STRESS-RELATED RACIAL DISCRIMINATION AND HYPERTENSION LIKELIHOOD IN A POPULATION-BASED SAMPLE OF AFRICAN AMERICANS: THE METRO ATLANTA HEART DISEASE STUDY

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INTRODUCTION

African Americans experience disproportionately higher rates of hypertension, as well as earlier age of onset and higher concomitant morbidity and mortality from hypertension when compared to any other race/ethnic group in the United States.1 Explanations for this disparity vary from theories of a slavery-based etiology to those associated with lower quality of health care and adverse environmental factors.2–4 Exposure to incidents of perceived racial discrimination has also been identified as a potential biopsychosocial risk factor that may account for higher rates of hypertension observed in African Americans.5–7 Given its ubiquitous historical and contemporary prevalence, stress derived from incidents of racial discrimination may be related to hypertension in African Americans.7–8 This theoretical concept is buttressed by studies that demonstrate the deleterious physiologic effects of racial discrimination as a stressor attributable to elevated blood pressure9–13 and those from survey findings that document a high prevalence of exposure to racial discrimination.14–17 Although overall rates of hypertension may indeed be higher when compared to other race/ethnic groups, hypertension is not uniformly prevalent in African Americans – despite the confounding high prevalence of exposure to racial discrimination.

Research related to various aspects of potential mechanistic relationships and etiologic correlates of stress-related racial discrimination and elevated blood pressure in African Americans is increasingly being investigated.6,18 Measurement of association, however, is complex, in light of the subjective construct of racial discrimination as a perceived individualized experience. Results from population-based, cross-sectional studies have been mixed.18 A number of studies, for instance, fail to demonstrate a positive correlation between perceptions of racial discrimination and blood pressure or hypertension.19–21 Others, on the other hand, demonstrate a positive association,13–22 while still others suggest that the relationship may be an independent function of social position or gender,12,23 as well as differential coping style.24,25 Although informative, findings from these studies are limited in that they are often based on a heterogeneous biracial study population of Black and White participants and generally include sociodemographic attributes or theoretical proxy indicators that do not measure the direct effects of the degree of perceived stress derived from an encounter of racial discrimination.

The magnitude of response to a stressful event is influenced by subjective cognitive perception and judgment of respective stress exposure.26 Surprisingly, we were unaware of any published investigations designed to yield insight regarding the confounding

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Objective: Exposure to racial discrimination has been identified as an adverse biopsychosocial stressor that may be related to the prevalence of hypertension in African Americans. The overall objective of this research was to estimate aspects of the effects of self-reported exposure to stress-related racial discrimination and hypertension likelihood in a sample of African Americans.

Data: Physiologic and self-reported cross-sectional data were collected on a stratified sample of hypertensive (n=174) and normotensive (n=182) African Americans, aged ≥21 years, residing in metropolitan Atlanta, Georgia from 1999 through 2001.

Method: Separate, multivariate logistic regression models were fit, including relevant covariates, to ascertain the effects of exposure to incidents of stress-related racial discrimination and level of generally derived stress with hypertension likelihood.

Results: Exposure to incidents of racial discrimination was not significantly associated with an increased likelihood of hypertension. More than 74% of respective normotensive and hypertensive respondents reportedly experienced stress-provoking encounters of racial discrimination. Magnitude of stress generally derived from exposure was, however, a highly significant predictor. Respondents reporting “moderate” and a “high to very high” level of derived stress were more than twice as likely to be hypertensive when compared to those reporting “no to low” derived stress (P value=.02 and .01, respectively).

Conclusion: Exposure to racial discrimination is a prevalent psychosocial stressor in African Americans but may not be significantly associated with hypertension prevalence; degree of stress derived from encounters may be an important determinant. More research is needed to clarify the complex relationship between stress-related racial discrimination and hypertension in African Americans. (Ethn Dis. 2005;15:585–593)

Key Words: African Americans, Blacks, Ethnicity, High Blood Pressure, Hypertension, Minority Health, Prejudice, Race, Racial Discrimination, Racism, Stress
paradoxical issue of higher exposure to incidents of stress-related racial discrimination and differential hypertension prevalence in African Americans. We hypothesized that differential perception regarding the magnitude of stress generally derived from incidents of racial discrimination may be related to hypertension likelihood.

