Journal of Aging and Health
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J Aging Health 2013 25: 839 originally published online 18 June 2013
DOI: 10.1177/0898264313491427

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http://jah.sagepub.com/content/25/5/839

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Journal of Aging and Health
25(5) 839–862

DOI: 10.1177/0898264313491427
jah.sagepub.com
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Abstract

Objective: The objective of this study was to consider race differences in age-trends of autonomic nervous system functioning, using a national data set with a broad age range. **Methods:** Measures of baseline heart rate variability (HRV) and HRV reactivity were derived from electrocardiograph (ECG) recordings taken at rest and during cognitive stress tasks. Age-trends in HRV and HRV reactivity were compared among 204 African Americans and 833 Whites ages 34 to 83 years (M = 53.7, SD = 11.4), before and after controlling for socioeconomic status (SES). **Results:** For HRV-reactivity, age-trends were steeper among African Americans and lower SES participants than Whites and higher SES participants. For baseline HRV, age-trends varied by SES but not race. **Discussion**: Results relating to HRV-reactivity (but not baseline HRV) were consistent with hypotheses suggesting that African Americans are exposed to higher levels of stress and

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experience accelerated declines in health across the life span. The relevance of the findings to research on social stress and health disparities is discussed.

Keywords
health disparities, African American, heart rate variability, stress reactivity, autonomic nervous system

Racial and socioeconomic disparities in health remain a pressing public health concern in the United States (Adler & Rehkopf, 2008; Adler & Stewart, 2010; U.S. Department of Health and Human Services (DHHS), 2011; Williams, Mohammed, Leavell, & Collins, 2010; Williams & Sternthal, 2010). Greater exposure to social stress and less access to coping resources have been hypothesized to result in accelerated dysregulation of physiologic systems across the life span among disadvantaged, historically marginalized, or currently stigmatized groups (Colen, 2011; Kuzawa & Sweet, 2009; Meyer, Schwartz, & Frost, 2008; Slopen et al., 2010; Thoits, 2010; Walsemann, Geronimus, & Gee, 2008). To address the paucity of research on this topic, and the need for a more detailed understanding of specific physiological systems in relation to health disparities, the current study considered age-trends in heart rate variability (HRV) and HRV reactivity among African Americans and Whites.

Despite clear disparities in heart disease between African Americans and Whites in the United States (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Frieden, 2011; Williams & Leavell, 2012), racial differences in cardiac autonomic control, and how these regulatory profiles change with age, are not well understood. HRV is one important measure of autonomic regulation, for which an understanding of racial differences is in its infancy. The magnitude of HRV at rest—as measured by the root mean square of successive differences between heart beats (RMSSD)—is an indicator of parasympathetic influence or vagal modulation of the heart (De Meersman & Stein, 2007; Stein & Kleiger, 1999). Higher levels of HRV are associated with reduced risk of recurrent events in patients following myocardial infarction and with heart failure (Camm et al., 2004; Gerritsen et al., 2001; Kleiger, Miller, Bigger, & Moss, 1987) and with a lower likelihood of incident heart disease in initially healthy participants in community studies (Liao et al., 1996; Liao, Carnethon, Evans, Cascio, & Heiss, 2002; Tsuji et al., 1994).

Another important measure of autonomic nervous system (ANS) functioning—for which race differences are also not well understood—is HRV

reactivity. Although extensive research has hypothesized that greater reactivity of particular cardiovascular responses, such as blood pressure, may be associated with worse health outcomes (Chida & Steptoe, 2010; Treiber et al., 2003), several recent perspectives on parasympathetic activity suggest that HRV reactivity in response to challenging tasks (vagal withdrawal) is indicative of better task performance and more adaptive emotion regulation strategies (Becker et al., 2012; Blair & Peters, 2003; Calkins, Graziano, & Keane, 2007; Calkins & Keane, 2004; Duschek, Muckenthaler, Werner, & Reyes del Paso, 2009). Furthermore, HRV reactivity is through to reflect flexibility or plasticity of the ANS, and thus greater cardiovascular health (Blascovich & Mendes, 2010; Duschek et al., 2009; Porges, 2007; Rottenberg, Salomon, Gross, & Gotlib, 2005). Although research on the health impact of HRV reactivity is needed, a conceptualization of HRV reactivity as a biomarker of healthy ANS functioning is consistent with studies indicating declines in HRV reactivity with age and chronic stress exposure (El-Sheikh & Erath, 2011; Katz, 2007; Steptoe, Kunz-Ebrecht, Wright, & Feldman, 2005).

