



Race and ethnic variation in college students' allostatic regulation of racism-related stress

Jacob E. Cheadle^{a,1}, Bridget J. Goosby^a, Joseph C. Jochman^b, Cara C. Tomaso^c, Chelsea B. Kozikowski Yancey^c, and Timothy D. Nelson^c

^aDepartment of Sociology, University of Texas at Austin, Austin, TX 78712; ^bDepartment of Sociology, University of Nebraska-Lincoln, Lincoln, NE 68588; and ^cDepartment of Psychology, University of Nebraska-Lincoln, Lincoln, NE 68588

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Racism-related stress is thought to contribute to widespread race/ethnic health inequities via negative emotion and allostatic stress process up-regulation. Although prior studies document race-related stress and health correlations, due to methodological and technical limitations, they have been unable to directly test the stress-reactivity hypothesis in situ. Guided by theories of constructed emotion and allostasis, we developed a protocol using wearable sensors and daily surveys that allowed us to operationalize and time-couple self-reported racism-related experiences, negative emotions, and an independent biosignal of emotional arousal. We used data from 100 diverse young adults at a predominantly White college campus to assess racism-related stress reactivity using electrodermal activity (EDA), a biosignal of sympathetic nervous system activity. We find that racism-related experiences predict both increased negative emotion risk and heightened EDA, consistent with the proposed allostatic model of health and disease. Specific patterns varied across race/ethnic groups. For example, discrimination and rumination were associated with negative emotion for African American students, but only interpersonal discrimination predicted increased arousal via EDA. The pattern of results was more general for Latinx students, for whom interpersonal discrimination, vicarious racism exposure, and rumination significantly modulated arousal. As with Latinx students, African students were particularly responsive to vicarious racism while 1.5 generation Black students were generally not responsive to racism-related experiences. Overall, these findings provide support for allostasis-based theories of mental and physical health via a naturalistic assessment of the emotional and sympathetic nervous system responding to real-life social experiences.

race and ethnicity | health disparities | discrimination | stress reactivity | allostasis

Racism in the United States shapes interracial social interactions (1) and, mirroring society-wide trends in the racialization of American politics (2, 3), the situation may be worsening with rising racial animosity (4, 5). Consequently, racism-related stress is hypothesized to contribute to widespread minority health disparities (6) and Black/White mortality differentials on a scale sufficient to influence election outcomes (7). One possible explanation for these disparities is that race-related stress increases negative emotions and physiological wear and tear via modulation of the sympathetic-adrenal-medullary (SAM) axis, hypothalamic-pituitary-adrenal (HPA) axis, and immune system (8). Up-regulation of these systems, often referred to generically as “the stress process,” is the outcome of the brain’s predictive modeling and regulation of the body’s energetic needs, or allostasis (9). Here, race-related stress is thought to increase energy demands as the brain prepares the body to deal with threats by marshaling oxygen, glucose, and other energetic mediators (10). Consequently, researchers have argued that ongoing allostatic up-regulation of the autonomic and endocrine systems, combined with immune activity modulation, in response to racism-related threats is physiologically taxing and over time increases

vulnerability to chronic disease (11, 12). For example, downstream physiological adaptations to a body energized on high alert are thought to increase risks for excess adiposity, hypertension, diabetes, and cardiovascular disease (13–15), traditionally the leading cause of death in the United States.

Despite the theoretical importance of regulatory allostatic processes for understanding racial health inequities, racism-related physiological responding has not been directly measured in real time in the real world. Conducting these assessments is challenging because the timing of many socially mediated experiences, such as racism-related incidents, occur in “social time”; that is, they are dependent upon actions beyond the control of the individual, are largely unobserved, and are highly variable and stochastic. Acute physiological changes are thus responses to temporally variable stimuli and are modulated on precise time scales in the order of seconds (i.e., SAM) and minutes (i.e., HPA). Although systematic and metaanalytic reviews find evidence that discriminatory experiences are correlated with mental and physical health both early (6) and later in life (16), most studies are cross-sectional, retrospective, and rely on behavioral and psychological self-reports, even when employing temporally sensitive methodologies (17). Although a few recent studies in natural settings incorporating biomarkers consistent with models of allostasis have begun filtering into the literature (18), their measurement strategies are not time-synchronized

Significance

Racial resentment, antiimmigration sentiment, and hate crimes, including on college campuses, have increased recently. Although African American (70%) and Hispanic/Latinx people (56%) report considerable discrimination exposure, there remains a critical need for research examining the pathways through which racism-related stress affects health. Here, we developed an ecological, intensive longitudinal design leveraging an independent biosignal of stress arousal collected from a wearable sensor. Our method documents both increased simultaneous negative emotion and stress arousal among African American, Latinx, and African students in response to racism-related stress. These findings provide support for allostasis-based theories of mental and physical health via a naturalistic assessment of emotional and physiological responding to real-life social experiences.

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¹To whom correspondence may be addressed. Email: j.e.cheadle@utexas.edu.

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with race-related experiences. These studies provide post hoc evidence of allostatic process modulation, but do not measure allostatic regulation concurrently as it transpires *in situ*.

Our study was designed to address these limitations and to directly test allostatic modulation of the sympathetic component of the SAM response to racism-related stress. To these ends, we developed a prospective protocol capturing events throughout the day, including the timing of racism-related experiences of perceived interpersonal discrimination, rumination on racism, and vicarious racism exposure, as well as negative emotions. Our design also included a wearable device that continuously tracked SAM activity using an electrodermal activity (EDA) sensor, a direct measure of the sympathetic nervous system (SNS) division of the autonomic nervous system that indexes affective arousal. This approach facilitated approximate time-synchronization of racism-related experiences with the SNS-mediated first-stage allostatic stress-response pathway. We were therefore able to operationalize two key aspects of stress-response dynamics temporally coupled with racism-related stressors: Negative emotion and SNS arousal. To our knowledge, this study provides a temporally coregistered and ecologically embedded assessment of the dynamic links between race-related stressors, negative emotions, and the SAM-mediated SNS component of a stress response among individuals of diverse ethnic backgrounds.

Racism-Related Stress and Negative Emotion

Racism is thought to foster racial disparities in health via inequities at multiple levels of social organization (11, 19). Because racism is a structural feature of the United States, it has influenced institutional, organizational, and interpersonal facets of life that together increase exposure to adverse circumstances and experiences (20). We focus on three racism-related conditions operating at the individual level. In this study, first, our “perceived interpersonal discrimination” measure emphasizes microaggressions, which are brief and commonplace daily verbal and behavioral indignities (whether intentional or not) that communicate hostile, derogatory, or prejudicial insults (21); second is “vicarious racism,” or the experiences of racist events indirectly encountered through observation or learning (22); and third is “rumination,” or perseverating thoughts or feelings on negative discriminatory experiences (23, 24).

Modern allostasis-informed neuroscientific theories of constructed emotion (TCE) (25) propose that our brains integrate sensory information within (interoception) and external (exteroception) to the body, make predictions about both what will happen in the external world and our internal bodily needs, and then prepares our bodies for action by regulating energetic and immune resources through allostasis. According to the TCE, these diverse inputs and predictions are interoceptively experienced in consciousness as affect (how positive/negative and low/high aroused one feels), which together with cognitive processes generates emotional perception and experience. Threat-related stressors are thought to be particularly damaging as they increase energy demands that are experienced as negatively valenced affective sensations feeding into negative appraisals and emotions (26). Consistent with the larger concern that racism-related stress is a contributor to health disparities, particularly among African Americans, research shows that exposures to racism-related stressors—such as discrimination, rumination, and vicarious racism—are associated with adverse psychological outcomes, including anger, anxiety, and depressive symptoms (22–24, 27). Associations with negative emotions have been found using cross-sectional (23, 28, 29), longitudinal (30), and daily diary designs (27, 31–33).

To our knowledge, only one study has previously linked the timing of discrimination to the timing of negative emotions throughout the day (33), while other racism-related factors have yet to be examined. Given the proposed links between acute

stress-related allostasis and negative emotions, the associations between daily racism-related experiences and mental health, and the threat-related nature of these social exclusionary experiences, we hypothesized that within-day race-related experiences would be concurrently and positively associated with negative emotions. Understanding these links is essential because fluctuations in negative emotion may contribute to risk for depression and social anxiety (34).

