Racial/Ethnic and Gender Differences in the Association between Self-Reported Experiences of Racial/Ethnic Discrimination and Inflammation in the CARDIA Cohort of 4 US Communities

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Abstract

Inflammation is etiologically implicated in cardiometabolic diseases for which there are known racial/ethnic disparities. Prior studies suggest there may be an association between self-reported experiences of racial/ethnic discrimination and inflammation, particularly C-reactive protein (CRP). It is not known whether that association is influenced by race/ethnicity and gender. In separate hierarchical linear models with time-varying covariates we examined that association among 901 Black women, 614 Black men, 958 White women, and 863 White men in the Coronary Artery Risk Development in Young Adults (CARDIA) study of young adults in four US communities. Self-reported experiences of racial/ethnic discrimination were ascertained in 1992–93 and 2000–01. Inflammation was measured as log-transformed CRP in those years and 2005–06. All analyses were adjusted for blood pressure, plasma total cholesterol, triglycerides, homeostatic model assessment for insulin resistance (HOMA-IR), age, education, and community. Our findings extend prior research by suggesting that, broadly speaking, self-reported experiences of racial/ethnic discrimination are associated with inflammation; however, this association is complex and varies for Black and White women and men. Black women reporting 1 or 2 experiences of discrimination had higher levels of CRP compared to Black women reporting no experiences of discrimination ($\beta = 0.141$, SE = 0.062, $P < 0.05$). This association was not statistically significant among Black women reporting 3 or more experiences of discrimination

and not independent of modifiable risks (smoking and obesity) in the final model. White women reporting 3 or more experiences of discrimination had significantly higher levels of CRP compared to White women reporting no experiences of discrimination independent of modifiable risks in the final model ($\beta = 0.300$, SE = 0.113, $P < 0.01$). The association between self-reported experiences of racial/ethnic discrimination and CRP was not statistically significant among Black and White men reporting 1 or 2 experiences of discrimination. Further research is needed.

Introduction

In the United States, Blacks have disproportionately higher rates compared to Whites of certain cardiometabolic diseases, including hypertension, type 2 diabetes, stroke, and coronary heart disease. Not only is the incidence of these diseases, which contribute to the leading causes of death, elevated among Blacks, but the diseases themselves appear to be more severe in Blacks than in Whites. For example, premature mortality due to cardiovascular disease, as measured by years of potential life lost before 75 years of age, among Blacks is twice that of Whites (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005). Likewise, the age-adjusted diabetes mortality rate among Blacks is slightly more than twice that of Whites ((U.S.), 2006). Although modifiable risks, such as current cigarette smoking, alcohol consumption, and obesity may explain some of these Black-White differences, factors contributing to these racial/ethnic disparities in health have not been elucidated fully. One important determinant of disparities in health that has been proposed is racial/ethnic differences in experiences of racial/ethnic discrimination (Krieger, Rowley, Herman, Avery, & Phillips, 1993).

Racial/ethnic discrimination unfairly limits the opportunities and agency of specific minority groups, especially Blacks and manifests on at least three levels in the United States: institutional, personally mediated, and internalized (Cunningham, Berkman, Gortmaker, Kiefe, Jacobs, Seeman et al., 2011; D. R. Williams & Mohammed, 2009). A growing body of evidence from epidemiologic analyses suggests that Blacks generally experience and report more racial/ethnic discrimination than Whites (Albert, Ravenell, Glynn, Khera, Halevy, & de Lemos, 2008; Hunte & Williams, 2009; Kessler, Mickelson, & Williams, 1999) and that experiences of racial/ethnic discrimination may result in toxic stress that places individuals at greater risk for cardiometabolic diseases (Clark, Anderson, Clark, & Williams, 1999; Krieger et al., 1993; D. R. Williams & Mohammed, 2009).

Several prior studies have shown that self-reported experiences of racial/ethnic discrimination are associated with several biomarkers and modifiable risks of cardiometabolic diseases across the life course, including preterm and low-birthweight deliveries (Mustillo, Krieger, Gunderson, Sidney, McCreath, & Kiefe, 2004), high blood pressure (Brondolo, Rieppi, Kelly, & Gerin, 2003; Krieger, 1990; Krieger & Sidney, 1996), insulin resistance (Chambers, Tull, Fraser, Mutunhu, Sobers, & Niles, 2004), obesity (Chambers et al., 2004; Gee, Ro, Gavin, & Takeuchi, 2008; Hunte & Williams, 2009), alcohol consumption (Borrell, Jacobs, Williams, Pletcher, Houston, & Kiefe, 2007; Yen, Ragland, Greiner, & Fisher, 1999), and current smoking (Bennett, Wolin, Robinson, Fowler, & Edwards, 2005; Borrell et al., 2007). Nonetheless, the results of prior studies have been inconsistent, with various studies reporting linear, curvilinear, or no associations.

