

Race and Ethnicity in Public Health Research: Models to Explain Health Disparities

William W. Dressler,¹ Kathryn S. Oths,¹
and Clarence C. Gravlee²

¹Department of Anthropology, The University of Alabama, Tuscaloosa, Alabama 35487; email: wdressle@tenhour.as.ua.edu, koths@tenhour.as.ua.edu

²Department of Anthropology, Florida State University, Tallahassee, Florida 32306; email: cgravlee@fsu.edu

Annu. Rev. Anthropol.
2005. 34:231–52

First published online as a
Review in Advance on
June 14, 2005

The *Annual Review of
Anthropology* is online at
anthro.annualreviews.org

doi: 10.1146/
annurev.anthro.34.081804.120505

Copyright © 2005 by
Annual Reviews. All rights
reserved

0084-6570/05/1021-
0231\$20.00

Key Words

cultural constructivism, psychosocial stress, racism, birth weight, blood pressure

Abstract

The description and explanation of racial and ethnic health disparities are major initiatives of the public health research establishment. Black Americans suffer on nearly every measure of health in relation to white Americans. Five theoretical models have been proposed to explain these disparities: a racial-genetic model, a health-behavior model, a socioeconomic status model, a psychosocial stress model, and a structural-constructivist model. We selectively review literature on health disparities, emphasizing research on low birth weight and high blood pressure. The psychosocial stress model and the structural-constructivist model offer greatest promise to explain disparities. In future research, theoretical elaboration and operational specificity are needed to distinguish among three distinct factors: (a) genetic variants contributing to disease risk; (b) ethnoracial or folk racial categories masquerading as biology; and (c) ethnic group membership. Such elaboration is necessary to move beyond the conflation of these three distinct constructs that characterizes much of current research.

Contents

INTRODUCTION.....	232
RACIAL AND ETHNIC HEALTH DISPARITIES: THE EPIDEMIOLOGIC EVIDENCE	232
PATTERNS IN THE LITERATURE ON RACIAL AND ETHNIC HEALTH DISPARITIES.....	234
EXPLAINING RACIAL AND ETHNIC HEALTH DISPARITIES.....	234
The Racial-Genetic Model.....	235
The Health-Behavior Model.....	236
The Socioeconomic Status Model	238
The Psychosocial Stress Model...	238
The Structural-Constructivist Model.....	241
RESEARCH NEEDS.....	243
CONCLUSION.....	245

racial and ethnic groups as well. Every field of study concerned with public health, including epidemiology, health services research, health psychology, medical sociology, clinical medicine, nursing, and medical anthropology, contributes to this research.

The issues are immense. Approaching this research in any sensible way demands that subjects from population genetics to the social and cultural construction of the concepts of race and ethnicity be addressed. And there are deeply entrenched ideologies on all sides of the issue. Separating ideology and theory is always difficult, but it seems to be particularly problematic in the study of the health effects of race and ethnicity. On the one hand, the biological reality of race continues to be obvious to many. On the other hand, it is equally clear to many researchers that race is a culturally constructed entity. Stating these two perspectives so simply obscures the profound differences in epistemology that generate them, differences that can overwhelm research progress on a topic that demands an integration of realist and constructivist perspectives. The study of racial and ethnic health disparities requires a biocultural perspective in the fullest sense of the term.

The aim of this chapter is to examine how some of these thorny questions have been approached. The literature is vast, so our discussion will be selective, out of necessity. First, we briefly review the nature and breadth of racial and ethnic health disparities. Second, we address the major alternative explanations for these disparities. Third, we turn to the question of the logical status of the constructs of race and ethnicity and consider how they might be more effectively defined for cross-cultural research.

RACIAL AND ETHNIC HEALTH DISPARITIES: THE EPIDEMIOLOGIC EVIDENCE

A research focus on health disparities can be traced to the *Black Report* examining social class and regional differences in health status

INTRODUCTION

Understanding health disparities is a major initiative of the public health research establishment in the United States (Woolf et al. 2004) and around the world (Almeida-Filho et al. 2003). Reducing such health disparities is one goal of the *Healthy People 2010* initiative, a program of the National Institutes of Health (NIH) intended to improve public health in the United States (U.S. Dep. Health Hum. Serv. 2000). “Health disparities” refers to differences in morbidity, mortality, and access to health care among population groups defined by factors such as socioeconomic status, gender, residence, and especially “race” or “ethnicity.” Research has examined differences in health status among diverse racial and ethnic groups; however, why the health disparities between black and white Americans are so large is the central question in this research. On nearly every index measured, African Americans suffer in relation to European Americans, and often in relation to other

Health disparities: differences in morbidity, mortality, and access to health care among population groups defined by factors such as socioeconomic status, gender, residence, and race or ethnicity

NIH: National Institutes of Health

in Britain (Townsend & Davidson 1982), but realization of the extent of ethnic and racial health disparities in the United States has been a result of reports published by the public health research establishment, especially the NIH and related agencies. Nickens (1986) drew attention to these disparities through a calculation of “excess deaths” in relation to race or ethnicity. This is an epidemiologic index in which predicted deaths are calculated for a population subgroup (e.g., black men) using data from another population subgroup (e.g., white men). Excess deaths are the number of deaths observed that exceed the predicted number. Nickens found substantial disparities between ethnic and racial groups: Black Americans in particular exhibited a large excess mortality compared with white Americans on virtually every cause of death. Other ethnic groups (e.g., Asian and Pacific Islanders) exhibited fewer than predicted deaths in some categories compared with white Americans, which indicates that health disparities can be observed in lower-than-expected rates for some groups as well.

Wong & associates (2002) conducted a similar analysis with more sophisticated techniques of adjustment. They also contrasted racial and ethnic disparities with educational disparities and found that different diseases contribute to different disparities. In terms of disparities in mortality between less well- and more well-educated persons, coronary artery disease, lung cancer, stroke, pneumonia, congestive heart failure, and lung disease contributed most to lost years of life. With respect to racial or ethnic disparities in mortality, however, hypertension, HIV, diabetes, and homicide were the greatest contributors. Therefore, there are a few diseases that contribute most to disparities in mortality.

Keppel et al. (2002) examined data between 1990 and 1998 using a set of ten health status indicators relevant to the *Healthy People 2010* goals. These indicators included a range

of health conditions (coronary heart disease, cancer, tuberculosis) as well as indicators with more direct social referents such as live births to teenagers ages 15–17 and suicide and homicide rates. Keppel et al. examined these indicators in relation to racial and ethnic categories employed in the U.S. census: non-Hispanic white; non-Hispanic black; Hispanic; American Indian or Alaska Native; and Asian or Pacific Islander. For mortality related to disease and traumatic injury, rates declined for all population subgroups between 1990 and 1998. With respect to racial and ethnic disparities, black Americans’ rates for six measures (total mortality, heart disease, lung cancer, breast cancer, stroke, and homicide) exceeded other groups’ rates by a factor ranging from 2.5 to almost 10 during both time periods. Other ethnic groups had higher rates for suicide (white Americans) and motor vehicle accidents (American Indian and Alaskan Natives). Overall, Asians and Pacific Islanders tended to have the lowest mortality rates, although Hispanics were lowest for strokes. Keppel et al. concluded,

Based on this analysis relatively little progress was made toward the goal of eliminating racial/ethnic disparities among the HSIs [health status indicators] during the last 10 years. Progress toward the goal of eliminating health disparities will require more concerted efforts during the next 10 years. (2002, p. 12)

Clearly, understanding ethnic and racial health disparities demands a careful examination of all groups in all societies in which such disparities exist. However, these results indicate that understanding the health status differences between black and white Americans is fundamental to understanding health disparities in general because this difference contributes most to overall health disparities. In the remainder of this review, we place emphasis on understanding these disparities.

Ethnicity: a dimension of sociocultural systems that defines essential group differences and structures the relations among persons classified in terms of those differences. The criteria of essential group difference will vary cross-culturally

HSIs: health status indicators

Racial-genetic

model: a model for the explanation of health disparities that emphasizes population differences in the distribution of genetic variants

Health-behavior

model: a model for the explanation of health disparities that emphasizes differences between racial and ethnic groups in the distribution of individual behaviors related to health, such as diet, exercise, and tobacco use

Socioeconomic

status model: a model for the explanation of health disparities that posits the over-representation of some racial and ethnic groups within lower socioeconomic statuses

Psychosocial stress

model: a model for the explanation of health disparities that emphasizes the stresses associated with minority group status, and especially the experience of racism and discrimination

PATTERNS IN THE LITERATURE ON RACIAL AND ETHNIC HEALTH DISPARITIES

A number of reviews have examined the extent to which race and ethnicity are used as variables in public health research. Systematic reviews of several key journals show that (a) race and ethnicity are among the most commonly used variables in public health research; (b) their use is on the rise; and (c) they tend to be used uncritically and without definition.

