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The Impact of Racial Discrimination and Hostility on Adrenergic Receptor Responsiveness in African Americans

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Abstract

Objective—Racial discrimination is increasingly recognized as a contributor to increased cardiovascular disease risk among African Americans. Previous research has shown significant overlap between racial discrimination and hostility, an established predictor of CVD risk including alterations in adrenergic receptor functioning. The present study examined the associations of racial discrimination and hostility with adrenergic receptor responsiveness.

Methods—In a sample (N=57) of young to middle-aged African American adults (51% female) with normal and mildly elevated blood pressure (BP), a standardized isoproterenol sensitivity test (CD_{25}) was used to evaluate β-AR responsiveness, while the dose of phenylephrine required to increase mean arterial pressure (MAP) by 25 mmHg (PD_{25}) was used to assess α_1-AR responsiveness. Racial discrimination was measured using the Perceived Racism Scale and hostility was assessed using the Cook-Medley Hostility scale.

Results—In hierarchical regression models, greater racial discrimination, but not hostility, emerged as a significant predictor of decreased β-adrenergic receptor responsiveness ($\beta = .38$, $p = .004$). However, moderation analysis revealed that the association between racial discrimination and blunted β-adrenergic receptor responsiveness was strongest among those with higher hostility ($\beta = .49$, 95% CI [.17, .82], $p = .004$). In addition, hostility, but not racial discrimination, significantly predicted α_1-AR responsiveness.

Conclusions—These findings suggest racial discrimination was associated with blunted β-adrenergic receptor responsiveness, providing further evidence of the potential contribution of racial discrimination to increased CVD risk among African Americans. The adverse effects of discrimination on cardiovascular health may be enhanced in individuals with higher levels of hostility.

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Conflicts of Interest:
The authors report no conflicts of interest.

Keywords

Adrenergic receptor responsiveness; discrimination; hostility; African Americans

INTRODUCTION

Racial discrimination has long been considered an important factor in explaining the higher incidence of hypertension and other disparities in rates of cardiovascular disease (CVD) among African Americans (1). Evidence from population-based investigations including the Cardiovascular Risk Development in Young Adults (CARDIA) and Jackson Heart Study (2, 3) has consistently shown an association between racial discrimination and hypertension prevalence. Despite an ever-increasing number of empirical studies, understanding of the mechanisms underlying this putative relationship remains limited. Indeed, recent findings have suggested that examining BP alone may be an inadequate approach to unraveling the complex linkages between racial discrimination and CVD risk (4, 5). For example, one recent meta-analysis showed that the relationship between racial discrimination and hypertensive status was more consistent than the link between discrimination and blood pressure (BP), *per se*. In addition, the association between racial discrimination and BP was moderated by a number of factors including age, sex and the setting in which BP measurements were obtained (i.e. clinic vs ambulatory) (6).

Several mechanisms have been implicated as being responsible for the greater CVD risk among African Americans including diminished nitric oxide bioavailability (7) and salt-sensitivity (8). In addition, research has consistently implicated heightened sympathetic nervous system (SNS) activity (9, 10). The adrenergic receptors play an important role in regulating cardiac and vascular responses to acute and chronic stressors (11, 12). Binding of epinephrine (EP) and norepinephrine (NOR) with either α_1-adrenergic receptors (α_1-AR) and/or β-adrenergic receptors (β-AR), trigger changes in BP via vasoconstriction and/or vasodilation, as well as through modulation of heart rate (HR) and cardiac contractility. Over time, receptor affinity diminishes or is enhanced due to repeated or prolonged catecholamine exposure (13). The resultant pattern of hyperactive α_1-ARs (i.e. excessive vasoconstriction) and/or hypoactive β-ARs (i.e. blunted vasodilation and cardiac response) is an important pathway in CVD pathogenesis (14, 15). Decreased β-AR sensitivity, in particular, is especially problematic as previous research has shown that β-ARs are down-regulated in hypertension (16, 17), ischemic heart disease and chronic heart failure (18–20). There also is evidence of diminished β-AR, but enhanced α_1-AR responsiveness, among African Americans (9, 21), while other research has shown that socio-demographic factors including obesity (22–24) and socioeconomic/social status (25, 26) may further impair β-AR functioning.

