Everyday Discrimination Prospectively Predicts Inflammation Across 7-Years in Racially Diverse Midlife Women: Study of Women's Health Across the Nation

Danielle L. Beatty[1], **Karen A. Matthews**[2,3], **Joyce T. Bromberger**[2,3], and **Charlotte Brown**[2]

[1]University of Maryland, Baltimore County, Baltimore, MD

[2]University of Pittsburgh, Department of Psychiatry, Pittsburgh, PA

[3]University of Pittsburgh, Department of Epidemiology, Pittsburgh, PA

Abstract

Self-reported discrimination has emerged as a predictor of negative psychological and physical health outcomes across racial/ethnic groups. The goals of this study were to determine whether C-reactive protein (CRP), a marker of inflammation and risk factor for future cardiovascular disease (CVD) was independently predicted by everyday discrimination or whether race or body mass index (BMI) modified this association over a 7-year period among 2,490 women from racially diverse backgrounds. At baseline, the 10-item Williams' measure of everyday discrimination was administered. Generalized estimating equations were used to assess these associations. Descriptive results showed that Black and Chinese women reported greater discrimination than White, Japanese, and Hispanic women, while Black and Hispanic women had the highest levels of CRP over the 7-year period. There was no main effect of everyday discrimination ($B = .003$, $SE = .005$, $p = .58$) and this association did not differ as a function of race (p's $> .05$). The everyday discrimination $\times$ BMI interaction term significantly predicted higher CRP levels over time in the full sample of women ($p = .03$). Specifically, in non-obese women (BMI less than 30), higher perceived everyday discrimination was associated with higher CRP levels over the 7-year period. These findings were independent of demographic, negative affect, biomedical, and behavioral factors. The results demonstrate that greater everyday discrimination is associated with increased inflammation over time in non-obese women. These findings highlight the implications of interpersonal sources of social stress for long-term physical health via their impact on intermediary biological pathways, specifically inflammation. Greater emphasis on such linkages is warranted as we work towards ameliorating health disparities exacerbated by individual-level factors.

Keywords

everyday discrimination; C-reactive protein; cardiovascular diseases; inflammation

Correspondence concerning this article should be addressed to Danielle L. Beatty, Ph.D., Assistant Professor, Department of Psychology, University of Maryland, Baltimore County. dlbeatty@umbc.edu, phone, 410-455-2426 mailing address, University of Maryland, Baltimore County 1000 Hilltop Circle, Math/Psychology Building, Office 328, Baltimore, MD 21250.

Introduction

Self-reported discrimination is associated with an increased risk for cardiovascular disease (CVD) and all-cause mortality across racial/ethnic groups (Barnes et al., 2008). Heart disease is the leading cause of death in women (Lloyd-Jones et al., 2010). Black women are at the greatest risk for CVD-related mortality, compared with Whites and other racial groups (Lloyd-Jones et al., 2010). In fact, Black women are at the highest risk for death from heart disease among all racial, ethnic, and gender groups (Roger et al., 2012). While Black women tend to have higher rates of the standard risk factors for CVD, including obesity, hypertension, and diabetes, (Henderson, Bretsky, Henderson, & Stram, 2001; Henderson, Haiman, Wilkens, Kolonel, Wan, & Pike, 2007) adjustment for these differences does not fully account for the disproportionate CVD burden observed in this group. Thus, potential alternative pathways linking discrimination to greater CVD risk should be explored to better understand the greater CVD risk profile of racial minorities, including Black and Hispanic women. A potential alternative pathway that may be implicated in the relation of self-reported discrimination to CVD risk is chronic inflammation. Inflammation has been identified as an important factor in the onset and progression towards subclinical CVD and a precursor to CVD events. C-reactive protein (CRP) is a known marker of chronic systemic inflammation that is linked to acute (Hamer, Gibson, Vuononvirta, Williams, & Steptoe, 2006; Miller, Rohleder, Stetler, & Kirschbaum, 2005; Nijm, Kristenson, Olsson, & Jonasson, 2007) and chronic stressors (Coussons-Read, Okun, & Nettles, 2007; Ranjit, Diez-Roux, Shea, Cushman, Ni et al., 2007; Fulgini et al., 2009; Ranjit, Diez-Roux, Shea, Cushman, Seeman et al., 2007; Shivpuri, Gallo, Crouse, & Allison, 2012) and prospectively predicts CVD in healthy men and women (Ridker, Buring, Cook, & Rifai, 2003). Indeed, in the Women's Health Study, CRP prospectively predicted a 5-fold increase in risk of any vascular event and a 7-fold increase in risk of myocardial infarction or stroke among apparently healthy women (Ridker et al., 1998; Ridker et al., 2003). Importantly, the American Heart Association and Centers for Disease Control and Prevention have identified CRP as a risk marker in monitoring CVD risk, setting cut points of <1 mg/L, 1–3 mg/L, and >3 mg/L to identify low to high individual risk for future CVD events (Pearson et al., 2003). Higher CRP levels are indicative of greater inflammation and thus greater CVD risk.

Chronic stressors are characterized by ongoing, or recurring events that persist over an extended period of time. Examples of chronic stressors include caregiving for elderly parents, job strain, negative interpersonal relationships, and everyday discrimination. Overall, the evidence linking chronic stress to poor health is compelling (Cohen, Janicki-Deverts, & Miller, 2007; Schneiderman, Ironson, & Siegel, 2005; Umberson, Liu, & Reczek, 2008). Specific to CRP, studies examining the impact of chronic stress have reported mixed findings (Davis et al., 2008; Marin, Martin, Blackwell, Stetler, & Miller, 2007; McDade, Hawkley, & Cacioppo, 2006; O'Connor et al., 2009). Nevertheless, there is some evidence that chronic stress is linked to CRP across multiple types of stressors. In 6,814 White, Black, Hispanic, and Latino adults participating in the Multi-Ethnic Study of Atherosclerosis (MESA) Ranjit et al. (2007), there was a positive association between chronic stress arising from the poor health of close others, one's employment situation, finances, and relationships and elevated CRP. In another MESA study, which examined the

role of gender, women who reported care giving stress had higher CRP than women who did not report this particular stressor (Shivpuri et al., 2012). Similar findings have been demonstrated when using a psychosocial stress score based on four indicators (lower social class, being divorced or separated from a partner, psychological distress, and less education; Hamer & Stamatakis, 2008) and when summing across daily negative interpersonal interactions in a sample of adolescents (Fulgini et al., 2009).

