

Chronic Stress Burden, Discrimination, and Subclinical Carotid Artery Disease in African American and Caucasian Women

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This study examined the association between a composite index of stress that included measures of life events, ongoing stress, discrimination, and economic hardship and subclinical carotid disease among 109 African American and 225 Caucasian premenopausal women. African Americans reported more chronic stress and had higher carotid intima-media thickness (IMT) as compared with Caucasians. Among African Americans only, the composite stress index and unfair treatment were associated with higher IMT. These effects were partially mediated by biological risk factors. African Americans who reported experiencing racial discrimination had marginally more carotid plaque than did those who did not report experiencing racial discrimination. The results suggest that African Americans may be particularly vulnerable to the burden of chronic stress.

Key words: carotid arteries, chronic stress, race, discrimination

For the past several decades, there has been a proliferation of research documenting the association between chronic psychosocial stressors and clinically manifest cardiovascular disease (CVD; for a review, see Rozanski, Blumenthal, & Kaplan, 1999). Most consistently, studies have shown that chronic occupational stress

(for a review, see Schnall, Landsbergis, & Baker, 1994) and low socioeconomic status or economic hardship (e.g., Adler et al., 1994; Matthews et al., 2002) are prospectively linked with increased risk of CVD morbidity and mortality, independent of other known risk factors, particularly in populations of Caucasian men. Less is known, however, about the impact of chronic stress on women's health in general, and on African American women in particular. Notably, lower status groups, including women and African Americans, appear to have greater exposure to stressful life events and to be more adversely affected by these stressors as compared with higher status groups (Davis, Matthews, & Twamley, 1999; Thoits, 1982; Turner, Wheaton, & Lloyd, 1995).

Investigating the unique chronic stressors that may be involved in the etiology of CVD in women is important for several reasons. First, evidence suggests that men and women may differ in the appraisal of chronic stress and the subsequent health consequences (Marmot, Bosma, Hemingway, Brunner, & Stansfeld, 1997; Verbrugge, 1986). Studies of physiological reactions to acute stress suggest that women have smaller physiological responses to acute stressors (Davis & Matthews, 1996; Girdler & Light, 1994), although interpersonal stressors can elicit larger responses in women (Kiecolt-Glaser, Glaser, Cacioppo, & Malarkey, 1998; Smith & Gallo, 1999). In addition, women may experience unique sources of chronic stress, stemming from occupying multiple roles both within the home and in the labor force. Finally, men and women may differ in the types of stressors encountered even within the same social roles, such as gender-based discrimination in the workplace.

Most research to date has focused on isolated stressors that influence the etiology and progression of disease. However, multiple stressors often co-occur in an ecological context and may interactively influence health outcomes (Evans & Cohen, 1987; Lepore & Evans, 1996). Individuals who are faced with multiple sources of stress may exhaust effective coping resources, thereby making them more susceptible to the deleterious health consequences of stress (Gallo & Matthews, 2003; Lepore, Evans, &

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Palsane, 1991; Rook, 1990). Researchers have posited that African American women's more rapid deterioration in health status relative to that of Caucasian women may be partially attributable to the cumulative effect of exposure to multiple sources of stress—an effect termed the *weathering hypothesis* (Geronimus, 1992). Thus, aggregating across multiple sources of stress may increase the predictive power of individual stress measures and may be a more valid representation of the impact of stress on disease. In contrast, studies that have examined discrete measures of stress, independent of their ecological context, may underestimate the actual stress–disease relationship (Cohen, Evans, Stokols, & Krantz, 1986; Evans & Cohen, 1987). In particular, previous research may not have adequately captured the overall stress burden experienced by women and ethnic minorities by failing to consider the effects of pervasive environmental stressors.

In particular, discrimination is a pervasive phenomenon in American culture, with more than 50% of African Americans attributing substandard housing, lack of skilled labor and managerial opportunities, and lower wages to ethnic discrimination (Sigelman & Welch, 1991). However, studies examining the association between discrimination and cardiovascular outcomes (primarily blood pressure and hypertension) are limited, and their results are equivocal (for a review, see Krieger, 1999).

An important limitation in the existing discrimination literature, specifically, and the chronic stress literature, generally, is the reliance on clinically manifest disease. Clinical outcomes are subject to a number of inherent biases (e.g., self-report, physicians' expectations), and they preclude definitive conclusions regarding the influence of risk factors on early stages of disease development. The emergence of new technology to evaluate the extent of subclinical atherosclerosis, such as B-mode ultrasound measures of carotid artery intima–media thickness (IMT) and plaque, eschews potential biases associated with clinical events and provides a valid and objective approach to understanding risk factors for CVD at an early stage of atherosclerotic burden. Positive associations have been reported between carotid artery IMT and cardiovascular risk factors (Bonithon-Kopp et al., 1991; Lassila, Sutton-Tyrrell, Matthews, Wolfson, & Kuller, 1997), and high levels of IMT have been prospectively linked with incident cases of clinical coronary heart disease and stroke, even after researchers have statistically controlled for standard cardiovascular risk factors (Chambless et al., 1997; Salonen & Salonen, 1991). Thus, the use of subclinical outcomes may elucidate key biological, behavioral, and psychosocial risk factors that may lead to accelerated disease progression in certain individuals and ethnic groups.

