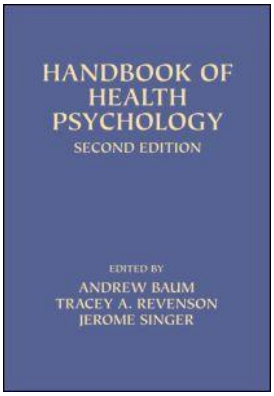

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Racial Disparities in Hypertension and Links Between Racism and Health

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Racial disparities in health continue to be a pressing problem in the United States. What accounts for these racial disparities? Are there race differences in both the individual-level and the community-level health risk factors and health-related behaviors? Do racism and ethnic discrimination contribute, directly or indirectly, to these disparities? To answer these questions, we examine racial disparities in high blood pressure, or hypertension, as a model for thinking about the complex issues driving health disparities in general.

We begin by documenting Black–White differences in the prevalence, awareness, treatment, and control of hypertension. This analysis clarifies the specific nature of the disparities that must be addressed. Next, we review the literature investigating Black versus White differences in the major modifiable risk factors for hypertension, including both individual- and community-level risks. Disparities in these risk factors may contribute to racial disparities in prevalence and could provide focal points for intervention. Yet, as we will demonstrate, studies investigating race differences in modifiable risk factors have significant methodological limitations. More important, without a clear understanding of the determinants of any race differences in risk exposure, simply documenting these race differences will not provide sufficient guidance for the development of risk-reducing interventions to reduce racial disparities in hypertension.

Risk-reducing interventions target the constellation of psychological experiences (e.g., affects, behaviors, and cognitions) and environmental circumstances (e.g., resources and barriers) that contribute to the development of the health-impairing conditions. However, these contributory factors are likely to vary substantially by race and ethnicity, among other factors. We will argue that a portion of the race differences in determinants of health outcomes is a function of differential exposure to racial and ethnic discrimination. In the third section of the chapter, we examine the evidence linking racism to hypertension both directly and by means of associations with each of the modifiable risk factors for hypertension. We consider different levels of racism, including cultural, institutional, interpersonal, and internalized or intrapersonal (Harrell, 2000; Krieger, 1999). Each of these levels may act independently and synergistically with racism at other levels and with associated psychosocial factors (e.g., socioeconomic status, or SES) to affect the development and course of hypertension (Myers, 2009). Understanding the multilevel mechanisms through which racism affects cardiovascular health is essential to help clinicians understand the range of barriers facing individuals from different racial or ethnic groups as they work together to improve health outcomes. This knowledge can promote the development of tailored and, consequentially, more effective integrated treatments for hypertension.

It is important to note that there is little consensus on the best terms to use to distinguish among groups based on phenotypic or cultural characteristics, and both scientific and political factors

influence the debate. Some terms—for example, *Black*—have been used to refer to "racial" groups despite the lack of biological evidence for distinct races; whereas other terms, among them the designation *Latinos*, are usually considered to be labels for ethnic groups but have also been used to refer to "races." In this chapter, we have chosen to use the terms *race* and *ethnicity* and *racism* and *ethnic discrimination* interchangeably to refer to groups based on notions of race, cultural background, and/or ethnicity.

Similarly, there are differences of opinion about the most appropriate terms to use when referring to different ethnic or racial groups. Because we discuss ethnic discrimination as maltreatment based on phenotypic or cultural characteristics, we have chosen the most general categories—that is, the terms *Black* and *Latino*—rather than more specific subcategories when referring to the participants' ethnic and racial groupings.

Our review focuses on the differences between African Americans and White Americans, because this has been the primary focus of empirical research on racial disparities in hypertension (HTN). However, it is important to note that there are also significant disparities for other ethnic groups (e.g., Angell, et al., 2008; Burt, et al., 1995; Glover, Greenlund, & Ayala, 2002; H. Kramer et al., 2004; Zhao, Ford, & Mokdad, 2008). Recently, investigators have also identified variations among subgroups within larger ethnic or racial categories (e.g., subgroups of Asians or Latinos/Latinas; Rosamond et al., 2008)

DOCUMENTING DISPARITY: VARIATIONS IN THE PREVALENCE, AWARENESS, TREATMENT, AND CONTROL OF HYPERTENSION

Estimates of the prevalence of hypertension in Blacks and Whites in the United States have been obtained through major epidemiological studies conducted using national or regional samples. At any given time, race-group variations in hypertension prevalence may be a function of differences in underlying pathology and symptoms presentation, as well as variations in access to diagnosis and treatment, in adherence to prevention and treatment strategies, and in response to treatment. Therefore, we will examine available data on each of these issues separately.

Although there have been variations in the standards used to consider individuals eligible for treatment, the major epidemiological studies have generally considered an individual to meet criteria for current hypertension if his or her clinic blood pressure (BP) is at or above 140/90 mmHg, or if he or she reports receiving antihypertensive treatment. National studies examining prevalence rates for hypertension among adults (> = 20 years of age) consistently report substantially higher rates for Blacks than Whites. Across studies, the prevalence rates for Whites range from 24.4% to 29%; whereas the prevalence rates for Blacks range from 30.6% to 40.5% (Borrell, Crawford, Barrington, & Maglo, 2008; Fiscella & Holt, 2008; Glover et al., 2002; Glover, Greenlund, Ayala, & Croft, 2005; Hajjar & Kotchen, 2003). Black–White differences in the prevalence of hypertension have actually increased over time for men. For example, comparisons of prevalence rates from NHANES III (collected between 1988 and 1994) and NHANES IV (collected between 1999 and 2004) indicate that the prevalence of hypertension increased for all groups, but the effects were significant for Black and White women and for Black but not White men (Cutler et al., 2008).

Black–White differences in BP levels emerge at an early age, prior to concerns about treatment adherence or treatment response. Studies of children and adolescents indicate race-group discrepancies as early as 8 years of age (Muntner et al., 2006). Among every age group, BP levels of Black individuals are higher than those of White individuals (Muntner et al., 2006). The effects of persistently elevated BP on overall health accumulate over time, with hypertension-related illnesses serving as a significant contributor to the gap in lifespan between Blacks and Whites in the United States (Fiscella & Holt, 2008). In addition, long-term differences in BP may contribute to changes in brain function that in turn modify response to antihypertensive treatment (Jennings & Zanstra, 2009).

Efforts to reduce BP depend on being aware of one's hypertensive status. Recent data indicate that Blacks are more likely than Whites to be aware of their hypertensive status. Rates of awareness

in Blacks range from 66.7% to 85.5%, whereas rates of awareness in Whites range from 62.9% to 82.4% (Angell et al., 2008; Glover et al., 2002; Hajjar & Kotchen, 2003; Hertz, Unger, Cornell, & Saunders, 2005; Victor et al., 2008).

There are a number of effective nonpharmacological and pharmacological treatments for hypertension (Lawes, Bennett, Feigin, & Rodgers, 2004; Schwartz, Neale, Marco, Shiffman, & Stone, 1999). Many current studies indicate that Blacks are more likely than Whites to be prescribed treatment for hypertension. Rates of treatment for hypertension in Blacks range from 53.6% to 68.2%; whereas rates of treatment for hypertension in Whites range from 48.6% to 60.4% (Glover et al., 2002; Hertz et al., 2005; Victor et al., 2008). However, at least one study in New York City (Angell et al., 2008) reported that Black adults were less likely to receive treatment than were Whites, in part because the Black individuals were less likely than Whites to receive routine primary care.

