Cardiovascular Reactivity During Conversations About Discrimination is Buffered by Social Support Among U.S. Latines

Hannah I. Volpert-Esmond[a]*

Jessica R. Bray[b]

Samantha M. Pages[a]

Chad Danyluck[c]

[a]University of Texas at El Paso, Department of Psychology, 500 W. University Ave, El Paso, TX, 79968

[b]University of Kentucky, Department of Psychology, Kastle Hall 115, Lexington, KY, 40506

[c]Carleton University, Department of Psychology, 1125 Colonel By Dr, Ottawa, Ontario, K1S 5B6, Canada

*corresponding author email: hivolpertes@utep.edu

Accepted for publication at Scientific Reports

Abstract

Racial discrimination is conceptualized as an acute and chronic stressor. Like other acute stressors, lab-based studies demonstrate acute effects of discrimination-related stressors on stress-related cardiovascular outcomes, including total cardiac output, blood pressure, and indices of sympathetic and parasympathetic nervous system activity. Critically, it is important to understand how individual and social factors buffer the experience of race-related acute stress. The current study extends existing work by measuring cardiovascular indices of stress during conversations about racial/ethnic discrimination and examines the moderating role of social support. Latine/Hispanic participants ($N = 97$) talked about personal discrimination experiences with either a close other or a research assistant they had never previously met. Participants in both conditions exhibited cardiovascular reactivity indicative of stress during the conversation. Additionally, patterns of reactivity reflected a more adaptive stress response and recovery profile when participants talked about discriminatory experiences with a close other relative to a stranger (less parasympathetic withdrawal during the stressor and more parasympathetic rebound during recovery). These patterns are consistent with a *stress buffering* account of social support, which suggests social bonds and community-level support are critical to consider in interventions to mitigate the harms of experiencing discrimination and prevent chronic health disparities.

Experiences of racial discrimination are pervasive for members of minoritized racial/ethnic groups. More than half of U.S. Latines, for example, report experiencing discrimination due to their race, perceived intelligence, and language proficiency [1]. Experiencing discrimination is linked to both psychological and physical health outcomes, including cardiovascular disease and mortality rates [2–5]. This relationship has been demonstrated for various minoritized racial/ethnic groups in the US, including Latine/Hispanic individuals [6–8]. To explain the relationship between discrimination and health, discrimination has been conceptualized as both an acute and chronic stressor (e.g., Clark et al., 1999). Within this perspective, repeated experiences of discrimination-related stress that activate momentary adaptive changes in cardiovascular, immune, and neuroendocrine functioning contribute over time to chronic dysregulation in physiological functioning, resulting in racial disparities in health across a broad spectrum of health outcomes. Critically, both individual-level and group-level factors have been identified that buffer the negative impacts of discrimination on health, including social support [10], although, the mechanisms by which social support may disrupt the negative impact of discrimination on health are understudied, especially for Latine/Hispanic people. The current study examines how culturally relevant social support among Latines reduces cardiovascular reactivity during conversations about discrimination, which has implications for reducing adaptive "wear and tear" on the body in the long term and may function as a protective factor for both physical and psychological health.

Discrimination as an Acute Stressor

Cardiovascular disease is among the leading causes of death in North America and is experienced more frequently by minoritized racial/ethnic groups [11,12]. Repeated experiences of racial/ethnic discrimination have been linked to increased risk for cardiovascular disease [4,13,14].

To understand this link, the *biopsychosocial model* [9] conceptualizes discrimination as a stressor similar to other physical or psychological stressors. In response to acute stressors, the human body activates the sympathetic nervous system and deactivates the parasympathetic nervous system, similar to patterns seen during increased metabolic demand, such as exercise [15]. This stress response is a state of high energy expenditure and, once the stressor has passed, the body rapidly returns to a more energy-efficient resting state [16]. When acute stressors accumulate over time, however, without adequate opportunity for the individual to recover, such stressors become chronic, contributing to load on systems that regulate responses to environmental changes and, ultimately, producing the kind of bodily wear and tear that can result in cardiovascular disease.

In support of the conceptualization of discrimination as an acute stressor, experimental lab studies demonstrate that perceiving discrimination results in stress reactivity: increased blood pressure and heart rate [17–19], decreased parasympathetic activity [20–22], increased sympathetic activity [22–24] and increased cortisol production [25]. Over time, repeated experiences of discrimination and resulting stress responses can contribute to cumulative load in these regulatory systems that leads to racial disparities in physical health, including in cardiovascular, immune, and neuroendocrine functioning [14,26]. Although much of this research has been done with Black participants, studies have shown both acute and chronic effects of discrimination among Latine/Hispanic participants, including acute effects on heart rate, blood pressure, sympathetic and parasympathetic activity, and cortisol [24,27–29], as well as chronic, lifetime effects on physical health [30–33]. Latines are the largest ethnic minority group in the U.S. and report experiencing discrimination at similar rates as other racial/ethnic minority groups within the U.S. [34,35] and thus compose an important but understudied group.

Conversations about Discrimination in the Lab

Lab-based research on the acute effects of discrimination often ask participants to imagine their responses to written or audio/visual vignettes of discrimination [17,19,20,23,36–39]. For example, an early study examining blood pressure had participants view video clips that were either neutral, racist, or anger-provoking [40]. Another study had Black participants listen to a 2-minute audio clip describing an incident involving unfair treatment of a person while shopping and give a 5-minute speech on how they would feel and react if they were in that scenario [18]. Other work has involved simulation of discrimination in the lab, typically using confederates [27–29,41–46]. For example, Hoggard et al[47] included a manipulation where Black participants "accidentally" overheard a conversation between the experimenter and confederate describing the participant as intellectually inferior because of their race. In another study, Latine participants were randomly assigned to an interaction with the experimenter that included a microinsult ("You speak English really well") or a microinvalidation ("Where are you really from?") or no discrimination [27]. Recently, additional technologies such as virtual reality have been used to increase the realism of such simulations [45].

Such lab-based work has the benefit of making the discrimination exposure consistent across participants; however, depending on participants' own experiences, imagined or simulated discrimination in the lab may or may not reflect a participant's own lived experiences. In response to this, some work has asked participants to recall their own experiences of discrimination [22,48], which has the benefit of making experiences salient that have personal relevance to the participant. The present study expands on this by examining Latine participants' own experiences of discrimination through discussion with a conversation partner while cardiovascular stress reactivity is measured.

Social support as a protective factor

Given the prevalence of experiencing discrimination and its negative consequences, it is important to consider protective buffering factors that can mitigate the negative effects of discrimination. Although Latines experience higher levels of obesity, diabetes, and other cardiometabolic risk factors than non-Latine Whites [49–51] and worse social determinants of health (e.g., income, access to health services, exposure to contagious illnesses),[52,53] some studies show that Latines and Mexican-Americans in particular do not experience worse cardiovascular health or mortality in general, possibly because of effective socio-cultural buffering factors [51]. Several protective factors that may mitigate the effect of stress and discrimination on health are particularly important for Latines, including cultural values of familism and resulting social support prevalent in Latine communities. Familism refers to the cultural emphasis on one's family as the main source of emotional and instrumental social support when needed and includes elements of loyalty, reciprocity, and solidarity within one's family [54–57]. Familism has been shown to have a protective effect by promoting resilience among those experiencing acculturative conflict and is related to lower levels of internalizing symptoms, depression, and suicidality [57–63]. However, it is unclear whether and how strong family relationships attenuate the negative effect of discrimination on cardiovascular stress and mental health.

One possibility is the *stress-buffering hypothesis,* which suggests that close others and social relationships affect cardiovascular, immune, and neuroendocrine functioning during the experience of stress, which reduces allostatic load and potentially prevents the development of chronic health disparities [64]. The ability for social relationships to buffer stress, including cardiovascular functioning, is well established with non-Hispanic participants and non-race-specific lab-based stressors [65,66]. Passive, physical, and verbal forms of social support have all been shown to buffer cardiovascular stress responses [67–69]. Additionally, both strangers (e.g., lab

confederates) as well as existing supports (e.g., friends, family members, significant others) have been shown to function as effective stress-buffers in the lab [70–72]. However, most experimental research testing the stress-buffering hypothesis has included only non-Hispanic White participants reacting to non-race-specific forms of stress.

Given the buffering effect of social support on cardiovascular stress reactivity among non-Hispanic White participants, as well as the positive effect of social support in other health domains among Latines, it stands to reason that familism and social support among Latines may buffer the negative effects of discrimination-related stress on physical health, specifically via cardiovascular reactivity to acute stressors. Two studies to date have examined the role of familism among Latines in moderating cortisol reactivity during a race-neutral lab-induced stressor with mixed results [73,74]. In both studies, familism was measured prior to the stressor via self-report, and there was no element of social support (i.e., the presence of another person) during the stressor. The current study applies a social support paradigm used in other stress buffering research to test the possibility that the presence of family support *during* a race-specific stressor (e.g., recalling a personal experience of racial/ethnic discrimination) reduces cardiovascular stress reactivity, which may mitigate the negative impact of discrimination on downstream health outcomes.