Jones and colleagues (1991) reviewed articles published between 1921 and 1990 in the *American Journal of Epidemiology* and found that roughly two thirds of articles made reference to race. By 1990 almost 80% of the research articles published included race as a variable. Comstock et al. (2004) updated these results and found that 77% of articles published from 1996 to 1999 in the *American Journal of Epidemiology* and the *American Journal of Public Health* made reference to race or ethnicity. Williams (1994) documents a similar pattern in the health services research literature from 1966 to 1990, and Drevdahl and colleagues (2001) show that more than 81% of reports published in 2000 in the journal *Nursing Research* referred to race or ethnicity.

Investigators have observed similar patterns outside the United States. For example, Ellison & de Wet (1997) report that roughly half of the articles published in the *South African Medical Journal* in 1992–1996 mentioned race, and the proportion was much higher (73.9%) in genetic studies. British health researchers also use racial and ethnic categories (Sheldon & Parker 1992), although there is a decided preference for the concept of ethnicity more than race in the United Kingdom.

Explicit definitions of race or ethnicity are rare. Williams's (1994) review revealed that none of the 121 empirical studies published between 1966 and 1990 in the health services research literature defined race. Just 5 of the 167 studies published in *Nursing Research* from 1952 to 2000 defined the racial and ethnic

categories used, and these definitions often failed to make clear the differences among categories (Drevdahl et al. 2001). Similarly, in a review of the *American Journal of Epidemiology* and the *American Journal of Public Health*, Comstock et al. (2004) found that most recent articles neither specify why race or ethnicity was included as a variable nor identify the method by which either was assessed. These patterns appear to hold true elsewhere in the biomedical literature (Kaplan & Bennett 2003, Osborne & Feit 1992).

Social scientists are often quick to recognize these shortcomings in the public health literature, but it remains to be seen if they approach race and ethnicity in any more sophisticated way. Preliminary results from an ongoing content analysis of *Medical Anthropology* and *Medical Anthropology Quarterly* (Gravlee et al. 2004) suggest that neither the concept of race nor ethnicity is used as commonly in medical anthropology as in public health. Medical anthropologists display a preference for ethnicity more than race, but an analytic distinction between these concepts is seldom made. In addition, as in public health, race and ethnicity typically are not defined, and the methods by which groups and individuals are assigned to racial or ethnic categories generally are not explicit.

EXPLAINING RACIAL AND ETHNIC HEALTH DISPARITIES

Dressler (1993) identified four general models in the literature to explain health disparities: a racial-genetic model; a health-behavior model; a socioeconomic status model; and a social structural model. Changes in emphasis in the literature in the intervening ten years require both expansion and slight modification of these categories as follows: a racial-genetic model; a health-behavior model; a socioeconomic model; a psychosocial stress model; and a structural-constructivist model.

In the following sections, we sample from the health disparities literature, but most

of our examples will come from research on low birth weight and essential hypertension/chronic high blood pressure. These are useful health indicators because they effectively bracket the life span, and they are among the health problems that contribute most to health disparities. Additionally, there is evidence that low birth weight and high blood pressure are associated, and hence may be linked in the biocultural processes that generate health disparities (Barker 2004).

The Racial-Genetic Model

There are large differences in rates of low birth weight (defined as birth weight less than 2500 grams) and rates of hypertension (blood pressure higher than 140 mm Hg systolic and/or 90 mm Hg diastolic) between black and white Americans. Currently 13.3% of black women, versus 6.9% of white women, give birth to a low birth weight baby. Similarly, 38.6% of black women and 34.8% of black men have high blood pressure, compared with 22.6% of white women and 24.8% of white men (Cent. Dis. Control Prev. 2004a,b).

Especially with respect to high blood pressure, there have been appeals by some researchers to racial-genetic factors to account for these disparities (Boyle 1970). For blood pressure, these appeals have been based on both the differences in hypertension prevalence between blacks and whites, and the gradient of blood pressure and hypertension prevalence in relation to skin color within the African American community (Harburg et al. 1978, Keil et al. 1977, Klag et al. 1991). Although poor birth outcomes are less often explicitly attributed to racial-genetic differences, some researchers have suggested as such (Wilcox & Russell 1990, and see critique by David 2001). In current literature it can be difficult to find overt attribution of disease risk to a racial-genetic component, perhaps because of widespread knowledge of the critique of race as a biological construct (Cooper 1984, Montagu 1962, Kittles & Weiss 2003). More often than not, as noted above, differ-

ences associated with race or ethnicity are simply reported.

Prior to technological advances in molecular biology, evidence had accumulated that a racial-genetic explanation was untenable for broad population differences in blood pressure. Literature reviews showed that prevalence rates were extremely variable across populations in Africa and people of African descent in the New World. Later, focused research (eliminating alternative measurement hypotheses for these differences) demonstrated an east-west gradient in hypertension prevalence: West African samples had the lowest prevalence (16%), West Indian populations had an intermediate prevalence (26%), and African American populations had the highest prevalence (33%) (Cooper et al. 1997). With respect to birth weight, David & Collins (1997) found that the rate of low birth weight infants of African-born black women in Chicago (3.6%) was closer to that of U.S.-born white women (2.4%) than to U.S.-born black women (7.5%). Similarly, Kleinman and associates (1991), using a national data set, found that the risk of neonatal mortality was 22% lower for foreign-born compared with U.S.-born black women, whereas there was no difference in risk for whites on the basis of birth country. More recently, Acevedo-Garcia et al. (2005) have shown that lower-educated foreign-born black women do not differ in birth outcomes from highly educated U.S.-born black women (less well-educated U.S.-born black women had the highest risk of low birth weight). These data at least suggest that birth weight and blood pressure are subject to substantial environmental influence and hence are not under strict racial-genetic control, given the range of variability in prevalence rates in genetically related populations living in different environments.

With the advent of technology for identifying genetic variants, the importance of a racial-genetic component in blood pressure has become even less tenable because the search for variant gene structures that contribute to blood pressure has not been

Structural-constructivist model: a model for the explanation of health disparities that emphasizes the intersection of racially stratified social structures with the cultural construction of routine goals and aspirations as the cause of differences in morbidity and mortality. Explicit in this model is the cultural construction of the notion of “race” itself.

particularly successful (Crews & Williams 1999, Harrap 2003, Oparil et al. 2003); and those candidate genes that do appear to be associated with blood pressure are not differentially distributed across conventional racial groups (Cooper et al. 1999, Daniel & Rotimi 2003), nor do they differ between African Americans and first-generation African immigrants (Bouzekri et al. 2004, Carlos Poston et al. 2001).

The racial-genetic model has not disappeared, however. Belief in its importance in the explanation of blood pressure disparities continues in the form of Grim's "slavery hypothesis" (Grim & Robinson 1996). This hypothesis posits that a salt-sparing genetic variant was selected for in Africa (a kind of "thrifty genotype") owing to chronic salt shortages. Then, enslaved Africans were subjected to extreme conditions of sodium deprivation in the Middle Passage and under conditions of slavery, leading to high mortality rates, and this salt-sparing genetic variant in New World African-descent populations was further selected for. According to the slavery hypothesis, owing to the higher prevalence of this racial-genetic trait, African Americans and other black populations in the western hemisphere retain more sodium when it is plentiful in the diet, resulting in high blood pressure (see below on the salt intake hypothesis for hypertension).

The slavery hypothesis is a controversial idea to account for racial and ethnic health disparities and has many critics (Curtin 1992, Jackson 1991). What is striking, however, is its wide acceptance based on virtually no empirical evidence. In a paper full of insight into how hypotheses diffuse, Kaufman & Hall (2002) demonstrate the level of credibility the hypothesis has received in both the professional and popular literature, even being incorporated into recommendations on how high blood pressure should be treated (Brownley et al. 1999), despite the lack of research. As Kaufman & Hall note, "The intellectual resilience of the Slavery Hypothesis may be attributable to several of its ideologic

components[. . .]perhaps the most influential of these in the modern era is the beguiling allure of a simplistic genetic determinism" (2002, p. 116). The slavery hypothesis may owe its persistence to its reinforcement of folk models of race. As Kittles & Weiss (2003, p. 34) point out, even specialists in genetics routinely confuse technical and folk uses of the term race. The slavery hypothesis may appear to be true simply because it is consistent with, and in turn reinforces, a Western European and American cultural construction of race as a biologic entity (see also Braun 2002, Sankar et al. 2004).

In sum, this model for the explanation of racial and ethnic health disparities that emphasizes genetic variants differentially distributed across these groups appears to have little explanatory power.

The Health-Behavior Model

Here we subsume hypotheses that account for health disparities on the basis of discrete behaviors voluntarily adopted by individuals. The health behaviors regarded as important usually include the combination of high caloric intake and low physical activity, which leads to obesity, smoking, excessive alcohol intake, and the intake of specific dietary items, such as high salt intake or low potassium intake.