To our knowledge, only one previous study (Muntaner, Nieto, Meyer, Szklo, & Tyroler, 1998) has investigated the cross-sectional relationship between chronic stress and carotid IMT in women. In a sample of African American and Caucasian men and women from the Atherosclerosis Risk in Communities Study, Muntaner et al. (1998) found a positive association between several measures of occupational stress and carotid IMT across all four race–gender strata, independent of socioeconomic status.

Despite the scarcity of direct evidence, previous research has identified a number of plausible mechanisms that may act as pathophysiological links between chronic stress and development of atherosclerosis. First, chronic exposure to stress may have direct negative effects on cardiovascular health by exhausting biological resources for adapting to stress or by inducing a persistently

heightened arousal state that may have pathogenic effects on the cardiovascular system (Krantz & Manuck, 1984; McEwen, 1998; Selye, 1956). Over time, physiological responses to chronic stress could affect resting blood pressure levels (Matthews et al., 2002) and metabolic risk factors, such as elevations in plasma lipids (Räikkönen, Keltikangas-Jarvinen, Adlercreutz, & Hautanen, 1996) and glucose (Vitaliano, Scanlan, Krenz, & Fujimoto, 1996), perhaps consequential to cortisol elevations. Anderson, McNeilly, and Myers (1992) reviewed a number of physiological factors, including sodium retention and heightened sympathetic nervous system activity, that might mediate the association between chronic stress and increased vascular activity among African Americans. Additionally, chronic stress could affect atherosclerotic progression indirectly by increasing exposure to behavioral and psychosocial risk factors (as reviewed by Rozanski et al., 1999). Finally, though the evidence is mixed (see Gump & Matthews, 1999), some researchers have argued that chronic stressors may heighten responses to acute stressors and exacerbate their negative effects.

Our primary aim in this study was to examine the association between a composite index of chronic stress that included measures of life events, ongoing stressors, unfair treatment, and economic hardship and the risk of subclinical carotid disease in African American and Caucasian women. Our primary hypothesis was that greater accumulated stress burden would be associated with greater IMT and plaque. On the basis of their “double minority status” of being African American and female, we predicted that African American women would report higher values on measures of life events, ongoing stress, economic hardship, and unfair treatment and, consequently, would have higher composite stress scores. We also explored whether the effect of chronic stress on subclinical outcomes depends on ethnicity. Finally, we examined biological and behavioral risk factors related to the chronic stress measures as potential pathways through which greater stress may lead to increased disease risk. To our knowledge, this is the first empirical investigation to assess the impact of chronic stressors other than occupational stress on subclinical end points.

Method

Participants

The 334 participants were drawn from the Pittsburgh site of a seven-site, longitudinal study of midlife aging called the Study of Women's Health Across the Nation (SWAN). A detailed description of the SWAN has been published in previous reports (e.g., Sowers et al., 2000). The present study's ethnic composition consisted of 225 non-Hispanic Caucasians and 109 African Americans. The ethnic breakdown of the sample reflects the Pittsburgh site's oversampling of African Americans by means of recruiting Caucasians and African Americans in the ratio of 2:1. At the Pittsburgh SWAN site, the primary recruitment methods were random digit dialing and voter registration lists. Eligibility criteria required that participants were between 42 and 52 years old, had not taken hormone replacement therapy, and had had a menstrual period in the prior 3 months. SWAN participants eligible for the subclinical disease evaluation included women who did not report having angina, intermittent claudication, cerebral ischemia, or revascularization and who were not taking specific medications for hypertension, diabetes, or heart arrhythmias. A total of 123 African American and 265 Caucasian women were identified as eligible to participate in the carotid ultrasound study. Of these women, 88.6% of the African American women ($n = 109$) and 84.9% of the Caucasian women ($n = 225$)

completed the subclinical protocol and completed psychosocial data for participation in the current study.

Procedure

Recruitment for the carotid ultrasound protocol began approximately 6 months after the baseline examinations had begun because of requirements of the National Institute on Aging. Therefore, 284 participants had the ultrasound evaluation within 7 months of the baseline visit ($M = 1.96$ months, $SD = 1.61$), and 50 participants had their scans an average of 14 months later ($SD = 3$), at the time of the first follow-up visit. Importantly, analyses restricted to the subset of women ($n = 284$) who had carotid and psychosocial data available within 7 months of the baseline visit revealed findings similar to those in the larger sample.¹ Written informed consent was obtained from participants prior to participation in the study procedures. The University of Pittsburgh's Institutional Review Board approved all study protocols. Participants were paid \$8 per hour for attending the baseline clinic visit, and an additional \$5 was paid to women who were eligible and completed the carotid ultrasound protocol.

