2016

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Custom Citation

Peterson, L., Matthews, K. A., Derby, C. A., Bromberger, J. T., and Thurston, R. C. 2016. "The Relationship Between Cumulative Unfair Treatment and Intima Media Thickness and Adventitial Diameter: The Moderating Role of Race in The Study of Women's Health Across the Nation." *Health Psychology* 35 (4): 313-321.

The Relationship Between Cumulative Unfair Treatment and Intima Media Thickness and Adventitial Diameter: The Moderating Role of Race in The Study of Women's Health Across the Nation

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Tables: Table 1.Characteristics of study population (N=1056)

Table 2. Race differences on intima media thickness, adventitial diameter, and cumulative unfair treatment

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Abstract

Objective: Unfair treatment may have a detrimental effect on cardiovascular health. However, little research on chronic health outcomes employs cumulative measures of unfair treatment. We tested whether cumulative unfair treatment was associated with greater subclinical cardiovascular disease in a diverse sample of African American, Caucasian, Chinese, and Hispanic women. We also examined whether this relationship varied by race. **Method:** The Study of Women's Health Across the Nation is a longitudinal study of midlife women. Cumulative unfair treatment was calculated as the average of unfair treatment assessed over 10 years at 6 time points. Subclinical cardiovascular disease, specifically carotid intima media thickness and adventitial diameter, was assessed via carotid ultrasound conducted at study year 12 in 1056 women. We tested whether cumulative unfair treatment was related to subclinical cardiovascular disease via linear regression, controlling for demographic factors including socioeconomic status and cardiovascular risk factors. **Results:** The relation between unfair treatment and subclinical cardiovascular disease significantly varied by race (*p*s < .05), with unfair treatment related to higher intima media thickness (B = .03, SE = .01, *p* = .009) and adventitial diameter (B = .02, SE = .009, *p* = .013) among Caucasian women only. No significant relations between unfair treatment and subclinical cardiovascular disease outcomes were observed for African American, Hispanic, and Chinese women. **Conclusions:** Our findings indicate that cumulative unfair treatment is related to worse subclinical cardiovascular disease among Caucasian women. These findings add to the growing literature showing that Caucasian women's experience of unfair treatment may have detrimental health implications. **Key words:** unfair treatment, discrimination, socioeconomic status, cardiovascular disease, intima media thickness, atherosclerosis, race

Cardiovascular disease (CVD) is the leading killer of women in the United States, accounting for 51% of women's deaths (Go et al., 2013). Overall, mortality rates for CVD are higher among African Americans compared to Caucasians (Gillespie, 2009). The burden of CVD morbidity among women varies by race, with prevalence rates of 36.6% among Caucasian women, 48.9% among African American women, and 30.7% among Mexican American women (Go et al., 2013). It is important to understand the key factors, including psychosocial factors, that contribute to racial and ethnic cardiovascular health disparities among women.

A large literature has established that psychosocial stressors contribute to CVD (Everson-Rose & Lewis, 2005; Steptoe & Kivimäki, 2012), including recent research demonstrating that chronic low socioeconomic status (SES) is associated with subclinical CVD (Thurston et al., 2014). Unfair treatment—which participants have described as inequality, injustice, denial of opportunities, being perceived of as incapable or underserving on the basis of one's identity (e.g., race, gender; Williams et al., 2012)—is a particularly relevant psychosocial stressor when considering racial and ethnic disparities. A wealth of research has demonstrated that unfair treatment is related to negative health outcomes even when accounting for stress and personality traits, such as stable negative affect (Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009; Williams, 2012). African Americans in particular are exposed to race-based unfair treatment due to discrimination and socio-historical oppression (Williams & Mohammed, 2009; Williams, 2012). Research indicates that African Americans experience higher discrimination compared to Caucasians (Borrell et al., 2010; Brown, Matthews, Bromberger, & Chang, 2006; Krieger et al., 2011) and other minority groups (Borrell et al., 2010; Brown et al., 2006). Some data indicate that among African Americans, discrimination is associated with more pronounced cardiovascular stress responses (Clark, 2006; Lepore et al., 2006), blunted nocturnal blood

pressure dipping (Beatty & Matthews, 2009; Brondolo et al., 2008; Dolezsar, McGrath, Herzig, & Miller, 2014; Ituarte, Kamarck, Thompson, & Bacanu, 1999), and hypertension (Dolezsar et al., 2014; Krieger & Sidney, 1996; Sims et al., 2012; c.f., Brown et al., 2006). However, unfair treatment may also impact CVD among majority groups and is understudied among racial and ethnic minorities other than African Americans (Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009; Williams, 2012).