The overall objective of this research was to estimate the effects of self-reported exposure to stress-related racial discrimination and perceived degree of stress generally derived from encounters with hypertension likelihood in a sample of African Americans. To the best of our knowledge, this study is among the first of its kind composed of a stratified sample of hypertensive and normotensive African Americans. Information gleaned from these findings provide new insights that may help advance the growing public health field of research aimed at understanding the potential relationship between racial discrimination and hypertension in African Americans.

MATERIALS AND METHODS

Study Population and Sampling Frame

Study findings are based on cross-sectional data collected on a stratified sample of hypertensive and normotensive respondents that participated in the Metro Atlanta Heart Disease Study (MAHDS). The MAHDS is a pilot study designed to comprehensively measure sociodemographic, psychosocial, lifestyle, physiologic, and anthropometric correlates of heart disease and hypertension in African Americans, including self-reported measures of stress and racial discrimination; some findings have been previously reported. Recruitment activities and data collection took place from 1999 to 2001 on individuals residing throughout metropolitan Atlanta, Georgia. All data elements were collected at the Morehouse School of Medicine Clinical Research Center. Inclusion criteria for participation in the study included the following parameters: English-speaking, self-identified as African American and born in the United States, 21 years of age or older, and hypertensive or normotensive. Individuals were excluded if they were pregnant, had congenital heart disease, kidney disease, or had severe physical or mental limitations. Customary guidelines in place at the time of data collection were used to define hypertension. Participants with a mean systolic or diastolic blood pressure reading of ≥140 mm Hg or ≥90 mm Hg, respectively, or currently taking prescribed antihypertensive medication were defined as hypertensive. Those with a mean systolic or diastolic blood pressure reading of <140 mm Hg or <90 mm Hg, respectively, and not currently taking prescribed antihypertensive medication were defined as normotensive.

A total of 521 individuals were prescreened and deemed eligible for participation. Six were later determined to be ineligible; 515 were contacted and scheduled for data collection. A total of 356 individuals subsequently reported for data collection, thereby resulting in an overall response rate of 69.1%. The final study population included 174 hypertensive respondents and 182 that were normotensive. The research protocol was reviewed and approved by Morehouse School of Medicine’s institutional review board. A signed informed consent was obtained prior to data collection.

Data Collection

All study indices were collected during a one-time, two-hour assessment. Trained interviewers administered a survey to individual participants via a structured interview questionnaire. The self-reported questionnaire assessed sociodemographic information, personal medical history, and psychosocial information. Psychosocial variables were defined as self-reported indices of experience with exposure to incidents of stress-related racial discrimination and level of perceived stress derived from exposure. Trained registered nurses and certified technicians collected physiologic data consisting of blood pressure and anthropometric measurements. Three blood pressure measurements were taken on the participant’s right arm, with a five-minute rest interval in between, by using a mercury sphygmomanometer. The last two measures were then averaged. The participant’s anthropometric status was assessed by body mass index (BMI) and waist circumference. Weight and height were measured with participants wearing light clothes and without shoes. Body mass index (BMI) was calculated as the ratio of weight (in kilograms) over height$^2$ (in meters$^2$).

