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## Interactive Relations Across Dimensions of Interpersonal-Level Discrimination and Depressive Symptoms to Carotid Intimal-Medial Thickening in African Americans

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### Abstract

**Objective:** This study aimed to examine within-race interactions of multiple dimensions of self-reported discrimination with depressive symptoms in relation to carotid intimal-medial thickness (IMT), a subclinical marker of atherosclerosis prospectively implicated in stroke incidence, in middle-aged to older African American and white adults.

**Methods:** Participants were a socioeconomically diverse group of 1941 African Americans (56.5%) and whites from the Healthy Aging in Neighborhoods of Diversity across the Life Span study (30–64 years old, 47% men, 45.2% with household income <125% federal poverty threshold) who underwent carotid IMT measurement. Discrimination was assessed across four dimensions (everyday, frequency across various social statuses, racial, and lifetime burden). The Center for Epidemiologic Studies Depression scale was used to assess depressive symptoms.

**Results:** In cross-sectional hierarchical regression analyses, two interactions were observed in African Americans: more frequent discrimination across various social statuses ( $b < 0.001$ ,  $p = .006$ ) and a higher lifetime discrimination burden ( $b < 0.001$ ,  $p = .02$ ) were each related to thicker carotid IMT in those with greater depressive symptoms. No significant findings were observed within whites.

**Conclusions:** Among African Americans, those reporting high levels of discrimination and depressive symptoms have increased carotid atherosclerosis and may be at greater risk for clinical end points compared with those reporting one or neither of these risk factors. Findings suggest

that assessment of interactive relationships among social and psychological factors may elucidate novel pathways for cardiovascular disease, including stroke, among African Americans.

### Keywords

atherosclerosis; health disparities; intimal-medial thickness; subclinical vascular disease; risk factors; psychosocial vulnerabilities

## INTRODUCTION

Black-white disparities in stroke risk in the United States exceed 300% between the ages of 45 and 65 years (1). For example, in 2014, it was estimated that 22,384 excess strokes occurred in African Americans relative to whites. Although this racial disparity attenuates at older ages, the ensuing disability, secondary stroke, and mortality risks contribute to a substantial and protracted burden of disease in African Americans. Traditional risk factors and socioeconomic status (SES) explain only ~50% of the excess risk observed in African Americans (1), prompting researchers to call for the identification of psychosocial factors that may be contributing (1). Ultimately, identifying novel factors associated with *early* indicators of disease may be a crucial step in reducing the disproportionate stroke burden among African Americans.

The prognostic disease process underlying stroke incidence is atherosclerosis, which can begin early in the life course (2). Carotid intimal-medial thickness (IMT), measured with B-mode carotid ultrasonography, provides an early marker of subclinical atherosclerosis (3). IMT is prospectively linked to stroke incidence in both middle-aged (4) and older adults (5), as well as in minority racial groups, including African Americans (6,7).

Interpersonal discrimination, which is broadly defined as unfair treatment experienced in one on one interactions (8), is an established social stressor and predictor of cardiovascular disease (CVD; (9–14)). African Americans report significantly higher rates of interpersonal discrimination across various race-and non-race-related dimensions than other race groups, especially whites (15–17). Some prior work has demonstrated that specific types of interpersonal discrimination and even expectations of future interpersonal discrimination (18) may be adversely linked to subclinical vascular disease, including IMT, in African Americans (18–20). For example, in the Study of Women’s Health Across the Nation (SWAN), everyday discrimination, which captures minute but chronic insults without specifying the reason for the discrimination, was associated with concurrently greater IMT in African American women (20). However, in a subsequent longitudinal analysis of the same sample, cumulative exposure to everyday discrimination for 10 years was not a significant predictor of IMT in African American women (19). Similarly, everyday discrimination and lifetime discrimination burden were unrelated to IMT in 3029 African Americans in the Jackson Heart Study (21). Studies examining interpersonal discrimination in relation to other subclinical markers of CVD have also been mixed, but largely demonstrate an adverse linkage (22–29).

At least four considerations may shed light on these inconsistent findings. First, interpersonal discrimination is a multidimensional construct that can be captured in several

ways. For example, one can focus on whether or not the reason for the discrimination is discerned as being due to specific attributes (e.g., race, age, or sex; (16)) or social statuses occupied by the targeted individual, the frequency of exposure to particular types of discriminatory events (e.g., weekly), the time frame of assessment (e.g., lifetime), and/or the subjective bearing or *weight* associated with these experiences altogether. Researchers have recently posited that variations in exposure to these different forms of discrimination may yield variations in health end points (8) and accordingly have called for simultaneous examination of various dimensions of discrimination in relation to health disparities (30). For instance, in a recent report from the Jackson Heart Study, major lifetime discrimination and the overall burden of discrimination were associated with greater hypertension risk, whereas everyday discrimination was not. Similarly, a recent report in 26,992 adults in the National Epidemiologic Survey of Alcohol and Related Conditions study demonstrated that reports of interpersonal discrimination from multiple sources (e.g., racial, weight, and sex discrimination) were more strongly predictive of self-reported CVD incidence compared with fewer sources (31). Most prior studies of cardiovascular-related end points, however, have focused on a single dimension of discrimination and therefore may not have fully captured the complexity of discriminatory experiences (9,32). Furthermore, the methodologies used to assess even one type of discrimination may vary between studies and contribute to discrepant results. For example, some CVD risk studies examining non-race-based discrimination (e.g., everyday discrimination; e.g., Refs. (20,23)) assess *attributions* for these experiences. That is, they use a two-stage approach where the questions initially assess “unfair treatment” without regard for the reason but then pose a follow-up item to ascertain a primary attribution (e.g., race) for these experiences. In contrast, the one-stage approach explicitly focuses on various aspects of the respondent’s experiences with racial discrimination in the primary measure. Emerging evidence demonstrates that these two approaches are distinct and not empirically equivalent (33), yielding significantly different respondent reports (e.g., Refs. (15,34,35)). Some confusion about the consistency of the relation between racial discrimination and CVD end points in the literature may therefore be due to the use of the term *racial discrimination* to refer to studies examining *attributions* to race for everyday discrimination, as well as studies explicitly examining racial discrimination.

Second, exploration of discrimination associated with interlocking social statuses may be critical. In this regard, the intersectionality framework posits that discriminatory events linked to the multiple identities that one occupies (e.g., African American race, female sex, and older age) are not independent or unidimensional but instead are interdependent (36,37). It has been suggested that an individual’s experiences with discrimination may be uniquely influenced by these mutually constitutive social statuses (38), and emerging evidence supports this proposition as related to health end points (e.g., Refs. (39,40)). However, to our knowledge, no prior work has examined discrimination related to multiply occupied social statuses (e.g., race, sex, and SES) in association with CVD end points, including subclinical vascular risk markers such as IMT.

Third, in a similar vein, elucidating within-race heterogeneity may yield critical insight into how experiences of discrimination shape CVD disparities between races. Although much of the literature examining interpersonal discrimination and CVD has focused on African

Americans, emerging research demonstrates adverse CVD-related outcomes in whites who report these experiences as well (19,41). Presumably, however, the sociohistorical context surrounding race in the United States has shaped divergent bases for the linkages between discrimination and health in whites versus African Americans (39,42). For instance, cognitive and social factors underlie individual perceptions of discrimination (43) and likely lead to qualitatively different experiences in majority versus minority groups (44). As an example, several studies show that whites' perceptions of being unfairly treated because of their race are driven by growing resentment due to shifts in the racial status hierarchy or the belief that gains by racial minorities reflect a zero-sum game for whites (45–47). In contrast, enduring evidence of race-based inequities in key domains including politics, education, health care, the criminal justice system, and the economy underscores and parallels African Americans' reports of discrimination in interpersonal-level interactions (48). Previous investigations of interpersonal discrimination and IMT have either focused solely on one racial group (49) or have investigated the moderating role of race (19,20), thereby emphasizing intergroup differences. Although such approaches add to the literature on race, discrimination, and cardiovascular risk, they may obscure potentially vital within-group processes. Therefore, we use a within-group framework to elucidate patterns in the linkages of discrimination with IMT separately within African Americans and within whites.