Discrimination is typically measured using questionnaires administered at a single time point. The traditional approaches to assessing unfair treatment are limited in their application to the study of chronic disease because they do not capture persistent exposure to unfair treatment. Chronic, rather than acute, stressors may be better predictors of the diagnosis and progression of chronic health outcomes, particularly for atherosclerosis, which develops over time (Cohen, Kessler, & Gordon, 1997; Thurston et al., 2014). Discrimination researchers have called for a more comprehensive approach toward discrimination assessment, such as using questionnaires administered repeatedly over time, to estimate a *cumulative* measure of discrimination (Smart Richman & Leary, 2009; Stock, Peterson, Gibbons, & Gerrard, 2013; Williams & Mohammed, 2009). For example, research among African American women demonstrated that cumulative, rather than cross-sectional, unfair treatment predicted the presence of coronary artery calcification (Lewis et al., 2006).

CVD develops over the lifespan. Subclinical CVD is detectable prior to clinical events and predictive of these events (Bots & Sutton-Tyrrell, 2012; Stein et al., 2008). CVD is associated with arterial restructuring, including thickening of the lumen-intima medial (IMT) and widening of the media-adventitia, which can be measured noninvasively using ultrasound of the carotid arteries. While IMT is more commonly researched than adventitial diameter, both are well-

validated, reproducible, indicative of vessel remodeling, and predictive of events even among low-risk populations (Bots & Sutton-Tyrrell, 2012; Polak et al., 2011; Stein et al., 2008). Subclinical CVD measures of atherosclerosis are particularly useful for assessing psychosocial influences in racial health disparities because they are subject to less provider bias in detection (Bots & Sutton-Tyrrell, 2012; Stein et al., 2008).

Racial disparities emerge in IMT prior to adulthood. In a sample of adolescents and young adults, African Americans had higher IMT than Caucasians (Kieltyka et al., 2003). Additional research has demonstrated these racial disparities in IMT among midlife (Troxel, Matthews, Bromberger, & Sutton-Tyrrell, 2003) and older women (Manolio et al., 1995). In research with additional ethnic groups, Chinese participants had lower IMT compared to Caucasian, African American, and Hispanic participants (Carnethon et al., 2005), and Caucasian participants had lower adventitial diameter compared to African American and Chinese participants (Polak et al., 2011).

Research has investigated whether unfair treatment is associated with subclinical CVD among multiracial samples in the Study of Women's Health Across the Nation (SWAN), a seven site longitudinal study of aging in midlife women. In a subsample of 225 women at the Pittsburgh site, reports of unfair treatment were related to IMT cross-sectionally among African Americans but not among Caucasians (Troxel et al., 2003). In another subsample of 181 SWAN participants (at the two SWAN sites that measured coronary calcification, an indicator of calcified plaques in the coronary arteries), African American women who reported cumulative unfair treatment assessed at 5 time waves were more likely to have coronary artery calcification; this relationship was not apparent among Caucasian women (Lewis et al., 2006).

Recently six of the seven SWAN sites measured IMT and adventitial diameter and accumulated up to six measures of unfair treatment. In consequence, the present study had the opportunity to examine the relationship between cumulative unfair treatment and subclinical CVD in 1056 older women from a greater variety of racial and ethnic groups than prior SWAN reports. Thus, the current research extends the existing literature in several ways. First, we used a comprehensive measure of cumulative unfair treatment derived over 10 years. Second, we explored the relationship between unfair treatment and subclinical CVD in a multiracial sample, which includes Hispanic and Chinese women in addition to African American and Caucasian women. We examined these relations while controlling for CVD risk factors as well as key demographic factors, such as SES. We predicted higher cumulative unfair treatment would be associated with higher IMT and adventitial diameter among the full sample. Second, we predicted that race would moderate the relationship between cumulative unfair treatment and subclinical CVD outcomes such that the relation between cumulative unfair treatment and subclinical CVD would be strongest among African Americans.

Method

Participants

Data for the present study were drawn from SWAN, an ongoing, multisite, epidemiological study of the menopause transition among a racial/ethnically diverse sample of women (see Matthews et al., 2009). In 1996-97, middle-aged women were recruited from seven sites across the United States. Women were recruited from lists of names or household addresses, and select sites supplemented primary sampling frames to obtain adequate numbers of racial/ethnic minority women. Seventy-three percent of the women selected were contacted and provided information to determine eligibility. Women were eligible at baseline if they were between the

ages of 42 and 52, reported having had a menstrual cycle within the last 3 months, had a uterus, had at least one ovary, and were not pregnant, lactating, using oral contraceptives or hormone therapy. More than half (51%; N = 3302) of eligible women enrolled. SWAN protocols were approved by the institutional review boards at each site, and each participant provided written informed consent.