Instrument

The instrument used in the study was adapted and developed from a combination of existing validated instruments as well as data generated from focus group assessments conducted among a sample of study participants. We were interested in assessing specific aspects of racial discrimination and stress in African Amer-
icans that had yet to be operationalized or captured in any existing instruments. We also discovered that existing instruments had been derived from a younger, highly educated, college-age sample and were principally aimed at assessing perceptions of racial discrimination initiated specifically by Whites.30–33 Our areas of interest were much broader and included estimates concerning general perception of racial discrimination as experienced by African Americans initiating from any race/ethnic group. We were also interested in assessing the variance effects of age and socioeconomic status (SES) within an African-American sample population. Inherent deficiencies thus required use of a modified, cohesive assessment tool to capture and address our specific measures of interests. The precedence for such a tailored measurement instrument has been previously required and subsequently implemented by other investigators in light of this growing contemporary area of public health research.19,23

The study instrument was empirically tested for internal validity and test/retest reliability and is available from the authors upon request. Cronbach coefficients were calculated on raw data for specified domains of racial discrimination and level of stress.34 Cronbach for these questions ranged from 0.73 to 0.80.

Outcome and Predictor Variables

The study’s main outcome included a dichotomous measure of hypertensive vs normotensive health status. The two primary predictor variables included self-reported exposure to: 1) ever having experienced incidents of racial discrimination in any aspect of life that produced stress and; 2) level of perceived stress generally derived from such exposure. Exposure to stress-related racial discrimination was assessed as a “yes” vs “no” dichotomous response to the question, “Have you ever experienced racism or discrimination from non-African Americans during your lifetime in any aspect of your life (eg, work, school, shopping) because of your skin color and race that caused you stress?” Respondents reporting, “don’t know/not sure” were aggregated with those reporting “no” to such exposure. Level of stress generally derived from exposure was assessed among the subset of respondents that responded “yes” to having experienced incidents of racial discrimination that caused stress and measured as a numeric rank-ordered response to the directive, “Please rank your general level of stress when you experience racism or discrimination from non-African Americans because of your skin color and race, (1=none, 2=very little, 3=some [moderate], 4=high, 5=very high).” Responses were collapsed into three rank-ordered measures due to skewness with “1” representing the lowest level of generally derived stress and “3” representing the highest.

Statistical Analyses

Two separate multivariate logistic regression models were fit to estimate the association of stress-related incidents of racial discrimination and magnitude of generally derived stress with hypertension likelihood. Age, BMI, gender, SES, and residential location have been demonstrated to be positively correlated with elevated blood pressure.35–40 We included these variables in each model as covariates to adjust for potential confounding outcome effects. Age in years, years of educational attainment (as a measure of SES), and BMI (kg/m²) were entered as continuous variables; gender and residential location (urban vs suburban) were entered as categorical variables. First-order interaction terms between each primary predictor and covariate were also entered to further assess the scope of potential effect with outcome findings. Odds ratios (OR), 95% confidence intervals (CI) and level of statistical significance are reported; a two-tailed P value of ≤.05 and ≤.10, respectively, were established for main and interaction effects.17 All analyses were performed by using Statistical Analyses Software, version 8.1.41

RESULTS

Table 1 presents the distribution of selected sociodemographic and physiologic characteristics based on the stratified sampling frame. The overall age of respondents ranged from 21 to 81 years. Hypertensive respondents were slightly older (mean 54.0 years) when compared to those that were normotensive (mean 42.0 years). Mean years of educational attainment and marital status were relatively evenly distributed between the two groups, as was BMI. Household annual income, on the other hand, was slightly higher among those that were normotensive ($44,717 vs $38,476, respectively); differences were not statistically significant. A higher proportion of hypertensive respondents reported living in an urban residential environment (63%).

A comparison of sociodemographic and physiologic characteristics between responders and non-responders is not presented. However, a review of information revealed that non-responders were slightly younger (43.0 mean years of age vs 49.0 mean years of age); years of educational attainment were comparable (13.0 and 14.0 mean years, respectively). Mean systolic blood pressure (SBP) was higher among non-responders (134.5 mm Hg) when compared to responders (129.0 mm Hg); mean diastolic blood pressure (DBP), on the other hand, was similar (83.3 mm Hg and 82.3 mm Hg, respectively).