Allostasis, Racism-Related Stress, and Health

The TCE proposes that the brain does not operate as a stimulus-response model. Rather, the brain (largely unconsciously) makes predictions (and calibrates prediction errors) about both the external world and internal milieu, seeking to match metabolic resources to external demands and internal needs (25). For example (35), when the brain predicts an acute threat-related stressor, the SNS directly innervates vascular and paravascular tissue while the SAM cascade releases adrenaline and activates proinflammatory immune response genes in anticipation of the need for wound healing. Over the course of minutes, via the HPA axis, glucocorticoids increase energy circulation and suppress the circulation of proinflammatory cytokines. With chronic activation, ongoing elevation of circulating glucocorticoids can desensitize or inhibit glucocorticoid receptors, reducing inflammation suppression and elevating basal inflammatory activity levels (36). Through these and related processes, SAM and HPA-axis up-regulation are thought to be key mechanistic pathways by which racism-related experiences become embodied in morbidities (37) such as cardiovascular disease (38–40), particularly among African Americans (41, 42).

In biologically informed research on racism-related stress, discrimination has received by far the most attention in observational studies. When not cross-sectional, studies tend to use biomarkers summarizing allostatic activity, often over long-time horizons (e.g., months, years), while measuring discrimination retrospectively (43). Although reflective of stress in general, these measurements are temporally decoupled from specific racism-related experiences. The majority of such studies either measure cortisol as a marker of HPA-axis activation and dysregulation (43, 44) or markers of inflammatory immune function (e.g., proinflammatory cytokines) (44–46). Therefore, these studies tend to capture statistical correlations indicative of allostatic modulation, but not precise time-coupled regulatory dynamics. Laboratory-based studies linking stress reactivity and discrimination build upon extensive social exclusion and stigma literatures and most commonly include retrospective discrimination measures followed by acute stress exposure and measurement (47, 48), while a few others introduce discriminatory treatments and measure allostatic parameters (49, 50). These studies are primarily limited to measuring interpersonal discrimination, although one notable exception found that vicarious racism exposure predicted elevated cortisol (39). Overall, laboratory studies document increased heart rate, lower heart rate variability, and elevated cortisol secretion in response to discrimination exposure (43, 44). Although a sparse set of laboratory studies document discrimination-based allostatic up-regulation, precise time-coupled dynamics have not been assessed in natural settings or for other forms of racism-related stress for which reactivity may vary.

We sought to build on this research by directly assessing time-coupled SAM-mediated allostatic responses to racism-related stress, combining the ecological validity of observational studies while approximating the temporal precision available in the laboratory. To this end, we developed a within-day design to identify the association between race-related stress and allostatic stress-response modulation using a wearable device that continuously tracked EDA (51–53), a measure of SNS arousal tracked by small changes in perspiration on the surface of the skin that increase electrical conductivity (54). Because EDA was

sampled at 4 Hz and responds to changes in arousal within seconds, the research protocol was designed to contextualize the signal by coregistering experiences through the day. Experimental studies document increased SNS arousal in response to acute negative social stressors, such as social exclusion, by means of increased heart rate, blood pressure, and EDA (55). SNS activity is therefore an important signal for capturing the first SAM-mediated stages of allostasis because of its role in the control of essential cardiac functions, respiration, blood pressure (42), and other physiological parameters (56).

To our knowledge, there have been no studies designed to document simultaneous exposure to race-related stressors and time-synchronized allostatic responding in natural settings, although experiences on short-time scales have been linked to negative emotions and decreased coping capacity (33). Based on the notion that discrimination represents a direct social exclusionary threat, laboratory and observational studies documenting acute stress responses to discrimination (44), laboratory work documenting direct links to heart rate variability (49), and following the TCE, we hypothesized that these acute experiences would increase energetic demands that would elevate time-coupled SNS arousal via allostatic regulation. Considering rumination and vicarious racism threat-related stressors, we also hypothesized that exposure would increase arousal. However, rumination and vicarious racism exposure have also been associated with depressive symptoms (24), which could lead to decreased arousal if such experiences (on average) promote withdrawal and decreased energy mobilization (57).

Racism-Related Stress and Race/Ethnicity

Given that systematic social background differences between race/ethnic groups could affect both differential predicted metabolic demand and appraisal of race-related experiences, we conducted separate analyses for each group in our study: African American, 1.5 generation Black (1.5 Gen Black) students who were primarily childhood refugee migrants (58), African, and Latinx. For example, international African students are adapting to a new cultural context and navigating it in a secondary language, which could alter both the time course of recognition patterns and allostasis (e.g., a discriminatory experience may be most commonly understood only later in a ruminative context). The 1.5 Gen Black

participants, largely from a local African refugee population, were embedded in a cohesive, strongly integrated local community. At the same time, Latinx groups were targeted by antiimmigrant election rhetoric and with punitive immigration enforcement policy proposals over the study period, which might have marshalled in a new era of heightened vigilance and sensitivity to racism-related stressors. Racialized experiences have always been a component of the African American experience. We therefore estimated group-specific associations.

Results

For our analysis, $n = 100$ racially diverse students ($n = 31$ African American, $n = 30$ 1.5 Gen Black, $n = 15$ African, $n = 24$ Latinx; $n = 61$ female, $n = 39$ male; age = 20.4 y) at a predominantly White Midwestern university wore Empatica E4 wristbands (59) and completed daily diaries sent via SMS messages for up to 2 wk (for descriptive statistics, see *SI Appendix*, Tables S1–S6). There were a number of advantages for our purposes to working with college students on a primarily White campus. Upwardly mobile students of color commonly attend predominantly White campuses, and the race/ethnic composition of such environments is likely to persist postgraduation in primarily White workspaces, possibly contributing to high levels of stress and adverse stress-related outcomes (60). Additionally, college students are generally more literate and have greater technological access and familiarity than the general population. The latter point was especially important given the complexity of our study protocols.

The Empatica E4 wristband was chosen for its EDA sensor. EDA indexes SNS arousal from small changes in sweating in a relatively linear fashion (51). As with the HPA-axis, SNS is up-regulated when individuals are confronted with environmental threat (56). The raw signal was processed in 5-min epochs using a reverse inference dynamic causal model that estimated sudomotor neuron activity (SNA) from nonspecific skin conductance fluctuations (61, 62). The 5-min epochs were then grouped into 15-min “moments” of the average, maximum, minimum, and maximum/minimum (max-min) difference in EDA–SNA/5-min rates (*SI Appendix*, Fig. S1). Each EDA–SNA measure summarizes different aspects of the momentary arousal time course. Average EDA–SNA provides a scaled summary of total activity throughout the moment, while the maximum and minimum

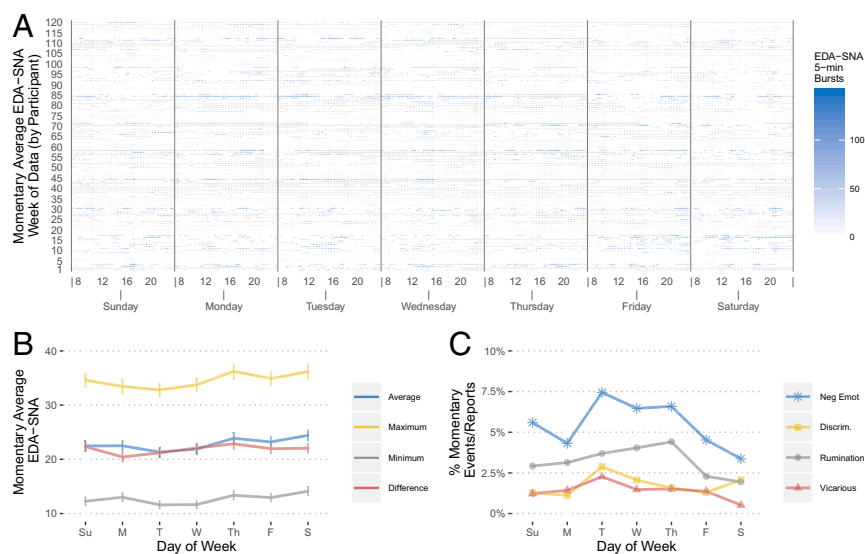


Fig. 1. Description of the key analysis data including EDA–SNA arousal, negative affect, and racism-related stress. (A) Heatmap representation of the EDA–SNA data used in this study by the weeks included in the analysis. Each cell is a 15-min moment and missing data are indicated in gray. (B) Summary of momentary EDA–SNA and (C) negative emotion and race-related stress over the week.

reflect the 5-min highs and lows within each moment. The maximum difference can then capture acute spiking within the moment or baseline shifts when considered in conjunction with the minimum. We refer to moments with very low EDA-SNA rates as “0-moments.” A heatmap of these data is presented by participant week in Fig. 1A. The weekly average arousal curves are plotted in Fig. 1B for each EDA-SNA operationalization. Arousal was lowest on average early in the week before rising and peaking on Friday.