Women were assessed annually at approximately one year intervals. The present investigation examined unfair treatment assessed at baseline (V0) through Visit 10 in relation to IMT and adventitial diameter data collected via carotid ultrasound at six sites (Boston, Chicago, New Jersey, Pittsburgh, Oakland, Michigan; Visit 12). Of the 1,552 women who underwent carotid measurements, data on IMT and adventitial diameter were successfully obtained for 1507 and 1511 women, respectively. We excluded women with a history of stroke (n = 49), angina (n = 73), myocardial infarction (n = 66), or diabetes (n = 288) prior to their carotid scan. An additional 131 women were not included in final adjusted models due to other missing data (race n = 4; age n = 3; income n = 93; education n = 13; BMI (body mass index) n = 13; diastolic blood pressure n = 11; smoking status n = 3; alcohol use n = 13). Compared to women included in the analyses, women with missing data were more likely to have higher HOMA (homeostatic model assessment) indexes, engage in less physical activity, and have a family income of less than $35,000 a year (*p*s < .05). There were 1,056 women in the final models.

Measures

Cumulative unfair treatment and chronic high unfair treatment. Unfair treatment was assessed at six time points (Visits 0, 1, 2, 3, 7, and 10) using The Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997; mean number of time points = 5.52, SD = .95, range 1 – 6). The Everyday Discrimination Scale is a widely-applied instrument (Williams &

Mohammed, 2009), with well-established convergent and divergent validity (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005; Taylor, Kamarck, & Shiffman, 2004). The scale includes 10 items assessing the frequency of experiencing unfair treatment in a respondent's day-to-day life (e.g., "In your day-to-day life…how often do you receive poorer service than other people in restaurants or stores," 1 = *often* to 4 = *never*) and provides options for indicating the perceived reasons why unfair treatment occurred (e.g., race, gender). Responses were reverse-scored so that higher numbers indicated greater unfair treatment. Reliabilities of scale items at each time point were high (αs > .88) and responses across items were averaged within time points. Reliability of unfair treatment scores across time points was high (α of scores averaged across time points = .91) and moderately stable (ICC = .66). Participants' *cumulative* unfair treatment scores were calculated by averaging their scores across time points (average cumulative unfair treatment score, M = 1.66, SD = .44, range 1 - 3.2). Participants' experiences of *chronic high* unfair treatment were calculated by summing the number of time points participants reported experiencing unfair treatment *often* or *sometimes* on any item divided by the total number of time points the participants completed the unfair treatment scale. This calculation resulted in the *percentage* (expressed in decimals) of time points participants reported experiencing unfair treatment across the study (M = .48, SD = .37, range .00 - 1.00).

Intima Media Thickness and Adventitial Diameter. Carotid ultrasound measures and readings were conducted at each site using a Terason t3000 Ultrasound System (Teratech Corp, Burlington, MA) equipped with a variable frequency 5 to 12 Mhz linear array transducer. Technicians were trained by the University of Pittsburgh Ultrasound Research laboratory and monitored during the study period for reliability. Technicians took two digitized images of the left and right distal common carotid artery. These four images were read using the AMS semi-

automated edge-detection software (Wendelhag, Gustavsson, Suurküla, Berglund, & Wikstrand, 1991). Digitized images for readings were obtained from the near and far wall of the left and right distal common carotid artery, 1 cm proximal to the carotid bulb. Near and far wall common carotid artery IMT measures were obtained by electronically tracing the lumen-intima interface and the media-adventitia interface across a 1-cm segment proximal to the carotid bulb. One measurement was generated for each pixel over the area resulting in approximately 140 measures for each segment. The average values for these measures were recorded for each of the four locations, and the mean of the average readings (in mm) at all four locations was used. Common carotid artery adventitial diameter was measured at the same four locations as the distance from the adventitial-medial interface on the near wall to the medial-adventitial interface on the far wall at end-diastole. Readings were completed by readers at the SWAN Ultrasound Reading Center (Ultrasound Research Laboratory, Department of Epidemiology, University of Pittsburgh). Reproducibility of IMT measures was good to excellent (ICC between sonographers $\geq .77$, between readers $> .90$). The scanning and reading protocols have established reliability (Sutton-Tyrrell et al., 1998) and have been used in numerous studies (e.g., Njoroge, Khoudary, Fried, Barinas-Mitchell, & Sutton-Tyrrell, 2011; Sekikawa et al., 2012; Sutton-Tyrrell et al., 2002).

