Respiratory Sinus Arrhythmia and Heart Rate Reactivity Moderate the Relationship Between Racial Discrimination and Anxiety

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Abstract

Racial discrimination may be conceptualized as a unique, chronic stressor that impacts the lives of Black individuals with multiple associations to negative mental and physical health outcomes. High rates of racial discrimination are typically associated with increased anxiety symptomology. The present study was designed to examine if this relation may be moderated by intraindividual differences in autonomic reactivity of Black undergraduate students, as reflected by cardiac regulation. Participants completed an online questionnaire to estimate lifetime experiences of racial discrimination and current anxiety symptomology. In a subsequent laboratory visit, electrocardiogram data were collected during a task designed to mimic a real-life encounter with racial discrimination. Heart regulation responses to this challenge were measured as change from baseline estimates of heart rate and respiratory sinus arrhythmia (heart rate reactivity and ΔRSA). Multiple regression analyses showed that both heart rate reactivity and ΔRSA moderate the relationship between reported frequency of racial discrimination and anxiety symptomology, even after controlling for the effects of age, gender, and parental education. Results also demonstrated that a high baseline heart rate can account for the same relation all by itself, as it overcomes the moderating effects of autonomic regulation. These results suggest that attention to processes of physiological regulation should be central to research aimed at identifying Black emerging adults most physically and mentally affected by racial discrimination throughout development.

Keywords: racial discrimination, anxiety, autonomic nervous system, RSA, heart rate
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The status of racial discrimination as a distinct stressor in the lives of people of color has been well documented. Experiences of racial discrimination are generally persistent and pervasive, chronically and negatively impacting daily functioning on interpersonal, institutional, and societal domains throughout the lifetimes of people of color (Sellers, Caldwell, Schmeelk-Cone, & Zimmerman, 2003; Hoggard, Jones, & Sellers, 2016). In addition to augmenting cumulative stress levels, acute experiences of racial discrimination may specifically impact health by generating psychological distress related to one’s race, causing individuals to seek out potentially unhealthy mechanisms to cope with this distress, and eliciting behavioral responses (e.g., hypervigilance, avoidance) that produce changes within physiological systems (Williams & Mohammed, 2009; Carter & Forsyth, 2010). Indeed, racial discrimination has been shown to be inherently harmful to both psychological and physiological health, regardless of one’s socioeconomic status (Williams & Mohammed, 2009). While there is substantial research associating negative health outcomes with racial discrimination, few studies have examined the role of heart rate and vagal activity in this relationship. The present study seeks to examine the connection between racial discrimination and internalizing symptomology in a college population of Black individuals, while investigating physiological factors that may influence the strength and direction of this relationship. The experience of racial discrimination often activates the stress response system (Harrell, Hall, & Taliaferro, 2003); it is thus quite possible that individual differences in stress reactivity play an important moderating role in the measured association between racial discrimination and internalizing symptomology. While past research has demonstrated that discrimination is associated with negative health outcomes, variables such as centrality of racial identity (Hoggard, Jones, & Sellers, 2016), private/public regard (Neblett & Roberts, 2013), and specific coping mechanisms (Harrell, Hall, & Taliaferro, 2003) have been
shown to moderate this relation. However, little attention has been paid to how individual differences in autonomic regulation may moderate this same relationship. The current study seeks to examine ΔRSA and heart rate reactivity as moderators of the relationship between racial discrimination and anxiety symptomology.

**Racial Discrimination**

Racial discrimination may be defined as “the unequal treatment of persons or groups on the basis of their race or ethnicity” (Pager & Shepherd, 2008, p.182). Discriminatory events that are solely attributed to an individual’s racial or ethnic background present a unique and pervasive source of stress to people of color (Sellers, Caldwell, Schmeelk-Cone, & Zimmerman, 2003). Racial discrimination may be conceptualized as a chronic stressor that manifests on societal, institutional, and interpersonal levels within a variety of contexts (Williams & Mohammed, 2009). Consequently, the experience of racial discrimination may be particularly salient and persistent throughout the lives of people of color.

Historically and contemporarily, Blacks\(^1\) have faced frequent experiences of racial discrimination in the workplace, the educational and justice systems, and countless other domains of daily life (Pager & Shepherd, 2008). Recurrent experiences of negative appraisal based on one’s race may have profound effects on biological, psychological and social functioning (Clark, Anderson, Clark, & Williams, 1999). Chronic activation of this stress response system is known to negatively impact health and psychological well-being. Individual differences in the recruitment of this system may explain why some individuals are more

\(^1\) Note: This honors thesis uses the term “Black” to refer to individuals who self-identify as African, Caribbean Black, Bi-/Multi-racial, African American, and Afro-Latino.
susceptible than others to internalizing symptomology as a result of life-long experiences of discrimination (Neblett & Roberts, 2013).

As Black adolescents transition to institutions of higher education, racial identity becomes more relevant to the sense of self, causing the impact of racial discrimination to be particularly salient during this time (Harper & Quaye, 2007). This may pose specific challenges to Black students who attend primarily white institutions (PWIs), as racist attitudes, prejudices and stereotypes may contribute to an increased perception of discriminatory experiences during a time of formative identity growth (Rucker, West, & Roemer, 2010). Several studies have examined the racially discriminative experiences of Black students at PWIs. Black college students perceived more differential treatment on campus compared with Caucasian, Hispanic, and Asian students, and reported more frequent instances of being treated differently by their peers and professors than other groups (Suarez-Balcazar, Orellana-Damacela, Portillo, Rowan, & Andrews-Guillen, 2003). Furthermore, racial prejudices often lead others to identify Black adolescents as dangerous or unintelligent, leading to negative police confrontations and inappropriate discipline at school, being called racial slurs/epithets by peers, and being rejected from activities (Fisher, Wallace, & Fenton, 2000).