The Role of the Social Environment in Race Differences Over Time

Structural inequalities resulting from historical marginalization, such as neighborhood and school segregation, result in less access to resources and greater exposure to chronic and acute stresses among African American individuals relative to Whites (Fix & Struyk, 1993; Pager & Shepherd, 2008; Smedley, Stith, & Nelson, 2003; Sternthal, Slopen, & Williams, 2011). For example, segregation of neighborhoods has led to increased exposure to health risks (LaVeist & Wallace, 2000; Morello-Frosch & Jesdale, 2005), decreased availability or resources (Gordon et al., 2011; Powell, Slater, Mirtcheva, Bao, & Chaloupka, 2007), and limited social capital (Cornwell & Cornwell, 2008; Small, Jacobs, & Massengill, 2008) in communities with a higher proportion of African American residents. Furthermore, the continued presence of negative stereotypes and racism in society, in addition to creating and sustaining neighborhood segregation, result in daily stressors for African Americans, which are experienced across the life span (Clark, Anderson, Clark, & Williams, 1999; Fuller-Rowell, Doan, & Eccles, 2012; Steele, 2010; Sue, 2010).

A growing body of research suggests that the physical and social environments in which we live, and the everyday experiences associated with these environments—whether they be stressful or positive—accumulate over time and get "under the skin" to influence physical health (Evans, Miller, Chen,

& Seeman, in press; Friedman & Herd, 2010; Juster, McEwen, & Lupien, 2010; Miller, Chen, & Cole, 2009; Slopen et al., 2010). Thus, differential exposure to social and environmental stresses, alongside poorer access to social resources is likely to be an important contributor to well-documented Black–White disparities in mortality and heart disease (Brondolo et al., 2011; Williams et al., 2010). For example, race-related stresses experienced across the life span are thought to lead to accelerated health declines among African Americans, which has been referred to as the weathering hypothesis (Geronimus et al., 2010; Geronimus, Hicken, Keene, & Bound, 2006).

Race and Aging of the Autonomic Nervous System

At least five studies have considered differences in HRV at rest between African Americans and Whites. Of these, four have found that resting levels of HRV—as measured by RMSSD and other measures of vagal modulation—are higher among African Americans than Whites (Li et al., 2009; Liao et al., 1995; Sloan et al., 2008; Wang, Thayer, Treiber, & Snieder, 2005), and one study has also found lower levels among African Americans (Lampert, Ickovics, Horwitz, & Lee, 2005). These findings are puzzling in light of previously noted racial disparities in heart disease. With regard to HRV-reactivity, at least two studies have been conducted, but findings are inconsistent, with one reporting greater reactivity among African Americans (Arthur, Katkin, & Mezzacappa, 2004), and the other reporting no race differences (Li et al., 2009).

A wide range of studies have shown that HRV declines with age (Liao et al., 1995; Sloan et al., 2008; Steptoe et al., 2005). There is also evidence to suggest a similar age trend in HRV-reactivity, with older adults showing a smaller magnitude of challenge-induced change in HRV than younger adults (Steptoe et al., 2005). Only two studies that we are aware of considered race differences in age-trends of HRV at rest (Choi et al., 2006; Sloan et al., 2008). However, neither study found evidence of accelerated health declines with age among African Americans. While these findings provide preliminary evidence against an accelerated decline hypothesis for HRV at rest, small sample size (Choi et al., 2006) and limited age-ranges (Sloan et al., 2008) render the findings inconclusive and underscore the need for additional research.

With respect to HRV-reactivity, to our knowledge no studies have considered race differences in age-trends, despite the potential importance of HRV-reactivity as a stress-sensitive biomarker of cardiac autonomic control (Becker et al., 2012; Blair & Peters, 2003; Blascovich & Mendes, 2010; Duschek et al., 2009; El-Sheikh & Erath, 2011; Porges, 2007; Rottenberg

et al., 2005). Indeed, because limited evidence supports African American vulnerability in resting HRV, HRV-reactivity may be particularly important in understanding possible racial differences in aspects of ANS functioning that may be linked to disease.