Inflammation, as reflected by circulating levels of C-reactive protein (CRP), is hypothesized to be an etiological determinant of cardiometabolic risk or "common soil"(Stern, 1995). Prior studies have shown that an elevated level of the inflammatory biomarker CRP is an independent predictor of both cardiovascular disease (Ridker, Buring, Shih, Matias, & Hennekens, 1998; Ridker, Hennekens, Buring, & Rifai, 2000) and diabetes (Pradhan, Manson, Rifai, Buring, & Ridker, 2001), for which there are known racial/ethnic and gender differences. The results of the two prior studies examining the association between self-reported experiences of racial/ethnic discrimination and inflammation were also mixed. In the first study, no association was observed in stratified analyses of middle-aged Blacks, Whites, and Hispanics (Albert et al., 2008). In the second study among elderly Black women and men, a positive, linear association between self-reported experiences of racial/ethnic discrimination and inflammation was observed (Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010). It remains unknown whether that association is influenced by race/ethnicity and gender.

Prior studies on inflammation have found elevated levels of CRP in Blacks, as compared to Whites (Khera, McGuire, Murphy, Stanek, Das, Vongpatanasin et al., 2005; K. A. Matthews, Sowers, Derby, Stein, Miracle-McMahill, Crawford et al., 2005; Nazmi & Victora, 2007). Moreover, Black women have been found to have higher inflammation marker levels than White women, who have higher levels than Black men, who in turn have higher levels compared to White men (Khera et al., 2005). Because of these well-established racial/ethnic and gender differences in inflammation and related cardiometabolic diseases, further examination of the association between self-reported experiences of racial/ethnic discrimination and inflammation among young Black and White women and men may be enlightening in understanding the etiology of disparities in cardiometabolic diseases.

Another issue is related to the possible mediating mechanisms through which experiences of racial/ethnic discrimination might impact inflammation, including psychosocial, socioeconomic, and modifiable risk factors in different racial/ethnic and gender groups. Other psychosocial and socioeconomic factors, such as chronic stress (Ranjit, Diez-Roux, Shea, Cushman, Seeman, Jackson et al., 2007), unemployment (Janicki-Deverts, Cohen, Matthews, & Cullen, 2008), education and income (Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009), are also associated with elevated levels of CRP and often vary by race/ethnicity and gender, possibly through physiological stress mechanisms and modifiable risks. These factors also deserve further examination.

The purpose of this study was to examine the influence of race/ethnicity and gender on the association between self-reported experiences of racial/ethnic discrimination and inflammation. We hypothesized that there would be a positive, linear association between self-reported experiences of racial/ethnic discrimination and CRP among young Black women and men and that this association would be stronger among Black women. Analyses were adjusted for two psychosocial factors (social desirability and personal control/mastery) and modifiable risks (current smoking, alcohol consumption, and obesity), which are possible mediating mechanisms that could contribute to inflammation among individuals who experience racial/ethnic discrimination.

Methods

Sample

CARDIA is a prospective, multicenter investigation of the natural history of cardiovascular risk development in young adulthood. In 1985–86, 5115 individuals were recruited from four study communities in the United States: Birmingham, Alabama; Chicago, Illinois; Minneapolis, Minnesota; and Oakland, California. A stratified random sampling procedure

was used to achieve balance at each center by gender, race/ethnicity (Black, White), age (18–24 and 25–30 years), and education (high school degree or less, some college or more). The sites institutional review boards approved this study with all procedures followed in accordance with institutional guidelines and informed consent was obtained from each participant. Of eligible participants, 50% enrolled in the study. Additional details about study design, eligibility requirements, and recruitment are available elsewhere (Friedman, Cutter, Donahue, Hughes, Hulley, Jacobs et al., 1988). Participants were reexamined in years 2, 5, 7, 10, 15, and 20. This analysis used data collected in year 7 (1992–93), year 15 (2000–01), and year 20 (2005–06). Follow-up rates at year 7, year 15, and year 20 were 81%, 74%, and 72%, respectively. This analysis was limited to 901 Black women, 614 Black men, 958 White women, and 863 White men.

Inflammation

CRP was measured from blood samples collected at years 7, 15, and 20 using a Behring Nephelometer II (BN II) (Dade Behring, Deerfield, Illinois). CRP levels less than 1mg/L are generally considered normal; levels between 1 and 3mg/L signify moderate risk for cardiometabolic diseases; and levels greater than 3mg/L signify high risk (Pearson, Mensah, Alexander, Anderson, Cannon, Criqui et al., 2003). Since CRP was used explicitly as an indicator of low-grade inflammation, 302 Black women, 277 Black men, 196 White women, and 162 White men with evidence of a possible infection as reflected by CRP concentrations above 10mg/L were not included in the analyses (Sabatine, Morrow, Jablonski, Rice, Warnica, Domanski et al., 2007). CRP is often elevated among participants with an infectious disease. The values of CRP were log transformed because of the skewed distribution of CRP and used in all analyses as a continuous variable.

Self-Reported Experiences of Racial/Ethnic Discrimination

During the examinations in years 7 and 15, participants completed seven-item, situation versions of the Experiences of Discrimination (EOD) index (Cunningham et al., 2011; Krieger, 1990; Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). In the year 7 examination, participants were asked whether they had "ever experienced discrimination, been prevented from doing something or been hassled or made to feel inferior … because of their race or color" in any of seven domains: "at school, getting a job, at work, getting housing, getting medical care, on the street or in a public setting, and from the police or in the courts." However, in the year 15 examination, the phrase "been prevented from doing something" was dropped from the discrimination question, and the domain "from the police or in the courts" was replaced with "at home." Therefore, we examined the EOD as a six-item index. At the year 7 examination, the Cronbach's alpha for(Krieger, 1990) this six-item index was 0.77 (Black women), 0.76 (Black men), .053 (White women), and 0.54 (White men). At the year 15 examination, the Cronbach's alpha was 0.78 (Black women), 0.77 (Black men), 0.60 (White women), and 0.57 (White men). While other versions of the EOD index have been shown to demonstrate construct validity and high internal consistency, it does not perform equally as well for Whites compared to Blacks here and may weaken the findings in the current study (Krieger et al., 2005). Responses to the EOD index were combined to form a 3-level categorical variable pertaining to experiences of racial/ethnic discrimination in 0, 1 or 2, or 3 or more of the specified domains.