Current study

The current study extends existing work by testing the possibility that the presence of close others during recall of a personal experience of racial/ethnic discrimination buffers the acute response typically experienced during a race-relevant stressor, thereby functioning as a protective factor for both physical and psychological health. In our paradigm, participants brought someone they were personally close with to the lab and had a conversation about their

own personal experiences with discrimination, either with that close other or with a Latine laboratory research assistant they had never met before. Indices of cardiovascular stress reactivity were measured during a 5-minute baseline, the 10-minute conversation about the participant's experiences with discrimination (Discrimination Speaking Task; DST), and a 5-minute recovery period. We hypothesized that the DST would induce stress reactivity and changes in momentary affect and that social support from close others would buffer stress. The moderating roles of ethnic identity, familism, and prior experiences with discrimination on stress reactivity were explored.

We report three indices relevant to cardiovascular stress reactivity: Interbeat interval (IBI), cardiac sympathetic index (CSI), and respiratory sinus arrhythmia (RSA). IBI is the inverse of heart rate, measured as the amount of time in milliseconds between R spikes in the electrocardiograph (ECG) waveform, and is a global measure of hemodynamic function reflecting influences of both sympathetic (SNS) and parasympathetic nervous system (PNS) activation. Acute psychological stress tasks consistently elicit decreases in IBI (less time between heart beats) in response to acute stress [15]. Since IBI is a multiply determined cardiovascular end point, we additionally indexed SNS and PNS influences separately using CSI and RSA, respectively. Both are derived from rhythmic fluctuations in heart rate during respiration and capture unique autonomic influences on the heart. SNS and PNS activity, which we measure with CSI and RSA respectively, have potentially differential consequences for long-term health[15].

CSI, a putative measure of SNS activity, is derived from a Lorenz plot, where each IBI is plotted against the subsequent IBI [75]. The transverse axis reflects beat-to-beat variation in the IBI time series while the longitudinal axis reflects the IBI range. CSI, the ratio of L/T, is unaffected by parasympathetic blockades but decreases with sympathetic blockades in healthy adults [75]. A

reasonable amount of evidence suggests that CSI is a useful index of sympathetic influence over the heart. CSI increases with administration of intravenous cocaine [76]. Changes in CSI relate to mental disorders linked with dysregulated SNS activity [77,78]. CSI also increases in response to novel, socially evaluative tasks [79]. Evidence for CSI changes across tasks is somewhat equivocal [75,80]; however, some have found that it reliably discriminates between resting and stressor tasks[81]. In contrast, RSA, thought to reflect PNS influences over the heart through the vagus nerve, is derived by performing spectral analysis on the high-frequency respiration band of the IBI time series data. RSA is often interpreted as a biomarker of self-regulation in response to rapidly changing environmental demands [82–84].

During acute stress, SNS activity increases due to beta-adrenergic sympathetic activation in a "fight or flight" response before returning to baseline once the stressor has passed. Over-activation of this response has been associated with anxiety disorders and PTSD [85,86]. In response to stress, PNS activity typically decreases, representing vagal withdrawal or an orienting response in preparation for motor or emotional responses to match the situation. PNS withdrawal itself can be adaptive, as it prepares an individual to react to a stressor or stimulus. However, heightened withdrawal is considered maladaptive, as it indicates an "over-preparation" for the stressor and results in over-expenditure of physiological resources that do not match the metabolic demand [83,87,88]. Once the stressor has passed, PNS activity typically increases past baseline levels ("vagal rebound") before returning to baseline[84,89]. Lack of vagal rebound predicts development of cardiovascular disease, including chronic hypertension[90].

Results

Manipulation Checks

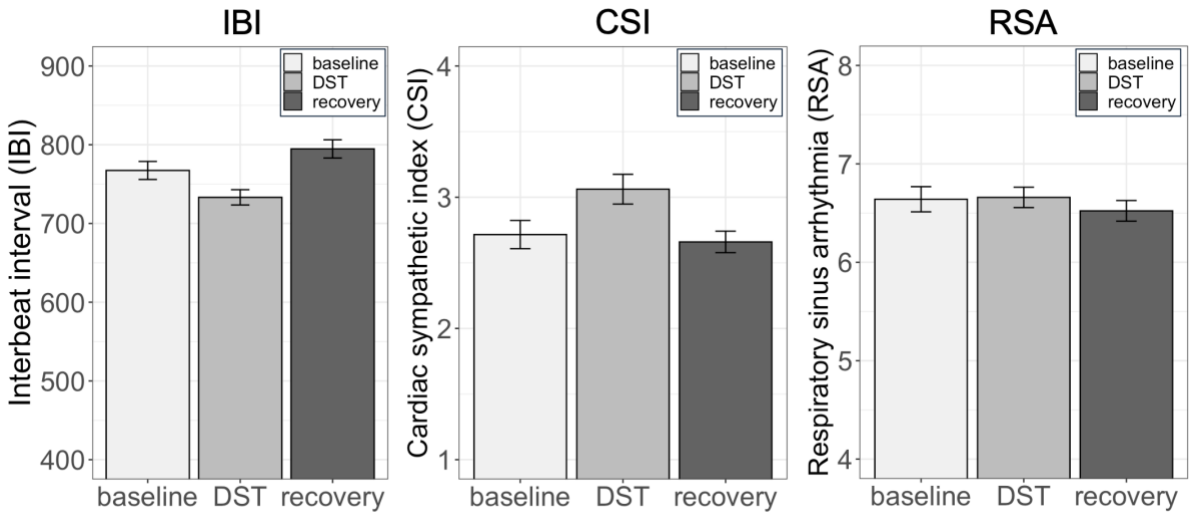
Was the DST stressful? First, we checked whether the Discrimination Speaking Task was stressful by comparing IBI, RSA, and CSI across the three periods (baseline, DST, and recovery; Figure 1). We used a mixed effects model to test each outcome separately, with a random intercept by Participant to account for the repeated measures nature of the data, along with Period as the fixed effect. The following covariates were additionally included: BMI (mean-centered), age (mean-centered), gender (0 = woman, 1 = man, 2 = trans/non-binary), alcohol in the last 24 hours (0 = no, 1 = yes), caffeine in the last 2 hours (0 = no, 1 = yes), and whether the participant ate in the last 2 hours (0 = no, 1 = yes). Analyses for unadjusted models are included in the Supplementary Material. We used the lme4 [91] and lmerTest [92] packages in R (version 4.2.1) to fit the models and the emmeans package [93] for post-hoc comparisons (all reported means are model-estimated means). Data and code for analysis can be found at [https://osf.io/28bnz/].

As expected, there were significant changes in IBI across time periods (Figure 1). IBI decreased significantly from the baseline period (M = 807 ms, SE = 40 ms) to the DST period (M = 773 ms, SE = 40 ms), $t(160) = -7.2$, $p < .001$, Cohen's $d = -1.13$. and then increased significantly to the recovery period (M = 834 ms, SE = 40 ms), $t(160) = 12.7$, $p < .001$, Cohen's $d = 1.99$, which is consistent with the conceptualization of the DST as an acute stressor. Our measure of sympathetic activity—CSI—was consistent with this conceptualization as well. CSI significantly increased from the baseline period (M = 2.78, SE = 0.30) to the DST period (M = 3.13, SE = 0.30), $t(160) = 3.1$, $p = .006$, Cohen's $d = 0.49$, and then significantly decreased to the recovery period (M = 2.73, SE = 0.30), $t(160) = -3.5$, $p = .002$, Cohen's $d = -0.55$. However, RSA did not significantly change from the baseline period (M = 6.89, SE = 0.37) to the DST period (M

= 6.90, *SE* = 0.37), $t(160) = 0.1$, $p = .997$, Cohen's $d = 0.01$, nor from the DST period to the recovery period ($M = 6.77$, $SE = 0.37$), $t(160) = -1.3$, $p = .406$, Cohen's $d = -0.20$.

Figure 1

IBI, CSI, and RSA, Separately by Period



Some significant effects of covariates were noted. There was a significant effect of eating within the last 2 hours on IBI, such that eating corresponded with a lower IBI, $b = -45.4$, $t(73) = -2.0$, $p = .046$, $d = -1.48$. There was a significant effect of age on CSI, $b = 0.06$, $t(73) = 2.8$, $p = .007$, $R^2_{semi\text{-}partial} = 0.053$, and on RSA, $b = -0.06$, $t(73) = -2.3$, $p = .027$, $R^2_{semi\text{-}partial} = 0.043$, where being older was associated with a higher CSI and a lower RSA. All other effects of covariates were not significant.

Did perceived support differ across experimental conditions? Our experimental manipulation (i.e., whether participants talked about their experiences of discrimination with a close other versus a stranger during the DST) was intended to manipulate the level of social support the participant received during the stressor. To check this manipulation, we compared level of perceived social support during the DST according to experimental condition.

