

Perceived Racism and Blood Pressure: A Review of the Literature and Conceptual and Methodological Critique

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ABSTRACT

Racial disparities in health, including elevated rates of hypertension (HT) among Blacks, are widely recognized and a matter of serious concern. Researchers have hypothesized that social stress, and in particular exposure to racism, may account for some of the between-group differences in the prevalence of HT and a portion of the within-group variations in risk for HT. However, there have been surprisingly few empirical studies of the relationship between perceived racism and blood pressure (BP) or cardiovascular reactivity (CVR), a possible marker of mechanisms culminating in cardiovascular disease. This article reviews published literature investigating the relationship of perceived racism to HT-related variables, including self-reported history of HT, BP level, or CVR. Strengths and weaknesses of the existing research are discussed to permit the identification of research areas that may elucidate the biopsychosocial mechanisms potentially linking racism to HT. We hope to encourage investigators to invest in research on the health effects of racism because a sound and detailed knowledge base in this area is necessary to address racial disparities in health.

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INTRODUCTION

Racial disparities in health status, including excess rates of hypertension (HT) among Blacks, are widely recognized (1,2), but the causes of these disparities are poorly understood. Researchers have hypothesized that social stress, and in particular exposure to racism, may account for some of the between-group differences in the prevalence of HT (3–8). However, there have been surprisingly few epidemiological studies of the relation-

ship between perceived racism and blood pressure (BP) level or hypertensive status (6,9–13). Similarly, although the literature is growing, there have been very few published articles investigating the relationship of perceived racism to cardiovascular reactivity (CVR) and stress, a potential risk factor for HT (14–24).

The purpose of this article is to provide a critical review of the literature investigating the relationship of perceived racism or ethnic discrimination to HT and related variables. The review is divided into two sections. The first section examines the findings and the methodological limitations of studies addressing the primary question: Is perceived exposure to racism associated with elevated BP? Particular attention is paid to issues related to the measurement of perceived racism and to the potential mediators and moderators of the relationship of racism to BP, including coping and socioeconomic status (SES). The second section of the paper provides a critical review of the literature examining the relationship of racism to CVR. These studies emerge from a research tradition investigating the hypothesis that the relationship of psychosocial stress to HT is mediated through changes in autonomic modulation (25–28) and may shed light on the biopsychosocial mechanisms linking perceived racism to elevated BP. When reviewing articles on CVR, particular attention is paid to the nature of laboratory models of racism and the interpretation of reactivity data.

The review is limited to published peer-reviewed articles on these topics. References were gathered using PsycINFO, MEDLINE, ERIC, and Sociology Abstracts and submitting the key words *ethnic discrimination, prejudice, racial discrimination, or racism* in combination with additional key words including *blood pressure, cardiovascular reactivity, heart rate, or hypertension*. Other references were obtained by checking the bibliographies of each article. This search strategy yielded a total of 6 articles (6,9–13) investigating the relationship of perceived racism or ethnic discrimination to self-reported history of HT or observed BP level and an additional 11 articles investigating the relationship of racism to CVR (14–24).

Definitions of Racism

Clark et al. (26) defined racism as “the beliefs, attitudes, institutional arrangements, and acts that tend to denigrate individuals or groups because of phenotypic characteristics or ethnic group affiliation” (p. 805). Contrada and others (29,30) used the more general term of *ethnic discrimination* and defined it as unfair treatment received because of one’s ethnicity, where *ethnic-*

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ity refers to various groupings of individuals based on race or culture of origin.

Related terms include *prejudice* and *discrimination* (31). Prejudice is an attitudinal dimension and consists of negative or stigmatizing beliefs about a particular group. Discrimination refers to behavioral practices ranging from social distancing (i.e., exclusion or rejection) to aggression. We prefer the term *racism* because recent usage (26,32,33) encompasses both attitudinal and behavioral phenomena.

Recently, investigators have suggested that the effects of racism can be examined within the framework of the stress and coping theories of Lazarus and others (34,35). These theories suggest that the effects of exposure to a stressor are mediated through the individual's appraisals of and coping responses to the stressor. Consequently, this article focuses on the effects of perceived or self-reported racism, specifically the perception that one is being targeted for negative treatment because of ethnicity.

