

## ORIGINAL ARTICLE

# Prior exposure to racial discrimination and patterns of acute parasympathetic nervous system responses to a race-related stress task among Black adults

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## Abstract

Black adults' prior exposure to racial discrimination may be associated with their acute parasympathetic reactivity to and recovery from a new race-related stressor. Existing analytical approaches to investigating this link obscure nuances in the timing, magnitude, and patterns of these acute parasympathetic nervous system (PNS) responses. In a re-analysis of a prior study, we utilize an hidden Markov model (HMM) approach to examine how prior experiences of racial discrimination are associated with intraindividual patterns of (1) physiological states of PNS activity and (2) patterns of and variability in transitions between these physiological states. Participants ( $N=118$ ) were Black young adults (range 18–29 years;  $M_{\text{age}}=19.67$ ,  $SD_{\text{age}}=2.04$ ) who completed an online survey to index prior racial discrimination exposure, followed by an in-person lab visit during which their PNS activity in response to a race-related stress task was measured via electrocardiogram and converted into respiratory sinus arrhythmia. HMMs indicated evidence for two states: baseline and a second state representing a significant reduction in respiratory sinus arrhythmia. Most participants (93.22%) demonstrated a blunted response to the task, indicating that they did not transition from baseline during the procedure. Prior racial discrimination was not associated with HMM states or state transition parameters. Blunted physiological responses may be an important area of future investigation that could inform early life course mental and physical health screenings.

## KEYWORDS

Black adults, hidden Markov model, intraindividual variability, parasympathetic nervous system, racial discrimination, respiratory sinus arrhythmia

## 1 | INTRODUCTION

The parasympathetic nervous system (PNS) is a division of the autonomic nervous system that has important implications for disease risk (Thayer et al., 2010). Higher PNS levels index engagement in bodily rest and digest functions controlled by the vagus nerve (Doucerain et al., 2022), which in turn regulate functions of bodily systems (Williams et al., 2019). However, dysregulation of the PNS (Thayer & Sternberg, 2006; Williams et al., 2019), is associated with cardiovascular and inflammatory morbidity and mortality (Thayer et al., 2010; Thayer & Sternberg, 2006).

Exposure to social stressors is one notable psychosocial influence on PNS activity (Hill et al., 2017; Hill & Thayer, 2019). According to Polyvagal Theory (Porges, 2007), during exposure to a stressful social interaction, reductions in PNS activity occur with the removal of the vagal break (i.e., PNS withdrawal), thus allowing the person to address situational demands. The degree of PNS reactivity to stressful social interactions varies by person, with greater PNS withdrawal suggesting more sensitivity to the interaction (Doucerain et al., 2022). Following exposure to the stressful social interaction, the degree to which cardiac vagal control is restored (i.e., PNS augmentation occurs) after the stressor is no longer present is often termed PNS recovery. Beyond reactivity to the social stressor, recovery is an important indicator of how quickly an individual's body resets following a challenge so they can self-regulate and be prepared to react to upcoming stressors (Doucerain et al., 2022). This PNS flexibility is an important indicator of health status (Brook & Julius, 2000; Thayer & Friedman, 2004; Thayer & Sternberg, 2006) and the experience of acute and chronic stress can dampen PNS flexibility over time (Hill et al., 2017). Although some studies do examine both PNS reactivity and recovery to social stressors using change score methods that flatten variability in these psychophysiological responses (e.g., Neblett Jr. & Roberts, 2013; Volpe et al., 2019), intraindividual nuances in the timing, magnitude, and patterns of PNS responses to stressful social interactions remain understudied. Therefore, we perform a re-analysis of our prior study to address these limitations. The first aim of the current study is to determine if there are different physiological states of acute PNS response to a race-related stress task, and if so, examine nuances in patterns of and variability in transitions between PNS states.

Racial discrimination, defined as the negative treatment of racially minoritized individuals, is one potent social stressor for Black people in the United States (Volpe et al., 2020; Williams et al., 2019) that disrupts physiological functioning (e.g., Neblett Jr. & Roberts, 2013; Thayer & Lane, 2007; Volpe et al., 2019). In this study, we focus

on interpersonal experiences of racial discrimination, defined as negative and stressful social interactions between Black and White individuals (following Williams et al., 2003). Research often examines the physiological effects of acute exposure (i.e., measuring physiological responses to racial discrimination in the moment, as in Neblett Jr. & Roberts, 2013) and prior exposure (i.e., self-reported prior experiences occurring over a longer period on overall basal PNS levels, as in Wagner et al., 2015) to racial discrimination separately. There is also some initial evidence that increased prior exposure to racial discrimination is associated with lower PNS responses to new acute instances of stressors for Black individuals (Neukrug et al., 2022; Sheehan et al., 2019; Volpe et al., 2019; Wagner et al., 2015). However, these studies differ in how they define prior exposure to racial discrimination, with some studies measuring past year or shorter indices (as in Volpe et al., 2019; Sheehan et al., 2019) and others using lifetime measures (as in Neukrug et al., 2022; Wagner et al., 2015). Further, many of these studies use change score approaches, obscuring individual variations in PNS responses. Given the importance of the role of PNS responses to social stress and subsequent long-term health, it is imperative to better understand how prior exposure to racial discrimination influences intraindividual nuances in the timing, magnitude, and patterns of acute PNS reactivity and recovery in response to a new race-related stress task. Therefore, the second aim of the current study is to examine if prior exposure to racial discrimination is associated with patterns of and variability in transitions between PNS states.

