Race and Resting-State Heart Rate Variability in Brazilian Civil Servants and the Mediating Effects of Discrimination: An ELSA-Brasil Cohort Study

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ABSTRACT

Objectives: African Americans are characterized by higher heart rate variability (HRV), a finding ostensibly associated with beneficial health outcomes. However, these findings are at odds with other evidence that blacks have worse cardiovascular outcomes. Here, we examine associations in a large cohort from the ELSA-Brasil study and determined whether these effects are mediated by discrimination.

Methods: Three groups were compared on the basis of self-declared race: "black" (n = 2,020), "brown" (n = 3,502), and "white" (n = 6,467). Perceived discrimination was measured using a modified version of the Everyday Discrimination Scale. Resting-state HRV was extracted from 10-minute resting-state electrocardiograms. Racial differences in HRV were determined by regression analyses weighted by propensity scores, which controlled for potentially confounding variables including age, sex, education, and other health-related information. Nonlinear mediation analysis quantified the average total effect, comprising direct (race–HRV) and indirect (race–discrimination–HRV) pathways.

Results: Black participants displayed higher HRV relative to brown (Cohen's d = 0.20) and white participants (Cohen's d = 0.31). Brown relative to white participants also displayed a small but significantly higher HRV (Cohen's d = 0.14). Discrimination indirectly contributed to the effects of race on HRV.

Conclusions: This large cohort from the Brazilian population shows that HRV is greatest in black, followed by brown, relative to white participants. The presence of higher HRV in these groups may reflect a sustained compensatory psychophysiological response to the adverse effects of discrimination. Additional research is needed to determine the health consequences of these differences in HRV across racial and ethnic groups.

Key words: discrimination, heart rate variability, HRV, mediation analysis, propensity score weighting, race.

INTRODUCTION

Noncommunicable diseases including neuropsychiatric disorders and cardiovascular disease (CVD) are leading global burdens of disease (1,2). These diseases are responsible for 63% of all deaths worldwide, and 80% of these occur in low- and middle-income countries (3). In Brazil, an upper middle income country, morbidity and mortality caused by noncommunicable diseases are greatest in the most socioeconomically disadvantaged groups and especially in black individuals (4–8). A potential candidate

ANOVA = analysis of variance, **CHD** = coronary heart disease, **CIS-R** = Clinical Interview Schedule-Revised, **CVD** = cardiovascular disease, **ELSA-Brasil** = The Brazilian Longitudinal Study of Adult Health, **HF-HRV** = high-frequency heart rate variability, **HRV** = heart rate variability, **TPR** = total peripheral resistance

SDC Supplemental Content

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marker for future ill-health is heart rate variability (HRV), an index of parasympathetic (vagal) function that may underpin psychophysiological flexibility, psychological well-being, health, and longevity (9–11). Reduced HRV is observed in the mood and anxiety disorders (12–17) and is associated with a 32% to 45% increased risk of a first cardiovascular event, even in populations without known CVD (18). The vagus nerve plays an important regulatory role over a variety of allostatic systems including the hypothalamic-pituitary-adrenal axis (19), inflammatory processes (20), and glucose regulation (21–25). Consequently, tonic vagal dysregulation—indexed using resting-state HRV—may lead to ill-health from a host of conditions and diseases including CVD (9,10,26,27).

It is unexpected therefore that vagally mediated HRV in African Americans is *higher*—not lower—than HRV in "whites" (28). These findings, based on a meta-analysis of 17 studies published between 1995 and 2013, were associated with a large effect size (Hedges $g = 0.93$, 95% confidence interval [CI] = 0.25 to 1.62). Higher HRV in African Americans represents a paradox because African Americans are also characterized by a high prevalence of cardiovascular morbidity and mortality (29,30). One potential explanation for increased HRV is a sustained, compensatory psychophysiological response to the adverse effects of discrimination, which include a worsening of blood pressure, cholesterol, body mass index, and self-assessed general health (31,32). Consistent with this possibility, African Americans display higher resting levels of systolic blood pressure and total peripheral resistance (TPR) in combination with greater HRV in comparison with European Americans (33). For the present study, we hypothesized that black and brown relative to white participants would display increased resting-state HRV in a large cohort from the Brazilian population. We also determined whether these racial differences in HRV would be mediated by discrimination consistent with a compensatory response.

