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Racial Discrimination and Acute Physiological Responses among Black Young Adults: The Role of Racial Identity

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Abstract

Purpose: Racial discrimination has long-term consequences for cardiovascular health, potentially by dysregulating acute physiological responses. However, the role of psychological factors that may be protective or increase vulnerability for dysregulated responses, such as racial identity, remains unclear. This study examines the association between racial discrimination and acute parasympathetic responses, and the role of racial centrality, private regard, public regard in this association.

Methods: Black young adults ($N = 119$, $M_{age} = 19.45$) recruited from a predominantly White institution in the southeastern United States completed an online survey (in which racial discrimination, racial identity, and control variables were reported) and a laboratory visit, during which they were exposed to a vignette of racial discrimination while their parasympathetic activity (indexed by respiratory sinus arrhythmia) was recorded.

Results: While racial discrimination was not associated with respiratory sinus arrhythmia reactivity or recovery, centrality moderated this association such that more frequent racial discrimination was associated with greater parasympathetic reactivity and recovery only among

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Implications and Contribution:

This study finds that the less central race is to one's self-concept, the more one is able to mitigate acute physiological responses when facing racial discrimination. Programs that help Black young adults cope with racism may thereby protect against cardiovascular risk.

participants low in racial centrality. Neither private regard nor public regard emerged as significant moderators.

Conclusions: This study is the first to show that lower levels of racial centrality can mitigate the association between discrimination and acute parasympathetic responses, which has important implications for initiatives aimed at reducing cardiovascular risk for Black young adults.

Keywords

respiratory sinus arrhythmia; racial discrimination; racial identity; Black Americans; young adults

Black young adults are at higher risk for developing precursors to cardiovascular disease than other racial/ethnic groups in the United States (1). Racial discrimination (RD), defined as actions by members of dominant racial groups that have negative and differential impacts on members of non-dominant racial groups (2), is a contributor to poor health in Blacks (3) and poses developmental risks for Black youth transitioning from adolescence to adulthood (4). RD may negatively impact Black young adults' physiological responses (5), a precursor to long-term cardiovascular health (6). However, not all Black young adults who encounter RD physiologically respond in unhealthy ways. Therefore, the present study examines racial identity, the significance and meaning that Black individuals place on race when defining themselves (7), as a protective or vulnerability factor in the link between RD and physiological functioning.

Racial Discrimination and Acute Physiological Responses

The Biopsychosocial Model of Racism affirms that RD engenders exaggerated and prolonged autonomic nervous system (ANS) responses to threat (8). Physiological overactivation or underactivation due to RD erodes health by conditioning cardiovascular responses to subsequently experienced acute stressors (9–12), preparing Black bodies for future social assaults (13). The present study extends the literature on RD and ANS activity by examining the parasympathetic nervous system (PNS), which has a wide range of control over heart rate regulation via the vagus nerve and is thereby a more sensitive marker of the impact of social stressors (14). We assessed PNS influence by calculating respiratory sinus arrhythmia (RSA) via the magnitude of heart rate variability (HRV) during the respiratory cycle. According to Porges' Polyvagal Theory (15), RSA is sensitive to social interactions and therefore may be especially important for understanding social stressors such as RD. During a stressful situation, RSA should decrease from a baseline level (RSA withdrawal) as individuals bolster psychological resources. The degree of RSA withdrawal could signal disease risk, as lower HRV is a predictor of cardiovascular disease and all-cause mortality (16). During recovery, after the stressor has been removed, RSA activity should rebound (RSA augmentation), evidencing higher levels of PNS activity. The absence of such augmentation is associated with ventricular valve health and subsequent risk for chronic hypertension (17). Therefore, there are two stages of a physiological response to acute stress: a reactivity period wherein the individual is attending to a stressor during which RSA decreases from baseline levels (RSA withdrawal), and a recovery period wherein the stressor is removed and RSA increases from stress level (RSA augmentation). Experimental studies have documented PNS withdrawal during reactivity to racism tasks (18) and less PNS

augmentation during recovery from these tasks (19). Negative associations between chronic RD and HRV have also been reported (5, 20). However, a paucity of studies consider the relationship between RD and RSA and examine differences in RSA during both reactivity and recovery periods.