A central aspect of stress exposure that many individuals face on a daily basis is self-reported, interpersonal discrimination. Whereas discrimination can be broadly defined as biased treatment or behavior, interpersonal discrimination represents those experiences of bias unfolding in one-on-one interactions between individuals. In turn, interpersonal discrimination is typically assessed via one of two constructs; racial discrimination – which explicitly examines bias, associated with race – or everyday discrimination – which broadly examines bias without reference to the specific reason (e.g., race or gender) for the bias. In the current study, we assess everyday discrimination, which captures chronic, typical, but often minor events that characterize day-to-day interpersonal interactions. These can include being treated with less respect, being excluded from social outings, or being verbally threatened (Essed, 1991). Everyday discrimination is posited to be a common experience across all groups and focuses broadly on these experiences without regard for the reason (e.g., phenotypical, demographic, cultural etc.). Mounting evidence demonstrates that everyday discrimination has adverse effects on health among racial minorities and non-minority racial groups, although research continues to demonstrate that these associations are usually more pronounced among racial minorities (Beatty & Matthews, 2009; Brown et al., 2006).

While the linkage of interpersonal discrimination to CVD is steadily being established, attention has turned to elucidating the biological mechanisms underlying this association. Notably, interpersonal discrimination is a specific chronic psychosocial stressor largely investigated within the context of the broader chronic stress-CVD research. In this regard, evidence from the broader chronic psychosocial stress literature points to inflammation as a pathway linking stress to CVD. In the current study, we test the hypothesis that everyday discrimination is prospectively linked to CRP, an inflammatory marker implicated in CVD in women from both minority and non-minority racial backgrounds.

Two studies have examined the association between self-reported discrimination and CRP. In a sample of 1,475 Black, White, and Hispanic adults (30-65 years old) participating in the Dallas Heart Study, Albert et al. (2008) used a single item to assess whether exposure (dichotomous choice response: yes or no) to racial discrimination ("Have you ever been mistreated because of your race/ethnicity) was related to CRP. They found no relation between exposure to racial discrimination and CRP levels in the full sample or when stratified by race. Whether the association of discrimination to CRP was conditional upon race is unclear as an interaction between discrimination and race was not tested. In contrast, Lewis, Aiello, Leurgans, Kelly, and Barnes (2010) reported a positive association between everyday discrimination and CRP in a sample of 296 predominantly female, older Blacks (mean age 73, 70% female). Here, participants were asked to report on the frequency of their

experiences with exclusion and mistreatment across different settings, such as restaurants and while shopping.

Body mass index (BMI) has been independently linked to discrimination and CRP. In a study of 59,000 Black women everyday discrimination was associated with an increased incidence of obesity over a 12-year period (Cozier, Yu, Coogan, Bethea, Rosenberg, & Palmer, 2014). It has been posited that discrimination acting as a chronic stressor results in neuroendocrine dysregulation that in turn influences the accumulation of excess body fat. Notably, the association between CRP and BMI is strong due to adipose tissue acting as an inflammatory site. Indeed, BMI is a known correlate of CRP (Barinas-Mitchell, Cushman, Meilahn, Tracy, & Kuller, 2001; Fransson et al., 2010; Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009; Nguyen, Lane, Smith, & Nguyen, 2009; Rexrode, Pradhan, Manson, Buring, & Ridker, 2003; Visser Bouter, McQuillan, Wener, & Harris, 1999). Thus, BMI may serve as an important pathway in the linkage between discrimination and CRP. Specifically, it is unclear whether the linkage between everyday discrimination and CRP is modified by BMI.

Goals of the Current Study

The current analyses will extend these previous studies of the association between everyday discrimination and CRP in three ways. First, we will examine this relationship prospectively. Using longitudinal measures of CRP will allow us to evaluate whether an inflammatory mechanism is involved in the causal processes underlying the role of self-reported discrimination as a chronic stressor and it's relationship to CVD. Indeed, it is critical to note the long induction period of CVD – it can take decades to produce observable clinical pathology (Ben-Shlomo & Kuh, 2002; Kittleson et al., 2006; Kuh, & Ben-Shlomo, 1997; Power & Hertzman, 1997; Ross, 1993). Thus, examining a link between everyday discrimination and CRP overtime can increase understanding of the impact of discrimination on CVD and the mechanisms that help explain this association. Using SWAN data collected across 7 annual time points in addition to baseline will allow us to assess the long-term influence of everyday discrimination on this endpoint.

Second, conducting these analyses in the Study of Women's Health Across the Nation (SWAN) which includes Black, White, Hispanic, Chinese, and Japanese women will allow us to address the association between everyday discrimination and CRP and whether there are racial differences. Third, we will explore whether BMI moderates the association between everyday discrimination and CRP. To our knowledge, no other studies have assessed these questions in a racially diverse sample, investigated potential pathways, or assessed whether these factors are prospectively linked.

Method

Participants and procedure

SWAN is a longitudinal study designed to examine health throughout midlife aging in a racially diverse sample of women. The study consists of 3,302 women drawn from seven cities across the U.S., where each study site recruited a cohort of approximately 450

participants that included White women and women of one other predetermined racial group. Specifically, by site; Blacks in Pittsburgh, Pennsylvania, Boston, Massachusetts, Chicago, Illinois, and the Detroit metropolitan area, Michigan; Chinese in Oakland, California; Hispanics in Newark, New Jersey; and Japanese in Los Angeles, California. At study entry, participants were between the ages of 42 to 52 years old, self-identified as one of the respective sites designated racial groups or White, reported recent menses (< 3 months prior to enrollment), were either premenopausal or early perimenopausal, and not using hormone replacement therapy (HRT).

SWAN participants were recruited using random digit dialing and registered voters, census data. "Snowballing" was also used to recruit Japanese women. Supplementary details of the SWAN study methodology have been previously reported (Sowers et al., 2000). Data were collected either via self-report measures or by trained SWAN technicians using standardized SWAN protocols. Regardless of the method used, research assistants were available to assist participants with the completion of measures that might be hindered by participants' vision, literacy, or language abilities.