### 1.1 | Patterns of PNS reactivity and recovery from stress

Psychophysiological models assert the importance of both reactivity to and recovery from social stress (e.g., Polyvagal Theory; Porges, 2007; Neurovisceral Integration Model, Thayer & Friedman, 2004), while the concept of vagal flexibility (Muhtadie et al., 2015) underscores the value of being able to modulate PNS activity to support behaviors appropriate to a variety of situations, ranging from innocuous to threatening. In the broadest sense, an “adaptive” pattern of PNS activity in response to a given situation will depend on the nature and severity of the challenge that the situation presents. When confronted with a social threat or challenge (i.e., a social stressor), an adaptive parasympathetic response would entail a reduction or withdrawal of PNS activity relative to baseline levels, followed by an increase or augmentation of activity once the threat was removed. However, even in this most basic account, questions arise about how to characterize

the adaptivity of different degrees of withdrawal and subsequent augmentation, including how to define an appropriate level of reactivity, how closely post-stressor levels of PNS activity should resemble baseline levels, and how quickly that recovery should occur.

These questions reflect, in part, the fact that there will typically be substantial variation in individuals' parasympathetic response to the same stressor, as suggested by multiple theories of both neurophysiological (Del Giudice et al., 2011) and parasympathetic function (Porges, 2007). For example, some individuals may exhibit modest to minimal reductions in PNS activity in response to a stressor, a phenomenon sometimes referred to as blunted withdrawal. Others may exhibit a robust reduction in PNS activity upon the onset of a stressor, but then not evidence a recovery of that activity to baseline levels once the stressor has passed.

Prior studies have employed a variety of different statistical approaches to examine individuals' parasympathetic responses during laboratory procedures designed to approximate experiences of discrimination, including multilevel (Hoggard et al., 2015), ANOVA (Neblett Jr. & Roberts, 2013), and hierarchical linear regression models (Hill et al., 2017). Regardless of the statistical method, a common approach has been to model change scores or residualized change (e.g., Casad & Petzel, 2018; Doucerain et al., 2022; Haft et al., 2022; Sosoo et al., 2022; Volpe et al., 2019; Williams et al., 2019) to examine individual differences in the magnitude of PNS withdrawal following the onset of a stressor (i.e., differences in reactivity), as well as the differences in the subsequent PNS augmentation once the stressor has passed (i.e., differences in recovery). By convention, for each individual, reactivity is typically defined as the mean within-person level of PNS activity during the portion of the procedure when the experimenter deemed the stressor to be present, minus the mean level of activity during the baseline period. Recovery may then be defined as the mean within-person level of activity for the period following the removal of the stressor, minus reactivity. With reactivity and recovery levels of PNS activity thus defined for each individual, between-person comparisons of those levels can be made.

This approach invokes two assumptions about the phenomenon of PNS reactivity to and recovery from stress. The first is that all individuals proceed from one state of PNS activity to the next in the same order: from a baseline state to a state of reactivity, and then from a state of reactivity to a state of recovery. The second is that the rate at which all individuals transition between states is precisely specified by the experimenter, who delineates, *a priori*, the temporal boundaries that define the baseline, reactivity, and recovery phases of a given procedure (Laborde et al., 2018). Given the confluence of life experiences that may

impinge upon each individual's PNS response to stress (c.f., Del Giudice et al., 2011), both of these assumptions are unlikely to be valid. Rather, there is likely to be considerable heterogeneity in the nature of individuals' parasympathetic reactions to and recovery from stress, and the rate at which individuals transition from one state to another (e.g., reactivity to recovery). This heterogeneity will reflect the concomitant variability in each individual's unique life experiences and how those experiences shaped that individual's parasympathetic function.

Hidden Markov models (HMMs) offer an alternative approach to examining patterns of PNS reactivity to and recovery from stress. These models do not use change scores or residualized change and do not require that all individuals progress from states of baseline parasympathetic function to reactivity and then recovery states, but rather allow for individual differences in this progression, such that individuals move from baseline to other states of PNS activity that are defined by the available data. Moreover, HMMs allow for individual variation in the temporal dynamics of the parasympathetic response to stress (i.e., heterochronicity), such that individuals may move from one state to another at different rates. Lastly, within the HMM framework, modeling the PNS state-conditional distributions of observed data allows for the specification of individual-specific fixed and/or random effects. Thus, the application of HMMs to the study of individuals' parasympathetic response to a race-related stress task may allow for a more nuanced understanding of individual differences in that response, and, ultimately, help explain why those individual differences are observed.

## 1.2 | Prior racial discrimination exposure and acute PNS activity

The Biopsychosocial Model of Racism asserts that Black people experience stress from exposure to racial discrimination, which can lead to dysregulated physiological responses (Clark et al., 1999). More frequent prior exposure to racial discrimination has been associated with lower resting PNS activity (Douceirain et al., 2022; Hill et al., 2017; Williams et al., 2019). Experimental evidence also indicates that acute exposure to race-related stress tasks specifically leads to acute PNS withdrawal for Black adults (Hoggard et al., 2015; Kort, 2016). However, less is known about how the amount of prior exposure to racial discrimination impacts PNS activity in response to a new acute exposure. Studies that explicitly examine recovery from race-related stressors have found mixed results. For instance, in one study, Black adults exposed to an explicit racial stereotype threat had decreased PNS recovery from baseline (Williams et al., 2019). In another study, some

Black college students exhibited PNS augmentation during recovery (Neblett Jr. & Roberts, 2013).

Theory suggests that prior exposure to racial discrimination, as a chronic stressor that Black adults experience, catalyzes a wear-and-tear on bodily systems that dampens physiological responsivity to environmental stimuli (Geronimus, 1992; McEwen & Stellar, 1993). In this way, more frequent prior exposure to racial discrimination may be associated with blunted acute PNS responses to a race-related stressor. A small body of existing literature has investigated these propositions. For instance, Volpe et al. (2019) did not find that prior racial discrimination was directly associated with PNS reactivity (operationalized via a change score subtracting composite PNS activity during a baseline resting period from composite PNS activity during a race-related stress task) or recovery (operationalized via a change score subtracting composite PNS activity during a race-related stress task from composite PNS activity during a following resting period). In contrast, Wagner et al. (2015) found that more frequent prior exposure to racial discrimination was associated with greater acute PNS withdrawal in a sample of Black and White women with diabetes who were exposed to a public speaking stressor in which they were falsely accused of shoplifting. Reliance on change scores that flatten intraindividual variability may be one reason why existing literature on prior exposure to racial discrimination and acute PNS activity has produced inconsistent results.