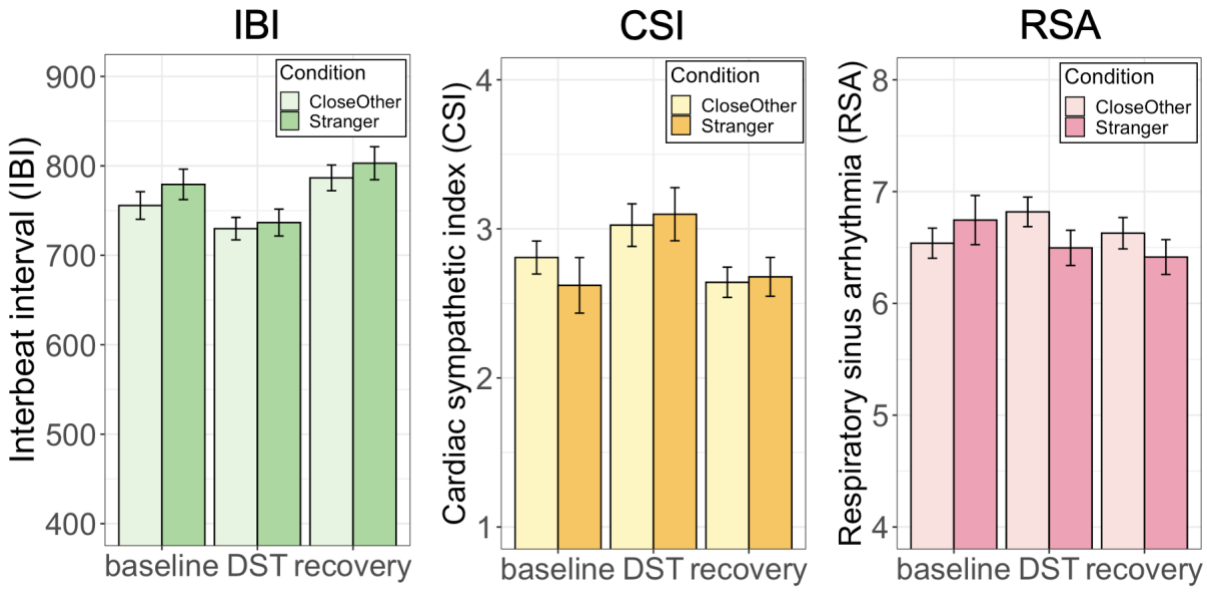
Participants in the stranger condition reported significantly lower levels of perceived support (M = 3.67, SE= 0.12) than participants in the close other condition (M = 4.45, SE = 0.12), $t(81)$ = 4.7, $p < .001$, Cohen's d = 1.04, suggesting strangers were perceived as less supportive than close others, as expected.

Effect of Condition on Cardiovascular Stress

To examine the effect of condition, we added condition as a fixed predictor to the previously describe multilevel models including all covariates and examined the Condition × Period interactions.

Figure 2

IBI, RSA, and CSI, Separated by Period and Experimental Condition (Close Other versus Stranger)



IBI. There were no significant interactions between period and condition predicting IBI (see Figure 2). Although there was a slightly larger decrease in IBI from baseline to DST in the stranger condition (M_{diff} = -42.7 ms, SE_{diff} = 6.8 ms) relative to the close other condition (M_{diff} = -26.7 ms, SE_{diff} = 6.9 ms), this difference was not significant, $t(158) = 1.7$, $p = .100$, $R^2_{semi\text{-}partial}$ =

0.00. There was also no significant difference across conditions in change from DST to recovery, $t(158) = 1.1$, $p = .283$, $R^2_{semi\text{-}partial} = 0.00$.

CSI. There were no significant interactions between period and condition predicting CSI, including no differences as a function of condition from baseline to DST, $t(158) = 1.06$, $p = .289$, $R^2_{semi\text{-}partial} = 0.00$, or from baseline to recovery, $t(158) = -0.16$, $p = .870$, $R^2_{semi\text{-}partial} = 0.00$.

RSA. There was a significant interaction between period and condition, specifically when considering change in RSA from baseline to DST, $t(158) = 2.52$, $p = .013$, $R^2_{semi\text{-}partial} = 0.01$. Participants in the stranger condition showed a decrease in RSA from baseline to the DST period ($M_{diff} = -0.25$, $SE_{diff} = 0.15$), whereas participants in the close other condition showed an increase ($M_{diff} = 0.27$, $SE_{diff} = 0.15$), indicating more PNS withdrawal to the stressor in the stranger condition. There was no significant difference between conditions in change scores from the DST to recovery, $t(158) = 0.52$, $p = .607$, $R^2_{semi\text{-}partial} = 0.00$. We additionally examined the difference between baseline and recovery to look for evidence of vagal rebound. There was a significant difference across conditions, such that participants in the close other condition experienced a pattern of vagal rebound where RSA was higher during recovery than baseline ($M_{diff} = 0.08$, $SE_{diff} = 0.15$), whereas participants in the stranger condition had lower RSA during recovery than baseline ($M_{diff} = 0.03$, $SE_{diff} = 0.23$). Although the effect of condition on recovery was significant (i.e. a cross-over interaction), $t(158) = -2.0$, $p = .046$, $R^2_{semi\text{-}partial} = 0.01$, each of the simple effects was not significant, making conclusions about vagal rebound difficult.

Moderators of Cardiovascular Reactivity

We additionally examined potential moderators of stress reactivity, focusing specifically on changes in IBI, CSI, and RSA from baseline to DST. Here, we used multilevel models, only including the baseline and DST periods, to look at the interaction between period and each

individual difference variable. All covariates were included, as before, and all individual difference variables were standardized. Perceived social support during the DST, ethnic identity, and familism values did not significantly moderate stress reactivity, *p*s > .05 (see Table 1). However, frequency of experiencing discrimination in the past year did significantly moderate CSI reactivity from baseline to stress, such that reporting more frequent discrimination corresponded to a stronger sympathetic stress response in the DST, relative to baseline. Exploratory follow-ups examining each discrimination item separately revealed that this relationship with CSI reactivity was driven primarily by vicarious experiences of discrimination ("In the past year, how often did you see or witness anti-Mexican or anti-Hispanic statements or behaviors in person?"), $b = 0.32$, $t(79) = 2.5$, $p = .016$, $R^2_{semi\text{-}partial} = 0.026$.

Table 1

Moderation of Cardiovascular Reactivity from Baseline to Stress by Individual Difference Variables

	ΔIBI			ΔCSI			ΔRSA		
	b	*p*	$R^2_{semi\text{-}partial}$	*b*	*p*	$R^2_{semi\text{-}partial}$	*b*	*p*	$R^2_{semi\text{-}partial}$
Perceived support	0.05	.993	.000	0.08	.541	.002	-0.01	.923	.000
Ethnic identity (commitment)	-7.71	.137	.002	0.16	.243	.006	-0.04	.764	.000
Ethnic identity (exploration)	-8.28	.109	.002	0.11	.422	.003	-0.20	.105	.008
Familism (support)	-2.01	.700	.000	0.18	.192	.008	-0.16	.196	.005
Familism (obligation)	-0.03	.996	.000	0.09	.506	.002	-0.10	.404	.002
Familism (referent)	-2.93	.573	.000	0.11	.441	.003	-0.11	.380	.003

Past-year Discrimination	-8.90	.084	.002	**0.30**	**.028**	**.022**	-0.11	.367	.003

Note. Beta estimates represent the interaction between the individual difference variable and period in multilevel models that include all covariates.

Interactions between individual difference variables and condition were additionally examined, but no moderating effects were significant, suggesting the effect of condition on reactivity did not significantly differ according to any individual difference variables.

Changes in Self-Reported Affect

In addition to cardiovascular measures of stress, we examined fluctuations in momentary affect, which participants reported during baseline and following the DST period at the beginning of recovery. We expected participants to show increases in negative affect, anxiety symptoms, and depressive symptoms, and decreases in positive affect, consistent with prior research showing an acute effect of discrimination-related stress on psychological wellbeing. Using paired t-tests, we did find a small decrease in positive affect, M_{diff} = -0.16, SD_{diff} = 0.71, $t(80) = -2.0$, $p = .049$, $d = .22$, but no change in negative affect, M_{diff} = 0.06, SD_{diff} = 0.48, $t(80) = 1.1$, $p = .297$, $d = 0.12$, or depressive symptoms, M_{diff} = 0.01, SD_{diff} = 0.51, $t(80) = 0.2$, $p = .886$, $d = 0.02$. Additionally, we found a significant *decrease* in anxiety symptoms from baseline to post-DST, M_{diff} = -0.26, SD_{diff} = 0.76, $t(80) = -3.1$, $p = .003$, $d = 0.35$, which was unexpected and not consistent with the conceptualization of the DST as an acute psychological stressor. There were no differences in change in affect by condition.

Discussion

Given the prevalence of racial discrimination and its consequences for both mental and physical health [4,14], it is important to identify buffering factors that reduce the negative impact of discrimination on health. Among Latine communities, familism and social support may be

particularly important to consider. In the current study, we brought participants to the lab to recall experiences with discrimination in a conversation with either a close other (such as family member or friend) or a stranger. Cardiovascular measures of stress reactivity were examined to test the stress-buffering hypothesis, which suggests that social support during a stressful event may reduce cardiovascular stress responses and alleviate the long-term effect of repeated race-related stress activation.

Overall, we found some evidence that having a conversation about past experiences of discrimination was stressful, in that participants displayed a typical physiological stress response during the DST relative to baseline (e.g., decreases in IBI and increases in CSI). These changes are consistent with the cardiovascular profile exhibited when confronting or encountering a stressor [94] and are similar to cardiovascular changes displayed in response to other racial discrimination stressors[21,22]. However, differences in condition (e.g., whether participants discussed the experience of discrimination with a close other versus a stranger) were only apparent in parasympathetic reactivity. Specifically, people who discussed past discrimination experiences with a close other displayed less PNS withdrawal and a more adaptive pattern of recovery than people who shared their experiences with a stranger.