The personal distress generated from frequent experiences of racial discrimination throughout time poses a threat to both the physiological and psychological well being of Black young adults. A meta-analysis of available research have revealed a direct relationship between perceived discrimination and mental/physical health, such that perceptions of high discrimination are associated with poorer mental and physical health. Those analyses further demonstrate that this relationship is partially mediated by heightened stress responses, assessed through cardiovascular reactivity and self-reported psychological stress (Pascoe & Smart Richman,
Other studies have shown that lifetime racial discrimination is associated with increased anxiety, somatization, and interpersonal sensitivity (Landrine & Klonoff, 1996), as well as depression and perceived stress (Brody, Lei, Chae, Yu, Kogan, & Beach, 2014). Gibbons et al., 2012 also showed that, high and sustained experiences of racial discrimination reduces resilience capacities and increases risk-taking behavior. Finally, quantitative physiological investigations have shown that high, stable levels of perceived racial discrimination are associated with increased levels of cortisol and norepinephrine, as well as high blood pressure, even when controlling for socioeconomic status and health risk behaviors (Brody et al., 2014).

**Associations Between Racial Discrimination and Anxiety**

Experiences of racial discrimination have been linked with internalizing symptoms, particularly anxiety (Landrine & Klonoff, 1996; Williams & Mohammad, 2009; Brody et al., 2014). Anxiety can be conceptualized as an emotional response to experiences involving challenge and pressure and may be accompanied by physiological symptoms such as sweating, trembling, muscle tension, rapid heart rate, increases in blood pressure, and dizziness (Kazdin, 2000). Chronic states of anxiety have wide-ranging effects on cognitive and behavioral functioning as well as health outcomes. In the cognitive domain, anxiety is associated with mental confusion, impaired decision-making, fearful thoughts, and memory deficits. Actions like fidgeting, pacing, handwringing and other agitated actions are behavioral indicators of anxiety (Creamer, Foran, and Bell, 1995). Finally, sustained, high levels of anxiety are associated with increased risk for negative health outcomes such as hypertension (Vlachakis, Schiavi, Mendlowitz, De Guia, & Wolf, 1974), coronary heart disease (Kawachi, Sparrow, Vokonas, & Weiss, 1994), gastrointestinal complications (Huang, Mykletun, & Dahl, 2002), and chronic breathing disorders (Kunik, et al., 2009).
The literature detailing associations between racism and anxious behavior has demonstrated that frequent occurrences of racial discrimination predict higher levels of anxiety symptomology. Numerous studies examining affective, cognitive, behavioral, and physiological symptoms of anxiety have identified a positive correlation between racial discrimination and anxious behavior, even when controlling for socioeconomic risk factors and levels of generalized stress (Landrine & Klonoff, 1996; Gaylord-Harden & Cunningham, 2008; Pascoe & Smart Richman, 2009; Williams & Mohammad, 2009). Racially discriminatory interactions are inherently negative and hostile, however individual differences in autonomic stress regulation may account for differential responses to the chronic stress of discrimination among young adult Black individuals (Neblett & Roberts, 2013). While the importance of race centrality in self-identity may accentuate the perception of race discrimination for some members of the Black community, this effect on health outcomes should be examined against the background of individual differences in autonomic reactivity to stress. These distinct differences in the evaluation of negativity in events involving racial discrimination indicate that a certain event may impact individuals in several ways, and affect the formation of co-morbid physiological and cognitive anxiety symptoms. While the literature has consistently demonstrated an association between the sustained experience of racism and internalizing symptoms such as anxiety, little is known regarding the moderating role the physiological stress response system may play in this relationship.

The Role of Vagal Activity in Stressful Situations

As racial discrimination has been identified as a pervasive stressor in the daily lives of Black individuals, these events may be perceived as threatening and challenging, requiring swift response. The Biopsychosocial Model (Clark et al., 1999) proposes that individual differences in
stress activation in response to racial discrimination are formed by complex interactions between constitutional, sociodemographic, psychological, and behavioral factors. Differences in these interactions not only determine the ways in which an individual is able to cope with racial adversity momentarily, but also condition how sustained racism influences health outcomes over time. Based on this model, occurrences of racial discrimination pose a challenge to an individual’s homeostatic balance that demands recruiting physiological resources to restore stability. The present study focuses specifically on individual differences in stress activation in the face of racial discrimination, as physiological regulatory processes are herein postulated as central mechanisms that mitigate/exacerbate long-term associations between health outcomes and a cumulative life experience with racism.

As the main branch of the parasympathetic nervous system, the vagus nerve offers critical information about individual autonomic resting states and reactivity to stressful challenge. With its source in the nucleus ambiguus (brain stem medulla), the vagus nerve extends to the cardiac sinoatrial node through which it regulates heart rate (Porges, 2007). Vagal activity is also regulated by the cortico-bulbar tracts that connect the cortex to the nucleus ambiguous, and thus contribute to the regulation of vagal activity. These pathways that allows for the integration of higher-order, executive information and autonomic functions, and thus allows conscious experience to also regulate cardiac activity.

The measure of respiratory sinus arrhythmia (RSA), defined as variable heart rate linked with spontaneous breathing, reflects the cardiorespiratory rhythm produced by output from the vagal nerve onto the sinoatrial node of the heart. This contribution to heart rate regulation introduces a degree of cardiac variability, the amplitude of which is measured as RSA. This easily accessible indicator of parasympathetic input to the heart allows for the observation of
RSA suppression under external demands, functioning to maintain cardiac activity within a safe homeostatic range (Porges, 1992).