Fourth, none of the previous studies on discrimination and IMT considered potential psychological risk moderators of this relationship. Specifically, depressive symptoms are an independent predictor of stroke and are more prevalent in those reporting discrimination. Findings from the Health and Retirement Study and the National Health and Nutrition Examination Survey demonstrated that in both African Americans and whites, a) increased depressive symptoms were prospectively associated with a 25% increased stroke risk, with evidence for a dose-response relationship (50); b) a high level of depressive symptoms conferred a 50% to 160% increased stroke risk during a 22-year follow-up compared with a low level of depressive symptoms (51); and c) this latter pattern was most pronounced among African Americans, as greater depression put them at  $\approx 3$ -fold increased stroke risk (51). Similarly, depressive symptoms predict progression of IMT over time after accounting for traditional cardiovascular risk factors (52,53). In addition, African Americans experience greater chronicity and severity of depressive symptoms (54,55), with symptoms more likely to go untreated (56), compared with whites. Notably, data across five longitudinal studies (57–61) examining the linkage between discrimination and depressive symptoms indicate that a) the two constructs are conceptually distinct and b) generally, various dimensions of interpersonal discrimination precede the onset and worsening of depressive symptoms rather than the reverse.

We posit that the coupling of an interpersonal, socially based susceptibility (discrimination) with an intrapersonal psychological vulnerability (depressive symptoms) may contribute to greater subclinical vascular disease. Broadly, this proposition of a “coupling” effect draws upon an integrative framework including the diathesis-stress model and the biopsychosocial model of racism, the weathering hypothesis, and social-evaluative threat theory (10,62–64). Altogether, this framework suggests that the synergistic effect of a stressful event combined with psychological distress may uniquely increase risk for CVD. Indeed, psychological stressors are thought to influence physiology via activation of specific cognitive and/or

affective processes and their related central nervous system underpinnings. Individuals with elevated depressive symptoms may have diminished resources for coping with the stress of chronic interpersonal discrimination and thus show exacerbations in physiological changes that contribute to disease (65). Similarly, events evaluated as threatening to an individual's social identity or standing, such as discriminatory acts, can mobilize emotive reactions, and individuals experiencing higher levels of depressive symptoms may be more vulnerable to the stress-related risk for these events. If such factors are chronically in play, a wear and tear of essential physiological systems would be expected and may emerge on an accelerated trajectory, such as in middle age. Consistent with this conceptualization, an analysis of more than 5000 African Americans in the National Survey of American Life study demonstrated that those who reported high levels of racial discrimination *and* a history of mood disorder, including depression, were at the greatest risk for CVD (65). However, to our knowledge, no prior studies have assessed whether depressive symptoms exacerbate the relation between interpersonal discrimination and carotid IMT.

The current study examines interactive associations among different dimensions of self-reported discrimination with depressive symptoms in relation to subclinical vascular disease as indicated by carotid IMT in middle-aged to older, community-dwelling African Americans in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study. We focus primarily on African Americans because of the disproportionate burdens of discrimination and stroke observed in this group. With regard to discrimination, we include a comprehensive assessment of both race- and non-race-based forms of discrimination. Specifically, we focus on measurements that capture everyday discrimination without attribution for the cause, frequency of experiences of discrimination that individuals attribute to various social statuses they concurrently occupy (e.g., age, race, sex, etc.), racial discrimination, and the subjective impact or *weight* of discrimination throughout the lifetime. We hypothesize that the positive relationships between these four dimensions of interpersonal-level discrimination and carotid IMT will be more pronounced among African Americans with greater depressive symptoms. We also anticipate that the strength of the relationships may vary across the specific dimensions of discrimination. However, we do not posit a priori hypotheses for each of the four dimensions assessed because of mixed findings across studies examining single dimensions of discrimination (14,19) and few studies examining multiple dimensions (65–68) in relation to CVD end points. Although African Americans are the primary focus, emerging findings document an adverse impact of discrimination in whites, as well. Thus, we also conduct exploratory analyses in whites that are analogous to the primary analyses in African Americans. In addition, we directly examine racial differences in these associations by testing three-way interactions among discrimination indices, depressive symptoms, and race with carotid IMT in the overall sample of African Americans and whites.

We anticipate that associations between discrimination, depressive symptoms, and carotid IMT will be independent of established cardiovascular risk factors (e.g., body mass index [BMI], hypertension, diabetes, CVD, lipid-lowering medication use, cigarette smoking, and alcohol and/or illicit drug use) and key sociodemographic factors (i.e., age, sex, SES). Alternatively, it is plausible that depressive symptoms could serve as an intermediate pathway (69–71) linking discrimination to poorer health. Therefore, we will

also explore the potential mediating effect of depressive symptoms on the association between interpersonal-level discrimination and carotid IMT following adjustment for the aforementioned cardiovascular and sociodemographic factors.

## MATERIALS AND METHODS

### Participants and Procedure

HANDLS is a longitudinal study of health disparities attributable to race and SES in an urban sample. The study's design and implementation has been described elsewhere (72). Briefly, HANDLS participants are a fixed cohort of African American and white adults recruited via household screenings from an area probability sample of 13 census segments in the city of Baltimore, Maryland, which were selected for their likelihood of yielding representative samples of participants who were African American or white, male or female, and with household incomes of  $\geq 125\%$  or  $<125\%$  of the 2004 federal poverty level. In addition, participants were deemed eligible for HANDLS if they were between 30 and 64 years of age at baseline. The institutional review board at the National Institute of Environmental Health Sciences approved the HANDLS study protocol.

The present study used data collected during the first wave of HANDLS, which occurred between 2004 and 2009 and consisted of two phases: a) recruitment, written informed consent, and an interview within participants' homes, and b) medical history, physical examination, and other assessments on mobile medical research vehicles (MRVs) parked within participants' neighborhoods. After initial screening, participants were excluded from HANDLS if they met any of the following criteria: a) unable to provide informed consent, b) currently pregnant, c) severe developmental disability, d) within 6 months of active cancer treatment (i.e., chemotherapy, radiation, or biological treatments), e) diagnosed as having AIDS, or f) were unable to provide valid government-issued identification or a verifiable address (72). Examinations were performed on those participants whose blood pressure was  $<160/100$  mm Hg. Of the 3720 participants who were recruited, 2801 were deemed eligible for continued participation in the study, of whom 2226 had valid carotid ultrasonography data (56.2% African American, 43.8% white). Participants were deemed ineligible for carotid ultrasonography if they had a) elevated blood pressure at the time of ultrasound ( $>200/100$  mm Hg), b) presence of carotid bruit, c) weight  $\geq 295$  lb, or d) inability to lie in a completely supine position for 15 minutes. In addition, participants were excluded if they reported a history of stroke ( $n = 37$ ), transient ischemic attack ( $n = 54$ ), heart failure ( $n = 43$ ), or carotid endarterectomy ( $n = 2$ ). No participants with valid carotid IMT data had a history of aneurysm. Finally, after medical exclusions were applied, participants were excluded if they were missing data for any variables used in the present analyses ( $n = 165$ ), resulting in a final analysis sample of 1941 participants, of whom 1097 were African American and 844 were white. Of note, missing data for depressive symptoms and total cholesterol were previously imputed in the larger HANDLS sample by regression, with groups stratified by race, poverty status, and sex ( $<10\%$  of HANDLS participants were missing these variables in the originally).