The baseline clinic visit and each subsequent follow-up visit occurred between the 2nd and 5th days of the participant's menstrual period, following a 12-hr fast. Anthropometric measurements included height, weight, waist and hip circumference, body mass index (BMI), systolic blood pressure (SBP), and diastolic blood pressure (DBP). A fasting blood draw was used to obtain measurements of total serum cholesterol, total high-density lipoproteins (HDL-c), triglycerides, calculated low-density lipoproteins, insulin, glucose, and clotting factors. Lipid and lipoprotein fractions were analyzed on EDTA-treated plasma (Warnick & Albers, 1978). Total cholesterol and triglycerides were analyzed using enzymatic methods on a Hitachi 747 analyzer (Boehringer Mannheim Diagnostics, Indianapolis, IN). HDL-c was isolated using heparin-2m manganese chloride (Warnick & Albers, 1978). Laboratory assays were conducted at a central laboratory that conforms to the quality control standards of the Centers for Disease Control and Prevention and the National Heart, Lung, and Blood Institute. Certified technicians measured blood pressure. Two blood pressure measurements were recorded, and the results were averaged. To ensure quality technician performance and compliance with a standard protocol, all SWAN interviewers were certified before they collected physical measures on participants.

At each clinic visit, participants provided information concerning medical history, health behaviors, and demographic and psychosocial characteristics thought to affect health. Descriptions of the psychosocial measures used in this study are reported below. Physical activity was assessed using an adaptation of Baecke's questionnaire, an instrument that has been used widely in the epidemiologic literature and has documented reliability and validity (Baecke, Burema, & Fritjers, 1982).

Psychosocial Measures

Composite stress. To create an overall measure of chronic stress burden, we developed a composite score consisting of four indicators of stress (life events, ongoing stressors, economic hardship, and unfair treatment). Analyses showed that with the exception of ongoing stressors and unfair treatment, all correlations among the individual stress measures were significant, with Pearson correlation coefficients ranging from .13 to .50. With the exception of economic hardship, which was dichotomized at the top 34% (due to its distribution), all of the other individual stress measures were dichotomized at the top 20% of their distributions, and we summed the dichotomous codes. Scores ranged from 0 to 4, with higher scores indicating greater accumulated stress. Percentages of participants with scores of 0, 1, 2, 3, and 4, respectively, on the composite stress measure were 46.1%, 27.5%, 17.1%, 6.9%, and 2.4% for the whole sample; 33.0%, 30.3%, 25.7%, 9.2%, and 1.8% for African Americans; and 52.4%, 26.2%, 12.9%, 5.8%, and 2.7% for Caucasians. Consequently,

individuals with scores of 3 or 4 on the composite stress measure were combined into a single group. The composite stress measure was modeled as a continuous variable ranging from 0 to 3 in the analyses.

Stressful life events. A modified version of the Psychiatric Epidemiology Research Inventory Life Events Scale (Dohrenwend & Dohrenwend, 1981; Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978) was used to evaluate events relevant to midlife women and different ethnic and socioeconomic environments. Respondents were asked to indicate whether they had experienced any of 36 events (e.g., "a close relative died," "had trouble with a boss or conditions at work," "major money problems") during the previous 12 months. Of the total sample, 51.2% indicated that they had not experienced any very stressful event in the previous 12 months, 30.0% indicated that they had experienced 1 or 2 events, and 18.8% indicated that they had experienced 3 or more events. Thus, we dichotomized this variable so that one group comprised women who reported 2 or fewer events (coded 0) and the other group comprised women who reported 3 or more events (top 20% of distribution; coded 1).

Ongoing stressors. Chronic, unremitting stressors were operationalized as events occurring in the lives of the participants that endured for 12 months or more and that were perceived as very stressful (e.g., health problems in a close relative or friend, difficulties with a job or ability to work, financial strains; Bromberger & Matthews, 1996). A total of nine items were rated on a 3-point scale ranging from *not very upsetting* to *very upsetting*. Participants who indicated at least one ongoing stressor as being "very upsetting" were coded as having a severe chronic stressor (1), whereas participants who rated the items as "not very upsetting," "moderately upsetting," or not endorsing any ongoing problems were defined as having no chronic stressors (0). Preliminary analyses indicated that 78.6% of the sample endorsed having no severe ongoing stressors, 13.8% had one stressor, and 7.6% endorsed having two or more ongoing stressors. Thus, we used a dichotomous variable coded as 1 if the participant endorsed at least one severe ongoing stressor or 0 if the participant did not endorse any item. Because the SWAN leadership approved the use of this measure after the baseline examinations had begun, 82 participants did not complete this measure at baseline. Consequently, we substituted data ($n = 82$) from the first annual follow-up visit, provided the data were collected within 12 months of the carotid measures.

Economic hardship. A single item was used to assess how difficult it was to pay for "basics," such as food, housing, medical care, and heating. The response format was a 3-point scale ranging from 1 (*very hard*) to 3 (*not very hard at all*). Analyses from the Coronary Artery Disease Risk Development in Young Adults study revealed that having difficulty paying for basics independently predicted incident hypertension over a 10-year follow-up period (Matthews et al., 2002). Eight percent of the current sample indicated that it was very hard to pay for basic necessities, 26% indicated that it was somewhat hard, and 66% indicated that it was not very hard at all. Thus, the top 34% of the distribution (i.e., "somewhat" or "very hard") was coded as 1, and participants who endorsed "not very hard at all" (66%) were coded as 0.