Polyvagal Theory (Porges, 1994) posits that vagal nerve input to the heart provides an index of autonomic, defensive responses to stressful environmental situations. According to this theory, the structure of the vagus has evolved in response to a “defensive world”, affording distinct capabilities that allow the individual to preserve homeostatic functioning in a challenging environment. When vagus myelination and the organization of the cortico-bulbar pathways take place over the first year of life in a supportive social context, a neural platform is established that both supports socially approved responses when challenged (Porges, 2011), and manages energetic resources through engagement or disengagement of the vagal break, without recruiting much of the more taxing sympathetic nervous system (see “Dicksonian dissolution principle” in Porges, 2009).

The mammalian vagal complex achieves homeostatic maintenance during challenge through the “vagal brake” (Porges, 2007), a mechanism by which the vagal nerve removes its input to the heart when threat is perceived. When the vagal brake is applied, the intrinsic rate of the sinoatrial node (~100 bpm) is restored, causing the heart rate to increase slightly from resting levels. Flexibility in vagal brake regulation ensures economy of metabolic resources and supports social engagement and cognitive functioning. During extended periods of challenge, the vagal brake may be applied, withdrawn, and reapplied in support of conflicting needs for homeostatic balance and ongoing demands on social/cognitive coping resources (Calkins, Graziano, & Keane, 2007). Substantial changes in RSA from baseline to challenge (ΔRSA) have often been reported in the literature as strong predictors of positive outcomes in childhood, such as greater ability to control emotions and attention and better success at school integration.
(Calkins et al., 2007). However, large changes in RSA have also been shown to be maladaptive, not only by “pausing” parasympathetic input via the vagal brake, but also by recruiting the sympathetic nervous system (SNS) to mobilize fight-or-flight behaviors (Kogan, Gruber, Shallcross, Ford, & Mauss, 2013). More recent literature, however, has explained these discrepant findings by suggesting that high vagal reactivity signals heightened biological sensitivity to context (Boyce & Ellis, 2005; Holochwost, Gariepy, Propper, Mills-Koonce, & Moore, 2014). From this perspective, a high vagal reactivity may be associated with positive outcomes in low stress/high social support environment and negative outcomes in high stress/low social support environment. Therefore large ΔRSA values may signal detrimental outcomes in the context of racial discrimination.

The Role of Heart Rate Reactivity in Stressful Situations

In addition to changes in RSA, cardiac reactivity may also be conceptualized as a physiological marker of stress vulnerability and emotional regulation. Defined as the “mean increase in heart rate observed in response to a task or stressor”, heart rate reactivity reflects the autonomic arousability of an individual, and may be a pertinent factor in considering how one responds to a stressor in the environment (Cacioppo, 1997). Early researchers in cardiac psychophysiology have demonstrated that environmentally induced anxiety is similarly linked with a marked increase in heart rate from resting levels (Deane & Zeaman, 1958; Barclay, 1961). Furthermore, they observed a great deal of inter-individual variability in the capacity of the heart to regulate itself; some individuals responded to stress with a large surge in heart rate, while others only exhibited a small increase from baseline. Thus, the impact of the autonomic nervous system on heart rate during stress is variable from individual to individual, and has clear associations with experiences of anxiety.
Furthermore, associations between heart rate and long-term negative health outcomes, such as heart disease and hypertension, have been the focus of a wealth of medical cardiovascular research for decades. One of the critical findings of this early medical research was well documented by Tinsley Randolph Harrison, revealing a distinct relationship between general anxiety and tachycardia, or pathologically high resting heart rate (Harrison, 1939). Harrison observed that his patients suffering from anxiety and nervousness almost always exhibited an overactive heart characterized by tachycardia of 80-120 beats per minute; he designated this state as “hyperkinetic syndrome”. Furthermore, Harrison noted that hyperkinetic syndrome did not typically lead to immediate cardiac failure, except through certain disturbances in the cardiovascular apparatus caused by pathology elsewhere in the body. Later research examining neurogenic factors contributing to cardiac functionality have revealed that individuals that experience high levels of anxiety and associated tachycardia may be at particular risk for heart conditions such as coronary heart disease and atherosclerosis (Rosenman & Friedman, 1973). Accordingly, high resting heart rate itself may indicate biological vulnerability to developing life-threatening heart conditions as a result of perceived stress and anxiety.

Possible Implications for Heart Rate and Vagal Activity in the Experience of Racial Discrimination

While few studies have examined the distinct role of cardiac vagal activity in the context of racial discrimination distress, research suggests that this index may be particularly useful to understand how racism negatively affects mental and physical health in the Black population (Neblett & Roberts, 2013). Specifically, these studies have confirmed that Black participants’ heart rate significantly increases in response to observing racially discriminatory images or to adding elements of racism to a neutral cognitive task (Myers, Stokes, & Speight, 1989; Harrell et
al., 2003). Social situations involving racism elicited higher heart rate reactivity in Black participants than Caucasian participants (Lepore et al., 2006). This study contributes to current research by examining the role of autonomic nervous reactivity during racially discriminatory events, as reflected through respiratory sinus arrhythmia and heart rate.

**The Present Study**

The current study seeks to examine the role of ΔRSA and heart rate reactivity in the relationship between racial discrimination distress and anxiety symptoms. Black undergraduate students participated in a two-part study that consisted of an online survey and an *in vivo* task. First, participants completed an online survey that included demographic data, as well as information about lifetime experiences of racial discrimination and current symptoms of anxiety. For the second part of the study, participants were exposed to an *in vivo* experience in the laboratory designed to stimulate psychological and physiological processes that often accompany real-life events of racial discrimination. Throughout the *in vivo* task, the heart rate of each participant was measured using electrodes to determine baseline cardiac functioning as well as cardiac response to the task. Heart rate analyses were completed after laboratory visits to derive measures of vagal activity as indexed by RSA and its relative suppression during the challenge task.