Body composition is clearly associated with higher blood pressure (Sowers et al. 2002), but differences in body composition do not explain health disparities. National Center for Health Statistics (NCHS) (Cent. Dis. Control Prev. 2004c) data show that in 1999–2000, 67.3% of white American men would be considered overweight (a body mass index >25.0), compared with 60.3% of African American men. This discrepancy therefore could not account for the differences in hypertension prevalence between black and white men. Fifty-seven percent of white women are overweight compared with 77.7% of black women. Although this seemingly could account for prevalence differences between

black and white women, it apparently does not. Bell and associates (2004) recently analyzed data from the NHANES (National Health and Nutrition Examination Survey) for women, and black women remained twice as likely to have high blood pressure after controlling for obesity. Furthermore, virtually every study of blood pressure routinely uses a measure of body composition (e.g., the body mass index) as a control variable, with little effect on black-white disparities.

With respect to birth weight, heavier women tend to have heavier babies (Inst. Med. 1990), so the higher prevalence of overweight among black women cannot explain the higher prevalence of low birth weight.

Physical activity levels affect both weight and overall risk of disease, and there is evidence of differences among racial and ethnic groups in levels of physical activity. Nationally, 34.4% of white men and 38.3% of white women report being physically inactive compared with 45.1% of black men and 55.1% of black women (Schoenborn et al. 2004, p. 42). Bell et al. (2004) found that controlling for reported levels of physical activity made no difference in the differential risk of hypertension between black and white women. With respect to low birth weight, strenuous occupational activity (such as standing for long periods) does not alter differences in low birth weight between black and white women (Homer et al. 1990, Teitelman et al. 1990).

Smoking is a risk factor that has been directly implicated in low birth weight (McCormick et al. 1990) but only tangentially, if at all, in association with blood pressure (Janzon et al. 2004). Again, smoking is not a factor likely to account for health disparities because there are virtually no differences in rates of smoking between black and white men (27.1% versus 25.2%) or between black and white women (19.5% versus 22.2%) (Schoenborn et al. 2004, p. 21).

Alcohol intake is discouraged during pregnancy because it contributes to low birth weight, whereas when considering blood

pressure there appears to be a J-shaped relationship between alcohol consumption and blood pressure; nondrinkers and mild drinkers (≤ 3 drinks/day) had comparably low blood pressures, and heavy drinkers had higher blood pressures (Estruch et al. 2004). According to NCHS survey data, 70.8% of white men versus 55.8% of black men report being drinkers; corresponding figures for white versus black women are 60.4% versus 39.4%. Rates of heavy drinking, defined as 14 or more drinks per week, are very similar across groups, ranging from 2.2% among African American women to 5.6% among white men (Schoenborn et al. 2004, p. 7). Again, there is little evidence that differences in alcohol intake account for the large disparities in health status; in studies that control for reported alcohol intake, racial and ethnic differences in blood pressure and birth weight remain unchanged, although there is some evidence that blood pressure may increase at a lower level of alcohol intake for black men versus other groups (Acevedo-Garcia et al. 2005, Bell et al. 2004, Fuchs et al. 2001).

With respect to specific factors and specific outcomes, high salt intake has been hypothesized to account for black-white differences in blood pressure (Sowers et al. 2002); however, the link of salt intake with high blood pressure continues to be controversial. As McCarron (2000) notes, the salt intake hypothesis has been based on inappropriate animal laboratory models; the reporting of highly selective cross-cultural data; and questionable and unsubstantiated concepts like a genetic salt sensitivity. Although reduction in dietary sodium can lead to small but sustained decreases in blood pressure in some persons with high blood pressure, the evidence for salt intake as an etiologic factor that accounts for racial and ethnic differences in hypertension is slim to nonexistent (Chrysant et al. 1997).

Some investigators have attempted to evaluate the combined effects of health behaviors in reducing racial and ethnic health disparities. These studies enter physical activity, smoking, alcohol consumption, body

NCHS: National Center for Health Statistics

NHANES: National Health and Nutrition Examination Survey

mass index, and various measures of nutrient intake simultaneously into an analysis, along with race or ethnicity. The results are mixed. In some studies, racial or ethnic differences are left unchanged (Bassett et al. 2002, Bell et al. 2004). With these variables, plus depressive symptoms and anxiety, Jones-Webb and coworkers (1996) were able to reduce differences in mean blood pressure for black and white men, but not for black and white women. Finally, using prospective data, Liu et al. (1996) reported that black-white mean blood pressure differences at a 7-year follow-up could be reduced by 40%–50% through the inclusion of body mass index, alcohol and tobacco use, physical activity, and dietary intake of calcium, potassium, and protein.

As observed ten years ago, these health behaviors can be potent contributors to disease risk (Dressler 1993); there is little evidence, however, that alone or in combination health behaviors can explain racial and ethnic health disparities.

The Socioeconomic Status Model

The socioeconomic status (SES) model subsumes research that sees racial and ethnic health disparities confounded with SES disparities in health. Race and SES are correlated (i.e., African Americans are overrepresented among lower SES groups), and some argue that controlling for SES will either reveal the “true” effect of race or ethnicity or, if secondary to SES disparities, cause racial disparities to disappear. Little or no consideration is given to why confounding occurs. This approach was encountered more often in earlier literature (e.g., Keil et al. 1977, Starfield et al. 1991) than recent studies (although see Dyer et al. 1999). In general, controlling for SES fails to account completely for racial and ethnic disparities, despite leading to a reduction in the magnitude of group differences. The failure of SES controls to account for racial or ethnic differences has then, in turn, been used as “evidence” of some kind of residual racial-genetic effect. As Kaufman

and colleagues (1997) show, however, such an inference is almost never warranted because of the problems associated with trying to untangle race, ethnicity, and SES. Various forms of residual confounding occur, which in turn render an inference regarding some kind of racial-genetic effect unlikely, even after controlling for SES (see also Davey Smith 2000).

Some researchers have argued that the confounding of SES and racial disparities is a function of the wider distribution of risky health behaviors among lower class people, such as those behaviors reviewed in the previous section (Liu et al. 1996, Stamler et al. 2003). This again is an argument that race or ethnicity is confounded with SES; however, as shown above, controlling for health behaviors does not explain racial and ethnic health disparities.

More promising directions in research on SES examine the effect of residence in low-income communities on health (Williams & Collins 2001), as well as how SES may moderate racial or ethnic differences (Acevedo-Garcia et al. 2005).

The Psychosocial Stress Model

In earlier literature, after showing that controls for SES failed to account for racial and ethnic disparities, reasoned speculation led to a consideration of the stresses associated with institutional and interpersonal racism as a cause of these disparities (Clark et al. 1999, Williams & Collins 1995). The extension of the psychosocial stress model to studies of racial and ethnic disparities was stimulated by the logical difficulties and empirical weakness of alternative racial-genetic and health-behavior models. As usual, the serious consideration of the social production of disease had to await the exhaustion of alternative approaches. Currently, this model looms large in the literature, and it is possible to categorize several approaches within this orientation.

The first approach can be best exemplified by the social epidemiologists Krieger (1999, 2003) and Williams (Williams & Collins 1995,

Wyatt et al. 2003). In this approach, there is a clear distinction made between institutional racism and perceived racism, the former referring to the system of structured inequality that places black Americans lower on all indicators of economic well-being, and the latter referring to the conscious perception of discriminatory acts and practices and the distress associated with that perception. Institutional racism results in the limited access of racial and ethnic minorities to resources, both in the sense of limited access to high-paying jobs or educational opportunities and in the sense of limited access to resources that would support the attainment of better health status (e.g., living in neighborhoods with markets that stock fresh fruits and vegetables, neighborhoods in which it is safe to walk for exercise). The concept of institutional racism has mainly offered a framework for the interpretation of racial and ethnic health disparities that is an alternative to other (e.g., racial-genetic) models, providing what Krieger (1999, p. 310) calls an “indirect” approach to the study of discrimination and health.

Perceived racism, by comparison, is measured directly by self-reports of respondents about their experiences of discriminatory acts, both in institutional settings (e.g., on the job) and in mundane social interactions (Krieger 1990, Krieger & Sidney 1996). The empirical record for measures of perceived discrimination is mixed. In a recent review, Williams and associates (2003) report eleven studies that examine the association of perceived discrimination and blood pressure. Of these studies, three find a direct association, three find no association, and five find associations that pertain only to particular subgroups (e.g., gender or occupational groups; see also Brondolo et al. 2003). A recent study reports a direct association of perceived discrimination and blood pressure, although data were collected from a convenience sample (Din-Dzietham et al. 2004). Two studies in the review by Williams et al. (2003) examined perceived discrimination and low birth weight; one found no association and one found a conditional as-

sociation. A more recent study found that controlling for self-reported discrimination reduced by half the risk for black women of reporting having had a low birth weight baby (Mustillo et al. 2004; see also Collins et al. 2004).