Data used in the current analyses were drawn from the SWAN baseline and the six annual follow up visits – 1 through 7, which took place between 1995 and 2001. At baseline, the 2,490 women included in the current analyses had 1) everyday discrimination and CRP data for baseline and for at least one of the follow up visits, 2) baseline CRP levels < 10 mg/L, and 3) reported being free of diabetes and CVD (as defined by a history of angina, myocardial infarction, or stroke). Although not mutually exclusive, the 812 women not included in the current analyses were excluded due to; 1) missing everyday discrimination data or CRP at baseline (n = 94) or CRP at one follow-up visit or more (n = 321), 2) high CRP levels at baseline (i.e., > 10; n = 294), or 3) evidence of affirmative diabetes or CVD status at baseline (n = 240). The Institutional Review Boards at all participating study sites approved the SWAN research protocol and participants provided the required consents for participation.

Measures

Primary Study Variables

Everyday discrimination: Participants completed an adapted, 10-item version of the Everyday Discrimination Scale (Williams et al., 1999), which utilizes a 4-point scale (1 = never, often = 4) to assess the frequency of experiences with specific forms of interpersonal maltreatment. Each item starts with the following question, "In your day-to-day life have you had the following experiences?" Example items include "You are treated with less courtesy than other people," "People act as if they think you are not smart," and "You receive poorer service than other people at restaurants or stores." The original 9-item measure was modified in the SWAN study protocol to include one additional item, "People ignore you or act as if you are not there." This measure has been included in the mean everyday discrimination score of previously published studies (e.g., Lewis et al., 2006; Lewis, Yang, Jacobs, & Fitchett, 2012). A mean score was calculated for the 10 items, with a higher score indicating more frequent perceived everyday discrimination. Internal test-retest reliability scores for the Everyday Discrimination Scale in the SWAN sample has been

very good ranging from 0.87 to 0.90 across several years (Lewis et al., 2006). Because the everyday discrimination variable has been shown to be relatively stable over time in the SWAN sample (Lewis et al., 2006; Lewis et al., 2012), we only use baseline scores in the current analyses.

C-Reactive Protein: CRP was ascertained and quantified via an ultrasensitive rate immunonepholemetry method (Dade-Behring, Marburg, Germany). Due to financial constraints, CRP was not assayed at follow-up visit 2. Thus, participants had up to seven CRP values across baseline and years 1, 3, 4, 5, 6, and 7 available for analysis. CRP levels > 10 were eliminated from the analyses as such levels indicate that they may be currently ill or have an infection.

Demographic and Biomedical Covariates: All covariates were selected for inclusion based on their established relationship with psychosocial factors, including self-reported discrimination or with inflammatory markers, particularly CRP (Friedman et al., 2009; Friedman & Herd, 2010; Lewis et al., 2010; Slopen et al., 2010). During the baseline examination self-reported race, age, socioeconomic status (SES), and negative emotions were assessed. SES was evaluated using education and financial strain. Educational attainment was coded as greater than or less than a 4-year college degree. Financial strain was assessed via a single question in the SWAN interview, "How hard is it for you to pay for the very basics like food, housing, medical care, and heating?" and coded – 1 = no difficulty paying for basics and 0 = somewhat or very hard paying for basics. The 13-item cynicism subscale of the Cook-Medley Hostility Scale (Barefoot et al., 1989) and the 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) were used to assess negative emotions; with higher scores on both measures indicating greater cynicism (scale range 0-13) and depressive symptomatology (scale range 0-60), respectively. Cynicism was only assessed at baseline in the SWAN study due to findings that this trait is relatively stable in middle to later life (Lewis et al., 2006; Shekelle, Gale, Ostfield, & Paul, 1983) and prior papers on SWAN subsamples have reported strong Cronbach's alpha (e.g., .91, Midei, Matthews, & Bromberger, 2010). Similarly, the CES-D has well-established reliability across epidemiological studies, and strong Cronbach's alpha in prior SWAN studies (e.g., .89; Midei et al., 2010) and other racially diverse samples (Roberts, 1980).

At baseline and across follow-up visits, participants reported their current alcohol consumption and cigarette smoking status. In clinical examinations, systolic and diastolic blood pressure (SBP and DBP), low- and high-density lipoprotein (LDL/HDL), and triglyceride and glucose levels and BMI were assessed. BMI was calculated as weight in kilograms divided by height in meters squared (kg/m2) and was treated as a continuous variable. Physcial activity (i.e., involvement in exercise, sports, household, caregiving, and daily routines) was assessed using an adapted version of the Kaiser Physical Activity Survey (Sternfeld, Ainsworth, & Quesenberry, 1999).

At each follow up visit, we assessed menopausal status as well as new medication use and disease onset. Menopausal status was based on menstrual bleeding patterns in the previous 12 months and was categorized as: a) premenopausal = menstrual period in the past 3

months with no change in regularity in the past 12 months; b) early perimenopausal = menstrual period in the past 3 months with some change in regularity over the previous 12 months; c) late perimenopausal = no menstrual period within the past 3 months, but some menstrual bleeding within the past 12 months; d) post menopausal = no menstrual period within the past 12 months; e) surgical menopause = hysterectomy or bilateral oophorectomy; f) indeterminant menopausal status = used hormone therapy before final menses or surgical menopause, thus, status could not be determined. Also across the follow-up period, participants were asked to report use of hormone therapy (including estrogen and progestin), prescribed (e.g., antihypertensive, anticoagulant, diabetic, heart, or birth control) and over the counter medications (e.g., pain), and CVD events (including heart attack and stroke).

Analysis Plan

Descriptive characteristics of baseline data for the full SWAN sample and stratified by race were assessed using analysis of variance with post-hoc contrasts and chi square analyses as appropriate. Primary study variables, everyday discrimination and CRP were assessed as continuous variables. Due to highly skewed values, CRP was log-transformed and verified by examining a normal probability plot of residuals. Log transformed CRP values were used in all analyses. However, in Table 1 the non-log transformed CRP mean values are reported.

Generalized estimating equations (GEE) were used to test; 1) the main effect of everyday discrimination, 2) the everyday discrimination × race interaction term, and 3) the everyday discrimination × BMI interaction term on CRP over time. GEE is an extension of the generalized linear model and is used to analyze longitudinal data where multiple observations from the same participant are likely to be correlated. The GEE method uses quasi-likelihood estimation that takes into account the within-subject correlation and is robust to misspecification of the unknown correlation structure.