Perceived racism can reflect exposure to individual or institutional racism, communicated through attitudes or behaviors (32,33). Reports of exposure to racism may also reflect perceptions of racial bias based on the outcomes of interactions with employers, the criminal justice system, or other institutions. These outcomes can lead to perceptions of being exposed to racism even when the direct interpersonal interactions with members of these institutions (e.g., supervisors, police officers, etc.) have not been perceived to be directly discriminatory.

It is important to note that perceived experiences of racism may generate only a portion of the health effects of racism. Discrimination that is not directly perceived may also produce changes in health status. For example, individuals may experience economic deprivation that is a result of racism, but they may not consider their economic circumstances as determined by race-related bias. Economic deprivation may impair cardiovascular health whether it is a function of racism or not (36–38). Thus, evidence based on self-reported perceptions of racism may underestimate its effects.

Epidemiological Studies

Studies of skin tone and BP. Early epidemiological studies indirectly examined the health effects of racism by studying the relationship of skin tone to HT (39–42). Initially, skin tone was regarded as a marker for genetic risk for HT by some researchers (42). However, it has also been used as an indirect index of exposure to discrimination (43), because darker skin tone has been associated with lower SES in large-scale studies (41,44). This economic deprivation was hypothesized to be partly a function of racial discrimination (44).

Some studies examining the relationship of skin tone to HT suggested that darker skin tone was associated with elevated BP (39,41,45). However, not all studies have found these effects for all groups (42,46,47), and other studies have suggested that the effects of skin tone on BP may be moderated by gender (48), social class (42,49), or country of residence (50), suggesting that the effects of skin tone on BP are at least partly a function of environmental circumstances, (i.e., the meaning and social consequences of darker skin).

Skin tone cannot serve as a proxy for perceived racism or discrimination because the data on the relationship of skin tone to perceived racism are mixed (43,44). Although a recent study reported that Black Americans with dark skin tones were 11 times more likely to report discrimination than those with relatively lighter skin tones (43), another large-scale epidemiological study reported that skin color was *not* significantly associated with the reports of discrimination in most venues (44). To date, studies of the relationship of skin tone to BP have not explicitly tested the hypothesis that the association of skin tone to BP is mediated by perceived racism.

Studies of perceived racism and BP. Six studies have explicitly investigated the relationship of perceived racism or discrimination to HT or BP level. One of these (13) evaluated the degree to which variations in exposure to discrimination accounted for Black–White differences in BP level. All six studies examined within-group effects, assessing the degree to which relative differences among minority group members in their exposure to racial discrimination were associated with variations in hypertensive status or BP level.

In the first of two studies, Krieger (6) asked 101 women (51 Black) in Alameda County California if they had “ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior because (they were) a woman/because of (their) race” (p. 1274). The dependent variable was self-reported history of HT. Black women who denied any experience with discrimination were 2.6 times more likely to be at risk for high BP than Black women who reported one or more occurrences.

In a more extensive investigation (CARDIA), Krieger and Sydney (13) studied a larger sample ($N = 4,086$) of young Black and White women and men. Participants were asked if they had ever been exposed to discrimination in one of seven venues. Within each gender, SES, and racial group, participants were divided into subgroups depending on their exposure to zero, one or two, or three or more incidents of racism. Careful measures of resting BP (using a random zero sphygmomanometer) served as the dependent measure.

To evaluate between-group effects, Black–White differences in BP were estimated at each level of exposure to discrimination. For working-class women, Black–White differences in BP were smallest at moderate levels of exposure to discrimination (i.e., exposure to one or two incidents). The greatest differences were seen at the lowest level of exposure, (i.e., there was almost a 7 mmHg gap between the systolic BP [SBP] of White vs. Black women who reported no discrimination).