Whereas resting PNS activity is consistently associated with positive outcomes (e.g., effective emotion- and self-regulation abilities),[95] patterns of PNS reactivity in response to stress can be more variable and dependent on the increased demands related to the stressor, including demands for top-down executive processing. When a higher activity level is required in response to a stressor and metabolic demands are important, a higher vagal withdrawal response is associated with more effective self-regulation. However, in situations that require more top-down executive functioning and top-down control, a smaller vagal control response may be more

adaptive [83,96]. In the case of conversations about discrimination, which can be a socially sensitive topic requiring emotion regulation and little physical metabolic demand, strong vagal withdrawal may be counterproductive to successfully navigating these conversations. Here, we saw a larger PNS withdrawal response in participants having a conversation with a stranger relative to a close other. This pattern is similar to prior studies observing higher PNS reactivity in situations where discrimination was seen as more threatening [20,21,23]. One possibility is that participants with social support from a close other positively reappraised the discrimination experience and felt they had more psychosocial resources to deal with the stressor, characterized by strong sympathetic activation and less parasympathetic withdrawal. This physiological profile of a challenge response is similar to challenge responses exhibited by participants who were administered oxytocin prior to a social stressor [97].

Additionally, patterns of recovery were significantly different in the close other condition relative to the stranger condition. Vagal rebound in recovery is characterized by an increase in PNS activity that exceeds resting levels following a stressor and is a compensatory mechanism to restore homeostasis. Enhanced vagal rebound is observed in individuals with greater aerobic fitness following exercise [89] and decreased in individuals with a family history of cardiovascular disease [90] or emotion regulation difficulties [98]. Participants in the stranger condition showed a lack of vagal rebound, suggesting more difficulty regulating emotionally following the conversation about discrimination and has been found in other studies examining recovery following a race-based stressor [20,21].

Together, these patterns suggest a more adaptive stress response and recovery profile when participants described their experiences of racial discrimination to a close other relative to a stranger, identifying an important pathway by which social support may modulate stress

responses experienced during recall of discrimination. This mechanism is especially important to consider given recent attention to rumination as a coping mechanism following experiences of discrimination, and how continued processing and reflection on a negative experience contributes to long-term health consequences [99–102].

In an exploratory way, we additionally investigated several individual difference factors that may affect cardiovascular reactivity when having conversations about racial discrimination, including ethnic identity, cultural values of familism, and frequency of past experiences of discrimination. The only significant moderating factor was how frequently an individual had experienced racial discrimination within the past year, and specifically, how often they had vicariously experienced discrimination ("How often did you see or witness anti-Mexican or anti-Hispanic statements or behaviors in person?"). Recent research on vicariously experienced discrimination distinguishes it from directly experienced discrimination and can encompass seeing, hearing, or learning about others' experiences with discrimination either in person from friends, family, or strangers, or through online social networks or the news[103]. People from racial/ethnic minority groups, including Latines, experience discrimination vicariously more often than directly [104,105], and can experience negative consequences as a result, including psychological distress, sleep disturbances, and other adverse health-related outcomes[106,107]. The current results are consistent with previous studies showing that frequent experiences of discrimination over time alters individuals' autonomic functioning and stress reactivity[13]. Although the current results are exploratory, future research should continue to examine the consequences of vicariously experienced discrimination and mechanisms by which vicarious discrimination influence both physiological and psychological outcomes.

Although this study provides preliminary evidence of an intriguing physiological mechanism by which discrimination-related stress may be buffered, there were several limitations that should be considered when interpreting results. First, during the Discrimination Speaking Task (DST), participants discussed their own experiences with discrimination, which were often variable in terms of content and emotional impact. In some cases, participants said they had never experienced discrimination and did not initially know what to talk about, possibly because of the unique characteristics of the El Paso area (e.g., 80% of the population is Latine, making Latines the majority group), or alternatively described experiences of intra-group discrimination (e.g., being excluded for not being "Mexican enough"), a less-studied form of racial discrimination. We did not audio or video record what participants talked about during the DST, so we have no way of controlling for or accounting for the variability in the conversations. Since reporting more experiences of discrimination in the past-year was related to greater increases in SNS activity, one possibility is that participants who had more frequently experienced discrimination recently had more emotionally laden experiences to discuss during the DST, prompting greater stress reactivity.

We expected increases in negative affect, depression, and anxiety related symptoms and decreases in positive affect following the DST to match psychological stress responses captured following other acute stressors, whether race-related or not [45,108]. However, we did not see this pattern and instead saw *decreases* in anxiety following the conversation. One possibility is that since participants completed the baseline questionnaire after physiological recording equipment had already been applied, the baseline period captured some anticipatory processes while preparing for the conversation or responses to the unfamiliar lab environment and equipment, including elevated anxiety. Thus, the baseline questionnaire may not have captured a true

baseline. Additionally, and importantly, the conversation about discrimination may not have been experienced as a purely negative stressor, as other lab-based manipulations of discrimination have been in the past. Instead, because of the nature of the give-and-take of a conversation, especially when a close other is present, these conversations may have been cathartic and affirming. This may especially be the case because in both conditions, participants talked with a racial/ethnic ingroup member (e.g., a Latine conversation partner). Thus, a conversation about shared experiences may have elicited a shared group identity that may have also buffered momentary negative affect, even when talking with a stranger. Other research has begun to examine the positive effects of difficult conversations about race on relationship quality and positive psychological outcomes, providing an opportunity for bonding and other long term positive effects [109–112]. Thus, conversations about discrimination may have more nuanced consequences than an isolated simulation of discrimination because of the complexity of interpersonal dynamics, which may be either mediated or moderated by stress responses and autonomic system related emotional regulation.[79]

Last, we did not measure respiration rate to adjust for respiratory influences when examining RSA. Because participants were having a conversation during the DST but were at rest during baseline, differences in respiration related to speech production create a possible confound in interpreting any differences in RSA between the baseline and DST periods, as RSA can reflect changes in respiration that are independent of central vagal effects [113,114]. However, we found no overall differences in RSA across periods and instead, our main effects of interest concerned differences in RSA reactivity across conditions. As systematic differences in speech production were not expected across conditions, issues related to controlling for respiration are

less likely to influence our conclusions. Future research should consider adjusting for respiratory influences related to speech production to strengthen conclusions.

Taken together, our work highlights the protective role of close social ties in managing the impacts of racial/ethnic discrimination on physiological mechanisms relevant to cardiovascular health. When people discuss their discrimination experiences in the presence of close others, they mitigate immediate harm and enhance recovery. Thus, the current study suggests that working with close others to reflect on or process instances of discrimination or racial trauma might be a resource for minoritized racial communities to draw from to improve their long-term health and well-being. Further work is needed to assess how and why conversations are beneficial, and what strategies can be used to maximize the positive benefits of social support.

Method

Participants

The project was approved by the University of Texas at El Paso IRB and was performed in accordance with all relevant guidelines and regulations. Participants were recruited from undergraduate psychology classes and the surrounding community via the university SONA system, flyers, and campus announcements. Interested participants completed an eligibility screening survey and were selected for participation if they 1) were 18 years of age or older, 2) identified as Latina, Latino, Latinx, or Hispanic, 3) were fluent in English, and 4) were able to bring another person to the lab with whom they had a close relationship that spoke English fluently ("close other") and also identified as Latina, Latino, Latinx, or Hispanic. Additionally, prospective participants were excluded from participating if they had a BMI over 35 or used medication likely to affect cardiovascular functioning, including antidepressant, antipsychotic, or

antihypertensive medication. Participants were compensated with course credit or a $30 electronic gift card for their participation. Additionally, the person who accompanied the participant to the study ("close other") received a $20 electronic gift card in compensation.

A total of 97 people participated in the study (78 female, 14 male, 3 non-binary, and 2 not reported) from October 2021 to December 2022. Sample size was not determined by an *a priori* power analysis; Instead, we simply recruited as many participants as possible within a given window of time, as recruitment of participants and their close others to come to an in-person lab session was challenging and slower than expected, especially due to ongoing COVID-related concerns. The age of participants ranged from 18 years to 48 years (M =20.9, SD =4.4). The close others that participants brought with them to the lab included romantic partners (n = 22), parents (n = 27), siblings (n =14), close friends (n = 24), cousins (n = 7), and their own children over 18 years (n = 3). Data from 1 participant were excluded due to equipment failure, and cardiovascular data from 13 participants were excluded due to artifacts in the data, resulting in a final sample size of 83 participants. To conduct a sensitivity power analysis, we used G*Power to determine that the smallest effect size detectable with 80% power for a within-between interaction (e.g., the hypothesized Period x Condition interaction) in a repeated-measures ANOVA with the final sample size (n = 83) was f = .18.

Procedure

Following informed consent, physiological recording equipment was applied to the participant. Participants then completed a baseline questionnaire that took approximately 10 minutes and included items related to demographics, ethnic identity, values of familism, and information about the person they brought with them to the lab, including their race/ethnicity and relationship to the participant (see Measures).