The EDA-SNA data were complemented with electronic surveys sent each evening that were used to annotate activities and experiences throughout the day. Following Daly et al.’s (63) study linking within-day heart rate and affective dynamics, we adapted the day reconstruction method (DRM) (64), a diary approach approximating discrete experience sampling methods. This approach allowed us to link activities and experiences to moments so that the behavioral and EDA-SNA data could be joined on short timescales. Participants first identified common anchoring activities that could also affect arousal levels assessed by EDA (e.g., class attendance, studying, meals, and so forth) and then indicated when these activities took place, with scales first utilizing broad temporal ranges (e.g., 7:00 to 10:00 AM) and then telescoping to momentary ranges (e.g., 8:00 to 8:15 AM) (SI Appendix, Fig. S2). After identifying anchoring activities, discrimination-related experiences—including discriminatory events, vicarious racism, and rumination on racism—were assessed. A momentary discrimination event was encoded based on one or more responses to 17 questions from the Racism and Life Experiences Scale (e.g., “being treated rudely or disrespectfully”) (65). Specific items were not linked to specific moments. Instead, a momentary indicator reflects one or more events from the list. Both vicarious racism [“Did you learn about racial injustices or the mistreatment of people of color on social media (such as Facebook, Twitter, etc.) today?”] and rumination (“Over the course of the day, did you think about racial injustices and the mistreatment of people of color in the US?”) were captured with single items. Independent assessments of momentary negative emotion were then collected (“Think about how negative, anxious, bad, or stressed you felt throughout the day”). Fig. 1C presents the percentage of moments for which these items were reported throughout the week. Discrimination and vicarious racism peaked early in the week, before declining later in the week, a pattern followed by negative emotion. Rumination tended

to be most prevalent in the middle of the week before declining into the weekend.

Analysis of Momentary Negative Emotion. We first test the hypothesis that racialized experiences are positively associated with concurrent momentary negative emotion, measured as a binary indicator of a negative affective state. We used participant-nested multilevel logistic regression models for momentary negative emotions, with results reported in Fig. 2 as odds ratios (OR) (SI Appendix, Table S6). ORs reflected the difference in the odds that negative affect was reported in a given moment when a racism-related experience (discrimination, rumination, vicarious racism) was reported versus moments when no such experience was recorded. Control variables included lagged negative emotion from the prior moment, race/ethnicity, meals, napping, exercise, in class, studying, at work, day number in the study, and fixed effects for hour of the day and day of week. Fig. 2A reports ORs by race with African American students as the reference group. Only the 1.5 Gen Black students were statistically less likely to report negative emotion (OR = 0.49, $P < 0.01$). ORs (Fig. 2B) indicate that discrimination (OR = 6.33, $P < 0.001$) and rumination (OR = 2.35, $P < 0.001$) moments were positively associated with concurrent negative-emotion reports in the moment, while vicarious racism trended in the same direction but did not achieve statistical significance. These results suggest that racism-related experiences are positively associated with negative emotion independently of prior momentary negative emotion.

As shown in Fig. 2C, these findings are largely consistent for African American and Latinx students. In contrast, only rumination was associated with negative emotion for 1.5 Gen Black students (OR = 6.49, $P < 0.01$). Both discrimination (OR = 103.3, $P < 0.001$) and rumination (OR = 12.81, $P < 0.01$) predicted negative emotion for African students, but are not shown graphically because of near-perfect prediction. A discrimination event was a strong predictor of negative emotion for all groups except the 1.5 Gen Black students, while rumination and vicarious racism had differential associations across groups, but of these two, only rumination was positively associated with negative emotion across groups.

Analysis of Momentary EDA-SNA. Having established that racism-related experiences are associated with negative emotions, we

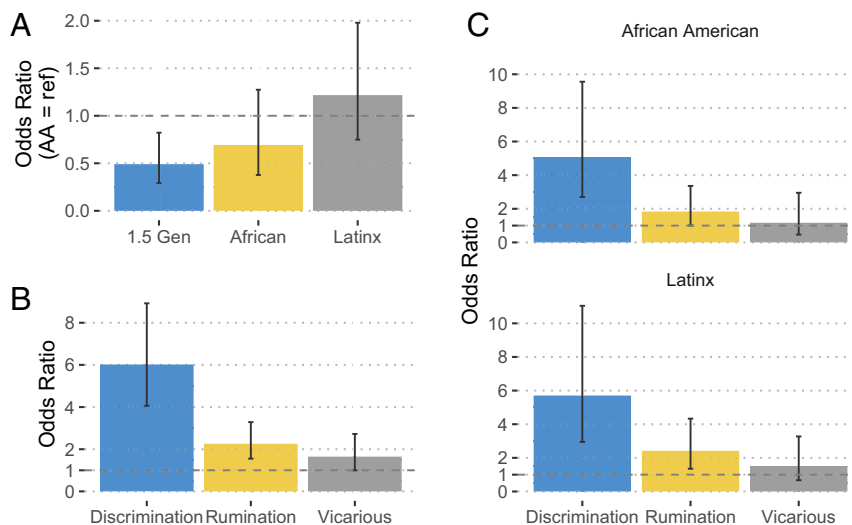


Fig. 2. ORs with confidence intervals for race/ethnicity and race-related stressors predicting the binary momentary negative emotion indicator using multilevel random intercept logistic regression models. (A) Results by race (AA = ref, African American as reference group) and (B) race-related stressor for the full sample. (C) Results for African American and Latinx participants (see SI Appendix, Table S6 for full results).

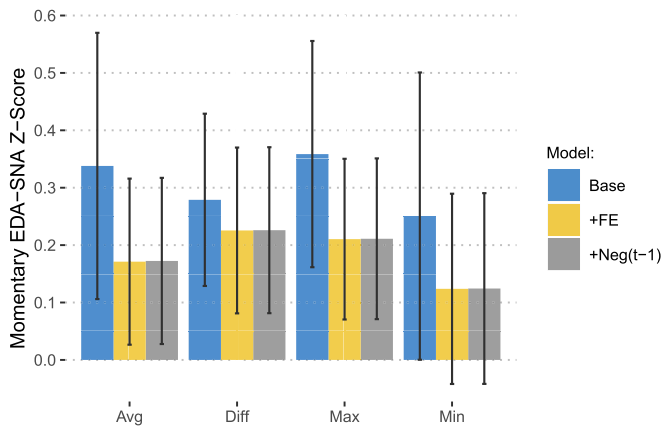


Fig. 3. Discrimination events and arousal for African Americans. Standardized momentary EDA-SNA estimates with confidence intervals from fixed effects linear regression with robust SEs (for full results see *SI Appendix, Fig. S3 and Tables S8–S11*). Avg, average; Diff, difference; Max, maximum; Min, minimum.

next sought to test the threat-related allostatic hypothesis that emotional arousal would be elevated during periods of racism-related stress. We present linear regression models using the standardized EDA-SNA measures with robust SEs over the following specifications: First, with the control variables and lagged momentary EDA-SNA from the prior moment with a random intercept, then including person fixed-effects, and finally adding lagged negative emotion from the prior moment to adjust for state negative emotionality biases, which have been offered as an alternative explanation for discrimination-health associations (66).

Results for discrimination events for African Americans are presented in Fig. 3 (see also *SI Appendix, Fig. S3 and Tables S8–S11*). Average EDA-SNA was statistically associated with discrimination across models, but the effect size decreased from 0.34 ($P < 0.01$) to 0.17 ($P < 0.05$) with the inclusion of the person fixed effects. In other words, average EDA-SNA was 0.17 SDs larger when comparing individuals to themselves (within-person due to inclusion of fixed effects) during moments when discrimination was reported versus moments when it was not reported, all else equal. This association was also not affected by the inclusion of lagged negative affect, suggesting that the response to perceived discrimination is not being driven by prior momentary negative emotions. Both the max-min difference and maximum EDA values were consistently statistically significant ($P < 0.01$) across equations with effect sizes >0.2 SD. The rise in the max-min difference (and lack of elevation for the minimum) are consistent with an acute stress response as individuals showed spiking during moments when discrimination was reported when compared to moments when it was not. Findings for rumination and vicarious racism were not consistently significant so are not reported here. When combined with the negative-emotion findings, these results point to discrimination experiences as a contributor to acute stress arousal. Neither rumination nor vicarious racism were associated with consistent patterns of either higher or lower levels of emotional arousal. Our second hypothesis is therefore only partly supported for African American students.