Covariates. Race/ethnicity was self-identified based on women's responses to an open-ended question from the SWAN screening interview, "How would you describe your primary racial or ethnic group?" Response classifications were adapted from the NHANES III and coded for use as a categorical variable (African American, Caucasian [reference group], Chinese, and Hispanic; Centers for Disease Control and Prevention, 1994). Socioeconomic status (SES) was assessed using education at baseline (categorized as low: $\leq$ high school, medium: some college or vocational school, high: $\geq$ college) and total self-report household income at Visit 12

(categorized based upon the sample distribution as low: ≤ $34,999, medium: $35,000-$74,999, high: ≥ $75,000).

Participants' ages and health behaviors were obtained from self-administered questionnaires and interviews at Visit 12. Health behaviors included smoking status (past/never, current); alcohol use based on weekly servings of beer, wine, liquor, or mixed drinks (categorized as low: < once a month, moderate, high: ≥ 2 times a week); and physical activity based on the Kaiser Physical Activity Survey, originally adapted from the Baecke physical activity questionnaire (Baecke, Burema, & Frijters, 1982; Sternfeld, Ainsworth, & Quesenberry Jr., 1999).

BMI was calculated (kg/m^2) from measured height and weight. Blood pressure was obtained from the average of two seated measurements; given the high correlation between systolic and diastolic blood pressure ($r = .64$, $p < .0001$), only diastolic blood pressure (DBP) was included. Analyses also controlled for reported use of medication during the SWAN study, specifically medication for lowering blood pressure, medication for lowering lipids, insulin, and anticoagulants (considered as separate variables, never versus ever used during the study).

Phlebotomy was performed in the morning following overnight (min 10-hour) fast within 90 days of the annual visit. Blood was separated, frozen (-80°C), and sent on dry ice to the University of Michigan Pathology Laboratory, CLIA-certified and accredited by the College of American Pathologists. Measurements were performed on a Siemens ADVIA 2400 automated chemistry analyzer utilizing Siemens ADVIA chemistry system reagents. Glucose was measured using a two-step enzymatic reaction utilizing hexokinase and glucose-6-phosphate dehydrogenase enzymes. Serum insulin was measured using radioimmunoassay. Insulin sensitivity was calculated using HOMA methodology [(fasting insulin*fasting glucose)/22.5] (Matthews et al., 1985). Lipid fractions were determined from EDTA-treated plasma. Total

cholesterol and triglycerides concentrations were determined by coupled enzymatic methods, HDL isolated based upon the method of Izawa et al. (Izawa, Okada, Matsui, & Horita, 1997) and LDL was measured directly (Okada, Matsui, Ito, Fujiwara, & Inano, 1998).

Data Analyses

IMT, adventitial diameter, triglyceride, and HOMA values were natural log transformed for analyses. Baseline differences between included/excluded participants were tested using chi-squares and independent sample t-tests. Race differences in cumulative unfair treatment and IMT/adventitial diameter were examined with general liner models. Associations between cumulative unfair treatment and IMT/adventitial diameter were estimated using linear regressions. Models were first adjusted for age, race, and site (minimal-adjusted model, Model 1); second for SES indicators (SES-adjusted model, Model 2);, and third for relevant covariates based upon their associations with outcomes at $p < .10$ (full-adjusted model, Model 3).[1] The interactions between cumulative unfair treatment [chronic high unfair treatment] and race/ethnicity were examined by cross product terms included in all models. Data were cleaned in SPSS v20 and analyses were performed in SAS v9.3 using the proc glm command (with class statement) for all main empirical analyses.

Results

Women were on average 59 years old at the time of the ultrasound and endorsed an average cumulative unfair treatment score of 1.66 (SD = .44; see Table 1). Slightly over half of the sample was comprised of non-Hispanic Caucasian women, 26.8% of African American women, and the remainder Chinese or Hispanic women. African American and Chinese women

[1] Several psychological covariates considered for Model 3 were not included because they were not significantly associated with cardiovascular outcome variables (e.g., depression measured via the Center for Epidemiological Studies Depression Scale (Radloff, 1977), anxiety measured via the Generalized Anxiety Disorder-7 scale (Spitzer, Kroenke, Williams, & Löwe, 2006), and negative affect measured via the Positive and Negative Affect Scale (Watson, Clark, & Tellegen, 1988); *p*s > .32).

reported significantly higher cumulative unfair treatment than did Caucasian and Hispanic women ($p < .0001$; see Table 2). The most common reported reason for unfair treatment within each racial/ethnic group was race/ethnicity for African American (83.4% of the African American subsample), Chinese (73.4% of the Chinese subsample), and Hispanic (39.3% of the Hispanic subsample) women, and gender for Caucasian (62.2% of the Caucasian subsample) women. Caucasian and Chinese women had the lowest IMT while Caucasian and Hispanic women had the lowest adventitial diameter ($p < .0001$; see Table 2). IMT and adventitial diameter were correlated with each other ($r = .56$, $p < .0001$).