Once the recording equipment was applied, ECG and EDA data were recorded during three distinct periods: baseline (5 minutes), Discrimination-Speaking Task (DST; 10 minutes), and recovery (5 minutes). Although EDA data were recorded, they are not included in this report because of equipment-related issues affecting the quality of data. During the baseline period, participants were asked to sit alone in the recording room with no distractions. Then, a conversation partner entered the room, and participants were given instructions for the Discrimination Speaking Task (DST). The conversation partner was randomly assigned to be either the "close other" that the participant had brought with them to the lab or a research assistant whom the participant had never previously met ("stranger" condition). For the first few minutes, participants were instructed to describe to their conversation partner an instance of racial/ethnic discrimination that they had experienced without interruption. The conversation partner was instructed to respond naturally once the participant had finished describing their experience, and the conversation was allowed to continue for the remainder of the ten minutes. In both conditions, the conversation partner matched the participant's race/ethnicity (e.g., identified as Latine). The conversation during the DST was not audio or video recorded to increase the comfortability of participants and their close others to speak openly about potentially sensitive topics. Research assistants who interacted with participants in the stranger condition did have knowledge of the experimental manipulation but were not instructed to respond in a particular manner and instead received the same instructions as the close other to respond in a way that felt natural, as if someone initiated a similar conversation outside the lab. Two research personnel were present at each lab session so that the research assistant who the participant interacted with during the DST was not the experimenter who conducted the consent

process and placed the physiological recording equipment on the participant at the beginning of the study. A total of seven research assistants acted as the stranger throughout data collection.

Following the DST, the conversation partner was asked to leave the room and the participant completed a short recovery questionnaire that took less than 2 minutes (see Measures). The participant was then left alone for a 5-minute recovery period with no distractions. Following the recovery period, the recording equipment was removed, and the participant was debriefed about the nature of the study. The entire procedure took about 1.5 hours.

Measures

Physiological Measures

The electrocardiogram (ECG) signal was measured with no online filter and a sampling rate of 512 Hz using 4 Ag/AgCl electrodes (right collarbone, left collarbone, CMS/DRL on left forearm) using an ActiveTwo BioSemi data acquisition system (BioSemi, Amsterdam, Netherlands). An offline band filter from 0.1-50 Hz was applied to the digitized data before automated detection of R spikes for extraction of interbeat intervals using EMSE (version 5.6.1). All automated event markers were visually inspected and misplaced markers were corrected manually. Participants with excessive missing markers (i.e., IBIs $>$ 1200 ms) within a period were excluded, rather than relying on imputation, which resulted in the exclusion of 13 participants. Once markers had been inspected, IBI series were produced separately for each participant and each time period. Mean IBI, CSI [75], and RSA (natural log of band-limited [.12-.40 Hz] variance of IBI series) were calculated for each time period using CMetX[81].

Baseline Questionnaire

Cardiovascular Covariates. To account for their effects on cardiovascular functioning and reactivity, we asked participants to report their height (inches) and weight (pounds) to calculate body mass index (BMI), age, and gender (man, woman, trans/non-binary, other, prefer not to say). Participants additionally reported if they had consumed alcohol within the last 24 hours, caffeine (e.g. coffee, tea, energy drinks) within the last 2 hours, and whether they had eaten within the last 2 hours.

Ethnic Identity. The Multigroup Ethnic Identity Measure - Revised (MEIM-R)[115] was used to measure both identity commitment (3 items) and identity exploration (3 items). Items assessing identity commitment included statements such as, "I have a strong sense of belonging to my ethnic group." Items assessing identity exploration included statements such as "I have often done things that will help me understand my ethnic background better." Participants were asked to indicate how much they agree with each statement on a scale of 1 (strongly disagree) to 5 (strongly agree). Internal consistency within our sample was acceptable for each subscale (commitment $\alpha = .86$; exploration $\alpha = .88$). Separate composite scores were created for each facet, such that higher scores represented more identity commitment or identity exploration.

Familism Values. The three familism subscales from the Mexican American Cultural Value Scale[59] were used to assess Mexican American cultural values related to support, obligation, and family as referent. Familism support values refer to the desirability to maintain close relationships (e.g., "Family provides a sense of security because they will always be there for you"; 6 items). Familism obligation values refer to the importance of tangible care giving (e.g., "If a relative is having a hard time financially, one should help them out if possible"; 5 items). Familism referent values refer to the reliance on communal interpersonal reflection to define the self (e.g. "A person should always think about their family when making important

517 decisions"; 5 items). Participants were asked to indicate how much they believe each statement
518 on a scale of 1 (not at all) to 5 (completely). Internal consistency within our sample was
519 acceptable for each scale (support $\alpha = .87$; obligation $\alpha = .78$; referent $\alpha = .80$). Separate
520 composite scores were created for each facet, such that higher scores represented stronger
521 familism values.

522 **Past-year Racial/Ethnic Discrimination.** We created 5 items to assess past-year
523 discrimination, including both direct experiences of discrimination and indirect (vicarious)
524 experiences. Additionally, items separately asked about online versus in-person experiences and
525 rumination about discrimination ("In the past year, how often…were you treated unfairly or
526 poorly because of your race/ethnicity in person?; …were you treated unfairly or poorly because
527 of your race/ethnicity on social media or the internet in general?; …did you see or witness anti-
528 Mexican or anti-Hispanic statements or behaviors in person?; …did you see or witness anti-
529 Mexican or anti-Hispanic statements or behavior on social media or in the news?; …did you
530 think about racial injustices and the mistreatment of Latino/a, Hispanic, or other people of color
531 in the U.S.?"). Participants were asked to indicate how often they experienced each item on a
532 scale of 1 (never) to 5 (almost every day). Internal consistency within our sample was acceptable
533 ($\alpha = .79$). A composite score was created by averaging across items, such that higher scores
534 represented more frequent experiences of discrimination.

535 **Momentary Affect.** The PANAS-X [116] was administered to assess momentary positive
536 and negative affect (10 items each), along with added items to assess momentary anxiety (3
537 items: "anxious", "worried", "restless") and depression-related symptoms (3 items: "depressed",
538 "sad", "downhearted"). Participants were instructed to indicate to what extent they felt each
539 emotion or feeling "right now" on a scale of 1 (very slightly or not at all) to 5 (extremely).

Internal consistency at baseline (negative affect α: .83; positive affect α: .91; anxiety α: .73; depression α: .93) was acceptable. A composite score was created separately for each facet, such that higher scores represented higher levels of affect/symptoms.

Recovery Questionnaire

Momentary Affect. The same PANAS-X items were administered following the DST as in the baseline questionnaire. Internal consistency at recovery (negative affect α: .83; positive affect α: .91; anxiety α: .81; depression α: .85) was acceptable.

Perceived Support. The emotional support subscale from the Berlin Social Support Scales [117] was adapted to assess perceived support during the DST. Participants were instructed to think about the person they interacted with during the DST and indicate their agreement with the following 6 statements: "This person showed me that he/she loves and accepts me", "This person comforted me when I was feeling bad", "This person made me feel valued and important", "This person expressed concern about my condition", "This person assured me I can rely completely on him/her", "This person encouraged me not to give up"). Responses were on a scale of 1 (strongly disagree) to 5 (strongly agree). Internal consistency within our sample was acceptable (α = .90). A composite was created by averaging the items, such that higher scores represented higher levels of perceived support.

Author Contributions

HV conceptualized the project and oversaw every stage of data collection and manuscript preparation. JB and SP coordinated data collection and processing and contributed to manuscript writing. CD contributed to manuscript writing. All authors reviewed the manuscript and contributed to revisions.

Additional Information

The authors have no competing interests to declare.

Data Availability

All data and code for analysis can be found at [https://osf.io/28bnz/].

References

1. Pew Research Center. Half of U.S. Latinos experienced some form of discrimination during the first year of the pandemic. 2021. https://www.pewresearch.org/race-and-ethnicity/2021/11/04/half-of-u-s-latinos-experienced-some-form-of-discrimination-during-the-first-year-of-the-pandemic/

2. Carter RT, Johnson VE, Kirkinis K, Roberson K, Muchow C, Galgay C. A Meta-Analytic Review of Racial Discrimination: Relationships to Health and Culture. *Race Soc Probl*. 2019;11(1):15-32. doi:10.1007/s12552-018-9256-y

3. Korous KM, Causadias JM, Casper DM. Racial discrimination and cortisol output: A meta-analysis. *Social Science & Medicine*. 2017;193:90-100. doi:10.1016/j.socscimed.2017.09.042

4. Pascoe EA, Richman LS. Perceived Discrimination and Health: A Meta-Analytic Review. *Psychol Bull*. 2009;135(4):531-554. doi:10.1037/a0016059

5. Schmitt MT, Branscombe NR, Postmes T, Garcia A. The consequences of perceived discrimination for psychological well-being: A meta-analytic review. *Psychological Bulletin*. 2014;140(4):921-948. doi:10.1037/a0035754

6. Brittian AS, Toomey RB, Gonzales NA, Dumka LE. Perceived Discrimination, Coping Strategies, and Mexican Origin Adolescents' Internalizing and Externalizing Behaviors: Examining the Moderating Role of Gender and Cultural Orientation. *Applied Developmental Science*. 2013;17(1):4-19. doi:10.1080/10888691.2013.748417

7. Cano MÁ, Castro Y, Dios MA de, et al. Associations of ethnic discrimination with symptoms of anxiety and depression among Hispanic emerging adults: a moderated mediation model. *Anxiety, Stress, & Coping*. 2016;29(6):699-707. doi:10.1080/10615806.2016.1157170