Latinx students displayed more consistent patterns of within-person EDA-SNA arousal differences than African Americans during moments in which racism-related experiences were reported versus those moments when such experiences were not reported, as shown in Fig. 4 (*SI Appendix, Tables S12–S15*). Microaggressive discrimination events were associated with elevated arousal across average, maximum, and max-min difference EDA-SNA operationalizations, with effect sizes of ~ 0.2 SD. As with African American students, this pattern of results is consistent with an acute

stress response to discrimination. Latinx students also showed elevated average and minimum arousal during periods of rumination, indicating more sustained arousal. Given the trend of antiimmigrant rhetoric over the study period, these findings are consistent with reports of increased race-related stress as students grappled with hostility and uncertainty about their future and acceptance in the United States (67). In contrast, vicarious racism was associated with lower emotional arousal across EDA-SNA operationalizations. Circumplex models of emotion posit that feelings of fatigue are associated with neutral valence low arousal states, while feelings of anger and frustration are associated with higher negatively valenced arousal states (68). The findings for Latinx students were therefore partially consistent with our second hypothesis. Arousal was increased for discrimination experiences and rumination on racism, possibly indicating threat-related responding, but with suppressed arousal possibly indicating fatigue in response to vicarious racism.

Findings for 1.5 Gen Black students showed few consistencies across models or robustness checks, and are therefore not shown here (*SI Appendix, Fig. S4 and Tables S16–S19*). African students had elevated EDA-SNA during rumination (average, maximum, minimum) (*SI Appendix, Fig. S5 and Tables S20–S23*). Interestingly, because of language and cultural differences, African students may not always recognize discriminatory or micro-aggressive experiences in the moment but come to recognize such experiences later when ruminating upon their social encounters. As with Latinx students, arousal was consistently lower in vicarious exposure moments across operationalizations. Taken together, these findings suggest that care should be taken when admixing different race/ethnic populations due to potential background differences that affect the way race-related stress is experienced, physiologically expressed, and emotionally categorized.

Robustness Checks. In addition to the results presented here, we also analyzed the data under different assumptions to determine if the results were robust. We therefore reanalyzed the data in the following ways (for results, see *SI Appendix*). First, EDA-SNA data were skewed with a long high-arousal state tail. For the most part, participants spent time in low-arousal states on the EDA-SNA index. To address the issue of extreme outliers, we Winsorized momentary EDA-SNA at the upper 99th percentile. Across parallel models to those presented above, results were very nearly identical, indicating that arousal outliers did not bias our results. Second, we also analyzed EDA-SNA in the raw 5-min impulse rate using both random intercept and fixed-effects linear regression, Tobit regression, and zero-inflated negative binomial regression (ZINB). Given the skew in the EDA-SNA rates, SEs in the linear random intercept and fixed-effects regressions were adjusted using robust SEs, as in all prior analyses. The Tobit model assumed that EDA-SNA 0-moment left-censoring reflected limited sensor sensitivity for lower arousal states. The ZINB model assumed overdispersed rate data with excess 0-moments. This model combined logistic regression with negative binomial regression to predict whether or not each moment was a 0-moment and then the conditional EDA-SNA rate. The presented results were largely consistent across these specifications and differing core modeling assumptions.

Third, the ZINB model provided estimates of the likelihood of a 0-moment (*SI Appendix, Table S24*). These results largely confirmed our prior findings for African American and Latinx students, and were consistent for vicarious racism exposure for African students. African Americans were less likely to report 0-moments for average, maximum, and max-min EDA-SNA activity during those times when a discrimination event was reported. For Latinx students, vicarious racism was associated with an increased 0-moment probability and the maximum, minimum, and max-min 0-moment probability were decreased when discrimination was reported. These results therefore supported the prior reported linear regression findings. Fourth, we

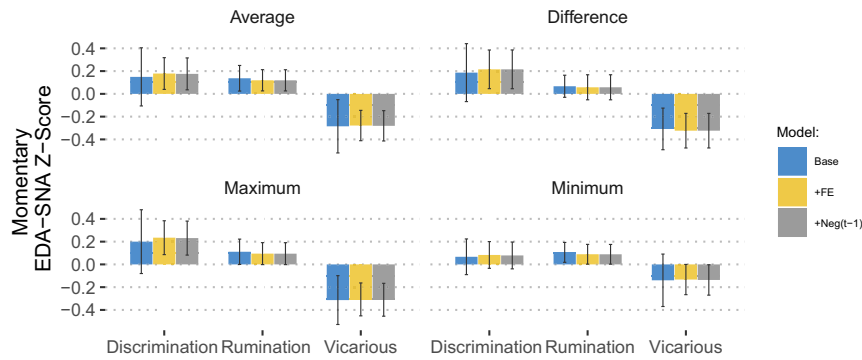


Fig. 4. Race-related stress and arousal for Latinx participants. Standardized momentary EDA-SNA estimates with confidence intervals from random and fixed effects linear regression with robust SEs (for full results, see *SI Appendix, Tables S12–S15*).

aggregated the results up from moments at the 15-min level to accumulation per hour and then reanalyzed parallel specifications of the accumulated z-score EDA-SNA/5-min rate using linear regression with robust SEs, and with the new accumulated raw EDA-SNA/5-min rate using the ZINB model. These analyses, which grouped moments into hours, generally confirmed our prior results, giving us greater confidence that the EDA-SNA and racism-related experience findings presented in the text are robust to a variety of model, distributional, and temporal specification assumptions.

Discussion

Racism is hypothesized to be an important contributor to health inequities via increased negative emotions and predictive allostatic modulation of an acute stress response that is responsible for energy mobilization and wound healing. Over time, repeated stress actuation increases physiological wear and tear above and beyond other forms of life stress (e.g., economic stress) via brain-regulated allostatic mechanisms (11, 69) that can come to adversely shift physiological response profiles over time (9). This study directly documents key aspects of the TCE model in the context of racism-related stress by temporally interlinking episodes of these stressors to negative emotions and the early SNS component of the acute stress response via EDA from a wearable sensor. Although our laboratory-inspired stimulus-locked approach organized around 15-min “moments” throughout the day is approximate, this limitation is balanced against the real-world significance of measuring these processes unscripted in the “social time” of day-to-day life.

Overall, our results support prior findings that perceived microaggressive discrimination increases negative emotion. Our study differed from—and went beyond—prior work by linking the timing of discrimination to concurrent negative emotion risk experienced in the real world, controlling for negative emotion reported a moment earlier. These associations held for all groups except for the 1.5 Gen Black students, who were less likely than others to report negative emotion and race-related stress in general. These students were primarily from a Sudanese refugee population (70), about whom very little is known with regard to their experiences with discrimination and the subsequent extent to which such experiences affect their psychological and physical health. It is possible that their membership in a close-knit, highly cohesive and integrated community was protective. African American, Latinx, and African international students were, however, more likely to report increased risk for negative emotion in response to discrimination events. Following this pattern of increased negative emotion, elevated SNS arousal was also shown for African American and Latinx students in response to discrimination. The strong association between discrimination and negative emotion points to the role of discrimination in

elevating negatively valenced arousal. These findings are provocative when viewed through the lens of TCE, which posits allostasis as a critical contributor to mental health (26), the idea being that the affective feelings generated via interoceptive monitoring of predictive allostatic regulation influences cognitive appraisal and emotion classification (25). From the TCE perspective, allostatic response profiles are endogenously implicated in both mental and physical health (26).

Latinx and African students also had increased average arousal during ruminative moments and suppressed arousal across EDA-SNA operationalizations in response to vicarious racism, although vicarious racism exposure was not associated with negative emotion. The lack of consistent results for African Americans’ arousal with respect to vicarious racism and rumination may seem surprising given their rates of reported interpersonal discrimination and media exposure to black deaths at the hands of police (e.g., George Floyd, Breonna Taylor, Philando Castille, Alton Sterling, Terence Crutcher, among others). The study covered a first period of peak activity in the Black Lives Matter movement, which may have provided collective spaces for political activism and social support that mediated the negative consequences of vicarious racism and rumination (71, 72). At the same time, a level of fear and uncertainty surrounded the punitive immigration policies targeting the Latinx community. Mass deportations and immigration enforcement heightened fear and uncertainty as 96% of deportations are Latin Americans and 36% of Latinos report knowing someone who has been deported (73). Consequently, Latinx youth in predominantly White spaces report more fear, anxiety, and concern around the security and safety of their family and community members (74, 75). With respect to vicarious exposure and decreased arousal, such experiences may increase fatigue, reflecting the chronicity of the persistent emotional toll from threat of disruption to their lives (76).

This study is also limited in important ways that may provide useful guidance for future research. Although the study was rich along the time dimension, the overall sample size was small, particularly for race/ethnic stratified analysis, and lacked the statistical power to explore moderation by nativity for Latinx participants or by gender. In addition, the sample was constrained to students on a single Midwestern college campus. We note that despite the geographic limitations, many colleges and universities are primarily White institutions, as are many of the workplaces that students of color will enter and spend significant amounts of time in after graduation. However, it remains true that future studies would benefit from random-sampling techniques, larger sample sizes to increase statistical power and representativeness, as well as increased diversity along other dimensions (e.g., age). Larger samples may also be important for providing new insights into how the treatment effects of race-related

stress vary both within and between race/ethnic groups. Exposure to racism could also result in differential allostatic response profiles at different times. For example, ruminating on racism may, at one occurrence, lead to anger and elevated arousal, but fatigue and decreased arousal at another time, depending on context. Moreover, we identified only group-specific average effects but there are important individual differences in stress reactivity that should be considered in future research (77).