## Measures

**Dimensions of Interpersonal-Level Discrimination**—The nine-item *Everyday Discrimination* scale (73) measures the frequency of routine experiences of discrimination without requiring the participant to make an explicit attribution (e.g., race/ethnicity) for the experience. Participants are asked, “In your day-to-day life, how often have any of the following things happened to you?” Examples of items are “How often are you treated with less courtesy than other people?” and “How often do you get worse service at restaurants and stores than other people?” Participants responded to items on a 6-point scale ranging from 1 (*almost every day*) to 5 (*less than once a year*), or 6 (*never*). Responses were reversed scored and summed, such that possible scores ranged from 9 to 54 and higher scores indicating greater everyday discrimination. This scale has been shown to have strong internal consistency ( $\alpha = .88$ ; (54)) and was similarly strong among African Americans and whites in our sample,  $\alpha = .85$ .

Participants completed the 10-item *Frequency of Discrimination across Sources* measure, which assessed how often they experienced various sources of discrimination. Specifically, these items asked, “Overall how much have you experienced prejudice or discrimination due to...” sex, race, ethnicity, income, age, religion, physical appearance, sexual orientation, health status, and disability. The items do not specify a time frame. Participants responded to items on a 4-point scale ranging from 1 (*not at all*) to 4 (*a lot*). Possible scores ranged from 10 to 40, with higher scores indicating greater frequency of discrimination from various sources. This measure was derived from a similar measure used in previous research to assess prevalence of discrimination in health care settings (74). This scale had high internal consistency among African Americans and whites in our sample,  $\alpha = .85$ .

*Racial discrimination* was assessed with a measure previously used in epidemiologic research (75). This six-item inventory ascertains whether participants ever (i.e., across their lifetimes) experienced racial discrimination at school, when getting a job, at work, when getting housing, when getting medical care, and from police or in courts. Participants responded *no* (0) or *yes* (1) to each item. Possible scores ranged from 0 to 6, with higher scores indicating greater racial discrimination. These six items were among those included in versions of the Experiences of Discrimination scale, which has strong internal consistency ( $\alpha = .74$ ) and test-retest reliability (0.70; (15)). In our sample of African Americans and whites, this scale had strong internal consistency,  $\alpha = .84$ .

*Lifetime discrimination burden* was assessed with a two-item measure (16,76,77). Specifically, these items asked, a) “Overall, how much harder has your life been because of discrimination?” and b) “Overall, how much has discrimination interfered with your life?” Participants responded to each item on a 4-point scale ranging from 1 (*not at all*) to 4 (*a lot*). Possible scores ranged from 2 to 8, with higher scores indicating greater lifetime discrimination burden. These items have previously been used in epidemiologic studies of African Americans (21,66). In the present sample of African Americans and whites, these two items were strongly correlated,  $r = 0.79$  ( $p < .001$ ).

**Depressive Symptoms**—Depressive symptoms were assessed with the 20-item *Center for Epidemiologic Studies Depression* (CES-D) scale, which has strong psychometric

properties (78). Participants responded to items on a 4-point scale ranging from 0 (*rarely*) to 3 (*mostly*). Possible scores ranged from 0 to 60, with higher scores indicating greater depressive symptoms. This measure had strong internal consistency among African Americans and whites in our sample,  $\alpha = .91$ .

**Carotid IMT**—High-resolution B-mode ultrasonography of the left common carotid artery was performed with a standard transducer (5.0L45) and equipment (Acuson CV 70; Siemens). The far arterial wall IMT was evaluated as the distance between the intimal-luminal interface and medial-adventitial interface in a region 1.5 cm proximal to the carotid bifurcation. Specific care was taken to measure IMT in areas devoid of plaque. IMT was measured on a frozen-frame image, magnified to achieve higher resolution of detail. The IMT measurement was obtained from five contiguous sites at approximately 1-mm intervals and averaged for analyses. Sonography measurements were performed by a single sonographer and reviewed by a board-certified cardiologist, who adjusted them as necessary.

**Adjustment Variables**—Similar to our prior work (79), adjustment variables included age, biological sex, SES, BMI, total cholesterol, hypertension, diabetes, CVD, lipid-lowering medication use, cigarette smoking, and alcohol and/or illicit drug use. Participants self-reported their age, and biological sex was coded as 0 (women) and 1 (men). SES was calculated from a composite score that included self-reported annual household income and years of education. Participants were considered *higher SES* (coded as 0) if they reported a) an annual household income (adjusted for household size)  $\geq 125\%$  of the 2004 federal poverty level and b)  $\geq 12$  years of education. Conversely, participants were classified as *lower SES* (coded as 1) if they reported a) an adjusted annual household income  $<125\%$  of the 2004 federal poverty level or b)  $<12$  years of education. The Wide Range Achievement Test (WRAT-3) Word Reading subtest was administered at the MRV visit and used as a measure of literacy (80).

During the MRV visit, participants completed a comprehensive physical examination and medical history with a physician or nurse practitioner. BMI was calculated as the ratio of weight to height squared (in kilograms per meter squared), both measured with calibrated equipment. Fasting venous blood specimens for total cholesterol assay were collected on the MRV and analyzed at the NIA Clinical Research Branch Core Laboratory (Baltimore, Maryland) and Quest Diagnostics Inc. (Baltimore, Maryland, and Chantilly, Virginia) using a spectrophotometer (AU5400 Immuno Chemistry Analyzer; Olympus, Center Valley, Pennsylvania). Hypertension and diabetes were represented as dichotomous variables (coded as 0 [absent] and 1 [present]) in the present study. Hypertension was based on self-reported diagnosed hypertension, self-reported use of antihypertensive medications (diuretics, blockers, angiotensin inhibitors, or vasodilators), or resting systolic blood pressure  $\geq 140$  mm Hg or diastolic blood pressure  $\geq 90$  mm Hg. Diabetes was based on self-reported diagnosed diabetes, self-reported use of diabetes medications, and/or glucose  $\geq 126$  mg/dl (7 mmol/L). Next, a dichotomous CVD cluster variable was created to indicate the absence or presence (coded as 0 and 1, respectively) of self-reported diagnoses of coronary artery disease, myocardial infarction, peripheral artery disease, atrial fibrillation, angioplasty, and/or coronary artery bypass surgery. Other dichotomous adjustment variables



were self-reported lipid-lowering medication use (0, never prescribed; 1, ever prescribed), self-reported cigarette smoking status (coded as 0 [never used regularly] and 1 [ever used regularly]), and self-reported alcohol and/or illicit drug use (i.e., alcohol, marijuana, cocaine, and/or opiate use status collapsed into one dichotomous variable; coded as 0 [never used regularly] and 1 [ever used regularly]).