8. Araújo BY, Borrell LN. Understanding the Link Between Discrimination, Mental Health Outcomes, and Life Chances Among Latinos. *Hispanic Journal of Behavioral Sciences*. 2006;28(2):245-266. doi:10.1177/0739986305285825

9. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist*. 1999;54(10):805-816. doi:10.1037/0003-066X.54.10.805

10. Finch BK, Vega WA. Acculturation Stress, Social Support, and Self-Rated Health Among Latinos in California. *Journal of Immigrant Health*. 2003;5(3):109-117. doi:10.1023/A:1023987717921

11. GBD. Global, regional, and national age–sex specific all-cause and cause-specific mortality for 240 causes of death, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *The Lancet*. 2015;385(9963):117-171. doi:10.1016/S0140-6736(14)61682-2

12. Chiu M, Maclagan LC, Tu JV, Shah BR. Temporal trends in cardiovascular disease risk factors among white, South Asian, Chinese and black groups in Ontario, Canada, 2001 to 2012: a population-based study. *BMJ Open*. 2015;5(8):e007232. doi:10.1136/bmjopen-2014-007232

13. Lockwood KG, Marsland AL, Matthews KA, Gianaros PJ. Perceived discrimination and cardiovascular health disparities: a multisystem review and health neuroscience perspective. *Annals of the New York Academy of Sciences*. 2018;1428(1):170-207. doi:10.1111/nyas.13939

14. Mays VM, Cochran SD, Barnes NW. Race, Race-Based Discrimination, and Health Outcomes Among African Americans. *Annual Review of Psychology*. 2007;58(1):201-225. doi:10.1146/annurev.psych.57.102904.190212

15. Brindle RC, Ginty AT, Phillips AC, Carroll D. A tale of two mechanisms: A meta-analytic approach toward understanding the autonomic basis of cardiovascular reactivity to acute psychological stress. *Psychophysiology*. 2014;51(10):964-976. doi:10.1111/psyp.12248

16. Thayer JF, Sternberg E. Beyond Heart Rate Variability: Vagal Regulation of Allostatic Systems. *Annals of the New York Academy of Sciences*. 2006;1088(1):361-372. doi:10.1196/annals.1366.014

17. Lepore SJ, Revenson TA, Weinberger SL, et al. Effects of social stressors on cardiovascular reactivity in black and white women. *Ann Behav Med*. 2006;31(2):120-127. doi:10.1207/s15324796abm3102_3

18. Merritt MM, Bennett GG, Williams RB, Edwards CL, Sollers JJ. Perceived racism and cardiovascular reactivity and recovery to personally relevant stress. *Health Psychology*. 2006;25(3):364-369. doi:10.1037/0278-6133.25.3.364

19. Guyll M, Matthews KA, Bromberger JT. Discrimination and unfair treatment: relationship to cardiovascular reactivity among African American and European American women. *Health Psychol*. 2001;20(5):315-325. doi:10.1037//0278-6133.20.5.315

20. Volpe VV, Lee DB, Hoggard LS, Rahal D. Racial Discrimination and Acute Physiological Responses Among Black Young Adults: The Role of Racial Identity. *Journal of Adolescent Health*. 2019;64(2):179-185. doi:10.1016/j.jadohealth.2018.09.004

21. Dorr N, Brosschot JF, Sollers JJ, Thayer JF. Damned if you do, damned if you don't: The differential effect of expression and inhibition of anger on cardiovascular recovery in Black and White males. *International Journal of Psychophysiology*. 2007;66(2):125-134. doi:10.1016/j.ijpsycho.2007.03.022

22. Cooper DC, Thayer JF, Waldstein SR. Coping with Racism: The Impact of Prayer on Cardiovascular Reactivity and Post-stress Recovery in African American Women. *Ann Behav Med*. 2014;47(2):218-230. doi:10.1007/s12160-013-9540-4

643 23. Neblett EW, Roberts SO. Racial identity and autonomic responses to racial discrimination.
644 *Psychophysiology*. 2013;50(10):943-953. doi:10.1111/psyp.12087

645 24. Sawyer PJ, Major B, Casad BJ, Townsend SSM, Mendes WB. Discrimination and the
646 Stress Response: Psychological and Physiological Consequences of Anticipating Prejudice
647 in Interethnic Interactions. *Am J Public Health*. 2012;102(5):1020-1026.
648 doi:10.2105/AJPH.2011.300620

649 25. Zeiders KH, Landor AM, Flores M, Brown A. Microaggressions and Diurnal Cortisol:
650 Examining Within-Person Associations Among African-American and Latino Young
651 Adults. *Journal of Adolescent Health*. 2018;63(4):482-488.
652 doi:10.1016/j.jadohealth.2018.04.018

653 26. Ginty AT, Kraynak TE, Fisher JP, Gianaros PJ. Cardiovascular and autonomic reactivity to
654 psychological stress: Neurophysiological substrates and links to cardiovascular disease.
655 *Autonomic Neuroscience*. 2017;207:2-9. doi:10.1016/j.autneu.2017.03.003

656 27. García J, Serpas D, Torres Y. Taking It to Heart: Preliminary Investigation on the
657 Cardiovascular Effects of Racial/Ethnic Microaggressions in Latinx. *Social Behavioral*
658 *Research and Practice*. 2020;5:1-7. doi:10.17140/SBRPOJ-5-120

659 28. Huynh VW, Huynh QL, Stein MP. Not just sticks and stones: Indirect ethnic discrimination
660 leads to greater physiological reactivity. *Cultural Diversity and Ethnic Minority*
661 *Psychology*. 2017;23(3):425-434. doi:10.1037/cdp0000138

662 29. Townsend SSM, Eliezer D, Major B, Mendes WB. Influencing the World Versus Adjusting
663 to Constraints: Social Class Moderates Responses to Discrimination. *Social Psychological*
664 *and Personality Science*. 2014;5(2):226-234. doi:10.1177/1948550613490968

665 30. Hagiwara N, Green TL, Moreno O, Smith D, Corona R. Ethnic discrimination and weight
666 outcomes among Latinx emerging adults: Examinations of an individual-level mediator and
667 cultural moderators. *Cultural Diversity and Ethnic Minority Psychology*. 2021;27(2):189-
668 200. doi:10.1037/cdp0000336

669 31. LeBrón AMW, Spencer M, Kieffer E, Sinco B, Piatt G, Palmisano G. Correlates of
670 Interpersonal Ethnoracial Discrimination Among Latino Adults with Diabetes: Findings
671 from the REACH Detroit Study. *Journal of Ethnic & Cultural Diversity in Social Work*.
672 2017;26(1-2):48-67. doi:10.1080/15313204.2016.1263820

673 32. Ryan AM, Gee GC, Laflamme DF. The Association between Self-Reported Discrimination,
674 Physical Health and Blood Pressure: Findings from African Americans, Black Immigrants,
675 and Latino Immigrants in New Hampshire. *Journal of Health Care for the Poor and*
676 *Underserved*. 2006;17(2):116-132. doi:10.1353/hpu.2006.0079

677 33. Flores E, Tschann JM, Dimas JM, Bachen EA, Pasch LA, de Groat CL. Perceived
678 Discrimination, Perceived Stress, and Mental and Physical Health Among Mexican-Origin
679 Adults. *Hispanic Journal of Behavioral Sciences*. 2008;30(4):401-424.
680 doi:10.1177/0739986308323056

34. Arellano-Morales L, Roesch SC, Gallo LC, et al. Prevalence and correlates of perceived ethnic discrimination in the Hispanic Community Health Study/Study of Latinos Sociocultural Ancillary Study. *Journal of Latina/o Psychology*. 2015;3(3):160-176. doi:10.1037/lat0000040

35. Lee RT, Perez AD, Boykin CM, Mendoza-Denton R. On the prevalence of racial discrimination in the United States. *PLOS ONE*. 2019;14(1):e0210698. doi:10.1371/journal.pone.0210698

36. Bennett GG, Merritt MM, Edwards CL, Sollers JJ. Perceived Racism and Affective Responses to Ambiguous Interpersonal Interactions among African American Men. *American Behavioral Scientist*. 2004;47(7):963-976. doi:10.1177/0002764203261070

37. Hermosura AH, Haynes SN, Kaholokula JK. A Preliminary Study of the Relationship between Perceived Racism and Cardiovascular Reactivity and Recovery in Native Hawaiians. *J Racial and Ethnic Health Disparities*. 2018;5(5):1142-1154. doi:10.1007/s40615-018-0463-4

38. Jones DR, Harrell JP, Morris-Prather CE, Thomas J, Omowale N. Affective and physiological responses to racism: the roles of afrocentrism and mode of presentation. *Ethn Dis*. 1996;6(1-2):109-122.

39. Morris-Prather CE, Harrell JP, Collins R, Leonard KL, Boss M, Lee JW. Gender differences in mood and cardiovascular responses to socially stressful stimuli. *Ethn Dis*. 1996;6(1-2):123-131.