The Role of Racial Identity

The transition from adolescence to young adulthood for Black individuals can be shaped by racial prejudice (4) and experiencing RD propels Black young adults to explore *racial* identities (21). The present study examines three dimensions of racial identity aligned with the Multidimensional Model of Racial Identity (7) - centrality (the degree to which one defines oneself in terms of race), private regard (how one views Black individuals and their own group membership), and public regard (how one believes others view Black individuals). Racial identity is believed to mitigate the adverse effects of RD on health (22, 23). However, examination of racial identity has produced inconsistent results, suggesting that racial identity could be a protective or vulnerability factor for health outcomes. For example, private regard has served as a protective factor in investigations spanning psychosocial and health outcomes for Black adolescents and young adults (24–25), but was not protective in others (26). While a preponderance of research documents a positive association between centrality and psychological distress (27–28), some studies have documented the inverse association (23). Low public regard is found to buffer individuals from the negative consequences of RD on well-being, specifically depressive symptoms (25). One of the first to examine past year RD and racial identity on RSA, this study helps contextualize these inconsistent results.

Although racial identity has received attention as a protective factor, few studies consider the role of racial identity in the association between RD and acute PNS responses. In the one study of racial identity as protective for acute PNS responses, Black college students high on private regard and low on public regard exhibited RSA withdrawal during reactivity to racism vignettes with a White perpetrator, but RSA augmentation during recovery (24). No significant interactions were found for racial centrality. As this study did not examine past year RD, the present study is an important addition as frequent past year RD may condition dysregulated acute physiological responses to stressors, increasing the potential for cardiovascular risk over time.

The Present Study

Grounded in psychobiological (8, 15) and racial identity (7) frameworks, our investigation adds to the nascent body of literature examining how RD experiences may relate to Black young adults' acute RSA responses to a lab-based racial stressor and the role of racial identity in this association. All moderation hypotheses were proposed in line with Neblett and Roberts (24). Our study examined the following questions:

- RQ1. Is RD associated with acute RSA reactivity (RQ1a) and recovery to a lab-based racial stressor (RQ1b)? We hypothesized that RD would be associated with acute RSA withdrawal during reactivity (RQ1a) and recovery (RQ1b).

- RQ2. Do racial identity dimensions (centrality, private regard, and public regard) moderate the association between RD and acute RSA reactivity (RQ2a) and recovery (RQ2b)?
 - RQ2a (RSA reactivity): For individuals high on private regard and low on public regard, we hypothesized that more frequent RD would be associated with RSA withdrawal, compared to no association between chronic RD and RSA reactivity for individuals low on private and high on public regard. We hypothesized that racial centrality would not moderate the association between RD and RSA reactivity.
 - RQ2b (RSA recovery): For individuals high on private regard and low on public regard, we hypothesized that more frequent RD would be associated with RSA augmentation, compared to no association with RSA recovery for individuals low on private regard. We hypothesized that racial centrality would not be a moderator.

Methods

Participants

Data were collected as part of a larger study of Black college students attending a large public predominantly White institution in the southeastern United States. The sample comprised 131 Black undergraduate students whose RSA was measured via a laboratory visit, 12 of which were excluded due to missing predictor values or equipment errors, resulting in a final analytic sample of 119 ($M_{\text{age}} = 19.45$, $SD_{\text{age}} = 1.42$, 76.5% female). The majority of students identified as first-year (34.5%) or sophomore (30.3%) students. A majority of the sample identified as Black/African American (84.9%), while 10.1% of participants identified as Bi-/multi-racial, .8% identified as African, and 4.2% identified as Afro-Central/South American or Caribbean Black. The analytic sample largely reported no history of cardiovascular concerns (95.8%).

Measures

Demographic measures.—Demographic measures of age, sex, highest level of parental education, and body mass index (BMI) were control variables in our analyses. Research identifies differences in RSA by age (28), sex (29), BMI (30), and parental education (as a proxy for socioeconomic status; 31). Participants were asked to identify their sex by indicating male, female, or other, and indicated highest level of education completed by their parents (mother or father) using the following options: did not finish high school, General Equivalency Diploma, Associate's degree/Vocational training or licensure, Bachelor's degree, or Master's degree or higher. BMI was calculated for each participant from self-reported height and weight using the Center for Disease Control's formula.

Racial discrimination.—The Daily Life Experiences Scale (DLE; sample $\alpha = .92$), a subscale of the Racism and Life Experiences Scale (32), was utilized to assess the frequency with which participants experienced 18 racial hassles in the past year (e.g., "In the past year, how often have you been ignored, overlooked, or not given service because of your race?").