An additional limitation worth considering is the challenge of linking experiences in real-world settings to short-term physiological response-pattern dynamics. Following prior work linking within-day affect variation to heart rate using the DRM diary technique (63), we adapted this method (64). Although we originally anticipated applying the experience sampling method (ESM) (78), we discovered during pilot testing that our goals were particularly challenging for ESM. The sensor-based signal provided continuous data along the time dimension, while ESM provided snapshots scattered discretely throughout the day. Moreover, ESM surveys still required retrospection as we did not anticipate concurrent experiential reports during survey responding. The amount of retrospection was also directly tied to the number of ESM survey snapshots needed to detail the day with respect to the ongoing EDA stream. ESM is most useful for questions related to concurrent individual states, but adding retrospection due to the social nature of the processes under study here dramatically increased both survey complexity and respondent burden. Burden was further exacerbated by the high-dimensionality racism-related experiences, which can take many forms, as well as inclusion of other experiences throughout the day to provide additional context of relevance to the EDA signal. In short, our ESM surveys became cumbersome, greatly risking response rates and therefore within-day coverage (79). It is important to acknowledge that not all studies find strong correlations between DRM and ESM reports (80) and that a number of outstanding questions related to DRM remain (81). Recall bias may have inflated estimated associations between discrimination and the likelihood of a negative emotion moment report. At the same time, linking experiences to clock time can be quite challenging, which could have biased associations between EDA-SNA and racism-related events. Here, it seems likely that the bias decreased effect sizes due to misalignments and our results may therefore provide underestimates of the average arousal response. Despite the potential biases, the discrimination findings in particular are consistent with theoretical expectations linking social exclusionary experiences and both negative affective valence and increased arousal (82). However, we believe there remains considerable opportunity for methodological refinement of low-burden methods interlinking individual states, social experiences, and physiological processes.

Finally, it is also worth noting that we were only able to measure the first stages of the physiological response, the SNS-mediated component of the SAM. A full acute stress response extends over longer periods of time with increased HPA-axis activity and immune response alterations (44, 83). However, the link between discrimination and negative emotional arousal is telling in this case, strongly suggesting an overall response pattern that may be adaptive on short time scales but could be health-damaging when chronic. Although common approaches to stress-response management suggest breathing, mindfulness, and related techniques to decrease arousal (84), the biopsychosocial model of threat and challenge poses an interesting framework (85–87). From this perspective, it is noted that downstream allostatic cascades can be altered by whether a stressor is appraised as a threat or challenge. Accordingly, challenge appraisals result in differential SAM activity patterns at the early stages and without the subsequent HPA activity associated with threats. Challenge appraisals appear to increase SAM activity, but with decreased peripheral vasculature resistance and increased cardiac output

(86). Challenge appraisals also appear to be associated with improved academic performance and decreased proinflammatory cytokines in the context of stereotype threat (88). Intervention approaches may therefore consider the importance of appraisal not just in terms of reducing activation, but in terms of reappraisals that motivate positive challenge orientations. Viewed from this angle, it is also worth considering that allostasis is not just a stress response so much as a mechanism of energy regulation, and that maintenance and restoration of flexible physiological response capacity is an important feature of health that should be one target of intervention efforts (9, 89).

Conclusion

This study has important implications for understanding racism-related health processes. Such research is of considerable importance because racial resentment (90) and divisive political rhetoric (91) are on the rise, hate crimes have become more frequent in recent years (5, 92, 93), and racist events have become more common on college campuses (94). Recognition of these and related issues, such as police violence, resulted in Black Lives Matter protests across the nation throughout the summer of 2020 and into the fall. At the same time, due to the systematic nature of racism in the United States, the chronicity of the allostatic stress-regulation processes studied here appear to contribute to the stark race/ethnic inequities in COVID-19 illness severity and mortality via contributions to health conditions associated with more aggressive disease progression (95, 96). We have documented allostatic responding in the context of negative emotions consistent with the SAM-mediated first stages of a classic acute stress response. In doing so, we demonstrated a method of physiological measurement using a signals approach that allowed us to borrow the concept of a time-coupled stimulus-response from laboratory research but applied outside of the laboratory as participants navigated the social ecology of their day-to-day lives. Perhaps due to concerns about the ethics of racial mistreatment in laboratory paradigms, most of the theoretical work underlying physiological explanations of racism-related health inequities has been tested in other contexts (e.g., social exclusion, general stress) or only via temporally imprecise and decoupled correlations. We have shown that key parts of the underlying mechanistic model translating racism-related exposures like discrimination to health via allostasis hold in consistent ways, particularly for African American and Latinx college students. By showing how such stress manifests emotionally and physiologically in real time, our findings ground prior work documenting correlations between physiologic wear and tear and race-related stress early in the life course (97–99). This embodiment is likely key for understanding how America's legacy of racism shapes psychological and physical health inequities.

Materials and Methods

Data and Methods. Data for this study were collected on a large, predominantly White Midwestern university campus during the 2016 to 2017 academic year. Participants were recruited via flyers and listserv emails to campus groups serving students of color. During the fall 2016 semester participants were enrolled for a 2-wk period, and for a 1-wk period during the spring 2017 semester (for additional information, see *SI Appendix*). Upon enrollment, students were invited to the laboratory where they were consented and administered an intake survey (basic sociodemographics, psychological characteristics, and prior discrimination experiences). During intake participants were trained to use the Empatica E4 wristbands (59, 100, 101), to upload data to the cloud via a provided laptop, and to complete the smartphone-delivered daily diaries. Links to morning and evening diaries were delivered via SMS text messages at 8:00 AM and 8:00 PM with a 6-h response window. The evening diary, used here, took ~8.5 min on average. The evening diary response rate was 87%.

During the fall semester students had the potential to receive up to \$270 in compensation for completing all study procedures over the 2-wk period, including two laboratory visits, all daily diaries, and for wearing the

wristbands throughout the day. The incentive was up to \$144 during the spring semester for the completion of all study procedures over the 1-wk period. This study uses data collected from the intake survey, evening diaries, and the E4 wristbands. The University of Nebraska Institutional Review Board approved the study protocol. All participants signed written informed consent documents. All study procedures were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national), as determined by the Helsinki Declaration of 1975, and revised in the year 2000.

Sample. The sample used for this analysis combined three sources of data: Daily diaries, transformed into within-day moments; the processed EDA sensor data; and the intake survey. Our final analysis sample of $n = 100$ participants provided $n_t = 37,654$ observations. To select the sample, there were initially 147 participants with sensor data of a total of 151 participants. However, only 142 participants were able to join together sensor, daily diary, and intake data. Of these, another 18 did not have valid sensor data between 7:00 AM and 12:00 PM, the waking day, and another 18 were either White or Asian, and so were removed from the study, reducing the sample size to 106 participants. There were another four participants who showed no evidence of an EDA response despite providing a valid sensor stream. Finally, after removing empty moments due to the construction of lags, an additional two participants did not have sufficient observations to remain in the study. The final sample size was reduced to 100 participants. The theoretical number of moments for this sample is $n_t = 60,984$. Some of the lost moments reflect hardware failures both for the wearable device as well as nondelivered or nonreceived SMS survey links. Participants also removed devices at different times for charging/uploading data, when participating in competitive sports, during activities they did not want to be tracked, and during periods when they were simply noncompliant as some found the stiff wristband to be uncomfortable. Basic descriptive statistics are shown in *SI Appendix, Tables S1 and S2*.

Within-Day Measures. The following measures were used to characterize student experiences, emotions, and activities throughout the day. For the surveys, activities/behaviors/experiences were indicated as having taken place during 3-h windows, after which the questions telescoped to momentary 15-min indicators within selected 3-h periods.

Sensor Data. This study used EDA (4 Hz) collected at the wrist (*SI Appendix*). The raw signal was median-filtered over a five-sample window and the timeseries was trimmed and aligned to 5-min intervals from the top of the hour. This window provided 1,200 data points for the general linear mixed model used in the processing routine in MATLAB using the PSPM (v3.02) dynamic causal model of spontaneous skin conductance fluctuations estimator (61, 102). This variational Bayesian model inversion algorithm infers SNA from spontaneous skin voltage fluctuations. Because biphasic decay of the skin voltage fluctuation signal builds up in a linear fashion (103) determined by peripheral factors in addition to SNA, the model was structured to return to 0 in the absence of SNA with a maximum of 30/min. The SNA threshold was set at 0.05 μs . The procedure therefore provided a single SNA activity summary estimate over the interval that we then combined into moments to assess different facets of the activity throughout the moment to provide insight into average activity, spiking (maximum, max-min difference), and baseline shifts throughout the moment (minimum). No SNA activity was detected in 61% of the average, maximum, and difference moments, suggesting that most moments are not high emotional intensity and the distribution of arousal is not normal in shape. Descriptive statistics are shown in *SI Appendix, Table S3*.