Even when treated, the majority of patients do not consistently achieve BP control (i.e., achieving levels below 140/90 with treatment). Nationally, across ethnic or racial groups, only about 31% of those diagnosed with hypertension meet criteria for good BP control (Wang & Vasan, 2005). There is some evidence that difficulties in achieving BP control are particularly problematic for Blacks in comparison to Whites, even when both groups receive treatment. Across a number of studies, the control rates among treated hypertensive Whites ranged from 55.6% to 86.3%; whereas rates for Blacks ranged from 44.1% to 65.2% (Angell et al., 2008; Hajjar & Kotchen, 2003; Hertz et al., 2005; Victor et al., 2008). However, other studies have revealed no significant race differences in BP control (Cutler et al., 2008; Wyatt et al., 2008).

There is some evidence that Black–White differences in BP control are partly a function of disparities in adherence. Several studies, including large-scale investigations emerging from samples of Veterans Administration patients, have reported that Blacks are less adherent to antihypertensive medication than are Whites, and that these differences in adherence largely account for Black–White differences in BP control (Bosworth et al., 2006; Krousel-Wood et al., 2009). In contrast, other population-based studies emerging from NHANES have indicated that Blacks are as adherent or more adherent than Whites and that Blacks continued to have poorer BP control despite adherence to treatment (Natarajan, Santa Ana, Liao, Lipsitz, & McGee, 2009). Still other studies have reported no race differences in adherence (Kressin & Peterson, 2001).

In sum, Black Americans are more likely to be aware of their hypertension and may be more likely to receive treatment for hypertension. Despite these gains, Black Americans are still more likely than White Americans to develop hypertension, and they may be less likely to achieve effective BP control. These findings suggest that influencing access to care and treatment may not be sufficient to reduce the excess burden of hypertension among Blacks. A greater understanding of the biopsychosocial determinants of hypertension may be necessary to address the racial disparities in the prevalence and course of hypertension and to modify the type and intensity of treatments offered.

RISK FACTORS FOR HYPERTENSION

Racial disparities in the prevalence and course of hypertension may be partly a function of disparities in exposure to risk factors for hypertension. Some of the risk factors are measured at an individual level (e.g., obesity), whereas others are measured at a community level (e.g., neighborhood crime). If race differences in exposure to particular risk factors account for race differences in BP, then interventions designed to reduce these risk factors should reduce racial disparities in hypertension. But the situation is not that simple or clear. As we will review, there are difficulties with the available evidence that make it difficult to evaluate the role of race differences in risk factors to the development of hypertension. Overall, the findings highlight the need for more complex models to explain racial disparities in the prevalence of hypertension (Dressler, Oths, & Gravlee, 2005).

The major *individual-level* and potentially modifiable risk factors associated with hypertension include dietary factors and obesity and exercise participation and fitness (Cossrow & Falkner, 2004; Dickson, Blackledge, & Hajjar, 2006; Hajjar & Kotchen, 2003; Halpert et al., 1997; Narkiewicz,

2006; Okosun, Choi, Dent, Jobin, & Dever, 2001; Wexler et al., 2008), as well as psychosocial stressors and stress-related personal characteristics (e.g., stressful events, negative affect–related traits; Jonas & Lando, 2000; Matthews et al., 2004). *Community-level* risk factors include neighborhood SES and neighborhood stressors (e.g., crime, marital instability; see Dressler et al., 2005, for review; Harburg, Gleiberman, Roeper, Schork, & Schull, 1978; Nguyen, Evans, & Zonderman, 2008; D. K. Wilson, Kliewer, & Domenic, 2004).

We briefly review evidence supporting the link between each individual-level and community-level risk factor and hypertension. Next, we examine the evidence documenting racial disparities in the level of exposure to the risk factor and/or suggesting racial differences in the relationship of this risk factor to BP levels. Finally, we evaluate the evidence investigating the degree to which these risk factors can explain racial disparities in hypertension.

HEALTH BEHAVIORS AND RELATED OUTCOMES

Obesity

Obesity as defined by body mass index (BMI) is a potent risk factor for hypertension (Cossrow & Falkner, 2004; Okosun et al., 2001). There are well-documented racial disparities in the prevalence of obesity, though there are variations depending on gender. The NHANES 1999–2000 survey revealed no race differences in the prevalence of obesity (i.e., BMI ≥ 30) for adult men, but substantial differences between Black and White women (White, 30.1%; Black, 49.7%; Flegal, Carroll, Ogden, & Johnson, 2002). Although BMI is associated with increased risk for hypertension in all groups, as obesity increases, the risks for hypertension grow more pronounced for Whites than Blacks (Oberg et al., 2007; Paeratakul, Lovejoy, Ryan, & Bray, 2002).

Abdominal obesity or visceral adipose tissue is a specific risk factor for hypertension (Fox et al., 2007). Studies of Black–White differences in abdominal obesity yield mixed findings (Cossrow & Falkner, 2004; Okosun et al., 2001; Perry et al., 2000). The limited available data suggest that the relationship of abdominal obesity to hypertension is stronger for Blacks than for Whites (Okosun et al., 2001).

Healthy Diets

Hypertension prevalence has been associated with several dietary factors, including low levels of consumption of fruits and vegetables, low levels of consumption of potassium, and high levels of consumption of sodium, among other factors (Beitz, Mensink, & Fischer, 2003; Dickson et al., 2006; John, Ziebland, Yudkin, Roe, & Neil, 2002; Kotchen & McCarron, 1998). There are racial disparities in patterns of consumption. In comparison with White Americans, African Americans consume significantly fewer fruits and vegetables (Dickson et al., 2006) and more dietary sodium (Ervin, 2008).

There are very limited direct data on race differences in the relationship of patterns of consumption to hypertension. There is indirect evidence that the ability to purchase a healthier diet (e.g., when individuals have access to supermarkets) is associated with better improvements in BP for Black individuals rather than for Whites individuals (Morland, Wing, & Diez-Roux, 2002). There are some data suggesting that Black individuals are more affected by high sodium intake than are Whites, and Blacks respond with larger reductions in BP when exposed to a low-sodium diet (Sacks et al., 2001). However, there is also evidence that ethnicity does not influence the relationship between sodium intake and BP (Madhavan & Alderman, 1994).

Alcohol Consumption

The evidence concerning the relationship of alcohol consumption to hypertension has been mixed, with some studies reporting that the number of alcoholic drinks per day is associated with

hypertension (Marmot et al., 1994; Russell, Cooper, & Frone, 1990; Sesso, Cook, Buring, Manson, & Gaziano, 2008; York & Hirsch, 1997). Other studies have not found a relationship of alcohol intake to hypertension (Koppes, Twisk, Van Mechelen, Snel, & Kemper, 2005). Daily alcohol consumption is higher among White adults than Black adults, but the association of alcohol intake to hypertension risk is greater for Black men. Race differences in the relationship of alcohol to hypertension are not seen in women (Svetkey et al., 2005).

Fitness and Exercise

Higher levels of fitness are associated with lower risk for hypertension (Fagard, 2005; Halpert et al., 1997). Data from national studies suggest that Whites are more likely than Blacks (33.7% vs. 25.3%) to report regular leisure-time physical activity (Rosamond et al., 2008). Black Americans are also more likely than their White counterparts to be "thinking about exercising" as opposed to actually exercising, according to one large-scale survey (Wexler et al., 2008). There is some evidence that although higher levels of fitness are associated with lower levels of hypertension for both Blacks and Whites, the relationship of fitness to hypertension was significant for Whites and not for Blacks (Bassett, Fitzhugh, Crespo, King, & McLaughlin, 2002).

Psychosocial Stressors

Individual-level psychosocial stressors include a wide variety of variables including life events and job strain. Psychosocial stressors also include personal characteristics, including negative affect and anger-related traits (i.e., trait anger, hostility, and anger expression) that may increase the frequency and intensity of stress exposure and distress responses. Community-level stressors can include neighborhood violence, marital instability, or housing instability.