Time-Varying Correlates of Inflammation

All time-varying correlates were measured at years 7, 15, and 20. Systolic blood pressure, plasma total cholesterol, and triglyceride measurements were assessed using standardized methods. The glucose and insulin measurements were performed at Linco Research (now Millipore, Inc., Billerica, MA, USA). The concentration of glucose in the stored serum samples was determined with a Cobas Mira Plus chemistry analyzer (Roche Diagnostics,

Indianapolis, IN, USA) and using the hexokinase ultraviolet method. The insulin measurements were performed by using a radioimmunoassay with an overnight, equilibrium-incubation format. The homoeostasis model for insulin resistance (HOMA-IR) was calculated as glucose (mmol/L)×insulin (mU/L)/22·5 (D. R. Matthews, Hosker, Rudenski, Naylor, Treacher, & Turner, 1985).

Current smoking status was assessed by an interviewer-administered questionnaire. Participants were classified as current smokers if they reported presently smoking at least five cigarettes per week almost every week. Alcohol consumption was assessed by separate questions regarding how many drinks of wine, beer, and liquor participants usually consumed in a week. The amount of alcohol consumed was determined by transforming the total number of drinks into milliliters of alcohol consumed in a typical week. BMI was used as a measure of obesity and evaluated using a standardized protocol for measuring height and weight. Height measurements were rounded to the nearest half centimeter. Weight was rounded to the nearest half pound.

Time-Fixed Correlates of Inflammation

Age, gender, and race/ethnicity were measured at baseline by an interviewer-administered questionnaire. The center was defined as one of the four study communities. Self-reported education was measured during the year 7 examination and assessed as a dichotomous variable: (1) high school degree or less; or (2) some college or more. Prior research suggests that psychosocial factors, such as social desirability and personal control/mastery, are associated with biomarkers of cardiometabolic risk and are potential confounders(Levesque, Bureau, Moskowitz, Tardif, Lavoie, Dupuis et al., 2009; Roepke & Grant, 2011; Rutledge & Linden, 2000; Seeman, 1991). Social desirability refers to a respondent's tendency to respond in a manner that aligns with expected social norms, although responses may not be true (Crowne & Marlowe, 1960). It was measured with a 20-item version of the Crowne-Marlowe Social Desirability Scale, a commonly used assessment of social desirability. The scale was analyzed by counting the number of responses reported as true. The Cronbach's alpha for this scale was 0.72 (Black women), 0.72 (Black men), 0.72 (White women), and 0.76 (White men). Personal control/mastery was assessed using a seven-item version of Pearlin and Schooler's (1978) Personal Mastery Scale, which evaluates feelings of personal control over life circumstances and outcomes versus feelings of helplessness. The Cronbach's alpha for this scale was 0.76 (Black women), 0.78 (Black men), 0.82 (White women), and 0.81 (White men).

Statistical Analyses

We estimated parameters for the association between self-reported experiences of racial/ethnic discrimination and the dependent variable CRP using hierarchical linear modeling (HLM) with PROC MIXED in SAS 9.2. Of note, HLM permits the modeling of time-varying and time-fixed correlates of inflammation over repeated measurement occasions. As multiple observations at different times are viewed as nested within the individual, each model has two levels: a within-subject level that specifies individual time paths, and a between subject level that considers whether group membership (e.g. category of self-reported experiences of racial/ethnic discrimination) accounts for differences in within-group intercepts. After examining variance in individual-level intercepts and slopes, a conditional model predicts intercept and slope terms using group as a predictor variable. These models allow control for potential confounding variables and can accommodate missing values of the dependent variable. The covariance structure chosen for these models was an autoregressive structure, which recognizes that more proximate observations are more correlated in comparison to more distant observations. We were primarily interested in the between-group effects and therefore present only the data for fixed effects.

Model 1 used self-reported experiences of racial/ethnic discrimination to predict log-transformed CRP, adjusting for systolic blood pressure, plasma total cholesterol, triglycerides, HOMA-IR, age, education, and center. Model 2 included the variables adjusted for in Model 1, and the psychosocial factors of social desirability and personal control/mastery. Model 3 included the variables adjusted for in Model 2, and the following modifiable risks: current smoking, alcohol consumption, and BMI.

Results

Table 1 shows the distribution of the biomarker of inflammation, time-varying correlates, and time-fixed correlates among Black women and men participants. Most Black women and men reported experiencing racial/ethnic discrimination, with 74.5 percent (Black women) and 80.5 (Black men) reporting any discrimination at year 7 and 72.6 percent (Black women) and 77.0 (Black men) reporting any discrimination at year 15. Table 2 shows the distribution of the biomarker of inflammation, time-varying correlates, and time-fixed correlates among White women and men participants. Racial/ethnic discrimination was considerably less frequent among White women and men, but not insubstantial, with 30.4 percent (White women) and 28.4 (White men) reporting any discrimination at year 7 and 23.4 percent (White women) and 23.4 (White men) reporting any discrimination at year 15. For all groups, current smoking rates decreased over time, but systolic blood pressure, plasma total cholesterol, triglycerides, HOMA-IR, and BMI increased.