Covariates were organized into two categories for entry into the GEE models; fixed and time-varying covariates. The fixed variables, which we treated as consistent over time included site, age, race, education, financial strain, cynicism, and depressive symptoms. The baseline values for these fixed variables were included in all analyses. The remaining covariates were treated as time-varying covariates, that is, the value entered could change over time and was assessed at baseline and at each of the annual follow up visits and the respective values across those seven time points. The time-varying covariates were time, smoking status and alcohol consumption, physical activity, menopausal status, medication use, hormone therapy, a new heart attack or stroke, levels of systolic blood pressure, triglycerides, LDL, HDL, and glucose. BMI, a time-varying variable, was treated as a covariate in the models testing the main effect of everyday discrimination and the *Everyday Discrimination × Race* interaction term. When the *Everyday Discrimination × BMI* interaction term was tested, BMI was treated as a dichotomous variable where there was a non-obese and obese (indicated by a BMI of 30 or greater) grouping.

In regard to the Hispanic sample, analyses were conducted on data from the women at the New Jersey site for everyday discrimination through follow-up 3 and for CRP through follow-up 5. New Jersey was the only site with Hispanic participants, thus data on this group were available only for analyses through follow-up 5. The results across all three hypotheses

did not change when Hispanic women were excluded, thus they were included in all reported results. All analyses were conducted using Statistical Analysis System, Version 9.2, software (SAS Institute, Inc., Cary, North Carolina). A $p < .05$ (two-tailed) was used to determine significance.

Results

The mean age of the 2,490 women included in the current sample was 46.3 ($SD = 2.7$) and the majority were White (49%) and Black (24.6%). As indicated by Table 1, there were significant racial differences in baseline characteristics. In post hoc analyses not shown, Black and Hispanic women had significantly higher CRP than Whites, Japanese, and Chinese $ps < .005$. Japanese and Chinese women tended to have very similar CRP level profiles, whereas Chinese women reported higher levels of everyday discrimination compared with Japanese women. As expected, Black women reported significantly higher levels of everyday discrimination than all other groups, whereas Hispanic women reported significantly lower levels than all other groups. Both Black and Hispanic women had significantly higher BMI compared with White, Chinese, and Japanese women, p .005.

Everyday Discrimination Predicts CRP Over Time, in Non-Obese Women

The findings indicate that everyday discrimination did not have a significant main effect on CRP, $B = .003$, $SE = .005$, $p = .58$, in the full sample or when testing an everyday discrimination × race interaction term (p's > .05). The *Everyday Discrimination × BMI* interaction term significantly predicted CRP, $p = .03$. To probe the significant finding, simple effects analyses were conducted stratified by non-obese (BMI < 30: 72%) and obese (BMI 30; 28%) status. The relation of everyday discrimination to CRP over time was found in women who were not obese, $B = .01$, $SE = .01$, $p = .04$, whereas there was no association in the obese women, $B = -.01$, $SE = .01$, $p = .25$.

Discussion

This study sought to elucidate the longitudinal relation of everyday discrimination to inflammation in a sample of racially diverse women. We observed that everyday discrimination was associated with greater inflammation as measured by CRP levels over a 7-year period. This relationship did not vary by race, but was observed only in women with a BMI of less than 30. Altogether, the current findings demonstrate that everyday discrimination can impact the inflammatory process implicated in CVD onset in women of different races or ethnicities.

This is the first study to test whether everyday discrimination is linked to CRP over time. The current findings underscore cross-sectional findings (Friedman et al., 2009; Lewis et al., 2010) as non-obese women reporting personal experiences of bias and incivility in interpersonal interactions unfolding across various situations and settings had higher CRP levels over the 7-year follow up period. These findings also extend the studies that have focused on other forms of chronic stress (e.g., daily interpersonal, caregiver, or job strain stress) and their respective links to inflammation outcomes. Similar findings have been demonstrated when using a psychosocial stress score based on four indicators (lower social

class, being divorced or separated from a partner, psychological distress, and less education; Hamer & Stamatakis, 2008) and when summing across daily diary negative interpersonal interactions in a sample of adolescents (Fulgini et al., 2009). Altogether these data present a compelling case for the link between chronic stress generally and everyday discrimination in particular and inflammation and suggest that further research explicating the role of inflammation as a pathway linking stress to CVD risk is warranted.

This study was the first to test whether the relation of everyday discrimination to CRP was conditional upon BMI. We found that everyday discrimination is related to increases in CRP over time for normal-weight and overweight women but not for the obese. These results were not completely surprising. The literature linking discrimination to BMI has been mixed (Cozier, Wise, Palmer, & Rosenberg, 2009; Hebl & Heatherton, 1998; Hunte & Williams, 2009; Lewis et al., 2010) whereas the relation of BMI to CRP is well-established (Barinas-Mitchell et al., 2001; Gruenewald et al., 2009; Rexrode et al., 2003; Visser et al., 1999). With regard to the link between discrimination and BMI, theoretically it is plausible that a stressor would be linked to weight through behavioral and physiological processes.

One explanation for the null relation of everyday discrimination to CRP in larger (BMI 30) women is based on the biological changes that ensue from obesity (Wang & Nakayama, 2010). As an individual becomes obese, their adipocytes enlarge contributing to substantial alterations in adipose tissue that subsequently affects systemic inflammation. The increase in levels of multiple proinflammatory markers may obscure any physiological impact that exposure to everyday discrimination has on individuals who are larger. We also examined other factors that might account for the relation of everyday discrimination to CRP including health status and diseases, behaviors, and demographic factors.

This study also provided an important opportunity to explore whether the link of everyday discrimination to CRP differed as a function of race. Researchers have long argued that discrimination acts as a unique stressor among Blacks and other racial minorities, which may account for the racially patterned disparities in multiple physical health outcomes (Clark, Anderson, Clark, & Williams, 1999). In more recent years, a burgeoning literature suggests everyday discrimination is a harmful stressor with negative implications for CVD related risk factors among racial minority and non-minority racial groups alike (Friedman et al., 2009; Hunte & Williams, 2009). Our results support these findings. We explored the role of race in the relation of everyday discrimination to CRP and found no differences, suggesting that the impact of this stressor may be detrimental for racial minority and non-minority racial groups.

It is no surprise that the literature examining the role of discrimination as a chronic stressor in relation to CVD outcomes has predominately focused on racial minorities. These groups have historically been disenfranchised and continue to experience greater rates of mistreatment as compared with non-minority racial groups (Byrd, 2012). A growing literature documents that the impact of discrimination, without regard to race may have a notable, negative impact, which may be observed in racial minorities and non-minority racial groups. The current findings support this notion as we examined everyday discrimination. Future research which explicitly delineates the various forms of

discrimination overall, as well as racial discrimination is warranted. Such research may yield the best understanding of how these forms of chronic stress overlap, are distinct, or perhaps exacerbate each other. Examining these factors in a racially diverse sample would be critical.