Negative Emotion. In the evening, diary participants were asked to report when they felt negative throughout the day. The specific wording of the question was: "Think about how negative, anxious, bad, or stressed you felt throughout the day. Were there instances during the following 3-hour periods when you felt very negative, anxious, bad, or stressed? You may select multiple options throughout the day." Descriptive results are presented in *SI Appendix, Table S4*.

Discrimination-Related Experiences. Race-related social experiences were measured in three ways. Discrimination events used Harrell's Racism and Life Experiences Scale (104). Participants indicated whether they experienced any of 17 largely microaggression-consistent items (e.g., "being treated rudely or disrespectfully," "others reacting to you as if they were afraid or intimidated," "being treated as if you were 'stupid' or 'talked down to'," and so forth). Participants were then asked when these experiences took place throughout the day. In order to make the delivery of the questions feasible, it was not possible to link specific items to moments. Discrimination is thus a momentary binary indicator of a perceived microaggressive experience. Participants were also asked about rumination ("Over the course of the day, did you think about racial injustices and the mistreatment of people of color in the US?") and vicarious racism ("Did you learn about racial injustices or the mistreatment of people of color on social media (such as Facebook, Twitter, etc.) today?"). These experiences were similarly mapped throughout the day and included in all statistical models (*SI Appendix, Table S4*).

Additional Variables. In the evening diary, participants were first asked to annotate their experiences and activities throughout the day with the adaptive 3-h to 15-min procedure that oriented participants to differing events throughout the day. In general, we were seeking to control for different settings college students inhabit as they go about their days, recognizing that arousal could vary throughout the day as many physiological processes related to mood, attention, and so on are both context-dependent but also follow (e.g., diurnal) curves (which is why we were careful to adjust for time). Many settings also have implications for physical movement as well as being indoors and outdoors and associated temperature fluctuations, all of which can influence EDA (51, 105). Indicators were included for when participants attended class, were studying, or were at work; when participants had breakfast, lunch, and dinner; as well as when they were exercising or napping. These indicators were used to help participants orient their experiences throughout the day as part of the survey procedure and to contextualize common reoccurring activities that could represent deviations in arousal states. For example, arousal as measured by EDA increases with exercise and decreases sitting stationary during class.

Analysis Approach. The binary indicator of momentary negative emotion was modeled using a two-level random intercept model (106). The model was structured over time points t for each of the participants i . Logit coefficients for the full sample and by race/ethnicity are shown in *SI Appendix, Table S6* for the presented results and under relaxed sample constructions in *SI Appendix, Table S7*. Momentary EDA-SNA parameters were first estimated using linear random intercept models with robust SEs by race/ethnicity, and then including person-fixed effects to control for temporally invariant confounds (107). Within-person y -standardized estimates are presented for the main results. Robustness checks included standardized Winsorized EDA-SNA, utilizing the unstandardized EDA-SNA burst estimate, a Tobit model of the burst rate, and the ZINB. In addition, data were also collated into a 60-min representation using the EDA-SNA z -score, EDA-SNA burst, and ZINB. These results are shown in *SI Appendix, Tables S8-S23*. The marginal discrete changes in the probability that a 0-moment EDA-SNA was recorded from the ZINB models are shown in *SI Appendix, Table S24*. Estimates showing that results are consistent when comparing the subset of results from a second week of participation to the first week are shown in *SI Appendix, Table S25* for EDA-SNA and *SI Appendix, Table S26* for negative emotions.

Data Availability. Preprocessed data and statistical modeling scripts are available on the Open Science Framework, <https://osf.io/sbhd7/> (108).

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1. E. Bonilla-Silva, The structure of racism in color-blind, "post-racial" America. *Am. Behav. Sci.* **59**, 1358-1376 (2015).
2. A. M. Enders, J. S. Scott, The increasing racialization of American electoral politics, 1988-2016. *Am. Polit. Res.* **47**, 275-303 (2019).
3. B. F. Schaffner, M. Macwilliams, T. Nteta, Understanding White polarization in the 2016 vote for president: The sobering role of racism and sexism. *Polit. Sci. Q.* **133**, 9-34 (2018).

4. D. Bauman, Hate crimes on campuses are rising, New FBI data show. *The Chronicle of Higher Education*, <https://www.chronicle.com/article/hate-crimes-on-campus-are-rising-new-fbi-data-show> (2018). Accessed 3 December 2019.
5. G. S. Edwards, S. Rushin, The effect of President Trump's election on hate crimes. *Social Science Research Network*, https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3102652 (2018). Accessed 5 November 2018.