Psychological traits and dispositions including anxiety (27–30), depression (31) and low social support (32) also have been related to AR functioning. The presence of high levels of hostility has been shown to be related to diminished β-AR responsiveness, especially among men (13, 32). There is a well-established literature linking hostility with increased CVD incidence and mortality risk (33), and previous studies have noted a consistent positive

association between hostility and racial discrimination (34, 35). Increased hostility is acknowledged as one potential response to frequent exposure to discrimination among African Americans (35–37); it also has been suggested that hostility may actually account for the relationship between discrimination and health outcomes (38). Although no studies have explored the vascular effects of both racial discrimination and hostility concurrently, Thomas and colleagues examined the interaction of ethnicity and perceived racial discrimination on the pressor response in a community-based sample of African American and White adults (39). Pressor responsiveness was defined as the difference between baseline BP and peak BP observed following administration of a 100μg dose of phenylephrine, a selective α_1-AR agonist that mimics the vasoconstrictive effects of norepinephrine to produce a short-term increase in BP. While African Americans exhibited a greater BP pressor response in comparison to Whites, perceived racial discrimination was positively associated with the diastolic pressor response, irrespective of ethnicity (39).

To our knowledge, the study by Thomas, et al., is the only previous investigation of the association between racial discrimination and AR function. In the present study, we sought to further examine the relationship between racial discrimination and AR functioning in African Americans. In particular, we hypothesized that greater racial discrimination would be associated with heightened α_1-AR sensitivity and blunted β-AR sensitivity. Also, because of the significant association between racial discrimination and hostility, as well as evidence linking hostility to adrenergic responsivity, we sought to examine the interactive effects of discrimination and hostility on AR responsiveness.

METHODS

Participants

Fifty-seven African American adults participating in the Biobehavioral Investigation of Hypertension (BIOH) study at Duke University Medical Center are included in the present study. Data were collected between August 1994 and August 1998 and all subjects were recruited via local media (i.e. newspapers, magazines) advertisements. Individuals with BP exceeding 160 mm Hg systolic (SBP) or 100 mm Hg diastolic (DBP), a history of antihypertensive medication or tobacco use, were excluded. The study protocol was approved by Duke University Medical Center's Institutional Review Board, and all participants provided verbal and written consent prior to participation.

Measures

Demographic and Anthropomorphic Characteristics—Demographic and socioeconomic information was obtained by participant self-report. Income was rated on a 6-point Likert scale (1 = less than $15k, 2 = $15k – $29,999, 3 = $30k – $44,999, 4 = $45k – $59,999, 5 = $60k – $74,999 and 6 = $75k and above). Education was similarly rated on a 7-point Likert scale (1 = less than 7^{th} grade, 2 = some high school, 3 = high school graduate, 4 = trade school, 5 = some college, 6 = college graduate and 7 = postgraduate work or degree). Clinic BP was assessed on 3 separate visits, approximately 1 week apart, to determine BP status and study eligibility. Three seated BPs were taken, 2 minutes apart, using an appropriate sized occlusion cuff, mercury column sphygmomanometer, and stethoscope. BP

readings were averaged across the three visits to define clinic BP status. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2).

Racial Discrimination—Racial Discrimination was assessed using the Perceived Racism Scale (40), a 51-item questionnaire that assesses experiences with racism across four domains: racism on the job, racism in academic settings, racism in public settings, and exposure to racist statements. Participants reported the frequency of exposure in each of these areas using a 6-point Likert scale (0 = Not Applicable, 1 = Almost never, 2 = Several times a year, 3 = Several times a month, 4 = Several times a week and 5 = Several times a day). In this study, an overall score was obtained by summing responses across the four domains (41). In the present sample, scores on the Perceived Racism Scale ranged from 0–91, with a higher score indicating a greater frequency of perceived exposure to racism over the course of one's life. The Perceived Racism Scale has demonstrated good internal consistency and Cronbach's alpha in the present study was .97.

Hostility—Hostility (Ho) was assessed using the Cook-Medley Hostility (Ho) Scale (42), 50-item true/false questionnaire derived from the Minnesota Multiphasic Personality Inventory. Scores range from 0 to 50 with higher scores indicating a greater degree of cynicism, mistrust of others and anger proneness. The Ho scale has shown acceptable reliability and validity and Cronbach's alpha in the present sample was .84.