Psychosocial stressors may affect risk for hypertension through both psychophysiological and behavioral pathways. Depression, anxiety, and anger-related traits all have been associated with disruptions in the autonomic and neuroendocrine systems that subserve BP regulation, though the pattern of effects differs depending on the characteristic under study (e.g., Jorgensen, Johnson, Kolodziej, & Schreer, 1996; Sloan et al., 2001). For example, there is substantial evidence that hostility and other anger-related traits are associated with increased BP or heart rate reactivity to stress, one index of altered autonomic function. Hostility and other psychosocial stressors also may contribute to poor health behaviors and may influence the relationship of particular health behaviors to hypertension (Matthews, 2005).

Many psychosocial stressors have been linked to hypertension prevalence in the general population, though the evidence is not completely consistent (Friedman et al., 2001). Work-related stressors are a significant correlate of hypertension (Levenstein, Smith, & Kaplan, 2001). There is also evidence linking chronic perceived stress (Sparrenberger et al., 2009), depression (Bosworth, Bartash, Olsen, & Steffens, 2003), anxiety (Jonas & Lando, 2000), hostility (Yan et al., 2003), and anger suppression (Schum et al., 2003) to either BP level or hypertension, (Friedman et al., 2001; Levenstein et al., 2001; Sparrenberger et al., 2009). Effects for job strain (Ohlin, Berglund, Rosvall, & Nilsson, 2007), anger, anxiety, and depression (Rutledge & Hogan, 2002) have been seen cross-sectionally as well as prospectively (i.e., in studies of changes from normotensive to hypertensive status).

There is evidence of Black–White differences in the levels of exposure to psychosocial stressors associated with hypertension, though the findings depend on the type and severity of the stressor (Jonas & Lando, 2000; Julius, 1988). For example, Black adults are more likely to suffer from unemployment and to face discrimination at work than are Whites (Mays, Coleman, & Jackson, 1996). Assessments of negative mood and overall psychological distress occur disproportionately among African Americans in comparison to Whites (Jonas & Lando, 2000); however, data on the prevalence of diagnosable depressive disorders do not yield consistent evidence of higher levels of diagnosable depression among African Americans (C. I. Cohen, Magai, Yaffee, & Walcott-Brown,

2005; Riolo, Nguyen, Greden, & King, 2005). There is also evidence of racial disparities in anger-related traits, with several studies indicating that Blacks have higher self-reported levels of trait hostility than do Whites (D. C. Cooper & Waldstein, 2004; R. B. Williams, Barefoot, & Schneiderman, 2009). There is some limited evidence that Blacks may be more likely than Whites to suppress rather than express anger (Harburg, Gleiberman, Russell, & Cooper, 1991).

The effects of stressor exposure on distress also appear to be greater for Blacks. Initial evidence cited by Williams (D. R. Williams & Collins, 1995) suggested that Blacks reported more distress from negative life events than did Whites. Prospective studies suggest that smaller changes in stress exposure are associated with greater changes in depressive symptoms for Blacks versus Whites (George & Lynch, 2003).

Stress and negative affect–related characteristics may also be more closely related to hypertension for Black adults than White adults, though the findings are not always consistent. Unemployment has been associated with greater rates of hypertension among Black men than among Whites (Levenstein et al., 2001). Prospective studies indicate that depression scores are more closely related to hypertension incidence in Blacks than Whites (Jonas & Lando, 2000; Krieger & Sidney, 1996); however, other studies have reported reverse effects (Yan et al., 2003). Some investigators have reported stronger relationships of hostility to BP for Black adults (R. S. Cooper, 2003); but Yan et al. (2003) reported that although hostility was a significant predictor of the development of hypertension, the effects were seen for both Black and White adults. Similarly, anger suppression has been associated with hypertension. Some studies suggest that these effects are more likely to emerge in studies of Blacks (Jorgensen et al., 1996), though other studies report no race differences (Schum et al., 2003).

Cardiovascular reactivity (CVR), one measure of physiological response to stress, has been prospectively associated with hypertension risk (Matthews et al., 2004). Many, though not all, studies of CVR suggest that Blacks show greater BP reactivity than do Whites across a range of stressors (Anderson, 1989; Duey et al., 1997; Light, Turner, Hinderliter, & Sherwood, 1993; Murphy, Stoney, Alpert, & Walker, 1995; Thomas, Nelesen, Malcarne, Ziegler, & Dimsdale, 2006; Treiber et al., 1993; Wilcox, Bopp, Wilson, Fulk, & Hand, 2005).

In sum, there are racial disparities in key health behaviors and related outcomes associated with hypertension risk. White individuals consume greater amounts of alcohol than do Black individuals; but Black individuals are more likely to be obese and to consume diets high in sodium but low in fruits and vegetables and are less likely to engage in physical activity than are Whites. However, the effects of these health behaviors and associated outcomes are not consistent across race and ethnic groups. The association of obesity and fitness may be slightly stronger for Whites than Blacks, whereas some evidence suggests that the association of sodium and alcohol consumption and abdominal obesity to hypertension is stronger for Blacks than Whites.

Black individuals are more likely to experience psychosocial stress, to experience distress as a function of stress exposure, and to display heightened physiological responses to these acute stressors; they may also be more likely to show slower recovery to certain stressors (Lepore et al., 2006). It is not clear if psychosocial stressors are more closely related to hypertension diagnosis in Black versus White adults.

COMMUNITY-LEVEL RISK FACTORS

In the next section, we examine contextual or community-level factors, including neighborhood SES and neighborhood measures of stress that have been identified as risk factors for hypertension. The bulk of studies of community-level factors have examined effects on coronary heart disease or mental health outcomes (Diez-Roux, Merkin, Hannan, Jacobs, & Kiefe, 2003; Leventhal & Brooks-Gunn, 2000), but there is a growing literature on the effects of neighborhood stress or economic resources on risk for hypertension or hypertension prevalence. These community-level risk factors may differentially influence the health behaviors and psychosocial stress exposure in Black

versus White individuals. These community-level variables may also moderate the relationship of individual-level risk factors to BP.

Neighborhood SES, Neighborhood Stress, and Hypertension

Neighborhood levels of SES are generally assessed with census-derived indices of income, education, and occupation level of the residents or the costs and types of housing. The findings from correlational studies examining neighborhood levels of SES and hypertension prevalence are clear. In general, indices associated with lower levels of SES are associated with higher levels of hypertension. Specifically, median housing values, controlling for individual-level measures of SES, have been negatively associated with hypertension prevalence in a national sample of Black women (Cozier et al., 2007). Similarly, rental costs have been inversely associated with hypertension in a regional study in the southern United States (Schlundt, Hargreaves, & McClellan, 2006). Block-group-level measures of income, education, and occupational status were associated with hypertension in a study of four different communities, though not all effects were significant and there were some variations by race (Diez-Roux et al., 1997). Neighborhood affluence (i.e., presence of educated individuals, proportion of individuals in professional or managerial occupations) was associated with decreased risk for hypertension in a national sample (Morenoff et al., 2007).

Low levels of neighborhood SES are associated with fewer nutritional and recreational resources. Specifically, in comparison to more affluent areas, low-SES neighborhoods have fewer stores selling low-cost and high-quality fruits and vegetables, more stores selling alcohol and fast food, and fewer facilities for recreation and fitness (Kwate, 2008; Schlundt et al., 2006). The lack of resources increases the barriers to positive health behavior and increases the likelihood that individuals will have one or more individual-level risk factors.

Data on the effects of community-level stress exposure are beginning to emerge and suggest that perceptions of social stress in the community, including experiences of violence, may increase risk for hypertension (see D. Wilson et al., 2004, for review). The studies of neighborhood stressors have evaluated the relationship of both objective and subjective ratings of different characteristics associated with stress to either BP level or self-reported hypertension. Neighborhood characteristics that have been assessed include residential or marital stability, cleanliness, crime, perceived safety, and violence. Crime- and violence-related stressors are likely to serve as chronic stressors, because fear of crime and behavioral preparations to prevent crime may be ongoing, even if actual personal or property damage occurs infrequently.

