Examining the Association Between Perceived Discrimination and Heart Rate Variability in African Americans

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Objective: Previous research attempting to delineate the role of discrimination in racial/ethnic disparities in hypertension has focused largely on blood pressure, which is chiefly governed by the sympathetic branch of the autonomic nervous system. Consequently, few studies have considered the role of the parasympathetic branch and particularly its regulation of the heart via the vagus nerve. *Method:* In the present cross-sectional study, we employed hierarchical linear regressions to examine associations between perceived ethnic discrimination and resting heart rate variability (HRV), an important biomarker of parasympathetic cardiac modulation and overall health, in a sample ($N = 103$) of young, healthy African American participants (58% female, $M_{age} = 19.94$ years, $SD = 2.84$). *Results:* After accounting for demographic factors and health status characteristics, lifetime discrimination emerged as an inverse predictor of HRV. When subdomains of discrimination were considered, discrimination attributable to threats or actual acts of aggression was also predictive of lower HRV. *Conclusions:* Our findings suggest that a greater lifetime burden of discrimination and discriminatory harassment and/or assault is associated with lower resting HRV in African Americans. The implications of these findings are discussed in the context of past, present and emerging research emphasizing biological linkages between discrimination and health.

Keywords: African Americans, discrimination, heart rate variability, threat

African Americans continue to face the greatest burden of hypertension in the world (Go et al., 2013). Disparities in cardiovascular disease (CVD) prevalence, morbidity, and mortality account for a substantial portion of observed differences in life expectancy between African Americans and Whites (Kochanek, Arias, & Anderson, 2013; Miniño, 2013), and hypertension alone accounts for 15% of all CVD deaths among African Americans (Wong, Shapiro, Boscardin, & Ettner, 2002). Amid growing recognition of the important contribution of psychosocial stressors in CVD etiology and progression (Dimsdale, 2008; Hamer & Malan, 2010; Mensah, Mokdad, Ford, Greenlund, & Croft, 2005; Steptoe & Kivimäki, 2012), a stalwart hypothesis has been the notion that frequent experiences of racism and discrimination play a crucial role in the development of hypertension and broader health disparities faced by African Americans (Anderson & Armstead, 1995; Anderson, McNeilly, & Myers, 1991; Clark, Anderson, Clark, & Williams, 1999). Notably, in a seminal work, Clark and colleagues (1999) posited that the experience of racism and racial discrimination across multiple domains (i.e., structural, institutional and interpersonal) represents a unique form of chronic psychosocial

stress. According to this Biopsychosocial Model of Racism, African Americans experience frequent exposure to discrimination, which may serve to initiate exaggerated psychological and physiological (i.e., fight-or-flight) responses. In addition, engaging in passive or other potentially maladaptive forms of coping (i.e., anger) may actually serve to prolong mental and physical reactions to the stressor. Over time, this chronic pattern of exposure, poor or dysfunctional coping, and exaggerated reactivity is argued to contribute to dysregulation of the cardiovascular system, ultimately giving rise to chronic elevations in blood pressure (BP) vis-à-vis hypertension (Anderson et al., 1991; Clark et al., 1999).

Consistent with the stress and coping (Lazarus & Folkman, 1984) and reactivity (e.g., Krantz & Manuck, 1984; Manuck, Kasprowicz, & Muldoon, 1990) frameworks on which it is based, the Biopsychosocial Model of Racism suggests a prominent role of the autonomic nervous system (ANS) as a major biological pathway linking discrimination-related stress to health and disease. In particular, the sympathetic branch of the ANS plays a substantial role in the regulation of BP and chronically heightened sympathetic nervous system (SNS) activity has been characterized as the primary driver of hypertension (Amerena & Julius, 1995; Brook & Julius, 2000; Julius, Schork, & Schork, 1988; Palatini & Julius, 2009), especially in African Americans (e.g., Hill, Sollers, Edwards, Thayer, & Whitfield, 2014; Taherzadeh, Brewster, van Montfrans, & VanBavel, 2010).

A number of studies have focused on the association between discrimination and BP, or hypertension (for reviews see Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Brondolo, Rieppi, Kelly, & Gerin, 2003; Couto, Goto, & Bastos, 2012; Cuffee, Hargraves, & Allison, 2012; Dolezsar, McGrath, Herzig, & Miller, 2014; Harrell, Hall, & Taliaferro, 2003; Paradies, 2006; Williams & Neighbors, 2001); however, there is an ongoing debate regarding the consistency of this relationship (e.g., Brondolo et al., 2011; Couto et al., 2012; Dolezsar et al., 2014; Paradies, 2006). For instance, in a recent meta-analysis of perceived racial discrimination and hypertension/BP studies spanning a 30-year period, Dolezsar and colleagues (2014) found that perceived discrimination was consistently associated with hypertensive status; however, the size of this effect was relatively small (Fisher's $z = .05$) and was moderated by such factors as Black race/ethnicity, male gender, older age and educational status. In addition, the extent to which discrimination was associated with BP also varied, as effects were much stronger for ambulatory blood pressure (ABP) among Blacks, and nighttime diastolic ABP, in particular, as compared with resting systolic (SBP) or diastolic (DBP) blood pressure (Dolezsar et al., 2014). As these findings suggest, a focus on sympathetic mechanisms alone may provide only a partial view of the complex, autonomic pathway between discrimination and cardiovascular outcomes. In the present study, we diverge from these previous approaches by examining the relationship between discrimination and *parasympathetic nervous system* (PNS) activity as a means of better understanding *how* discrimination experiences may erode health.