To evaluate within-group effects (i.e., the degree to which variations among Blacks in the intensity of exposure to racism were associated with variations in BP), analyses estimated differences in average BP among groups varying in exposure, as well as coping style, gender, and SES. Within Blacks, groups varying in their level of exposure to discrimination displayed differences in average SBP ranging from 2 to 6 mmHg. In a finding similar to that seen in the earlier Krieger (6) study, among Black working-class men and women, and among pro-

fessional-class women, those who reported no incidents of racial discrimination had higher SBP and diastolic BP (DBP) than those who reported moderate levels of exposure (i.e., one or two incidents). However, members of these groups who reported higher levels of exposure (i.e., three or more incidents) also had higher BP than those who reported moderate levels. The findings varied for professional-class men. In this group, exposure to no discrimination or to relatively high levels of discrimination was associated with lower SBP and DBP than moderate exposure. Additional variations among groups depended on coping style. Krieger and Sidney (13) interpreted the findings as suggesting that the strategies used to cope with racism, including denial and anger suppression, complicate attempts to assess levels of exposure and influence the relationship of exposure to BP.

Broman (9) conducted a cross-sectional study to examine the relationship of discrimination to health problems. Members of a random sample of 312 African American adults (67% female) in the Detroit, Michigan area were asked if they had ever experienced discrimination (*yes* or *no*) in several settings (e.g., getting a job, at work, while shopping, from the police, etc.) over the past 3 years. Respondents were also asked if they were ever told that they had either high BP or heart disease. After controlling for SES, reports of discrimination did not predict health status. There were no moderating effects of gender.

Three studies examined the effects of work-related discrimination on BP (10–12). James, LaCroix, Kleinbaum, and Strogatz (11) performed the first study to examine the relationship of perceived racism at work to BP in a sample of 112 Black men. Perceived racism at work was measured with a single dichotomous item, one level of which reflected the belief that “being Black hindered them at work,” and the other level reflected the belief that “being Black had helped them at work.” The average of two BP measures taken during a structured interview served as the dependent measure. There was no main effect of perceived racism on BP, however, there were interactions among perceived racism, job success, and “John Henryism,” a measure of persistent, active coping. Among the men who had obtained relatively greater job success and who were high in John Henryism, those who reported perceiving discrimination at work had higher DBP than those who did not report discrimination.

James, Lovato, and Khoo (12) examined the relationship of perceived prejudice and discrimination on the job and resting BP in a sample of 89 employees, who were primarily Mexican American (64%) or Black (18.1%). Participants completed a psychometrically sound 16-item organizational prejudice–discrimination measure that assessed the degree to which employees felt targeted for prejudice and discrimination at the worksite. The average of two BP measurements (SBP and DBP were combined to form a single index) was the dependent measure. Analyses revealed a small positive correlation between the perceived intensity of exposure to prejudice and BP levels ($r = .20, p < .05$), controlling for weight, age, organization type, and self-esteem.

Dressler (10) examined the association of perceived racism at work in 90 Black individuals. The measure of perceived racism included four items assessing the degree to which participants perceived that pay raises and other work-related issues

were based on race. Although perceived racism scores were associated with other indices of stress, they were not directly associated with BP measures taken at the participants’ homes using aneroid sphygmomanometers.

In sum, six studies explicitly examined the relationship of perceived discrimination or racism to HT-status or BP level, with three of the studies focusing on workplace discrimination. The CARDIA study (13) suggested that the level of exposure to racism influenced Black–White differences in BP level. Mixed findings were reported among studies that examined within-group relations of perceived racism to BP. Three studies (11–13) revealed a positive association of perceived racism to BP in at least one group of participants, but for two of these studies the effects depended on either the coping style (11) or the gender and social class of the participants (13). In contrast, three studies (including one described previously) found no main effects of perceived racism on BP (9–11), and two studies reported an inverse effect, with lack of exposure to discrimination associated with higher BP for some groups of participants (6,13). Three studies provided evidence suggesting that coping may influence the relationship of racism to BP, and that the effects of racism on BP vary by both gender and social class (6,11,13).

These pioneering studies provided some insight into the complexity of the relationship of racism to BP; however, the methodological limitations concerning the measurement of racism and BP limit their interpretability. There are also difficulties with the strategies used to analyze the roles of coping and SES, which may mediate, moderate, or add to the racism–BP relationship. The methodological and interpretive issues are addressed in the following section.

METHODOLOGICAL ISSUES

Issues of Measurement

Assessing exposure to discrimination. In several of the epidemiological studies described earlier, the ability to examine within-group relations was limited by the measures of perceived racism. In three studies (6,9,13), the measures asked participants if they had been exposed to discrimination in one of several settings. Participants were divided into two or three categories of exposure, depending on the number of venues in which they had been exposed. A fourth article (11) used a single item measure inquiring if the participant believed that “being black had hindered their success.”