METHODS

Participants

ELSA-Brasil is a cohort of 15,105 civil servants aged 35 to 74 years enrolled between August 2008 and December 2010 at six different sites in Brazil (Belo Horizonte, Porto Alegre, Rio de Janeiro, Salvador, Sao Paulo, and Vitoria). The ethics committees of the participating universities approved the research protocol. All participants provided written informed consent after a complete description of the study. Exclusion criteria comprised current or recent pregnancy (within 4 months of first interview), intention to quit working at the institution in the near future, severe cognitive or communication impairment, and, if retired, residence outside of a study center's metropolitan area. The study design and sampling procedures of ELSA-Brasil have been reported previously (34–36). The present study focused on participants who self-reported their race as "white" (6,467), "brown" (3,502), and "black" (2,020) consistent with the classification adopted by the 2010 National Brazilian Census. Racial measurement in the Brazilian census refers to phenotype (skin color), not ancestry (origin), and the census has consistently included a term for the admixed population ("*Pardo"* or brown), unlike in the United States (37). Self-declared race is a construct with sociopolitical significance that may capture unmeasured factors affecting health outcomes. Although we do not focus on health disparities here per se, our outcome measure (HRV) is medically important and may mediate downstream changes in a variety of allostatic systems. A total of 11,989 participants were available from the ELSA-Brasil cohort after removing participants who refused to self-report their race ($n = 184$), participants who self-reported Asian ($n = 374$) or indigenous ancestry ($n = 157$), participants without available HRV examinations ($n = 1813$, including 504 with ectopic beats, reflecting either extra or skipped beats on the ECG trace), and those missing data on other variables used in analysis ($n = 653$).

Electrocardiogram Assessment

Resting-state data from the electrocardiogram was recorded for 10 minutes during spontaneous breathing without task demands, in the supine position. The electrocardiogram was collected in the morning (8:00 A.M. to noon) in a temperature-controlled room (21°C-24°C) and was sampled at 250 Hz with a digital electrocardiograph (Micromed, Brazil), consistent with international standards for the collection of HRV (38,39). Wincardio (4.4a) software generated the R-R interval series from a selected lead (D2), which is associated with higher R-wave amplitude. Data were then processed to obtain high-frequency (HF-HRV) using the autoregressive method as described elsewhere (40). The HF-HRV (0.15 to 0.40 Hz) component was expressed in absolute units, and then, log-transformed data as a normalization strategy (41). The focus of our study is on HF-HRV, a specific marker of vagal function (42).

Discrimination Questionnaire

Perceived discrimination was determined on the basis of a modified version of the Everyday Discrimination Scale (43) (see also http://scholar.harvard.edu/davidrwilliams/book/export/html/32495). This measure captures unfair treatment on the following five common experiences including: (1) the workplace such as being fired or not recommended for promotion (employment item), (2) difficulty in renting property or living within the community (housing item), (3) unfair accusation, being searched or assaulted by the police (interactions with police item), (4) receiving poorer service within a public place such as a bank, shop, hospital, or government office (discrimination in public places item), or (5) being (unfairly) discouraged within school or college (education item). For each situation, participants were asked whether they had experienced discrimination and responded on an ordinal scale. Available responses were "no," "yes, one time" and "yes, more than one time." Participants were then given a score of 0 if they did not experience discrimination, 1 if they reported experiencing any of these forms of discrimination once only, and a score of 2 if they had experienced any of these forms of discrimination on more than one occasion. Test-retest reliability ($n = 92$, 2 weeks apart (44)) relating to overall discrimination is associated with a κ coefficient of 0.85 (95% CI = 0.72 to 0.98).