Informed by the Biopsychosocial Model (Clark et al., 1999) this study examined the role of autonomic regulation in the relationship between experiences of racial discrimination and anxiety symptomology. Given the prevalence of racially discriminatory events in the lives of people of color, as well as the known adverse effects these occurrences may have on health, it is imperative to understand more precisely the mechanisms by which racial discrimination impacts psychological health in the Black community.
Based on prior research and prevailing theories, I expect the relationship between self-reported lifetime racial discrimination and anxiety symptoms to be moderated by vagal regulation such that ΔRSA values will mitigate this relationship. Specifically, I predict that individuals with high ΔRSA will demonstrate increased levels of anxiety symptomology when exposed to frequent instances of racial discrimination throughout their lifetimes. By contrast, individuals with lower ΔRSA should exhibit less anxiety symptoms when exposed to similar levels of racial discrimination.

Additionally, I also expect heart rate reactivity to moderate the relationship between self-reported lifetime racial discrimination and anxiety symptoms. Specifically, I hypothesize that individuals with large increases in heart rate from baseline to the challenge task will exhibit high levels of anxiety when reporting frequent racial discrimination. This relation is expected to be substantially weaker for individuals whose heart rate did not change much from baseline to challenge.

Finally, I hypothesize that the moderating effect of heart rate reactivity on the relationship between self-reported lifetime racial discrimination and anxiety symptoms will be observed only among individuals with low baseline heart rates. This moderating effect is not expected among individuals with a high resting heart rate, as the well-established relationship between tachycardia and general anxiety should override this effect.

**Method**

**Participants**

Participants consisted of 210 Black-identifying undergraduate students attending a public, predominantly White/Caucasian, Southern university in the United States. For the purposes of the present study, “Black identifying” encompassed individuals who self-identified as African, Caribbean, Bi-/Multi-racial, African American, and Afro-Latino. All participants were 18 years
of age or older and were fluent in both written and spoken English. Participants were recruited through flyers, or announcements presented in campus organizations, class notices, and the university’s Psychology Department. A smaller subset of participants \((n=120)\) completed both the online portion and laboratory visit components of the study, receiving either $20 or 2.5 participant pool credits for their contribution. The present study was conducted using this subset of participants exclusively, and will be the sample currently discussed. Approximately 77% of this sample was female, reflecting a slightly greater percentage of Black female students attending the university in 2015 (about 65%), and 23% of the sample was male. The ages of participants encompassed 18 to 29 years of age, although 95% of participants were 18 to 22 years old. Approximately 84% of the participant pool identified as African American, 11% identified as bi-racial or multi-racial, and 4% identified as Afro-Latino. Less than 1% of the sample identified as Native African/African immigrant.

**Procedure**

The present study took place in two sessions—an online component and a laboratory visit. In the online session, participants completed a Qualtrics survey including information about the study, consent, and eligibility, as well as the mental health and racial discrimination measures previously discussed. Participants then scheduled a laboratory visit at least 72 hours after completing the online questionnaire. Upon arrival to the laboratory visit, participants were greeted by a Black research assistant and a White research assistant. The Black research assistant led the participant into a room in which the subject was informed about the nature of the study and his/her consent was obtained. Exchanges between the research assistants and the participants during the session were audiorecorded at the participant’s informed consent. After this initial phase, the Black research assistant applied ECG leads to the participant in a triangle configuration consistent with Einthoven’s method. The Black research assistant then instructed
the participant to relax, and try not to think of anything in particular before leaving the participant alone in the room. Upon the Black research assistant’s exit from the room, a timer set for 5 minutes began. In this period of 5 minutes, the participant’s resting baseline heart rate was recorded. After the baseline period ended, a White research assistant entered the room and conducted the challenge phase of the session in which the participant was presented with an in-vivo scenario designed to mimic an experience of racial discrimination. The stress task incorporated elements racial discrimination vignettes (Neblett & Roberts, 2013), in order to elicit physiological and psychological reactions that may be stimulated as a result of a racist encounter.

To commence the stress task, the White research assistant then reached into a box of slips of paper, telling the participant that each slip contained an everyday possible scenario. This assistant then instructed the participant to imagine himself/herself in this real life situation and to respond verbally as if he/she were actually reacting to the scenario in real life. While the assistant led the participant to believe a scenario would be selected at random, in reality, all slips of paper contained the same vignette, which was then read aloud to the participant:

“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?”

The participant was then asked to respond verbally to the scenario. While the precise language of their response was not evaluated for the purposes of the present study, the heart rate of the participant during this time was used to determine RSA during the stress period. After the participant finished replying to the situation, the White research assistant asked the subject to relax, and try not to think of anything in particular before leaving the participant alone in the room for 5 minutes. This period of time allowed the participant to recover from the stressful scenario, however this interval was not utilized in the current study. After the 5-minute recovery
period, the Black research assistant rejoined the participant in the room. At this point, the participant was debriefed regarding the aims of the study, given the opportunity to provide feedback and ask questions, and offered campus health and community resources that may help him/her further explore the topics of the study and practice self-care and wellness.

**Measures**

**Demographic Measures.** Self-reported demographic information concerning participant age, sex, and degree of parental education were gathered via Qualtrics. To assess highest level of parental education, participants were asked to indicate if at least one of their parents had earned a Masters degree or higher, a Bachelors degree, an Associates degree or vocational training, a GED, or did not complete high school. These variables were later examined as potential covariates of the predictors of interest to the present study.