These measures may be relatively insensitive to within-group or individual differences in exposure because the prevalence of exposure to racism was very high. The vast majority of Black individuals in these studies had been exposed to discrimination at some point in their lifetime. For example, Krieger and Sidney (13) reported that 77% of Black women and 84% of Black men reported discrimination in at least one setting. Broman (9) found that, overall, 60% reported discrimination in any setting over the past 3 years.

Adequate within-group tests of the relationship of exposure to racism to BP require more sensitive and well-studied measures. In five of the six studies (6,9–11,13), no information was provided regarding the psychometric properties of the mea-

asures. The one study employing a psychometrically sound measure of workplace racial discrimination yielded the clearest evidence of a small, but significant, positive within-group relationship of perceived racism to BP level (12). Specifically, the measure used in the James et al. (12) study included 16 items evaluating a range of discriminatory experiences in the workplace. Previous psychometric testing had indicated that this scale was likely to be a reliable and valid index of individual differences in level of exposure to discrimination (51).

A number of new self-report measures, including The Schedule of Racist Events (52) and the Perceived Racism Scale (53), among others (30,51,54–61) can also permit a more sensitive measurement of exposure to racism across everyday situations, including the workplace. Detailed psychometric testing has been performed on several of these measures (see 62 for review). Several of these scales (30,52,61,63) measure the frequency, intensity, and duration of exposure (see 62). Items inquire about exposure to both obvious and subtle forms of discrimination and assess exposure to a variety of attitudes and behaviors including harassment, rejection, stigmatization, and discrimination. This is useful because even relatively limited exposure to overtly discriminatory events may have health consequences, given that these events may alter the individual's appraisals of, and response to, future events (64,65). In addition, chronic exposure to subtle or low-intensity episodes of race-related rejection, exclusion, or aggression may have deleterious effects as well (66).

Despite adequate psychometrics and comprehensive surveys, assessing exposure to racism can be problematic. There are a variety of personal factors that may result in the tendency to minimize or maximize reports of exposure to racism. Research indicates that individuals are reluctant to report being victims of discrimination of any kind because they associate victim status with loss of control (67). Personality dimensions such as a tendency toward defensiveness or denial may also be related to the tendency to minimize reports of stress (68) or racism (6). On the other hand, variables including negative affectivity and trait anxiety have been associated with increased reporting of stress-related symptoms (69) and may increase reports of racism or racism-related distress as well. Trait anger may increase sensitivity to interpersonal racism because anger is the emotion most commonly associated with exposure to racism (52).

The direction and nature of the relationship between exposure to stress, personality characteristics, and subjective reports of distress is not known and is a source of controversy (35). Exposure to racism has been associated with distress and anxiety (29,70), which may mediate the relationship of racism to BP. However, distress and anxiety may also increase the tendency to perceive situations as threatening and potentially racist. Additional research is required to understand the effects of personal characteristics, including attitudes, affective state, and coping style on self-reports of racism. The inclusion of measures of these personal characteristics may aid in the interpretation of the effects of perceived racism on BP and permit investigators to

disentangle the effects of exposure to stress from those associated with efforts to cope with the stress.

Assessing BP and HT. There also have been limits to the assessment of BP. In two of the studies (6,9) the measures are limited to self-reports of a history of HT or to a history of any kind of cardiovascular disease (CVD). These self-report measures are problematic because there have been several reports that a significant proportion of individuals are unaware that they have high BP (71,72). In both the James studies (11,12), multiple automated readings of BP were obtained, enhancing the reliability of measurement over self-reports. Unfortunately, in the 1994 study (12), measures of SBP and DBP were combined into a single variable, a relatively unusual strategy and one that obscures the ability to compare across studies. In the 1984 study (11), a commercial device measuring BP from a fingertip was used; these devices are limited in their ability to provide valid BP measurements.

In future research, measures of ambulatory BP (ABP) may be useful for obtaining a more reliable index of elevated pressure. Average 24-hr ABP and BP load have been shown to be more closely related than clinic readings to cardiovascular morbidity and mortality (73). Researchers have successfully used ABP monitoring to examine the cardiovascular correlates of a variety of other psychosocial stressors (see 74 for a review).