Cumulative unfair treatment was positively correlated with IMT ($r = .096$, $p = .001$) and adventitial diameter ($r = .12$, $p = .0001$) in bivariate analyses. Unfair treatment was marginally associated with IMT in the minimal-adjusted model (Model 1; B = .01, SE = .008, $p = .09$), significantly associated in the SES-adjusted model (Model 2; B = .02, SE = .009, $p = .03$), but was not significant in the full-adjusted model (Model 3; B = .01, SE = .009, $p = .20$). Similarly, unfair treatment was marginally associated with higher adventitial diameter in the minimal-adjusted model (Model 1; B = .01, SE = .007, $p = 06$), significantly associated in the SES-adjusted model (Model 2; B = .02, SE = .007, $p = .02$), but was no longer significant in the full-adjusted model (Model 3; B = .007, SE = .007, $p = .33$).

We next examined whether the relation between cumulative unfair treatment and subclinical CVD outcomes varied by race/ethnicity. Evidence of a moderating effect of race/ethnicity on the relationship between discrimination and subclinical CVD was apparent for IMT (cumulative unfair treatment by race interaction, ($F(3, 1052) = 2.92$, $p = .03$; Table 3) and for adventitial diameter ($F(3, 1052) = 2.62$, $p = .049$; Table 4). In models stratified by race, higher cumulative unfair treatment was associated with higher IMT among Caucasian women (B

= .03, SE = .01, p = .009), but the association between unfair treatment and IMT was not significant for African American, Hispanic, and Chinese women (ps > .25). A similar pattern emerged for adventitial diameter, as results indicated a significant relationship with cumulative unfair treatment among Caucasian women (B = .02, SE = .009, p = .013), but this relationship failed to emerge among the other races (ps > .28).

Several exploratory analyses were conducted. First, given that we were interested in the chronicity of exposure to unfair treatment, we considered the role of chronically high unfair treatment in relation to outcomes. Analyses were conducted examining whether high chronicity (percentage of times participants rated experiencing high levels of unfair treatment across visits) was related to subclinical CVD. No main effect of high chronicity emerged for IMT or adventitial diameter (ps > .33), but moderate/significant race by high chronicity interactions emerged for IMT (p = .08) and adventitial diameter (p = .02), with follow up analyses demonstrating that high chronicity was associated with higher IMT (B = .03, SE = .01, p = .01) and adventitial diameter (B = .03, SE = .01, p = .006) among Caucasians, but this relationship did not emerge for other races (ps > .24). Second, we were interested in further exploring whether cumulative versus single time-point discrimination predicted subclinical CVD. Analyses were conducted examining whether discrimination assessed at Visit 10 (the most proximal visit to the carotid scan) predicted IMT and adventitial diameter. No main effect of non-cumulative discrimination emerged for IMT or adventitial diameter (ps > .46), and race did not moderate the relationship between non-cumulative discrimination and IMT/adventitial diameter (ps > .13).

Discussion

The present study examined the relationship between cumulative unfair treatment and subclinical CVD in a diverse sample of midlife women. The results failed to confirm our first

hypothesis; there was no significant relationship between cumulative unfair treatment and IMT or adventitial diameter among the aggregate sample when controlling for relevant covariates. Our test of the second hypothesis had unexpected results; although the relationship between cumulative unfair treatment and subclinical CVD was moderated by race, cumulative unfair treatment predicted subclinical CVD among *Caucasian* rather than the other groups of women. Exploratory analyses revealed chronic high exposure to unfair treatment also predicted subclinical CVD among Caucasians, but a single time point measure of unfair treatment failed to predict subclinical CVD outcomes. These results emerged despite African American and Chinese women reporting significantly higher discrimination, and African American women having significantly higher IMT and adventitial diameter.