This study has some limitations, which should be considered when interpreting the findings. First, it is possible that our assessment of everyday discrimination did not fully capture the comprehensive nature of such experiences among women (Essed, 1991). Self-report measures of discrimination are focused on those experiences which individuals ascertain as being biased and thus, may not include those experiences they either cannot recall or that were ambiguous in intent or delivery. Additionally, the focus on individual perception can possibly be influenced by personality characteristics or negative emotions. Here, we were able to assess the relation of everyday discrimination to CRP independent of trait cynicism and depressive symptomatology. We believe the utilization of a well-validated measure of everyday discrimination and accounting for relevant behavioral, physiological, and demographic factors outweighs these limitations.

This study makes several contributions. Our focus on perceptions of global discrimination versus a specific form of discrimination perhaps allowed us to capture experiences that participants could not ascribe to a specific attribute phenotypic or otherwise (e.g., race, gender, weight, religion, or SES). The assessment of CRP across multiple time points allowed us to more comprehensively examine this inflammatory marker. Indeed, research indicates that chronic stress may impact CVD risk through low-grade, but chronic inflammation (Shivpuri et al., 2012). This suggests that the ability to gauge the implications of CRP may be limited if assessed at a single time point and the potential impact of this factor on CVD risk may be missed. To date, most studies have been cross-sectional, limiting our ability to understand how the CRP profile may be affected over time by self-reported discrimination. The inclusion of women from racial minority and non-minority groups allowed us to better understand whether the discrimination-health link documented in the literature (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Paradies, 2006; Pascoe & Richman, 2009; Williams & Mohammed, 2009) was applicable to groups other than Black women. Exploratory analyses on the role of race in the relation of everyday discrimination to CRP revealed no race differences suggesting that the impact of this chronic stressor on inflammation is present regardless of race. This finding contributes to a burgeoning recognition in the literature that discrimination can be deleterious for all.

Acknowledgments

References

Albert M, Glynn R, Buring J, Ridker P. C-reactive protein levels among women of various ethnic groups living in the United States (from the Women's Health Study). The American Journal of Cardiology. 2004; 93(10):1238–1242. [PubMed: 15135696]

Albert M, Ravenell J, Glynn R, Khera A, Halevy N, de Lemos J. Cardiovascular risk indicators and perceived race/ethnic discrimination in the Dallas Heart Study. American Heart Journal. 2008; 156(6):1103–1109. [PubMed: 19033005]

Allport, GW. The nature of prejudice. Reading, MA: Addison-Wesley; 1954.

Arnlöv J, Ingelsson E, Sundström J, Lind L. Impact of body mass index and the metabolic syndrome on the risk of cardiovascular disease and death in middle-aged men. Circulation. 2010; 121(2):230–236. [PubMed: 20038741]

Barefoot JC, Beckham JC, Haney TL, Siegler IC, Lipkus IM. Age differences in hostility among middle-aged and older adults. Psychology and Aging. 1993; 8(1):3–9. [PubMed: 8461112]

Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG, Williams RB Jr. The Cook-Medley hostility scale: Item content and ability to predict survival. Psychosomatic Medicine. 1989; 51(1):46–57. [PubMed: 2928460]

Bandura, A. Social cognitive theory of mass communication. In: Bryant, J.; Zillmann, D., editors. Media effects: Advances in theory and research. 2nd. Mahwah, NJ: Lawrence Erlbaum Associates, Inc; 2002. p. 121-154.

Barinas-Mitchell E, Cushman M, Meilahn EN, Tracy RP, Kuller LH. Serum levels of C-reactive protein are associated with obesity, weight gain, and hormone replacement therapy in healthy postmenopausal women. American Journal of Epidemiology. 2001; 153(11):1094–1101. [PubMed: 11390329]

Barnes LL, De Leon CF, Lewis TT, Bienias JL, Wilson RS, Evans DA. Perceived discrimination and mortality in a population-based study of older adults. American Journal of Public Health. 2008; 98(7):1241–1247. [PubMed: 18511732]

Barnes LL, De Leon CF, Wilson RS, Bienias JL, Bennett DA, Evans DA. Racial differences in perceived discrimination in a community population of older blacks and whites. Journal Of Aging & Health. 2004; 16(3):315–337. [PubMed: 15155065]

Beatty DL, Matthews KA. Unfair treatment and trait anger in relation to nighttime ambulatory blood pressure in African American and white adolescents. Psychosomatic Medicine. 2009; 71(8):813–820. [PubMed: 19661190]

Beiner L, Albers AB. Young adults: Vulnerable new targets of tobacco marketing. American Journal of Public Health. 2004; 94(2):326–330. [PubMed: 14759950]

Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. International Journal of Epidemiology. 2002; 31(2):285–293. [PubMed: 11980781]

Brondolo E, Love E, Pencille M, Schoenthaler A, Ogedegbe G. Racism and hypertension: A review of the empirical evidence and implications for clinical practice. American Journal Of Hypertension. 2011; 24(5):518–529. [PubMed: 21331054]

Brown C, Matthews K, Bromberger J, Chang Y. The relation between perceived unfair treatment and blood pressure in a racially/ethnically diverse sample of women. American Journal Of Epidemiology. 2006; 164(3):257–262. [PubMed: 16777930]

Byrd DR. Race/ethnicity and self-reported levels of discrimination and psychological distress, California, 2005. Preventing Chronic Disease. 2012; 9:e156. [PubMed: 23078667]

Camara SK, Orbe MP. Understanding interpersonal manifestations of "reverse discrimination" through phenomenological inquiry. Journal of Intercultural Communication Research. 2009; 40(2):111–134.

Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans: A biopsychosocial model. American Psychological Association. 1999; 54(10):805–816.

Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. Journal of the American Medical Association. 2007; 298(14):1685–1687. [PubMed: 17925521]

Coussons-Read ME, Okun ML, Nettles CD. Psychosocial stress increases inflammatory markers and alters cytokine production across pregnancy. Brain, Behavior, and Immunity. 2007; 21(3):343–350.