Black Women

Table 3 presents results for Black women. Model 1 suggested a curvilinear association between self-reported experiences of racial/ethnic discrimination and CRP among Black women. Black women reporting 3 or more experiences of discrimination had significantly lower levels of CRP compared to women reporting no experiences of discrimination ($\beta = -0.119$, SE = 0.061, $P < 0.05$). In contrast, Black women reporting 1 or 2 experiences of discrimination had higher levels of CRP compared to those women reporting no experiences of discrimination ($\beta = 0.118$, SE = 0.059, $P < 0.05$). In model 2, which adjusted for social desirability and personal control/mastery, Black women reporting 1 or 2 experiences of discrimination had higher levels of CRP compared to Black women reporting no experiences of discrimination ($\beta = 0.141$, SE = 0.062, $P < 0.05$). Social desirability was also statistically significant for Black women ($\beta = 0.035$, SE = 0.010, $P < 0.001$). In model 3, which also adjusted for current smoking, alcohol consumption, and BMI, the association between self-reported experiences of racial/ethnic discrimination and CRP was not statistically significant. Current smoking ($\beta = 0.169$, SE = 0.057, $P < 0.01$) and BMI ($\beta = 0.084$, SE = 3.74E-03, $P < 0.001$) were statistically significant for Black women.

Black Men

Table 3 presents results for Black men. Model 1 suggested a negative, linear association between self-reported experiences of racial/ethnic discrimination and CRP among Black men. Black men reporting 3 or more experiences of discrimination also had lower levels of CRP compared to those reporting no experiences of discrimination ($\beta = -0.130$, SE = 0.066, $P < 0.05$). The association between self-reported experiences of racial/ethnic discrimination and CRP was not statistically significant in models 2 and 3. Current smoking ($\beta = 0.360$, SE = 0.059, $P < 0.001$) and BMI ($\beta = 0.078$, SE = 5.16E-03, $P < 0.001$) were statistically significant for Black men.

White Women

Table 4 presents results for White women. Model 1 suggested a positive, linear association between self-reported experiences of racial/ethnic discrimination and CRP among White

women. White women reporting 3 or more experiences of discrimination had significantly higher levels of CRP compared to women reporting no experiences of discrimination (β = 0.244, SE = 0.118, P < 0.05). In model 2, this association remained statistically significant for White women reporting 3 or more experiences of discrimination (β = 0.303, SE = 0.124, P < 0.05). In model 3, this association remained statistically significant for White women reporting 3 or more experiences of discrimination (β = 0.300, SE = 0.113, P < 0.01). BMI was also statistically significant for White women (β = 0.096, SE = 4.26E-03, P < 0.001).

White Men

Table 4 presents results for White men. Model 1 suggested no association between self-reported experiences of racial/ethnic discrimination and CRP among White men. Self-reported racial/ethnic discrimination was not statistically significant in any of the models. Personal control/mastery was statically significant for White men (β = −0.018, SE = 6.82E-03, P < 0.01). Current smoking (β = 0.290, SE = 0.058, P < 0.001) and BMI (β = 0.058, SE = 4.42E -03, P < 0.001) were also statistically significant for White men.

Discussion

The results of our epidemiologic analyses did not support our hypothesis. There was not a positive, linear association between self-reported experiences of racial/ethnic discrimination and CRP among Black women and men. Instead, we observed a curvilinear association between self-reported racial/ethnic discrimination and CRP among Black women and a negative, linear association among Black men. These associations between self-reported experiences of racial/ethnic discrimination and CRP were not independent of modifiable risks. Additionally, we observed a positive, linear association among White women and no association among White men.

An explanation for the racial/ethnic and gender differences in the observed associations is not readily available. Our results differ from prior studies examining the association between self-reported experiences of racial/ethnic discrimination and inflammation. In contrast to the curvilinear association among Black women, the negative, linear association among Black men, and the positive, linear association among White women we observed, Albert and colleagues (2008) did not find an association among middle-aged Black, White, and Hispanic women and men in the Dallas Heart Study, and Lewis and colleagues (2010) found a positive, linear association in among elderly Black women and men in the Minority Aging Research Study. Other studies have shown curvilinear associations (Krieger & Sidney, 1996; Ryan, Gee, & Laflamme, 2006), positive, linear associations (Din-Dzietham, Nembhard, Collins, & Davis, 2004; Mustillo et al., 2004), or no association (Albert et al., 2008; Broman, 1996) between self-reported experiences of racial/ethnic discrimination and biomarkers of cardiometabolic risk. One potential reason for the mixed results in the literature is differences in the psychometric instruments used to measure self-reported experiences of racial/ethnic discrimination (Paradies, 2006; D. R. Williams & Mohammed, 2009).