Cardiovascular Measurements During AR Responsiveness Testing—All AR responsiveness testing was conducted while participants were reclined. As previous research has shown that caffeine increases BP and HR (43), we elected to minimize the contribution of potential individual differences in BP and HR increases due to caffeine consumption by invoking a 6-hour abstinence period (44). BP was measured continuously using the Finapres Model 2300 (Ohmeda, Madison, WI) noninvasive BP monitor. This instrument utilizes the vascular unloading technique to measure SBP, DBP and mean arterial pressure (MAP) on a beat-by-beat basis, and has been validated against intra-arterial measures under various conditions including pressor responses to phenylephrine (45). Heart rate (HR) was derived via electrocardiogram (ECG) as the interval between successive R-waves.

β-AR Responsiveness—The standardized isoproterenol sensitivity test (CD_{25}) was used to evaluate β-AR responsiveness in terms of the chronotropic dose of isoproterenol required to increase HR by 25 beats per minute (bpm) (46). Progressively increasing bolus-doses of isoproterenol (0.125, 0.25, 0.5, 1.0, 2.0, 4.0 μg) were injected into a vein until an increase in HR of at least 25 bpm was observed. HR responses following each dose were computed as the shortest 3 successive ECG R-R intervals following drug injection, compared to the shortest 3 R-R intervals at rest (pre-injection). Following each dose, the next higher dose was not injected for at least 5 minutes, or until cardiovascular activity had returned to resting levels, usually within 5–l0 minutes. The linear regression model of log-dose/HR response for each subject was used to determine CD_{25} exactly by interpolation. The CD_{25} measure provides an index of β-AR responsiveness that is inversely related to AR responsiveness (i.e., *higher* CD_{25} values are indicative of *reduced* or *blunted* β-AR responsiveness).

α_1-AR responsiveness—A procedure analogous to the β-AR responsiveness test described above was used for assessing α_1-AR responsiveness, using the α_1-AR pharmacological agonist phenylephrine, to stimulate vascular α_1-AR (47). In this test, the criterion response is defined as the dose required to increase MAP by 25 mmHg (PD_{25}). An initial dose of 25 μg phenylephrine was used, with successive doses doubled until the 25 mmHg response was exceeded, or until a maximum dose of 800 μg. Again, at least 5-minutes, or longer if required for recovery of MAP to resting levels, preceded administration of successive doses. The linear log-dose/MAP response curve was used to determine the exact PD_{25} dose. The PD_{25} index is inversely related to vascular α_1-AR responsiveness (i.e., *higher* PD_{25} indicates *reduced* α_1-AR responsiveness, or more importantly *lower* PD_{25} indicates *heightened* α_1-AR responsiveness).

Statistical Analysis—Descriptive statistics were computed to characterize sample characteristics and to assess initial patterns of association among the study variables. Hierarchical regression analyses were used to evaluate the effects of discrimination and hostility on β-AR responsiveness to isoproterenol (CD_{25}) and α_1-AR responsiveness to phenylephrine (PD_{25}). Covariates were selected based on previous literature (22, 23,25,27–29, 31, 48) and included age, sex (0 = female, 1 = male), BMI, education, income and clinic BP. Model 1 included the aforementioned covariates only. Discrimination was added as the single predictor in Model 2, followed by the inclusion of hostility in Model 3 and the racial discrimination × hostility interaction in Model 4. The significant interaction of racial discrimination and hostility on CD_{25} was further probed in a separate model using the PROCESS conditional effects macro developed by Hayes (49). This utility provides estimates and tests of significance for simple slope effects and boot-strapped estimates of the confidence interval. All statistical analyses were conducted using the SAS 9.3 system (SAS Institute, Cary, NC) with significance set at $p = .05$.

RESULTS

Descriptive statistics (i.e. means, standard deviations and Pearson's correlations) are presented in Table 1. The sample had an average age of 33.2 (standard deviation = 5.9) years and was approximately half female (51%). Participants reported an average level of education consistent with having completed some college and endorsed an average income range of between $30,000 and $44,999. In correlational analyses, age was positively associated with DBP ($r = .38$, $p = .003$). Sex was inversely associated with HR ($r = -.49$, $p < .001$) and positively correlated with PD_{25} ($r = .27$, $p = .045$), indicating lower HR in women and heightened α_1-AR responsiveness in men. BMI was positively associated with SBP ($r = .32$, $p = .016$), DBP ($r = .28$, $p = .036$), HR ($r = .28$, $p = .037$) and CD_{25} ($r = .28$, $p = .038$). PD_{25} was inversely associated with SBP ($r = -.34$, $p = .011$), DBP ($r = -.26$, $p = .049$) and HR ($r = -.28$, $p = .036$). CD_{25} was positively associated with both hostility ($r = .28$, $p = .033$) and racial discrimination ($r = .43$, $p = .001$), and racial discrimination was moderately correlated with hostility ($r = .50$, $p < .001$).