6. N. Priest *et al.*, A systematic review of studies examining the relationship between reported racism and health and wellbeing for children and young people. *Soc. Sci. Med.* **95**, 115–127 (2013).
7. J. M. Rodriguez, A. T. Geronimus, J. Bound, D. Dorling, Black lives matter: Differential mortality and the racial composition of the U.S. electorate, 1970–2004. *Soc. Sci. Med.* **136–137**, 193–199 (2015).
8. R. Clark, N. B. Anderson, V. R. Clark, D. R. Williams, Racism as a stressor for African Americans. A biopsychosocial model. *Am. Psychol.* **54**, 805–816 (1999).
9. P. Sterling, Allostatics: A model of predictive regulation. *Physiol. Behav.* **106**, 5–15 (2012).
10. J. Fridman, L. F. Barrett, J. B. Wormwood, K. S. Quigley, Applying the theory of constructed emotion to police decision making. *Front. Psychol.* **10**, 1946 (2019).
11. B. J. Goosby, J. E. Cheadle, C. Mitchell, Stress-related biosocial mechanisms of discrimination and African American health inequities. *Annu. Rev. Sociol.* **44**, 319–340 (2018).
12. A. D. Ong, L. Benson, A. J. Zautra, N. Ram, Emodiversity and biomarkers of inflammation. *Emotion* **18**, 3–14 (2018).
13. H. E. R. Hunte, D. R. Williams, The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. *Am. J. Public Health* **99**, 1285–1292 (2009).
14. C. M. Dolezar, J. J. McGrath, A. J. M. Herzig, S. B. Miller, Perceived racial discrimination and hypertension: A comprehensive systematic review. *Health Psychol.* **33**, 20–34 (2014).
15. W. M. Troxel, K. A. Matthews, J. T. Bromberger, K. Sutton-Tyrrell, Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. *Health Psychol.* **22**, 300–309 (2003).
16. E. A. Pascoe, L. Smart Richman, Perceived discrimination and health: A meta-analytic review. *Psychol. Bull.* **135**, 531–554 (2009).
17. L. N. Potter, E. Brondolo, J. M. Smyth, Biopsychosocial correlates of discrimination in daily life: A review. *Stigma Health* **4**, 38–61 (2019).
18. A. D. Ong, D. R. Williams, U. Nwizu, T. L. Gruenewald, Everyday unfair treatment and multisystem biological dysregulation in African American adults. *Cultur. Divers. Ethnic Minor. Psychol.* **23**, 27–35 (2017).
19. D. R. Williams, S. A. Mohammed, Racism and health I: Pathways and scientific evidence. *Am. Behav. Sci.* **57**, 1152–1173 (2013).
20. B. Reskin, The race discrimination system. *Annu. Rev. Sociol.* **38**, 17–35 (2012).
21. D. W. Sue *et al.*, Racial microaggressions in everyday life: Implications for clinical practice. *Am. Psychol.* **62**, 271–286 (2007).
22. K. A. Truong, S. D. Museus, K. M. McGuire, Vicarious racism: A qualitative analysis of experiences with secondhand racism in graduate education. *Int. J. Qual. Stud. Educ.* **29**, 224–247 (2016).
23. A. Borders, C. T. H. Liang, Rumination partially mediates the associations between perceived ethnic discrimination, emotional distress, and aggression. *Cultur. Divers. Ethnic Minor. Psychol.* **17**, 125–133 (2011).
24. S. Nolen-Hoeksema, B. E. Wisco, S. Lyubomirsky, Rethinking rumination. *Perspect. Psychol. Sci.* **3**, 400–424 (2008).
25. L. F. Barrett, The theory of constructed emotion: An active inference account of interoception and categorization. *Soc. Cogn. Affect. Neurosci.* **12**, 1–23 (2017).
26. L. F. Barrett, K. S. Quigley, P. Hamilton, An active inference theory of allostasis and interoception in depression. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **371**, 20160011 (2016).
27. J. C. Jochman *et al.*, Mental health outcomes of discrimination among college students on a predominately White campus: A prospective study. *Socius* **5**, 2378023119842728 (2019).
28. K. Sanders-Phillips *et al.*, Perceived racial discrimination, drug use, and psychological distress in African American youth: A pathway to child health disparities. *J. Soc. Issues* **70**, 279–297 (2014).
29. C. L. Woods-Giscombe, M. Lobel, C. Zimmer, C. Wiley Cené, G. Corbie-Smith, Whose stress is making me sick? Network-stress and emotional distress in African-American women. *Issues Ment. Health Nurs.* **36**, 710–717 (2015).
30. N. M. Hurd, F. A. Varner, C. H. Caldwell, M. A. Zimmerman, Does perceived racial discrimination predict changes in psychological distress and substance use over time? An examination among Black emerging adults. *Dev. Psychol.* **50**, 1910–1918 (2014).
31. N. J. Heard-Garris, M. Cale, L. Camaj, M. C. Hamati, T. P. Dominguez, Transmitting trauma: A systematic review of vicarious racism and child health. *Soc. Sci. Med.* **199**, 230–240 (2018).
32. L. Torres, A. D. Ong, A daily diary investigation of Latino ethnic identity, discrimination, and depression. *Cultur. Divers. Ethnic Minor. Psychol.* **16**, 561–568 (2010).
33. N. T. Joseph, L. M. Peterson, H. Gordon, T. W. Kamarck, The double burden of racial discrimination in daily-life moments: Increases in negative emotions and depletion of psychosocial resources among emerging adult African Americans. *Cultur. Divers. Ethnic Minor. Psychol.*, 10.1037/cdp0000337 (2020).
34. R. J. Thompson, M. T. Boden, I. H. Gotlib, Emotional variability and clarity in depression and social anxiety. *Cogn. Emotion* **31**, 98–108 (2017).
35. N. I. Eisenberger, S. W. Cole, Social neuroscience and health: Neurophysiological mechanisms linking social ties with physical health. *Nat. Neurosci.* **15**, 669–674 (2012).
36. N. I. Eisenberger, M. Moieni, T. K. Inagaki, K. A. Muscatell, M. R. Irwin, In sickness and in health: The co-regulation of inflammation and social behavior. *Neuropsychopharmacology* **42**, 242–253 (2017).
37. B. S. McEwen, T. Seeman, Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Ann. N. Y. Acad. Sci.* **896**, 30–47 (1999).
38. C. J. P. Harrell *et al.*, Multiple pathways linking racism to health outcomes. *Du Bois Rev.* **8**, 143–157 (2011).
39. V. W. Huynh, Q.-L. Huynh, M.-P. Stein, Not just sticks and stones: Indirect ethnic discrimination leads to greater physiological reactivity. *Cultur. Divers. Ethnic Minor. Psychol.* **23**, 425–434 (2017).
40. M. M. Merritt, G. G. Bennett Jr, R. B. Williams, C. L. Edwards, J. J. Sollers 3rd, Perceived racism and cardiovascular reactivity and recovery to personally relevant stress. *Health Psychol.* **25**, 364–369 (2006).
41. T. T. Lewis, D. R. Williams, M. Tamene, C. R. Clark, Self-reported experiences of discrimination and cardiovascular disease. *Curr. Cardiovasc. Risk Rep.* **8**, 365 (2014).
42. K. G. Lockwood, A. L. Marsland, K. A. Matthews, P. J. Gianaros, Perceived discrimination and cardiovascular health disparities: A multisystem review and health neuroscience perspective. *Ann. N. Y. Acad. Sci.* **1428**, 170–207 (2018).
43. D. Busse, I. S. Yim, B. Campos, C. K. Marshburn, Discrimination and the HPA axis: Current evidence and future directions. *J. Behav. Med.* **40**, 539–552 (2017).
44. M. Berger, Z. Sarnyai, “More than skin deep”: Stress neurobiology and mental health consequences of racial discrimination. *Stress* **18**, 1–10 (2015).
45. R. L. Simons *et al.*, Discrimination, segregation, and chronic inflammation: Testing the weathering explanation for the poor health of Black Americans. *Dev. Psychol.* **54**, 1993–2006 (2018).
46. G. H. Brody, T. Yu, G. E. Miller, E. Chen, Discrimination, racial identity, and cytokine levels among African-American adolescents. *J. Adolesc. Health* **56**, 496–501 (2015).
47. J. Wagner, R. Lampert, H. Tennen, R. Feinn, Exposure to discrimination and heart rate variability reactivity to acute stress among women with diabetes. *Stress Health* **31**, 255–262 (2015).
48. T. Lucas *et al.*, Perceived discrimination, racial identity, and multisystem stress response to social evaluative threat among African American men and women. *Psychosom. Med.* **79**, 293–305 (2017).
49. L. S. Hoggard, L. K. Hill, D. L. Gray, R. M. Sellers, Capturing the cardiac effects of racial discrimination: Do the effects “keep going”? *Int. J. Psychophysiol.* **97**, 163–170 (2015).
50. P. J. Sawyer, B. Major, B. J. Casad, S. S. M. Townsend, W. B. Mendes, Discrimination and the stress response: Psychological and physiological consequences of anticipating prejudice in interethnic interactions. *Am. J. Public Health* **102**, 1020–1026 (2012).
51. W. Boucsein, *Electrodermal Activity* (Springer, 2012).
52. S. D. Kreibitz, Autonomic nervous system activity in emotion: A review. *Biol. Psychol.* **84**, 394–421 (2010).
53. T. Reinhardt, C. Schmahl, S. Wüst, M. Bohus, Salivary cortisol, heart rate, electrodermal activity and subjective stress responses to the Mannheim Multicomponent Stress Test (MMST). *Psychiatry Res.* **198**, 106–111 (2012).
54. M. E. Dawson, A. E. Schell, D. L. Filion, “The electrodermal system” in *Handbook of Psychophysiology*, J. T. Cacioppo, L. G. Tassinari, G. G. Bernston, Eds. (Cambridge University Press, ed. 2, 2000), pp. 200–223.
55. H. D. Critchley, Electrodermal responses: What happens in the brain. *Neuroscientist* **8**, 132–142 (2002).
56. P. Sterling, “Principles of allostasis: Optimal design, predictive regulation, pathophysiology and rational therapeutics” in *Allotasis, Homeostasis, and the Costs of Adaptation*, J. Schulkin, Ed. (Cambridge University Press, 2004) pp. 17–64.
57. A. C. Phillips, A. T. Ginty, B. M. Hughes, The other side of the coin: Blunted cardiovascular and cortisol reactivity are associated with negative health outcomes. *Int. J. Psychophysiol.* **90**, 1–7 (2013).
58. A. Portes, R. G. Rumbaut, *Legacies: The Story of the Immigrant Second Generation* (University of California Press, Berkeley, CA, 2001).
59. M.-Z. Poh, N. C. Swenson, R. W. Picard, A wearable sensor for unobtrusive, long-term assessment of electrodermal activity. *IEEE Trans. Biomed. Eng.* **57**, 1243–1252 (2010).
60. C. G. Colen, D. M. Ramey, E. C. Cooksey, D. R. Williams, Racial disparities in health among nonpoor African Americans and Hispanics: The role of acute and chronic discrimination. *Soc. Sci. Med.* **199**, 167–180 (2018).
61. D. R. Bach, J. Daunizeau, N. Kuelzow, K. J. Friston, R. J. Dolan, Dynamic causal modeling of spontaneous fluctuations in skin conductance. *Psychophysiology* **48**, 252–257 (2011).
62. D. R. Bach, J. Daunizeau, K. J. Friston, R. J. Dolan, Dynamic causal modelling of anticipatory skin conductance responses. *Biol. Psychol.* **85**, 163–170 (2010).
63. M. Daly, L. Delaney, P. P. Doran, C. Harmon, M. MacLachlan, Naturalistic monitoring of the affect-heart rate relationship: A day reconstruction study. *Health Psychol.* **29**, 186–195 (2010).
64. D. Kahneman, A. B. Krueger, D. A. Schkade, N. Schwarz, A. A. Stone, A survey method for characterizing daily life experience: The day reconstruction method. *Science* **306**, 1776–1780 (2004).
65. S. P. Harrell, A multidimensional conceptualization of racism-related stress: Implications for the well-being of people of color. *Am. J. Orthopsychiatry* **70**, 42–57 (2000).
66. S. O. Lilienfeld, Microaggressions. *Perspect. Psychol. Sci.* **12**, 138–169 (2017).
67. A. D. Martinez, L. Ruelas, D. A. Granger, Household fear of deportation in relation to chronic stressors and salivary proinflammatory cytokines in Mexican-origin families post-SB 1070. *SSM Popul. Health* **5**, 188–200 (2018).
68. J. A. Russell, A circumplex model of affect. *J. Pers. Soc. Psychol.* **39**, 1161–1178 (1980).
69. D. R. Williams, S. A. Mohammed, Discrimination and racial disparities in health: Evidence and needed research. *J. Behav. Med.* **32**, 20–47 (2009).
70. M. S. Willis, O. Nkwocha, Health and related factors for Sudanese refugees in Nebraska. *J. Immigr. Minor. Health* **8**, 19–33 (2006).
71. E. C. Hope, L. S. Hoggard, A. Thomas, Emerging into adulthood in the face of racial discrimination: Physiological, psychological, and sociopolitical consequences for African American youth. *Transl. Issues Psychol. Sci.* **1**, 342–351 (2015).
72. E. C. Hope, M. B. Spencer, “Civic engagement as an adaptive coping response to conditions of inequality: An application of phenomenological variant of ecological systems theory (PVEST)” in *Handbook on Positive Development of Minority Children and Youth*, N. J. Cabrera, B. Leyendecker, Eds. (Springer International Publishing, 2017), pp. 421–435.