As well, other studies have also observed heterogeneity in the association between self-reported experiences of racial/ethnic discrimination and health by race/ethnicity (Cooper, Mills, Bardwell, Ziegler, & Dimsdale, 2009; Hunte & Williams, 2009) and gender (Banks, Kohn-Wood, & Spencer, 2006). For example, Banks and colleagues (2006) observed that gender moderated the relation between racial/ethnic discrimination and symptoms of anxiety. They asserted that Black men may have been buffered from experiencing symptoms of anxiety associated with racial/ethnic discrimination due to gender differences in coping responses. The former assertion may be relevant to our findings and also may explain to some degree why in this study Black men appear to respond differently with respect to the

effect of self-reported experiences of racial/ethnic discrimination on inflammation compared to Black women.

Although our results show that the highest levels of CRP are among Black women and men reporting no experiences of discrimination compared to those women and men reporting 3 or more experiences of discrimination, it is unlikely that discrimination is beneficial to health of Black women and men. Three possible explanations for these contradictory findings are denial of experiences of discrimination, internalized oppression, and variation in socioeconomic factors, such as education. One explanation for our findings relates to denial, whereby people refuse to see themselves as a target of discrimination. Despite the fact that the denial of experiences of racial/ethnic discrimination may bring some benefits, prior studies have observed that there may also be negative health consequences (Moghaddam, Taylor, Ditto, Jacobs, & Bianchi, 2002; Nyklicek, Vingerhoets, Van Heck, & Van Limpt, 1998). Another reasonable explanation for our findings is internalized oppression. Krieger and Sidney (1996), as well as others (Peters, 2004; Ryan et al., 2006), have argued that some people who experience discrimination may not report it due to internalized oppression, whereby members of an oppressed group accept the notion that they are powerless and inferior. In this current study, the higher levels of CRP among Black women and men reporting no experiences of racial/ethnic discrimination compared to those reporting 3 or more experiences of discrimination might reflect denial or internalized.

Our inclusion of social desirability and personal control/mastery were avenues by which we could consider aspects of denial and internalized oppression, respectively, which were available in the CARDIA study. Therefore, we examined whether the association might be related to social desirability and personal control/mastery. We observed gender differences in the effect of social desirability, which had statistically significant effect for Black women only. Prior research indicates that threatening and anxiety-arousing questions about behaviors or experiences that are not usually discussed openly often lead respondents to answer in a more socially desired manner (Bradburn, Sudman, Blair, & Stocking, 1978). The addition of social desirability magnified the effect of reporting 1 or 2 experiences of discrimination on CRP and reduced the effect of reporting 3 or more experiences of discrimination among Black women. The addition of personal control/mastery, however, did not have an effect among Black women or men. Although the denial of experiences of racial/ethnic discrimination and internalized oppression may be relevant to explaining why we found elevated levels of CRP, neither of these psychosocial factors helped bring clarity to this paradox of our findings.

Since prior studies have observed variation in self-reported experiences of racial/ethnic discrimination by socioeconomic factors, we considered possible effect modification by education as a third potential explanation for our findings among Black women and men (Borrell et al., 2007; Borrell, Kiefe, Williams, Diez-Roux, & Gordon-Larsen, 2006; Krieger & Sidney, 1996; Yen et al., 1999). Both stratified analysis and formal tests for interactions were conducted among Black women and men to assess this possibility (data not shown). These analyses did not provide evidence of effect modification by education on the association.

The associations observed between self-reported experiences of racial/ethnic discrimination and CRP among Black women and men were not independent of modifiable risks. It appears that these modifiable risks —current smoking and obesity—may be mediating mechanisms. Though we did not conduct formal tests of mediation, our analyses and prior studies suggest that self-reported experiences of racial/ethnic discrimination may be associated with both current smoking (Bennett et al., 2005; Borrell et al., 2007) and obesity (Chambers et al., 2004; Gee et al., 2008; Hunte & Williams, 2009). Sequentially, these modifiable risks may

influence inflammation. These possible mediating mechanisms warrant further investigation in future studies.

Among the methodological strengths of our study is the utilization of the EOD index. Versions of the EOD index have been psychometrically validated for population-based studies and demonstrate evidence of good construct validity, high internal consistency reliability, and test-retest reliability (Cunningham et al., 2011; Krieger et al., 2005). Another strength is our use of HLM, which has the capacity to incorporate repeated of measures nested within individuals and handle missing data flexibly using likelihood-based estimation. Some limitations warrant consideration. The Cronbach's alphas for the EOD index for White women and men were low and may weaken the findings. However, this is not unexpected given the lower prevalence of self-reported experiences of racial/ethnic discrimination across the various items of the EOD index for Whites. Lastly, although reciprocal causation is unlikely, we did not establish temporal precedence because self-reported experiences of racial/ethnic discrimination was modeled as a time-varying predictor and collected contemporaneously with the outcome over repeated measurement occasions (Singer & Willett, 2003).

What is most intriguing about our observations are the higher levels of inflammation among Black women and men reporting no experiences of discrimination compared to those reporting 3 or more experiences and the positive, linear association among White women. A precise explanation for this is currently unavailable. To better understand the role of race/ethnicity, gender, and experiences of racial/ethnic discrimination in the etiology of disparities in cardiometabolic diseases, more research is needed with other population-based studies, including Blacks, Whites, and other racial/ethnic groups. It may be beneficial for future work on biomarkers of cardiometabolic risk to consider aspects of experiences of racial/ethnic discrimination not captured by the version of the EOD index used in our analyses, to include the use of the frequency version of the EOD index (Krieger et al., 2005), other commonly used psychometric instruments for self-reported racial/ethnic discrimination in population-based studies, such as the Williams Major and Everyday discrimination measures (D.R. Williams, Yu, Jackson, & Anderson, 1997), or more novel analytical approaches, such as inclusion of implicit measures of discrimination (Krieger, Carney, Lancaster, Waterman, Kosheleva, & Banaji, 2010; Krieger, Waterman, Kosheleva, Chen, Carney, Smith et al., 2011).