In a seminal set of studies, Harburg and colleagues (1978) reported that neighborhood stress (a composite of marital and residential instability and crime rates) was associated with higher levels of BP for younger, but not older, Black men and for younger Black women, but only for those who were underweight. The effects of neighborhood stress were not seen for Whites (Harburg et al., 1978). However, criteria for determining that an area was "high stress" were set separately for Blacks and Whites, and they lived in distinct neighborhoods. Consequently, it is difficult to determine if Whites would also display similar levels of BP if they were living in neighborhoods with the absolute stress levels seen in Black communities.

More recent work supports the notion that neighborhood stressors are associated with BP levels or hypertension. In adolescents, evidence suggests that reports of neighborhood levels of violence are associated with nocturnal blood pressure (D. K. Wilson, Kliewer, Teasley, Plybon, & Sica, 2002). In a study of managed-care patients with diabetes (Gary et al., 2008), perceived neighborhood problems (i.e., a combination of perceptions of crime, litter, trash, lighting, and access to transportation, exercise and supermarkets) were associated with poorer BP control. It is important to note that this was the case even when making comparisons controlling for individual-level indices of objective SES. Similarly, in the MESA Study, low levels of perceived safety and social cohesion, walkability, and access to healthy foods were associated with higher rates of hypertension (Mujahid et al., 2008).

There is substantial evidence that Blacks are more likely than Whites to live in areas with higher levels of perceived stress, lower levels of SES, and fewer health-promoting resources (Mujahid et al., 2008). This situation is particularly problematic because low-income Black individuals tend to live in more segregated neighborhoods with fewer resources than do low-income White individuals (Massey & Eggers, 1990). As D. R. Williams and Jackson (2005) have pointed out, the living conditions in impoverished Black neighborhoods are substantially worse than those in even the poorest neighborhoods in which the majority of residents are White.

The limited available evidence suggests minimal race difference in the deleterious effects of stressed or impoverished neighborhoods. In the MESA Study, low levels of key neighborhood characteristics (i.e., safety, social cohesion, walkability, and access to healthy foods) were associated with hypertension, but there were no race differences in these effects (Mujahid et al., 2008). Similarly, other researchers report that the risks of relative neighborhood disadvantage on hypertension hold for Whites as well as Black adults (Grady & Ramirez, 2008; Morenoff et al., 2007).

In sum, there is clear evidence that neighborhood SES is associated with higher levels of hypertension. There is also growing evidence of an association between neighborhood stressors and hypertension prevalence. Neighborhood-level variables (e.g., deficiencies in access to healthy food or recreational facilities or excess exposure to stress) appear to contribute to hypertension prevalence for both Blacks and Whites. However, Black individuals are more likely than White individuals to live in neighborhoods characterized by high levels of environmental stress and low levels of resources, increasing their exposure to both acute and persistent stressors and decreasing their ability to recover from stress exposure.

It is of note that there may also be other important pathways through which race affects hypertension risk, pathways not addressed here. For example, there may also be effects through the quality of medical care or through the patterns of distribution of physicians throughout poor communities (Bach, Hoangmai, Schrag, Tate, & Hargraves, 2004; Benkert, Peters, Clark, & Keves-Foster, 2006; Klonoff, 2009). There may also be other individual-level factors that serve as mediators of the relationship of racism to hypertension, including potential epigenetic influences or low birth weight (Oberg et al., 2007).

DO RACIAL DIFFERENCES IN RISK FACTORS ACCOUNT FOR RACIAL DISPARITIES IN THE PREVALENCE OF HYPERTENSION?

There is simply insufficient evidence to determine if racial differences in risk factors account for racial disparities in the prevalence of hypertension. There is, however, some evidence suggesting that racial disparities in hypertension persist despite controlling for individual-level health behaviors and even SES (see Dressler et al., 2005, for review). For example, in a population-based sample, racial disparities in untreated HTN persist when controlling for individual-level risk factors, including health behaviors (e.g., smoking, obesity, fitness), as well as individual-level education and income (Bell, Adair, & Popkin, 2004). Racial disparities in hypertension risk emerge at every education level (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005; Pickering, 1999). However, the limited number of studies that have included measures of perceived stress as well as health behavior and SES find more significant reductions in racial disparities (Jones-Webb, Jacobs, Flack, & Liu, 1996).

Nonetheless, there are limits to the interpretation of data on the degree to which individual-level risk factors account for racial disparities in hypertension, because the methods of analysis may not be adequate to test the hypothesis. In most studies, unweighted scores for each of the risk factors are entered into the analyses (e.g., Bell et al., 2004). This type of modeling assumes that the impact of each individual risk factor is the same for Blacks and Whites. Yet as we have presented, there is evidence that there are Black–White differences in the association of most risk factors with hypertension. Consequently, the assumption of equivalence is likely to be incorrect, and different quantitative models may be needed to fully evaluate these effects.

There may also be moderator effects in which one risk factor influences the expression of another. Many moderator effects involve individual-level stress exposure. Stress appears to change the relationships between sympathetic nervous system activity and BP regulation (Joyner, Charkoudian, & Wallin, 2008); stress affects sodium excretion by means of the renin-angiotension-aldosterone systems, altering BP through changes in blood volume (Harshfield, Pulliam, & Alpert, 1994); and stress influences the expression of genes involved in BP control (Esler et al., 2008). Black adults are more likely than Whites to be exposed to certain psychosocial stressors and, consequently, may be more likely to be subject to these moderator effects. Variations in stress exposure may partly account for race-related differences in the association of certain risk factors (e.g., abdominal obesity) to hypertension. Therefore, an analysis of the contribution of individual-level risk factors to racial disparities in hypertension that fails to include tests for interactions among these factors may underestimate their effects in explaining Black–White differences in hypertension.

There may also be "third variable" problems, given that race and social class are closely but not uniformly linked. Social class, and in particular education level, is associated with hypertension prevalence (see review by Colhoun, Hemingway, & Poulter, 1998). There are documented Black–White disparities in educational attainment, though the effects vary by educational subject as well as by individual age and generational cohort (U.S. Census Bureau, 2008; Jacobson, Olsen, Rice, Sweetland, & Ralph, 2001).

Education may affect hypertension through its association with health behaviors, including alcohol use and obesity (Colhoun et al., 1998) and exposure to psychosocial stressors (Diez-Roux, Northridge, Morabia, Bassett, & Shea, 1999). There is a need for further research on the prospective effects of changes in education or income on BP. This evidence could provide a clearer understanding of the causal nature of this relationship and help identify aspects of education that confer health benefits or contribute to racial disparities in risk.

The "third variable" problem is linked to a more problematic issue: The antecedents of any particular health behavior may vary by race, and these variations may also confound estimates of the relationship of each risk factor to hypertension. We can examine these issues in more detail by considering the factors that motivate individuals to exercise and by noting the effect of these factors on estimates of the relationship of exercise to hypertension.

When exercise facilities are easily accessible and the barriers to regular participation are low, then individuals may exercise even if they have only moderate levels of motivation or support. In contrast, when barriers to participation are high (e.g., limited access to child care, no peer support, insufficient leisure time, restricted ability to access safe exercise facilities), then any participation may reflect very high levels of motivation. In turn, on an individual basis, the level of motivation may be a function of the individual's level of conscientiousness, social support, and/or negative affect. Each of these antecedent variables—that is, variables that influence motivation and predict exercise participation—may have independent effects on hypertension risk.