Cozier YC, Wise LA, Palmer JR, Rosenberg L. Perceived racism in relation to weight change in the Black Women's Health Study. Annals of Epidemiology. 2009; 19(6):379–387. [PubMed: 19364665]

Cozier YC, Yu J, Coogan PF, Bethea TN, Rosenberg L, Palmer JR. Racism, segregation, and risk of obesity in the Black Women's Health Study. American Journal of Epidemiology. 201410.1093/aje/kwu004

Davis MC, Zautra AJ, Younger J, Motivala SJ, Attrep J, Irwin MR. Chronic stress and regulation of cellular markers of inflammation in rheumatoid arthritis: Implications for fatigue. Brain, Behavior, and Immunity. 2008; 22(1):24–32.

Essed, P. Understanding everyday racism: An interdisciplinary theory. Newbury Park, California: SAGE Publications; 1991.

Evans WD. Reverse discrimination claims: Growing like kudzu. Maryland Bar Journal. 2004; 37(1): 48–51.

Fransson EI, Batty GD, Tabák AG, Brunner EJ, Kumari M, Shipley MJ, et al. Kivimäki M. Association between change in body composition and change in inflammatory markers: An 11-year follow-up in the Whitehall II Study. Journal of Clinical Endocrinology and Metabolism. 2010; 95(12):5370–5374. [PubMed: 20719833]

Fuligni AJ, Telzer EH, Bower J, Cole SW, Kiang L, Irwin MR. A preliminary study of daily interpersonal stress and C-reactive protein levels among adolescents from Latin American and European backgrounds. Psychosomatic Medicine. 2009; 71(3):329–333. [PubMed: 19196810]

Friedman E, Williams D, Singer B, Ryff C. Chronic discrimination predicts higher circulating levels of E-selectin in a national sample: The MIDUS study. Brain, Behavior, and Immunity. 2009; 23(5): 684–692.

Friedman EM, Herd P. Income, education, and inflammation: Differential associations in a national probability sample (The MIDUS study). Psychosomatic Medicine. 2010; 72(3):290–300. [PubMed: 20100883]

Gee GC. A multilevel analysis of the relationship between institutional and individual racial discrimination and health status. American Journal of Public Health. 2008; 98(1):48–56.

Gruenewald TL, Cohen S, Matthews KA, Tracy R, Seeman TE. Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. Social Science & Medicine. 2009; 69(3):451–459. [PubMed: 19524346]

Hamer M, Chida Y. Active commuting and cardiovascular risk: A meta-analytic review. Preventive Medicine. 2008; 46(1):9–13. [PubMed: 17475317]

Hamer M, Gibson E, Vuononvirta R, Williams E, Steptoe A. Inflammatory and hemostatic responses to repeated mental stress: Individual stability and habituation over time. Brain, Behavior, and Immunity. 2006; 20(5):456–459.

Hamer M, Stamatakis E. The accumulative effects of modifiable risk factors on inflammation and haemostasis. Brain, Behavior, and Immunity. 2008; 22(7):1041–1043.

Hebl MR, Heatherton TF. The stigma of obesity in women: The difference is black and white. Personality and Social Psychology Bulletin. 1998; 24(4):417–426.

Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among U.S children, adolescents, and adults, 1999-2002. JAMA: Journal of the American Medical Association. 2004; 291(23):2847–2850.

Henderson SO, Bretsky P, Henderson BE, Stram DO. Risk factors for cardiovascular and cerebrovascular death among African Americans and Hispanics in Los Angeles, California. Academic Emergency Medicine. 2001; 8(12):1163–72. [PubMed: 11733295]

Henderson SO, Haiman CA, Wilkens LR, Kolonel LN, Wan P, Pike MC. Established risk factors account for most of the racial differences in cardiovascular disease mortality. Plos One. 2007; 2(4):e377. [PubMed: 17440613]

Hughes D, Rodriguez J, Smith EP, Johnson DJ, Stevenson HC, Spicer P. Parents' ethnic—racial socialization practices: A review of research and directions for future study. Developmental Psychology. 2006; 42(5):747–770. [PubMed: 16953684]

Hunte HR, Williams DR. The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. American Journal of Public Health. 2009; 99(7):1285–1292. [PubMed: 18923119]

Kelley-Hedgepeth A, Lloyd-Jones DM, Colvin A, Matthews KA, Johnston J, Sowers MR, et al. SWAN Investigators. Ethnic differences in C-reactive protein concentrations. Clinical Chemistry. 2008; 54(6):1027–1037. [PubMed: 18403563]

Kittleson MM, Meoni LA, Wang NY, Chu AY, Ford DE, Klag ME. Association of childhood socioeconomic status with subsequent coronary heart diseases in physicians. Archives of Internal Medicine. 2006; 166(21):2356–2361. [PubMed: 17130389]

Kuh, DL.; Ben-Shlomo, Y. A life course approach to chronic disease epidemiology; Tracing the origins of ill-health from early to adult life. Oxford: Oxford University Press; 1997.

Lewis TT, Aiello AE, Leurgans S, Kelly J, Barnes LL. Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. Brain, Behavior, and Immunity. 2010; 24(3):438–443.

Lewis TT, Everson-Rose SA, Powell LH, Matthews KA, Brown C, Karavolos K, et al. Wesley D. Chronic exposure to everyday discrimination and coronary artery calcification in African-American women: The SWAN heart study. Psychosomatic Medicine. 2006; 68(3):362–368. [PubMed: 16738065]

Lewis TT, Yang FM, Jacobs EA, Fitchett G. Racial/ethnic differences in responses to the everyday discrimination scale: A differential item functioning analysis. American Journal of Epidemiology. 2012; 175(5):391–401. [PubMed: 22306556]

Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, Simone GD, et al. Wylie-Rosett J. Heart disease and stroke statistics-2010 update: A report from the American Heart Association. Circulation. 2010; 121:e46–e215. [PubMed: 20019324]

Maibach, EW.; Rothschild, ML.; Novelli, WD. Social marketing. In: Glanz, K.; Rimer, BK.; Lewis, FM., editors. Health behavior and health education: Theory, research, and practice. 3rd. San Francisco, CA: Jossey-Bass; 2002. p. 437-461.