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Highlights

- Experiences of racial/ethnic discrimination may result in toxic stress that places people at greater risk for cardiometabolic diseases.

- We examine if race/ethnicity and gender influences the association between racial/ethnic discrimination and inflammation.

- We find that this association varies among Black and White women and men using separate hierarchical linear models.

Table 1

Characteristics of Black Women and Men Participants: The CARDIA Study (1992–2006)

	Black women					
	N	1992–93	N	2000–01	N	2005–06
Biomarker of inflammation						
CRP: mean (SD) (mg/L)	754	2.73 (2.57)	746	2.97 (2.66)	687	2.76 (2.65)
Time-varying correlates of inflammation						
Self-reported racial/ethnic discrimination	901		901			
No experiences of discrimination (%)		25.53		27.41		
1 or 2 experiences of discrimination (%)		29.08		30.63		
3 + experiences of discrimination (%)		45.39		41.95		
Systolic blood pressure: mean (SD)	900	108.70 (12.94)	896	116.14 (16.31)	786	118.25 (16.78)
Total cholesterol: mean (SD)	876	174.76 (32.03)	878	180.03 (32.98)	775	184.25 (33.99)
Triglycerides: mean (SD)	876	71.13 (50.62)	878	83.79 (56.60)	775	91.16 (65.75)
HOMA-IR: mean (SD)	819	3.96 (6.99)	873	3.81 (3.89)	774	4.31 (3.57)
Current smoking status	899		900		779	
Non-smoker (%)		71.41		75.22		78.18
Smoker (%)		28.59		24.78		21.82
Alcohol consumption: mean (SD)	900	1.71 (0.45)	900	1.68 (0.47)	771	1.68 (0.47)
BMI: mean (SD) (kg/m²)	878	29.09 (7.60)	886	31.59 (7.92)	779	32.43 (7.94)
Time-fixed correlates of inflammation						
Age: mean (SD) (years)	901	31.64 (3.84)				
Education	901					
High school graduate or less (%)				39.09		
Some college or more (%)				60.91		
Social desirability: mean (SD)	891			12.70 (3.28)		
Personal control: mean (SD)	900			28.60 (4.45)		

	Black men					
	N	1992–93	N	2000–01	N	2005–06
Biomarker of inflammation						

| | **Black women** | | | | | |
	N	1992–93	N	2000–01	N	2005–06
CRP: mean (SD) (mg/L)	570	1.66 (1.74)	565	2.16 (2.12)	468	2.02 (2.07)
Time-varying correlates of inflammation						
Self-reported racial/ethnic discrimination	614		614			
No experiences of discrimination (%)		19.54		22.96		
1 or 2 experiences of discrimination (%)		32.41		30.78		
3 + experiences of discrimination (%)		48.05		46.25		
Systolic blood pressure: mean (SD)	614	114.98 (12.38)	612	118.38 (15.51)	491	122.15 (13.96)
Total cholesterol: mean (SD)	606	180.52 (36.39)	610	186.26 (40.70)	486	182.08 (37.53)
Triglycerides: mean (SD)	606	89.23 (66.44)	610	107.08 (68.89)	486	112.36 (75.84)
HOMA-IR: mean (SD)	575	3.88 (5.56)	605	3.54 (3.14)	483	4.41 (3.94)
Current smoking status	614		612		489	
Non-smoker (%)		64.66		67.97		71.98
Smoker (%)		35.34		32.03		28.02
Alcohol consumption: mean (SD)	613	1.80 (0.40)	614	1.77 (0.42)	475	1.74 (0.44)
BMI: mean (SD) (kg/m^2)	610	27.21 (5.32)	613	29.07 (6.20)	487	29.97 (6.83)
Time-fixed correlates of inflammation						
Age: mean (SD) (years)	614	31.51 (3.68)				
Education			614			
High school graduate or less (%)				39.09		
Some college or more (%)				60.91		
Social desirability: mean (SD)			612	12.36 (3.34)		
Personal control: mean (SD)			613	29.04 (4.35)		

Table 2

Characteristics of White Women and Men Participants: The CARDIA Study (1992–2006)