The second approach to the study of psychosocial stress and health disparities employs a more general understanding of the term stress as negative affect (depression, anxiety) experienced by individuals, which in turn can be associated with deleterious health outcomes. This approach has been taken in the incorporation of psychosocial data into large national studies, such as CARDIA (Cardiovascular Disease in Young Adults) and the various waves of the NHANES (see Williams 1999 for a useful discussion of national data sets). Jonas et al. (1997) and Jonas & Lando (2000) looked at overall negative affect as a prospective predictor of incident hypertension in two different follow-up waves of the NHANES, finding that those who report negative affect are at a higher risk for developing hypertension and that this association is greater for African Americans. Davidson et al. (2000) found a similar pattern of results using the CARDIA data. Finally, using a subset of the CARDIA data, Knox and colleagues (2002) found that young African Americans who were more reactive to stressful stimuli in the laboratory in turn had higher ambulatory blood pressures three years later.

The third approach to the study of psychosocial stress and health outcomes is best represented by the early work of Harburg and associates (1973), and the subsequent work of James on the John Henryism hypothesis (James et al. 1983). These researchers adapted general models of the stress process to the specific ethnographic realities of the African American community. For example, Harburg et al. (1973) argued that persons, black or white, living in high “socioecologic stress” areas (characterized by low SES and high rates of social instability as measured by crime) were at a higher risk for stressful experiences on a daily basis, increasing

the likelihood of high blood pressure. For African Americans, and especially darker-skinned black men, there was the added insult of racist interactions (with police or other representatives of the white power establishment). These racist interactions were in turn likely to provoke hostility on the part of the black participant in the interaction, who may then suppress that hostility to avoid negative repercussions. The model thus predicted that darker-skinned black men who lived in high stress areas and suppressed hostility would have the highest blood pressures. Research results have been generally consistent with these predictions, although the strength of the anger expression and suppression effect has been found to be modest (Schum et al. 2003).

The John Henryism hypothesis (James et al. 1983) is named for the mythic black steel driver who, in the face of seemingly insurmountable odds, refused to be deterred in his efforts. In a series of studies, James found that persons in the black community who exhibit this tenacious and active coping style have higher blood pressure and a higher prevalence of hypertension if they also have fewer resources, such as higher educational attainment, for achieving their goals. While the findings in research on John Henryism have generally been consistent, there have been negative findings, and in some research the association has been obscured by other factors (James 1994) or was found to be contingent on variables such as gender (Dressler et al. 1998a). This suggests that the John Henryism model may be in need of further specification and elaboration.

Another example of this line of research is the study of job-related stressors and incident-treated hypertension by Levenstein and colleagues (2001). This study is relevant because institutional racism will result in individual-level factors such as differential job security for blacks and whites, which in turn may account for differences in disease rates. Levenstein et al. (2001) found that job insecurity and unemployment were potent risks for developing hypertension over a 20-year

follow-up, especially for men; however, as in other types of research, controlling for these factors did not substantially reduce the differences in risk between blacks and whites.

Oths et al. (2001) used a similar line of reasoning in the development of a model to account for ethnic differences in low birth weight. They adapted Karasek's model of job strain, which, in its original form, was developed to examine job stressors among white-collar workers. The central hypothesis of this model is that stress results from the imbalance of demands on the job and the resources a worker has to deal with those demands (see Markovitz et al. 2004). Oths et al. (2001) adapted this model to be appropriate for poor and working-class black and white women in Alabama who work in entry-level service jobs (e.g., fast-food restaurants and convenience stores) and in factories such as poultry processing, as well as for middle-class women in white-collar jobs. Prospectively, they found that women under high demands on the job who had little control (in terms of being able to take a break, for example) had lower birth weight babies and that the effect was enhanced for black women versus white women. The differences in birth weights between black and white women under low strain conditions were minimal; as job strain increased, the gap between black and white birth weights widened. Those who felt discriminated against on the job were nearly three times more likely to suffer job strain as those not discriminated against.

Finally, studies by Dressler (1990, 1991a) are relevant here. Like James's studies of John Henryism, Dressler eschewed the attempt to account for racial or ethnic differences in disease risk, focusing instead on factors within the African American community. On the basis of ethnographic observations, he adapted the concept of status incongruity, arguing that individuals' struggles to achieve a middle-class lifestyle in the face of limited economic resources would be a potent stressor. At the same time, traditional features of social organization in the

black community, especially reliance on the extended family for social support, would moderate that stressor. He found that the interaction of status incongruence and social support was associated with blood pressure within a Southern black community; however, the interaction of kin support and status incongruence was significant only for older (>45 years) respondents. For younger respondents, nonkin support buffered the pressor effect of status incongruence.

Studies of psychosocial stress processes represent a conceptual advance over racial-genetic, health behavior, and SES models in one important sense: In the psychosocial stress model, there is an explicit attempt to integrate, at least on some level, what is unique about the experience of the African American community in the United States and how that singular experience generates a particular configuration of stressors that in turn is associated with health and disease. At the same time, psychosocial stress models to explain racial and ethnic health disparities are subject to all of the same criticisms of the conventional stress model. First, the measurement of many stressors, especially perceived racism, suffers from all the difficulties of distinguishing the accurate perception of a stressor from cognitive and emotional efforts on the part of an individual to cope with that stressor (Meyer 2003), which can lead to complex interpretations of results (Krieger 1990). Second, despite considerable refinement over the years, this emphasis on the individual and individual perception tends to deflect attention away from broader social and cultural fields that generate stressors in the first place.

The Structural-Constructivist Model

The next approach to the study of racial and ethnic disparities will be referred to as a structural-constructivist model, following Bourdieu (1990) and Dressler (2001). This approach to research can be distinguished from previous approaches on several levels. First,

it takes into account explicitly the dual nature of human existence. On one hand, what is taken to be the reality of life is in large part a cognitive representation, constructed out of an amalgam of socially shared understandings distributed within a society (this is termed a constructivist perspective). On the other hand, individuals are constrained by external structures in which they are embedded, especially the ecology of social relationships created by the shared and distributed expectations of others. Social, psychological, and biological processes occur within this intersection of social structure and cultural construction.

The need for such a perspective can be found throughout the literature on racial and ethnic health disparities in the repeated assertion that race is a socially or culturally constructed concept (Krieger 2003); however, the study of how racial and ethnic categories are constructed, and the implication for health of those constructions, is rarely attempted. Furthermore, the importance of a constructivist perspective can be carried a step further. The logic underlying most research on health disparities is that finding and controlling for the ways in which blacks and whites differ will undo observed disparities. This logic seems unremarkable until examined further, as Kaufman & Cooper (1999) have done. These investigators argue that regarding race or ethnicity per se as having true causal potential is misplaced because the logical counterfactual argument cannot be made. The counterfactual argument asks what the effect of race or ethnicity would be if everyone who is “black” became “white,” and vice-versa. Kaufman & Cooper suggest that this argument makes sense for some variables such as obesity because persons who are overweight can logically be envisioned to lose weight and those who are thin can be imagined to gain weight. In what sense can race or ethnicity be imagined to fit this counterfactual argument? Kaufman & Cooper argue that race or ethnicity is such a dominant status category in the United States that the counterfactual logic

fails because every aspect of life is dominated so completely by racial or ethnic status, from birth to death, that entering race or ethnicity into a statistical analysis cannot be readily causally interpreted (see Berk 2004, pp. 81–103, for a similar but more general discussion of causal logic and categories such as race or ethnicity).

Krieger (2003, p. 196) argues that the notion of “exchangeability” denied by Kaufman & Cooper does in fact exist because it is possible to imagine a situation in which people, irrespective of their skin color, are not subject to the same racist interactions or, in keeping with the true counterfactual, in which the racist interactions would be reversed. But perhaps the issue here is not the subtleties of counterfactual logical rigor, but rather the ethnographic realities; that is, is it reasonable to assume that the same understanding or meaning of social interaction can be extended to persons in different racial or ethnic groups? Probably not. Take for example the phenomenon of DWB (driving while black; see West 1993). The likelihood and implication of being black and being stopped by police while driving in a white neighborhood are different, for example, than being white and being stopped by police while driving in a black neighborhood. In this example, and in many others we suspect, the counterfactual argument fails. These are simply incommensurate phenomena.

Again, the issue is not so much the rigor of causal inference as it is how to examine the phenomenon at hand. It is, perhaps, more important to understand, in an ethnographically nuanced way, how the goals and aspirations that structure mundane social interaction are constructed within racial and ethnic groups, and how these cultural constructions collide with the social structure in which they are played out.