## Statistical Analyses

**Race-Stratified Moderation Analyses**—Statistical analyses were conducted with the Statistical Package for the Social Sciences (SPSS) version 24. Moderation analyses were stratified by race. That is, primary analyses were first run in the African American participants, followed by parallel, exploratory analyses in the white participants. Hierarchical-entry multiple linear regression was used to examine main effects and interactive relations of multiple forms of discrimination (see Dimensions of Interpersonal-Level Discrimination measures) and depressive symptoms with carotid IMT. Three models were run for each discrimination measure. Model 1 included the main effect of discrimination and all covariates. Next, model 2 added the main effect of depressive symptoms. Finally, model 3 added the interaction effect of discrimination by depressive symptoms. If the interaction term was significant, model 3 was retained, and main effects of discrimination and depressive symptoms were not interpreted further (irrespective of their significance across models). Conversely, if the interaction term was nonsignificant, it was removed from the analysis and model 2 was retained as the final model.

The PROCESS macro for SPSS, Version 2.16 (81), was used to probe significant interaction effects using model template 1 (moderation with one moderator) within the package. Within PROCESS, the Johnson-Neyman technique (81) was used to detect regions of significance for the conditional effect of discrimination with carotid IMT across a wide range of CES-D scores. Significant interactions were also plotted at the mean  $\pm$  1 SD of depressive symptoms to assist with interpretation.

All adjustment variables were mean centered before analyses. To assist with interpretation of the Johnson-Neyman findings, we present findings from analyses without mean-centering discrimination and depressive symptoms. However, subsequent analyses confirmed that mean centering these variables did not change the results.

**Combined-Sample Moderation Analyses**—To test racial differences in the discrimination by depressive symptoms interaction, three-way interactions of discrimination measures, depressive symptoms, and race were tested in the overall sample consisting of African Americans and whites.

**Mediation Analyses**—Alternative mediation analyses were also run to determine whether depressive symptoms mediated associations between discrimination and carotid IMT. Mediation analyses were race stratified, with primary analyses run in the African American participants first, followed by parallel, exploratory analyses in the white participants. Mediation analyses were conducted using a bootstrapping confidence interval approach to examine potential mediating effects of depressive symptoms on the association between multiple forms of discrimination and carotid IMT. Within PROCESS, model template 4

(mediation with one mediator) was used within the sample, and 5000 bootstrap samples and a 95% confidence interval were requested. Mediation analyses adjusted for all covariates described previously.

## RESULTS

### Sample Descriptives

African Americans were more likely to have lower SES ( $\chi^2(1) = 27.09, p < .001$ ), hypertension ( $\chi^2(1) = 10.18, p = .001$ ), and a self-reported history of cigarette use ( $\chi^2(1) = 5.86, p = .017$ ) than their white counterparts (Table 1). Conversely, white participants were more likely to be women ( $\chi^2(1) = 4.25, p = .043$ ) and have a self-reported history of lipid-lowering medication use ( $\chi^2(1) = 8.20, p = .005$ ) than African Americans. Whites also had greater total cholesterol ( $t(1,939) = 4.35, p < .001$ ) and BMI,  $t(1,939) = 2.76, p = .006$ , whereas African Americans had greater carotid IMT ( $t(1,939) = -5.61, p < .001$ ). African Americans endorsed more interpersonal-level discrimination than did whites across all discrimination measures ( $p$  values  $< .001$ ). In the overall sample, 468 (24.1%) of participants self-reported a history of depression, with whites reporting greater rates of depression than African Americans, ( $\chi^2(1) = 55.73, p < .001$ ). We also observed that the discrimination measures were moderately to strongly correlated with one another, ranging from  $r = 0.39$  to  $r = 0.67$  (all,  $p$  values  $< .001$ ; Supplementary Table 1, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>). Correlations between depressive symptoms and each of the discrimination measures were small, ranging from  $r = 0.12$  to  $r = 0.26$  (all,  $p$  values  $< .001$ ).

### Moderation Results

**Race-Stratified Moderation Results**—Among African Americans, findings revealed a significant main effect of frequency of discrimination across sources before adjustment for depressive symptoms ( $b = 0.001, \beta = 0.06, p = .045$ ), such that greater scores on the measure were associated with greater carotid IMT (see model 1 in Table 2). This main effect attenuated to nonsignificance after adjustment for depressive symptoms ( $b = 0.001, \beta = 0.06, p = .052$ ; see model 2 in Table 2). In addition, this main effect was not interpreted further after subsequent analyses revealed it to be subsumed within a significant two-way interaction of frequency of discrimination across sources by depressive symptoms ( $b < 0.01, \beta = 0.26, p = .006$ ; see model 3 in Table 2). Subsequent analyses using the Johnson-Neyman technique revealed that greater frequency of discrimination across sources was associated with greater carotid IMT at all CES-D scores  $> 16.7$  (62nd percentile; all,  $p$  values  $< .05$ ; Supplementary Table 2, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>).

We observed no main effect of lifetime discrimination burden on carotid IMT among African Americans before adjustment for depressive symptoms ( $b < 0.01, \beta = 0.01, p = .807$ ) or after adjustment for depressive symptoms ( $b < 0.01, \beta < 0.01, p = .955$ ; see models 1 and 2 in Table 3). However, findings revealed a significant two-way interaction of lifetime discrimination burden by depressive symptoms ( $b < 0.01, \beta = 0.19, p = .021$ ; see model 3 in Table 3) in this group. The Johnson-Neyman technique demonstrated that greater lifetime

discrimination burden was associated with greater carotid IMT at CES-D scores  $>20.82$  (75th percentile; all,  $p$  values  $< .05$ ; Supplementary Table 3, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>). As shown in Figure 1, among African American participants with high depressive symptoms (i.e., CES-D score of 25.53, +1 SD above mean), greater carotid IMT was associated with greater a) frequency of discrimination from various sources ( $b = 0.003$ ,  $p = .001$ ) and b) lifetime discrimination burden ( $b = 0.01$ ,  $p = .018$ ).

Further analyses revealed a significant main effect of everyday discrimination with carotid IMT among African Americans before adjustment for depressive symptoms ( $b = 0.001$ ,  $\beta = 0.07$ ,  $p = .017$ ) and after adjustment for depressive symptoms ( $b = 0.001$ ,  $\beta = 0.07$ ,  $p = .021$ ; see models 1 and 2 in Table 4, respectively). As shown in Figure 2, greater everyday discrimination was associated with greater carotid IMT in this group. The interaction of everyday discrimination by depressive symptoms was nonsignificant ( $b < 0.01$ ,  $\beta = 0.08$ ,  $p = .221$ ; see model 3 in Table 4).

Findings also revealed a) nonsignificant main effects of racial discrimination before adjustment for depressive symptoms ( $b < .01$ ,  $\beta = -0.01$ ,  $p = .825$ ) and after adjustment for depressive symptoms ( $b < 0.01$ ,  $\beta = -0.01$ ,  $p = .757$ ; see models 1 and 2 in Supplementary Table 4, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>), and b) a nonsignificant interaction of racial discrimination by depressive symptoms ( $b < 0.01$ ,  $\beta = 0.22$ ,  $p = .095$ ; see model 3 in Supplementary Table 4, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>), in association with carotid IMT among African Americans. Finally, across analyses, there were no significant main effects of depressive symptoms in the African American participants before adjustment for interaction terms (all,  $p$  values  $> .514$ ; see model 2 in Tables 2–4 and Supplementary Table 4, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>).