Stress-Related Incidents of Racial Discrimination and Hypertension Likelihood

Findings related to the association of exposure to stress-related incidents of
racing discrimination and hypertension likelihood are presented in Table 2. Results reveal no statistically significant relationship. Advancing age and increasing BMI were, however, demonstrated to be significant independent covariate predictors of hypertension (OR/95% CI = 2.23/1.33–3.73; P < .001 and 1.05/1.01–1.10; P = .003, respectively). Results also show that respondents who resided in an urban location were twice as likely to be hypertensive when compared to those residing in a suburban environment (OR/95% CI = 2.23/1.33–3.73; P < .002). No significant first-order interaction effects were seen between the primary predictor, covariate, and outcome variables.

Level of Stress Generally Derived from Exposure and Hypertension Likelihood

Unlike the previous outcome finding, results from this analysis show that magnitude of stress derived from exposure to racial discrimination is positively associated with an increased likelihood of hypertension (Table 3). Respondents reporting a “moderate” level of generally derived stress were more than twice as likely to be hypertensive than those reporting “no to low” stress, as were those reporting “high to very high” generally derived stress (OR/95% CI = 2.35/1.14–4.83; P = .02 and OR/95% CI = 2.50/1.17–5.34; P = .01, respectively). Findings regarding the effects of covariates on outcome demonstrated a similar pattern as those observed in the previous analysis, including the lack of any interaction effects.

DISCUSSION

Findings reveal that exposure to stress-related racial discrimination is not significantly associated with an increased likelihood of hypertension in African Americans. Other studies found somewhat similar findings. An investigation by Broman, for instance, showed that experience with racially based discrimination was not significantly associated with physician-diagnosed hypertension.19 Poston et al20 and Dressler21 also found that perceived racial discrimination was unrelated to elevated blood pressure or hypertension. The results of our findings may be due in part to a number of factors, including an equivalent high prevalence of exposure reportedly experienced by the general study population. As Figure 1 illustrates, ≈74% of the overall study population, including hypertensive and normotensive persons, reported encounters with stressful incidents of racial discrimination. Magnitude of stress perceived to be derived from exposure among this population was, on the other hand, a strong and significant predictor of hypertension. Evidence shows that respondents reporting higher levels of generally derived stress were twice as likely to be hypertensive when
Table 2. Results from an adjusted logistic regression model predicting the effects of exposure to stress-related incidents of racial discrimination and hypertension likelihood in a sample of African Americans: MAHDS, 1999–2001

<table>
<thead>
<tr>
<th>Outcome Variable</th>
<th>Hypertensive n=174 vs Normotensive n=182</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary predictor</strong></td>
<td>OR 95% CI P value</td>
</tr>
<tr>
<td>Encountered incidents of racial discrimination (per 1 unit increment)</td>
<td>Referent 1.15 0.64, 2.13 .63</td>
</tr>
<tr>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td><strong>Covariate</strong></td>
<td>(per 1 unit increment)</td>
</tr>
<tr>
<td>Age, years</td>
<td>1.09 1.06, 1.11 &lt;.0001</td>
</tr>
<tr>
<td>Education, years</td>
<td>1.04 0.93, 1.15 .42</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>1.05 1.01, 1.10 .003</td>
</tr>
<tr>
<td>Gender</td>
<td>0.89 0.53, 1.49 .67</td>
</tr>
<tr>
<td>Residual location</td>
<td>2.23 1.33, 3.73 .002</td>
</tr>
</tbody>
</table>

* Data were derived from a cross-sectional physiologic and self-reported survey conducted from 1999 through 2001 among a stratified sampling of hypertensive and normotensive adult African Americans residing in Metro-Atlanta, Georgia—the Metro Atlanta Heart Disease Study (MAHDS).