Responses were made on a 6-point Likert-type scale ranging from 0 (*never*) to 5 (*once a week or more*). Scores were computed by averaging responses resulting in a continuous variable with higher numbers equaling greater experiences of discrimination.

Racial identity.—Three subscales of the Multidimensional Inventory of Black Identity (MIBI; 33) were used to assess private regard, public regard, and centrality. The private regard subscale (sample $\alpha = .79$) consisted of six items that measure participants' feelings about being Black and Black people (e.g., "I am happy that I am Black"). The public regard subscale (sample $\alpha = .85$) consisted of six items that measure participants' feelings about how others view Black people (e.g., "Overall Blacks are considered good by others"). The centrality subscale (sample $\alpha = .82$) consisted of eight items that measure the degree to which participants' define themselves in terms of their race (e.g., "In general, being Black is an important part of my self-image"). Responses to the MIBI are rated from 1 (*strongly disagree*) to 7 (*strongly agree*) and sub-scale scores were computed by averaging sub-scale items, resulting in continuous variables with higher numbers equaling higher private regard, public regard, and centrality.

Respiratory Sinus Arrhythmia.—Electrocardiogram data were collected using a Biopac (Goleta, CA) MP150 amplifier. Interbeat intervals were extracted from the electrocardiogram waveform in the Biopac AcqKnowledge program. The waveform and interbeat interval sequence for each participant were aligned and visually inspected for movement artifacts in CardioEdit. Edits were minimal (no more than 1% of data per file), adhering with CardioEdit editing rules. Edited interbeat intervals were then submitted to the CardioBatch program, which calculated three RSA values for each participant (baseline, stress, and recovery) using the Porges-Bohrer Method for extracting the high frequency component of each interbeat interval sequence from the .12-.4 Hz spectral frequency band-pass for adults (34). From these three RSA scores, RSA change scores were calculated for each participant. RSA reactivity was calculated by subtracting baseline RSA from stress RSA, and RSA recovery was calculated by subtracting stress RSA from recovery RSA.

Procedure

The data were from a two-part study, with part one being an online Qualtrics survey and part two being a laboratory visit. Participants were recruited for the two-part study via campus organizations, class announcements, psychology department participant pool, and flyers. Eligibility criteria for each part of the study included: 18 years or older, fluent in written and spoken English, and undergraduate student. After electronic consent, participants self-reported demographic information, past year RD, and racial identity in part one. After in-person consent, part two measured participants' RSA in response to a task. Participants must have completed part one in order to begin part two. A subset of participants completed both parts of the study. There were no meaningful differences on study variables between participants who completed the laboratory visit and those who did not (see Appendix A, which can be found online). Power analyses for the current investigation indicated that a sample size of 113 was necessary to detect a small effect ($f^2 = .15$) at a power level of .80 ($\alpha = .05$). Participants earned \$10 or participant pool credit for each part of the study. The

study was approved by the University of North Carolina at Chapel Hill's institutional review board.

During the laboratory visit, a Black research assistant administered an electrocardiogram and a White research assistant conducted a stress task. Three leads were secured to the participant's body following a modified Lead III Einthoven's equilateral triangle configuration. Participants were instructed to relax for five minutes (baseline period). Next, participants were presented with the stress task (stress period), which entailed being read a vignette about a student who experiences RD in a classroom setting (see Appendix B, which can be found online) and being asked to respond to the vignette as though they were experiencing it. This task was designed based on previous literature indicating that vignettes of RD produce acute physiological stress responses (24), and the vignette was based on data that indicates perceptions of affirmative action are common and frequent experiences for Black students (35, 36). Each participant was presented with the same vignette. Afterward, the participant engaged in another five minutes of relaxation (recovery period) and was debriefed.

Analysis Plan

Descriptive statistics and inter-correlations were first examined. Significant differences in RSA during baseline, stress, and recovery periods were then assessed to confirm the validity of the laboratory task as a measure of physiological stress. To address each research question, multivariate linear regression models (one for recovery and another for reactivity) were estimated using full information maximum likelihood (FIML) with robust standard errors to address non-normality in RSA change scores. FIML estimation was used to handle missing data. To address the first research question, the association between RD and RSA reactivity net the effects of control variables and baseline RSA score (RQ1a) and RD and RSA recovery net the effects of control variables, baseline RSA score, and stress RSA score (RQ1b) were examined. To address the second research question, we assessed whether centrality, private regard, and public regard moderated the associations between RD and RSA reactivity (RQ2a) and RSA recovery (R2b). Interaction terms were constructed using mean-centered variables. Significant interactions were probed at low (-1 *SD*), mean, and high ($+1$ *SD*) levels.