Although BP forms the essential basis for a diagnosis of hypertension, the heart is an essential determinant of BP. Regulation of the heart is complex, involving, among other factors, dynamic interactions of sympathetic and parasympathetic activity. In general terms, during periods of arousal, sympathetic neural activation drives an increase in heart rate (HR), which is important for

mobilization of an adequate response (i.e., to fight or flee) to stressors. In contrast, parasympathetic modulation of the heart (via the vagus nerve) is primarily inhibitory, promoting a slower HR and the conservation of energy. Additionally, under resting conditions, parasympathetic (i.e., vagal) activity is predominant over sympathetic influence (Saul, 1990). Thus, parasympathetic activity is a more sensitive marker of the relative impact of acute and chronic stressors on cardiovascular functioning and health.

Heart rate variability (HRV) is a quantification of parasympathetic cardiac influence, determined from the continuous intervals in time from one heartbeat to the next (i.e., interbeat interval, IBI). Higher HRV is generally considered to be cardio-protective and is regarded as an indicator of better physical and mental health and well-being (Kemp & Quintana, 2013). Lower HRV has been related to established risk factors for CVD, including smoking, obesity, total and low density cholesterol, positive family history of CVD, and age (Thayer, Yamamoto, & Brosschot, 2010). Moreover, lower HRV has been shown to predict the onset of hypertension (Schroeder, Liao, Chambless, Prineas, Evans, & Heiss, 2003), as well as increased CVD risk and all-cause mortality (Thayer & Lane, 2007). Further, lower HRV has been linked to poorer mental health outcomes including depression (Larsen & Christenfeld, 2009) and anxiety (Friedman, 2007; Tully, Cosh, & Baune, 2013). Given the high rates of comorbidity between depression and CVD, some researchers have proposed that HRV may be a common mechanism linking mental and physical health (Larsen & Christenfeld, 2009).

Researchers have similarly hypothesized that lower HRV may, at least partially, explain how stressors arising from minority status and socioeconomic disadvantages "get under the skin" and contribute to the greater rates of CVD among African Americans (Lampert, Ickovics, Horwitz, & Lee, 2005). Indeed, some research has reported findings of lower HRV in African Americans relative to Whites (e.g., Choi et al., 2006; Lampert et al., 2005), whereas other research has shown higher HRV in African Americans (Liao et al., 1995; Wang et al., 2005). More recently, Hill and colleagues (2015) conducted a meta-analysis of 17 studies comprising more than 11,000 total participants, reporting ethnic differences in basal HRV between African Americans and Whites. These authors reported that African Americans actually exhibit higher resting HRV, or a relative 'Vagal Advantage,' compared with Whites. Importantly, this effect was robust even when accounting for moderating factors such as age or whether studies were conducted in healthy or nonhealthy samples. Interestingly, this effect was observed consistently in African American women, but was not significant for African American men (Hill et al., 2015). In previous research, resting HRV has been characterized as a stable, trait-like measure that reflects individual differences in the capacity to navigate challenges and demands in the environment (Appelhans & Luecken, 2006; Segerstrom & Nes, 2007; Thayer & Lane, 2000). In this regard, individuals with higher HRV are thought to possess better or more efficient emotion regulation capabilities (e.g., Williams, Cash, Rankin, Bernardi, Koenig, & Thayer, 2015) that arguably buffer against the psychological and physiological experience of myriad stressors, including racial discrimination.

Although there is a growing literature on the relationship between psychosocial stressors and HRV, very few studies have examined this association in African Americans. Even fewer stud-

ies have considered the relationship between discrimination and HRV. One notable exception is a study by Dorr et al. (2007) wherein the researchers examined hemodynamic and autonomic cardiovascular responses to a racist versus nonracist interaction in a sample of African American men. These researchers found that men instructed to express versus inhibit their anger following the racist interaction exhibited delayed HRV recovery during the 10-min postinteraction period (Dorr, Brosschot, Sollers, & Thayer, 2007). In another laboratory-based study, Neblett and Roberts (2013) found African American participants to exhibit decreases in HRV during a blatantly racist imaginal task. In particular, among individuals with moderate levels of private regard (i.e., moderately positive feelings about one's racial group membership), there was a greater decrease in HRV during the blatantly racist imaginal scenarios when the perpetrator was White versus Black (Neblett & Roberts, 2013).