### 1.3 | Study aims

Previous investigations of PNS activity in response to social stressors have been limited in two interrelated ways. First, they assume that PNS activity measured in response to experimentally-induced social stressors results in separate underlying states of reactivity and recovery for all participants, and therefore analyze their data with this untested assumption in mind. Second, resultant of this assumption, they use change scores to model each of these underlying states. In the current study, we innovate by modeling Black adults' PNS activity data before, during, and after exposure to a race-related stress task in the laboratory via the specification of an HMM to introduce flexibility in the assumption of a priori states such that we can uncover important nuances in the timing of PNS activity. Therefore, the first aim of the current study is to use continuous repeated-measures data modeled using an HMM to determine if there are different physiological states of acute PNS response to a race-related stress task, and if so, examine nuances in patterns of and variability in transition from PNS states. Since the HMM approach is data-driven, we do not make specific a priori hypotheses about

the states that will be uncovered and their characteristics. However, we anticipate individual variability in states and the rates at which individuals transition between states.

Black adults' prior exposure to racial discrimination may influence their acute PNS responses to a race-related stress task. Conventional approaches have used change scores to determine if prior exposure is linked with the magnitude of PNS reactivity and recovery relative to others in a given sample. The use of an HMM approach also allows us to link prior experiences of racial discrimination with intraindividual patterns of PNS activity states. For instance, we can examine how prior racial discrimination is associated with rates of transition from one state to the next and how much of a transition Black adults experience (i.e., the degree of change in people's PNS responses in different states). Therefore, the second aim of the current study is to examine if prior exposure to racial discrimination is associated with patterns of and variability in transition between PNS states. We hypothesize that prior exposure to racial discrimination may be associated with these parameters, but we do not make directional hypotheses based on a paucity of prior research using this methodological approach.

## 2 | METHODS

### 2.1 | Participants

Data came from a larger investigation of racial discrimination and health conducted from 2014 to 2016 with Black adults from one large university in the southeastern United States (see Volpe et al., 2019 for details). This study received Institutional Review Board approval from the University of North Carolina at Chapel Hill. The analytic sample for the current study comprises 118 Black adults (range 18–29 years;  $M_{\text{age}} = 19.67$ ,  $SD_{\text{age}} = 2.04$ ) who completed both an online survey in which prior racial discrimination was self-reported and a laboratory visit in which PNS activity was measured. This number excludes seven participants who completed both parts of the study but had missing PNS data due to equipment errors.

Among those included in the analytic sample, a majority were African American (85.6%), followed by Multiracial (10.2%), Afro-Latine or Caribbean Black (3.4%), and African (0.8%). As PNS activity varies by biological sex (Yaghouby et al., 2020), we also asked participants to report their sex rather than gender. A majority of the sample (75.4%) were women. Over a third of our participants were first-year students (35.6%) followed by second-year (28.8%), fourth-year or beyond (18.6%), third-year (14.4%), and other (2.5%) which included students taking summer classes or prerequisites for nursing school. The median



highest level of parental education—one proxy for socioeconomic status for young adults (Ensminger et al., 2000; Williams et al., 2016)—was a Bachelor's degree. Most of the participants in our sample did not have cardiovascular disease or high blood pressure (94.9%). A little over half of our sample reported that they did not have a family history of cardiovascular disease or problems (55.9%), however, a little over a third did have a family history (38.1%) while the rest were unsure (5.9%). Most of our sample was not on cardiac medications or medications that caused drowsiness (99.2%) and none of our participants were on medications that affected their central nervous system. On average, participants experienced instances of racial discrimination at least once a year ( $M = 1.33$ ,  $SD = 0.86$ ).

## 2.2 | Procedure

Eligible participants first completed an online survey on Qualtrics, which included information about the study and an electronic informed consent form. Participants self-reported sociodemographic and health information as well as their exposure to prior racial discrimination using established scales. At least 72 h after completion of the online survey, participants attended a laboratory visit. During this visit, their PNS activity was measured via electrocardiogram (ECG) while the participant was sitting in a chair. PNS activity was measured across a 5-min resting period, a race-related stress task in which the participant was presented with an imagined scenario of racial discrimination, and a final 5-min resting period. The stress task involved the participant being read the scenario, asked to imagine that they were in the scenario, and asked to respond in the moment. For a detailed description of the scenario and its convergent validity see Volpe et al. (2019). For completing both parts of the study, participants were compensated with US \$20 or credit for their coursework. There were no differences in study variables between participants who completed the laboratory visit and those who did not (see Volpe et al., 2019).

### 2.2.1 | PNS data collection and processing

Biopac (Goleta, CA) MP150 acquisition hardware and AcqKnowledge software were used to transmit and record an ECG for each participant onto a laboratory computer. Single-use ECG electrodes were placed on the chest (one on the left and one on the right) and ankle (i.e., grounding electrode) in an Einthoven's triangle configuration to prevent movement artifacts. We selected respiratory sinus arrhythmia (RSA) as the PNS activity measure of interest, as RSA is a commonly used measure of PNS