73. G. R. Sanchez, E. D. Vargas, H. L. Walker, V. D. Ybarra, Stuck between a rock and a hard place: The relationship between Latino/a's personal connections to immigrants and issue salience and presidential approval. *Polit. Groups Identities* **3**, 454–468 (2015).
74. C. Patler, W. Laster Pirtle, From undocumented to lawfully present: Do changes to legal status impact psychological wellbeing among latino immigrant young adults? *Soc. Sci. Med.* **199**, 39–48 (2018).
75. L. Wray-Lake *et al.*, Being a Latinx adolescent under a Trump presidency: Analysis of Latinx youth's reactions to immigration politics. *Child. Youth Serv. Rev.* **87**, 192–204 (2018).
76. R. J. Hernández, M. T. Villodas, Overcoming racial battle fatigue: The associations between racial microaggressions, coping, and mental health among Chicana/o and Latina/o college students. *Cultur. Divers. Ethnic Minor. Psychol.* **26**, 399–411 (2020).
77. P. G. Williams, T. W. Smith, H. E. Gunn, B. N. Uchino, "Personality and stress: Individual differences in exposure, reactivity, recovery, and restoration" in *The Handbook of Stress Science: Biology, Psychology, and Health*, R. J. Contrada, A. Baum, Eds. (Springer Publishing Company, 2011), pp. 231–245.
78. T. S. Conner, H. Tennen, W. Fleeson, L. F. Barrett, Experience sampling methods: A modern idiographic approach to personality research. *Soc. Personal. Psychol. Compass* **3**, 292–313 (2009).
79. G. Eisele *et al.*, The effects of sampling frequency and questionnaire length on perceived burden, compliance, and careless responding in experience sampling data in a student population. *Assessment*, 10.1177/1073191120957102 (2020).
80. R. E. Lucas, C. Wallsworth, I. Anusic, M. B. Donnellan, A direct comparison of the day reconstruction method (DRM) and the experience sampling method (ESM). *J. Pers. Soc. Psychol.*, 10.1037/pspp0000289 (2020).
81. E. Diener, L. Tay, Review of the day reconstruction method (DRM). *Soc. Indic. Res.* **116**, 255–267 (2014).
82. S. S. Dickerson, P. M. Zoccola, "Cortisol responses to social exclusion" in *The Oxford Handbook of Social Exclusion*, C. N. DeWall, Ed. (Oxford Library of Psychology, Oxford University Press, 2013), pp. 143–151.
83. N. L. Sin, J. E. Graham-Engeland, A. D. Ong, D. M. Almeida, Affective reactivity to daily stressors is associated with elevated inflammation. *Health Psychol.* **34**, 1154–1165 (2015).
84. A. J. Shallcross, T. M. Spruill, The protective role of mindfulness in the relationship between perceived discrimination and depression. *Mindfulness (N Y)* **9**, 1100–1109 (2018).
85. J. P. Jamieson, "Challenge and threat appraisals" in *Handbook of Competence and Motivation Theory and Application*, A. J. Elliot, C. S. Dweck, D. S. Yeager, Eds. (Guilford Publications, ed. 2, 2017) pp. 175–191.
86. J. P. Jamieson, E. J. Hangen, H. Y. Lee, D. S. Yeager, Capitalizing on appraisal processes to improve affective responses to social stress. *Emot. Rev.* **10**, 30–39 (2018).
87. M. D. Seery, The biopsychosocial model of challenge and threat: Using the heart to measure the mind. *Soc. Personal. Psychol. Compass* **7**, 637–653 (2013).
88. N. A. John-Henderson, M. L. Rheinschmidt, R. Mendoza-Denton, Cytokine responses and math performance: The role of stereotype threat and anxiety reappraisals. *J. Exp. Soc. Psychol.* **56**, 203–206 (2015).
89. P. Sterling, Homeostasis vs allostasis: Implications for brain function and mental disorders. *JAMA Psychiatry* **71**, 1192–1193 (2014).
90. M. Romero, Trump's immigration attacks, in brief. *Contexts* **17**, 34–41 (2018).
91. C. Miller, A. Werner-Winslow, *Ten Days After: Harassment and Intimidation in the Aftermath of the Election* (Southern Poverty Law Center, 2016).
92. Federal Bureau of Investigation, Hate Crime Statistics, <https://ucr.fbi.gov/hate-crime/> 2018 (2018). Accessed 25 September 2019.
93. K. Müller, C. Schwarz, From hashtag to hate crime: Twitter and anti-minority sentiment, *Social Science Research Network*, https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3149103 (2018). Accessed 30 May 2020.
94. L. Musu-Gillette, A. Zhang, K. Wang, J. Zhang, B. A. Oudekerk, Indicators of school crime and safety: 2018. *Homeland Security Digital Library*, <https://ies.ed.gov/pub-search/pubsinfo.asp?pubid=2019047> (2019). Accessed 30 May 2020.
95. M. Shah, M. Sachdeva, R. P. Dodiuk-Gad, COVID-19 and racial disparities. *J. Am. Acad. Dermatol.* **83**, e35 (2020).
96. M. Madjid, P. Safavi-Naeini, S. D. Solomon, O. Vardeny, Potential effects of coronaviruses on the cardiovascular system: A review. *JAMA Cardiol.* **5**, 831–840 (2020).
97. G. H. Brody *et al.*, Perceived discrimination among African American adolescents and allostatic load: A longitudinal analysis with buffering effects. *Child Dev.* **85**, 989–1002 (2014).
98. A. T. Geronimus, M. Hicken, D. Keene, J. Bound, "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United States. *Am. J. Public Health* **96**, 826–833 (2006).
99. B. J. Goosby, S. Malone, E. A. Richardson, J. E. Cheadle, D. T. Williams, Perceived discrimination and markers of cardiovascular risk among low-income African American youth. *Am. J. Hum. Biol.* **27**, 546–552 (2015).
100. M. Garbarino, M. Lai, D. Bender, R. W. Picard, S. Tognetti, "Empatica E3—A wearable wireless multi-sensor device for real-time computerized biofeedback and data acquisition" in 2014 4th International Conference on Wireless Mobile Communication and Healthcare—Transforming Healthcare Through Innovations in Mobile and Wireless Technologies (MOBIHEALTH) (IEEE, 2014), pp. 39–42.
101. R. W. Picard, S. Fedor, Y. Ayzenberg, Multiple arousal theory and daily-life electrodermal activity asymmetry. *Emot. Rev.* **8**, 62–75 (2016).
102. D. R. Bach *et al.*, Psychophysiological modeling: Current state and future directions. *Psychophysiology* **55**, e13214 (2018).
103. M. Benedek, C. Kaernbach, A continuous measure of phasic electrodermal activity. *J. Neurosci. Methods* **190**, 80–91 (2010).
104. E. K. Seaton, T. Yip, R. M. Sellers, A longitudinal examination of racial identity and racial discrimination among African American adolescents. *Child Dev.* **80**, 406–417 (2009).
105. W. Boucsein *et al.*; Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures, Publication recommendations for electrodermal measurements. *Psychophysiology* **49**, 1017–1034 (2012).
106. S. W. Raudenbush, A. S. Bryk, *Hierarchical Linear Models: Applications and Data Analysis Methods* (SAGE Publications, Inc, ed. 2, 2001).
107. P. D. Allison, *Fixed Effects Regression Methods for Longitudinal Data Using SAS* (SAS Institute, ed. 1, 2014).
108. J. E. Cheadle *et al.*, Race and ethnic variation in college students' allostatic regulation of racism-related stress. Open Science Framework. <https://osf.io/sbhd7/>. Deposited 6 November 2020.