In another study, Wagner and colleagues (2015) found an inverse association between lifetime discrimination and HRV during a stressful speech task in a sample of 32 White and African American women with type II diabetes (Wagner, Lampert, Tennen, & Feinn, 2015). Finally, more recently, Hoggard and colleagues (2015) examined the effects of intergroup and intragroup discrimination on HRV across a 2-day period in a sample of African American women. They found that racial discrimination involving an African American perpetrator (i.e., confederate) was actually associated with an increase in HRV during the following 20-min recording period whereas racial discrimination involving a White perpetrator was associated with no change in HRV. On Day 2, those participants who had experienced discrimination from the White perpetrator exhibited lower HRV and higher HR than those who had experienced discrimination involving the African American perpetrator. The researchers concluded that merely returning to the environment in which one has previously experienced a discriminatory event may trigger a shift in cardiac autonomic functioning (Hoggard, Hill, Gray, & Sellers, 2015). Such a pattern could have significant implications for individuals living and working in settings where they frequently perceive or experience racial discrimination.

Fortunately, there have been some positive findings regarding the link between discrimination and HRV. Notably, Utsey and Hook (2007) assessed the impact of resting HRV on the relationship between race-related stress and psychological distress in 215 African American college students. Higher levels of institutional racism were associated with greater psychological distress in both men and women; however, the strength of this association was weaker in men with higher HRV (Utsey & Hook, 2007). In another report, Cooper and colleagues (2014) examined the association between cardiovascular function and the use of prayer coping in response to discrimination in a sample of 81 African American women. In this study, greater prayer coping was positively associated with HRV following a racism recall task (Cooper, Thayer, & Waldstein, 2014). These findings are consistent with the notion that higher HRV may serve to buffer the effects of discrimination-related stress in African Americans. To our knowledge, this handful of studies represents the current literature regarding the potential effects of perceived discrimination on HRV. Thus, the goal of the present study is to further elucidate an important biological mechanism by which accumulative racial discrimination experiences may compromise health.

Although it is evident that racial discrimination is a noxious stressor, researchers are unclear about whether the various forms or facets of interpersonal racial discrimination differentially impact health and well-being (Brondolo et al., 2005; Contrada et al., 2001; Sue et al., 2007). Researchers conducting correlational and survey studies have typically operationalized African Americans' experiences with racial discrimination as the composite frequency and/or impact score for one of the various racial discrimination scales (i.e., Everyday Discrimination Scale, Schedule of Racist Events, Daily Life Experience Scale). Few, however, have examined racial discrimination as a multidimensional construct with multiple subdomains. Such an examination would clarify whether the various forms or subdomains of racial discrimination (e.g., being harassed or threatened vs. being excluded) differentially impact cardiac functioning. Similarly, evaluating the different dimensions of racism and the psychological and physiological correlates of these dimensions can facilitate an understanding of psychobiological mechanisms linking racism to health (Brondolo et al., 2005). The present study attempts to facilitate this understanding by examining whether the relationship between perceived racial discrimination and HRV is consistent across different subdomains or facets of perceived racial discrimination.

The Present Study

The present study examines the association between lifetime and subdomains of perceived racial discrimination and resting HRV in a sample of African American college students. Previous research on discrimination and HRV has either focused on this relationship in exclusively female (Cooper, Thayer, & Waldstein, 2014; Hoggard et al., 2015; Wagner et al., 2015) or male samples (Dorr et al., 2007); or has conceptualized HRV mainly as a moderator with little attention to its importance as an outcome (Utsey & Hook, 2007). Thus, it is unclear whether perceived racial discrimination has a direct negative impact on parasympathetic cardiac control, or whether this potential relationship is the same for both African American men and women. We hypothesized that perceived racial discrimination would be associated with lower HRV. We were particularly interested in examining whether this relationship was consistent across different aspects of perceived racial discrimination. Indeed, prior research suggests that discrimination related to threats and/or actual instances of harassment or physical harm may be more burdensome on health (Brondolo et al., 2008). Finally, we were interested in examining whether the relationship between perceived racial discrimination and HRV was moderated by gender, as previous research suggests that African American women may be more vulnerable to the psychological effects of racial discrimination (e.g., anxiety; Banks et al., 2006; Greer et al., 2009) whereas African American men may be more vulnerable to the physiological effects of racial discrimination (Morris-Prather et al., 1996).