In sum, given existing support for the hypothesis that African Americans experience faster declines in health with age relative to Whites (Geronimus, 2001; Geronimus et al., 2010, 2006), and the need for a more detailed mechanistic understanding of racial disparities (Williams et al., 2010), the main focus of this study was to examine race differences in age-trends of baseline HRV and HRV-reactivity, before and after controlling for SES. Thus, we hypothesized steeper age-related declines in HRV and HRV-reactivity among African Americans relative to Whites, and that some of these race differences would remain after accounting for SES. More specifically, with respect to HRV-reactivity, we expected that (1) the magnitude of reactivity to challenge tasks (vagal withdrawal) would be smaller with increasing age and (2) that this age-related reduction in HRV reactivity would be greater among African Americans than Whites.

Method

Data and Sample

This study draws on data from Midlife in the United States (MIDUS), an ongoing national study of health and aging with baseline assessments conducted in 1995 and 1996 on individuals aged 25 to 74 (MIDUS I), and follow-up assessments conducted in 2004 and 2005 (MIDUS II). MIDUS II also included a city-specific sample of African Americans from Milwaukee, Wisconsin. Following MIDUS II surveys, detailed biomarker assessments, and a stress-reactivity protocol, were conducted on a subsample of participants in a 2-day visit to a General Clinical Research Center at one of three MIDUS study sites (UW, UCLA, Georgetown). The biomarker sample was comparable to the full MIDUS II sample on most sociodemographic factors and a range of health characteristics (see Love, Seeman, Weinstein, & Ryff, 2010 for a detailed sample description).

The initial sample for analyses focused on individuals who participated in the MIDUS II stress reactivity protocol and who self-identified as Black (N = 212) or White (N = 890). Of these participants, 1037 (94%) had valid HRV data. The final sample for analyses included 204 Black and 833 White participants with valid HRV data. Individuals of Hispanic origin were not included in either racial category, and were not analyzed separately due to small sample size. Sample descriptives are shown in Table 1.

Table 1. Descriptive Statistics.

	Overall		Black		White	
	(*N* = 1037)		(*N* = 204)		(*N* = 833)	
	Mean (%)	*SD*	*Mean* (%)	*SD*	*Mean* (%)	*SD*
Race (Black)	(19.7)	—	—	—	—	—
Gender (male)	(42.8)	—	(32.8)	—	(45.3)	—
Age (years)	53.8	11.4	50.9	10.6	54.5	11.6
Marital status (Married)	(69.0)	—	(42.2)	—	(75.5)	—
BMI	29.8	6.7	33.0	8.5	29.0	5.9
SBP (mmHg)	130.5	17.1	132.1	18.7	130.1	16.7
Med1 (Blood pressure)	(35.2)	—	(45.6)	—	(32.7)	—
Med2 (SNS-)	(4.0)	—	(3.0)	—	(4.0)	—
Med3 (SNS+)	(11.0)	—	(11.0)	—	(10.0)	—
Med4 (PNS-)	(22.0)	—	(21.0)	—	(22.0)	—
Med5 (PNS+)	(14.0)	—	(14.0)	—	(14.0)	—
Physical Activity	3.75	1.10	3.77	1.27	3.75	1.05
Income (dollars)	71,300	59,400	39,700	35,000	79,200	61,600
Parent education	5.07	2.46	3.96	2.98	5.31	2.43
Childhood poverty	(9.4)	—	(28.1)	—	(4.9)	—
RMSSD (log msec)	2.92	0.63	3.17	0.66	2.86	0.61
HF-HRV (log msec^2)	4.90	1.29	5.43	1.35	4.77	1.24
RMSSD reactivity	–0.16	0.29	–0.19	0.30	–0.15	0.29
HF reactivity	–0.38	0.62	–0.49	0.66	–0.36	0.61

Note: BMI = body mass index. SBP = systolic blood pressure. HF-HRV = high frequency-heart rate variability. Med1 = blood pressure medication. Med2 = medication with sympathetic nervous system (SNS) inhibitory influence. Med3 = medication with SNS excitatory influence. Med4 = medication with parasympathetic nervous system (PNS) inhibitory influence. Med5 = medication with PNS excitatory influence. Reactivity scores were calculated by subtracting baseline levels from stress task levels; negative reactivity scores thus indicate decreases in HRV in response to stress tasks.