Unfair treatment. Participants completed a 10-item questionnaire that assessed how frequently in their day-to-day lives they had experienced a variety of forms of interpersonal mistreatment. Examples include "How frequently are you treated with less respect than other people?" and "How frequently do people act as if they're better than you?" The items were presented in general terms of mistreatment, without making any reference to race, prejudice, or discrimination. Previous research has found that these items are positively correlated with self-reports of poor health, particularly among African Americans (Williams, Yu, Jackson, & Anderson, 1997). Participants were asked to rate the frequency with which they experienced each type of mistreatment on a 4-point scale (1 = *often*, 2 = *sometimes*, 3 = *rarely*, and 4 = *never*). For the present study, the items were reverse

¹ Analyses are available from Karen A. Matthews on request.

coded (i.e., 1 = *never* and 4 = *often*) so that higher values corresponded to higher frequency of experiences of unfair treatment. In the current sample, internal reliability (α) of this measure was estimated at .87 for the whole sample, .90 for African Americans, and .84 for Caucasians. For the composite stress measure, we dichotomized the unfair treatment scale, such that participants who scored in the top 20% of the distribution were coded as 1 ($n = 67$) and the remainder were coded as 0 ($n = 267$).

Racial discrimination. Sixty-six percent ($n = 72$) of the African American participants and 45% ($n = 102$) of the Caucasian participants responded either “sometimes” or “often” to at least 1 of the 10 unfair treatment items and were asked to complete an additional item for which they indicated to what reason they attributed the cause of mistreatment. Response categories included *race*, *ethnicity*, *gender*, *age*, *income level*, *language*, *physical appearance*, *sexual orientation*, and *other*. Participants who indicated either race or ethnicity as the cause for the mistreatment were categorized as having experienced racial discrimination. African Americans (54.2%; $n = 39$) were significantly more likely than Caucasians (3.9%; $n = 4$) to endorse ethnicity or race versus other factors as the main reason for mistreatment, $\chi^2(1, N = 174) = 57.27, p < .001$.

Carotid Ultrasound

Carotid measurements were assessed using a Toshiba SSA-270A scanner (Toshiba, Tustin, CA) equipped with a 5-MHz linear array-imaging transducer. During the scanning protocol, the sonographer obtained detailed B-mode images of the right and left common carotid arteries, the carotid bulb, and the first 1.5 cm of the internal and external carotid arteries. The sonographer imaged the vessel in several planes for each location and then focused on the interfaces required to measure IMT and any areas of focal plaque.

Trained readers at the University of Pittsburgh site measured the average IMT across 1-cm segments of the near and far walls of the distal common carotid artery, the far wall of the carotid bulb, and the internal carotid artery on both right and left sides. An overall measure of IMT (in millimeters) was calculated as the average of the measures from each location. Plaque scores were assessed from the images of the proximal common artery, distal common artery, carotid bulb, internal carotid artery, and external carotid artery. Plaque was defined as a discrete focal protrusion into the vessel lumen with at least 50% greater thickness than surrounding normal areas. Summary scores based on the size and number of plaques at each location were computed to produce an overall measure of focal plaque, called the plaque index. Plaque scores can range from 0 to 30 in older populations; however, in populations such as those in the SWAN, plaque is commonly analyzed as a categorical variable because of the small percentage of participants with any measurable plaque.

Reproducibility was determined in 15 participants who had their scans performed by two different sonographers. Intraclass correlations were .88 and .93 for IMT and the plaque index, respectively. In addition, previous reports that have used the same duplex scanning protocol as that described herein have shown high reproducibility of plaque and IMT scores (Sutton-Tyrrell, Wolfson, Thompson, & Kelsey, 1992; Thompson & Sutton-Tyrrell, 2001).

Statistical Analyses

Biological variables with substantially skewed distributions (i.e., fasting glucose, fasting insulin, and triglycerides) were analyzed using logarithmic transformations. Preliminary analyses revealed that the IMT scores were normally distributed, with no significant outliers. IMT scores ranged from 0.49 to 1.10 mm. The distribution of plaque scores was extremely positively skewed, as very few women had focal plaques. Thus, plaque scores were analyzed as a dichotomy, so that one group comprised women with plaque scores of 0 ($n = 290$) and the other group comprised women with plaque scores of 1 or higher ($n = 44$). A series of *t* tests and chi-square

analyses explored racial differences in demographics, biological and behavioral risk factors, and the major study variables (i.e., chronic stress measures and subclinical outcomes). The main analyses of this research used forced-entry stepwise linear and logistic regression models, for IMT and plaque, respectively, with age adjusted in the first step, to test the effect of the composite chronic stress measure on subclinical outcomes. To disentangle the effects of race and chronic stress on subclinical outcomes, we conducted a multiple regression analysis in which IMT was regressed on age, race, and composite stress in the first step and the interaction of race and composite stress in the second step. We performed similar analyses with plaque as the outcome. To further probe significant interaction effects and to determine which of the individual stress measures were contributing to the interaction, we conducted race-stratified analyses for the composite stress measure and for each individual stress measure separately.