Next, among whites, there were no significant main effects of a) everyday discrimination ( $b < 0.01$ ,  $\beta < 0.01$ ,  $p = .900$ ), b) frequency of discrimination across sources ( $b < 0.01$ ,  $\beta = -0.01$ ,  $p = .870$ ), (c) racial discrimination ( $b < 0.01$ ,  $\beta < 0.01$ ,  $p = .972$ ), or d) lifetime discrimination burden ( $b < .01$ ,  $\beta = 0.01$ ,  $p = .807$ ) with carotid IMT; these effects remained nonsignificant after adjustment for depressive symptoms (all,  $p$  values  $> .744$ ). Main effects of depressive symptoms with carotid IMT among whites were also nonsignificant across analyses (all,  $p$  values  $> .414$ ). Likewise, among whites, there were no significant interactions of a) everyday discrimination by depressive symptoms ( $b < 0.01$ ,  $\beta = 0.01$ ,  $p = .824$ ), b) frequency of discrimination across sources by depressive symptoms ( $b < 0.01$ ,  $\beta = 0.02$ ,  $p = .535$ ), c) racial discrimination by depressive symptoms ( $b < 0.01$ ,  $\beta = 0.02$ ,  $p = .557$ ), or d) lifetime discrimination burden by depressive symptoms ( $b < 0.01$ ,  $\beta = 0.03$ ,  $p = .400$ ) with carotid IMT. Results of these exploratory analyses are shown in Supplementary Tables 5–8, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>.

**Combined-Sample Moderation Results**—In the overall sample, there were no significant three-way interactions among a) everyday discrimination by depressive symptoms by race ( $b < .01$ ,  $\beta = .05$ ,  $p = .645$ ), b) frequency of discrimination across sources

by depressive symptoms by race ( $b < .01$ ,  $\beta = .18$ ,  $p = .196$ ), c) racial discrimination by depressive symptoms by race ( $b < .01$ ,  $\beta = .10$ ,  $p = .672$ ), and (d) lifetime discrimination burden by depressive symptoms by race ( $b < .01$ ,  $\beta = .11$ ,  $p = .334$ ) with carotid IMT. Results of these analyses are shown in Supplementary Tables 9–12, Supplemental Digital Content 1, <http://links.lww.com/PSYMED/A595>.

**Mediation Results**—Finally, across all analyses, depressive symptoms did not significantly mediate associations between any of the discrimination indices and carotid IMT among African Americans or whites (all 95% confidence intervals overlapped with 0; data not shown).

## DISCUSSION

In an urban sample of middle-aged and older adults, we identified interaction effects between dimensions of interpersonal-level discrimination and depressive symptoms in relation to early stage carotid atherosclerosis in African Americans. Specifically, African Americans reporting either a) greater exposure to discrimination across multiple sources or b) greater lifetime discrimination burden, in conjunction with increased levels of depressive symptoms, had greater average carotid IMT than did those reporting lower levels of discrimination or depressive symptoms. In secondary analyses, race did not moderate the relations among depressive symptoms, discrimination, and IMT across whites and African Americans, and depressive symptoms did not mediate any of the relationships between discrimination and IMT.

Discrimination, depressive symptoms, and their interaction were not associated with IMT among white participants. There are several potential reasons for the lack of analogous findings in whites. For one, reports of all types of discrimination, along with IMT levels, were higher among African Americans than among whites, which may suggest the presence of threshold effects. African Americans, in facing more severe, frequent, or long-standing interpersonal discrimination, may begin to develop atherosclerosis on an earlier or more accelerated trajectory compared with whites, especially when considered alongside other risk factors (i.e., elevated rates of hypertension in African Americans). In addition, or alternatively, the processes underlying perceptions and reports of discrimination could be qualitatively different between these two racial/ethnic groups (45,46,82), such that these experiences have more of a deleterious cardiovascular impact in African Americans. Continuing to examine the associations between psychosocial factors and cardiovascular risk factors from a within-race perspective may contribute to our understanding of racial disparities in cardiovascular outcomes. This approach may be particularly useful when seeking to explicate factors uniquely linked to the lived experience of particular racial groups, as discrimination is for African Americans.

Relatedly, race was not a statistically significant moderator of the associations among discrimination indices, depressive symptoms, and carotid IMT in the overall sample. This may suggest that there are truly no racial differences between African Americans and whites in the linkages of interpersonal discrimination and depressive symptoms to IMT. Alternatively, the underlying process linking interpersonal discrimination to IMT may be

dissimilar in African Americans and whites. The literature is quite clear that African Americans experience more discrimination (15–17) and also have greater carotid IMT compared with whites, with racial differences in IMT potentially stronger in younger adult samples (83–86), such as HANDLS participants. In addition, the overarching hypothesis in health disparities research is that African Americans are more severely affected by CVD end points and that the related subclinical disease processes begin at younger ages. Thus, it may not be appropriate to directly contrast these two racial groups when examining relationships among these variables. We also know that assessment of race alone is not an adequate proxy for the lived and dynamic experience of race, especially among historically marginalized racial groups such as African Americans. Thus, factors more closely aligned with the experience of a minority racial group may be best assessed for within-group variability, as critical distinctions may be lost if emphasis is only placed on interpreting differences across or between a majority and minority racial group. Here, owing to the racial disparities in discrimination and IMT, and limitations in the contribution of traditional risk factors, beyond the question of whether *there are race differences*, we sought to elucidate *whether and how* discrimination, a central aspect of life for African Americans, impacts IMT (86,87). Learning more about such within-group variability may lead to the development of more effective policies specific to minorities.

To our knowledge, no previous studies have examined depression as a moderator of the relationship between interpersonal discrimination and IMT. However, our results are consistent with a prior study of African Americans, in which the combination of both racial discrimination and a history of a mood disorder (depression, dysthymia, or bipolar disorder) was associated with self-reported CVD, as defined by hypertension, atherosclerosis, myocardial infarction, or stroke (57). Several biopsychosocial mechanisms could account for this effect. With regard to psychosocial factors, theoretical models of discrimination and health among African Americans have emphasized the potential moderating role of psychological factors, which may influence how individuals recognize, appraise, and react to stressors (10,88). For instance, prior work has suggested that relationships between discrimination and health outcomes may be buffered by an active coping style or increased social support (89,90), both of which may be limited in those with elevated depressive symptoms. Frequent exposure to discrimination may also promote *psychological weathering* and/or heightened perceptions of social threat (61,64). Individuals who are simultaneously experiencing depressive symptoms along with exposure to discrimination may be more attuned to instances of discrimination and/or experience them as more psychologically taxing than those with fewer symptoms. Over time, this may lead to physiological changes that harm the cardiovascular system. Specifically, depressive symptoms are associated with a host of neuroendocrine, immune, and autonomic perturbations that heighten cardiovascular risk (91,92). Thus, in those with symptoms of depression, physiological responses to the stress of discrimination may be amplified and, when experienced repeatedly, could contribute to an accelerated progression of atherosclerosis (93–95). Finally, depressive symptoms are linked to a variety of unhealthy behaviors such as smoking (61,89), difficulty with medication adherence (96), and poor sleep (97,98), any of which could exacerbate cardiovascular risk among those experiencing discrimination. Additional work exploring

the behavioral and physiological sequelae of the combination of higher discrimination and greater depressive symptoms could shed light on these potential pathways.