A number of studies have employed this perspective. For example, Dressler and associates (Dressler 1991b, 1999; Dressler et al. 1998b, 1999; Dressler & Bindon 2000) used this logic in studies of blood pressure in Brazil and the United States. These investigators

examined the everyday goals and aspirations for a good life shared within communities. In Brazil, both Afro-Brazilians and white Brazilians were included, and a general cultural consensus on a lifestyle (combining material goods and social behavior) representing a good life was found in two separate studies. In the United States, research was carried out exclusively within a Southern black community, and a general cultural consensus on what constituted a good life was also found. In each setting, a lifestyle of domestic comfort, not conspicuous consumption, was the consensus model, and this notion of an inconspicuous consumption was also emphasized in narrative data. Then, the degree to which individuals were able in their own behaviors to approximate this valued lifestyle was examined, which is referred to as cultural consonance in lifestyle. Individuals who were able to approximate better the valued lifestyle had lower blood pressure. Furthermore, in Brazil, there was an interaction effect between cultural consonance and skin color, such that the persons with darker skin color and higher cultural consonance had blood pressures lower than white Brazilians at any level of cultural consonance. In the United States, these researchers found a similar interaction effect between cultural consonance and skin color within the African American community, but this interaction effect was specific to men aged 25–44. These results suggest that the race of an individual is malleable and subject to interpretation in a given social field. Specifically, where individuals can present themselves in mundane social interaction as having achieved widely shared goals for socioeconomic attainment, in the way that these goals are encoded in culturally constructed lifestyles, the biosocial significance of skin color recedes.

Gravlee (2002, 2005; Gravlee & Dressler 2005) extended this logic in a study of skin color and blood pressure in Puerto Rico. An earlier paper (Costas et al. 1981) showed that higher blood pressure was associated with darker skin color in Puerto Rico, after controlling for a variety of other factors. Gravlee

investigated the cultural construction of skin color in Puerto Rico using cultural domain analysis and cultural consensus analysis. Using a standardized set of facial drawings derived from Harris's (1970) work in Brazil, he found that respondents agreed on the allocation of phenotypic descriptors to standardized faces, and, like Harris in Brazil, he found that these attributions were a function of skin color and hair type. He then investigated the association of skin color and blood pressure using distinct measures of skin color that included (a) direct measurement of skin pigmentation by reflectance spectrophotometry, (b) self-rated skin color on a nine-point scale, and (c) an estimate of ascribed color derived from linking survey respondents to the cultural model of skin color, as determined by cultural consensus analysis. In bivariate and multivariate analyses, skin reflectance was not associated with blood pressure; however, both self-rated and ascribed color were associated with blood pressure through interactions with SES. Individuals in lower SES groups had similar blood pressures irrespective of their attributed skin color category, whereas individuals in higher SES groups had higher blood pressures if they were also assigned to the category "black," according to the cultural consensus model (Gravlee 2002). Gravlee & Dressler (2005) also report that the discrepancy between self-rated color and skin pigmentation is associated with blood pressure through an interaction with SES. They interpret this finding as a status incongruity effect.

These studies take seriously the idea of the cultural construction of race or ethnicity, as well as how life goals are culturally constructed within communities of color and the implications for health when those goals are limited by racial stratification.

RESEARCH NEEDS

To this point, we have used the terms race or ethnicity in the same way they are generally employed in the literature, that is, without attempting to define them. The lack of explicit

definitions is widely recognized as a significant barrier to progress in research on health disparities. As Crews & Bindon (1991) point out, biomedical researchers are expected to define key concepts and to establish the validity and reliability of measurement operations, but the same researchers routinely assign participants to racial or ethnic categories without further comment (see also Hahn & Stroup 1994). This pattern obscures health researchers' responsibility to distinguish what they know about race as biological scientists from what they know about race as enculturated members of society, and it illustrates how much the reality of race is taken for granted in the United States. More important, it impedes efforts to understand the causes of health disparities among racially defined groups. When race is treated as a proxy for some unspecified combination of environmental, behavioral, and genetic factors, rigorous tests of the precise causal mechanisms involved are the exception, not the rule. For research to progress, a conceptual model of race and ethnicity is required; indeed, there is some movement in the biomedical literature to require the definition of race and ethnicity, and to specify their relevance to the study, when the terms are used in publication (Davidoff 2000). We examine some of the ways in which such a conceptual model might be developed and operationalized.

We assume that the term race has no universal biological referent when applied to the human species (Kittles & Weiss 2003); therefore, as it is asserted frequently in the literature, race is a cultural construct used by members of a society to explain perceived biological differences among humans in specific ethnographic settings. That being the case, the most suitable terms for use in research would be "ethnoracial categories," or perhaps "folk racial categories." It is then incumbent on researchers to demonstrate, in any given setting, the cultural model that generates ethnoracial categories and how these categories are employed in that ethnographic context. Research models exist, notably

Ethnoracial categories: culturally constructed or folk categories that denote essential differences conceived in terms of biological ancestry

Harris (1970), Harris et al. (1993), Byrne et al. (1995), and Gravlee (2005). Using elicitation techniques from cognitive anthropology, these researchers have systematically described ethnoracial descriptors where they are relevant and demonstrated how those descriptors are or can be applied in specific ethnographic contexts. These techniques, especially when combined with intensive ethnographic methods, can provide a clear picture of the folk racial model when the concept is employed in a given setting (e.g., the link of Harris's work with Burdick's (1998) in Brazil). Then, in epidemiologic surveys, when an individual self-selects an ethnoracial category, or when such an ethnoracial category is applied to an individual, the real sociocultural import of that attribution can be understood (Gravlee 2005). Although it may seem like a daunting task to include such an ethnographically detailed analysis in every study, it is probably unnecessary to replicate these analyses each time health research is conducted in that setting. It is more important that researchers are aware that folk racial categories are emic, ethnographically contingent constructs, and that they make every effort to link their specific operational procedures to what is understood about prevailing ethnoracial models in that specific setting (e.g., Dressler et al. 1999).

In contrast, the term ethnicity can be applied universally as an analytic construct. The ethnographic record suggests that there may be a universal tendency to differentiate individuals and social groups on the basis of factors generally associated with attributions of traditions and ancestry and the way in which those attributions are realized in the present (Gil-White 2001). Ultimately, ethnicity becomes a fundamental way to define social boundaries (Barth 1969). In a sense, ethnicity becomes a primary category in the analysis of any society. It is a category of sociocultural systems analysis on par with the economic system, the kinship system, or the system of religious belief and practice. Elaborated further, ethnicity is a social institution like kinship or marriage, but a higher-level one in that, as an

organizing tool that assigns identity to members of a group, it actually incorporates, or is partly constituted by, aspects of other institutions (kinship, economy). To be sure, ethnic group differences are culturally constructed within any given society. But what separates ethnic group differences and ethnoracial differences is that ethnic group differences may or may not include a folk racial component. The use of the term race, in contrast, demands that an ethnobiological theory (or folk model of essential biological difference) be demonstrated.

Elements of a definition of ethnicity have been offered by many, notably Montagu (1962), Barth (1969), Dominguez (1986), Crews & Bindon (1991), Gaines (2005), Oths (1999), and Gravlee (2005). The definition of ethnic groups within a society will incorporate any of a number of dimensions that can be placed into three broad categories—the cultural, the ancestral, and the referential—the salient features of which will vary between groups. The cultural includes shared models for both the mundane (e.g., language use, diet, dress, marriage rituals) and the more abstract (e.g., concepts of self, supernatural beliefs) aspects of life. A sense of shared ancestry includes territorial homeland, common history (which may include ethnoracial discrimination), and kinship (whether construed biologically), which may or may not incorporate phenotypic or genotypic characteristics such as hair type, body build, or skin tone. With respect to the referential, as ethnic group labels fundamentally separate people into in- and out-groups (i.e., “we” versus “they”), personal (or self-defined) and social (or other-defined) identity is an integral component of ethnic definition. Folk racial categories may then be indexed here, when relevant, as an emic self-categorization, or as an eticly imposed descriptor used by others.

In summary, rather than retaining the term race with any kind of etic biological connotation, it can be seen as a part of a meaningful folk taxonomy that may (if present)

be incorporated into a more robust classification with worldwide applicability. Therefore, instead of finding that “current racial categories capture ethnic status,” as Williams (1997, p. 325) holds, we prefer a model in which ethnic categories would subsume ethnoracial categories.

It should be clear that the scheme we suggest here invites the study of genetic factors in the distribution of disease, in terms of the explicit identification of genetic variants. Such a scheme would distinguish the distribution of genetic variants, folk racial categories, and ethnicity; each term would carve out a distinctive phenomenon for analysis in a theoretically and operationally explicit way. We might then be able, for example, to describe the distribution of disease separately in relation to genetic variants, ethnoracial categories, and ethnicity, and in relation to the combination of these factors. Furthermore, the associations of genetic variants, ethnoracial categories, and ethnicity can themselves become a focus of study. More to the point, however, our review of existing literature suggests that these three dimensions of human biology and social life have been routinely offered as risk factors for disease, while simultaneously being routinely conflated. The proposed scheme identifies factors believed important in existing literature and offers the potential to distinguish among them in future research.