Level of significance was established as a two-tailed P value of <.05. The outcome variable included a dichotomous measure of hypertensive vs normotensive respondents, with hypertensives as the referent group. Hypertensives were defined as respondents with a mean systolic or diastolic blood pressure reading of $140 \text{mm Hg}$ or $90 \text{mm Hg}$, respectively, or currently taking prescribed antihypertensive medication. The primary predictor included a dichotomous “yes” vs “no” response to the query of ever experiencing an incident of racial discrimination in any aspect of life that caused stress. The following covariates were included in the model to control for potential confounding effects: age (in years), educational attainment (in years as a measure of socioeconomic status), body mass index (kg/m²), gender (men vs women), and residential location (urban vs suburban). All first-order interaction terms with the primary predictor variable were also offered (including age, body mass index, and residential location).

As such, findings in our sample population failed to yield a statistically significant relationship, suggesting perhaps that higher SES, as measured by years of educational attainment, may not be equally protective in African Americans as observed in other racial/ethnic groups.

Overall study information confirms the conceptual premise that exposure to racial discrimination is a psychosocial factor that is stress provoking and highly prevalent in African Americans; it also suggests that, although prevalent, exposure to stress-related racial discrimination may not significantly influence hypertension likelihood. Evidence rather suggests that differences in the perceived degree of stress may be a more significant determinant than exposure per se. Our observation may be explained by a number of competing factors such as coping response, personality, and cardiovascular reactivity.

Caveats

Results from this pilot study provide important new salient insight regarding the probable association between stress-related racial discrimination and hypertension in African Americans. However, broad extrapolation of study findings may not be feasible because of a number of inherent constraints and limitations. Outcome findings, for instance, were based on a sample of individuals drawn from a large southern region of the country composed predominantly of African Americans. Results from Finch et al regarding acculturation in Hispanics suggest potential protective health effects derived from residing in a racially homogenous environment. The lack of a significant correlation between...
exposures to racial discrimination and hypertension likelihood observed in our study population may be due to similar sociocultural factors that may have thus influenced our findings. A replication of the study in another region of the country with a different racial/ethnic composition may produce different outcome findings. However, we have no reason to believe that our findings will significantly differ from studies replicated in similar sociodemographic communities of the United States.

Reporting and recall bias inherent in self-reported indices is another methodologic issue that requires acknowledgment. We attempted to control for recall bias between the primary predictor and outcome variables during the recruitment phase of the study by generally informing potential participants that the study was designed to assess aspects of hypertension and heart disease in African Americans and that a comprehensive questionnaire was therefore required.

Outcome findings were also based on cross-sectional observations and do not provide evidence concerning the cumulative physiologic effect of chronic exposure to racial discrimination and subsequent change in blood pressure over time. We were cognizant of this limitation and aware that a longitudinal (or nested) design that included an objective measure of stress, such as cortisol, would be optimal and more robust; unfortunately, resource limitations associated with this pilot precluded such an elaborate research design. We attempted to address an aspect of this issue by incorporating a modified probe adapted from validated proxy measures of chronic lifetime exposure with a response to the query “Have you ever experienced racism or discrimination from non-African Americans during your lifetime in any aspect of your life because of your skin color and race that caused you stress?”

Table 3. Results from an adjusted logistic regression model predicting the effects of level of stress generally derived from exposure to stress-related racial discrimination and hypertension likelihood in a sample of African Americans: MAHDS, 1999–2001

<table>
<thead>
<tr>
<th>Outcome Variable</th>
<th>Hypertensive</th>
<th>Normotensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary predictor</td>
<td>n=130</td>
<td>vs</td>
</tr>
<tr>
<td>Level of stress generally derived from exposure to racial discrimination (per 1 unit increment)</td>
<td>OR</td>
<td>95% CI</td>
</tr>
<tr>
<td>No – low stress referent</td>
<td>2.35</td>
<td>1.14, 4.83</td>
</tr>
<tr>
<td>Moderate stress</td>
<td>2.50</td>
<td>1.17, 5.34</td>
</tr>
<tr>
<td>High – very high stress</td>
<td>2.33</td>
<td>1.33, 3.73</td>
</tr>
<tr>
<td>Covariate (per 1 unit increment)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, years</td>
<td>1.09</td>
<td>1.06, 1.11</td>
</tr>
<tr>
<td>Education, years</td>
<td>1.04</td>
<td>0.93, 1.15</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>1.05</td>
<td>1.01, 1.10</td>
</tr>
<tr>
<td>Sex</td>
<td>0.89</td>
<td>0.53, 1.49</td>
</tr>
<tr>
<td>Residential location</td>
<td>2.23</td>
<td>1.33, 3.73</td>
</tr>
</tbody>
</table>