Cumulative unfair treatment did not predict subclinical CVD for the sample as a whole, and a significant relationship failed to emerge among African Americans in race moderation analyses. The results of the study are surprising given the wealth of research reporting a relationship between perceived discrimination and negative health outcomes (Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009). However, reviews on cardiovascular outcomes specifically suggest that conflicting results may depend on measurement of both the exposure (unfair treatment) and the particular CVD outcome of interest (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011). Null or inverse relationships between discrimination and cardiovascular outcomes are reported in the research (e.g., coronary artery calcification; Everage, Gjelsvik, McGarvey, Linkletter, & Loucks, 2012 and systolic blood pressure; Ryan, Gee, & Laflamme, 2006). Multiple indicators of subclinical CVD are used in research and, while related, are far from analogous. Similarly, discrimination can occur across multiple levels that span the individual to the institution. Brondolo and colleagues note that a more consistent relationship

between interpersonal discrimination and cardiovascular outcomes is reported for ambulatory monitoring of blood pressure, perhaps due to capturing a cardiovascular stress response to day-to-day events. Institutional discrimination (e.g., racial segregation; impoverished environments) may be more associated with clinical CVD outcomes (e.g., hypertension diagnosis), and possibly via a health behavior pathway (e.g., weight; Brondolo et al., 2011). Research on institutional discrimination is an important area for future research.

The unexpected results may also stem from how discrimination is perceived and reported among different groups. Discrimination is commonly conceptualized as an interpretation of the social environment, but the construct bears greater complexity. For example, perceptions of discrimination could function more as a trait variable, which may stem from response to the early social environment. There is a rich discussion in the discrimination literature surrounding the *perceptions* of discrimination, and whether individuals of oppressed groups may be vigilant or minimizing when reporting discrimination (Kaiser & Major, 2002). Minimizing the experience of discrimination can have negative implications for health outcomes, especially among individuals with high internalized racism who report no perceived discrimination (Williams, 2012). Our study contributes to the literature in that it is a large, diverse, well-characterized cohort of women with multiple assessments of discrimination and several well-validated subclinical CVD indicators. Future cardiovascular research requires continued discussion surrounding the conceptualization and operationalization of unfair treatment.

Our second hypothesis, that the relationship between cumulative unfair treatment and subclinical CVD would be strongest among the African American women, was unsupported. Results indicated that race moderated the association between both cumulative and chronicity of high unfair treatment and subclinical CVD, but the relationship was significant for Caucasians

only. Our findings indicating significant associations among Caucasians but not among African Americans are surprising. They add to the growing literature showing that despite reporting less unfair treatment, Caucasian women's experience of unfair treatment has detrimental implications for their health (Pascoe & Smart Richman, 2009; Williams & Mohammed, 2009). Caucasian women in the sample reported gender as the most common reason for unfair treatment. Notably, recent research indicates that experiences of different types of discrimination (e.g., racial, gender) are similarly conceptualized as injustice and regardless of the source contribute to stress among Caucasians as well as African Americans (Williams et al., 2012). However, women may vary in their construal of unfair treatment and subsequent behavioral responses (Smart Richman & Leary, 2009). Although speculative, one possibility is that Caucasian women's less frequent experience of unfair treatment, while beneficial, may result in limited opportunities to develop effective coping resources (e.g., active, prosocial, group identification). Lack of experience of coping with unfair treatment may have particularly deleterious cardiovascular consequences among Caucasian women.

The lack of association observed here between unfair treatment and cardiovascular outcomes among African Americans diverges from other findings, suggesting that the relationship between unfair treatment and subclinical cardiovascular outcomes is more detrimental for African Americans (Beatty & Matthews, 2009; Troxel et al., 2003). One major methodological difference between the present study and former research is that both of the former studies assessed unfair treatment at one time period rather than cumulatively. However, among African Americans in the present study, neither cumulative, high chronicity, nor single time point measurements of unfair treatment were significantly associated with subclinical CVD. There may be an unmeasured variable, which has greater bearing on African American women's health than

unfair treatment (e.g., racism vigilance; Clark, Benkert, & Flack, 2006). African American women in the study reported race as the most common source of unfair treatment, and measuring unfair treatment explicitly attributed to racism in the survey questions may have elicited different results (Shariff-Marco et al., 2011). Given that the present study includes participants who maintained enrollment for over 10 years and were not lost to follow up, an unmeasured variable may be related both to maintaining participation in SWAN and resilience to the effects of discrimination. The African American women may have developed coping strategies to maintain resilience when confronted with unfair treatment and racial discrimination. Future research should measure cumulative unfair treatment, subclinical CVD, as well as sources of resilience relevant for race-based unfair treatment.