Procedures

The stress reactivity protocol included an 11-min resting baseline period, followed by a 6-min stress task (mental arithmetic or Stroop task), a 6-min rest period, and a second 6-min stress task (mental arithmetic or Stroop task). The order of the stress tasks was counterbalanced and participants remained seated for the duration of the protocol. Each task was administered on a computer, with difficulty levels adjusted based on performance. A detailed description of the tasks is available at http://www.midus.wisc.edu/midus2/.

Outcome Measures

Analyses focus on a time-domain measure of HRV: the root mean square of successive differences (RMSSD).[1] This measure is a known indicator of the parasympathetic or vagal modulation of the heart, and can be reliably scored from 5-min data intervals (De Meersman & Stein, 2007; European Society of Cardiology Task Force, 1996; Stein & Kleiger, 1999). Baseline levels of HRV were calculated by averaging RMSSD scores from two 5-min data blocks taken at rest. Stress-task levels were calculated by averaging RMSSD scores from two 5-min stress-task data blocks. Reactivity scores were then calculated by subtracting baseline levels from stress-task levels (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 2007). Since HRV generally decreases in response to challenging cognitive tasks, mean reactivity scores are negative; hence, larger negative numbers indicate greater reactivity. In total, the analyses shown focus on two outcome measures (RMSSD and RMSSD reactivity).[2] RMSSD variables were log transformed to normalize the distributions and outliers were censored at 3 *SD*s from the mean.

Demographic and Control Measures

Age was coded as a continuous variable, measured in years, in order to test for linear age-trends in HRV and HRV reactivity. Curvilinear age-trends were also explored but were not found to be significant. Gender was coded as 0 for female and 1 for male. Race was coded as 0 for White and 1 for Black. Marital status was coded as 1 for married or living with a partner and 0 if otherwise. BMI (kg/m^2) was calculated from body measurements taken by nursing staff during the study visit. Resting systolic blood pressure was scored by averaging together the two most similar of three readings taken in a seated position. The MIDUS II Biomarker project also included comprehensive information on participant medication use. Medications were categorized based on pharmaceutical classification codes, and four dummy variables were created based on their known excitatory (+) or inhibitory (–) influence on the sympathetic nervous system and parasympathetic nervous system (SNS+, SNS–, PNS+, PNS–). A separate dummy variable was also coded to indicate blood pressure medication use. Physical activity was measured from 18 items in the MIDUS II self-administered questionnaire assessing the frequency of vigorous, moderate, and light physical activity on a 6-point scale ranging from *never* (coded as 1) to *several times a week* (coded as 6). Three 6-item subscales relating to each type of physical activity (vigorous, moderate, and light) were averaged and taken as a measure of overall activity level ($\alpha = .748$).

Four measures of SES were considered: education, income, parent education, and childhood poverty. Education was measured on a 12-point scale

ranging from some grade school or less (1) to PhD, MD, JD or other professional degree (12). Total household income was calculated as the sum of all household income from wages, pensions, SSI, or other government assistance and was censored at US$300,000 to avoid outliers. Parent education was scored as the mean of participant's reports of their mother's and father's education level and was measured on the same scale described above.[3] If education level was only reported for one parent, then that parent's education level was used. Childhood poverty was coded as 1 if the participant indicated that was there was ever a period of 6 months or more during their childhood or adolescence when their family was on public assistance, and 0 if otherwise.

Analyses

A series of regression models was estimated for each continuous outcome variable. Model 1 assessed the main effects of race, gender, and age, controlling for marital status, BMI, systolic blood pressure, and medication use. Model 2 added the main effects of four SES indicators (education, income, parent education, childhood poverty). Model 3 added a race by age interaction to assess differences in age-trends between Blacks and Whites. A race by gender interaction was also considered in Model 3. Model 4 added age by SES interaction terms to assess SES differences in age-trends, and the extent to which the effects of race were explained by SES.

All models were estimated in Mplus using maximum likelihood estimation with robust standard errors (MLR estimator) in order to account minor deviations from normality (Muthén & Muthén, 2010). Full information maximum likelihood estimation was used so that the sample size ($N = 1037$) was consistent across models (Enders, 2010). Education, income, parent education, childhood poverty, and physical activity were the only variables that contained missing data (.2%, 2.4%, 3.1%, 1.1%, and 1.7% respectively). To simplify interpretation of model parameters, all continuous predictor variables were z-scored to have a mean of 0 and a standard deviation of 1 in the models shown (Cohen, Cohen, Aiken, & West, 2003).