More concretely, any given score on a measure of exercise participation includes some variance associated with motivation to exercise. However, race differences in the relationship of exercise to hypertension may be obscured if the factors that affect motivation are not assessed as well. For example, some White individuals are less likely to face barriers to participating in exercise or consuming healthy diets because, for example, they are more likely to reside in areas with accessible opportunities for physical activity, and/or to have supports that promote exercise adherence (Mechanic, 2005). Therefore, for Whites living in environments that present few barriers to participation, low levels of exercise compliance (e.g., walking one day a week) may be a function of low levels of individual motivation. In contrast, for Blacks living in environments that present high barriers to participation, an equivalent level of participation (i.e., walking one day a week) may reflect a much higher level of motivation. If motivation is partly a function of mood, then for Whites, but not necessarily for Blacks, low absolute levels of participation may be paired with high levels of negative mood. The determinant of exercise behavior or any given health behavior is likely to vary by community depending on the actual barriers and supports for those behaviors. Consequently,

it may be important to conduct sample-specific evaluations of the predictors of any given health behavior.

Although studies of community variables as predictors of health behavior and outcome represent a new area of research, there is some evidence that community variables may at least partly explain racial disparities in hypertension. To underscore the importance of neighborhood resources, a recent study reports on a comparison of risk factors for hypertension in two neighborhoods in which the levels of resources available to Blacks and Whites were roughly equivalent. In this study, racial disparities in some of the variables related to hypertension were minimized (La Veist, Sellers, & Neighbors, 2001). In contrast, the one experimental study in which very low-income individuals from high-poverty neighborhoods were randomly assigned to new, less impoverished neighborhoods indicated that moving did not affect rates of hypertension over a five-year period, though there were reductions in obesity and in perceived stress and distress (Kling, Liebman, & Katz, 2007).

However, it is difficult to fully evaluate the hypothesis that community-level variables explain race differences, given that race-related and neighborhood-level variables are so heavily confounded. For example, in the Four Neighborhoods Study, the African American community was different from the fully or mostly White communities on every socioeconomic index (Diez-Roux et al., 1997). Similarly, the effects of specific indices of neighborhood stress are difficult to identify because some investigators have combined measures of access to resources (e.g., supermarkets) with measures more explicitly tied to psychosocial stress (e.g., perceptions of safety, violence, marital instability). These variables tend to cluster together, but it will be difficult to identify targets for intervention without a more systematic evaluation of their unique effects. Studies of the ways in which neighborhood or contextual factors moderate the effects of individual-level risk factors are also crucial.

The research on the effects of neighborhood stressors is much more developed in studies of childhood academic performance and mental disorders (see Leventhal & Brooks-Gunn, 2000, for review). It will be important to extend the methodologies from these studies to investigate the specific neighborhood stressors or resources that predict the development of hypertension.

IS RACISM A RISK FACTOR?

In the next section, we consider the possibility that racism contributes to the prevalence and course of hypertension. Racism is a psychosocial stressor that is disproportionately experienced by African Americans in comparison to European Americans (Brondolo, Brady, Libby, & Pencille, 2010; Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). Because it is a complex and multilevel stressor, racism may affect risk for hypertension through multiple mechanisms (Mays, Cochran, & Barnes, 2007). Figure 24.1 illustrates ways in which racism may operate to directly and indirectly influence risk.

Specifically, there may be direct relationships of racism to hypertension. Racism may also contribute to hypertension through effects on risk factors for hypertension (e.g., obesity, stress, or fitness). If racism influences the development and course of hypertension, risk-reducing interventions must consider the role of racism if they are to be successful in changing health behavior for targeted groups.

To fully evaluate the role of racism in the development of hypertension, it is necessary to first consider the complex nature of the actions and circumstances linked to racism and ethnic discrimination. There have been a number of definitions of racism and ethnic discrimination. One widely used definition, presented by Rodney Clark and colleagues in a paper published in 1999 in *American Psychologist* is that racism consists of "the beliefs, attitudes, institutional arrangements, and acts that tend to denigrate individuals or groups because of phenotypic characteristics or ethnic group affiliation" (Clark, Anderson, Clark, & Williams, 1999, p. 805). Contrada has used the more general term *ethnic discrimination* and defined it as unfair treatment received because of one's ethnicity, where "ethnicity" refers to various groupings of individuals based on race or culture of origin (Contrada et al., 2001; Contrada et al., 2000). In line with these definitions, we consider racism or

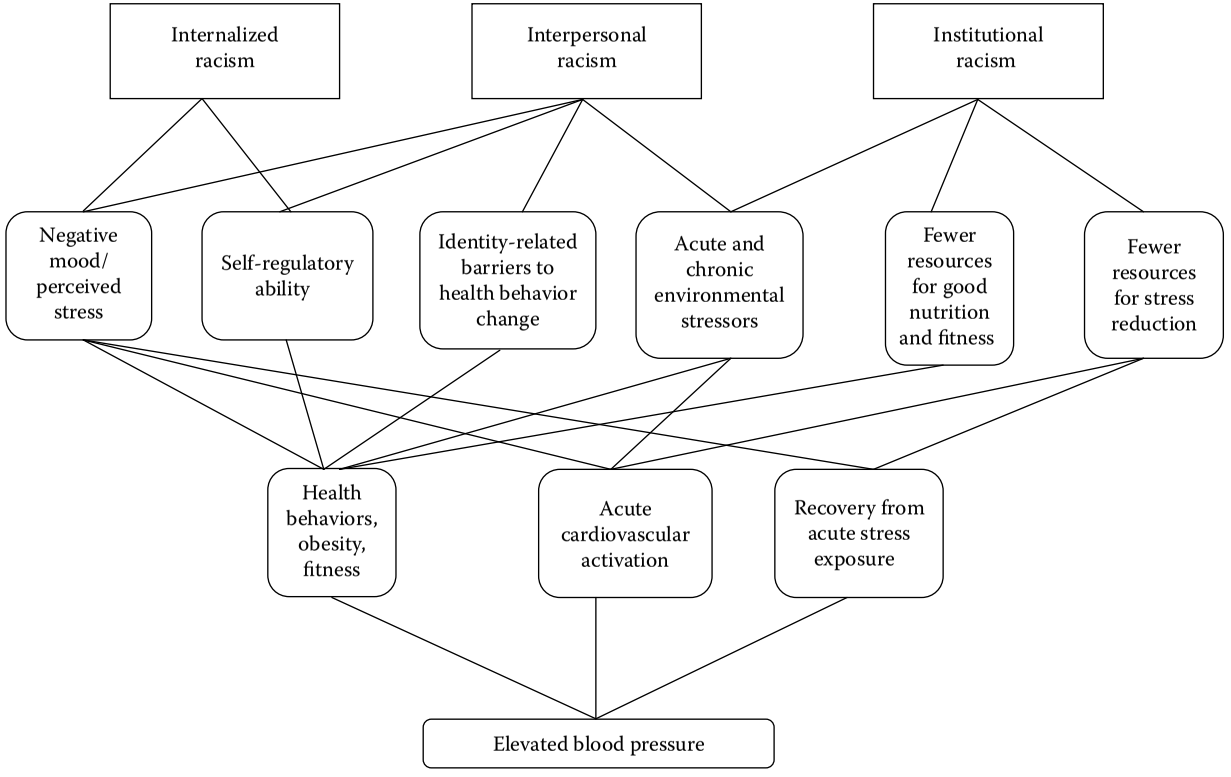

FIGURE 24.1 Possible relationships among different levels of racism, hypertension risk factors, and hypertension prevalence.

ethnic discrimination to be a form of social ostracism in which phenotypic or cultural characteristics are used to render individuals outcasts, making them targets of social exclusion, unfair treatment, and threat or harassment (Brondolo et al., 2010; Brondolo, Brady, et al., 2008).