Results

Study analyses were conducted in Mplus version 8 (37). Descriptive statistics and inter-correlations are reported in Table 1. Repeated measures ANOVA indicated significant differences in RSA during baseline, stress, and recovery ($F(1.58, 187.97) = 14.04, p < .01$) after Greenhouse-Geisser corrections for violation of sphericity assumptions ($\chi^2(2) = 36.51, p < .01$). Post-hoc pairwise comparison revealed that RSA during stress was lower than RSA during baseline (M difference = $-.32$, 95% C.I. [$-.46, -.18$]; Hedge's $g = -.31$), RSA during recovery was higher than RSA during stress (M difference = $.21$, 95% C.I. [$.07, .34$]; Hedge's $g = .19$), and RSA during baseline was higher than RSA during recovery (M difference = $.11$, 95% C.I. [$.02, .20$]; Hedge's $g = .10$). Magnitude of the effect sizes

were relatively small (Hedge's $g < .20$), with the exception of the difference between RSA baseline and RSA stress (Hedge's $g = -.31$; small-moderate).

RD and RSA Reactivity and Recovery

After control variables were incorporated, RD was not associated with RSA reactivity or RSA recovery (see Table 2).

Racial Identity Dimensions and RSA Reactivity

The interaction between RD and centrality ($b = -.18$) was associated with RSA reactivity (see Table 3). In particular, more frequent RD was associated with augmentation in RSA reactivity from baseline to stress at low levels of racial centrality ($b = .25$), but not at moderate and high levels of racial centrality (see Figure 1). The interactions between RD and private regard and RD and public regard were nonsignificant.

Racial Identity Dimensions and RSA Recovery

The interaction between RD and centrality ($b = -.11$) was associated with RSA recovery (see Table 3). In particular, more frequent RD was associated with augmentation in RSA recovery from stress to recovery at low levels of racial centrality ($b = .19$), but not at moderate and high levels of racial centrality (see Figure 2). The interactions between RD and private and public regard were nonsignificant.

Discussion

Contrary to our first hypothesis, RD was not associated with RSA reactivity or recovery. This was unexpected given that RD is found to produce dysregulated physiological systems (38). However, the few studies that link RD with acute PNS stress responses find that higher lifetime RD is associated with lower PNS activity at baseline (5) and withdrawal of PNS during experimental stress tasks (20). Our findings suggest that self-reporting frequency of RD during the past year may not be as predictive of RSA as lifetime RD, which future research should examine. Nevertheless, this study is one of the first to explore the connection between past year RD and RSA reactivity and recovery. Alternatively, one study found that only discrimination attributable to threat was a significant predictor of low HRV (5), indicating that certain aspects of RD may have differential effects on physiology. However, studies that find a significant association between RD and the PNS often only examine resting rates rather than reactivity and recovery. Perhaps we were unable to detect a significant association because a majority of participants reported infrequent experiences of racial discrimination, a consideration meriting further research. Finally, perhaps the association between RD and RSA variables is not direct but can be better understood within the context of racial identity (24).

Contrary to our hypotheses, only the dimension of racial centrality had a significant role in the association between RD and acute RSA reactivity and recovery. This is surprising given that Neblett and Roberts (24) found that both private regard and public regard were associated with RSA, while racial centrality was not. However, their study examined racial identity and physiology in tandem with situational factors (e.g., race of perpetrator, variation

of task), which is different from our assessment of RD. Taken together, while we based our hypotheses on prior empirical results (24) future research is necessary to clarify the role of racial identity.