Method

Participants

One hundred three self-identified African American college students were recruited at a large public university in the Midwest through the Research Experience Program (REP) pool, which

allows students to participate in research for partial class credit in an introductory level psychology course. Participants were also recruited, outside of the REP pool, using flyers, campus newspaper ads, and email listservs; these participants received a small monetary compensation (i.e., $15.00) for their participation. All participants were instructed to not smoke, engage in vigorous physical activity, or consume caffeine for at least two hours prior to their scheduled appointment. The study was conducted in compliance with the university Institutional Review Board, and all participants provided their written informed consent. Physiological data (i.e., heart rate) were missing for four participants due to equipment failure. The final sample included 99 African American participants (58% female, M_{age} = 19.94 years, SD = 2.84) with complete data.

Procedure

The present data represent baseline physiological recordings from a larger psychophysiological protocol. All participants were greeted by a study researcher in a designated waiting area. The participants were escorted to a soundproof experimental room that was equipped with a camera and a microphone for observational and instructional purposes as well as a high definition TV for stimuli presentation. Participants were provided with a general description of the study purpose (i.e., to examine the relationship between stress and physiological activity) as well as a detailed explanation of the procedures for psychophysiological recording in both verbal and written form. After providing consent, participants completed a preliminary packet of study questionnaires including the perceived discrimination measure. Thereafter, participants were outfitted with equipment for physiological recording (i.e., ECG, continuous BP monitoring device). Participants were then instructed to sit quietly and breathe as they normally would, while their baseline physiological activity was recorded for a 5-min period.

Measures

Heart rate variability. Continuous heart rate (HR) data was assessed using a 3-lead ECG at a sampling rate of 1kHz. Electrodes were placed below the right clavicle and on the left and right lower abdomen. Data were visually inspected for artifacts and corrected per recommended guidelines (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), using a custom software package (*HRV 2.51,* Mindware Technology, Gahanna, OH). The series of interbeat intervals (IBI), or time in milliseconds between successive R-spikes on the ECG waveform, were then written to a single text file. These files were then imported to the Kubios HRV analysis package 2.0 (Tarvainen, Niskanen, Lipponen, Ranta-aho, & Karialainen, 2014) and subjected to spectral analysis yielding time and frequency domain measures of HRV. In the present study, we focus on the high frequency (HF) HRV power band (HF-HRV, 0.15–0.4 Hz), which has previously been shown to be a valid and reliable measure of vagal activity (Thayer, Hansen, & Johnsen, 2010). As the distribution of HF-HRV was positively skewed, the data were natural log-transformed resulting in a normal distribution.

Discrimination. Racial discrimination was assessed using the brief Perceived Ethnic Discrimination Questionnaire-Community

Version (PEDQ-CV; Brondolo et al., 2005). The brief PEDQ-CV is a 17-item questionnaire designed to assess lifetime experiences of racial discrimination and maltreatment, particularly in interpersonal and social contexts. The measure contains four subscales that assess differing domains of discrimination including: social exclusion (Exclusion), stigmatization (Stigma), discrimination at work or school (Work/School), and threats or actual acts of harassment and/or harm (Threats). Each subscale consists of four items and each item is preceded by the stem phrase: "Because of your ethnicity/race, how often . . . ," followed by statements reflecting each domain: "have others ignored or not paid attention to you" (Exclusion); "have others hinted that you must be lazy/not clean" (Stigma); "have you been treated unfairly by co-workers or classmates" (Work/School); "have others threatened/actually hurt you" (Threats). Participants rated the frequency (1 = *never* to 7 = *very often*) with which they have experienced unfair treatment in one of the four domains. A higher score for each subscale is indicative of more frequent experiences with racial discrimination. In addition, all 17 items are summed to create a Lifetime Discrimination scale. In the present sample, internal consistency for the total scale was (α = .90) and ranged from .68 to.78 for the subscales.

Covariates. Covariates for the present study were selected based on previously reported relationships with HRV, including age, gender, body mass index (BMI), and physical activity level (Thayer, Yamamoto, & Brosschot, 2010). Although there was no evidence of hypertension among study participants, we also included resting BP. Indeed, BP has been shown to be inversely related to HRV in African American adolescents and adults (e.g., Keen et al., 2015; Urbina et al., 1998); and is a relative marker of SNS activity. Resting BP was collected continuously during the baseline period using a noninvasive photoplethysmography device.

Demographics. Participants self-reported their age and gender.

Health characteristics. Participant height and weight were measured using a medical grade stadiometer and scale. BMI was calculated as weight in kilograms (kg) divided by height in meters squared (m^2). Physical activity was assessed via self-report using the University of Houston Non-Exercise Questionnaire (Jackson et al., 1990), which assesses activity over the previous month on a 0 (i.e., avoid walking or exertion) to 7 (i.e., run 10 miles or 3 hours of comparable weekly physical activity) scale. Higher scores on this measure have been correlated with a greater level of cardiorespiratory fitness (Jackson et al., 1990).