β-AR Responsiveness

Hierarchical regression analysis was used to examine the hypothesis that racial discrimination would be independently related to β-AR responsiveness. As a reminder, *higher* CD_{25} values are indicative of *reduced* or *blunted* β-AR responsiveness.

Covariates including age, sex, BMI, education, income and BP were included in the initial model (Table 2). BMI ($\beta = .29$, $p = .049$) was the only significant predictor in Model 1, which accounted for 10% of the variance in CD_{25}. Discrimination emerged as a significant predictor of CD_{25} ($\beta = .38$, $p = .004$) in Model 2, explaining an additional 13% of the variance in CD_{25}, and suggesting that greater perceived discrimination is associated with blunted β-AR responsiveness. This effect was only modestly attenuated with inclusion of hostility in Model 3, as hostility was not a significant predictor ($\beta = .05$, $p = .76$) and did not account for any additional variance in CD_{25}. Importantly, the racial discrimination × hostility interaction (Model 4) was significant ($\beta = 1.23$, $p = .046$). As depicted in Figure 1, the effect of discrimination on CD_{25} was greatest among individuals with the higher (+1 standard deviation) hostility, ($\beta = .49$, 95% CI [.17, .82], $p = .004$); a similar trend was observed for individuals with average levels of hostility, ($\beta = .22$, 95% CI [−.11, .55], $p = .18$) but not among those individuals with lower hostility, ($\beta = -.05$, 95% CI [−.55, .45], $p = .83$).

α_1-AR responsiveness

Regression analyses were repeated with α_1-AR responsiveness as the focal outcome (Table 3). As a reminder, *higher* PD_{25} indicates *reduced* α_1-AR responsiveness, or more importantly *lower* PD_{25} indicates *heightened* α_1-AR responsiveness. In Model 1, sex ($\beta = 134.80$, $p = .004$), and SBP ($\beta = -5.68$, $p = .009$), were the only explanatory factors in a model explaining 20% of the variance in PD_{25}. The effects for sex and SBP were virtually unchanged in Model 2, as discrimination ($\beta = .07$, $p = .91$) did not account for significant additional variance in PD_{25}. In Model 3, hostility was negatively associated with PD_{25} ($\beta = -.34$, $p = .027$), suggesting that greater hostility is associated with α_1-AR responsiveness. There was no significant interaction effect for racial discrimination and hostility on PD_{25} (Model 4), ($\beta = -.06$, $p = .92$).

DISCUSSION

Increased interest and a growing literature have heightened awareness on the importance of racial discrimination as a unique predictor of CVD risk among African Americans. The primary goal of the present study was to further examine the relationship between perceived racial discrimination and AR functioning. Consistent with our hypotheses, we found that racial discrimination was associated with diminished β-AR sensitivity, and that this effect was magnified among those with higher levels of hostility. Contrary to our expectations, hostility did not emerge as a significant predictor of β-AR responsiveness. However, hostility was associated with heightened α_1-AR responsiveness.

Previous studies have suggested that exposure to racial discrimination contributes to the development of dysfunctional patterns of immune, neuroendocrine and cardiovascular

regulation leading to tissue and organ damage, and ultimately disease onset (50). One ongoing criticism of past research evaluating the relationship between discrimination and health outcomes has been the failure to account for the potential influence of negative affect, such as anger and hostility, or personality factors (51). Indeed, hostility has been linked with an increased risk of CVD events and mortality (33, 52). African Americans have been shown to report higher levels of hostility compared to Whites (53, 54); and hostility has been considered to be a likely response to chronic experiences of prejudice and racial discrimination (35). Given evidence that hostility appears to be a potent predictor of diminished β-AR (i.e. vasodilatory) activity, as well as recent work linking discrimination with a heightened pressor response among African Americans, we examined whether discrimination and hostility would interactively predict AR responsiveness. While it initially appeared that the effect of racial discrimination on β-AR sensitivity was independent of hostility, results of the moderation analysis indicated that hostility enhanced the effect of racial discrimination. In particular, individuals with the highest levels of both hostility and racial discrimination exhibited the greatest reductions in β-AR responsiveness. Moreover, given our observation that higher hostility was associated with heightened α_1-AR responsiveness, racial discrimination in this context would convey a combination of blunted β-AR responsiveness and heightened α_1-AR responsiveness, a pattern that would favor reduced vasodilation and heightened vasoconstriction. Indeed, this hemodynamic profile has been shown to be especially common amongst African Americans and has been linked to the early onset and heightened risk of CVD (9, 15, 55). It has been postulated that reduced β-AR sensitivity among individuals higher in hostility may reflect a greater chronic stress burden and/or heightened stress-related catecholamine activity (32). Chronic stress associated with racial discrimination may be similarly related to increased catecholamine activity, with one recent study reporting positive but non-significant associations between racial discrimination assessed in childhood with overnight urinary epinephrine and norepinephrine assessed in early adulthood (56). Overall, there is limited available data regarding the association between racial discrimination and the stress hormones and further research in this area would be informative.