The study had several limitations. First, subclinical cardiovascular measures were assessed at one point in the study, and we cannot conclude that discrimination relates to change in IMT or adventitial diameter over time. Future research would benefit from gathering synchronous unfair treatment and subclinical cardiovascular data to allow for examination of changes in discrimination in relation to changes in cardiovascular outcomes and whether any patterns vary by race. Second, the sample included only women and it is unknown if the results would be similar among men. Third, Hispanic women made up a small subsample in the dataset, and this group was comprised of women of different countries of origin and immigration statuses.

This study adds to the literature because it is one of the first research studies to investigate the relationship between unfair treatment and subclinical CVD among a diverse racial and ethnic sample. Additionally, the experience of discrimination was assessed cumulatively over the course of 10 years, providing comprehensive assessment of women's unfair treatment. These results suggest that discrimination is related to subclinical cardiovascular health among women

but that this relationship functions differently depending on women's racial/ethnic background. Unfair treatment was related to higher IMT and adventitial diameter among Caucasian women only, suggesting that Caucasian women's experience of unfair treatment may have implications for their health. These findings suggest the importance of continued investigation as to how and why identity-based unfair treatment affects cardiovascular outcomes and highlight the importance of the moderating impact of women's race and ethnic identity on risk factors for CVD.

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Table 1. Characteristics of study population (N = 1056)

Variable	Descriptive Indicator
Cumulative unfair treatment, Mean (SD)	1.66 (.40)
Age, years, Mean (SD)	59.48 (2.66)
Study Site, n (%)	
Michigan	186 (17.63%)
Boston, MA	161 (15.25%)
Chicago, IL	164 (15.53%)
Oakland, CA	258 (24.43%)
New Jersey	83 (7.86%)
Pittsburgh, PA	204 (19.32%)
Race/ethnicity, n (%)	
Caucasian	580 (54.92%)
African American	283 (26.8%)
Chinese	142 (13.45%)
Hispanic	51 (4.83%)
Education, n (%)	
≤ High school	205 (19.41%)
Some college	324 (30.68%)
≥ College	527 (49.91%)
Income, n (%)	
< $35,000	217 (20.55%)
$35,000 through < $75,000	376 (35.61%)
≥ $75,000	463 (43.84%)
BMI, Kg/m^2, Mean (SD)	29.09 (6.90)
DBP, mmHg, Mean (SD)	74.1 (9.95)
HDL, mg/dL, Mean (SD)	63.9 (16.34)
LDL, mg/dL, Mean (SD)	123.49 (30.30)
Triglycerides, mg/dL, Mean (SD)	109.80 (53.78)

HOMA index, Mean (SD)	2.61 (2.91)
Physical activity score, Mean (SD)	7.87 (1.83)
Current smokers, n (%)	85 (7.86%)
Alcohol consumption, n (%)	
<1/month	513 (48.58%)
Moderate	280 (26.52%)
≥ 2/week	263 (24.91%)
Anticoagulant medication, n (%)	113 (10.7%)
Cholesterol / lipid lowering medication, n (%)	294 (27.84%)
Blood pressure medication, n (%)	384 (36.36%)
Insulin/ pills for sugar, n (%)	12 (1.14%)
Intima media thickness, mm, Mean (SD)	0.78 (.11)
Adventitial diameter, mm, Mean (SD)	7.12 (.64)

Note. All responses derived from Visit 12. Medication use was derived from ever reporting medication use over the course of the study. Triglycerides, HOMA, IMT, adventitial diameter all natural log transformed for analyses due to non-normality; raw values are reported here

Table 2. Means and standard deviations of subclinical cardiovascular disease outcomes and unfair treatment by race/ethnicity

	Overall Test	Caucasian	African American	Chinese	Hispanic
Intima media thickness	$F(3, 1052)= 19.19$, $p<.0001$	.77 (SD = .11)$_a$	.82 (SD = .12)$_b$	.75 (SD = .11)$_{a\ c}$	.79 (SD = .11)$_{a\ b\ c}$
Adventitial diameter	$F(3, 1052)= 12.17$, $p<.0001$	7.04 (SD = .62)$_a$	7.29 (SD = .68)$_b$	7.17 (SD = .58)$_{a\ b\ c}$	6.94 (SD = .59)$_{a\ c}$
Cumulative unfair treatment	$F(3, 1052)= 48.80$, $p<.0001$	1.59 (SD =.37)	1.82 (SD = .42)$_a$	1.78 (SD = .38)$_a$	1.24 (SD = .30)

Note. Subscripts indicate row values which do not significantly differ. Although analyses were conducted with natural log transformed IMT and adventitial diameter, raw values are reported here.