Covariates

The present study sought to provide an important extension to the literature on the association between self-reported race and HRV. Although the decision over what variables should be used as covariates is controversial (45), our decision was driven by the need to set the distributions of certain variables as equal across racial groups (46), allowing us to draw conclusions that the outcome—HRV—is affected by race, rather than some other common, potentially confounding variable.

Age, sex, and education (less than high school, high school, or college education) are commonly employed covariates. Each of these variables

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statistically differs by race (see participant characteristics), highlighting some degree of structural confounding, and is known to affect HRV. Health-related information also differs by race and therefore represents possible alternate pathways to alterations in HRV. Health-related variables that were controlled in our study included smoking status (current versus past or never), physical activity (measured using the International Physical Activity Questionnaire and categorized according to low activity versus moderate or high activity as determined using scoring guidelines: http://www.ipaq.ki.se/scoring.pdf), body mass index (weight in kilograms divided by height in meters squared, kg/m^2). Additional health information on which groups were controlled included a continuous measure of psychiatric symptom severity determined using the Portuguese version (47) of the Clinical Interview Schedule-Revised (CIS-R (47)), hypertension (yes versus no; defined as systolic blood pressure ≥140 mm Hg, or diastolic blood pressure ≥ 90 mm Hg, or use of antihypertensive medications), diabetes mellitus (yes versus no; defined as self-reported or fasting blood glucose ≥ 126 mg/dl or 2-hour oral glucose tolerance test ≥ 200 mg/dl or glycated hemoglobin ≥ 6.5%), dyslipidemia (yes versus no; defined as low-density lipoprotein cholesterol ≥ 130 mg/dl or use of lipid-lowering medication), "hard" coronary heart disease (CHD, including myocardial infarction and coronary revascularization) (yes versus no), and use of antidepressant medication (yes versus no).

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics Version 21 and R Version 3.0.1. Participant characteristics (Table 1) were examined using one-way analyses of variance (ANOVAs) for contrasts involving continuous dependent measures, and χ^2 statistics for categorical variables in IBM SPSS Statistics Version 21. Tukey's HSD (honest significant difference) test is reported to correct for multiple comparisons and aid interpretation of ANOVAs, whereas post hoc analyses were inspected to determine those groups that statistically differed from "whites" in χ^2 statistics. Propensity score–weighted analysis and subsequent mediation analysis were conducted using R. These procedures are further described below and the R-code is provided in Supplemental Digital Content 1, http://links.lww.com/PSYMED/A302 [see also 48, 49].

Propensity score–weighted analysis was used to control for potentially confounding variables (see previous section on covariates) after which

TABLE 1. Participant Characteristics by Race Before Propensity Score Weighting (N = 11,989)