Lower racial centrality was found to be protective for both acute RSA reactivity and recovery, yet RD and RSA reactivity and recovery were not significantly associated at moderate or high levels of racial centrality. This contradicts the centrality hypothesis (39), which suggests that high racial centrality exacerbates psychological distress when experiencing RD. Yet support for this hypothesis is mixed (26, 40), previous research on the protective role of racial centrality is unclear, and few studies examine physiological outcomes. Young adulthood for Black students at a predominantly White institution is a time of increased risk of experiencing RD (4). Lower centrality may be associated with the perception that unfair treatment experienced is not race-related, as lower centrality individuals may be less likely to attribute much importance to race in their lives (23, 26, 40). Indeed, centrality was positively associated with racial discrimination in our sample. If a vignette of RD is not appraised as stressful for individuals with low racial centrality because they perceive the event as irrelevant, the RD vignette may be appraised as positive, reminding them that they are “not like other Black Americans” who would be especially affected by such an event. The current study did not examine appraisals, therefore future research should explore this possibility.

Several limitations of the present study should be noted. The analytic sample size was limited. Although we were able to detect effects, it is important that our findings are replicated. Our sample was predominantly female and sex was associated with racial identity and RSA reactivity, suggesting that future research should examine sex as a moderator. Due to the cross-sectional nature of the study, causation cannot be inferred from our results. Longitudinal research should examine how racial identity may protect Black young adults’ physiological responses from the deleterious effects of discrimination across time and examine other components of racial identity – salience and ideology. Due to the nature of the laboratory task in which vignettes were not manipulated, we cannot conclude whether responses to this task were equivalent to responses to *in vivo* experiences of RD 1) above and beyond another type of potentially stress-inducing task, and 2) outside the laboratory setting. However, results comparing RSA levels during baseline, stress, and recovery suggest that the task elicited a stress-related physiological response. The task was designed to elicit participant reactivity via the use of a White research assistant to deliver the vignette, and therefore the magnitude of participant reactivity may not be representative of reactivity to other experiences or situations. Fourth, in the Qualtrics survey, questions were not randomized and attention check items were not included. Although surveys were visually inspected for unusual data patterns, participants’ responses may have been impacted by order effects and/or inattentiveness.

Despite these limitations, this study has important implications for the health of Black young adults. The centrality of one’s racial identification may play an important role in shaping acute physiological responses to racism-related stress. While racial centrality is generally salutary, our study suggests that lower racial centrality can reduce risk in the context of RD. Thus, it is important for health professionals and college counseling centers to have an

understanding of the racial identities of their Black young adult clients who attend predominantly White institutions. They should discuss, consider, and evaluate the impact of racial discrimination experiences in the context of the significance of race with their clients, which would allow them to tailor and individualize their strategies and interventions that aim to better equip Black young adults to thrive in the face of RD.

Our investigation begins to inform understandings of racial identity as resilience-promoting factors. If race centrality does serve as a protective factor for Black young adults in the context of the environmental demands of RD, it is imperative to examine the processes through which racial centrality is associated with situational characteristics of RD in the moment. Such an understanding can help health practitioners and researchers promote the resilience of Black young adults, such that we may work to reduce cardiovascular health disparities.

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Appendix A

Only a subset of participants completed both parts of the study ($n = 131$; online survey, laboratory visit), whereas the remainder of the participants completed only the online survey ($n = 103$; no RSA data) as the laboratory portion was inaccessible due to a change in facilities in 2014. Among those who completed only the online survey, missing data ranged from .40% to 10.3% across study variables. Moreover, there were no differences between participants who completed the laboratory stress task and participants who only completed the online survey: sex ($\chi^2(1) = 3.81$; $p = .051$), parental education ($t(231) = .49$, $p = .622$), public regard ($t(220) = 1.46$, $p = .146$), racial centrality ($t(220) = -1.15$, $p = .252$), BMI ($t(123.72) = .67$, $p = .505$), and chronic racial discrimination ($t(227) = .47$, $p = .636$).

Although there was a difference in private regard between participants who completed the laboratory stress task ($M = 6.34$) versus participants who did not ($M = 5.96$; $t(151.67) = -3.05$, $p = .003$), the difference is not conceptually meaningful as both groups identically report high levels of private regard.

Appendix B

Vignette: It is the first day of class. The instructor asks you to exchange contact information and get to know the person sitting next to you. The person sitting next to you looks like me. After exchanging contact information, the person sitting next to you says, "Oh wow, you got into (college name)! So, are you actually really smart or did they just have to let a certain number of Black people in?"