	White women					
	N	1992–93	N	2000–01	N	2005–06
Biomarker of inflammation						
CRP: mean (SD) (mg/L)	878	1.76 (2.03)	857	1.97 (2.19)	833	1.88 (2.25)
Time-varying correlates of inflammation						
Self-reported racial/ethnic discrimination	958		958			
No experiences of discrimination (%)		69.62		76.62		
1 or 2 experiences of discrimination (%)		27.04		20.04		
3 + experiences of discrimination (%)		3.34		3.34		
Systolic blood pressure: mean (SD)	957	101.96 (9.54)	956	106.59 (12.16)	873	108.54 (12.11)
Total cholesterol: mean (SD)	945	175.29 (32.49)	944	182.31 (32.10)	864	187.15 (31.88)
Triglycerides: mean (SD)	945	77.27 (56.77)	944	94.12 (61.35)	864	97.82 (61.37)
HOMA-IR: mean (SD)	901	2.55 (1.99)	932	2.52 (2.04)	862	2.95 (3.68)
Current smoking status	956		957		868	
Non-smoker (%)		81.59		84.33		87.56
Smoker (%)		18.41		15.67		12.44
Alcohol consumption: mean (SD)	955	1.87 (0.34)	957	1.86 (0.35)	865	1.86 (0.35)
BMI: mean (SD) (kg/m^2)	925	24.99 (5.86)	940	26.78 (6.78)	871	27.34 (6.97)
Time-fixed correlates of inflammation						
Age: mean (SD) (years)	958	32.63 (3.37)				
Education			958			
High school graduate or less (%)				13.15		
Some college or more (%)				86.85		
Social desirability: mean (SD)			953	10.92 (3.57)		
Personal control: mean (SD)			956	28.79 (4.24)		

	White men					
	N	1992–93	N	2000–01	N	2005–06
Biomarker of inflammation						

	White women						
	N	1992–93	N	2000–01	N	2005–06	
CRP: mean (SD) (mg/L)	829	1.39 (1.62)	805	1.49 (1.65)	771	1.29 (1.52)	
Time-varying correlates of inflammation							
Self-reported racial/ethnic discrimination	863		863				
No experiences of discrimination (%)		71.61		76.59			
1 or 2 experiences of discrimination (%)		25.14		20.28			
3 + experiences of discrimination (%)		3.24		3.13			
Systolic blood pressure: mean (SD)	863	110.96 (10.76)	861	113.47 (12.42)	784	116.41 (11.57)	
Total cholesterol: mean (SD)	860	181.54 (36.19)	857	191.40 (37.79)	781	188.22 (36.92)	
Triglycerides: mean (SD)	860	111.83 (112.75)	857	139.77 (147.36)	781	140.35 (104.37)	
HOMA-IR: mean (SD)	818	3.17 (5.24)	851	3.20 (2.83)	780	3.79 (3.31)	
Current smoking status	863		863		774		
Non-smoker (%)		80.07		83.20		85.66	
Smoker (%)		19.93		16.80		14.34	
Alcohol consumption: mean (SD)	862	1.89 (0.31)	859	1.86 (0.34)	771	1.87 (0.34)	
BMI: mean (SD) (kg/m^2)	862	26.11 (4.14)	861	27.73 (4.74)	784	28.50 (6.15)	
Time-fixed correlates of inflammation							
Age: mean (SD) (years)	863	32.57 (3.30)					
Education			863				
High school graduate or less (%)				15.99			
Some college or more (%)				84.01			
Social desirability: mean (SD)			861	10.73 (3.63)			
Personal control: mean (SD)			862	28.68 (4.03)			

Table 3

Beta Coefficients and Standard Errors for Self-Reported Racial/Ethnic Discrimination as a Predictor of Log CRP, Black Women and Men: The CARDIA Study (1992–2006)

	Black women						Black men					
	Model 1[a]		Model 2[b]		Model 3[c]		Model 1[d]		Model 2[e]		Model 3[f]	
Effect on log (CRP)	b	SE	b	SE	B	SE	b	SE	b	SE	b	SE
Time-varying correlates												
Self-reported racial/ethnic discrimination												
No experiences of discrimination	Reference		Reference		Reference		Reference		Reference		Reference	
1 or 2 experiences of discrimination	0.118*	0.059	0.141*	0.062	0.099	0.056	−0.078	0.066	−0.071	0.069	−0.054	0.065
3 + experiences of discrimination	−0.119*	0.061	−0.056	0.064	−0.053	0.058	−0.130*	0.066	−0.123	0.070	−0.089	0.065
Time	−1.47E-03	3.52E-03	−2.27E-03	3.61E-03	−0.019***	3.44E-03	0.015***	3.67E-03	0.015***	3.75E-03	2.95E-03	3.73E-03
Systolic blood pressure	3.72E-03**	1.46E-03	2.88E-03*	1.50E-03	−2.70E-04	1.38E-03	3.09E-03	1.60E-03	2.96E-03	1.63E-03	−1.60E-04	1.56E-03
Total cholesterol	1.41E-03*	7.34E-04	1.70E-03*	7.63E-04	8.62E-04	7.09E-04	2.19E-03**	6.78E-04	1.82E-03**	7.10E-04	1.48E-03*	6.68E-04
Triglycerides	2.89E-03***	3.92E-04	2.32E-03***	4.01E-04	1.60E-03***	3.69E-04	3.82E-04*	3.37E-04	7.00E-04*	3.55E-04	2.78E-04	3.38E-04
HOMA-IR	0.020***	4.08E-03	0.042***	5.84E-03	0.015**	6.08E-03	0.025***	5.08E-03	0.023***	5.31E-03	5.56E-03	5.20E-03
Current smoking status												
Non-smoker					Reference						Reference	
Smoker					0.169**	0.057					0.360***	0.059
Alcohol consumption (SD)					9.28E-03	0.048					−0.078	0.055
BMI					0.084***	3.74E-03					0.078***	5.16E-03
Time-fixed correlates												
Age	1.77E-03	7.90E-03	−1.05E-03	8.38E-03	−7.06E-03	7.40E-03	8.43E-03	8.14E-03	0.010	8.85E-03	6.37E-03	7.94E-03
Education												
High school graduate or less	Reference		Reference		Reference		Reference		Reference		Reference	
Some college or more	0.067	0.064	2.39E-03	0.073	−0.024	0.065	−0.058	0.063	−0.029	0.071	−0.027	0.065
Social desirability			0.035***	0.010	0.028**	9.04E-03			−6.57E-03	0.010	−0.013	9.38E-03
Personal control/mastery			−4.62E-03	7.42E-03	5.11E-03	6.54E-03			2.27E-03	7.99E-03	−0.006	7.18E-03