Characteristics, M (SD) or N (%)	Self-Identified Race			Statistic
	White (n = 6467)[a]	Brown (n = 3502)[a]	Black (n = 2020)[a]	
Demographic variables				
Age	52.46 (9.25)	51.20 (8.62)[b]	51.69 (8.59)[b]	$F(2,11986) = 23.52$, $p < .001$
Female	3483 (53.9)	1810 (51.7)	1228 (60.8)[b]	$\chi^2(2) = 44.44$, $p < .001$
Education				
Less than high school	475 (7.3)	608 (17.4)[b]	435 (21.5)[b]	$\chi^2(2) = 379.07$, $p < .001$
High school	1683 (26.0)	1505 (43.0)[b]	1034 (51.2)[b]	$\chi^2(2) = 577.78$, $p < .001$
Lifestyle characteristics				
Physical inactivity	4782 (73.9)	2780 (79.4)[b]	1678 (83.1)[b]	$\chi^2(2) = 87.49$, $p < .001$
Current smoker	773 (12.0)	480 (13.7)[b]	307 (15.2)[b]	$\chi^2(2) = 16.43$, $p < .001$
Body mass index, kg/m^2	26.81 (4.68)	27.01 (4.61)	28.05 (5.11)[b]	$F(2,11986) = 52.77$, $p < .001$
Discrimination[d]	1383 (21.4)	852 (24.3)[b]	675 (33.4)[b]	$\chi^2(4) = 158.89$, $p < .001$
Health characteristics				
CIS-R score	7.55 (7.54)	8.84 (8.30)[b]	9.38 (8.66)[b]	$F(2,11986) = 55.00$, $p < .001$
Hypertension	2017 (31.2)	1276 (36.4)[b]	974 (48.2)[b]	$\chi^2(2) = 196.25$, $p < .001$
Diabetes mellitus	1045 (16.2)	692 (19.8)[b]	555 (27.5)[b]	$\chi^2(2) = 128.80$, $p < .001$
Dyslipidemia	3824 (59.1)	1980 (56.5)[b]	1113 (55.1)[b]	$\chi^2(2) = 12.96$, $p = .002$
Hard CHD[c]	306 (4.7)	128 (3.7)[b]	86 (4.3)	$\chi^2(2) = 6.38$, $p = .020$
Antidepressant use	473 (7.3)	183 (5.2)[b]	59 (2.9)[b]	$\chi^2(2) = 57.78$, $p < .001$
Heart rate measures				
Heart rate, beats per minute	67.02 (9.06)	66.91 (9.25)	66.70 (9.52)	$F(2,11986) = .95$, $p = .390$
HF-HRV [ln (ms^2)]	5.22 (1.20)	5.40 (1.17)[b]	5.58 (1.18)[b]	$F(2,11986) = 75.92$, $p < .001$
LF-HRV [ln (ms^2)]	5.41 (1.17)	5.42 (1.16)	5.33 (1.19)[b]	$F(2,11986) = 3.99$, $p = .019$

SD = standard deviation; CIS-R = Clinical Interview Schedule-Revised; CHD = coronary heart disease.

[a] Percentages refer to participants within each racial grouping.

[b] Refers to one-way ANOVA in which each group is compared with "whites" (Tukey's HSD (honest significant difference) test, $p < .05$) or the post hoc analysis from χ^2 statistics indicating those groups that statistically differed from "whites" ($p < .05$).

[c] Hard coronary heart disease includes myocardial infarction and coronary revascularization.

[d] Experience of major discrimination in any of the five major experiences on more than one occasion. This combined measure of discrimination is used in the mediation model.

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racial differences in HRV were determined. Propensity score analyses have several advantages over traditional regression-based analyses (48,50). Firstly, all covariates are summarized as a single propensity score providing an important dimension reduction tool for evaluating differences on the independent variable (e.g., race). Secondly, a formal model is explicitly specified that is not conflated with the modeling approach (i.e., the analysis on which conclusions are drawn). Thirdly, the propensity score is a function only of covariates, not outcomes (i.e., HRV); therefore, repeated analyses attempting to balance covariate distributions across groups (race) do not bias estimates of the outcome (HRV). Fourthly, propensity score methods avoid extrapolating beyond the observed data unlike traditional parametric modeling when groups (race) are disparate on covariates.

Weighted regression analyses were conducted using the "twang" and "survey" packages in the R statistical environment. Effective sample sizes (reported as "*n*") were obtained from propensity score weighting, reflecting the numbers of participants with similar features on covariates in each of the groups. These effective sample sizes are smaller than the numbers of participants included in analyses because they capture the adverse impact of increased variance on precision and power (48). Omnibus F tests of statistical significance were conducted, followed by *t* tests to determine between group differences using the *regTermTest* and *svydesign* functions of the *survey* package (51). The figure displays group means and 95% CIs after propensity score weighting, extracted using the *svyby* function in the same package. Cohen's *d* effect sizes were calculated using an online calculator (available here: http://www.campbellcollaboration.org/escalc/html/EffectSizeCalculator-SMD8.php; based on the book by Lipsey et al. (52)). A sensitivity analysis was then conducted to rule out any possibility that increased HRV in "blacks" is due to survival bias and underrepresentation of more severe disease due to premature cardiovascular death. Therefore, sensitivity analysis involved redoing our main analysis on younger individuals, as defined by those participants under the median age of the entire cohort.