List of abbreviations:

RD	Racial discrimination
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ANS	Autonomic nervous system
PNS	Parasympathetic nervous system
HRV	Heart rate variability
RSA	Respiratory sinus arrhythmia
BMI	Body mass index
RQ1	Research question 1
RQ1a	Research question 1a
RQ1b	Research question 1b
RQ2	Research question 2
RQ2a	Research question 2a
RQ2b	Research Question 2b
MIBI	Multidimensional Inventory of Black Identity
FIML	Full information maximum likelihood

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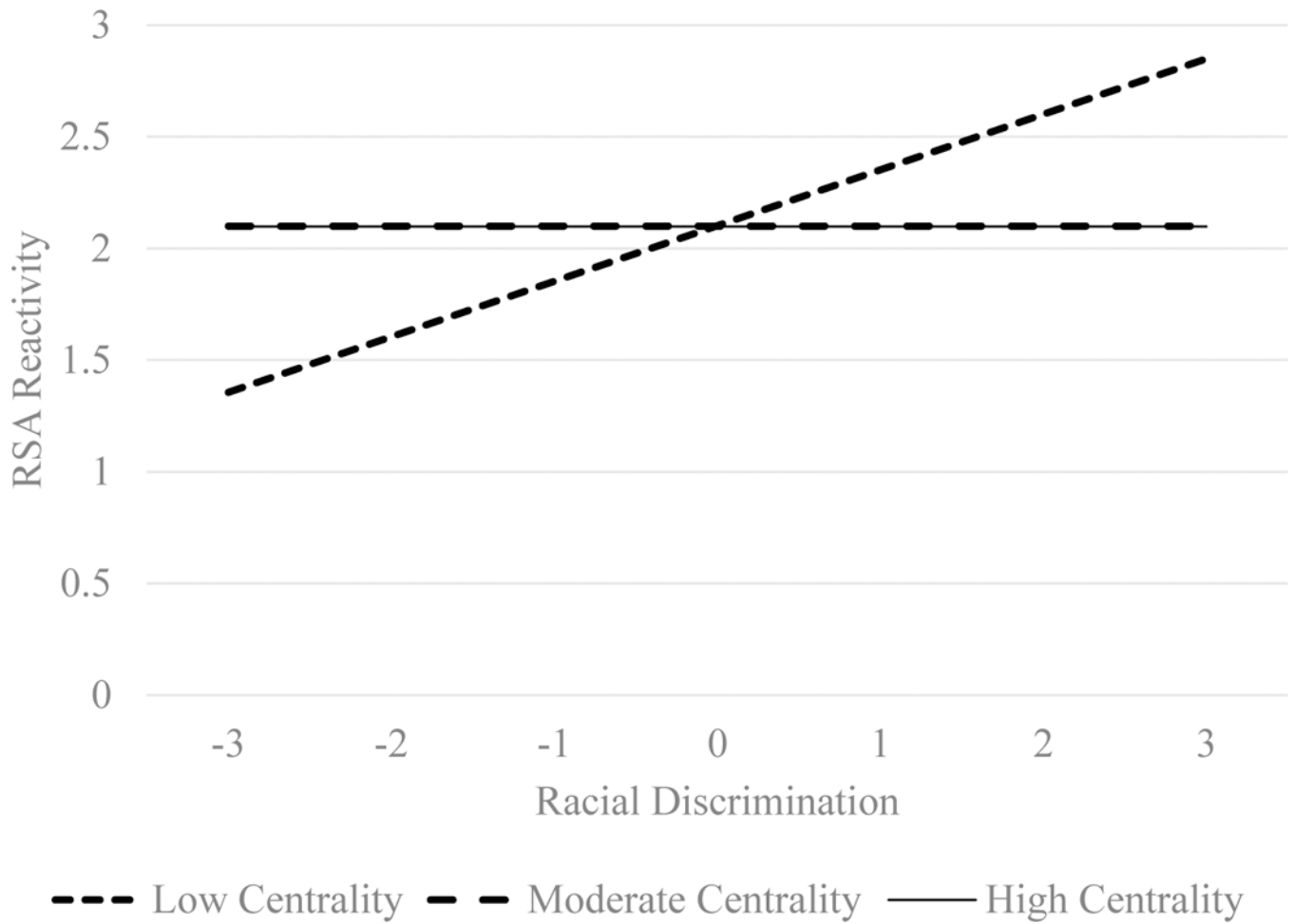


Figure 1. Moderation of Racial Discrimination and RSA Reactivity by Racial Centrality.
Note. RSA = respiratory sinus arrhythmia.