Data were derived from a cross-sectional physiologic and self-reported survey conducted from 1999 through 2001 among a stratified sampling of hypertensive and normotensive adult African Americans residing in Metro-Atlanta, Georgia—the Metro Atlanta Heart Disease Study (MAHDS). The distribution of hypertensives and normotensives (130 and 135, respectively) reflect the number of respondents that answered “yes” to experiencing incidents of racial discrimination that caused stress.

Level of significance was established as a two-tailed P value of <.05. The outcome variable included a dichotomous measure of hypertensive vs normotensive respondents (hypertensives=referent group). Hypertensives were defined as respondents with a mean systolic or diastolic blood pressure reading of ≥140 mm Hg or ≥90 mm Hg, respectively, or currently taking prescribed antihypertensive medication. Normotensive respondents were defined as those with a mean systolic or diastolic blood pressure reading of <140 mm Hg, <90 mm Hg, respectively, and not taking prescribed antihypertensive medication. The primary predictor included a rank-ordered trichotomous response to the query concerning level of perceived stress derived from an encounter of racial discrimination. The following covariates were included in the model to control for potential confounding effects: age (in years), educational attainment (in years as a measure of socioeconomic status), body mass index (kg/m²), sex (men vs women), and residential location (urban vs suburban). All first-order interaction terms with the primary predictor variable were also offered in the model (including × age, body mass index and residential location).

OR=odds ratio, CI=confidence intervals.

Fig 1. Prevalence of self-reported exposure to incidents of racial discrimination that caused stress
Genetics have been well established as a risk factor for hypertension and have recently been shown to be a relevant marker for stress reactivity.\textsuperscript{51} Inherent limitations associated with our study, however, precluded the ability to account for independent and/or potential interactive effects.\textsuperscript{52,53} We finally acknowledge the concept of racial discrimination as a subjective construct and that any attempt to measure an empiric correlation with hypertension likelihood may not be precise when compared to established physiologic risk factors. We further acknowledge that any observed association between measures of racial discrimination and hypertension may be a function of assessment instruments and probes.\textsuperscript{54} Despite these observations or research limitations, we nevertheless are confident that information produced by this report will contribute to the existing body of knowledge and subsequent development of research concerning stress-related racial discrimination and the etiology of hypertension in African Americans.

CONCLUSION

The unique milieu within the African-American context includes a convergence of social, environmental, and biologic factors that individually influence and interact as biopsychosocial risk factors that contribute to the risk of hypertension and its subsequent onset. The physiologic effects of stress derived from exposure to racial discrimination within this experience are complex and cannot be easily quantified; indeed, study results are competing and inconsistent. The primary objective of our research was to advance public health discourse and relevant research related to racial discrimination as a prevalent stressor and its effects on the etiology and excess burden of hypertension in African Americans.

Our findings suggest that stress-related incidents of racial discrimination are highly prevalent and equally experienced by African Americans, but may not be a significant factor associated with an increased likelihood of hypertension.

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REFERENCES


**AUTHOR CONTRIBUTIONS**

Design and concept of study: Davis

Acquisition of data: Davis, Liu, Collins

Data analysis and interpretation: Davis, Liu, Din-Dzietham

Manuscript draft: Davis, Collins, Din-Dzietham

Statistical expertise: Liu, Din-Dzietham