**Racial Discrimination Measures.** Information concerning experiences of racial discrimination was gathered using a slightly modified online version of the Daily Life Experiences subscale of the Racial and Life Experience Scales (RaLES; Harrell, Merchant, & Young, 1997). For the purposes of the present study, this subscale evaluates the frequency of perceived racial discrimination distress of 18 daily experiences over the lifetime of the participant in order to assess cumulative risk. The RaLES requires participants to indicate how frequently these events occur on a 6-point Likert Scale, which “0” signifying “never”, and “5” signifying “once a week or more”. Preceding each item, each participant was asked, “Please think about how often you have experienced each event because of your race or racial background. How often have you experienced this in your entire life?” RaLES items included situations such as “had someone treat me badly because I was Black,” “being observed or followed in public places,” and “being insulted, called a name, or harassed”. The scores indicated
on each of the 18 subscale items are summed to obtain a score reflecting occurrences of racial
discrimination in every day life. Psychometric properties of the Daily Life Experiences subscale
of the RaLES have been well established throughout literature (Center for Disease Control,
2007) and the RaLES has been demonstrated as an effective tool for measuring racial
discrimination (Harrell, Merchant, & Young, 1997). Internal reliability for the present sample
was robust, with Cronbach’s α = .92.

Physiological Measures. During the laboratory portion of the study, research assistants
collected participant psychophysiological data utilizing electrocardiographic measures.
Electrodes were applied directly to the participant’s skin in a trilateral lead placement
configuration, correspondent with Einthoven’s triangle technique. Throughout the laboratory
visit, electrodes recorded ongoing participant heart rate by way of electrocardiogram (ECG)
technology. The ECG signal was conveyed to and recorded by a laboratory computer utilizing
the AcqKnowledge program belonging to Biopac’s MP 100 data acquisition software.

Anxiety Measures. Anxiety symptomology was assessed using the Beck Anxiety
Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The inventory utilizes a Likert-type scale
to assess 21 symptoms related to anxiety in the past month. The BAI asks participants to indicate
how “bothered” they have been by each symptom, with 0 representative of “not at all”, and 3
signifying “severely-it bothered me a lot”. BAI anxiety items include “unable to relax,” “terrified
or afraid,” and “fear of worst happening.” Scoring of the BAI was accomplished by adding the
scores of each item to compute an overall account of anxiety symptoms. The BAI has been
demonstrated as a reliable measure of anxiety within non-clinical college populations (Creamer,
Foran, & Bell, 1995). Furthermore, the BAI offers clinically relevant cutoffs to anxiety
symptoms; a score of 0-21 is indicative of low anxiety, 22-35 represents moderate anxiety, and a
score of 36 or more may be a possible basis of clinical psychopathology. Internal reliability of the present sample was robust, with Cronbach’s α = .89.

**Data Analyses.**

The Statistical Package for the Social Sciences 22.0 (SPSS) was used to analyze data and produce relevant statistics. Electrocardiogram data for each participant was converted into an interbeat-interval (IBI) file, then cleaned and edited using CardioEdit software. During the initial, 5-minute baseline phase of the experiment, RSA was averaged over each 30-second interval using CardioBatch software, in order to estimate individual values of baseline RSA. RSA during challenge was averaged over each 30-second interval during the full “challenge” phase when the participant was read the racial discrimination vignette and given time to respond to the scenario. The duration of this “challenge” period was variable from participant to participant depending on the length of the response. Utilizing these values of RSA during both baseline and challenge, I calculated individual measures of ΔRSA (an estimate of RSA suppression) for each participant by subtracting the “challenge RSA” value from the “baseline RSA” value. Heart rate measures were also computed at “baseline” and “challenge” periods for each IBI file utilizing CardioBatch. Heart rate reactivity was calculated by subtracting average heart rate during “challenge” from average heart rate during baseline.

**Missingness.** While 210 participants completed the online portion of the study via Qualtrics, 84 of these participants were unable to schedule a laboratory visit to provide physiological measures that were of key interest in the current study. Additionally, two participants were missing data due to computer malfunctions during administration of the protocol that did not enable participants’ IBI data to be correctly recorded during the stress period. Furthermore, four participants had missing data on a measure used to calculate ΔRSA
(RSA value at time of stress) due to similar computer malfunctions which affected recording during the stress period. Therefore, only a smaller subset of participants (n=120) fully completed both parts of the study. To ensure that this subset did not differ from the larger sample, an independent samples t-test was conducted. The analysis showed that the participants who completed both parts of the study did not significantly differ on the outcome variables, lifetime racial discrimination ($t(207)=1.03, p=.297, d=.145$) and anxiety symptomology ($t(208)=-.398, p=.803, d=-.055$) from those who only completed the Qualtrics portion of the study. Thus it is assumed that data are Missing at Random (MAR).

Results

To test my first hypothesis regarding the moderating role of ΔRSA in the relation between racial discrimination and anxiety, I conducted a hierarchical multiple linear regression that estimated the main effects of racial discrimination, ΔRSA and their interaction in the prediction of anxiety levels, while controlling for age, sex, and parental education. The second hypothesis assessing if heart rate reactivity would moderate the relationship between racial discrimination and anxiety, was tested in a similar way examining discrimination by heart rate reactivity as the interaction term. Finally, a third regression model was tested to determine if heart rate baseline values would constrain the moderating effects of heart rate reactivity on the relation between racial discrimination and anxiety symptomology. To evaluate this potential effect, the predictors used in the second model were again entered first, with the addition of a three-way interaction term involving discrimination, baseline heart rate, and heart rate reactivity. Thus, this model examined the independent effects of heart rate reactivity, baseline heart rate, and racial discrimination, the contribution of two-way interactions amongst these variables, and their three-way interaction in the prediction of anxiety symptomology. In all three regression
analyses, I mean-centered the variables of racial discrimination and anxiety symptomology, as well as the covariates of age, sex, and parental education. The moderating variables of heart rate reactivity and ΔRSA were left uncentered to reflect their true zero value.