The present results add to a growing literature demonstrating that racial discrimination is associated with adverse patterns of vascular functioning among African Americans. It has long been established that the vascular component plays a more prominent role in pathological BP regulation among African Americans (35, 57). Yet, with some exception, there has been comparatively little research connecting discrimination to more direct measures of cardiovascular function. Notably, unfair treatment, attributed to multiple factors including race, has been found to be associated with greater intima-media thickness (IMT) among a subsample of African American women from the SWAN study (58). While this and other previous studies (59, 60) provide evidence that racial discrimination is associated with structural measures of vascular dysfunction, racial discrimination also has been linked to biochemical factors associated with local vascular regulation. For instance, Cooper and colleagues examined the relationship between perceived discrimination and Endothelin-1, a vasoconstrictive agent found throughout the central nervous system that has been implicated in vascular changes including small vessel remodeling and left ventricular hypertrophy, in a sample of African American and White adults. Notably, while there was no association

among Whites; perceived discrimination was positively associated with Endothelin-1 among African Americans, even after accounting for gender, physical activity and socioeconomic status (61). Discrimination also has been linked to higher levels of C-reactive protein (CRP) among African Americans (62–65). CRP has been characterized as an important marker of both systemic and vascular-related inflammation (66). It has been suggested that heightened inflammation is one consequence of SNS hyperactivity and some work has shown a positive association between circulating levels of CRP and diminished β-AR sensitivity (48). Collectively, our results add to this prior evidence in suggesting that discrimination is associated with a broad range of both structural and functional indicators of vascular impairment among African Americans.

In contrast to our expectations, we did not find racial discrimination to predict α_1-AR responsiveness. While our sample was larger than the African American subsample in the study by Thomas et al (39), it is noted that we employed a different measure of racial discrimination, incorporated a wider range of covariates, and assessed a slightly different outcome (i.e. the pressor dose of phenylephrine required to elevate blood pressure by 25 mmHg, in contrast to the prior study's index of the absolute change in BP to a single 100μg dose of phenylephrine). In addition, our study was comprised solely of African Americans, while the study by Thomas and colleagues was composed of both African American and White adults. Thus, it is possible that these differences contributed to the lack of a significant main effect of racial discrimination as well as the non-significant interactive effect of racial discrimination and hostility on α_1-AR responsiveness.

Intriguingly, we did observe a significant main effect of hostility on α_1-AR responsiveness. While the association between hostility and β-AR responsiveness has been more clearly documented (13, 27,28, 32, 67), less is known regarding the relationship between hostility and α_1-AR responsiveness. Indeed, some studies examining this association have reported null findings (13, 32). In contrast, hostility accounted for approximately 7% of the variance in PD_{25} in our sample, indicating that increasing levels of hostility were associated with enhanced α_1-AR responsiveness (i.e. greater vasoconstriction). Importantly, this effect was independent of sex and clinic BP, both of which have been recently shown to account for differences in α_1-AR responsiveness (21). Notably, several studies have demonstrated that African Americans exhibit heightened α_1-AR responsiveness compared to Whites (9, 21) and α_1-AR responsiveness also has been shown to be independent of blunted β-AR sensitivity among African Americans (68).