CONCLUSION

As shown in quantitative analyses of the social science and public health literature, ethnoracial categories and ethnicity have become nearly standard variables included in research on the distribution of disease. And, on nearly every indicator, but especially on several of the most important contributors to early mortality, black Americans and white Americans differ; black Americans suffer higher mortality and morbidity. These comparisons have, however, generated little understanding of these health disparities because they

SOCIAL STRUCTURE AND CULTURAL CONSTRUCTIVISM

The dual nature of human existence has been prominent in the history of social theory. Other terms could easily be substituted for social structure and cultural construction, but the basic issue has remained the same for more than 200 years: Is human behavior fundamentally a function of the external constraints imposed on groups and individuals, or is it a function of the way in which groups and individuals impose a meaningful structure on the world? Although most working social scientists probably understand their subject matter as an interaction of these aspects of human life, surprisingly little formal theory has tried to integrate the two. An exception to this is the earlier work of Pierre Bourdieu, who proposed a set of constructs (e.g., “habitus,” “social field,” “cultural space”) in which human behavior could be understood at the intersection of social structure and cultural construction. This is a particularly fruitful approach in the study of health disparities because it draws attention to the cultural construction of, for example, racial categories, while being sensitive to the causal potential of these arbitrary categories when there is a collective acceptance of their “reality.”

are reported mostly without comment or, perhaps worse, with vague comments implicating racial-genetic differences. A careful review of the literature indicates that such imputing of a racial-genetic basis for disease is without foundation, yet such suggestions cannot but reinforce the general American cultural model of ethnoracial categories.

Other studies have sought the nonracial-genetic basis for these differences, and research on discrete health behaviors, although important in part to be sure, suggests that these cannot account for overall group differences. What appears to offer a more potent explanation for ethnoracial and ethnic health disparities is a model in which other forms of inequality, especially social and economic inequalities, generate life conditions that are chronically stressful over the life course of black Americans. What Geronimus (1992) refers to as “weathering,” or the chronic, allostatic load generated by this continuing

adaptation to enduring structures of inequality, then generates observed health disparities. It is a collision of the cultural construction of mundane life goals with a social structure of ethnoracial stratification.

Understanding this process in terms of all its empirical complexity ultimately will require a reconceptualization of key factors in the process, notably the basic concepts of folk racial categories and ethnicity.

ACKNOWLEDGMENTS

The authors have benefited over the years from discussion of these issues with Russ Bernard, Jim Bindon, Woody Gaines, Sherman James, and the late Marvin Harris. The authors alone are responsible for any errors.

SUMMARY POINTS

1. Differences in morbidity and mortality between conventionally defined racial and ethnic groups have been widely documented, but these differences continue to be poorly understood.
2. Although overall racial and ethnic group differences are complex, the largest disparities are those between African Americans and European Americans. On virtually every indicator of morbidity and mortality, blacks suffer in relation to whites.
3. A review of the literature on health disparities reveals five types of explanatory models that have been employed to account for these differences; each explanatory model emphasizes different sets of variables.
4. Models that emphasize both psychological and sociocultural factors in the causes of health disparities appear to be most promising.
5. Future progress in this area will depend on the development of a satisfactory theory of ethnic differences.

FUTURE DIRECTIONS/UNRESOLVED ISSUES

1. The conceptualization and measurement of the social and cultural dimensions of race and ethnicity need to be improved.
2. More cross-cultural research is needed to accumulate empirical results outside the specific sociocultural constraints of North American and Western European societies.
2. The degree to which ethnoracial and other traits construct ethnicity in a given group is an empirical question that should be examined cross-culturally.
4. Future research needs to specify tests of hypotheses that include measures of both genetic and nongenetic differences between ethnic groups.

LITERATURE CITED

- Acevedo-Garcia D, Soobader MJ, Berkman LF. 2005. The differential effect of foreign-born status on low birth weight by race/ethnicity and education. *Pediatrics* 115:20–30
- Almeida-Filho N, Kawachi I, Filho AP, Dachs JWD. 2003. Research on health inequalities in Latin America and the Caribbean. *Am. J. Public Health* 93:2037–43
- Barker DJP. 2004. The developmental origins of well-being. *Philos. Trans. R. Soc. London Ser. B* 259:1359–66
- Barth F. 1969. *Ethnic Groups and Boundaries: The Social Organization of Culture Difference*. Boston, MA: Little, Brown
- Bassett DR, Futzhugh EC, Crespo CJ, King GA, McLaughlin JE. 2002. Physical activity and ethnic differences in hypertension prevalence in the United States. *Prev. Med.* 34:179–86
- Bell AC, Adair LS, Popkin BM. 2004. Understanding the role of mediating risk factors and proxy effects in the association between socio-economic status and untreated hypertension. *Soc. Sci. Med.* 59:275–83
- Berk RA. 2004. *Regression Analysis: A Constructive Critique*. Thousand Oaks, CA: Sage
- Bourdieu P. 1990. In *Other Words: Essays Towards a Reflexive Sociology*. Transl. M Adamson. Stanford, CA: Stanford Univ. Press (From French)
- Bouzekri N, Zhu X, Jiang Y, McKenzie CA, Luke A, et al. 2004. Angiotensin I-converting enzyme polymorphisms, ACE level and blood pressure among Nigerians, Jamaicans and African-Americans. *Eur. J. Hum. Genet.* 12:460–68
- Boyle E. 1970. Biological patterns in hypertension by race, sex, body weight, and skin color. *JAMA* 213:1637–43
- Braun L. 2002. Race, ethnicity, and health: Can genetics explain disparities? *Perspect. Biol. Med.* 45:159–74
- Brondolo E, Rieppi R, Kelly KP, Gerin W. 2003. Perceived racism and blood pressure: a review of the literature and conceptual and methodological critique. *Ann. Behav. Med.* 25:55–65
- Brownley KA, Hurwitz BE, Schneiderman N. 1999. Ethnic variations in pharmacological and nonpharmacological treatment of hypertension. *Hum. Biol.* 71:607–40
- Burdick J. 1998. *Blessed Anastácia: Women, Race, and Popular Christianity in Brazil*. New York: Routledge
- Byrne B, Harris M, Consorte JG, Lang J. 1995. What's in a name? The consequences of violating Brazilian emic, color-race categories in estimates of social well-being. *J. Anthropol. Res.* 51:389–97
- Carlos Poston WS, Pavlik VN, Hyman DJ, Ogbonnaya K, Hanis CL, et al. 2001. Genetic bottlenecks, perceived racism, and hypertension risk among African Americans and first-generation African immigrants. *J. Hum. Hypertens.* 15:341–51
- Cent. Dis. Control. 2004a. *National Center for Health Statistics*. <http://www.cdc.gov/nchs/data/hus/tables/2003/03hus066.pdf>
- Cent. Dis. Control. 2004b. *National Center for Health Statistics*. http://www.cdc.gov/nchs/fastats/pdf/nvsr52_10t43.pdf
- Cent. Dis. Control. 2004c. *National Center for Health Statistics*. <http://www.cdc.gov/nchs/data/hus/04trend.pdf#069>
- Chrysant SG, Weir MR, Weder AB, McCarron DA, Canoss-Terris M, et al. 1997. There are no racial, age, sex, or weight differences in the effect of salt on blood pressure in salt-sensitive hypertensive patients. *Arch. Int. Med.* 157:2489–94
- Clark R, Anderson NB, Clark VR, Williams DR. 1999. Racism as a stressor for African Americans: a biopsychosocial model. *Am. Psychol.* 54:805–16

A historically important paper because it was an early challenge to conventional racial thinking in the professional biomedical literature, drawing especially on the anthropological work of Ashley Montagu.

Researchers demonstrate unambiguously that groups of African descent exhibit different rates of hypertension in different environmental settings, thus challenging the notion that hypertension is under exclusive racial-genetic control.