*
  $p < 0.05$,

**
  $p < 0.01$,

***
  $p < 0.001$

Note: Model 1 includes experiences of discrimination (for Year 20 the last value was carried forward); time from Year 7; systolic blood pressure, total cholesterol; triglycerides; HOMA-IR; age; education; and center. Model 2 includes all the variables in Model 1, as well as social desirability; and personal control. Model 3 includes all the variables in Model 2, as well as current smoking status; alcohol consumption; and BMI.

[a] 693 observations are excluded because of missing values

[b] 886 observations are excluded because of missing values

[c] 932 observations are excluded because of missing values

[d] 358 observations are excluded because of missing values

[e] 539 observations are excluded because of missing values

[f] 568 observations are excluded because of missing values

Table 4

Beta Coefficients and Standard Errors for Self-Reported Racial/Ethnic Discrimination as a Predictor of Log CRP, White Women and Men: The CARDIA Study (1992–2006)

	White women						White men					
	Model 1[a]		Model 2[b]		Model 3[c]		Model 1[d]		Model 2[e]		Model 3[f]	
Effect on log (CRP)	b	SE	b	SE	b	SE	b	SE	b	SE	b	SE
Time-varying correlates												
Self-reported racial/ethnic discrimination												
No experiences of discrimination	Reference		Reference		Reference		Reference		Reference		Reference	
1 or 2 experiences of discrimination	0.040	0.053	0.049	0.055	0.072	0.051	−0.071	0.051	−0.069	0.053	−0.048	0.051
3 + experiences of discrimination	0.244 *	0.118	0.303 *	0.124	0.300 **	0.113	−0.031	0.118	−0.054	0.122	−0.036	0.117
Time	−0.018 ***	3.32E-03	−0.018 ***	3.38E-03	−0.026 ***	3.15E-03	−8.66E-03 **	3.09E-03	−8.76E-03 **	3.13E-03	−0.016 ***	3.15E-03
Systolic blood pressure	0.012 ***	1.93E-03	0.012 ***	1.98E-03	2.98E-03	1.85E-03	9.44E-03 ***	1.76E-03	9.44E-03 ***	1.79E-03	7.40E-03 ***	1.76E-03
Total cholesterol	2.42E-03 ***	7.20E-04	2.44E-03 ***	7.37E-04	1.16E-03	6.94E-04	1.95E-03 ***	5.81E-04	1.92E-03 ***	5.94E-04	1.77E-03 *	5.76E-04
Triglycerides	5.65E-03 ***	4.04E-04	5.71E-03 ***	4.19E-04	3.99E-03 ***	3.98E-04	5.67E-04 ***	1.72E-04	5.40E-04 **	1.74E-04	2.82E-04	1.71E-04
HOMA-IR	0.014 **	5.10E-03	0.025 ***	7.72E-03	−2.29E-03	7.42E-03	0.030 ***	5.01E-03	0.030 ***	5.03E-03	0.016 ***	5.04E-03
Current smoking status												
Non-smoker					Reference						Reference	
Smoker					−0.093	0.058					0.290 ***	0.058
Alcohol consumption (SD)					−0.010	0.061					−0.057	6.15E-02
BMI					0.096 ***	4.26E-03					0.058 ***	4.42E-03
Time-fixed correlates												
Age	−0.014	8.14E-03	−0.011	8.46E-03	−0.013	7.47E-03	2.00E-03	7.76E-03	1.26E-03	8.11E-03	4.9E-05	7.48E-03
Education												
High school graduate or less	Reference		Reference		Reference		Reference		Reference		Reference	
Some college or more	−0.029	0.070	0.020	0.088	0.032	0.079	−0.082	0.062	−0.076	0.073	0.032	0.070
Social desirability			3.50E-03	8.35E-03	5.48E-03	7.36E-03			−1.35E-03	7.70E-03	0.01609	5.04E-03
Personal control/mastery			−1.36E-03	7.04E-03	3.51E-04	6.23E-03			−0.018 **	6.82E-03	1.71E-03	7.13E-03 **

*
p < 0.05,

**
p < 0.01,

***
p < 0.001

Note: Model 1 includes experiences of discrimination (for Year 20 the last value was carried forward); time from Year 7; systolic blood pressure, total cholesterol; triglycerides; HOMA-IR; age; education; and center. Model 2 includes all the variables in Model 1, as well as social desirability; and personal control. Model 3 includes all the variables in Model 2, as well as current smoking status; alcohol consumption; and BMI.

[a]
404 observations are excluded because of missing values

[b]
539 observations are excluded because of missing values

[c]
598 observations are excluded because of missing values

[d]
267 observations are excluded because of missing values

[e]
385 observations are excluded because of missing values

[f]
413 observations are excluded because of missing values