Results

Resting Heart Rate Variability

RMSSD. Model results for RMSSD are shown in Table 2. In line with previous research, findings from Model 1 indicated that, net of other variables in the model, older participants had lower levels of RMSSD than younger

Table 2. Model Parameter Estimates Showing the Effects of Age, Race, and SES on Baseline HRV (as Measured by RMSSD).

	Model 1		Model 2		Model 3		Model 4	
	B	*(SE)*	*B*	*(SE)*	*B*	*(SE)*	*B*	*(SE)*
Intercept	2.904	(0.042)***	2.904	(0.044)***	2.882	(0.044)***	2.873	(0.044)***
Med1 (BP)	–0.040	(0.052)	–0.040	(0.051)	–0.043	(0.051)	–0.044	(0.051)
Med2 (SNS-)	–0.364	(0.107)***	–0.369	(0.106)***	–0.380	(0.106)***	–0.396	(0.107)***
Med3 (SNS+)	–0.159	(0.066)*	–0.157	(0.066)*	–0.155	(0.066)*	–0.155	(0.065)*
Med4 (PNS-)	–0.119	(0.050)*	–0.118	(0.050)*	–0.119	(0.050)*	–0.111	(0.050)*
Med5 (PNS+)	0.244	(0.069)***	0.243	(0.069)***	0.247	(0.069)***	0.248	(0.069)***
Physical Activity	0.030	(0.019)	0.031	(0.019)	0.027	(0.019)	0.029	(0.019)
BMI	–0.012	(0.022)	–0.012	(0.022)	–0.018	(0.022)	–0.018	(0.022)
SBP	–0.006	(0.021)	–0.005	(0.021)	–0.007	(0.021)	–0.007	(0.021)
Marital Status	–0.003	(0.043)	0.012	(0.045)	0.018	(0.045)	0.020	(0.045)
Gender	–0.007	(0.038)	–0.011	(0.038)	0.032	(0.041)	0.027	(0.041)
Race	0.283	(0.053)***	0.297	(0.059)***	0.387	(0.073)***	0.377	(0.073)***
Age	–0.110	(0.021)***	–0.115	(0.022)***	–0.125	(0.023)***	–0.130	(0.024)***
Education			0.005	(0.022)	–0.001	(0.021)	0.000	(0.021)
Income			–0.025	(0.020)	–0.027	(0.020)	–0.028	(0.020)
Parent Educ.			–0.003	(0.022)	–0.002	(0.022)	–0.005	(0.022)
Child Poverty			–0.112	(0.070)	–0.112	(0.069)	–0.116	(0.067)
Race×Gender					–0.228	(0.102)*	–0.222	(0.102)*
Race×Age					0.044	(0.056)	0.059	(0.058)
Educ.×Age							0.046	(0.020)*
Inc.×Age							–0.018	(0.022)
Par. Ed.×Age							–0.036	(0.022)
Child Pov.×Age							–0.036	(0.061)

Note: N = 1,037 for all models. RMSSD = Root Mean Square of Successive Differences. BMI = body mass index. SBP = systolic blood pressure. Med1 = blood pressure medication. Med2 = medication with sympathetic nervous system (SNS) inhibitory influence. Med3 = medication with SNS excitatory influence. Med4 = medication with parasympathetic nervous system (PNS) inhibitory influence. Med5 = medication with PNS excitatory influence.
***$p < .001$. **$p < .01$. *$p < .05$.