To probe the moderating effects predicted in this study, all significant interactions were probed using an online tool created by Preacher, Curran, and Bauer (2006). This platform allows for examining the relations of interest at sample-specific cut-off points of the moderators. It also provides simple slopes and intercepts, regions of significance, and graphical plots of two-way and three-way interactions via R-output that allow testing the robustness and potential generalizability of moderating effects.

Descriptive Statistics.

Descriptive statistics for the study variables collected using the Qualtrics questionnaire with the larger sample are presented in Table 1 (n=210). Means and standard deviations, as well as values of Cronbach’s alpha for the variables included in analyses are presented in Table 2 (n=120). Intercorrelations amongst study variables are provided in Table 3 (n=120).

ΔRSA as a Moderator of the Relationship between Discrimination and Anxiety.

A multiple linear regression was conducted to develop a model for predicting participants’ anxiety symptomology based on their experiences with lifetime racial discrimination and ΔRSA during the in vivo stress task. This model showed a significant interaction effect between racial discrimination and ΔRSA \((p = .000)\). The two-predictor model accounted for just over 70% of the variance in participant anxiety symptomology, \(F(4, 116) = 70.61, p = .000, R^2 = .71, 95\% CI [2.95, 7.30]\). Furthermore, the model was significant even when accounting for age, gender, and parental education as covariates in a hierarchical multiple linear regression analysis, the results of which are summarized in Table 4. Probing the interaction between ΔRSA and racial
discrimination using Preacher’s Multiple Linear Regression (MLR) two-way interaction tool, I entered specific values of ΔRSA to assess the effects of racial discrimination and anxiety at conditional values of the moderator; in this instance, at the 25th, 50th, and 75th percentiles, (i.e., ΔRSA = -.1381, .3122, .8929). As ΔRSA increases, the slope related to racial discrimination and anxiety becomes more strongly positive. The simple slope is 1.1938 at the 25th percentile ($p = 0.235$, not significant), 4.6724 at the 50th percentile ($p = 0.000$), and 7.775 at the 75th percentile ($p = 0.000$). Submitting the R-syntax for the simple regression lines to the Rweb server creates a plot of these effects shown in Fig. 1. The confidence bands for observed sample values of ΔRSA are provided in Figure 2. Here, the region of significance on the moderator (ΔRSA) encompasses values -1.12 to -.017, indicating that any simple slope outside of this range is statistically significant.

**Heart Rate Reactivity as a Moderator of the Relationship between Discrimination and Anxiety.**

A multiple linear regression was conducted to develop a model for predicting participants’ anxiety symptomology based on their experiences with lifetime racial discrimination and heart rate reactivity during the in vivo stress task. This model showed a significant interaction effect between racial discrimination and heart rate reactivity ($p = .003$). The two-predictor model accounted for 67% of the variance in participant anxiety symptomology, $F(4, 116) = 60.06$, $p = .000$, $R^2 = .67$, 95% CI [-.782, -.161]. Furthermore, the model remained significant even when age, gender, and parental education were partialed out in hierarchical analyses, the results of which are presented in Table 5. The significant two-way interaction between heart rate reactivity and racial discrimination was again probed using Preacher’s MLR tool. To this end, specific values of heart rate reactivity were entered to assess the effects of racial discrimination and anxiety at conditional values of the moderator; in this instance, at the 25th, 50th, and 75th
percentiles, (i.e., reactivity = -9.997, -4.967, -1.964). As shown in Figure 3, with large increases in heart rate reactivity, the slope related to racial discrimination and anxiety becomes more strongly positive. The simple slope is 6.7043 at the 25th percentile ($p = 0$), 4.9537 at the 50th percentile ($p = 0$), and 2.4392 at the 75th percentile ($p = 0.016$). The confidence bands for observed sample values of heart rate reactivity are presented in Figure 4. Here, the region of significance on the moderator (heart rate reactivity) encompasses values -1.27 to 23.9, indicating that any simple slope outside of this range is statistically significant.

Heart Rate Reactivity: Accounting for Baseline Heart Rate.

To determine how mean baseline heart rate may impact the moderating effects of heart rate variability, a multiple linear regression analysis was conducted that, in addition to heart rate reactivity and frequency of racial discrimination, included baseline heart rate. I assessed the three-way interaction between racial discrimination, heart rate reactivity, and baseline heart rate as predictors of anxiety symptomology. This model produced a marginally significant three-way interaction effect ($p = .066$). This three-predictor model accounted for 69% of the variance in participant anxiety symptomology, $F(8, 112) = 31.07, p = .000, R^2 = .69$, and remained significant after accounting for age, gender, and parental education in a hierarchical regression analysis, the results of which are summarized in Table 6. To probe this interaction using the Preacher’s MLR three-way interaction tool, I used the 25th and 75th percentiles of heart rate reactivity (i.e., reactivity = -9.997, -1.967) and at the 25th and 75th percentiles of mean baseline heart rate (at values of -7.807, 7.339) to generate “high” and “low” indices of each moderator. For participants with low baseline heart rate, as heart rate reactivity increases, the slope related to racial discrimination and anxiety becomes more strongly positive; the simple slope is 64.108 at the 25th percentile ($p = 0.000$), and 1.307 at the 75th percentile ($p = 0.192$, not significant). These
effects are shown in Fig. 5. For participants with high baseline heart rate, the relation between racial discrimination and anxiety was strongly positive regardless of heart rate reactivity; its simple slope is 6.77 at the 25th percentile ($p = 0.000$), and 3.626 at the 75th percentile ($p = .0004$). These effects are shown in Fig. 6.