responses to social stressors like racial discrimination (Neblett Jr. & Roberts, 2013) and one that especially aligns with Polyvagal Theory (Porges, 2007). To index RSA, we extracted interbeat intervals (IBIs) from the ECG waveform in AcqKnowledge and then exported the underlying IBI sequence and ECG waveform files into CardioEdit Plus (Brain–Body Center for Psychophysiology and Bioengineering, 2022). Manual editing of ECG data was performed by research assistants who first had to submit edited training files to establish reliability for independent editing via the Brain–Body Center training program. Two independent research assistants used the CardioEdit program to overlay each participant's IBI sequence onto their underlying ECG waveform. They visually inspected and corrected the underlying IBI file to match the ECG waveform and smoothed any movement artifacts in accordance with conservative CardioEdit editing rules. Research assistants needed to achieve at least 90% agreement with their edits, or the file was edited by the Principal Investigator who resolved any disagreements. Edits were minimal—no more than 1% of data per participant file was manually edited, in alignment with data quality and processing guidelines (Brain–Body Center for Psychophysiology and Bioengineering, 2022). Edited IBI files were then processed using CardioBatch Plus (Brain–Body Center for Psychophysiology and Bioengineering, 2016), which calculates RSA values using the Porges Bohrer method (Lewis et al., 2012). This method extracts the high-frequency component using the 0.12 to 0.4 Hz spectral frequency band-pass filter for adults, from which we exported RSA values for each participant in 30-s epochs. We selected this epoch length in line with previous research (Lewis et al., 2012; Neblett Jr. & Roberts, 2013). It was short enough to allow us to capture the nuanced rapid changes in RSA and long enough to capture at least one respiration cycle.

## 2.3 | Measures

Prior racial discrimination was measured using the Daily Life Experiences of Racism subscale of the Racism and Life Experiences Scale (Harrell, 1997; Lee et al., 2021). Using this subscale, participants rated the frequency with which they experienced each type of racial discrimination (18 items) in the past year on a Likert-type scale from 0 (never) to 5 (once a week or more). An example item is “ignored, overlooked, or not given service because of your race.” A total score was computed by averaging responses, such that higher numbers represent more frequent racial discrimination (sample Cronbach's  $\alpha = .93$ ).

We also included covariates in our analyses. Research suggests that demographic measures like age (Umetani

et al., 1998), sex (Koenig & Thayer, 2016), and parental education (Tonhajzerova et al., 2008) can impact RSA. Participants were asked to report their age in years. They reported their sex by indicating whether they were men or women. Lastly, participants indicated the highest level of parental education using the following options: did not finish high school, high school or General Equivalency Diploma, Associate's degree or Vocational training or licensure, Bachelor's degree, or Master's degree or higher. We coded these responses as earned at least a Bachelor's degree (0 = No, 1 = Yes).

## 2.4 | Data analytic approach

We fit an HMM to the RSA data from Volpe et al. (2019) using R and C++. The HMM has two major components: (1) a time-dependent latent state sequence (e.g., RSA states), and (2) a response variable (e.g., RSA) in which observations are independent conditional on the latent state sequence. For a thorough introduction to HMMs, see Rabiner (1989); for other contemporary applications of HMMs to health and social sciences see Kendall et al. (2024) and Williams et al. (2020, 2024).

To address our first study aim, we assumed that at any time point during the study, a participant's latent RSA state could be one of up to three states. The choice to model up to three states stems from previous literature in which a maximum of three states are defined by the three methodological portions of the task (Hoggard et al., 2015; Neblett Jr. & Roberts, 2013; Volpe et al., 2019; Williams et al., 2019). For our examination of latent RSA states, we identified a baseline state as associated with all participant observations prior to the specific moment at which each participant was exposed to a new stimulus (i.e., the stressor); this was the only state that was reliably characterized by the design. By fitting the HMM, we both estimated the parameters of the baseline state, as well as defined and characterized the other two potential RSA states, which we denoted as state 2 and state 3. States 2 and 3 need not coincide with the phases traditionally termed reactivity and recovery. We fit the HMM such that all transitions between baseline, state 2, and state 3 were possible.

All HMM parameters, including the latent RSA state sequences, were estimated via a Bayesian Markov chain Monte Carlo (MCMC) sampling routine. Estimates of all parameters are summarized by posterior medians and credible sets of the MCMC samples from their respective marginal posterior distributions. In the context of the HMM, for each participant, the progression from one latent RSA state to the next was modeled as a discrete time, discrete state Markov process with at most three RSA states

(i.e., the probability of transitioning from the current RSA state to another state in one unit of time is dependent on the current RSA state, and no past RSA states). The RSA response was modeled as a Gaussian random variable with a Gaussian random effect mean, conditional on the RSA state at a given time such that state-specific parent means and parent standard deviations were defined for each RSA state. Our HMM incorporated covariates (i.e., age, sex, and parental education) in two ways: (1) as linear fixed effects added to the mean of the response model, and (2) to specify the functional form of the transition rates governing the latent RSA state Markov process. Temporal dependencies within each participant's RSA measurements were accounted for via the latent RSA state Markov process.

As our goal was to model intraindividual variation in RSA responses relative to a participant's individual baseline RSA, we used random effects to control for heterogeneity in the baseline mean RSA values across participants, rather than assuming all participants had the same mean RSA at baseline. To allow for interindividual differences in RSA at baseline and *also* following the stressor, we modeled each participant's mean RSA as a state-conditional random effect, with the following random effect parameters: mean of the mean RSA for baseline state ( $\mu$ ), mean difference in mean RSA between baseline and state 2 ( $\alpha$ ), mean difference in mean RSA between baseline and state 3 ( $\beta$ ), standard deviation of the mean RSA for baseline ( $\sigma_1$ ), standard deviation of the mean RSA for state 2 ( $\sigma_2$ ), standard deviation of the mean RSA for state 3 ( $\sigma_3$ ).