- Collins JW Jr, David RJ, Handler A, Wall S, Andes S. 2004. Very low birthweight in African American infants: the role of maternal exposure to interpersonal racial discrimination. *Am. J. Public Health* 94:2132–38
- Comstock RD, Castillo EM, Lindsay SP. 2004. Four-year review of the use of race and ethnicity in epidemiologic and public health research. *Am. J. Epidemiol.* 159:611–19
- Cooper RS. 1984. A note on the biologic concept of race and its application in epidemiologic research. *Am. Heart J.* 108:715–23
- Cooper RS, Rotimi C, Ataman S, McGee D, Osotimehin B, et al. 1997. The prevalence of hypertension in seven populations of West African origin. *Am. J. Public Health* 87:160–68
- Cooper RS, Rotimi C, Ward R. 1999. The puzzle of hypertension in African-Americans. *Sci. Am.* 280:56–63
- Costas R Jr, Garcia-Palmieri MR, Sorlie P, Hertzmark E. 1981. Coronary heart disease risk factors in men with light and dark skin in Puerto Rico. *Am. J. Public Health* 71:614–19
- Crews DE, Bindon JR. 1991. Ethnicity as a taxonomic tool in biomedical and biosocial research. *Ethn. Dis.* 1:42–49
- Crews DE, Williams SR. 1999. Molecular aspects of blood pressure regulation. *Hum. Biol.* 71:475–504
- Curtin PD. 1992. The slavery hypothesis for hypertension among African Americans: the historical evidence. *Am. J. Public Health* 82:1681–86
- Daniel HI, Rotimi CN. 2003. Genetic epidemiology of hypertension: an update on the African diaspora. *Ethn. Dis.* 13:S53–66
- Davey Smith G. 2000. Learning to live with complexity: ethnicity, socioeconomic position, and health in Britain and the United States. *Am. J. Public Health* 90:1694–98
- David RJ. 2001. Birthweights and bell curves. *Int. J. Epidemiol.* 30:1241–43
- David RJ, Collins JW Jr. 1997. Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. *N. Engl. J. Med.* 337:1209–14
- Davidoff F. 2000. News from the international committee of medical journal editors. *Ann. Intern. Med.* 133:229–31
- Davidson K, Jonas BS, Dixon KE, Markovitz JH. 2000. Do depressive symptoms predict early hypertension incidence in young adults in the CARDIA study? *Arch. Intern. Med.* 160:1495–500
- Din-Dzietham R, Nembhard WN, Collins R, Davis SK. 2004. Perceived stress following race-based discrimination at work is associated with hypertension in African-Americans. *Soc. Sci. Med.* 58:449–61
- Dominguez VR. 1986. *White by Definition: Social Classification in Creole Louisiana*. New Brunswick, NJ: Rutgers Univ. Press
- Dressler WW. 1990. Lifestyle, stress, and blood pressure in a Southern black community. *Psychosom. Med.* 52:182–98
- Dressler WW. 1991a. Social support, lifestyle incongruity, and arterial blood pressure in a Southern black community. *Psychosom. Med.* 53:608–20
- Dressler WW. 1991b. Social class, skin color, and arterial blood pressure in two societies. *Ethn. Dis.* 1:60–77
- Dressler WW. 1993. Health in the African-American community: accounting for health inequalities. *Med. Anthropol. Q.* 7:325–45
- Dressler WW. 1999. *Culture, skin color and arterial blood pressure*. Presented at Annu. Meet. Am. Anthropol. Assoc., 98th, Chicago
- Dressler WW. 2001. Medical anthropology: toward a third moment in social science? *Med. Anthropol. Q.* 15:455–65

- Dressler WW, Balieiro MC, Santos JED. 1999. Culture, skin color, and arterial blood pressure in Brazil. *Am. J. Hum. Biol.* 11:49–59
- Dressler WW, Bindon JR. 2000. The health consequences of cultural consonance: cultural dimensions of lifestyle, social support and arterial blood pressure in an African American community. *Am. Anthropol.* 102:244–60
- Dressler WW, Bindon JR, Neggers YR. 1998a. John Henryism, gender, and arterial blood pressure in an African American community. *Psychosom. Med.* 60:620–24
- Dressler WW, Bindon JR, Neggers YR. 1998b. Culture, socioeconomic status, and coronary heart disease risk factors in an African American community. *J. Behav. Med.* 21:527–44
- Drevdahl D, Taylor JY, Phillips DA. 2001. Race and ethnicity as variables in *Nursing Research, 1952–2000*. *Nurs. Res.* 50:305–13
- Dyer AR, Liu K, Walsh M, Kiefe C, Jacombs DR Jr, Bild DE. 1999. Ten-year incidence of elevated blood pressure and its predictors. *J. Hum. Hyperten.* 13:13–20
- Ellison GTH, de Wet T. 1997. The use of ‘racial’ categories in contemporary South African health research. A survey of articles published in the *South African Medical Journal* between 1992 and 1996. *S. Afr. Med. J.* 87:1671–79
- Estruch R, Coca A, Rodicio JL. 2004. High blood pressure, alcohol, and cardiovascular risk. *Eur. Soc. Hyperten. Sci. Newsl.* 5:1–3
- Fuchs FD, Chambless LE, Whelton PK, Nieto FJ, Heiss G. 2001. Alcohol consumption and the incidence of hypertension. *Hypertension* 37:1242–50
- Gaines AD. 2005. Race: Local biology and culture in mind. In *Companion to Psychological Anthropology*, ed. C Casey, RB Edgerton, pp. 255–78. Oxford: Blackwell
- Geronimus AT. 1992. The weathering hypothesis and the health of African-American women and infants. *Ethn. Dis.* 2:207–21
- Gil-White FJ. 2001. Are ethnic groups “biological species” to the human brain? Essentialism in our cognition of some social categories. *Curr. Anthropol.* 42:515–54
- Gravlee CC. 2002. *Skin color, blood pressure, and the contextual effect of culture in southeastern Puerto Rico*. PhD thesis. Univ. Fla., Gainesville
- Gravlee CC. 2005. Emic ethnic classification in southeastern Puerto Rico: cultural consensus and semantic structure. *Soc. Forces* 83:949–70
- Gravlee CC, Dressler WW. 2005. Skin pigmentation, self-perceived color, and arterial blood pressure in Puerto Rico. *Am. J. Hum. Biol.* 17:195–206
- Gravlee CC, Sweet E, Abts M. 2004. *Race, ethnicity, and racism in medical anthropology, 1977–2002*. Presented at Soc. Appl. Anthropol., Dallas, TX
- Grim CE, Robinson M. 1996. Blood pressure variation in blacks: genetic factors. *Semin. Nephrol.* 16:83–93**
- Hahn R, Stroup DF. 1994. Race and ethnicity in public health surveillance: criteria for the scientific use of social categories. *Public Health Rep.* 109:7–15
- Harburg EL, Erfurt JC, Hauenstein LS. 1973. Socioecologic stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosom. Med.* 35:276–96
- Harburg E, Gleiberman L, Roeper P, Schork MA, Schull WJ. 1978. Skin color, ethnicity, and blood pressure I: Detroit blacks. *Am. J. Public Health* 68:1177–83
- Harrap SB. 2003. Where are all the blood pressure genes? *The Lancet* 361:2149–51
- Harris M. 1970. Referential ambiguity in the calculus of Brazilian racial identity. *Southwest. J. Anthropol.* 26:1–14
- Harris M, Consorte JG, Lang J, Byrne B. 1993. Who are the whites—imposed census categories and the racial demography of Brazil. *Soc. Forces* 72:451–62
- Homer CJ, Beresford SAA, James SA, Siegel E, Wilcox S. 1990. Work-related physical exertion and risk of preterm, low birthweight delivery. *Paediatr. Perinat. Epidemiol.* 4:161–74

An articulation of the so-called “slavery hypothesis” that posits a racial-genetic propensity to hypertension among African Americans.

An early paper articulating a nongenetic hypothesis to account for black-white differences in blood pressure, notable for its sensitivity to the ethnographic context.

A critique of the so-called “slavery hypothesis” for hypertension, notable in the way that it traces the intellectual diffusion of a hypothesis that has little empirical support.