Racism can occur on multiple levels: cultural, institutional, interpersonal, and intrapersonal or internalized (Harrell, 2000). Cultural racism occurs when the methods used to communicate cultural values (e.g., film, television, advertisements) convey negative information about a particular group. Through a variety of media, individuals are exposed to messages that influence the perceptions of a targeted group, affecting the behaviors and attitudes of all individuals both in and out of the targeted group (Major & O'Brien, 2005). The term *institutional racism* is generally used to refer to specific policies and/or procedures of institutions (i.e., government, business, schools, churches) that consistently result in unequal treatment for particular groups (Better, 2002; Griffith, Childs, Eng, & Jeffries, 2007; Lea, 2000). Jim Crow laws, forcing segregation of the races, were clear examples of institutional racism. Residential racial segregation can also be considered an outcome related to institutional racism (Williams & Mohammed, 2009).

Individual-level or interpersonal racism has been defined as "directly perceived discriminatory interactions between individuals, whether in their institutional roles or as public and private individuals" (Krieger, 1999, p. 301). Interpersonal racism may encompass different types of experiences, ranging from social exclusion, workplace discrimination, and stigmatization to physical threat, harassment, and aggression (Brondolo et al., 2005). Internalized racism has been defined as "the acceptance, by marginalized racial populations, of the negative societal beliefs and stereotypes about themselves" (D. R. Williams & Williams-Morris, 2000, p. 255). For example, individuals may come to believe themselves to be less intelligent by virtue of their group membership if they belong to a group stereotyped as inferior in intelligence.

Measuring Racism

Although cultural influences on the development of health attitudes and behaviors have been documented (Castro, Shaibi, & Boehm-Smith, 2009), to our knowledge there is no direct research examining the relationship of the effects of racial bias in media presentation to hypertension prevalence. Therefore, we confine our discussion of the strategies for measuring racism to assessments of institutional, interpersonal, and internalized racism.

Many studies of the health effects of institutional racism have examined outcomes of policies, rather than the policies or law themselves. Residential racial segregation (RRS) is one outcome of institutional policies and interpersonal practices that isolated African Americans and influenced patterns of housing (see M. R. Kramer & Hogue, 2009, for a review). In RRS, the types of exclusion and rejection associated with interpersonal racism are manifest in a community setting. Across all income groups, Blacks tend to live in more racially segregated areas than do Whites, but race-based residential segregation is most pronounced among low-income individuals (D. R. Williams & Mohammed, 2009). Population-based studies of attitudes toward housing confirm the degree to which race-based social ostracism drives RRS (Emerson, Yancey, & Chai, 2001).

Several different strategies used to assess RRS have been reviewed in Kramer and Hogue (2009). The indices assess the degree to which an individual is likely to encounter an individual of a different race. One of the most commonly used metrics is the proportion of Black residents in the census tract (or other defined area).

There are also investigators who have used self-reported measures of individual racism that occur in institutional settings (i.e., at work or in the criminal justice context) to serve as indices of institutional racism. However, because these scales measure directly perceived experiences, we have included them with measures of individual-level racism. Typically, interpersonal or individual-level racism is assessed with self-report questionnaires (e.g., Perceived Ethnic Discrimination Questionnaire–Community Version; Brondolo et al., 2005; Racism and Life Experiences Survey, Harrell, 1997;

Experiences of Discrimination Questionnaires, Krieger et al., 2005; Schedule of Racist Events, Landrine, Klonoff, Corral, Fernandez, & Roesch, 2006). These scales measure the frequency of exposure to either different types of race-based maltreatment or overall perceptions of exposure to discrimination in different settings. In published studies of hypertension and related risk factors, internalized racism has been assessed with the Nadolization Questionnaire, which measures agreement with common stereotypical beliefs (Taylor & Grundy, 1996).

ASSOCIATIONS OF INSTITUTIONALIZED, INTERPERSONAL AND INTERNALIZED RACISM TO HYPERTENSION AND HYPERTENSION-RELATED RISK FACTORS

Internalized Racism

To our knowledge, there is only one study directly assessing the effects of internalized racism to resting BP, and this study did not find a relationship (S. E. Tull et al., 1999). Three studies, including one of adolescents, suggest a relationship of internalized racism to abdominal obesity, a known risk factor for HTN (Butler, Tull, Chambers, & Taylor, 2002; Chambers et al., 2004; S. E. Tull et al., 1999). However, this relationship has not been found in every study (E. S. Tull, Sheu, Butler, & Cornelious, 2005). One study also reports a relationship of internalized racism to perceived stress (E. S. Tull et al., 2005).

Interpersonal Racism

In our earlier review (Brondolo, Rieppi, Kelly, and Gerin, 2003), we reported very limited evidence of a direct relationship of interpersonal racism to blood pressure. Since that publication, new studies have emerged, and several studies have included assessments of ambulatory blood pressure as well as clinic BP. To date, there have been 13 studies examining the relationship of self-reported exposure to interpersonal racism to resting BP level or self-reported or doctor-diagnosed hypertensive status in Black adults (Barksdale, Farrug, & Harkness, 2009; Broman, 1996; J. Collins, David, Handler, Wall, & Andes, 2004; K. James, Lovato, & Khoo, 1994; S. A. James, LaCroix, Kleinbaum, & Strogatz, 1984; Krieger, 1990; Krieger & Sidney, 1996; Peters, 2004, 2006; Poston et al., 2001; Ryan, Gee, & Laflamme, 2006). There are only two studies that find a positive relationship between self-reported racism and either BP level or self-reported hypertensive status in the group overall (K. James et al., 1994) or in one subgroup (i.e., non-U.S.-born women; Cozier et al., 2006). Seven studies did not find a direct relationship of perceived racism to BP when the investigators examined the sample as a whole (Barksdale et al., 2009; Broman, 1996; Cozier et al., 2006; Din-Dzietham, Nembhard, Collins, & Davis, 2004; Dressler, 1996; Peters, 2006; Poston et al., 2001); two studies found a negative relationship either among older participants (Peters, 2004) or among the participant group as a whole (Krieger, 1990).

In contrast, the data from ambulatory blood pressure (ABP)–monitoring studies are more consistent. ABP, and in particular nocturnal ABP, is regarded as a more reliable predictor of target organ damage than are clinic measures (Pickering et al., 2005). The five studies investigating these effects in adults reported positive relationships for either daytime ABP (Steffen, McNeilly, Anderson, & Sherwood, 2003), or nighttime (Brondolo, Libby, et al., 2008; Singleton, Robertson, Robinson, Austin, & Edochie, 2008), or both (Hill, Kobayashi, & Hughes, 2007; Tomfohr, Cooper, Mills, Nelesen, & Dimsdale, 2010).

It is surprising that although evidence shows that racism is related to obesity among Asian individuals (Gee, Ro, Gavin, & Takeuchi, 2008), there has been little research on the effects among African Americans. A recent study suggests that racism may be associated with changes in weight gain over an eight-year period (Cozier, Wise, Palmer, & Rosenberg, 2009). The one study investigating the effects of racism on exercise participation did not find an association (Shelton Puleo, Bennett, McNeill, Goldman, & Emmons, 2009).

There is clear evidence that interpersonal racism is associated with negative affect, anger, depression, and anxiety, all potential risk factors for HTN (Brondolo, Brady, et al., 2008; Kessler, Mickelson, & Williams, 1999; Moghaddam, Taylor, Ditto, Jacobs, & Bianchi, 2002). Some data confirm the relationship of interpersonal racism to CVR, itself a risk factor for HTN and a potential mediator of the relationship between psychosocial stress and HTN (Clark, 2000; Guyll, Matthews, & Bromberger, 2001).