**Discussion**

Despite considerable literature linking negative health outcomes with racial discrimination, there is a paucity of research that examines the role of autonomic reactivity in this relationship. The present study evaluated the association between lifetime racial discrimination and anxiety symptoms within a population of Black college students, with consideration to the ways in which autonomic functioning may influence this relationship. In this research, autonomic functioning was measured as changes in RSA ($\Delta$RSA) and heart rate (heart rate reactivity) from baseline levels during an *in vivo* task designed to elicit physiological and psychological reactions to racial discrimination.

The results of the current study supported my first hypothesis, predicting that $\Delta$RSA would moderate the relationship between frequency of lifetime racial discrimination and anxiety symptomology. There was a significant moderating effect of $\Delta$RSA detected for individuals at the 75th (high $\Delta$RSA) and 50th (average $\Delta$RSA) percentiles, such that individuals who exhibited high $\Delta$RSA experienced elevated anxiety symptoms when exposed to frequent racial discrimination, as compared to individuals who displayed average $\Delta$RSA. Thus, with a strong autonomic response to challenge, the high $\Delta$RSA group appears to be more negatively affected by lifetime experiences with discrimination than the low $\Delta$RSA group. In fact, in the latter group, incremental experiences of lifetime discrimination did not predict heightened levels of self-reported anxiety symptoms. This analysis suggests that frequent experiences of discrimination
may place individuals who display a strong autonomic reaction to challenge are at greater risk of developing internalizing symptoms.

My second hypothesis postulating that heart rate reactivity would moderate the relationship between lifetime racial discrimination and anxiety was also supported by the data. The results demonstrated a significant moderating effect of heart rate reactivity at 25\textsuperscript{th} (high heart rate reactivity), 50\textsuperscript{th} (average heart rate reactivity) and 75\textsuperscript{th} (low heart rate reactivity) percentiles. In environments presenting infrequent encounters with racial discrimination, individuals experience few anxiety symptoms regardless of heart rate reactivity. Conversely, when faced with frequent occurrences of racial discrimination, individuals with high heart rate reactivity experienced the highest levels of anxiety. Those with average heart rate reactivity showed moderate anxiety symptomology when exposed to frequent discrimination, while individuals with low heart rate reactivity experienced minimal anxiety symptoms. These results indicate that individual differences in heart rate reactivity strongly affect the relation between experiences of discrimination and anxiety symptomology. In this second analysis we observe, as we did in the first analysis, that anxiety is lessened at higher levels of experienced discrimination for individuals whose heart rate varies less in response to challenge.

The final hypothesis tested in this research predicted that the moderating effect of heart rate reactivity on the relationship between lifetime racial discrimination and anxiety would only be observed amongst individuals with low baseline heart rate. While this moderating effect was only marginally significant, it is still important to discuss the possible implications of this finding. Participants who displayed a low baseline heart rate only reported high levels of anxiety in response to frequent experiences of discrimination when their heart rate changed substantially from baseline during the stress task (high heart rate reactivity). Those individuals displaying a
high baseline heart rate experienced elevated levels of anxiety when exposed to frequent
discrimination regardless of heart rate reactivity, consistent with medical literature linking
tachycardia with general anxiety. It appears that a high heart rate at baseline overrides any
moderating effect that may be obtained through differences in heart rate reactivity. These effects
suggest that individuals with a high baseline heart rate may not only predisposed to developing
general anxiety, but may also be vulnerable to developing higher anxiety levels when exposed to
frequent occurrences of racial discrimination during their lives. This could indicate that a
combination of high baseline heart rate and frequent lifetime racial discrimination might place
these individuals at much greater risk for anxiety symptomology and associated long-term health
outcomes than those who do not have high baseline heart rate, although further research on this
topic is needed.

Due to the large proportion of female participants (77%) in relation to male participants
assessed in this research, it is important to discuss how this sample may have impacted the
results. Literature examining gender differences in health outcomes in response to discrimination
has clearly demonstrated that Black women face regular discrimination stemming from both their
race and their gender; this added chronic stress is evident in psychological and physiological
health outcomes (Greer, 2011; Perry, Harp, & Oser, 2013). When faced with frequent
discrimination, Black women may be at a higher risk for negative health outcomes and poor
well-being than males; specifically, cancer (Taylor et al., 2007), hypertension (Roberts, Vines,
Kaufman, & James, 2008) and anxiety symptoms (Banks, Kohn-Wood, & Spencer, 2006). Thus,
it is possible that the negative health effects observed in this study were overstimulated as a
result of a female-biased sample.
Due to the pervasive, chronic nature of discriminatory experiences, it is also critical to
discuss the ability of the RaLES measure to accurately capture lifetime experiences of racial
discrimination. In this study, this scale assessed the frequency of 18 daily “microexperiences”
(i.e., daily hassles) related to race throughout the lifetime of the individual. Because this method
only includes experiences involving microaggressions that are recognizable, it may not capture the
full extent of racially discriminatory experiences, as it does not ask participants about acute
violent/traumatic, emotionally-charged instances of racism (Williams & Mohammed, 2009).
Furthermore, due to the inherent limits of recall memory, it is possible that participants were
unable to remember certain experiences that may have occurred several years ago; future studies
may seek to refine the time frame in which participants are asked to remember experiencing
racial discrimination. Thus, the current tool used to measure lifetime racial discrimination may
not comprehensively assess experiences of racial discrimination, and may not fully capture
chronic exposure to discrimination over extended periods of time.