We used output from this model to determine evidence for the number of states that were supported by the data. If either of the following criteria were met, we could infer the presence of distinct RSA states in the data. The first criterion was that the estimated mean of the random effect difference in mean RSA between baseline and state 2 ( $\hat{\alpha}$ ) or the estimated mean of the random effect difference in mean RSA between baseline and state 3 ( $\hat{\beta}$ ) was statistically significantly different from zero. The second criterion was that the estimated standard deviation of the random effect for state 2 ( $\hat{\sigma}_2$ ) or the estimated standard deviation of the random effect for state 3 ( $\hat{\sigma}_3$ ) was statistically significantly different from the estimated standard deviation of the random effect for baseline ( $\hat{\sigma}_1$ ). We determined statistical significance at the 95% level (i.e., if 95% credible sets excluded zero or were non-overlapping). After determining empirical support for the number of distinct RSA states in the data, we described the number and nature of all potential state transitions. We estimated the proportion of participants who transitioned between states. We also estimated transition probabilities for each RSA state transition (representing the rate of transitioning from one state to another in a 30s epoch) in order to describe the relative speed of transitioning between states. Lastly, we

incorporated covariates in the HMM to determine if they were statistically significantly associated with RSA, and to control for their effects on RSA.

To investigate Aim 2 regarding the role of prior racial discrimination, we used the same HMM structure, with the addition of prior racial discrimination as a predictor variable as a RSA-state-specific fixed effect. We used this modeling approach to examine if prior racial discrimination exposure is associated with variation in the magnitude of RSA in each of the three potential RSA states (baseline:  $\delta_1$ , state 2:  $\delta_2$ , state 3:  $\delta_3$ ). Additionally, to determine if prior racial discrimination affects variability in transition rates between RSA states, we assessed the association of the predictor variable with the transition probabilities between states. We determined if the effect of prior racial discrimination on state-specific RSA magnitudes and state transition rates were statistically significant at the 95% level (i.e., if 95% credible sets excluded zero).

On the second author's website (<https://ebkendall.github.io/research.html>), find the **Supplemental Appendix** for a full description of the HMM parameterization and statistical descriptions.

### 3 | RESULTS

#### 3.1 | Aim 1: Physiological states of acute PNS responses to a race-related stress task

Our first aim was to determine support for different physiological states of acute RSA response to a race-related stress task in our sample, and, if supported, to examine nuances in patterns of and variability in transitions to/from RSA states. Results of the fitted HMM to determine support for different physiological states are presented in **Table 1**. At the 95% level, only one RSA state was statistically significantly different from the baseline RSA state according to its estimated mean of the random effect

difference in mean RSA from baseline ( $\hat{\alpha} = -.66$ ). This indicated the presence of a state 2, which is distinct compared to the baseline state. According to **Table 1**, the baseline state was characterized by an estimated parent mean RSA of 6.89 (and parent standard deviation  $\hat{\sigma}_1 = 1.11$ ) in the current sample. State 2 was characterized by a 0.66 unit statistically significant reduction in parent mean RSA compared to baseline parent mean RSA (and parent standard deviation  $\hat{\sigma}_2 = 0.62$ ). In terms of covariates, age and parental education did not have a statistically significant effect on the mean RSA. Sex did have a statistically significant effect on the mean RSA, however, such that the RSA for women is 0.60 units lower ( $\hat{\gamma}_2$ ).

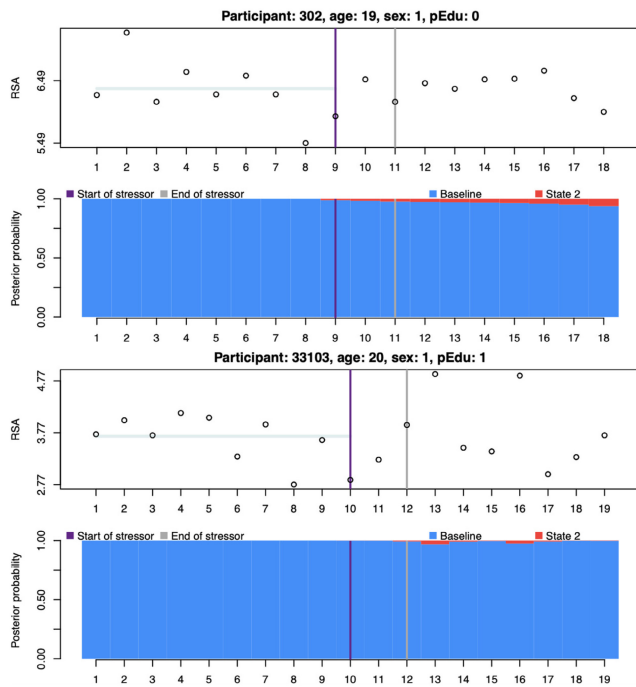
Given the presence of the two states (baseline, state 2), there exists two categories of state transition patterns that are of particular interest: (1) remaining in baseline for the duration of the task, and (2) transitioning from baseline to state 2 at any point during the experiment. Extending the second pattern into sub-categories, we are also interested in: (2.a) transitioning from baseline to state 2 and remaining in state 2 for the rest of the task, and (2.b) transitioning from state 2 back to baseline. Examining the estimated proportion of participants in the sample with these transitions indicated: (1) 93.22% of participants ( $n = 110$ ) remained in baseline, (2) 6.78% ( $n = 8$ ) of participants transitioned from baseline to state 2, (2.a) 1.69% ( $n = 2$ ) of participants remained in state 2 for the rest of the task after transitioning from baseline, and (2.b) 5.08% ( $n = 6$ ) of participants transitioned from state 2 back to baseline.