We conducted forced-entry stepwise regression analyses to assess whether biological and behavioral risk factors functioned as a pathway through which chronic stress is associated with subclinical disease. Consistent with Baron and Kenny's (1986) recommendations for testing statistical mediation, we considered evidence of at least partial mediation if the following criteria were met: (a) the predictor variable and the proposed mediator or mediators were correlated with the outcome variable, (b) the predictor variable was associated with the proposed mediator or mediators, and (c) the magnitude of the association between the predictor variable and the outcome variable was attenuated (i.e., substantial reduction in R^2 for the predictor variable) when the proposed mediator or mediators were statistically controlled.

To specifically address whether racial discrimination was associated with IMT and plaque, we conducted additional regressions to compare African American women who attributed the cause of mistreatment to racial or ethnic discrimination and African American women who attributed the cause to factors other than racial or ethnic discrimination or who did not report experiencing unfair treatment. Only data from African American women were used in these analyses because only 4 Caucasian women attributed the cause of mistreatment to racial or ethnic discrimination. As with all of the analyses, we adjusted age in the first step of the regression, followed by the reason for unfair treatment in the second step.

Results

Participant Characteristics

Descriptive statistics for demographic and psychosocial risk factor characteristics of this sample are presented in Table 1. The average age of the sample was 46.24 years ($SD = 2.45$), a value on which African American and Caucasian participants did not differ. A larger percentage of African Americans had household incomes less than \$35,000 per year compared with their Caucasian counterparts, but there were no racial differences in educational attainment. African Americans had higher baseline measures of BMI, waist-hip ratio, insulin, SBP, and DBP and lower triglyceride levels than Caucasians ($ps < .01$). African American women were also significantly less physically active than Caucasian women ($p < .001$). Of the total sample, 45.6% of the women had ever smoked, and the percentage of women who had ever smoked did not differ between Caucasian and African American women. African Americans had significantly higher scores on the composite measure of stress, $t(332) = -3.25, p < .001$, than did Caucasians. Analyses of the individual stress measures further revealed that African American women experienced significantly more unfair treatment, $t(332) = -4.05, p < .001$, and were more likely to report economic hardship (i.e., “somewhat or very hard to pay for basics”) in comparison to Caucasians, $\chi^2(1, N = 330) = 12.79, p < .001$. The groups did not differ in number of stressful life

Table 1
Study Characteristics for the Total Sample and by Ethnicity

Variable	Total sample (N = 334)	Caucasians (n = 225)	African Americans (n = 109)	p
Mean age at carotid scan (years)	46.24 (2.45)	46.11 (2.51)	46.00 (2.33)	.69
Household income < \$35,000/ year (%)	30.0	22.8	44.4	.001
High school education or less (%)	23.6	22.0	26.9	.48
Mean BMI (kg/m ²)	28.27 (6.10)	27.38 (5.63)	30.16 (6.62)	.001
Mean waist-hip ratio (cm)	.80 (.07)	.79 (.06)	.83 (.08)	.001
Mean insulin ^a (uLU/ml)	9.82 (6.30)	8.90 (4.79)	11.78 (8.37)	.001
Mean SBP (mm/Hg)	113.37 (15.37)	110.15 (12.10)	120.02 (18.94)	.001
Mean DBP (mm/Hg)	72.03 (9.21)	70.89 (8.29)	74.39 (10.51)	.003
Mean HDL-c (mg/dl)	53.70 (12.21)	53.52 (12.21)	54.07 (12.25)	.70
Mean LDL-c (mg/dl)	122.66 (31.03)	123.27 (30.65)	121.37 (31.92)	.61
Mean triglycerides ^a (mg/dl)	109.76 (63.12)	115.47 (66.41)	97.75 (53.91)	.01
Mean physical activity	8.06 (1.65)	8.27 (1.62)	7.61 (1.62)	.001
Ever smoked (%)	45.6	44.0	49.1	.39
Life event stress > 2 (%)	18.9	18.7	19.3	.90
Ongoing stress ≥ 1 (%)	21.4	22.4	19.4	.55
Unfair treatment	18.00 (4.67)	17.22 (4.00)	19.63 (5.53)	.001
Somewhat or very hard to pay for basics (%)	33.3	26.9	46.7	.001
Mean composite stress	0.90 (1.00)	0.77 (0.97)	1.15 (1.01)	.001
Mean IMT (mm)	0.64 (0.09)	0.69 (0.09)	0.62 (0.08)	.01
Plaque ≥ 1 (%)	9.5	8.3	15.6	.06

Note. Standard deviations are in parentheses. BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; HDL-c = high-density lipoprotein cholesterol; LDL-c = low-density lipoprotein cholesterol; IMT = intima-media thickness.

^a Analysis based on log-transformed data. Means and standard deviations reflect nontransformed values.

events experienced or ongoing stressors ($ps > .10$). Analyses of subclinical outcomes indicated that African Americans had a significantly higher average IMT in comparison to Caucasians, $t(1, 331) = -7.52, p < .01$, whereas a marginally greater percentage of Caucasians had any plaque in comparison to African Americans, $\chi^2(1, N = 334) = 3.42, p < .10$.

