–8.
147. **Davidson K, Jonas B, Kison K, et al.** Do depression symptoms predict early hypertension incidence in young adults from the CARDIA study? *Arch Intern Med* 2000;160:1495–500.

148. **Armstead C, Lawler K, Gorden G, et al.** Relationship of racial stressors to blood pressure responses and anger expression in black college students. *Health Psychol* 1989;8:541–56.
149. **Klonoff EA, Landrine H.** Acculturation and cigarette smoking among African American adults. *J Behav Med* 1996;19:501–14.
150. **Tull ES, Wickramasuriya T, Taylor J, et al.** Relationship of internalized racism to abdominal obesity and blood pressure in Afro-Caribbean women. *J Natl Med Assoc* 1999; 91:447–52.
151. **Airhihenbuwa CO, Kumanyika SK, TenHave TR, et al.** Cultural identity and health lifestyle among African Americans: a new direction for health intervention research? *Ethn Dis* 2000;10:148–64.
152. **Torres A, Bowens L.** Correlation between the internalization theme of Racial Identity Attitude Survey-B and systolic blood pressure. *Ethn Dis* 2000;10:375–83.
153. **Thompson HS, Kamarck TW, Manuck SB.** The association between racial identity and hypertension in African-American adults: elevated resting and ambulatory blood pressure as outcomes. *Ethn Dis* 2002;12:20–8.
154. **Koenig HG, George LK, Hays JC, et al.** The relationship between religious activities and blood pressure in older adults. *Int J Psychiatry Med* 1998;28:189–213.
155. **Williams DR.** Racism and health: a research agenda. *Ethn Dis* 1996;6:1–6.
156. **Utsey SO.** Assessing the stressful effects of racism: a review of instrumentation. *J Black Psychol* 1998;24:269–88.
157. **Deleted in proof.**
158. **Wyatt SB, Williams D, Boss B, et al.** Social factors in cardiovascular disease: design for the Jackson Heart Study. *Ethn Dis* 1999;9:306.
159. **Lenfant C.** Report of the NHLBI working group on research in coronary heart disease in blacks. *Circulation* 1994;90:1613–23.