A more nuanced summary of the patterns of transitions among RSA states is provided by examining the posterior probability distribution of each participant's latent RSA state sequence (i.e., how likely is the participant in each state at each instance in time). To illustrate, **Figures 1–3** present the observed RSA time series and the corresponding, fitted posterior probability distributions of the latent RSA states, for two participants each. Recall from before that the results indicated state 2 is statistically significantly

**TABLE 1** Hidden Markov model with covariates.

Random effect parent mean parameters			Fixed covariate effects on mean RSA		
$\mu$	$\alpha$	$\beta$	$\gamma_1$ : age	$\gamma_2$ : sex = 1	$\gamma_3$ : parental education = 1
<b>6.89*</b>	<b>-.66*</b>	-.34	-0.09	<b>-0.60*</b>	-0.03
Random effect standard deviations					
$\sigma_1$	$\sigma_2$		$\sigma_3$		
1.11	0.62		0.96		

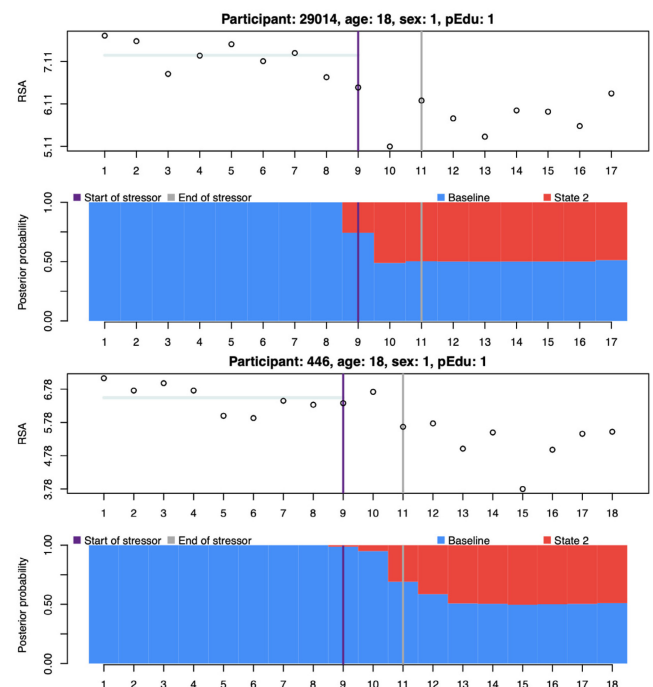
*Note:* The posterior medians of the RSA response parameters from the fitted HMM with all covariates. The bold\* is an indication that zero is in the 0.025 right or left tail region of the marginal posterior distribution for the parameter (not applied to the standard deviation terms). Sex is coded such that 1 indicates women. Level of parental education is coded such that 1 indicates if the participant's parent earned a Bachelor's degree or higher. The age covariate is centered, thus its parameter estimate is interpreted with respect to a change from the sample mean age.



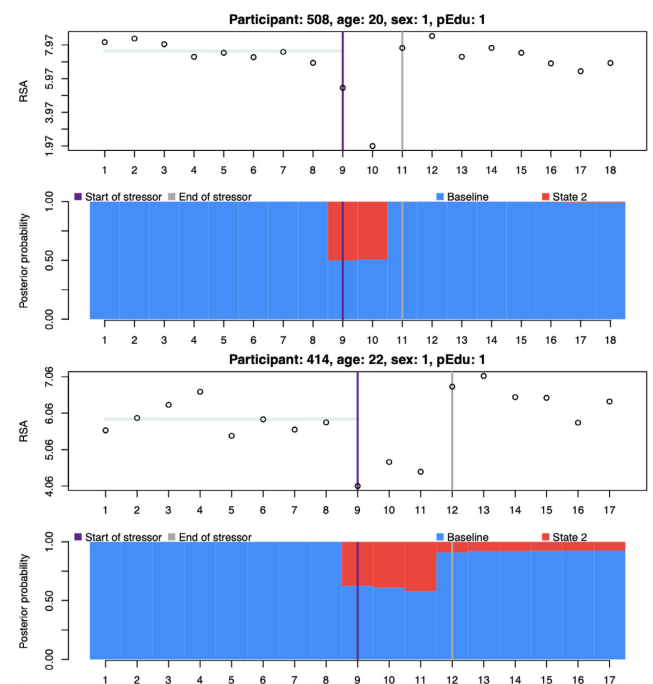
**FIGURE 1** The RSA time series and posterior probability distributions for two participants who were classified as having a blunted response from the fitted HMM with all covariates. The posterior probability distribution is color coordinated according to each of the states. Each time point has an associated vertical bar where the relative size of each color corresponds to the posterior probability of that state (analogous to a pie chart). The light blue horizontal line in the time series corresponds to the empirical mean RSA for that participant during the baseline period. HMM, hidden Markov model; RSA, respiratory sinus arrhythmia.

different from baseline, whereas state 3 showed no statistically significant difference from the baseline RSA state. As a result, these figures present results where any transitions to and/or from state 3 are assumed to be transitions to and/or from baseline (i.e., state 3 is treated as equivalent to baseline). Figures 1–3 correspond to three general categories of PNS responses to racial stressors (namely categories 1, 2.a, and 2.b, respectively, from above) that we inferred from surveying such plots for all 118 participants. Note that per the study design, participants' RSA state sequences are fixed in baseline for all time points before the racial stressor occurs, and so the posterior probability of the baseline state is always one at these time points. In the non-blunted response category, following an initial shift in RSA above or below a participant's random effect baseline mean, some participants' RSA values never returned to baseline, as observed in Figure 2, whereas other participants do exhibit a shift back to baseline RSA values, as observed in Figure 3.

Furthermore, we examined how quickly or slowly participants transitioned from state to state (i.e., the transition



**FIGURE 2** The RSA time series and posterior probability distributions for two participants who were not classified as having a blunted response (all else same as in Figure 1). RSA, respiratory sinus arrhythmia.



**FIGURE 3** The RSA time series and posterior probability distributions for two participants who were not classified as having a blunted response (all else same as in Figure 1). RSA, respiratory sinus arrhythmia.



rates). Transitioning out of baseline to state 2 occurs at a rate of 0.0149 per 30s and transitioning out of state 2 back to baseline occurs at a rate of 0.1979 per 30s. The probability of remaining in baseline in a given 30s epoch is 0.9851 and the probability of remaining in state 2 in a given 30s epoch is 0.8021.