In a new area of research, investigators have consistently documented a link between interpersonal racism and low birth weight (Collins et al., 2004; Collins, et al., 2000; Dole et al., 2004; Lespinasse, David, Collins, Handler, & Wall, 2004; Mustillo et al., 2004). Low birth weight is also a risk factor for HTN, possibly through its association with heightened stress responsivity (Feldt et al., 2007). These effects may be stronger for African Americans than for European Americans (Oberg et al., 2007).

Institutionalized Racism

There has been limited research specifically examining the degree to which HTN prevalence is increased as a function of RRS, as assessed by the degree to which Black individuals live primarily with other Blacks. In two studies, the proportion of individuals who were African American was associated with increased risk for hypertension (Grady & Ramirez, 2008). However, in only one study did the investigators control for neighborhood poverty and related factors (Grady & Ramirez, 2008). Consequently, it is difficult to determine if other characteristics of the neighborhood, rather than simply the presence of Black individuals, account for the higher prevalence of HTN. In contrast, two studies, one of adults (Morenoff et al., 2007) and one of adolescents (McGrath, Matthews, & Brady, 2006), report that a higher percentage of Blacks in the neighborhood was not associated with greater prevalence of HTN, controlling for other neighborhood conditions, including affluence and disadvantage.

Data from several studies suggest that living in neighborhoods with high concentrations of Black individuals is associated with a higher prevalence of obesity, higher rates of distress, and lower levels of fitness (Diez-Roux et al., 1999; Schlundt et al., 2006). However, there have been very little data examining these effects while controlling for the concomitant presence of high levels of neighborhood stress and disadvantage. Therefore, it is difficult to determine if these effects are directly associated with the presence of Blacks in the neighborhood or if they are a function of the economic and social conditions that ensue when resources are not available.

Indirect Effects of Institutional, Interpersonal, and Internalized Racism

Most individual-level behavior change interventions to reduce weight or improve fitness require some intrinsic motivation, self-awareness, and the ability to experience positive mood in response to progress and to anticipate positive affect when achieving goals. Exposure to racism may affect each of these intrapersonal motivation-related factors. Consequently, interventions may need to target these racism-related outcomes prior to considering health behavior change.

All levels of racism may indirectly affect the intrinsic motivation to engage in some activities to change health behavior through their effects on stigma sensitivity and racial identity. Cultural racism may influence the development and maintenance of stereotypes of African Americans and may engender the development of concerns about *stereotype threat*. Stereotype threat is experienced when individuals are anxious that their performance in a particular arena will confirm the potentially biased and overgeneralized beliefs other people hold about their group (Speight, 2007; Steele, 1997). Direct exposure to racism may intensify awareness of the dangers associated with racial-group membership and strengthen concerns about stereotype threat (Contrada et al., 2001).

Concerns about stereotype threat may influence health behavior when individuals may be concerned that they are not capable of changing these health-related behaviors for fear of confirming a

weakness associated with the group as a whole (G. L. Cohen & Garcia, 2005). Similarly, if individuals have internalized stereotypes about their group's ability to engage in health behavior change, self-stereotyping may undermine individuals' beliefs in their self-efficacy or ability to engage in health behavior change.

Racism may also influence motivation through processes related to identity. Adhering to behaviors that are associated with one's own racial or ethnic group and rejecting behaviors associated with other groups can be seen as a strategy for promoting in-group identification and esteem (Fordham & Ogbu, 1986). There is evidence suggesting that experiences of discrimination serve to shift internal schemas about the self, increasing the investment in and attention paid to racial or ethnic aspects of identity (Quintana, 2007). This shift can limit the motivation to participate in activities that do not conform to the individual's ethnic or racial identity. For example, investigators have reported that some urban African Americans view efforts to control weight and to exercise as characteristics that are associated with middle-class and White individuals (Oyserman & Harrison, 1998). The more participants viewed these behaviors as inconsistent with their racial or ethnic identity, the less likely they were to engage in healthy eating habits.

Studies of RRS, a form of institutional racism, suggest that neighborhoods with higher percentages of African Americans are also associated with higher prevalence of obese individuals and with lower rates of exercise participation. These concentrations of obesity or inactivity can change group norms about health eating and fitness and alter the motivation or perceived need to change health behaviors (Kwate, 2008).

Racism may also influence risk through its effects on self-regulation and self-awareness. For example, we and others have underscored the importance of considering racism to be a form of race-based social ostracism (Brondolo et al., 2010; Brondolo, Gallo, Myers, & Hector, 2009). Ostracism of any kind, even potential ostracism, has been demonstrated to be associated with impairments in self-awareness as individuals seek to avoid the pain of social exclusion and rejection. This lack of self-awareness contributes to ostracism-related deficits in behavioral self-regulation (see Baumeister, DeWall, Ciarocco, & Twenge, 2005). Inzlicht and others have demonstrated specific effects of race-based ostracism on behavioral self-regulation, including eating behavior (Inzlicht, McKay, & Aronson, 2006). The impairments in self-awareness that are a function of ostracism may be particularly problematic when they are combined with high levels of negative mood. Negative mood can make self-evaluation more painful and consequently increase the level of effort required to initiate behavioral self-control.

Negative mood and subjective experiences of stress can diminish the experience of positive affect that can fuel efforts at health behavior change. Research from our laboratory (Brondolo et al., 2005) as well as others suggests that racism changes perceptions of new events, making a broader range of experiences capable of evoking distress. Specifically, the degree to which individuals have been exposed to racism in their past influences the ways in which they view new episodes of race-based maltreatment and other ongoing interpersonal exchanges. Ambulatory monitoring studies indicate that the more individuals had been exposed to racism over the course of their lifetime, the more likely they were to view ongoing interpersonal interactions as harassing and unfair (Brondolo, Brady, et al., 2008) and to view new episodes of ethnicity-related maltreatment as threatening and harmful (Brondolo et al., 2005). This reaction increases the type and intensity of events that can evoke acute stress responses and may contribute to higher levels of ongoing stress exposure.

Racism may exhaust the available coping resources required to promote health behavior change (Brondolo, ver Halen, Pencille, Beatty, & Contrada, 2009). Targeted individuals must address the specific threats presented by actual or anticipated episodes of ethnicity-related maltreatment, such as interpersonal conflict, blocked opportunities, and social exclusion. They must also manage the emotional consequences, including painful feelings of anger, nervousness, sadness, and hopelessness, and their physiological correlates, as well as potential damage to self-concept and self-esteem (Mellor, 2004). Targeted individuals must manage their concerns about the effects of racism on other individuals who share their phenotypic or cultural characteristics, including their friends and

family members. The effects of RRS, including greater exposure to neighborhood poverty and stress, may require additional coping efforts.

Consequently, individuals who are targeted for racism must develop a broad range of racism-related coping responses to permit them to respond to different types of situations and to adjust the response depending on factors that might influence the effectiveness of any particular coping strategy. This level of coping flexibility is beneficial, but it is difficult to achieve (Cheng, 2003). The effort to develop and deploy all the coping strategies necessary to manage responses to different situations is an obvious burden and may limit the cognitive, affective, and self-control resources that are available to tackle additional challenges (Baumeister et al., 2005).

Finally, if race-based ostracism results in the creation of communities in which Black individuals are relatively isolated, fewer social, education, and economic resources will be available to support health and positive health behaviors. This lack of resources will make the barriers to health promotion efforts much higher. Relative isolation can lead to disenfranchisement from mainstream political and economic processes (Emerson et al., 2001; Feldt et al., 2007). Withdrawal from these processes may limit the ability of residents to attract additional health-promoting resources into the area, contributing to restricted access to a broader range of care (Bach et al., 2004).