Marin TJ, Martin TM, Blackwell E, Stetler C, Miller GE. Differentiating the impact of episodic and chronic stressors on hypothalamic-pituitary-adrenocortical axis regulation in young women. Health Psychology. 2007; 26(4):447–455. [PubMed: 17605564]

McDade TW, Hawkley LC, Cacioppo JT. Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: The Chicago Health, Aging, and Social Relations Study. Psychosomatic Medicine. 2006; 68(3):376–381. [PubMed: 16738067]

Messer LC, Shoe E, Canady M, Sheppard BK, Vincus A. Reported adolescent sexual norms and the development of a social marketing campaign to correct youth misperceptions. Journal of Children and Poverty. 2011; 17(1):45–63.

Midei AJ, Matthews KA, Bromberger JT. Childhood abuse is associated with adiposity in mid-life women: Possible pathways through trait anger and reproductive hormones. Psychosomatic Medicine. 2010; 72(2):215–223. [PubMed: 20064904]

Miller G, Rohleder N, Stetler C, Kirschbaum C. Clinical depression and regulation of the inflammatory response during acute stress. Psychosomatic Medicine. 2005; 67(5):679–687. [PubMed: 16204423]

Minior T, Galea S, Stuber J, Ahern J, Ompad D. Racial differences in discrimination experiences and responses among minority substance users. Ethnicity & Disease. 2003; 13(4):521–527. [PubMed: 14632272]

Nguyen XT, Lane J, Smith BR, Nguyen NT. Changes in inflammatory biomarkers across weight classes in a representative U.S. population: A link between obesity and inflammation. Journal of Gastrointestinal Surgery. 2009; 13(7):1205–1212. [PubMed: 19415399]

Nijm JJ, Kristenson MM, Olsson AG, Jonasson LL. Impaired cortisol response to acute stressors in patients with coronary disease. Implications for inflammatory activity. Journal of Internal Medicine. 2007; 262(3):375–384. [PubMed: 17697159]

O'Connor DB, Hendrickx H, Dadd T, Elliman TD, Willis TA, Talbot D, et al. Dye L. Cortisol awakening rise in middle-aged women in relation to psychological stress. Psychoneuroendocrinology. 2009; 34(10):1486–1494. [PubMed: 19520518]

Pager D, Western B, Bonikowski B. Discrimination in a low-wage labor market: A field experiment. American Sociological Review. 2009; 74(5):777–799. [PubMed: 20689685]

Paradies Y. A systematic review of empirical research on self-reported racism and health. International Journal of Epidemiology. 2006; 35(4):888–901. [PubMed: 16585055]

Pascoe EA, Smart Richman L. Perceived discrimination and health: A meta-analytic review. Psychological Bulletin. 2009; 135(4):531–554. [PubMed: 19586161]

Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO III, Criqui M, et al. Vinicor F. Markers of inflammation and cardiovascular disease: Application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation. 2003; 107(3):499–511. [PubMed: 12551878]

Power C, Hertzman C. Social and biological pathways linking early life and adult disease. British Medical Bulletin. 1997; 53(1):210–221. [PubMed: 9158295]

Radloff LS. The CES-D Scale: A self-report depression scale for research in the general population. Applied Psychological Measurement. 1977; 1(3):385–401.

Ranjit N, Diez-Roux AV, Shea S, Cushman M, Ni H, Seeman T. Socioeconomic position, race/ethnicity, and inflammation in the Multi-Ethnic Study of Atherosclerosis. Circulation. 2007; 116(21):2383–2390. [PubMed: 18025402]

Ranjit N, Diez-Rouz AV, Shea S, Cushman M, Seeman T, Jackson SA, Ni H. Psychosocial factors and inflammation in the multi-ethnic study of atherosclerosis. Archives of Internal Medicine. 2007; 167(2):174–181. [PubMed: 17242319]

Rexrode KM, Pradhan A, Manson JE, Buring JE, Ridker PM. Relationship of total and abdominal adiposity with CRP and IL-6 in women. Annals Of Epidemiology. 2003; 13(10):674. [PubMed: 14599731]

Ridker P. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation. 2003; 107(3):363–369. [PubMed: 12551853]

Ridker P, Rifai N, Pfeffer M, Sacks F, Moye L, Goldman S, Flaker GC, Braunwald E. Inflammation, pravastatin, and the risk of coronary events after myocardial infarction in patients with average cholesterol levels. Circulation. 1998; 98(9):839–844. [PubMed: 9738637]

Ridker P, Buring J, Cook N, Rifai N. C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events: An 8-year follow-up of 14719 initially healthy American women. Circulation. 2003; 107(3):391–397. [PubMed: 12551861]

Rivas-Drake D, Hughes D, Way N. A preliminary analysis of associations among ethnic-racial socialization, ethnic discrimination, and ethnic identity among diverse urban sixth graders. Journal of Research on Adolescence. 2009; 19(3):558–584.

Roberts RE. Reliability of the CES-D scale in different ethnic contexts. Psychiatry Research. 1980; 2(2):125–134. [PubMed: 6932058]

Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WJ, Bravata DM, et al. Turner MB. Heart disease and stroke statistics-2012 update: A report from the American Heart Association. Circulation. 2012; 125:e2–e222. [PubMed: 22179539]

Ross R. The pathogenesis of atherosclerosis: A perspective for the 1990s. Nature. 1993; 362(6423):801–809. [PubMed: 8479518]

Schneiderman N, Ironson G, Siegel S. Stress and health: psychological, behavioral, and biological determinants. Annual Review of Clinical Psychology. 2005; 1:1607–628.

Shekelle RB, Gale M, Ostfeld AM, et al. Hostility, risk of coronary heart disease, and mortality. Psychosomatic Medicine. 1983; 45(2):109–114. [PubMed: 6867229]

Shivpuri S, Gallo L, Crouse J, Allison M. The association between chronic stress type and C-reactive protein in the multi-ethnic study of atherosclerosis: Does gender make a difference? Journal of Behavioral Medicine. 2012; 35(1):74–85. [PubMed: 21503709]

Signorielli N. Minority representation in prime time: 2000 to 2008. Communication Research Reports. 2009; 26(4):323–336.

Slopen N, Lewis T, Gruenewald T, Mujahid M, Ryff C, Albert M, Williams D. Early life adversity and inflammation in African Americans and Whites in the midlife in the United States survey. Psychosomatic Medicine. 2010; 72(7):694–701. [PubMed: 20595419]

Sowers, M.; Crawford, S.; Sternfeld, B. SWAN: A multicenter, multiethnic, community-based cohort study of women and the menopausal transition. In: Lobo, RA.; Kelsey, J.; Marcus, R., editors. Menopause: Biology and Pathobiology. San Diego, CA: Academic Press; 2000. p. 175-188.