### 3.2 | Aim 2: The role of prior racial discrimination exposure

Table 2 presents the estimated parameters for the HMM which includes prior racial discrimination as a predictor variable. For all parameters associated with prior racial discrimination, the 95% credible sets contained zero, indicating a lack of evidence that prior racial discrimination exposure is associated with (1) the mean RSA in a given state ( $\delta_1$ ,  $\delta_2$ ,  $\delta_3$ ), or (2) the speed at which individuals transition from state to state.

## 4 | CONCLUSIONS

The current study advances the literature on acute PNS stress responses in two notable ways. Existing experimental research uses change scores or statistical approaches that assume participants experience three separate physiologically distinct states during the task period: a baseline period, a reactivity period, and a recovery period. We

utilized an HMM approach that allowed us to test if this assumption of three distinct states held, and to model patterns of intraindividual variability in states in our sample of Black young adults using a race-related stress task. Second, we linked two bodies of literature—one on self-reported prior racial discrimination and another on acute physiological responses to racial discrimination stress—by examining if prior exposure to racial discrimination impacts nuances in acute PNS responses to a race-related stress task. We used the HMM approach to examine if prior experiences of racial discrimination were associated with intraindividual patterns of states and state transitions.

Our results indicated strong support for only one additional PNS state beyond a baseline period. This second state was defined by PNS withdrawal, consistent with a stress reactivity response (Porges, 2007). At the same time, only a very small percentage of participants in the current sample transitioned out of baseline and to this reactivity state (i.e., state 2). These findings could suggest a high prevalence of participants being physiologically unresponsive, a high prevalence of modest to minimal changes in PNS activity, or some combination of the two. Importantly, there were no significant differences in the ratings of the stressfulness of the task between those who did or did not transition from baseline ( $t(116) = -0.26$ ,  $p = .796$ ), suggesting that the response is unlikely to be a methodological limitation of the task itself. The observed result most closely corresponds with what has been characterized as a blunted PNS response to acute stress, which has received increasing attention in the literature as a state of physiological dysregulation with poor health consequences (Carroll et al., 2012). A blunted response signals an inability to flexibly mount a physiological response to a stressor due to lack of successful integrated regulation of the peripheral nervous system, brainstem, and brain regions such as the hypothalamus and limbic-prefrontal junction (Carroll et al., 2017; Ginty et al., 2013). In line with allostatic load theory (McEwen & Stellar, 1993), this blunted physiological response could index the wear-and-tear on physiological systems resulting from chronic stress exposure (O' Riordan et al., 2023). Most research on acute PNS responses to stress has focused on support for the reactivity hypothesis—the assertion that hyper-reactivity, defined as prolonged or exaggerated cardiovascular reactivity (Obrist, 1981), to stressors negatively impacts health (Phillips et al., 2013). However, hypo-reactive responses such as blunting have received less consideration. Existing attempts to understand acute PNS responses to stress often quantify the magnitude of a reactivity response along a continuum, yet the numerical quantification of a prolonged or exaggerated PNS response is currently unclear (e.g., Volpe et al., 2019),

TABLE 2 HMM with prior racial discrimination as a predictor.

Random effect parent mean parameters			Fixed covariate effects on mean RSA		
$\mu$	$\alpha$	$\beta$	$\gamma_1$ : age	$\gamma_2$ : sex = 1	$\gamma_3$ : pEdu = 1
6.92*	-.65*	-.26	-0.09	-0.62*	-0.05
Random effect standard deviations			State-dependent prior racial discrimination effects on mean RSA		
$\sigma_1$	$\sigma_2$	$\sigma_3$	$\delta_1$ : baseline	$\delta_2$ : state 2	$\delta_3$ : state 3
1.11	0.91	0.72	-0.09	0.31	0.18

Note: The prior racial discrimination variable is centered, thus the parameter estimates are interpreted with respect to a change from the sample mean prior racial discrimination. The note indicates that the bold\* is an indication that zero is in the 0.025 right or left tail region of the marginal posterior distribution for the parameter. These are Bayesian statistics, so traditional significance (i.e.,  $p = .XXX$ ) does not apply. In the method it indicates that statistical significance is at the 95% level (i.e., if the 95% credible sets excluded zero or were non-overlapping).

Abbreviation: HMM, hidden Markov model.

thereby limiting the utility of this approach for clinical practice. The current study allowed us to consider what proportion of PNS responses in the given sample were consistent with a blunting response based on a number of latent states and transitions (or lack thereof) between them. In this way, our approach is much more consistent with existing theory on hyporeactivity and hyperreactivity, because our conceptualization of these states is based on intraindividual variability and the degree to which distinct states are supported in the data, rather than creation of change scores based on assumed underlying states. Therefore, the results of the current study suggest that acute PNS blunted responses observed by the majority of participants during a race-related stress task may be a common physiological pattern for Black young adults that merits further investigation.

Using the HMM approach also sheds light on patterns of acute PNS transitions during the task based on the two observed states rather than the potential three imposed states (i.e., baseline, reactivity, recovery). In the current study, we did not have strong evidence for a separate, distinct PNS state of augmentation that would correspond to the traditional concept of a separate period of recovery during the task. However, after observing the states present in the data, we used state transitions to more accurately match theory about hyperreactive and recovery patterns, and describe their prevalence for this sample and this task. We found that very few participants exhibited what would be considered a hyperreactivity pattern (i.e., staying in state 2 characterized by decreased PNS and remaining in that state) or a recovery pattern (i.e., transitioning from state 2 back to baseline). Describing the prevalence of state transitions allows us to characterize recovery more accurately as a return to baseline, compared to a unique state. In this conceptualization, transitioning from state 2 back to baseline is most aligned with recovery as a return to homeostasis (McEwen & Stellar, 1993; Porges, 2007) rather than a separate quantification of a period imposed by the task methodology. Once again, the high proportion of participants in our current sample (which was comprised exclusively of Black young adults) with blunted responses to stress raises the question of whether these responses represent a form of physiological dysregulation particularly common among Black individuals.