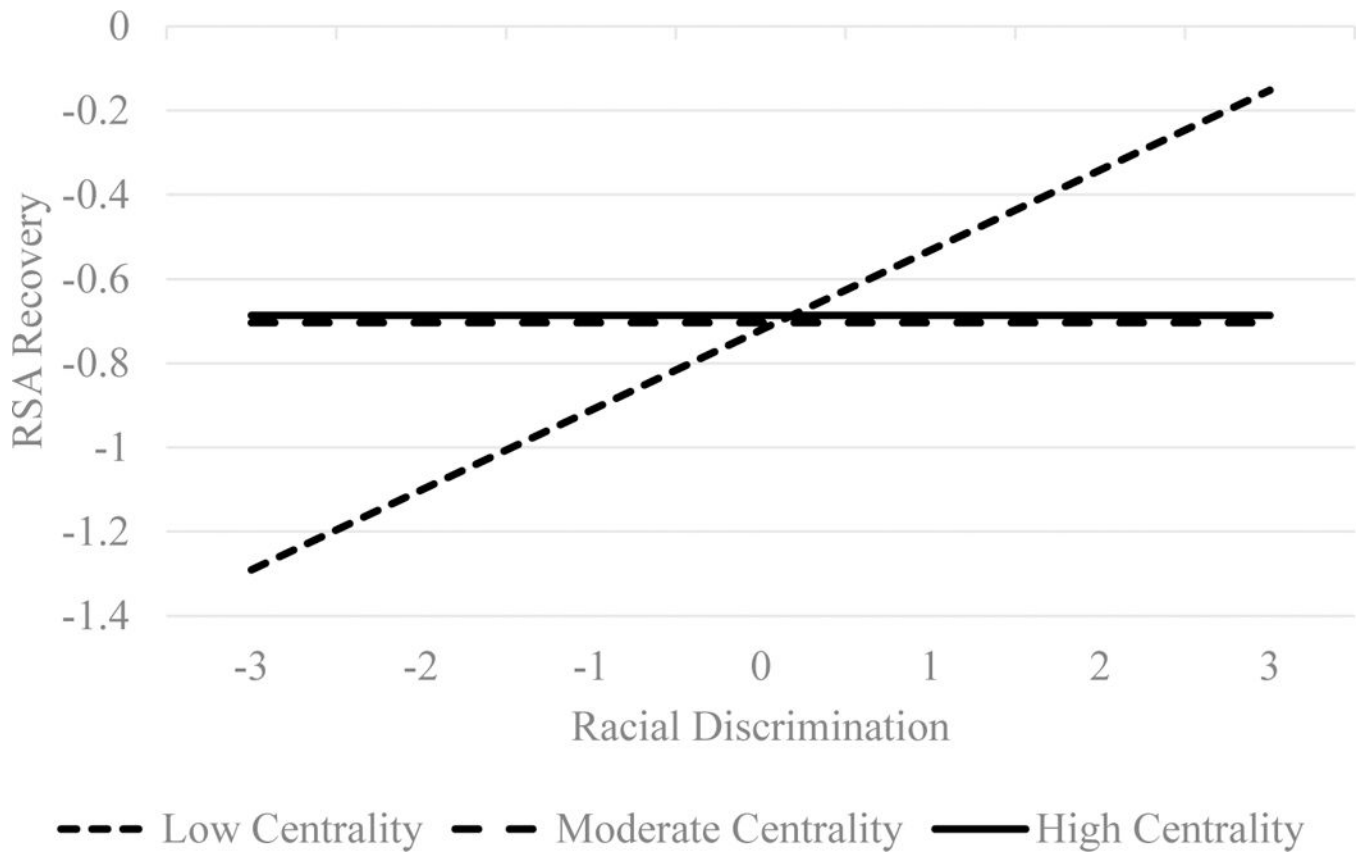


Figure 2. Moderation of Racial Discrimination and RSA Recovery by Racial Centrality.
Note. RSA = respiratory sinus arrhythmia.