Statistical analysis. We sought to test whether perceived racial discrimination predicts lower HRV, and whether the various aspects of perceived racial discrimination were associated with lower HRV to the same degree. We tested these questions in a hierarchical regression analysis using *STATA 12*. Specifically, we conducted two hierarchical regression analyses in which HRV served as the dependent variable. All variables were entered in blocks so that coefficients specified at subsequent steps predicted residual variance unexplained by the variables in previous steps. In terms of centering, we standardized all continuous variables. For the first hierarchical regression, gender and the standardized covariates of age, BMI, physical activity, SBP, and DBP were entered at Step 1. In Step 2, we added lifetime experiences of perceived racial discrimination (standardized). Finally, the cross-product of the gender and the standardized version of perceived

racial discrimination was entered at Step 3 to assess whether the effect of lifetime experiences of perceived racial discrimination was contingent on whether the individual is male or female. The second hierarchical regression mirrored the first hierarchical regression with one exception: we replaced the lifetime experiences of discrimination predictor with the discrimination subscales (i.e., Exclusion, Stigma, Work & School, and Threat).

Results

Participant Characteristics

Descriptive statistics (means, standard deviations) are presented in Table 1. There were no gender differences in age, $F(1, 98) = 1.129$, $p = .29$, $\eta_p^2 = .01$; BMI, $F(1, 98) = .538$, $p = .47$, $\eta_p^2 = .01$; SBP, $F(1, 98) = 1.022$, $p = .32$, $\eta_p^2 = .01$; DBP, $F(1, 98) = .104$, $p = .75$, $\eta_p^2 = .00$; Lifetime Discrimination $F(1, 98) = 2.405$, $p = .124$, $\eta_p^2 = .02$; or in the Exclusion $F(1, 98) = .086$, $p = .77$, $\eta_p^2 = .00$; Work/School $F(1, 98) = 2.329$, $p = .13$, $\eta_p^2 = .02$; or Threat $F(1, 98) = .948$, $p = .33$, $\eta_p^2 = .01$ subscales (see Table 1). Males reported higher levels of physical activity $F(1, 98) = 8.350$, $p = .005$, $\eta_p^2 = .08$; and marginally higher levels of discrimination due to stigma $F(1, 98) = 4.565$, $p = .06$, $\eta_p^2 = .01$, compared with females. There was also a marginal trend, $F(1, 98) = 3.106$, $p = .08$, $\eta_p^2 = .04$; for higher resting HF-HRV in females, compared with males.

Hierarchical Regressions

Results of the first hierarchical regression are displayed in Table 2. In Step 1, gender was found to be the only significant predictor of HRV, $b = .56$, 95% CI [.08, 1.04]. Being female was associated with higher HRV—accounting for 5% of the variance in HRV, $sr^2 = .054$. At Step 2, and consistent with predictions, lifetime experiences of discrimination significantly predicted lower HRV, $b = -.26$, 95% CI [−.51, −.02]. Approximately 4% of the variance in HRV was attributable to lifetime experiences of discrimination. Gender also remained a significant predictor of HRV after accounting for lifetime experiences with discrimination, $b =$

Table 1
Means and Standard Deviations

Variable	Male	Female	Total
N	43	56	99
Age (years)	19.63 (1.89)	20.25 (3.46)	19.98 (2.89)
BMI (kg/m^2)	26.48 (6.17)	27.49 (7.16)	27.05 (6.73)
Phy. Activity	4.95 (2.16)**	3.79 (1.86)	4.29 (2.07)
SBP (mm Hg)	112.61 (16.69)	109.10 (17.43)	110.63 (17.12)
DBP (mm Hg)	57.64 (14.02)	56.59 (17.27)	57.05 (15.87)
PD-Lifetime	34.00 (10.34)	30.55 (11.41)	32.05 (11.04)
PD-Exclusion	9.58 (3.01)	9.79 (3.74)	9.70 (3.43)
PD-Stigma	8.05 (3.45)*	6.59 (3.3)	7.22 (3.42)
PD-Work/School	8.35 (2.95)	7.38 (3.29)	7.80 (3.17)
PD-Threat	5.65 (2.45)	5.20 (2.19)	5.39 (2.30)
HF-HRV	6.18 (1.15)	6.63 (1.09)	6.43 (1.13)

Note. BMI = body mass index; Phy. = physical; SBP = systolic blood pressure; DBP = diastolic blood pressure, PD = perceived discrimination, HF-HRV = high frequency heart rate variability.
* $p < .05$. ** $p < .01$.

.46, 95% CI [.02, .94]. The cross-product of gender and lifetime experiences of discrimination did not significantly predict HRV at Step 3, $b = -.10$, 95% CI [−.59, .39].