- Inst. Med. Comm. Nutr. Status During Pregnancy Lactation. 1990. *Nutrition During Pregnancy: Part I: Weight Gain. Part II: Nutrient Supplements*. Washington, DC: Natl. Acad. Press
- Jackson FL. 1991. An evolutionary perspective on salt, hypertension, and human genetic variability. *Hypertension* 17:1129–32
- James SA. 1994. John Henryism and the health of African Americans. *Cult. Med. Psychiatry* 18:163–82
- James SA, Hartnett SA, Kalsbeek WD. 1983. John Henryism and blood pressure differences among black men. *J. Behav. Med.* 6:259–78**
- Janzon E, Hedblad B, Berglund G, Engstrom G. 2004. Changes in blood pressure and body weight following smoking cessation in women. *J. Intern. Med.* 255:266–72
- Jonas BS, Franks P, Ingram DD. 1997. Are symptoms of anxiety and depression risk factors for hypertension? *Arch. Fam. Med.* 6:43–49
- Jonas BS, Lando JF. 2000. Negative affect as a prospective risk factor for hypertension. *Psychosom. Med.* 62:188–96
- Jones CP, LaVeist TA, Lillie-Blanton M. 1991. “Race” in the epidemiologic literature: an examination of the *American Journal of Epidemiology*, 1921–1990. *Am. J. Epidemiol.* 134:1079–84
- Jones-Webb R, Jacombs DR, Flack JM, Liu K. 1996. Relationships between depressive symptoms, anxiety, alcohol consumption, and blood pressure. *Alcohol. Clin. Exp. Res* 20:420–27
- Kaplan JB, Bennett T. 2003. Use of race and ethnicity in biomedical publication. *JAMA* 289:2709–16
- Kaufman JS, Cooper RS. 1999. Seeking causal explanations in social epidemiology. *Am. J. Epidemiol.* 154:291–98
- Kaufman JS, Cooper RS, McGee DL. 1997. Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. *Epidemiology* 8:621–28
- Kaufman JS, Hall SA. 2002. The slavery hypertension hypothesis: dissemination and appeal of a modern race theory. *Epidemiology* 14:111–18**
- Keil JE, Tyroler HA, Sandifer SH, Boyle E Jr. 1977. Hypertension: effects of social class and racial admixture. *Am. J. Public Health* 67:634–39
- Keppel KG, Pearch JN, Wagener DK. 2002. Trends in racial and ethnic-specific rates for the health status indicators: United States, 1990–98. *Healthy People Statistical Notes No. 23*. Hyattsville, MD: Natl. Cent. Health Stat.
- Kittles RA, Weiss KM. 2003. Race, ancestry, and genes: implications for defining disease risk. *Annu. Rev. Genomics Hum. Genet.* 4:33–67
- Klag MJ, Whelton PK, Coresh J, Grim C, Kuller LH. 1991. The association of skin color with blood pressure in U.S. blacks with low socioeconomic status. *JAMA* 265:599–602
- Kleinman JC, Fingerhut LA, Prager K. 1991. Differences in infant mortality by race, nativity status, and other maternal characteristics. *Am. J. Dis. Child.* 145:194–99
- Knox SS, Hausdorff J, Markovitz JH. 2002. Reactivity as a predictor of subsequent blood pressure. *Hypertension* 40:914–19
- Krieger N. 1990. Racial and gender discrimination: risk factors for high blood pressure? *Soc. Sci. Med.* 30:1273–81
- Krieger N. 1999. Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int. J. Health Serv.* 29:295–352
- Krieger N. 2003. Does racism harm health? Did child abuse exist before 1982? On explicit questions, critical science, and current controversies: an ecosocial perspective. *Am. J. Public Health* 93:194–99
- Krieger N, Sidney S. 1996. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am. J. Public Health* 86:1370–78

- Levenstein S, Smith MW, Kaplan GA. 2001. Psychosocial predictors of hypertension in men and women. *Arch. Intern. Med.* 161:1341–46
- Liu K, Ruth KJ, Flack JM, Jones-Webb R, Burke G, et al. 1996. Blood pressure in young blacks and whites. *Circulation* 93:60–66
- Markovitz JH, Matthews KA, Whooley M, Lewis CE, Greenlund KJ. 2004. Increases in job strain are associated with incident hypertension in the CARDIA Study. *Ann. Behav. Med.* 28:4–9
- McCarron DA. 2000. The dietary guideline for sodium. *Am. J. Clin. Nutr.* 71:1013–19
- McCormick MC, Brooks-Gunn J, Shorter T, Holmes JH, Wallace CY, Heagarty MC. 1990. Factors associated with smoking in low-income pregnant women: relationship to birth weight, stressful life events, social support, health behaviors and mental distress. *J. Clin. Epidemiol.* 3:441–48
- Meyer IH. 2003. Prejudice as stress: conceptual and measurement problems. *Am. J. Public Health* 93:262–65
- Montagu A. 1962. The concept of race. *Am. Anthropol.* 64:919–28**
- Mustillo S, Krieger N, Gunderson EP, Sidney S, McCreath H, Kiefe CI. 2004. Self-reported experiences of racial discrimination and black-white differences in preterm and low-birthweight deliveries: the CARDIA Study. *Am. J. Public Health* 94:2125–31
- Nickens H. 1986. Health problems of minority groups: public health's unfinished agenda. *Public Health Rep.* 101:230–31
- Oparil S, Zaman MA, Calhoun DA. 2003. Pathogenesis of hypertension. *Ann. Intern. Med.* 139:761–76
- Osborne NG, Feit MD. 1992. The use of race in medical research. *JAMA* 267:275–79
- Oths KS. 1999. *Who's who in Alabama prenatal clinics*. Presented at Annu. Meet. Am. Anthropol. Assoc., 98th, Chicago
- Oths KS, Dunn LL, Palmer NS. 2001. A prospective study of psychosocial job strain and birth outcomes. *Epidemiology* 12:744–46
- Sankar P, Cho MK, Condit CM, Hunt LM, Koenig B, et al. 2004. Genetic research and health disparities. *JAMA* 291:2985–89
- Schoenborn CA, Adams PF, Barnes PM, Vickerie JL, Schiller JS. 2004. *Health Behaviors of Adults: United States, 1999–2001*. Natl. Cent. Health Stat., Vital Health Stat. 10(219). Washington, DC: US GPO
- Schum JL, Jorgensen RS, Verhaeghen P, Sauro M, Thibodeau R. 2003. Trait anger, anger expression, and ambulatory blood pressure: a meta-analytic review. *J. Behav. Med.* 26:395–415
- Sheldon TA, Parker H. 1992. Race and ethnicity in health research. *J. Public Health Med.* 14:104–10
- Sowers JR, Ferdinand KC, Bakris GL, Douglas JG. 2002. Hypertension-related disease in African Americans. *Postgrad. Med.* 112:24–34
- Stamler J, Elliott P, Appel L, Chan Q, Buzzard M, et al. 2003. Higher blood pressure in middle-aged American adults with less education—role of multiple dietary factors. *J. Hum. Hyperten.* 17:655–75
- Starfield B, Shapiro S, Weiss J, Liang KY, Ra K, et al. 1991. Race, family income, and low birth weight. *Am. J. Epidemiol.* 134:1167–74
- Teitelman AM, Welch LS, Hellenbrand KG, Bracken MB. 1990. Effect of maternal work activity on preterm birth and low birth weight. *Am. J. Epidemiol.* 131:104–13
- Townsend P, Davidson N. 1982. *Inequalities in Health: The Black Report*. Harmondsworth, UK: Penguin**

A classic paper that challenges the validity of conventional racial categories.

The analysis of socioeconomic and regional disparities in health in Britain that was central in articulating the issue of health disparities as an important topic of research.

- US Dep. Health Hum. Serv. 2000. *Healthy People 2010: Understanding and Improving Health*. Washington, DC: US GPO. 2nd ed.
- West C. 1993. *Race Matters*. Boston, MA: Beacon
- Wilcox AJ, Russell I. 1990. Why small black infants have a lower mortality rate than small white infants: the case for population-specific standards for birth weight. *J. Pediatr.* 116:7–10
- Williams DR. 1994. The concept of race in *Health Services Research: 1966 to 1990*. *Health Serv. Res.* 29:261–74
- Williams DR. 1997. Race and health: basic questions, emerging directions. *Ann. Epidemiol.* 7:322–33
- Williams DR. 1999. Monitoring racial/ethnic status in the U.S.: data quality issues. *Ethn. Health* 4:121–37
- Williams DR, Collins C. 1995. U.S. socioeconomic and racial differences in health: patterns and explanations. *Annu. Rev. Sociol.* 21:349–86
- Williams DR, Collins C. 2001. Racial residential segregation: a fundamental cause of the racial disparities in health. *Public Health Rep.* 116:404–16
- Williams DR, Neighbors HW, Jackson JS. 2003. Racial/ethnic discrimination and health: findings from community studies. *Am. J. Public Health* 93:200–8
- Wong MD, Shapiro MF, Boscardin WJ, Ettner SL. 2002. Contribution of major diseases to disparities in mortality. *N. Engl. J. Med.* 347:1585–92
- Woolf SH, Johnson RE, Fryer GE Jr, Rust G, Satcher D. 2004. The health impact of resolving racial disparities: an analysis of US mortality data. *Am. J. Public Health* 94:2078–81
- Wyatt SB, Williams DR, Calvin R, Henderson FC, Walker ER, Winters K. 2003. Racism and cardiovascular disease in African Americans. *Am. J. Med. Sci.* 325:315–31
-

RELATED RESOURCES

The Problem of Race

F Boas

Anthropology and Modern Life. 1960. Pages 18–62. New York: Norton

On the Race Concept

CL Brace

Current Anthropology. 1964. Volume 5, pages 313–18

Dusk of Dawn: An Essay Toward an Autobiography of a Race Concept

WEB DuBois

1940. New York: Harcourt, Brace and Co.

Inconsistencies in Coding of Race and Ethnicity Between Birth and Death in US Infants. A New Look at Infant Mortality, 1983 through 1985

RA Hahn, J Mulinare, SM Teutsch

Journal of the American Medical Association. 1992. Volume 267, Issue 2, pages 259–63

Man's Most Dangerous Myth: The Fallacy of Race

A Montagu

1942. New York: Columbia University Press