After the propensity score–weighted analysis, mediation analysis was conducted to determine whether discrimination mediates the effects of race on HRV. Although mediation modeling has typically been conducted within a linear structural equation modeling framework, linear models cannot be used on ordinal data such as the discrimination variable in the present study. We employed the "mediation" package (49) for mediation analyses, because this package provides a robust and flexible option for modeling nonlinearity. Mediation allows the average total ($\bar{\tau}$) effect to be decomposed into average direct ($\bar{\zeta}$) and average indirect ($\bar{\delta}$) effects, as well as for hypotheses relating to the mediating effects of discrimination to be tested statistically. Direct, indirect, and total effects are reported along with their uncertainty estimates on the basis of 1000 nonparametric bootstrap resamples. Like that for our propensity score analyses (previously described), we also conducted sensitivity analysis to determine whether mediation could be replicated in the younger cohort and in a cohort, in which potential confounding variables will be of less concern, especially ill-health and medication. Additional sensitivity analyses were conducted on heart rate to determine whether discrimination mediated any relationship between race and this variable (see supplementary information).

RESULTS

Participant Characteristics

Participant characteristics are presented in Table 1. Unadjusted analyses on the impact of race on HRV indicated that all groups significantly differed from each other, such that black participants display higher HF-HRV than brown participants and that brown participants display higher HF-HRV than white participants (See also the Supplementary Table S1, Supplemental Digital Content 2, http://links.lww.com/PSYMED/A303, which provides unadjusted correlations between each participant characteristic and HRV measures). Propensity score weighting analyses (reported below) confirmed these unadjusted findings after controlling for potentially confounding variables. Black participants also displayed a higher level of repeated discrimination, followed by brown relative to white participants. This finding provides the foundation on which results for mediation analyses are interpreted.

Inspection of participant characteristics (Table 1) indicates that, in comparison with white participants, black and brown participants were slightly younger and displayed more psychiatric symptoms. However, none of the averaged CIS-R scores (which indexed psychiatric symptoms) for race-based groupings reached the threshold required for diagnostic status (threshold score = 12 (47)). Black relative to white participants were also heavier, more likely to be female, to have a lower level of education, to be physically inactive, to be current smokers, and to have a higher prevalence of hypertension, diabetes mellitus, and dyslipidemia. Black participants relative to white and brown participants were also less likely to be using antidepressants. Brown relative to white participants were more likely to have a lower level of education, to be physically inactive, to be current smokers, and to have hypertension, diabetes mellitus, dyslipidemia, and "hard" CHD. Like black participants, they were also less likely to be using antidepressants relative to white participants. These findings highlight the need for propensity score weighting, which allowed for differences on these covariates to be equalized across racial groupings.

Propensity Score Analysis

Here, we examined the impact of race on resting-state HRV after propensity score weighting. Effective sample sizes for the groups were as follows: white, n = 5,747; brown, n = 3,139; and black, n = 1,314. Significant effects were observed for HF-HRV ($F(2,11986) = 67.23$, $p = <.001$). Black participants displayed higher vagally mediated HF-HRV (M = 5.60, 95% CI = 5.54 to 5.66) relative to white participants ($p < .001$, Cohen's $d = 0.31$; M = 5.23, 95% CI = 5.20 to 5.26). In addition, black participants displayed higher vagally mediated HF-HRV relative to brown participants ($p < .001$, Cohen's $d = 0.20$; M = 5.39, 95% CI = 5.35 to 5.44), whereas brown participants also displayed higher HF-HRV (M = 5.39, 95% CI = 5.35 to 5.44) than white participants (HF-HRV, $p < .001$, Cohen's $d = 0.14$; Fig 1).