40. Armstead CA, Lawler KA, Gorden G, Cross J, Gibbons J. Relationship of racial stressors to blood pressure responses and anger expression in Black college students. *Health Psychology*. 1989;8(5):541-556. doi:10.1037/0278-6133.8.5.541

41. Fischer S, Nater UM, Strahler J, et al. Psychobiological impact of ethnic discrimination in Turkish immigrants living in Germany. *Stress*. 2017;20(2):167-174. doi:10.1080/10253890.2017.1296430

42. Goodwin SA, Williams KD, Carter-Sowell AR. The psychological sting of stigma: The costs of attributing ostracism to racism. *Journal of Experimental Social Psychology*. 2010;46(4):612-618. doi:10.1016/j.jesp.2010.02.002

43. Hoggard LS, Byrd CM, Sellers RM. The lagged effects of racial discrimination on depressive symptomology and interactions with racial identity. *Journal of Counseling Psychology*. 2015;62(2):216-225. doi:10.1037/cou0000069

44. Hoggard LS, Jones SCT, Sellers RM. Racial Cues and Racial Identity: Implications for How African Americans Experience and Respond to Racial Discrimination. *Journal of Black Psychology*. 2017;43(4):409-432. doi:10.1177/0095798416651033

45. Lui PP, Gobrial S, Stringer E, Jouriles EN. Effects of racial discrimination on stress, negative emotions, and alcohol craving: A registered report of a virtual reality experiment.

Cultural Diversity and Ethnic Minority Psychology. Published online 2024:No Pagination Specified-No Pagination Specified. doi:10.1037/cdp0000636

46. Masten CL, Telzer EH, Eisenberger NI. An fMRI Investigation of Attributing Negative Social Treatment to Racial Discrimination. *Journal of Cognitive Neuroscience*. 2010;23(5):1042-1051. doi:10.1162/jocn.2010.21520

47. Hoggard LS, Hill LK, Gray DL, Sellers RM. Capturing the cardiac effects of racial discrimination: Do the effects "keep going"? *International Journal of Psychophysiology*. 2015;97(2):163-170. doi:10.1016/j.ijpsycho.2015.04.015

48. Franco M, O'Brien KM. Taking Racism to Heart: Race-Related Stressors and Cardiovascular Reactivity for Multiracial People. *Journal of Multicultural Counseling and Development*. 2020;48(2):83-94. doi:https://doi.org/10.1002/jmcd.12167

49. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014;311(8):806-814. doi:10.1001/jama.2014.732

50. Mozaffarian D, Benjamin EJ, Go AS, et al. Heart Disease and Stroke Statistics—2016 Update. *Circulation*. 2016;133(4):e38-e360. doi:10.1161/CIR.0000000000000350

51. Ruiz JM, Hamann HA, Mehl MR, O'Connor MF. The Hispanic health paradox: From epidemiological phenomenon to contribution opportunities for psychological science. *Group Processes & Intergroup Relations*. 2016;19(4):462-476. doi:10.1177/1368430216638540

52. Cleveland JC, Espinoza J, Holzhausen EA, Goran MI, Alderete TL. The impact of social determinants of health on obesity and diabetes disparities among Latino communities in Southern California. *BMC Public Health*. 2023;23(1):37. doi:10.1186/s12889-022-14868-1

53. Salgado de Snyder VN, McDaniel M, Padilla AM, Parra-Medina D. Impact of COVID-19 on Latinos: A Social Determinants of Health Model and Scoping Review of the Literature. *Hispanic Journal of Behavioral Sciences*. 2021;43(3):174-203. doi:10.1177/07399863211041214

54. Cortes DE. Variations in Familism in Two Generations of Puerto Ricans. *Hispanic Journal of Behavioral Sciences*. 1995;17(2):249-255. doi:10.1177/07399863950172008

55. Sabogal F, Marín G, Otero-Sabogal R, Marín BV, Perez-Stable EJ. Hispanic Familism and Acculturation: What Changes and What Doesn't? *Hispanic Journal of Behavioral Sciences*. 1987;9(4):397-412. doi:10.1177/07399863870094003

56. Steidel AGL, Contreras JM. A New Familism Scale for Use with Latino Populations. *Hispanic Journal of Behavioral Sciences*. 2003;25(3):312-330. doi:10.1177/0739986303256912

57. Valdivieso-Mora E, Peet CL, Garnier-Villarreal M, Salazar-Villanea M, Johnson DK. A Systematic Review of the Relationship between Familism and Mental Health Outcomes in Latino Population. *Front Psychol*. 2016;7. doi:10.3389/fpsyg.2016.01632

58. Calderón-Tena CO, Knight GP, Carlo G. The Socialization of Prosocial Behavioral Tendencies Among Mexican American Adolescents: The Role of Familism Values. *Cultural Diversity & Ethnic Minority Psychology*. 2011;17(1):98-106. doi:10.1037/a0021825

59. Knight GP, Gonzales NA, Saenz DS, et al. The Mexican American Cultural Values Scale for Adolescents and Adults. *The Journal of Early Adolescence*. 2010;30(3):444-481. doi:10.1177/0272431609338178

60. Piña-Watson B, Gonzalez IM, Manzo G. Mexican-descent adolescent resilience through familismo in the context of intergeneration acculturation conflict on depressive symptoms. *Translational Issues in Psychological Science*. 2019;5(4):326-334. doi:10.1037/tps0000210

61. Santiago CD, Torres SA, Brewer SK, Fuller AK, Lennon JM. The Effect of Cultural Factors on Daily Coping and Involuntary Responses to Stress Among Low-Income Latino Adolescents. *Journal of Community Psychology*. 2016;44(7):872-887. doi:https://doi.org/10.1002/jcop.21814

62. Volpert-Esmond HI, Marquez ED, Camacho AA. Family relationships and familism among Mexican Americans on the U.S.-Mexico border during the COVID-19 pandemic. *Cultur Divers Ethnic Minor Psychol*. 2023;29(2):145-151. doi:10.1037/cdp0000547

63. Zeiders KH, Updegraff KA, Umaña-Taylor AJ, Wheeler LA, Perez-Brena NJ, Rodríguez SA. Mexican-Origin Youths' Trajectories of Depressive Symptoms: The Role of Familism Values. *Journal of Adolescent Health*. 2013;53(5):648-654. doi:10.1016/j.jadohealth.2013.06.008

64. Uchino BN. Social Support and Health: A Review of Physiological Processes Potentially Underlying Links to Disease Outcomes. *J Behav Med*. 2006;29(4):377-387. doi:10.1007/s10865-006-9056-5

65. Gunnar MR, Hostinar CE. The social buffering of the hypothalamic-pituitary-adrenocortical axis in humans: Developmental and experiential determinants. *Soc Neurosci*. 2015;10(5):479-488. doi:10.1080/17470919.2015.1070747

66. Thorsteinsson EB, James JE. A Meta-analysis of the effects of experimental manipulations of social support during laboratory stress. *Psychology & Health*. 1999;14(5):869-886. doi:10.1080/08870449908407353

67. Kamarck TW, Manuck SB, Jennings JR. Social support reduces cardiovascular reactivity to psychological challenge: A laboratory model. *Psychosomatic Medicine*. 1990;52(1):42-58. doi:10.1097/00006842-199001000-00004

68. Doom JR, Doyle CM, Gunnar MR. Social stress buffering by friends in childhood and adolescence: Effects on HPA and oxytocin activity. *Social Neuroscience*. 2017;12(1):8-21. doi:10.1080/17470919.2016.1149095

69. Kirsch JA, Lehman BJ. Comparing Visible and Invisible Social Support: Non-evaluative Support Buffers Cardiovascular Responses to Stress. *Stress and Health*. 2015;31(5):351-364. doi:https://doi.org/10.1002/smi.2558

70. Edens JL, Larkin KT, Abel JL. The effect of social support and physical touch on cardiovascular reactions to mental stress. *Journal of Psychosomatic Research*. 1992;36(4):371-381. doi:10.1016/0022-3999(92)90073-B

71. Roberts MH, Klatzkin RR, Mechlin B. Social Support Attenuates Physiological Stress Responses and Experimental Pain Sensitivity to Cold Pressor Pain. *Annals of Behavioral Medicine*. 2015;49(4):557-569. doi:10.1007/s12160-015-9686-3

72. Ditzen B, Schmidt S, Strauss B, Nater UM, Ehlert U, Heinrichs M. Adult attachment and social support interact to reduce psychological but not cortisol responses to stress. *Journal of Psychosomatic Research*. 2008;64(5):479-486. doi:10.1016/j.jpsychores.2007.11.011

73. Campos B, Yim IS, Busse D. Culture as a Pathway to Maximizing the Stress-Buffering Role of Social Support. *Hispanic Journal of Behavioral Sciences*. 2018;40(3):294-311. doi:10.1177/0739986318772490

74. Gonzales NA, Johnson M, Shirtcliff EA, Tein J, Eskenazi B, Deardorff J. The role of bicultural adaptation, familism, and family conflict in Mexican American adolescents' cortisol reactivity. *Dev Psychopathol*. 2018;30(5):1571-1587. doi:10.1017/S0954579418001116

75. Toichi M, Sugiura T, Murai T, Sengoku A. A new method of assessing cardiac autonomic function and its comparison with spectral analysis and coefficient of variation of R–R interval. *Journal of the Autonomic Nervous System*. 1997;62(1):79-84. doi:10.1016/S0165-1838(96)00112-9

76. Newlin DB, Wong CJ, Stapleton JM, London ED. Intravenous Cocaine Decreases Cardiac Vagal Tone, Vagal Index (Derived in Lorenz Space), and Heart Period Complexity (Approximate Entropy) in Cocaine Abusers. *Neuropsychopharmacology*. 2000;23(5):560-568. doi:10.1016/S0893-133X(00)00135-4