Sternfeld B, Ainsworth BE, Quesenberry CP. Physical activity patterns in a diverse population of women. Preventive Medicine. 1999; 28(3):313–323. [PubMed: 10072751]

Barack Obama; 2009. The White House. Retrieved from http://www.whitehouse.gov/about/presidents/barackobama

Umberson D, Liu H, Reczek C. Stress and health behaviour over the life course. Advances in Life Course Research. 2008; 13:19–44.

Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. Journal of the American Medical Association. 1999; 282(22):2131–2135. [PubMed: 10591334]

Wallace R, Wallace D, Wallace R. Coronary heart disease, chronic inflammation, and pathogenic social hierarchy: a biological limit to possible reductions in morbidity and mortality. Journal of The National Medical Association. 2004; 96(5):609–619. [PubMed: 15160975]

Wang Z, Nakayama T. Inflammation, a link between obesity and cardiovascular disease. Mediators of inflammation. 2010; 2010

Williams DR. Miles to go before we sleep: Racial inequities in health. Journal of Health and Social Behavior. 2012; 53(3):279–295. [PubMed: 22940811]

Williams DR, Mohammed SA. Discrimination and racial disparities in health: Evidence and needed research. Journal of Behavioral Medicine. 2009; 32(1):20–47. [PubMed: 19030981]

Biographies

Dr. Danielle L. Beatty Moody is an Assistant Professor in the Department of Psychology and the Principal Investigator of the Social Determinants of Health Inequities (SoDHI) lab at the University of Maryland, Baltimore County, Baltimore, Maryland. She earned her PhD in Social and Personality Psychology at the City University of New York, Graduate Center, in 2007. Her research focuses on individual level social and psychological factors, which contribute to racial/ethnic disparities in cardiovascular disease risks. She has (co)authored articles in the *APA journal Health Psychology*, *Journal of Applied Social Psychology*, *and Psychosomatic Medicine.*

Dr. Karen A. Matthews is a distinguished Professor of Psychiatry, and Professor of Epidemiology, Psychology, & Clinical and Translational Science at the University Of Pittsburgh School Of Medicine, Pittsburgh, Pennsylvania. She earned her PhD in Psychology at the University of Texas at Austin in 1976. Her research interests are in development of cardiovascular risk in adolescents, Pathways connecting socioeconomic status and health, Women's health/menopause, Psychobiological determinants of cardiovascular risk.

Dr Joyce T. Bromberger is an Associate Professor of Epidemiology and Psychiatry at the University of Pittsburgh, Pittsburgh, Pennsylvania. Her work focuses on the relationship between physical and mental health in middle-aged women and the mechanisms that might explain the association between depression and cardiovascular disease in particular.

Dr. Charlotte Brown is an Associate Professor of Psychiatry and Health and Community Systems (Nursing) at the University of Pittsburgh, Pittsburgh, Pennsylvania. She earned her PhD from The American University in Washington D.C. Her research and clinical interests include depression recognition and treatment in primary care practice, psychosocial factors affecting women's health; and the impact of race and ethnicity on depression recognition and management.

Table 1

Baseline characteristics for full sample of SWAN participants and stratified by race

Characteristics	Total N=2,490		Black N=613		White N=1,233		Chinese N=221		Hispanic N=178		Japanese N=245	
Age (years)	46.3	2.7	46.2	2.7	46.3	2.7	46.5	2.6	46.4	2.7	46.8	2.7
4-year College degree (%)	47.0		9.0		28.0		5.0		1.0		5.0	
Have difficulty paying for basics (%)	36.0		10.0		15.0		3.0		5.0		3.0	
Premenopausal status [a]	55.0		13.0		27.0		5.0		4.0		6.0	
Systolic blood pressure (mmHg)	116.0	15.9	124.0	19.1	113.0	13.7	112.7	15.4	122.8	11.6	110.0	11.2
Diastolic blood pressure (mmHg)	74.9	10.2	76.7	11.6	73.2	9.4	73.1	10.7	81.8	7.7	74.9	8.6
Glucose (mg/dl)	92.9	16.2	95.5	17.7	91.7	14.9	92.6	8.7	96.6	29.1	92.2	7.9
HDL cholesterol (mg/dl)	56.9	14.1	56.4	13.7	56.7	14.3	60.6	12.8	50.0	11.3	60.8	14.4
LDL cholesterol (mg/dl)	115.2	30.6	116.8	33.1	115.9	30.6	107.4	26.2	121.3	26.8	111.0	28.5
Triglycerides (mg/dl)	107.1	71.1	95.6	55.3	107.3	67.8	106.8	59.5	131.0	93.9	118.5	100.8
Physical activity (score)	7.8	1.8	7.3	1.7	8.1	1.8	7.3	1.7	6.8	1.5	7.9	1.6
Body mass index, kg/m²	27.2	6.4	30.2	6.7	27.0	6.2	23.2	3.7	28.4	5.4	23.0	3.7
CES-D (score)	10.0	9.3	10.3	9.7	9.8	9.0	8.2	8.2	15.5	10.7	8.3	8.1
Cynicism (score)	3.9	3.0	4.9	3.2	3.2	2.6	4.0	3.0	6.1	3.5	3.6	2.8
Current Smoker (yes)	15.0		5.0		7.0		0.1		1.0		1.0	
Current Drinker (yes)	72		63		84		47		36		70	
Other medication use (yes) [b]	32		36		34		11				83	
Hypertensive (yes) [c]	13.0		6.0		3.0		1.0		2.0		1.0	
Everyday discrimination (score)	7.2	4.9	9.2	5.2	6.8	4.3	8.5	4.7	2.5	4.1	5.9	4.8
C-reactive protein (mg/L) [d]	2.2	2.4	3.0	2.7	2.2	2.3	1.3	1.8	2.8	2.5	.95	1.4

Data are means ± SD for continuous variables and % for categorical variables.

[a] Regarding menopausal status at baseline, all women were either premenopausal or early perimenopausal.

[b] The other medication use variable included use of anticoagulant, heart, and blood pressure medications, insulin, birth control pills or over the counter medications for pain.

[c] Hypertensive status at baseline was determined as follows; systolic blood pressure, 140 mmHg or diastolic blood pressure, 90 mmHg.

[d] For ease of interpretation, the C-reactive protein (mg/L) reported here is not log-transformed. All characteristics varied by race at p-value < .0001 (except for age which was at p = .06).