An additional sensitivity analysis was conducted on participants younger than the median of the entire cohort (median age = 51.00 years) to rule out the possibility that greater HRV observed in black and brown participants relative to white participants is due to survival bias. Effective sample sizes for the groups were as follows: white, n = 2,959; brown, n = 1,807; and black, n = 792. Again,

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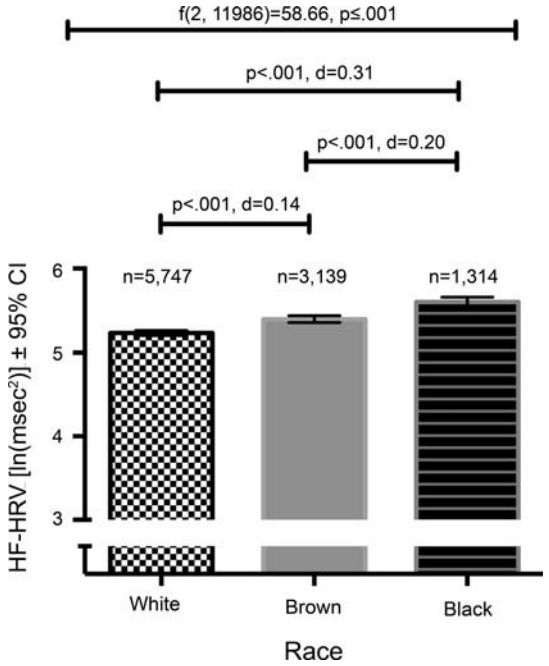

FIGURE 1. HF-HRV (±95% CI) by group. Black participants display significantly higher values than brown participants, who display significantly higher values than white participants.
HF-HRV = high-frequency heart rate variability.

significant effects were observed for HF-HRV ($F(2,6126) = 32.20$, $p < .001$). Black participants displayed higher vagally mediated HF-HRV (M = 5.82, 95% CI = 5.75 to 5.90) relative to white participants ($p < .001$, Cohen's $d = 0.29$; M = 5.50, 95% CI = 5.45 to 5.54). In addition, black participants displayed higher vagally mediated HF-HRV relative to brown participants ($p < .001$, Cohen's $d = 0.16$; M = 5.65, 95% CI = 5.60 to 5.70). Finally, brown participants also displayed higher HF-HRV (M = 5.65, 95% CI = 5.60 to 5.70) than white participants ($p < .001$, Cohen's $d = 0.14$). In summary, analyses revealed that the three groups differed from each other on HRV such that black participants displayed the highest HRV, followed by brown and white participants, the group that displayed the lowest values.

Mediation Analysis

Here, we examined whether the relationship between race and HRV is mediated by discrimination. Results focusing on "whites" and "blacks" revealed significant total ($\bar{\tau} = 0.3590$, 95% CI = 0.2980 to 0.4147, $p < .01$), direct ($\bar{\zeta} = 0.3521$, 95% CI = 0.2900 to 0.4086, $p < .01$), and indirect effects ($\delta = 0.0069$, 95% CI = 0.0010 to 0.0143, $p = .02$). Analysis on white and brown participants also revealed significant total ($\bar{\tau} = 0.1778$, 95% CI = 0.1492 to 0.2063, $p < .01$) and direct effects ($\bar{\zeta} = 0.1751$, 95% CI = 0.1462 to 0.2034, $p < .01$), but no indirect effect ($\delta = 0.0027$, 95% CI = −0.0006 to 0.0057, $p = .10$). An analysis was also conducted on brown and black participants; results indicated significant total ($\bar{\tau} = 0.1820$, 95% CI = 0.1521 to 0.2078, $p < .01$) and direct ($\bar{\zeta} = 0.1767$, 95% CI = 0.1475 to 0.2028, $p < .01$) effects, as well as an indirect effect at the statistical threshold ($\delta = 0.0053$, 95% CI = 0.0001 to 0.0100, $p = .05$).