Potential Mediators and Moderators

Coping. Models of the relationship of stress to health suggest that the health effects of a stressor such as racism may depend in part on the strategies used to cope with exposure. However, the dimensions of coping have been limited. The mediating, moderating, or additive nature of the relationship of coping to racism has yet to be determined.

In studies of the association of coping to racism and BP, investigators have focused primarily on anger coping and active coping. In two studies (6,13) participants were asked to report how they typically coped with discrimination or unfair treatment, indicating whether they tended to actively cope with discrimination (i.e., "accept it as a fact of life" vs. "try to do something about it") and identifying their anger management style (i.e., "talk to other people about it" vs. "keep it to yourself"). Broman (9) asked participants to indicate how they typically respond to discrimination. James et al. (11) assessed coping with the John Henryism scale, a measure of active coping.

Data from three studies provide partial support for the notion that anger and active coping play a role in determining the cardiovascular correlates of racism. For example, in two studies (6,13), Black working-class women who accepted and kept quiet about unfair treatment were much more likely to report a history of HT or to have relatively higher BP levels than were those who reported that they were more active and spoke to others. In the third study, James et al. (11) reported that the effects of racism depend on the level of John Henryism, such that among men high, but not low, in John Henryism, perceived rac-

ism is associated with relatively elevated DBP. Of the remaining studies, one study found no effects of coping (9), and two did not explicitly examine the effects of coping on the relationship of racism to BP (10,12).

The nature of the relationship between exposure to racism and coping remains unclear. Coping may mediate or moderate the relationship of racism to BP, or coping may add to the effects of racism on BP. A mediator model suggests that the stress of perceived racism determines the nature of the coping responses, and these coping responses explain any observed association of racism to BP (75). For example, Krieger (6) suggested that exposure to racism fosters a need to suppress awareness and expression of feelings and has reported that Black women who reported no exposure to discrimination were more likely to report that they accepted mistreatment and kept quiet about it. These findings are consistent with the work of theorists who argue that racism limits access to power, status, and resources—all variables that influence the use and effectiveness of different coping responses (64).

Alternatively, coping may moderate the relationship of racism to BP, with the choice of coping style independent of exposure to racism. Some coping styles would be expected to attenuate the relationship of racism to elevated BP, whereas others would be expected to strengthen it. James et al. (11) provided explicit tests supportive of the moderator hypothesis, but the sample and measures are too limited to draw definitive conclusions. Finally, the effects of coping may simply add to those associated with exposure to racism. Individuals who employed strategies such as anger suppression, independently associated with elevated BP (76), would be expected to have higher BP than those who did not suppress anger at any level of perceived racism. In the CARDIA study, Krieger and Sidney (13) reported that the effects of discrimination were independent of the choices of coping strategy and that the association of racism to BP varied depending on coping strategy. These data are more consistent with an additive or moderator model.

There is still very limited empirical research on the relationship of race-related stressors to coping strategies, and the existing findings appear to conflict. In addition, in several studies (6,13), the measures of anger coping are confounded with those of active coping. Better measures of coping will permit the separation of effects associated with anger management from those associated with active coping. In addition, improved measures will permit investigators to determine if the relationships among racism, coping, and ABP depend on either the type of perceived racism or the type of coping strategy.

Explicit tests of the nature of the relationship of exposure to racism and coping strategies are needed. A variety of new strategies, including structural equation modeling, can explicate the relations among racism, coping, and BP (77). These data can provide guidance for clinicians developing programs to reduce potential health effects of racism.

Evaluating the role of SES. Two studies suggest that SES influences the relationship of racism and coping to BP, although

the effects are not consistent (11,13). James et al. (11) reported that the interactions of racism and John Henryism emerged only among men who reported relatively greater job success. Krieger and Sidney (13) subdivided large groups of participants into working and professional classes. Complex SES group differences in the relationship of both exposure to racism and coping to BP were observed. For example, Black–White differences in BP are greater in working- versus professional-class participants. In particular, the consequences of speaking out about racism appear to differ by class, such that accepting racism as “a fact of life and keeping it to oneself” was associated with higher BP among working-class but not executive-class Black women. However, explicit tests of interactions among SES, racism, and coping were not performed.