Table 1

Descriptive Statistics and Intercorrelations Between Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	M(SD) or %
1. Male	--													24.2%
2. Age	.18*	--												19.43 (1.42)
3. BMI	<.01	.17	--											25.85 (5.23)
4. Parent Education Attainment	.09	-.23*	-.12	--										3.82 (1.18)
5. Racial Discrimination	.05	-.05	.12	.19*	--									1.35 (.87)
6. Centrality	-.13	-.08	.12	.10	.42*	--								5.03 (1.11)
7. Private Regard	-.21*	-.18*	-.15	.11	.05	.34*	--							6.36 (.70)
8. Public Regard	.19*	-.13	-.07	.12	-.27*	-.18*	.10	--						2.90 (1.07)
9. RSA (Baseline)	.07	-.11	.04	.02	-.08	.13	-.01	.03	--					6.48 (1.08)
10. RSA (Stress)	.20*	-.13	.00	-.03	-.08	-.09	-.11	.11	.74**	--				6.17 (1.03)
11. RSA (Recovery)	.15	-.06	.05	.05	-.06	-.11	-.12	-.04	.90**	.77**	--			6.35 (1.14)
12. RSA (Baselineto Stress)	.14	.01	-.04	-.01	-.01	.03	-.17	.08	-.39**	.29**	-.20*	--		-.32 (.74)
13. RSA (Stress to Recovery)	-.09	.04	.03	.04	.05	.01	.02	-.18*	.31**	-.24**	.40**	-.76**	--	.17 (.71)

Note.

* is $p < .05$.** is $p < .01$.

Table 2

Research Question 1: Associations between Racial Discrimination and RSA Changes

Predictors	RSA Reactivity			RSA Recovery		
	<i>b</i>	<i>s.e</i>	<i>p</i>	<i>b</i>	<i>s.e</i>	<i>p</i>
Male	.34	.14	.02	.02	.11	.86
Age	-.05	.05	.30	.03	.03	.34
Parental Education	-.02	.06	.75	.002	.05	.97
BMI	.001	.01	.96	-.001	.01	.86
RSA Baseline	-.29	.06	< .01	.71	.07	< .01
RSA Stress	--	--	--	-.71	.08	< .01
Racial Discrimination	-.04	.07	.63	.03	.04	.52

Notes. BMI = body mass index, RSA = respiratory sinus arrhythmia.

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Table 3

Research Question 2: Racial Identity Dimensions as Moderators of the Association between Discrimination and RSA Changes

	RSA Reactivity			RSA Recovery		
	<i>b</i>	<i>s.e</i>	<i>p</i>	<i>b</i>	<i>s.e</i>	<i>p</i>
Centrality						
Male	.32	.14	.02	.02	.11	.83
Age	-.04	.05	.43	.04	.03	.23
Parental Education	-.03	.05	.62	-.004	.04	.93
BMI	-.002	.01	.88	-.003	.01	.71
RSA Baseline	-.29	.06	< .01	.73	.07	< .01
RSA Stress	--	--	--	-.74	.08	< .01
Racial Discrimination	.03	.08	.67	.06	.05	.22
Centrality	-.002	.06	.98	.01	.04	.72
Racial Discrimination X Centrality	-.18	.06	< .01	-.11	.04	.01
Conditional Effects for Centrality						
-1 SD of Centrality	.25	.12	.04	.19	.09	.02
Mean of Centrality	.03	.08	.67	.06	.05	.22
+1 SD of Centrality	-.18	.11	.10	-.07	.05	.21
Private Regard						
Male	.29	.14	.03	.01	.11	.92
Age	-.06	.04	.14	.03	.03	.39
Parental Education	-.02	.06	.78	.003	.05	.94
BMI	-.01	.01	.72	-.002	.01	.81
RSA Baseline	-.29	.06	< .01	.71	.06	< .01
RSA Stress	--	--	--	-.71	.08	< .01
Racial Discrimination	-.02	.08	.84	.03	.05	.53
Private Regard	-.21	.09	.01	-.04	.07	.57
Racial Discrimination X Private Regard	-.10	.13	.43	.004	.10	.97
Public Regard						
Male	.31	.16	.05	.05	.12	.69
Age	-.05	.04	.29	.02	.04	.58
Parental Education	-.02	.06	.74	.01	.05	.79
BMI	< .001	.01	.98	< .001	.01	.99
RSA Baseline	-.29	.06	< .01	.71	.06	< .01
RSA Stress	--	--	--	-.71	.08	< .01
Racial Discrimination	-.01	.09	.88	.02	.05	.71

	RSA Reactivity			RSA Recovery		
Public Regard	.03	.07	.63	-.06	.05	.23
Racial Discrimination X Public Regard	.04	.07	.60	.06	.06	.27

Notes. BMI = body mass index, RSA = respiratory sinus arrhythmia.

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