We predicted that more frequent exposure to prior racial discrimination would be associated with acute PNS states, in accordance with allostatic load theory and the weathering hypothesis (Geronimus, 1992; McEwen & Stellar, 1993). However, prior racial discrimination was not associated with the probability of being in a given state or the rate of transition from state to state. At first glance, it may appear that the findings of the

current study mirror those of Volpe et al. (2019), with no association between prior racial discrimination and patterns of RSA reactivity or recovery in the same sample. However, the HMM found unambiguous support for two latent states, while the dominant approach has been to calculate change scores between researcher-imposed portions of a stress task without questioning if these portions represent distinct physiological states. In this way, the HMM approach remains a more precise and valuable way to understand the association between prior racial discrimination and acute PNS responses to a race-related stress task.

Prior racial discrimination may not have been associated with acute PNS states for several reasons. First, we did not consider how these prior experiences were appraised or coped with, despite the fact that these factors can change emotional (e.g., Neukrug et al., 2022) and physiological (e.g., Clark, 2003; Clark et al., 1999) responses to racial discrimination. For instance, in one experimental study with Black young adults, Sosoo et al. (2022) found that participants exposed to violent racial discrimination conditions versus neutral or non-violent racial discrimination conditions did not exhibit significant PNS changes, positing that this null effect may be indicative of an active coping response. In this way, perhaps frequency of exposure itself is not the most important aspect of these prior experiences that determines PNS responses to new acute exposures. Examining appraisal and coping moderators and mediators may illuminate the contexts and processes in which prior racial discrimination exposure does or does not impact acute PNS responses. Second, there may not have been concordance between the types of prior racial discrimination that were self-reported using an established measure and the acute race-related stress scenario to which participants were exposed in the laboratory. The Daily Life Experiences of Racism measure of prior racial discrimination includes events that are relatively subtle (i.e., “being ignored or overlooked”), while the acute scenario involved a very overt statement of an assumption due to one’s race (i.e., getting into a prestigious college “just because you are Black”). Different types of racial stressors may be coped with or responded to differently (e.g., Volpe et al., 2020, 2021), and therefore the potential misalignment between the type of prior experiences that were self-reported and the acute stressor that was experienced may be why we did not observe an association. Third, it may be that young adults, being relatively early in the life course, may have relatively less variability in PNS responses to stress (Yang & Kozloski, 2011) or report relatively less exposure to racial discrimination (White et al., 2020), compared to older populations. Therefore, we may not yet be seeing the full scale of accumulative effects of exposure to racial discrimination on physiological dysregulation.

The current study is not without limitations. First, it did not utilize an experimental manipulation of exposure to racial discrimination. Although the race-related stress task was a valid and acceptable way to measure Black young adults' acute responses to exposure to race-related stress (see Volpe et al., 2019), differences in the mode, delivery, or nature of the task may impact the physiological states and their transitions that were estimated in the current study. Although most participants exposed to the race-related stress task exhibited a blunted PNS response, it is possible that this could be ubiquitously observed among others who do not report exposure to race-related stress. At the same time, a design in which acute PNS responses are observed over time in response to the same stimuli for all participants may be especially optimal for an HMM, as one goal of the model is to evaluate evidence for a number of potential states and describe intraindividual patterns and variability in this intensive repeated-measures data sequence. Second, the sample is composed of college-attending Black young adults. Although we did not observe differences in results by age or parental education (i.e., proxy for socioeconomic status), or any differences between those who completed both visits compared to only the online survey, it may be that the context of being a Black young adult attending a predominantly white institution in which racial stress may be especially heightened (Hope et al., 2015) would impact the proportions and patterns of acute PNS states of responses to a race-related stress task present in our data. Third, we did not examine the role of other potential predictive factors such as coping styles and mental health on patterns of physiological responding, given the overwhelming proportion of blunted responses observed in the current non-clinical sample. However, as these factors may impact psychophysiological regulatory capacity (Brownlow et al., 2023), future research could purposely sample Black young adults with different coping styles or mental health statuses in order to examine if they predict different PNS states and transitions.

In conclusion, our results suggest that use of an HMM approach to model acute PNS responses to a race-related stress task is one innovative way to understand nuanced patterns of physiological dysregulation that may already be evidenced by young adulthood, beyond traditional change score approaches. Blunted physiological responses may be an important area of future investigation that could inform early life course clinical mental and physical health screenings. The HMM was also able to provide evidence of sex differences in PNS responses to an acute race-related stress task among Black young adults, such that women had lower average RSA than Black young adult men. These differences will be important to consider when determining what levels of physiological reactivity

(or lack thereof) are important for cardiovascular disease risk.

## AUTHOR CONTRIBUTIONS

**Vanessa V. Volpe:** Conceptualization; data curation; funding acquisition; investigation; methodology; project administration; resources; supervision; writing – original draft; writing – review and editing. **Emmett B. Kendall:** Data curation; formal analysis; methodology; software; visualization; writing – original draft; writing – review and editing. **Abbey N. Collins:** Conceptualization; investigation; project administration; writing – original draft; writing – review and editing. **Matthew G. Graham:** Conceptualization; writing – original draft; writing – review and editing. **Jonathan P. Williams:** Conceptualization; funding acquisition; project administration; software; supervision. **Steven J. Holochwest:** Conceptualization; supervision; writing – original draft; writing – review and editing.

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## CONFLICT OF INTEREST STATEMENT

Authors have no conflicts of interest to disclose.

## DATA AVAILABILITY STATEMENT

De-identified study data may be made available for research purposes via a data use agreement by emailing the first author. Code to replicate the results can be found at <https://ebkendall.github.io/research.html>.

## ETHICS STATEMENT

This is a secondary data analysis using data from a study that received institutional review board approval from the University of North Carolina at Chapel Hill (PI: Vanessa Volpe).

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

**Appendix S1.** Supplemental Appendix.

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