As was the case for coping, it is not yet known if SES exerts a mediating, moderating, or additive effect on the relationship of racism to BP. Poverty comes with its own set of stressors (e.g., overcrowding), which may independently affect risk for HT (8,37). These stressors may combine with the stressors associated with racism (e.g., interpersonal maltreatment) to produce overwhelming demands for coping. At the same time, poverty may limit the availability of coping resources or their effectiveness (78,79). If SES either moderates or adds to the effects of racism on BP, one possibility is that racism would be associated with poorer health outcomes (e.g., high BP) primarily among individuals with a low versus a high SES.

Because racism and poverty have been closely intertwined, it is also feasible that SES mediates the relationship of racism to health. Racism may block economic access and partly account for the finding that racial and ethnic minority groups in the United States—particularly Blacks and Latinos—are disproportionately represented among low SES populations (80).

Despite the close interconnection of racism and poverty, the complexities of the relationship can be articulated and investigated. Krieger, Williams, and Moss (81) highlighted the importance of measuring SES through multiple indices (e.g., education, occupation, and income) and on multiple levels (e.g., individual, family, and neighborhood). This comprehensive approach is necessary to capture the full range of resources available to the individual (82). Using multiple indicators is particularly important when studying diverse racial and ethnic groups because indicators of SES are not equivalent across groups (81). In addition, studies of perceived SES, or related variables such as lifestyle incongruity, may also shed light on the influence of SES on health (83).

Developing a More Comprehensive Model

Five of the six studies reviewed here focused on the effects of discrimination on BP in Blacks (6,9–11,13). The emphasis on the effects of discrimination in Blacks is reasonable given the high prevalence of HT in this group. However, it is important to build a more comprehensive model of the effects of discrimination.

Members of many different ethnic groups have experienced aspects of both institutional and interpersonal discrimination

(84), and it is reasonable to expect that social and economic discrimination will have health effects across groups. Yet, there has been no empirical research examining ethnic group differences in the relationship of racism to BP. Similarly, no data have been reported examining the differences among subcultures within minority populations (i.e., foreign born vs. American born, or West Indian vs. European Blacks). This may be relevant because immigration status (e.g., voluntary vs. involuntary) can also affect perceptions of discrimination (85,86).

The causes for disparities in HT among minority groups remains unclear. One possibility is that the relationship between exposure to racism and BP is not linear. Instead there may be threshold effects, with minimal health consequences below a certain level of exposure. Alternatively, ethnic differences in diet, SES, social support, or other factors may moderate the relationship of racism to BP, potentially mitigating some of the harmful effects.

One of the difficulties facing investigators has been a lack of tools for measuring exposure to discrimination in multiple ethnic groups. Most measures of exposure to racism were developed for Blacks (62). A new measure, the Perceived Ethnic Discrimination Questionnaire–Community Version, can be used across ethnic groups, and assesses the types of everyday experiences of discrimination common to members of all minority groups (29,30,85,87). Research on the effects of discrimination on BP in multiple ethnic groups would permit the development of a body of research assessing the generalized effects of discrimination, while permitting evaluation of effects specific to each group.

LABORATORY RESEARCH

Much important early work on the determinants of race differences in risk for HT in the area focused on Black–White differences in CVR to stress (5,25). This literature has been reviewed elsewhere (5). In general, research has shown that Blacks have greater BP and heart rate (HR) reactivity than Whites in response to most, but not all, lab stressors (88–93). There also appears to be some evidence of race differences in the pattern of cardiovascular responding, with Whites displaying greater myocardial activation and Blacks displaying greater vascular responsivity (94). However, these studies do not explicitly test the hypothesis that Black–White differences in psychophysiological response are accounted for by race differences in exposure to racism.

Researchers have also used CVR studies to directly examine the association of racism to risk for HT (14–24). Three types of questions have been addressed by psychophysiological studies of racism: Is acute exposure to laboratory models of racism associated with an increase in BP or HR? Is exposure to racist versus nonracist stressors associated with greater reactivity in minority participants? Are past experiences of racism associated with greater physiological responses to current stress, and do the effects depend on the racist versus nonracist nature of the stressor?