Results of the second hierarchical regression are displayed in Table 3. Female gender remained the only significant predictor of HRV in Step 1. When the perceived discrimination subscales were entered into the model at Step 2, discrimination attributable to threat predicted lower HRV, $b = -.28$, 95% CI [−.55, −.01]. Approximately 4% of the variance in HRV was attributable to discrimination due to threat, $sr^2 = .041$. Besides gender, no other predictors significantly predicted HRV, $b = .53$, 95% CI [.02, 1.05]. At Step 3, tests of interactions revealed that there were no gender-contingent associations between discrimination subscales and HRV.[1]

Discussion

Disparities in health and life expectancy persist for African Americans. Hypertension and related complications account for the largest proportion of CVD-related deaths in African Americans, and there are emerging data linking discrimination to mortality (e.g., Chae et al., 2015), as well. HRV is an important index of cardiovascular function and health, and has also been linked to psychological health and functioning. In the present study, we examined the relationship between perceived racial discrimination, across multiple domains, and resting HRV in a sample of African American emerging adults. We hypothesized that perceived discrimination would be associated with lower HRV. Consistent with our hypothesis, we found a direct, inverse effect of perceived discrimination on resting HRV. This association was not contingent on gender, or other covariates previously associated with lower HRV. In addition, the effect was most robust for more frequent experiences of discrimination involving being threatened or actually being physically harassed as a result of one's ethnicity. These findings clearly extend the fledgling literature regarding racial discrimination and HRV by demonstrating that discriminatory experiences may gradually reduce an individual's "first line of defense," or the initial capacity for coping with chronic racial and nonracial stressors.

Being mindful of the relatively limited nature of our results, it is worth noting that our findings complement and give further context to previous research on discrimination and BP. In particular, early hypertension research revealed that elevations in BP were driven by increased, or hyperactive, SNS activity, whereas PNS activity was hypoactive (Julius, Pascual, & London, 1971). These observations formed the basis of the Autonomic Imbalance model of hypertension (Amerena & Julius, 1995). Additional findings of consistent elevations in SNS vascular activity, both under resting

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[1] Although, we focus on HF-HRV we also considered time domain measures, particularly, the root mean square of successive differences (RMSSD) and the percentage of successive R–R intervals differing by more than fifty milliseconds (pNN50). HF-HRV was strongly associated with both RMSSD, $r = .90$, $p < .001$, and pNN50, $r = .85$, $p < .001$. With the exception of lifetime discrimination on pNN50 ($b = -.16$, $SE = 2.30$, 95% C.I. [−8.18, .88], $p = .112$), regression results for discrimination were largely consistent with findings for HF-HRV, particularly for the influence of lifetime discrimination on RMSSD ($b = -.21$, $SE = .06$, 95% C.I. [−.24, −.001], $p = .049$), and discrimination attributable to threat on both RMSSD ($b = -.33$, $SE = .07$, 95% C.I. [−.31, −.04], $p = .01$) and pNN50 ($b = -.32$, $SE = 2.60$, 95% C.I. [−11.69, −1.38], $p = .01$).

Table 2

First Hierarchical Regression Predicting Heart Rate Variability

Variable	Step 1 b	SE	95% CI lower	95% CI upper	Step 2 b	SE	95% CI lower	95% CI upper	Step 3 b	SE	95% CI lower	95% CI upper
Intercept	**6.12**	**.18**	**5.76**	**6.47**	**6.16**	**.18**	**5.81**	**6.51**	**6.15**	**.18**	**5.80**	**6.51**
Age	.00	.12	−.23	.24	.02	.12	−.22	.25	.01	.12	−.22	.25
Female	**.56**	**.24**	**.08**	**1.04**	**.46**	**.24**	**.02**	**.94**	.46	.24	−.02	.95
BMI	.05	.12	−.19	.28	.08	.12	−.15	.32	.08	.12	−.16	.32
Phy. Activity	.21	.13	−.06	.47	.22	.13	−.04	.48	.22	.13	−.04	.48
SBP	.10	.18	−.27	.46	.13	.18	−.23	.49	.13	.18	−.24	.49
DBP	−.20	.17	−.54	.14	−.26	.17	−.59	.08	−.26	.17	−.59	.08
Discrimination (Lifetime)					**−.26**	**.13**	**−.51**	**−.02**	−.21	.19	−.58	.16
Interaction: Female × Lifetime									−.10	.25	−.59	.39

Note. Gender is coded as Male = 0, Female = 1. SBP = systolic blood pressure; DBP = diastolic blood pressure. All continuous variables are standardized. Standardized discrimination variable was used when creating the interaction term. Bold coefficients are significant at $p < .05$.