In addition, while we did not examine the extent to which individuals expressed their hostility in the present study, both inhibition and expression of hostile feelings (i.e. anger) have been associated with a blunted decline in nocturnal BP (41, 69), as well as delayed cardiovascular recovery following stress exposure (70). For example, in the study by Dorr et al (70), African American and White men were instructed to either express or inhibit their anger following a race- or non-race related debate. Irrespective of debate topic, African American men who expressed their anger exhibited the most prolonged elevations in BP relative to African American inhibitors, as well as relative to White men in either condition. Further, African American men who inhibited their anger also demonstrated delayed recovery in systemic vascular resistance (SVR), a measure of peripheral vasoconstriction.

While recovery was only assessed up to ten minutes following the stressor in this study, other research has shown that increases in SVR may persist for as long as 45 minutes following exposure to a psychological stressor (71). It has been established that SVR plays a more prominent role in short-term and diurnal BP regulation among African Americans (9, 72, 73), and recent evidence suggests that SVR is more strongly related to an index of vascular hypertrophy among African Americans compared to Whites (74). Importantly, previous work also has shown that α_1-receptors are more strongly implicated in SNS-mediated vasoconstriction (75). While it is clear that additional research is needed to further evaluate the relationship between hostility and α_1-AR responsiveness, our findings do suggest that hostility may play an important role in hypertension risk among African Americans, contributing directly through heightened vasoconstriction and interactively via attenuated vasodilation.

There are several limitations which should be considered in interpreting our findings. First, given the cross-sectional nature of our study, little inference can be drawn regarding the cause-effect relationship between discrimination and hostility with AR responsivity. Our sample was relatively small and comprised entirely of young-to-middle-aged African Americans with normal or mildly elevated BP. Thus, our findings may have limited generalizability to older and/or more diverse populations or to individuals with more chronically elevated BP. While SBP emerged as a significant predictor of α_1-AR responsiveness, neither SBP nor DBP approached significance in models predicting the CD_{25} (i.e. β-AR) response. Although we accounted for markers of cardiovascular risk (i.e. BP, HR, BMI), we did not assess physical activity in the present study. Future research should determine whether physical activity may influence the association of racial discrimination and hostility with AR functioning; however, previous studies have indicated only a weak, inverse association, particularly with β-AR responsiveness (25, 48). Lastly, while we examined β-AR responsiveness using a methodology in accord with previous studies (25, 28,29, 31, 48), it should be noted that isoproterenol is a non-selective β-AR agonist, which stimulates both increased cardiac activity and vascular dilation. Previous work has shown that changes in systemic vascular resistance mirror those in heart rate as captured by the CD_{25} response, using an identical protocol as the current study (13, 76). Other research also has documented both greater β-receptor sensitivity and density among hypertensive African Americans (77). Our CD_{25} β-AR responsiveness index may also be a manifestation of altered β-AR density and/or sensitivity, with available evidence suggesting that reduced sensitivity is the more likely mechanism (77).

Despite these limitations, there are also several strengths of the present work. Most notably, we accounted for a number of additional factors which have previously been shown to influence adrenergic receptor sensitivity. For instance, research has shown that obesity is associated with β-AR down-regulation (22). While BMI was indeed a significant predictor of CD_{25}, its inclusion in the models did not diminish the influence of racial discrimination. Moreover, amid growing evidence that socioeconomic factors are related to AR functioning, we also considered the potential influence of income and education; however, neither of these indicators emerged as significant predictors of the CD_{25} response. In contrast, we did find income to be an inverse predictor of PD_{25}, raising the possibility that SES may be more strongly related to α_1-adrenergic functioning. Lastly, because we evaluated both α- and β-

responsiveness, our findings provide a more complete view of the association (or lack thereof) of racial discrimination with at least one important mechanism of vascular functioning.

Overall, our findings suggest that the adverse impact of racial discrimination on vascular function is due, in part, to blunted β-AR responsiveness and that this effect is enhanced by high levels of hostility. In addition, we found that hostility was directly related to heightened α_1-AR responsiveness, a pattern consistent with excessive stressor-related vasoconstriction among African Americans (68). Further research is needed to better characterize the relationship between racial discrimination and hostility with global SNS functioning, as well as to determine whether other negative psychosocial traits have a similar exacerbating influence.