The current study demonstrates the importance of examining discrimination as a multidimensional construct. Here, we observed that discriminatory experiences linked to multiple social categories one concurrently occupies, including race, ethnicity, sex, age, and SES, as well as the overall *weight* or burden of discriminatory experiences across one's lifetime were implicated in early atherosclerosis. This work echoes recent findings that suggest that reports of interpersonal discrimination from multiple sources (e.g., racial, weight, and sex discrimination) were related to self-reported CVD incidence (23). It is plausible that the synergistic nature of holding various social statuses for which one may be targeted for discrimination in varying or consistent degrees is especially potent. This particular finding may exemplify the need for further examination of discrimination and health within an intersectionality framework. This framework may be apt for such work as it posits that multiple social categories intersect at the microlevel of the individual experience to reproduce various macrolevels and mesolevels, interconnecting systems of disadvantage (36,37), in turn, promoting and maintaining health disparities. Next steps should seek to explicate whether and how specific social-status based discriminatory experiences, such as racial discrimination, may be especially deleterious in the context of exposure to other social status-based discrimination (e.g., sex) and if combinations of particular social statuses may further compromise or protect against adverse effects. In this regard, examination of the intersectionality paradox—where individuals occupy low (e.g., racial minority and physically challenged) and high (e.g., higher SES) status social identities—may further reveal subgroups for whom greater or lesser health disparities have previously been unknown.

We also observed that the burden of discrimination across one's lifetime may be particularly meaningful in understanding the impact of discrimination on subclinical vascular disease. This finding is consistent with prior reports demonstrating that the weight of discrimination is adversely linked with CVD end points (66–68). For instance, prior studies in African Americans observed that greater lifetime burden was associated with greater white matter lesion volume (subclinical marker of cerebrovascular disease; [68]), hypertension prevalence (66), and poorer health behaviors (i.e., smoking and sleep; (67)). This finding may reflect the importance of explicitly capturing the individuals' perception of how their experiences with discrimination have influenced them, in addition to assessing the frequency and types of discrimination they have faced. Specifically, understanding one's sense of how discriminatory experiences have blocked opportunities and created difficulties for him/her may shed light on the lived consequences or fallout of discriminatory experiences not fully captured in measures that take more of a quantitative focus.

Notably, everyday discrimination and racial discrimination, which assess the frequency of experiences with relatively minor discriminatory events and whether one has experienced racial discrimination across specific settings, respectively, were *not* related to carotid IMT in this sample. With regard to everyday discrimination, mixed findings have emerged across prior studies (19,66,67,99). Although there is some evidence that attributions to race for experiences of everyday discrimination are linked with greater IMT in African

Americans (20), studies explicitly examining racial discrimination in relation to IMT are lacking. Drawing on the discrimination findings overall, it is possible that exposure to discrimination associated with multiple social statuses, including racial discrimination, is particularly noxious for African Americans in ways that everyday discrimination and racial discrimination alone may not fully capture.

The varied findings for different dimensions of discrimination warrant further examination. For instance, a multimethod approach that uses daily diaries via ecological momentary assessment and retrospective self-report measured longitudinally overall multiple time points to assess various dimensions of discrimination and their related burden would provide rich insight into whether and how specific discriminatory experiences compound one another, especially when related to other social statuses. Future work aimed at disentangling discrimination in relation to various social identities may hold promise in forwarding understanding of differential linkages to CVD risk.

Our findings have several clinical and public health implications. Given the propensity for CVD in African Americans, these findings suggest that implementing assessments to capture a broadened definition of self-reported discrimination alongside depressive symptoms at routine health visits would be beneficial, with the hope that optimizing management of these stressors would indirectly improve cardiovascular health vis à vis psychological, behavioral, and physiological pathways. Periodic IMT testing to assess the progression of arterial narrowing and the presence of plaques in African Americans and other populations with an established greater burden of CVD risk and social stressors may also be a consideration.

Although individual-level interventions may provide some support, the challenges highlighted by the current findings and the related mounting body of literature (9,12,14,69,90,100–103) demonstrate that a multilevel response is required. Such a response must target the various systemic and long-standing sources of social inequity, which sustain health disparities. For instance, local state and city policies to ameliorate residential segregation and related neighborhood economic disenfranchisement, race-based police stops, and inequity in economic and resource distribution across school districts would be realistic and key targets for change. In sum, the accumulating evidence documenting individual-and structural-level discrimination and racism as critical bases for the protracted social, economic, and health inequities African Americans endure should serve as a catalyst for recognizing discrimination and racism as social determinants of health and in turn greater investment in their reduction to promote equity.

We did not observe independent associations of discrimination or depressive symptoms with IMT, which is inconsistent with some existing data (4,51). However, this may not be completely surprising given the biopsychosocial model (10) and other conceptualizations of mechanistic pathways linking psychosocial factors to biological processes and subsequent disease (104), which emphasize the complex interplay among multiple variables. Thus, a single level of analysis focused on any one specific psychological or social factor may constrain observation of existing linkages.

This study had some limitations. The use of a dichotomous SES variable may not have captured the full range of socioeconomic variability among the sample. However, HANDLS investigators based their initial area probability recruitment on a division of household income at 125% of the 2004 federal poverty level, with the goal of recruiting sufficient numbers of participants from both low-and moderate-income levels. In addition, HANDLS investigators could not add additional measures of SES, given that many HANDLS participants were unable to accurately estimate their annual incomes or overall wealth, and were used only sporadically. As a result, our most comprehensive estimate of individual-level SES depends on the investigators' initial ascertainment of poverty status and self-reported high school or greater educational attainment. Of note, methods of SES measurement vary widely in epidemiologic research owing to several study-specific factors, such as social groups and outcomes of interest and feasibility of measurement (105).

In addition, because the observed associations were cross-sectional, temporal associations cannot be established. It is important to determine whether the interactive association between discrimination and depressive symptoms predicts increases in IMT over time. Replication in other samples of African Americans in various regions across the United States is recommended, as geographic variations in experiences with discrimination (106) and CVD, including stroke (107), are well documented among African Americans. Examination of these linkages in other racial minority groups is also recommended before clinical application of these findings. Strong evidence demonstrates that reports of depressive symptoms are fairly stable over time (108). However, assessment of discrimination and depressive symptoms across similar time frames may be helpful as well. Also, similar to prior reports our findings suggest that depressive symptoms and discrimination are two distinct constructs (58–61). However, it is plausible that depressive symptoms may influence an individual's recall and interpretation of interpersonal interactions. Cigarette smoking was assessed using a dichotomous variable, which is unlikely to fully capture variability in lifetime cigarette smoking. However, asking about ever versus never smoking is a standard epidemiologic item used in nationally representative studies (e.g., Ref. (109)). Finally, use of a single sonographer is a limitation, as we could not estimate reliability with interrater agreement. In addition, the sonographer was not blind to participants' race or sex, which may have introduced bias. However, use of a single sonographer increases confidence that consistent procedures were followed within and between participants, and therefore may also be viewed as a strength. Sonography was also reviewed by a board-certified cardiologist to ensure validity.

The primary strength of this investigation involves its assessment of the interactive relation between self-reported interpersonal discrimination, a chronic social stressor for racial minorities, and depressive symptoms, an established CVD risk factor, in relation to carotid IMT. Concurrent assessment of multiple dimensions of discrimination is especially important, as it allows for greater precision in identifying the aspects of this chronic stressor that may be particularly pernicious with regard to atherosclerosis. Finally, we focused on these linkages in a sample of middle-aged and older African Americans, a group at increased risk for thicker IMT and subsequent cardiovascular events.