Several studies have examined the effects of simulated exposure to racism on CVR. Investigators exposed participants to

racist stimuli in a laboratory setting using a variety of methods including racist video or film scenes (14,16,22), slides depicting interactions among individuals of different races (20,23), speech or debate tasks (15,18,21), imagery tasks (19,24), and harassment from a White experimenter (17). These strategies were effective in producing significant increases in electrodermal activity (23), corrugator muscle tension (19,24), SBP and DBP (14,16–18,21,22), and HR (18,19,21,24).

Six studies examined the degree to which racist stimuli elicited greater or more sustained increases in BP or HR than anger-evoking but nonracist stimuli. Findings of larger increases in BP associated with racist stimuli would strengthen the claim that exposure to racism is a particularly pathogenic influence on BP. In four of the studies (14,18,21,23), racist stimuli provoked greater increases in sympathetic activation than the nonracist stimuli. However, the Fang and Myers (16) study failed to find differences in CVR between the racist and nonracist provocations, and there were no significant differences in the level of anger evoked by these two stimuli for Blacks. Similarly, Sutherland and Harrel (24) reported no significant differences in HR reactivity in responses to a fearful versus racially noxious scene.

A relatively new and promising research strategy examines the relationship between past exposure to racism and current CVR in response to both race-related and non-race-related stressors. These studies test the hypothesis proposed by Matthews and others (95,96) that background stressors, including racism, may sensitize individuals to future stressors. This model specifically predicts that past exposure to a stressor may be associated with increased reactivity to a new stressor. Data from two studies provide support for this hypothesis.

A recent study by Clark (15) is the first to examine the relationship of perceived racism to CVR in response to an interpersonal stressor. Participants included 39 African American female students. Perceived racism was assessed using the Perceived Racism Scale (63). BP was measured at baseline as well as before, during, and after participants spoke about their personal views on a topic unrelated to racism (e.g., animal rights). Clark found that past exposure to perceived racism was positively related to DBP changes during speech and associated with reduced recovery following speech.

Guyll, Matthews, and Bromberger (18) examined the effects of past exposure to both blatant and subtle interpersonal maltreatment, and the effects of past exposure to discrimination on CVR. Their measure of past exposure to discrimination was somewhat limited (i.e., participants were asked if they attributed their prior exposure to interpersonal maltreatment to racism vs. a number of other unrelated variables, such as age or SES). Participants included both Black and White women. They completed a nonracist laboratory task (i.e., mirror tracing) and a potentially race-related task (i.e., a speech stressor in which the participants must think about and then deliver a speech defending themselves against an unfounded charge of shoplifting.)

Blacks reported higher levels of exposure to maltreatment than Whites. Only 39 of 91 Black participants reported that this maltreatment could be attributed to racism. The 39 women who reported exposure to racism displayed higher average baseline

HRs and had higher average DBP reactivity than the 62 who did not attribute maltreatment to racism. These effects were specific to the potentially racist speech-stressor task. It was during this task, but not the nonracist mirror-tracing task, that Black women who reported attributing maltreatment to racism had higher DBP reactivity than those who did not.

Laboratory studies of racism permit the examination of changes in cardiovascular activation under controlled circumstances. All studies reported that acute exposure to depictions of racism was associated with increases in several indices of autonomic activation. Among Blacks, most, although not all, findings indicate that racist stimuli are associated with greater increases in activation than other types of provocations. In the cases in which participants did not demonstrate greater arousal to the racist stimuli, the contrasting stimuli were designed to evoke high levels of fear or anger. Recent work suggests that past exposure to racism may influence perceptions of, and psychophysiological response to, new challenges, including both racist and nonracist stressors (15,18).

LIMITATIONS

In-Vivo Measures of Exposure to Racism

Characteristics of the racist stimuli may influence the affective or coping responses elicited, and consequently affect the cardiovascular response. Most of these laboratory studies included presentations of overt racism (e.g., accusations of shoplifting or Ku Klux Klan, etc.). However, overt discrimination is only one dimension of racism. Strategies for classifying dimensions of racism, reviewed in detail elsewhere (32,33), suggest that racism can be expressed both overtly and covertly and as attitudes and behaviors.

Each dimension or expression of racism may elicit different patterns of coping with different autonomic correlates. For example, the emotional and cardiovascular correlates of exposure to ethnicity-related aggression may differ from those associated with ethnicity-related exclusion or rejection. Only one study examined the differences between blatant and subtle racism (19) and reported that blatant racism evoked higher HR reactivity. Further studies are needed to clarify the patterns of psychological and physiological coping associated with these different types of racist provocations.