conditions and in response to stressors among African Americans, greatly informed the hypothesis that hypertension was predominately driven by greater SNS activity in this group. Thus, the focus of previous research on the relationship between discrimination and BP was well-grounded. Indeed, it has been relatively recent that scholars have broadened their view to reconsider the role of the PNS. Typically, during the initiation of the fight or flight response, PNS modulation decreases, allowing SNS tone to trigger an increase in HR and further facilitating the full cascade of physiological changes needed to respond to the stressor. Once the threat or challenge has passed, PNS dominance of HR is reasserted. It may be that African Americans have an intrinsically higher SNS vascular tone, and have adapted to this state by developing greater resting HRV (i.e., a Vagal Advantage). Hypothetically, once this rebalanced system is altered, perhaps as a result of chronic psychological distress (i.e., anxiety, Friedman, 2007), HRV is irreversibly diminished, paving the way for relatively unchecked SNS activity and chronic disease. In context, our findings are complementary to previous results showing a positive

relationship between racial discrimination and increased BP, as it is possible, though speculative, that HRV may also have been lower among African Americans in these studies. Although HRV declines with age, longitudinal research will be invaluable to ultimately determining *whether*, *how*, and *when* the hypothesized shift from higher to lower resting HRV begins in African Americans.

Scholars have continually emphasized the importance of considering contextual factors that may influence the relationship between discrimination and health (Brondolo, 2015). The Biopsychosocial Model of Racism also acknowledges that factors such as age, gender, individual differences in coping styles (i.e., active vs. passive) and other personality characteristics (i.e., trait anger), socioeconomic status, differing types of discrimination, and the race/ethnicity of the perpetrator may all have important mediating and moderating effects on this relationship. Although we did not find that gender moderated the impact of discrimination on resting HRV, our finding of a relatively consistent gender difference in favor of females is consistent with previous work (Hill et al., 2015)

Table 3

Second Hierarchical Regression Predicting Heart Rate Variability

Variable	Step 1 b	SE	95% CI lower	95% CI upper	Step 2 b	SE	95% CI lower	95% CI upper	Step 3 b	SE	95% CI lower	95% CI upper
Intercept	**6.12**	**.18**	**5.76**	**6.47**	**6.13**	**.18**	**5.77**	**6.50**	**6.10**	**.19**	**5.73**	**6.47**
Age	.00	.12	−.23	.24	−.02	.13	−.27	.23	.00	.13	−.26	.25
Female	**.56**	**.24**	**.08**	**1.04**	**.55**	**.25**	**.04**	**1.05**	**.53**	**.26**	**.02**	**1.05**
BMI	.05	.12	−.19	.28	.07	.12	−.18	.31	.07	.12	−.18	.32
Phy. Activity	.21	.13	−.06	.47	.24	.14	−.04	.51	.24	.14	−.04	.52
SBP	.10	.18	−.27	.46	.12	.18	−.24	.48	.16	.19	−.21	.54
DBP	−.20	.17	−.54	.14	−.27	.17	−.61	.07	−.29	.18	−.64	.06
Discrimination (Exclusion)					−.13	.16	−.45	.19	−.34	.26	−.86	.18
Discrimination (Stigma)					.07	.18	−.28	.42	.09	.25	−.40	.58
Discrimination (Work & School)					.01	.18	−.34	.37	.17	.29	−.40	.75
Discrimination (Threat)					**−.28**	**.14**	**−.55**	**−.01**	−.26	.20	−.66	.14
Interaction: Female × Exclusion									.43	.35	−.26	1.13
Interaction: Female × Stigma									−.12	.36	−.84	.60
Interaction: Female × Work & School									−.26	.35	−.96	.43
Interaction: Female × Threat									−.14	.28	−.71	.42

Note. Gender is coded as Female = 0, Male = 1. SBP = systolic blood pressure; DBP = diastolic blood pressure. All continuous variables are standardized. Standardized discrimination variable was used when creating the interaction term. Bold coefficients are significant at $p < .05$.

as well as a more general pattern in the larger HRV literature. At first glance, this pattern, at least generally, comports with previous research indicating that African American women may be less susceptible to the physiological impacts of discrimination than African American men (Morris-Prather et al., 1996). Additional research is needed to further explicate the biopsychosocial underpinnings of gender differences in relation to the experience and impact of discrimination.