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Abbreviations

AR	Adrenergic Receptors
BP	Blood Pressure
EP	Epinephrine
NOR	Norepinephrine
CVD	Cardiovascular Disease
CD_{25}	Dose of Isoproterenol to increase heart rate by 25 bpm
BMI	Body Mass Index
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
MAP	Mean Arterial Pressure
PD_{25}	Dose of Phenylephrine to increase mean arterial pressure by 25 mm Hg

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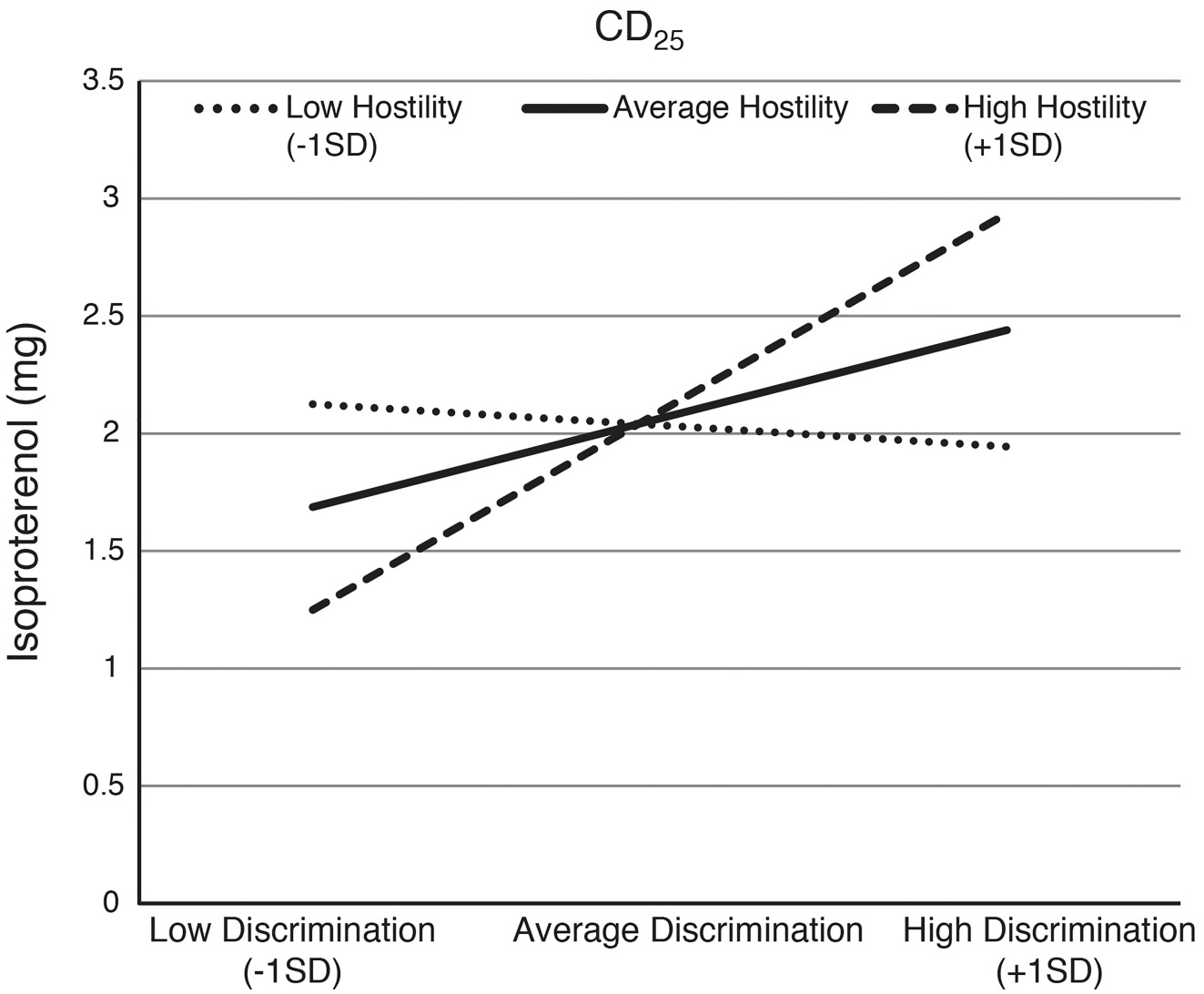

Figure 1. Interaction of Perceived Discrimination and Hostility on Beta-adrenergic Responsivity (i.e. CD_{25})

Higher CD_{25} values are indicative of *reduced* or *blunted* β-AR responsiveness. The effect of discrimination on CD_{25} was greatest among individuals with higher hostility, (β =.49, *SE* = .16, p =.004); compared to individuals with average (β = .22, *SE* = .16, p = .18) and lower (β = −.05, *SE* =.25, p =.83) hostility.