Limitations to the Interpretation of BP Reactivity Data

The interpretation of the BP reactivity data is unclear because it is not known if the BP reactivity seen in the laboratory in response to depictions of racist interactions would be elicited during "real-world" interactions perceived to be discriminatory. It is also not known if these stress-induced increases in autonomic activation are sufficient to produce significant and ongoing changes in cardiovascular function (97). Equally problematic is the fact that there is almost no epidemiological evidence indicating how often individuals face different types of racism. If these events are experienced relatively frequently, even a small increase in activation might have significant health effects. However, if these events are experienced relatively rarely,

then even major increases in arousal might not increase risk for HT.

Studies of multiple markers of autonomic changes are also needed. The biological pathways by which exposure to stress leads to CVD, including HT and cardiac events, still remain controversial (98). This is due, in part, to the multiple physiologic pathways that may be involved and that operate in concert. CVR may simply be a marker for other processes that have not yet been studied in this context, including platelet aggregation and endothelial function lipid metabolism (99–101).

SUMMARY AND CONCLUSIONS

The existing data on the relationship of racism to BP level or HT status are mixed. These pioneering studies provide important insights and guidance, but methodological limitations limit their interpretability and are likely to account for the inconsistent and relatively weak findings. A comprehensive evaluation of the relationship between racism and BP status has yet to be conducted. Findings from CVR studies are clearer. These studies suggest that acute exposure to racism is associated with increases in cardiovascular activation. In addition, past exposure to racism may influence current CVR to race-related and other stressors.

Progress in understanding the health effects of racism will not be made without adequate attention to issues of measurement. Strategies for assessing perceived racism must include more systematic assessment of exposure to multiple dimensions of racism, including discrimination, stigmatization, social distancing, and threat and aggression. Improving the measurement of HT-related variables, possibly through the use of ABP monitoring, may also help clarify the acute and chronic effects of race-related stress on BP. Ethnicity, SES, coping, and personal characteristics may influence the effects of racism on health. Consequently, we need to systematically assess these constructs to fully understand the ways they may influence the relationship of racism to HT.

Racism is a multifaceted experience likely to affect health through several different pathways (5,8,26,102). Frequent, negative, and potentially race-related interpersonal exchanges can have a cumulative toll on health because they may elicit repeated demands for both anger and active coping. Attributional processes may influence the degree to which individuals anticipate these encounters and believe they can cope with them. Studying racism and race-related negative interpersonal interactions can provide a model system for the investigation of the ways in which our cardiovascular system regulates and is regulated by our social behavior and our perceptions of others.

Coping may influence the relationship of racism to BP, but the nature of this relationship is not yet clear. Coping may affect both the reporting of racist events and the impact of these events on health. It may be difficult to separate these effects using cross-sectional designs, and intervention studies may be required. Better measures of distinct dimensions of coping and new tests of the relationship of racism to coping will provide the knowledge base needed to develop appropriate coping-based interventions.

SES may mediate, moderate, or add to the effects of racism on BP, however, the measurement and data analytic strategies used in previous research were not sufficient to clearly articulate the relationships among racism, SES, and HT. Further research using structural equation modeling or longitudinal paradigms is necessary to clarify these relationships and to help target interventions to those most in need.

The laboratory work on the effects of acute exposure to racism has great promise as well. An examination of PsycINFO reveals that there were many dissertations on this topic in the last 5 years, and as these studies are published, they will help to clarify many important questions. To help interpret these reactivity data, basic epidemiological data are needed to generate reliable estimates of the frequency and intensity of exposure to episodes of racism during the course of everyday life. In addition, further studies comparable to that of Clark (15) and Guyll et al. (18) are needed to understand the ways in which past exposure influences cardiovascular response to a range of everyday stressors.

Although progress has been made, exposure to racism is still a painful part of life for many Americans. We know too little about the psychological and physiological consequences of exposure to this stressor. Behavioral medicine researchers can shine the light of empiricism on the difficult and serious problem of racism, and in doing so, generate the knowledge needed to reduce racial disparities in health.

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