We then conducted a sensitivity analysis to determine whether mediation could be replicated in the younger cohort. Results from this sensitivity analysis focusing on "whites" and "blacks" revealed significant total ($\bar{\tau} = 0.3347$, 95% CI = 0.2572 to 0.4090, $p < .01$), direct ($\bar{\zeta} = 0.3234$, 95% CI = 0.2487 to 0.3973, $p < .01$), and an indirect effect at trend levels ($\delta = 0.0113$, 95% CI = −0.0001 to 0.0203, $p = .06$). Analysis on white and brown participants also revealed significant total ($\bar{\tau} = 0.1623$, 95% CI = 0.1256 to 0.1981, $p < .01$) and direct effects ($\bar{\zeta} = 0.1593$, 95% CI = 0.1230 to 0.1943, $p < .01$), but no indirect effect ($\delta = 0.0030$, 95% CI = −0.0016 to 0.0080, $p = .21$). The sensitivity analysis on brown and black participants indicated significant total ($\bar{\tau} = 0.1704$, 95% CI = 0.1357 to 0.2063, $p < .01$), direct ($\bar{\zeta} = 0.1629$, 95% CI = 0.1281 to 0.1992, $p < .01$), and indirect effects ($\delta = 0.0075$, 95% CI = 0.0008 to 0.0152, $p = .03$). In summary, mediation analysis revealed a small effect of discrimination with regard to the proportion of the mediated effect. Although small, this mediating effect of discrimination significantly contributes to the race-related increase in HRV. Finally, additional sensitivity analysis (reported in Supplemental Digital Content 3, http://links.lww.com/PSYMED/A304) revealed no significant mediating effect of discrimination for the relationship between race and heart rate highlighting the specificity of HRV findings that we report here.

DISCUSSION

Recently published meta-analytic findings (28) indicate that African Americans display higher HRV relative to "whites," despite increased risk for cardiovascular morbidity and mortality among "blacks." The present study examined whether these findings extend to a large cohort from the Brazilian population, which may be less prone to residual confounding because of substantial racial admixture (53). We also examined whether these findings are mediated by discrimination consistent with a compensatory response to the adverse effects of discrimination including hypertension (32) (Table 1). We observed that HRV is higher in black and brown participants, relative to white participants, and that this finding is unlikely to be a consequence of survival bias as indicated by the sensitivity analysis on younger participants. Therefore, as per findings in African Americans (28), our findings in a Brazilian sample are a conundrum given the increased risk for morbidity and mortality that is most prominent in black individuals. We also present the first evidence—to our knowledge—that racial

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differences in HRV may be partially underpinned by the experience of repeated discrimination.

Although discrimination is strongly associated with adverse physiological effects including hypertension (Table 1 (31,32)), our findings also show that it may be associated with a sustained, compensatory psychophysiological response. Although the parasympathetic (vagal) and sympathetic nervous systems are typically conceptualized as two opposing components, this is not correct. Vagal activity—indexed by HRV— may be coactivated or codeactivated (54,55). While coactivation of the parasympathetic (vagal) and sympathetic nervous systems activity may help mitigate the deleterious effects of increased sympathetic nervous systems activity (56), sympathetic-parasympathetic deactivation may reflect passive sensory intake (57). Higher HRV in "blacks" may therefore reflect a compensatory response to the adverse physiological responses associated with perceived discrimination, which may include increased sympathetic nervous systems activity. It is important to note that although "blacks" may display increased HRV, these alterations do not ameliorate other adverse physiological effects of discrimination (31,32), which contribute to higher rates of morbidity and mortality caused by CVD. An earlier study (33) demonstrated that African Americans—relative to European Americans—display greater HRV, in combination with higher resting levels of systolic blood pressure and TPR. TPR was also increased and its recovery was delayed when anger was inhibited (33); TPR may contribute to elevations in blood pressure and increased risk of morbidity and mortality (58,59). These and other differences in vascular activity may therefore underpin the increased risk for CVD in "blacks" versus "whites."