Table 1

Sample Descriptives and Correlations (N=57)

Variable	Mean(SD)	1	2	3	4	5	6	7	8	9	10	11	12
1. Age (years)	33.12 (5.91)	-	−.10	.10	−.07	.00	.19	.38**	.14	.19	.05	.17	.10
2. Sex (% male)	.49		-	−.18	−.08	.16	.19	−.03	−.49***	.21	.27*	.01	.20
3. BMI (kg/m^2)	26.39 (3.61)			-	−.17	.10	.32*	.28*	.28*	.28*	−.14	.10	−.02
4. Education	5.18 (1.3)				-	.18	−.10	−.06	.01	−.17	.00	−.21	.00
5. Income	2.68 (1.09)					-	.05	.16	−.08	.17	−.15	−.05	.31*
6. SBP (mmHg)	131.12 (14.22)						-	.72***	.02	.20	−.34**	.02	.01
7. DBP (mmHg)	81.96 (11.48)							-	.19	.23	−.26*	−.03	.10
8. HR (bpm)	65.42 (12.51)								-	.25	−.28*	.21	.05
9. CD$_{25}$ (µg)	2.26 (1.71)									-	−.11	.28*	.43**
10. PD$_{25}$ (µg)	264.37 (154.37)										-	−.17	.05
11. Hostility	21.56 (7.8)											-	.50***
12. Racial Discrimination	48.12 (31.46)												-

Income was rated on a 6 point scale: 1 = less than $15k, 2 = $15k – $29,999, 3 = $30k – $44,999, 4 = $45k – 59,999, 5 = $60k – $74,999 and 6 = $75k and above. Education was rated on a 7 point scale: 1 = less than 7th grade, 2 = some high school, 3 = high school graduate, 4 = trade school, 5 = some college, 6 = college graduate and 7 = postgraduate work or degree. CD$_{25}$, dose (in micrograms) of isoproterenol to increase heart rate by 25 bpm; PD$_{25}$, dose (in micrograms) of phenylephrine to increase mean arterial pressure by 25mmHg.

*** $p \leq .001$,

** $p \leq .01$,

* $p \leq .05$

Table 2

Regression Model of Discrimination and Hostility Predicting CD_{25} (N = 57)

	Model 1		Model 2		Model 3		Model 4	
	Estimate	*p*	*Estimate*	*p*	*Estimate*	*p*	*Estimate*	*p*
Age	0.16	.25	0.11	.39	0.10	.43	0.12	.36
Sex	0.25	.080	0.19	.17	0.19	.16	0.18	.19
BMI	0.29	.049	0.28	.038	0.28	.043	0.26	.052
Education	−0.10	.46	−0.09	.46	−0.08	.52	−0.06	.63
Income	0.10	.46	0.004	.98	0.003	.98	−0.05	.72
SBP	−0.06	.75	−0.01	.96	−0.02	.93	0.03	.87
DBP	0.12	.55	0.08	.68	0.09	.65	0.02	.90
RD			0.38	.004	0.36	.024	−0.54	.25
HO					0.05	.76	−0.40	.13
RD×HO							1.23	.046
R^2		.10		.23		.22		.27

Note: Estimates represent standardized beta coefficients. RD=racial discrimination, HO = Hostility

Table 3

Regression Model of Discrimination and Hostility Predicting PD_{25} (N = 57)

	Model 1		Model 2		Model 3		Model 4	
	Estimate	*p*	*Estimate*	*p*	*Estimate*	*p*	*Estimate*	*p*
Age	0.16	.22	0.16	.23	0.22	.102	0.22	.107
Sex	0.44	.002	0.44	.003	0.40	.005	0.40	.005
BMI	0.10	.46	0.10	.47	0.13	.33	0.13	.34
Education	0.05	.68	0.05	.68	0.00	.98	0.00	.97
Income	−0.22	.091	−0.23	.100	−0.28	.039	−0.28	.047
SBP	−0.52	.009	−0.52	.010	−0.46	.018	−0.46	.020
DBP	0.08	.69	0.08	.70	−0.02	.91	−0.02	.93
RD			0.02	.91	0.21	.17	0.26	.59
HO					−0.34	.027	−0.31	.25
RD×HO							−0.06	.92
R^2		.20		.18		.25		.23

Note: Estimates represent standardized beta coefficients. RD = racial discrimination, HO = Hostility