Table 3. Relation between cumulative unfair treatment and intima media thickness, full sample and models stratified by race

	Full sample	Caucasians	African Americans	Chinese	Hispanics
	N= 1056	n= 580	n= 283	n= 142	n= 51
	F(31, 1024)= 6.77**	F(25, 554)= 3.39**	F(23, 259)= 1.19	F(20, 121)= 2.68**	F(20, 30)= 1.60
	R^2= .17	R^2= .15	R^2= .10	R^2= .31	R^2= .51
	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
Discrimination	.01 (.009)	.03 (.01)**	.006 (.02)	-.01 (.02)	-.07 (.06)
Race	F(3, 1052)= 3.26*				
African American	.03 (.01)**				
Chinese	.01 (.01)				
Hispanic	.01 (.02)				
Caucasian	--				
Discrimination by Race	F(3, 1052)= 2.92*				

Note. All models adjusted for site, age, socioeconomic status, BMI, DBP, cholesterol, HOMA, physical activity, smoking, alcohol use, and medication use. Numerator degrees of freedom differ between racial groups because the number of sites controlled for varied across racial groups, the control variables across all models were identical.
†p<.1
*p<.05
**p<.01

Table 4. Relation between cumulative unfair treatment and adventitial diameter, full sample and models stratified by race

	Full sample	Caucasians	African Americans	Chinese	Hispanics
	N= 1056	n= 580	n= 283	n= 142	n= 51
	F(31, 1024)= 6.71**	F(25, 554)= 4.82**	F(23, 259)= 2.33**	F(20, 121)= 1.96*	F(20, 30)= 1.51
	R^2= .17	R^2= .18	R^2= .17	R^2= .24	R^2= .50
	B (SE)	B (SE)	B (SE)	B (SE)	B (SE)
Discrimination	.007 (.007)	.02 (.009)*	-.009 (.01)	-.006 (.02)	-.05 (.04)
Race	F(3, 1052)= 5.35**				
African American	.01 (.007)†				
Chinese	.04 (.01)**				
Hispanic	-.01 (.02)				
Caucasian	--				
Discrimination by Race	F(3, 1052)= 2.62*				

Note. All models adjusted for site, age, socioeconomic status, BMI, DBP, cholesterol, HOMA, physical activity, smoking, alcohol use, and medication use. Numerator degrees of freedom differ between racial groups because the number of sites controlled for varied across racial groups, the control variables across all models were identical.

†p<.1
*p<.05
**p<.01

Funding Sources. SWAN has grant support from the National Institutes of Health (NIH), DHHS, through the National Institute on Aging, the National Institute of Nursing Research and the NIH Office of Research on Women's Health (Grants U01NR004061; U01AG012505, U01AG012535, U01AG012531, U01AG012539, U01AG012546, U01AG012553, U01AG012554, U01AG012495). The content of this manuscript is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute on Aging, National Institute of Nursing Research, Office of Research on Women's Health or the NIH.

Acknowledgements. SWAN Clinical Centers: University of Michigan, Ann Arbor-Siobán Harlow, PI 2011-present, MaryFran Sowers, PI 1994-2011; Massachusetts General Hospital, Boston, MA-Joel Finkelstein, PI 1999-present; Robert Neer, PI 1994-1999; Rush University, Rush University Medical Center, Chicago, IL-Howard Kravitz, PI 2009-present; Lynda Powell, PI 1994-2009; University of California, Davis/Kaiser-Ellen Gold, PI; University of California, Los Angeles-Gail Greendale, PI; Albert Einstein College of Medicine, Bronx, NY-Carol Derby, PI 2011- present, Rachel Wildman, PI 2010-2011; Nanette Santoro, PI 2004-2010; University of Medicine and Dentistry-New Jersey Medical School, Newark-Gerson Weiss, PI 1994-2004; and the University of Pittsburgh, Pittsburgh, PA-Karen Matthews, PI. NIH Program Office: National Institute on Aging, Bethesda, MD-Winifred Rossi 2012-present; Sherry Sherman 1994-2012; Marcia Ory 1994-2001; National Institute of Nursing Research, Bethesda, MD-Program Officers. Central Laboratory: University of Michigan, Ann Arbor-Daniel McConnell (Central Ligand Assay Satellite Services). Coordinating Center: University of Pittsburgh, Pittsburgh, PA – Maria Mori Brooks, PI 2012 - present; Kim Sutton-Tyrrell, PI 2001 – 2012; New England Research Institutes, Watertown, MA - Sonja McKinlay, PI 1995 – 2001. Steering Committee: Susan Johnson, Current Chair; Chris Gallagher, Former Chair Assistance with data analysis: Yue-Fang Chang, Hassen Khan Special thanks: Maryann Peterson