We found that both lifetime discrimination and discrimination attributable to threat or actually being physically harassed were associated with lower HRV. This supports that broadband measures of discrimination may be useful for future studies of HRV, but also illustrates the additional informative value of multidomain measures. Our findings for the Threat subscale are consistent with previous research indicating that discrimination related to threats and/or actual instances of harassment or physical harm may be more salient and detrimental to health (Brondolo et al., 2008). Although the items on the Threat subscale assess personal experiences, it is tantalizing to consider whether witnessing threats or overt acts of violence, and particularly events interpreted as discriminatory or race-related in origin, has a vicarious physiological impact. For instance, both news and social media have facilitated the broad and rapid sharing of information and profound imagery from recent events in Sanford, Jacksonville, Ferguson, Detroit, North Charleston, Tulsa, Staten Island, Baltimore, and unfortunately, numerous other locales, that may be experienced collectively by African Americans as racially motivated and/or discriminatory. Although far beyond the scope of the present study, it is nonetheless feasible that discriminatory threat and violence, experienced indirectly, influence subsequent behavior (e.g., Himmelstein, Young, Sanchez, & Jackson, 2015) and possibly physiological activity. For example, as Hicken and colleagues (2013) have suggested, heightened vigilance is one possible behavioral consequence of such events and attributions (Hicken, Lee, Ailshire, Burgard, & Williams, 2013). Chronic vigilance for threat is also a common feature of generalized anxiety disorder (GAD) as well as posttraumatic stress disorder (PTSD), and autonomic dysregulation, including a shift in resting HRV, is a well-established consequence of chronic anxiety (Friedman, 2007; Tully et al., 2013). Current and future research exploring the parallels between racial discrimination and anxiety (e.g., Graham, Calloway, & Roemer, 2015; Hunter & Schmidt, 2010) may be particularly helpful in further determining the physiological costs of race-related vigilance.

Although we have focused primarily on HRV as an outcome, it is also a significant correlate and mediator of other biological mechanisms that have been additionally related to discrimination. In particular, there is emerging interest in the linkages between inflammatory processes and sleep with discrimination. For example, HRV has been inversely related to C-reactive protein (CRP), an important biomarker of systemic and vascular-related inflammation (Johnson, Abbasi, & Master, 2013). African Americans have been shown to exhibit higher CRP levels than Whites (Johnson et al., 2013), and there is growing evidence of a positive association between discrimination and CRP (Beatty Moody, Brown, Mathews, & Bromberger, 2014; Cunningham et al., 2012; Goosby, Malone, Richardson, Cheadle, & Williams, 2015; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010) as well as other inflammatory markers

(Brody, Yu, Miller, & Chen, 2015). Similarly, there is significant correspondence between HRV and both objective and subjective measures of sleep (Stein & Pu, 2012). It is well established that African Americans experience poorer sleep than other groups in the United States (e.g., Durrence & Lichstein, 2006). Previous research has examined the influence of discrimination on nighttime BP (i.e., Hill, Kobayashi, & Hughes, 2007; Tomfohr, Cooper, Mills, Nelesen, & Dimsdale, 2010), and growing evidence suggests that sleep may be another important pathway through which the vigilance associated with discrimination negatively impacts health (e.g., Hicken et al., 2013; Slopen, Lewis, & Williams, 2015). It is not yet clear whether reduced HRV is the cause or consequence of dysfunction in these additional biological pathways. The disruption of multiple biological processes and systems is consistent with the notion of Weathering, or the accumulative impact of multiple co-occurring disruptions in health due to chronic stressors, which contribute to premature biological aging and increased disease risk in African Americans (e.g., Geronimus, Hicken, Keene, & Bound, 2006). Thus, it is crucial that future investigations more closely examine the impact of discrimination on the intersecting relationships among these important overlapping mechanisms.

We acknowledge that our findings reflect a cross-sectional 'snapshot' of the relationship between perceived racial discrimination and resting HRV that limits any inferences that can be drawn regarding causality. Moreover, recall bias may have influenced participants' self-reported experiences of discrimination. However, we incorporated a multidimensional measure of discrimination and the items for some subscales (i.e., Threats) assessed discrete events that may be more salient in memory and thus retrieved with greater specificity. In addition, although our sample size was consistent with previous studies in this area (e.g., Neblett & Roberts, 2013; Utsey & Hook, 2007), our sample composition was relatively homogenous with respect to age and apparent health status. Although these characteristics pose a potential challenge to the generalizability of our findings, it is notable that similar effects were observed in studies of adults with greater variability in age and health status (i.e., Cooper et al., 2014; Dorr et al., 2007; Wagner et al., 2015). In addition, focusing on the impact of racial discrimination among emerging adults has become increasingly important as research suggests that discrimination may begin taking a serious biological toll on African Americans before or by emerging adulthood (Brody et al., 2014; Williams & Mohammed, 2009).

It has been suggested that higher HRV is an index, not only of cardiovascular health, but also of individual resources to adaptively cope with chronic stressors. Our findings suggest that above and beyond the influence of other plausible biological factors, greater lifetime burden of discrimination, and particularly having experienced discriminatory harassment and/or assault, is associated with lower resting HRV in African Americans. These results complement previous research on discrimination and BP, and extend the current literature regarding the relationship between discrimination and HRV. Collectively, these insights may prove informative for future research examining the unique and overlapping biological underpinnings of racial discrimination as a salient and pervasive form of chronic stress.

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