77. Toichi M, Kubota Y, Murai T, et al. The influence of psychotic states on the autonomic nervous system in schizophrenia. *International Journal of Psychophysiology*. 1999;31(2):147-154. doi:10.1016/S0167-8760(98)00047-6

78. Weinberg A, Klonsky ED, Hajcak G. Autonomic impairment in Borderline Personality Disorder: A laboratory investigation. *Brain and Cognition*. 2009;71(3):279-286. doi:10.1016/j.bandc.2009.07.014

79. Danyluck C, Page-Gould E. Social and Physiological Context can Affect the Meaning of Physiological Synchrony. *Scientific Reports*. 2019;9(1):8222. doi:10.1038/s41598-019-44667-5

80. Toichi M, Kamio Y. Paradoxical Autonomic Response to Mental Tasks in Autism. *J Autism Dev Disord*. 2003;33(4):417-426. doi:10.1023/A:1025062812374

81. Allen JJB, Chambers AS, Towers DN. The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology*. 2007;74(2):243-262. doi:10.1016/j.biopsycho.2006.08.005

82. Berntson GG, Cacioppo JT, Quigley KS. Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology*. 1993;30(2):183-196. doi:10.1111/j.1469-8986.1993.tb01731.x

83. Laborde S, Mosley E, Mertgen A. Vagal Tank Theory: The Three Rs of Cardiac Vagal Control Functioning – Resting, Reactivity, and Recovery. *Front Neurosci*. 2018;12. doi:10.3389/fnins.2018.00458

84. Porges SW. The Polyvagal Perspective. *Biol Psychol*. 2007;74(2):116-143. doi:10.1016/j.biopsycho.2006.06.009

85. Campbell AA, Wisco BE. Respiratory sinus arrhythmia reactivity in anxiety and posttraumatic stress disorder: A review of literature. *Clinical Psychology Review*. 2021;87:102034. doi:10.1016/j.cpr.2021.102034

86. Craske MG, Rauch SL, Ursano R, Prenoveau J, Pine DS, Zinbarg RE. What Is an Anxiety Disorder? *FOC*. 2011;9(3):369-388. doi:10.1176/foc.9.3.foc369

87. Beauchaine T. Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology*. 2001;13(2):183-214. doi:10.1017/S0954579401002012

88. Thayer JF, Lane RD. A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*. 2000;61(3):201-216. doi:10.1016/S0165-0327(00)00338-4

89. Stanley J, Peake JM, Buchheit M. Cardiac Parasympathetic Reactivation Following Exercise: Implications for Training Prescription. *Sports Med*. 2013;43(12):1259-1277. doi:10.1007/s40279-013-0083-4

90. Mezzacappa ES, Kelsey RM, Katkin ES, Sloan RP. Vagal rebound and recovery from psychological stress. *Psychosom Med*. 2001;63(4):650-657. doi:10.1097/00006842-200107000-00018

91. Bates D, Mächler M, Bolker B, Walker S. Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*. 2015;67(1):1-48. doi:10.18637/jss.v067.i01

92. Kuznetsova A, Brockhoff PB, Christensen RH. lmerTest Package: Tests in Linear Mixed Effects Models. *Journal of Statistical Software*. 2017;82(13):1-26. doi:10.18637/jss.v082.i13

93. Lenth R, Singmann H, Love J, Buerkner P, Huerve M. emmeans: Estimated marginal means. Published online 2018.

94. Seery MD. Challenge or threat? Cardiovascular indexes of resilience and vulnerability to potential stress in humans. *Neuroscience & Biobehavioral Reviews*. 2011;35(7):1603-1610. doi:10.1016/j.neubiorev.2011.03.003

95. Beauchaine TP, Bell Z, Knapton E, McDonough-Caplan H, Shader T, Zisner A. Respiratory sinus arrhythmia reactivity across empirically based structural dimensions of psychopathology: A meta-analysis. *Psychophysiology*. 2019;56(5):e13329. doi:10.1111/psyp.13329

96. Thayer JF, Åhs F, Fredrikson M, Sollers JJ, Wager TD. A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews*. 2012;36(2):747-756. doi:10.1016/j.neubiorev.2011.11.009

97. Kubzansky LD, Mendes WB, Appleton AA, Block J, Adler GK. A heartfelt response: Oxytocin effects on response to social stress in men and women. *Biological Psychology*. 2012;90(1):1-9. doi:10.1016/j.biopsycho.2012.02.010

98. Berna G, Ott L, Nandrino JL. Effects of Emotion Regulation Difficulties on the Tonic and Phasic Cardiac Autonomic Response. *PLOS ONE*. 2014;9(7):e102971. doi:10.1371/journal.pone.0102971

99. Borders A, Hennebry KA. Angry rumination moderates the association between perceived ethnic discrimination and risky behaviors. *Personality and Individual Differences*. 2015;79:81-86. doi:10.1016/j.paid.2015.01.047

100. Borders A, Liang CTH. Rumination partially mediates the associations between perceived ethnic discrimination, emotional distress, and aggression. *Cultural Diversity and Ethnic Minority Psychology*. 2011;17:125-133. doi:10.1037/a0023357

101. Dondanville AA, Bordewyk A, Pössel P. Role of Rumination in the Association between Discrimination and Adolescents' Mental and Physical Health. *J Child Fam Stud*. 2022;31(12):3302-3313. doi:10.1007/s10826-022-02401-2

102. Otto MW, Lubin RE, Rosenfield D, et al. The association between race- and ethnicity-related stressors and sleep: the role of rumination and anxiety sensitivity. *Sleep*. 2022;45(10):zsac117. doi:10.1093/sleep/zsac117

103. Holloway K, Varner F. Forms and frequency of vicarious racial discrimination and African American parents' health. *Social Science & Medicine*. Published online July 24, 2021:114266. doi:10.1016/j.socscimed.2021.114266

104. Volpert-Esmond HI, Aboargob M. Direct and vicarious experiences of discrimination and rumination among Latinos before and during the beginning of the COVID-19 pandemic. *Social and Personality Psychology Compass*. 2023;17(11):e12864. doi:10.1111/spc3.12864

105. Quinn EB, Ross JD, Boston PQ, Committee HS, Mulligan CJ, Gravlee CC. The social patterning of vicarious discrimination: Implications for health equity. *Social Science & Medicine*. 2023;332:116104. doi:10.1016/j.socscimed.2023.116104

106. Yip T, Chung K, Chae DH. Vicarious racism, ethnic/racial identity, and sleep among Asian Americans. *Cultural Diversity & Ethnic Minority Psychology*. 2024;30(2):319-329. doi:10.1037/cdp0000534

107. Moody MD, Browning WR, Hossain M, Clay OJ. Vicarious experiences of major discrimination, anxiety symptoms, and mental health care utilization among Black Adults. *Social Science & Medicine*. 2023;316:114997. doi:10.1016/j.socscimed.2022.114997

108. Allen AP, Kennedy PJ, Cryan JF, Dinan TG, Clarke G. Biological and psychological markers of stress in humans: Focus on the Trier Social Stress Test. *Neuroscience & Biobehavioral Reviews*. 2014;38:94-124. doi:10.1016/j.neubiorev.2013.11.005

109. Coard SI, Kiang L, Martin Romero MY, Gonzalez LM, Stein GL. Talking through the tough: Identifying facilitating factors to preparation for bias and racial–ethnic discrimination conversations among families from minoritized ethnic–racial groups. *Family Process*. 2023;n/a(n/a). doi:10.1111/famp.12878

110. DeLaney EN, Williams CD, Elias MJ, et al. Racial discrimination and depressive symptoms mediated by conversations about race among students of color. *Journal of American College Health*. 2023;71(9):2835-2839. doi:10.1080/07448481.2021.1998071

111. Moffitt U, Syed M. Ethnic-Racial Identity in Action: Structure and Content of Friends' Conversations about Ethnicity and Race. *Identity*. 2021;21(1):67-88. doi:10.1080/15283488.2020.1838804

112. Sanchez KL, Kalkstein DA, Walton GM. A threatening opportunity: The prospect of conversations about race-related experiences between Black and White friends. *Journal of Personality and Social Psychology*. 2022;122(5):853-872. doi:10.1037/pspi0000369

113. Grossman P, Kollai M. Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within- and between-individual relations. *Psychophysiology*. 1993;30(5):486-495. doi:10.1111/j.1469-8986.1993.tb02072.x

114. Grossman P, Karemaker J, Wieling W. Prediction of Tonic Parasympathetic Cardiac Control Using Respiratory Sinus Arrhythmia: The Need for Respiratory Control. *Psychophysiology*. 1991;28(2):201-216. doi:10.1111/j.1469-8986.1991.tb00412.x

115. Phinney JS, Ong AD. Conceptualization and measurement of ethnic identity: Current status and future directions. *Journal of Counseling Psychology*. 2007;54(3):271-281. doi:10.1037/0022-0167.54.3.271

932 116. Watson D, Clark LA. *The PANAS-X: Manual for the Positive and Negative Affect Schedule,*
933 *Expanded Form.* University of Iowa Press; 1999.

934 117. Luszczynska A, Boehmer S, Knoll N, Schulz U, Schwarzer R. Emotional support for men
935 and women with cancer: Do patients receive what their partners provide? *International*
936 *Journal of Behavioral Medicine*. 2007;14(3):156-163.

937