Chronic Stress and IMT

After adjustment for age, higher values on the composite stress measure were associated with greater average IMT in the total

sample, $\beta = .12, \Delta R^2 = .015, p < .05$. There was a significant Race \times Composite Stress interaction, $\beta = .26, \Delta R^2 = .023, p < .005$. As depicted in Figure 1, separate analyses by race revealed a linear relationship between increasing stress and IMT in African Americans but virtually no association in Caucasians. In addition, as shown in Table 2, higher scores on the individual measures of life events, ongoing stress, and economic hardship were each associated with marginally higher mean IMT in African Americans, but there was no association in Caucasians. Finally, unfair treatment showed a significant positive association only for Afri-

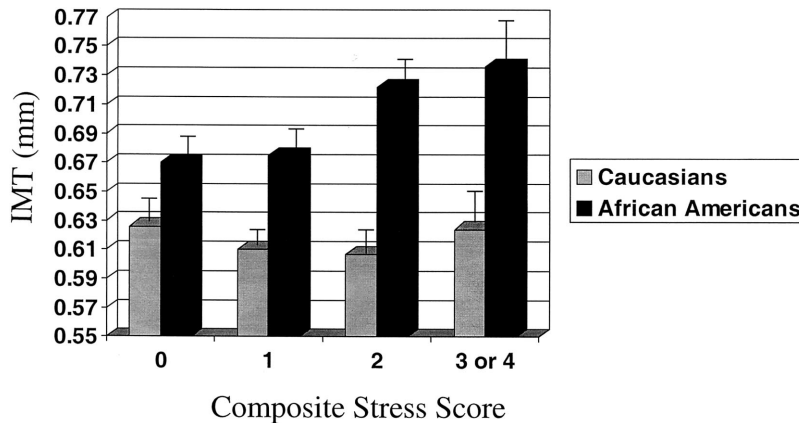


Figure 1. Age-adjusted average carotid intima-media thickness (IMT) with standard errors for African Americans and Caucasians (N = 334) according to increasing levels of composite stress.

Table 2
Separate Regressions of Average Carotid Intima–Media Thickness on Chronic Stress Measures for African Americans and Caucasians, After Adjustment for Age

Variable	African Americans				Caucasians			
	β	ΔR^2	ΔF	p	β	ΔR^2	ΔF	p
Composite stress ^a	.28	.079	9.09	.003	-.05	.003	0.64	.42
Life events ^b	.16	.026	2.84	.10	-.02	.000	0.06	.80
Ongoing stress ^b	.16	.026	2.76	.10	-.09	.007	1.58	.21
Economic hardship ^b	.17	.030	3.25	.07	-.02	.000	0.10	.75
Unfair treatment ^c	.21	.045	4.93	.03	.03	.001	0.18	.68

^a Scores range from 0 to 3. ^b Scores are dichotomous (0 or 1). ^c Scores range from 3 to 10.

can Americans. Thus, we conducted mediation analyses only in African Americans, for whom there were significant effects of the composite stress and unfair treatment measures.

Mediation Analyses

Forced-entry stepwise regression analyses tested the degree to which biological and lifestyle risk factors mediated the observed effects of composite stress and unfair treatment on IMT in African Americans. Variables that were correlated with both the outcome variable (IMT) and the predictor variables (unfair treatment and composite stress) at $p \leq .10$ were considered as potential mediators. Risk factors correlated with IMT in African Americans included SBP, BMI, HDL (inversely), waist–hip ratio, and insulin (log-transformed). Furthermore, SBP, DBP, HDL, and waist–hip ratio were correlated with composite stress, and DBP, BMI, and HDL were associated with unfair treatment. Thus, SBP, HDL, and waist–hip ratio were tested as mediators of the composite stress effect, and BMI and HDL were tested as mediators of the unfair treatment effect.

Table 3 shows the stepwise regression models for composite stress and unfair treatment on IMT, with and without statistical control for biological mediators in African Americans. In the model that adjusted only for age, composite stress accounted for 7.9% of the variance in IMT ($p < .01$). After we partialled out SBP, HDL-c, and waist–hip ratio, the percentage of variance in IMT accounted for by composite stress decreased by 38.0% (R^2 changed from .079 to .049); however, the association between composite stress and IMT remained significant, $\beta = .23$, $t(5, 101) = 2.35$, $p < .05$. The omnibus statistic for the full model was significant, $F(5, 101) = 3.11$, $p < .05$, but composite stress was the only independent predictor.

Unfair treatment accounted for 4.5% of the variance in IMT in the age-adjusted model. After we statistically controlled for HDL-c and BMI, the percentage of variance in IMT accounted for by unfair treatment decreased by 29% (R^2 changed from .045 to .032). Additionally, the effect of unfair treatment was reduced to marginal significance, $\beta = .18$, $t(4, 104) = 1.92$, $p = .06$. The omnibus statistic for the full model was significant, $F(4, 104) = 3.99$, $p < .01$, and both of the biological risk factors—HDL-c, $\beta = -.13$, $t(4, 104) = -1.94$, $p = .06$, and BMI, $\beta = .002$, $t(4, 104) = 1.68$, $p = .10$ —were also only marginally related to IMT.

Chronic Stress and Plaque

We examined the association between chronic stress and plaque through logistic regression analyses that modeled the probability of the presence of any focal plaque. Neither the composite stress measure nor any of the individual stress measures were related to the probability of having a higher plaque score, nor were there any significant race interactions ($ps > .10$). Thus, we did not conduct mediational analyses for plaque.