## SUMMARY

In this urban, community-dwelling, socioeconomically diverse cohort of adults, African Americans who reported both higher levels of discrimination across several dimensions and greater depressive symptoms demonstrated the greatest carotid IMT. Discrimination and depressive symptoms were not associated with carotid IMT in whites. These findings suggest that singular consideration of social and psychological factors may obscure optimal conceptualization of subclinical vascular risk among racial minorities, as well as clinical end points, such as stroke. In the clinical setting, interventions focused on alleviating depressive symptoms among individuals who are experiencing discrimination may benefit from addressing the inequity of their social experiences. Ultimately, consideration of psychosocial risk factors central to the experience of African Americans may be warranted to intervene on their greater risk for atherosclerosis and stroke.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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## Glossary

<b>BMI</b>	body mass index
<b>CES-D</b>	Center for Epidemiologic Studies Depression scale
<b>HANDLS</b>	Healthy Aging in Neighborhoods of Diversity across the Life Span
<b>IMT</b>	intimal-medial thickness
<b>MRV</b>	medical research vehicle
<b>SES</b>	socioeconomic status

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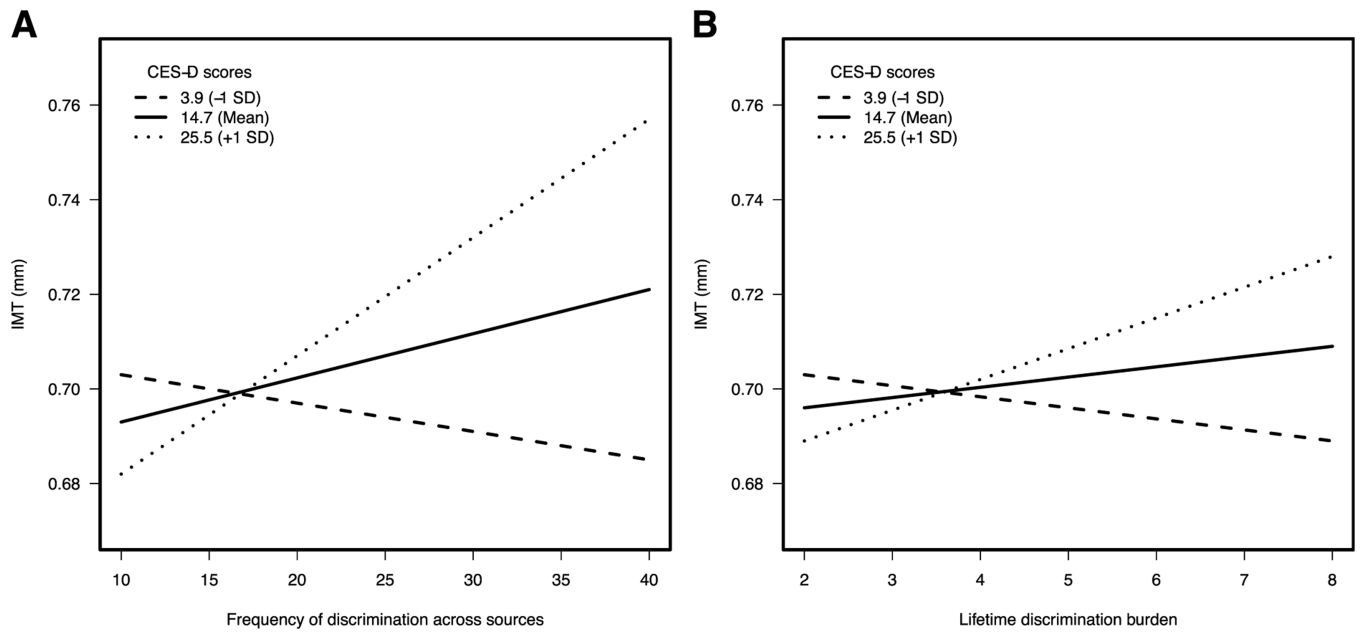
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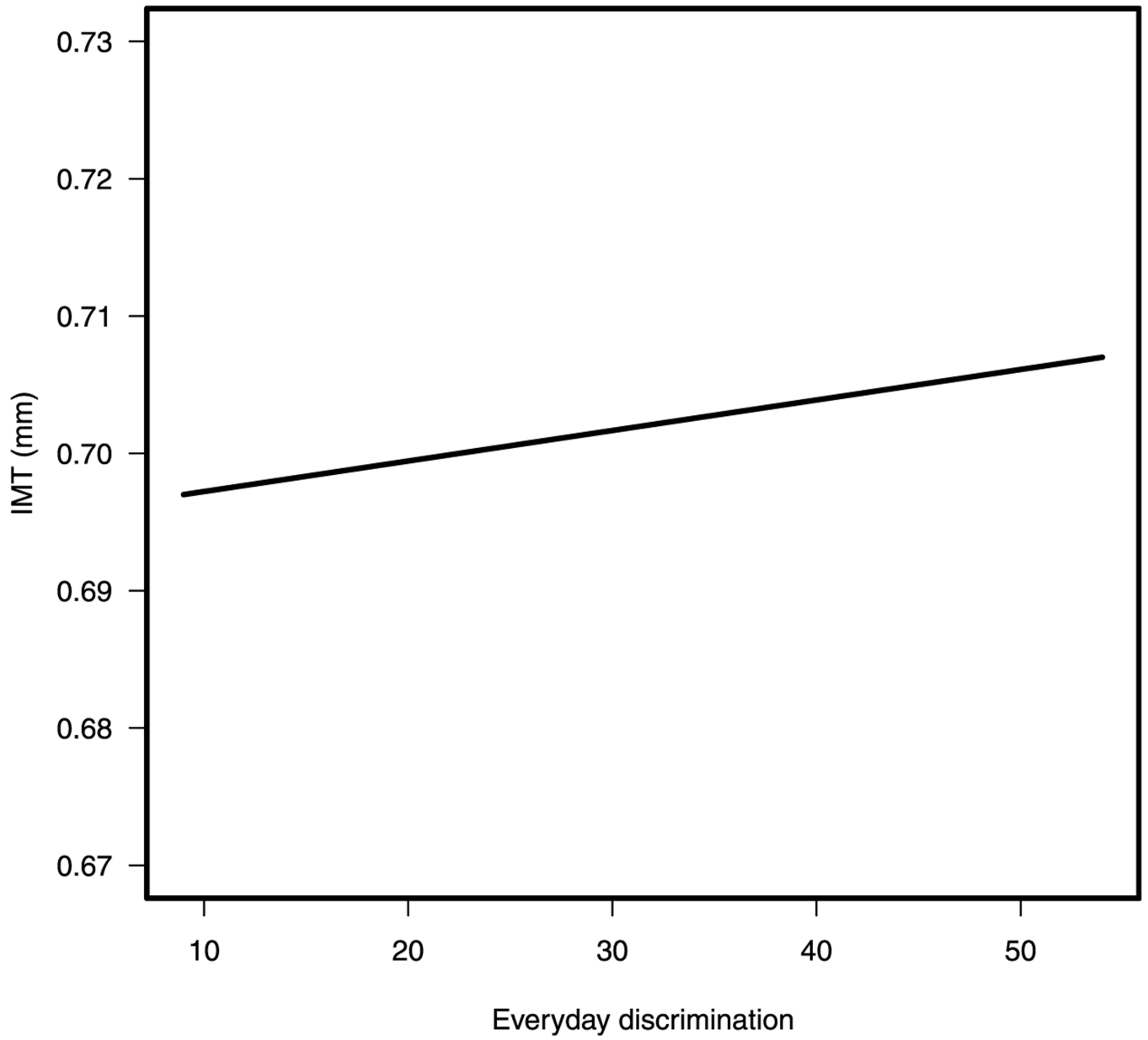
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**FIGURE 1.**

Significant moderating effect of depressive symptoms on the association between carotid IMT and frequency of discrimination across sources (A) and lifetime discrimination burden among African American participants (B). \*  $p < .05$ . IMT = intimal-medial thickness; CES-D = Center for Epidemiologic Studies Depression scale.