There are several limitations to the research presented that may impact the interpretation of
results. While the in vivo task designed to mimic elements of real life situations of racial
discrimination appeared to elicit a stress response in participants (91% of participants exhibited
an increase in heart rate), it cannot be definitively concluded that this scenario fully replicates
physiological reactivity to discrimination threat in real life. As the vignette was deemed ethically
acceptable through institutional review board approval, it may not accurately reflect the highly
traumatic nature of some racist experiences. It thus remains possible that, at least for some
individuals, the laboratory challenge constructed for the purpose of the study underestimated
autonomic reactivity that may occur in real life. If this were the case, the strength of ΔRSA as a
moderator of the relation of interest may also be underestimated in the present study. Similarly,
we cannot assume that the physiological responses observed during the laboratory visit were specific to stress resulting from racial discrimination; it is possible that fluctuations in heart rate and ΔRSA could be attributed to other sources of stress, such as being in an unfamiliar laboratory with strangers or being sensitive to having one’s responses measured in the context of a psychological experiment, for example.

Another limitation to the present study regards the time frames over which autonomic responses were measured during of the stress task. Because participants were asked to respond verbally to the racial discrimination vignette, the length of the task varied from participant to participant, with the average duration being around five minutes. However, considering the transient changes normally observed in autonomic reactivity in response to stress, this time frame may in fact be too long to accurately detect changes resulting specifically from the in vivo task. While the results of the current study still seem to support hypotheses, a narrower window of time that captures the immediate response to the vignette may more precisely capture physiological changes induced by a response to racial discrimination.

Finally, the generalizability of the present research is limited to the extent that its subjects were all Black young adults enrolled in program of higher education. According to the 2015 census on educational attainment, 35% of Black young adults never attend a 2-year or 4-year college or university, leaving a large portion of this population unaccounted for in this particular research (U.S. Census Bureau, 2015). Nonetheless, in spite of the potentially protective factors black individuals attending an institution of higher education may derive from their upbringing (e.g., higher parental education, higher SES, higher living standards), the results of the current study still reveal a substantial negative impact of racial discrimination, especially for individuals prone to strong autonomic responses to challenge. Examining a non-college population of Black
young adults may extend our knowledge of how the experience of discrimination affects mental health in the wider population of Blacks.

Future research should focus on refining the current model to control for participants’ generalized stress levels while accounting for perceived stressfulness of the laboratory task, narrowing the time frame of the stress task to better isolate transitory autonomic changes specific to the challenge, and including Black participants who are not attending an institution of higher education. Because high RSA suppression and large augmentations in heart rate indicate a possible recruitment of sympathetic nervous activity, future research may complement the present study by also measuring SNS activity during the stress task. This could be accomplished non-invasively through the examination of circulating adrenaline levels through concentrations of alpha amylase in saliva samples. As the recruitment of the SNS during challenge is more metabolically costly, those individuals who display SNS input during the lab task may not only be vulnerable to problems in psychosocial adjustment (such as anxiety), but also may be at higher risk for medical conditions such as cardiovascular disease and hypertension. As the autonomic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis often operate in tandem to influence the stress response, future research may also seek to measure cortisol levels in order to assess the contribution of this related stress mechanism. Sustained levels of high HPA activity in response to stress may further exacerbate negative health effects by causing cell death in the ventral tegmental area (Tsigos & Chrousos, 2002). The consideration of high cortisol levels and the HPA axis may expand the clinical value of the current research by identifying individuals at risk for diseases that are related to dopaminergic cell death, such as Parkinson’s and Alzheimer’s (Snyder, Stricker, & Zigmond, 1985; Davis, Davis, Greenwald, Mohs, Mathe, Johns, & Horvath, 1986).
The present study offers several valuable contributions to the literature detailing the negative impact of racial discrimination within Black individuals. Most notably, this research demonstrates the importance of accounting for physiological reactivity when assessing the relationship between racial discrimination and long-term health outcomes. One advantage of focusing on autonomic reactivity is that current technological advances offer easily obtainable indices of its activity. Using such measures, the present study demonstrated that in combination with frequent discriminatory experiences, individuals displaying high autonomic reactivity are placed at a heightened risk of developing anxiety symptoms.

Additionally, young adults who experience high levels of anxiety may not currently exhibit associated physical health problems, such as coronary heart disease and hypertension, but may present these issues as they age. Thus, it may be important to distinguish at-risk individuals in order to provide them with preventative intervention to lessen the effects demonstrated within the current study, as well as health conditions that may develop later as individuals age.

In conclusion, the findings reported in the present study contribute new evidence supporting the view that racial discrimination has the potential to negatively affect mental health. Its results are also consistent with a large body of medical literature that has linked measures of cardiac functioning to clinical symptoms of anxiety. The models tested in the present research were motivated by the goal of refining our ability to identify, via easily obtainable physiological indices, those Black individuals who are most at risk for developing internalizing symptoms as a result of frequent experiences of racial discrimination. It is hoped that the present research will facilitate future prevention and intervention programs aimed at alleviating the very real health risks faced by Black individuals in our society.
References


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*p<.05, **p<.01
Table 4
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Note: Age, Sex, Parental Education, and Discrimination were centered at their means.
* $p < .05$, ** $p < .01$
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Note: Age, Sex, Parental Education, and Discrimination were centered at their means.
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Note: Age, Sex, Parental Education, and Discrimination were centered at their means.

* p < .05, ** p < .01
**Figure 1.** *Multiple Linear Regression 2-Way Interaction: Racial Discrimination x ΔRSA*
Figure 2. Confidence Bands for 2-Way Interaction: Racial Discrimination x ΔRSA
Figure 3. Multiple Linear Regression 2-Way Interaction: Racial Discrimination x Heart Rate Reactivity
Figure 4. Confidence Bands for 2-Way Interaction: Racial Discrimination x Heart Rate Reactivity
Figure 5. Multiple Linear Regression 3-Way Interaction: Racial Discrimination x Heart Rate Reactivity x Low Baseline Heart Rate

![Figure 5](image-url)
Figure 6. Multiple Linear Regression 3-Way Interaction: Racial Discrimination x Heart Rate Reactivity x High Baseline Heart Rate