Racial Discrimination

There was a nonsignificant effect for African American women who attributed the cause of unfair treatment to race or ethnicity to have higher average IMT in comparison to those who reported no unfair treatment or those who attributed the cause to other factors, $\beta = .15$, $t(107) = 1.51$, $p = .14$. Furthermore, African American women who attributed the cause of unfair treatment to race or ethnicity had marginally higher odds of having a plaque score greater than or equal to 1 in comparison to those who reported no unfair treatment or those who attributed the cause to other factors (odds ratio = 4.02, 95% confidence interval = 0.94–17.14, $p = .06$). Specifically, 15.4% of African American women who attributed unfair treatment to race or ethnicity had any plaque, whereas 4.3% of those who did not report unfair treatment or who attributed the cause to other reasons had any plaque.

Discussion

In this study, we evaluated the extent to which greater accumulated chronic stress is associated with early stages of atherosclerotic development in African American and Caucasian women. Consistent with our hypothesis, we found that the combined stress from life events, ongoing stressors, economic hardship, and unfair treatment was associated with greater average IMT, but only among African Americans. With the possible exception of attributions of racial discrimination, none of our other stress measures were associated with plaque. These null findings, however, may be due to the small percentage (i.e., 13%) of individuals with any measurable plaque and the subsequent loss of power.

Our findings also elucidate possible biological mechanisms through which greater chronic stress may lead to increased risk for CVD in African American women. In particular, we found that biological mediators, specifically, metabolic risk factors (i.e., dys-

Table 3
Regression of Average Carotid Intima-Media Thickness on Chronic Stress Measures, With and Without Adjustment for Biological Risk Factors

Step and variable entered	<i>B</i>	<i>SE B</i>	β	ΔR^2 block	ΔF
Model 1: Composite stress, adjusted for age only (<i>n</i> = 108)					
1					
Age at time of carotid scan (years)	.002	.003	.05	.001	$F(1, 107) = 0.09$
2					
Composite stress	.024	.008	.28**	.079	$F(1, 106) = 9.09**$
Model 1a: Composite stress, adjusted for age and biological risk factors (<i>n</i> = 101)					
1					
Age at time of carotid scan (years)	.001	.004	.03	.001	$F(1, 100) = 0.09$
2					
Biological risk factors				.089	$F(3, 97) = 3.17*$
Systolic blood pressure	.000	.000	.12		
HDL-c	.000	.001	-.13		
Waist-hip ratio	.098	.121	.09		
3					
Composite stress	.002	.009	.23*	.049	$F(1, 96) = 5.50*$
Model 2: Unfair treatment, adjusted for age only (<i>n</i> = 107)					
1					
Age at time of carotid scan (years)	.002	.004	.01	.001	$F(1, 106) = 0.07$
2					
Unfair treatment	.003	.002	.21*	.045	$F(1, 105) = 4.93*$
Model 2a: Unfair treatment, adjusted for age and biological risk factors (<i>n</i> = 101)					
1					
Age at time of carotid scan (years)	-.003	.003	-.01	.000	$F(1, 103) = 0.01$
2					
Biological risk factors				.106	$F(2, 101) = 5.98**$
HDL-c	-.001	.001	-.19†		
Body mass index	.002	.001	.16†		
3					
Unfair treatment	.003	.002	.18†	.032	$F(1, 100) = 3.68†$

Note. HDL-c = high-density lipoprotein cholesterol.
 † $p < .10$. * $p < .05$. ** $p < .01$.

lipidemia, elevated blood pressure, and obesity), accounted for a substantial portion of the variance in the composite stress and IMT relationship. After we controlled for SBP, HDL-c, and waist-hip ratio, the variance accounted for by composite stress was reduced by 38%, but it remained the only independent predictor of IMT in the final model. IMT is sometimes considered to represent thickening specifically caused by cumulative exposure to elevated blood pressure (Barnett, Spence, Manuck, & Jennings, 1997); however, this is unlikely to be the case in the present analyses, given that SBP was not a significant predictor of IMT in the full model. We also demonstrated that partialing out the effects of BMI and HDL-c reduced the percentage of variance in IMT that unfair treatment accounted for by 29% and reduced the effect to marginal significance. Thus, these results suggest that chronic stress may lead to increased risk of disease through its association with other known cardiovascular risk factors, namely, blood pressure, cholesterol, and obesity; however, accumulated stress still exerts an independent effect on disease risk.

Why is the relationship between chronic stress and IMT evidenced only in African Americans? One possibility is that African

Americans' greater exposure to chronic stress leads to an accelerated risk trajectory early on in life, which results in greater cumulative exposure to standard cardiovascular risk factors in midlife. In contrast, the effect of accumulated chronic stress on subclinical disease may not be manifested in Caucasian women until later in life, due to their lesser exposure to stressors. This reasoning is consistent with our findings as well as with previous reports that have found that African Americans have greater carotid IMT as compared with Caucasians (Burke et al., 1994; Sutton-Tyrrell, Zeigler-Johnson, Lassila, Holmes, & Kuller, 1998). Furthermore, race differences in the natural histories of risk factor development (e.g., blood pressure, obesity, hyperinsulinemia, and dyslipidemia) may contribute to an acceleration of risk in African Americans, at least in the very early stages of atherosclerotic progression. That is, despite the fact that African Americans and Caucasians in this sample were comparable in chronological age, the African American women may have shown greater signs of "physiological aging" based on a prolonged exposure to key risk factors. Moreover, exposure to stress may have a more adverse effect in African Americans in the early stages of atherosclerotic development by

potentiating the effects of preexisting risk factors, including metabolic processes.