**FIGURE 2.** Significant main effect of everyday discrimination with carotid IMT among African Americans. IMT = intimal-medial thickness.

TABLE 1.

## Participant Characteristics Stratified by Race

Variable	AA (n = 1097)	Whites (n = 844)	Sig.
Female, n (%)	571 (52.1)	479 (56.8)	*
Lower SES, n (%) <sup>d</sup>	654 (59.6)	403 (47.7)	***
<High school education/GED, n (%)	350 (31.9)	265 (31.4)	***
Below 125% poverty level, n (%)	521 (47.5)	264 (31.3)	***
Age, y	46.67 ± 9.13	46.97 ± 9.25	
Body mass index, kg/m <sup>2</sup>	28.85 ± 7.13	29.78 ± 7.54	**
Total cholesterol, mg/dl	182.33 ± 38.78	190.26 ± 41.10	***
Hypertension, n (%)	475 (43.3)	305 (36.1)	**
Diabetes mellitus, n (%)	155 (14.1)	105 (12.4)	
1 Cardiovascular disease, n (%) <sup>b</sup>	118 (10.8)	77 (9.1)	
Lipid-lowering medications, n (%)	140 (12.8)	147 (17.4)	**
Ever used cigarettes, n (%) ever used	539 (49.1)	368 (43.6)	*
Ever used alcohol or illicit drugs, n (%) <sup>c</sup>	635 (57.9)	539 (60.3)	
Depression history, n (%)	195 (17.8)	273 (32.3)	***
Everyday discrimination	21.50 ± 8.83	19.90 ± 8.12	***
Frequency of discrimination across sources	17.39 ± 5.94	14.49 ± 4.82	***
Racial discrimination	7.68 ± 1.92	6.41 ± 1.04	***
Lifetime discrimination burden	3.77 ± 1.77	2.85 ± 1.50	***
Depressive symptoms (CES-D scale)	14.71 ± 10.83	14.86 ± 11.42	
Carotid IMT, mm	0.70 ± 0.13	0.67 ± 0.13	***

AA = African Americans; SES = socioeconomic status; GED = General Educational Development; CES-D = Center for Epidemiologic Studies Depression scale; IMT = intimal-medial thickness.

Values are presented as mean ± SD, unless otherwise indicated. Racial differences were examined with independent-samples *t* tests and  $\chi^2$  tests of independence.

\*  $p < .05$ .

\*\*  $p < .01$ .

\*\*\*  $p < .001$ .

<sup>d</sup> Lower SES reflects an annual household income (adjusted for household size) <125% of the 2004 Health and Human Services poverty level and/or educational attainment <high school diploma or GED.

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<sup>b</sup> Cardiovascular diseases assessed were coronary artery disease, myocardial infarction, peripheral artery disease, atrial fibrillation, angioplasty, and coronary artery bypass surgery.

<sup>c</sup> Illicit drugs assessed were marijuana, cocaine/crack, and opiates.

**TABLE 2.**

Independent and Interactive Relations of Frequency of Discrimination Across Sources and Depressive Symptoms With Carotid Intima-Medial Thickness Among African Americans ( $n = 1097$ )

Variable	Model 1	Model 2	Model 3
Age	0.004 ***	0.004 ***	<b>0.004</b> ***
Male sex	0.025 **	0.026 **	<b>0.027</b> **
Lower SES	-0.007	-0.007	<b>-0.007</b>
Body mass index	0.003 ***	0.003 ***	<b>0.003</b> ***
Total cholesterol	<0.001	<0.001	<b>&lt;0.001</b>
Hypertension	0.019 *	0.018 *	<b>0.019</b> *
Diabetes mellitus	0.012	0.012	<b>0.013</b>
Cardiovascular disease	0.006	0.006	<b>0.006</b>
Lipid-lowering medication use	0.002	0.002	<b>0.002</b>
Ever used cigarettes	0.006	0.005	<b>0.006</b>
Ever used alcohol and/or illicit drugs	0.014	0.014	<b>0.013</b>
Frequency of discrimination across sources	0.001 *	0.001	<b>-0.001</b>
Depressive symptoms		<0.001	<b>-0.002</b> *
Frequency of discrimination across sources by depressive symptoms			<b>&lt;0.001</b> **

SES = socioeconomic status.

Model 3 (shown in bold) was retained as the final regression model.

\*  $p < .05$ .

\*\*  $p < .01$ .

\*\*\*  $p < .001$ .

**TABLE 3.**

Independent and Interactive Relations of Lifetime Discrimination Burden and Depressive Symptoms With Carotid Intima-Medial Thickness Among African Americans ( $n = 1097$ )

Variable	Model 1	Model 2	Model 3	Sig.
Age	0.004	0.004	<b>0.004</b>	***
Male sex	0.025	0.026	<b>0.027</b>	**
Lower SES	-0.007	-0.008	<b>-0.008</b>	
Body mass index	0.003	0.003	<b>0.003</b>	***
Total cholesterol	<0.001	<0.001	<b>&lt;0.001</b>	
Hypertension	0.01	0.019	<b>0.018</b>	*
Diabetes mellitus	0.014	0.014	<b>0.014</b>	
Cardiovascular disease	0.007	0.007	<b>0.008</b>	
Lipid-lowering medication use	0.002	0.001	<b>0.001</b>	
Ever used cigarettes	0.006	0.006	<b>0.006</b>	
Ever used alcohol and/or illicit drugs	0.014	0.014	<b>0.013</b>	
Lifetime discrimination burden	0.003	0.003	<b>-0.004</b>	
Depressive symptoms		<0.001	<b>&lt;0.001</b>	
Lifetime discrimination burden by depressive symptoms			<b>&lt;0.001</b>	*

SES = socioeconomic status.

Model 3 (shown in bold) was retained as the final regression model.

\*  
 $p < .05$ .

\*\*  
 $p < .01$ .

\*\*\*  
 $p < .001$ .

**TABLE 4.**

Independent and Interactive Relations of Everyday Discrimination and Depressive Symptoms With Carotid Intima-Medial Thickness Among African Americans ( $n = 1097$ )

Variable	Model 1	Model 2	Model 3	Sig.
Age	0.004	<b>0.004</b>	0.004	***
Male sex	0.026	<b>0.026</b>	0.026	**
Lower SES	-0.007	<b>-0.007</b>	-0.007	
Body mass index	0.003	<b>0.003</b>	0.003	***
Total cholesterol	<0.001	<b>&lt;0.001</b>	<0.001	
Hypertension	0.019	<b>0.019</b>	0.019	*
Diabetes mellitus	0.013	<b>0.013</b>	0.013	
Cardiovascular disease	0.007	<b>0.007</b>	0.007	
Lipid-lowering medication use	0.003	<b>0.002</b>	0.002	
Ever used cigarettes	0.006	<b>0.006</b>	0.006	
Ever used alcohol and/or illicit drugs	0.013	<b>0.013</b>	0.013	
Everyday discrimination	0.001	<b>0.001</b>	0.001	*
Depressive symptoms		<b>&lt;0.001</b>	<0.001	
Everyday discrimination by depressive symptoms			<0.001	

SES = socioeconomic status.

Model 2 (shown in bold) was retained as the final regression model.

\*  
 $p < .05$ .

\*\*  
 $p < .01$ .

\*\*\*  
 $p < .001$ .