Summary

In sum, the evidence directly linking internalized, interpersonal, and institutionalized racism directly to hypertension is limited, though there are variations in findings depending on the ways in which these variables are operationalized and assessed (see D. R. Williams & Mohammed, 2009). The data suggest that interpersonal racism is associated with ABP, a measure that is sensitive to stress-related variations in BP. There are some studies that suggest that RRS is associated with elevated BP, but further research is needed to understand the degree to which the findings are a function of racial segregation specifically versus a function of the lack of resources and high levels of stress commonly associated with highly racially segregated communities. This is an important issue to understand, because there are some data suggesting that when individuals gain access to neighborhoods with more resources, they show a decrease in risk factors for hypertension (i.e., distress and obesity), though not in hypertension incidence itself. In addition, when comparisons are made between predominantly Black versus predominantly White neighborhoods with roughly equivalent resources, racial differences in the rates of some risk factors decrease. This finding suggests that examining resources and stress may be more critical than considering racial composition.

The clearest evidence suggests that racism may affect risk for hypertension through two primary pathways: (1) by increasing stress exposure and decreasing opportunities for recovery, and (2) by both directly and indirectly compromising the ability to engage in health-promoting behaviors.

All types of racism appear to increase stress exposure. Internalized racism has been directly associated with higher levels of perceived stress (E. S. Tull et al., 2005). Interpersonal racism has been directly, consistently, and uniformly associated with depressive symptoms and anger. Both stereotype threat and interpersonal racism also affect perceptions of new situations, such that a broader range of events become capable of eliciting a stress response. The physiological correlates of stress also appear to be influenced by exposure to racism, as evidence suggests that interpersonal racism is associated with greater cardiovascular reactivity to both race- and non-race-related stressors. This may partly explain the evidence linking racism to ABP, which captures daily stress reactivity.

As a consequence of institutionalized racism, Black individuals, even those with relatively high levels of income or education, are more likely to reside in segregated neighborhoods (D. R. Williams & Jackson, 2005). In comparison to neighborhoods in which White individuals predominate, neighborhoods in which Black individuals comprise a majority of the population are more likely to be deprived of social and economic resources. In part, this deprivation is a function of the withdrawal of the economic and social resources of Whites and other races unwilling to live in areas with

substantial proportions of Black residents. These effects are compounded by the relative economic disadvantage of Black households. Disadvantaged, or low-resource, neighborhoods increase exposure to acute and persistent stressors, including greater violence, crime, and noise.

Equally important, high-stress, low-income communities also present many fewer opportunities for stress reduction and recovery. The aesthetics of the environment do not provide respite, there are fewer recreational or cultural facilities that provide opportunities for casual relaxation, and there is often less social cohesion to promote social support among residents. This combination of greater stress exposure and fewer opportunities or supports for recovery is likely to create an overall stress burden that is much higher and more sustained for Black Americans than that experienced by members of other, less stigmatized ethnic groups.

There is also substantial evidence that racism influences health behaviors that increase the risk for hypertension. Internalized racism has been directly associated with abdominal obesity. Institutionalized racism indirectly affects health behavior through its effects on neighborhood SES. Low levels of community resources make the barriers to effective health promotion high. Consequently, very high levels of motivation and initiative are necessary for individuals to surmount these barriers and pursue beneficial health behaviors.

However, interpersonal and internalized racism raise the internal barriers to health promotion. Racism may indirectly influence the motivation to participate in health-promoting behaviors that either engender concerns about stereotype threat (i.e., because Blacks have been portrayed as incapable of succeeding in these arenas) or are seen as irrelevant to Black identity (i.e., because they are overly identified with White individuals or individuals of other races). Continued ostracism limits the type of self-awareness and self-regulation required to make consistent efforts at behavior change. The depressed and angry moods that are a persistent effect of interpersonal racism can sap the will to change.

CONCLUSION

Despite improvements in awareness and treatment, Black individuals remain at higher risk for the development of hypertension than do White individuals. At least a portion of this disparity is likely to be a function of racial disparities in individual- and community-level risk factors. However, some of the determinants of risk exposure appear to differ for Blacks and Whites, and racism may play a substantial role in fostering conditions that increase the likelihood that individuals will develop hypertension.

The risk factors for hypertension are interrelated. For example, variations in individual SES are linked to exposure to neighborhood stressors, as well as to individual psychosocial stressors and health behaviors. When individuals face multiple risk factors, a single-focus intervention (e.g., exercise training) may be inadequate. Multiple-risk-factor interventions (e.g., simultaneously addressing diet, exercise, and stress) may be needed. In addition, race-based variations in the relationship of each risk factor to HTN may affect the intensity of intervention required. The duration and level of support offered to individuals facing high environmental and intrapersonal barriers should be greater than those offered to individuals with more resources (Castro et al., 2009).

The effect of each risk factor may be moderated by other, co-occurring variables (e.g., stress may moderate the effect of diet on insulin levels). Stress reduction interventions, on an individual as well as community level, may be needed before other types of health promotion interventions can be successful. However, the sources of stress may vary by race or ethnic group, affecting the types of interventions needed. For example, risk reduction interventions for highly stigmatized groups may need to consider the broad range of interpersonal stressors that emerge as a function of exposure to racism. Interventions may need to address interpersonal relations, conflict management, and generalized depressive symptoms before they address health behavior.

In addition, the presence of some stressors may be a function of environmental or personal circumstances that are disproportionately associated with one group versus the other. Black

Americans are more likely than White Americans to live in high-density neighborhoods characterized by the presence of environmental-social stressors. Individual-level interventions to decrease stress responses may not be sufficient to manage persistent and high-intensity neighborhood-level stressors (e.g., crime or crowding). It may not be sufficient or appropriate to encourage relaxation in the face of some unjust environmental exposures if the intervention does not also provide an opportunity to advocate for changes in the social environment.

Individual-level interventions to change motivation depend on the ability to increase self-awareness and generate enthusiasm. These interventions may fail if they do not first consider the role race-based social ostracism plays in self-awareness and negative mood. Self-awareness can increase negative affect, and this may be intolerable for individuals who are already experiencing high levels of negative affect as a function of exposure to racism.

It may be necessary to first develop and implement individual-level interventions to identify and ameliorate the effects of racism on mood and self-regulation. In highly ostracized groups, interventions designed to change health behaviors may also need to consider the degree to which hypertension-related health behaviors are portrayed in the media and incorporated into notions of Black identity. Preliminary assessments of the degree to which individuals associate certain health behaviors with particular types of identity may need to be evaluated and addressed before motivational interventions can be implemented.

Individual-level motivational interventions may be irrelevant without community level interventions to increases access to health-promoting resources and to challenge race-related stereotypes about health behavior. Increasing access to improved neighborhood conditions appears to be associated with reductions in obesity and improvements in mental health (Kling et al., 2007; Morland et al., 2002). Targeted efforts to address identity-based or stereotype threat–based concerns about health promotion on a community-wide level may reduce intrapersonal barriers to motivation (Oyserman & Harrison, 1998; Steele, 1997).

In sum, data on ethnic and racial or class differences in the determinants of health behaviors have clear implications for interventions. For individuals facing low environmental barriers to making health-related changes, individual-level interventions focused on motivation and adherence may be appropriate. In contrast, for individuals facing high environmental barriers to health-related change, individual-level motivational interventions may fail or potentially lead to further distress or self-hatred. Environmental interventions aimed at barrier reduction may be both more appropriate and effective.

Careful research can guide the development and implementation of effective risk reduction efforts. More consistent methods and use of assessment tools will help clarify the causes for the mixed findings in much of the literature on race and hypertension. Research that examines effects both across groups and within specific groups is needed. Multidisciplinary approaches considering intrapersonal, interpersonal, and community-level risk factors are needed to achieve the goal of reducing hypertension across all groups.

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