Finally, consistent with contemporary stress models (e.g., Gallo & Matthews, 2003; Lazarus & Folkman, 1984), African Americans may be at a greater risk for the harmful consequences of stress because of their greater exposure to environmental stressors and the concomitant depletion of stress-buffering resources. Some researchers have argued that African American women in particular are at increased risk for psychological and physical health consequences because of their double minority status of being Black and female in American society (Carrington, 1980; Hauenstein, 1996). For instance, despite equivalent educational levels, African American women in the present study reported lower income levels and greater difficulty paying for basics than did Caucasian women. In a study assessing the impact of gender discrimination on hypertension in the Coronary Artery Disease Risk Development in Young Adults sample, Krieger (1990) found that reports of gender discrimination were associated with high blood pressure among African American participants but not among Caucasian participants. Similarly, in the present study, unfair treatment was associated with higher IMT only among African American women. Moreover, 54% of African American women attributed the cause of unfair treatment to racism, and there was a marginal association between the experience of racism and IMT and plaque. Discrimination could impact health directly through interpersonal interactions that facilitate pathogenic processes by provoking anger and exacerbating physiological reactions to stress (Clark, Anderson, Clark, & Williams, 1999) or indirectly through sociological mechanisms, such as reduced access to health care, segregation into crowded or unhealthy environments, and discriminatory employment and educational practices (e.g., Hummer, 1996; Williams & Collins, 1995).

These findings, however, must be interpreted within the context of the study limitations. First, the number of analyses conducted must temper the results due to the potential inflation of Type I errors. Second, the stress measures were based on self-report. Although we attempted to capture the participants' overall stress burden by creating a composite measure of stress, this measure was by no means a comprehensive measure of stress. The relatively poor temporal proximity between the measurement of risk factors and subclinical outcomes, due to the recruitment protocol, is another methodological limitation. Statistical criteria were used to determine potential mediators, thus maximizing the possibility of sample-specific findings. Moreover, our analysis of mediators was limited to biological and lifestyle variables measured in the study; however, it is plausible that chronic stress may impact subclinical disease through other unmeasured variables, such as neuroendocrine responses or psychosocial functioning. It is also plausible that unmeasured third variables, including coping strategies and personality style, may moderate the effect of chronic stress on subclinical disease. Finally, the use of cross-sectional data precluded us from determining the directionality of the relationship, although women were unaware of their disease state.

Despite these limitations, the present research has several notable strengths. First, the demographics of the sample are particularly notable, given that women in general, and African American women in particular, have traditionally been underrepresented in studies of psychosocial risk factors and cardiovascular health. In addition, the fact that these women were middle-aged and ap-

proaching menopause provides an important opportunity to investigate how psychosocial stress influences women's cardiovascular health at a time of increasing risk. The methods for recruitment in this sample, random digit dialing and voter registration lists, also increase the likelihood that the findings in the sample population will generalize to healthy, premenopausal women in the population at large. Finally, as we mentioned previously, this study extends the existing body of evidence examining the role of chronic stress in cardiovascular outcomes by considering a composite measure of stress that included measures of unfair treatment and economic hardship, in the context of an objective subclinical outcome. This is the first study to examine the effect of unfair treatment on a subclinical end point and, as such, provides an important extension to the limited and mixed results regarding an association between discrimination and health. Interestingly, African Americans who reported experiencing racial discrimination specifically had marginally more plaque, and a generalized measure of unfair treatment was significantly associated with IMT. The findings clearly suggest that the experience of being unfairly treated, regardless of the attributed cause, is a prevalent stressor for African Americans that may have substantial health implications.

In conclusion, this study provides an objective estimate of the association between chronic stress and subclinical outcomes in a biracial sample of healthy, premenopausal women and suggests several important strategies for future research. First, this study demonstrates the utility of using an aggregate measure of diverse indicators of stress, comprised of scores at the upper end of the distribution of stress measures, in order to create a "high-risk group." In addition, this study highlights the profound effects of race on exposure to stress and the effect of stress on disease. Importantly, African Americans, particularly women, have higher rates of cardiovascular morbidity and mortality as compared with non-Hispanic Caucasians (Gillum, 1991; Lewis, Raczynski, Oberman, & Cutter, 1991), but this excess risk can be explained only partially by standard biological risk factors. Despite the fact that our sample consisted of healthy, premenopausal women, the findings of racial differences in IMT clearly suggest that African American women are on an accelerated trajectory of disease development as compared with Caucasian women. Thus, future empirical investigations are needed to evaluate the extent to which racial differences in exposure to accumulated stress burden, including the effects of discrimination and economic burden, account for racial disparities in cardiovascular health.

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