A notable feature of HRV is that it reflects psychological (e.g., emotion regulation) as well as physiological processes (e.g., control over allostatic systems), providing a structural link between mental and physical health (9). Although emotion regulation is a complex and multidimensional construct that may also include rumination, we consider resting-state HRV to index capacity for emotion regulation, reflecting effective prefrontal control of visceromotor, neuroendocrine, and behavioral responses, critical for goal-directed behavior, adaptability, and health (11,60,61). Therefore, higher HRV in black individuals may reflect a heightened yet forced capacity to regulate emotions associated with day-to-day stressors and discrimination against black and brown individuals. Some previous studies support this possibility. Firstly, "blacks" may be conditioned from an early age to inhibit their expression of anger in an effort to reduce or avoid aggression (33), a consequence of early conditioning of avoidance learning in a social context (62,63). While inhibition of anger may be associated with more rapid blood pressure recovery, it is also associated with delayed TPR recovery, which may increase risk from hypertension-related death and disability (33). Although we did not focus on hypertension here, it is notable in our own sample that black individuals display greater frequency of hypertension, followed by brown participants and white participants (Table 1). Furthermore, earlier research has demonstrated that racism-related vigilance represents an important source of chronic stress that contributes to this higher prevalence in hypertension (32). Secondly, "blacks" are associated with *a lower lifetime risk of mood and anxiety disorders*, and that associated protective factors likely originate in childhood (before the age of 10 years (64)), as does learning to regulate emotions, according to the avoidance-learning hypothesis (33,62,63).

Our study has several strengths including the investigation of HRV race-related differences in a large cohort characterized by substantial genetic admixture (53), the application of robust statistical techniques including propensity score–weighted regression analyses and nonlinear mediation analysis on which results are based, and confirmation of findings when only focusing on the younger cohort. We also demonstrate that although heart rate was decreased in black participants relative to the other groups, discrimination did not mediate the relationship between race and heart rate (see Supplemental Digital Content 3, http://links.lww.com/PSYMED/A304), highlighting the specificity of the HRV findings that we report here. It is important to recognize that resting-state HRV and the HF-HRV component in particular—unlike heart rate—are relatively pure indicators of vagal activity (11,65).

Several limitations of our work should also be noted. We did not collect data on respiratory rate or depth, which may influence estimates of HRV. However, the question over whether they should be controlled—especially during resting-state recordings—remains a divisive one in the field of psychophysiology (27,66–68). The use of a self-report measure of discrimination may also be considered a limitation, and we do not have any measure of emotion regulation, the ability to avoid anger, or the frequency of anger episodes. In the future, researchers may wish to consider the possibility of using a variant of the implicit association test for assessing the unconscious experience of discrimination (69,70). Another limitation is the difficulty in quantifying the degree to which mediation analysis was robust to the possibility of unobserved confounding. Therefore, we adopt a balanced approach to inferring causation, presenting a theoretical set of relationships within a cross-sectional dataset and interpret findings within the context of the available literature. Studies with longitudinal designs are necessary to further examine the pathways through which HRV is increased in black and brown individuals, as well as the downstream mechanisms that may underpin increased risks for cardiovascular morbidity in these individuals.

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We must also acknowledge that the proportion of variance explained by discrimination in our mediation analysis was small (the mediating effect was only 2%–3% of the total effect). This small effect is perhaps unsurprising considering that 62% of the sample reported no experience of discrimination and that race-associated differences in HRV is likely to be multideterminable. As suggested previously, it is possible that the conscious report of no discrimination in persons with fewer socioeconomic resources reflects a conscious decision not to report discrimination or even internalized oppression that is not consciously perceived (69).

In conclusion, we demonstrate that black and brown individuals display higher HRV relative to white individuals, findings that are partly mediated by discrimination. The race-based differences we observed in the present study may reflect a compensatory response to the adverse physiological effects of discrimination. They may also reflect other factors including gene-environment interactions and psychosocial issues on which further study is required. Future work in this area is necessary to further clarify the differences underpinning the observed race-related differences in HRV, as well as the mechanisms linked to race-related health disparities. Our findings therefore lay an important foundation for future work that seeks to better understand the relationship between race and HRV and differential pathways to CVD.

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