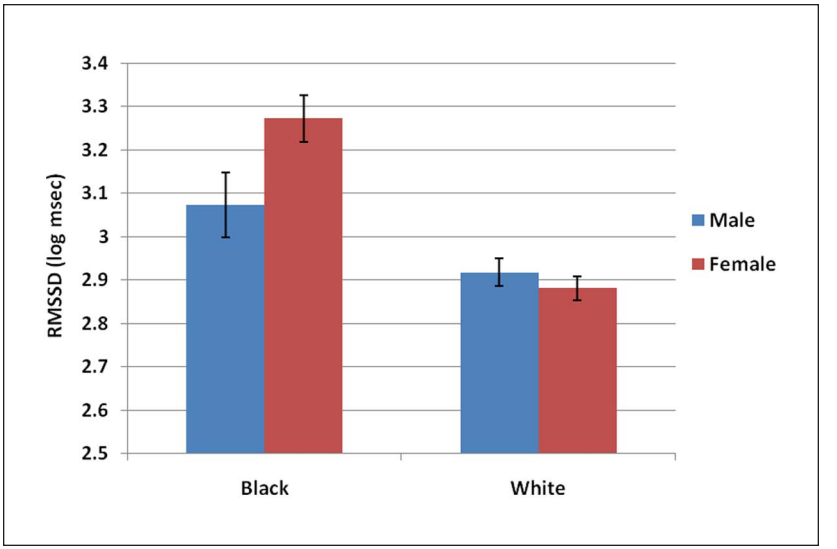

Figure 1. Fitted interaction plot showing gender differences in baseline heart rate variability for Blacks and Whites.
Note: Error bars represent plus and minus 1.96 standard errors of each point estimate.

participants ($p < .001$). Specifically, for each 10-year increase in age, RMSSD levels were .15 *SD* units lower. Findings also indicated .45 *SD* units higher levels of RMSSD among Black participants than Whites ($p < .001$). Model 2 did not show direct effects of the SES variables (education, income, parental education, childhood poverty) on RMSSD. Model 3 added a race by age interaction but did not find differences in age-trends between Blacks and Whites ($p = .435$). Model 3 also considered a race by gender interaction, which indicated that the higher levels of RMSSD among Blacks shown in Model 1 were primarily driven by differences between Black and White females ($p = .025$). The race by gender interaction is shown in Figure 1. Model 4 considered SES differences in the age trend by adding the SES by age interactions. The education by age interaction indicated steeper age-trends among less educated participants ($p = .023$). No additional differences in the age-trend were found for the other SES variables.

Heart Rate Variability Reactivity

RMSSD Reactivity. Results for RMSSD reactivity are shown in Table 3. Findings from Model 1 indicated that, net of other variables in the model, the

Table 3. Model Parameter Estimates Showing the Effects of Age, Race, and SES on HRV Reactivity to Cognitive Challenge Tasks (as Measured by Changes in RMSSD).

	Model 1		Model 2		Model 3		Model 4	
	B	*(SE)*	*B*	*(SE)*	*B*	*(SE)*	*B*	*(SE)*
Intercept	–0.157	(0.020)***	–0.154	(0.020)***	–0.152	(0.021)***	–0.154	(0.021)***
Med1 (BP)	–0.038	(0.025)	–0.038	(0.025)	–0.043	(0.025)	–0.043	(0.025)
Med2 (SNS-)	0.093	(0.058)	0.097	(0.058)	0.100	(0.058)	0.103	(0.057)
Med3 (SNS+)	–0.035	(0.030)	–0.035	(0.031)	–0.037	(0.030)	–0.039	(0.030)
Med4 (PNS-)	0.026	(0.023)	0.025	(0.023)	0.022	(0.023)	0.018	(0.023)
Med5 (PNS+)	0.076	(0.034)*	0.075	(0.034)*	0.078	(0.034)*	0.078	(0.034)*
Physical Activity	0.002	(0.010)	0.001	(0.010)	–0.001	(0.010)	–0.001	(0.010)
BMI	–0.008	(0.010)	–0.008	(0.010)	–0.006	(0.010)	–0.005	(0.010)
SBP	0.003	(0.011)	0.003	(0.011)	0.003	(0.011)	0.002	(0.010)
Marital Status	0.007	(0.020)	0.001	(0.021)	0.001	(0.021)	0.002	(0.021)
Gender	–0.003	(0.019)	–0.002	(0.019)	–0.002	(0.021)	0.002	(0.021)
Race	–0.025	(0.025)	–0.027	(0.027)	–0.025	(0.030)	–0.014	(0.030)
Age	0.026	(0.011)*	0.028	(0.011)*	0.018	(0.012)	0.016	(0.012)
Education			–0.008	(0.010)	–0.009	(0.010)	–0.007	(0.010)
Income			0.010	(0.009)	0.008	(0.009)	0.007	(0.009)
Parent Educ.			0.003	(0.011)	0.004	(0.011)	0.003	(0.011)
Child Poverty			0.016	(0.035)	0.029	(0.034)	0.037	(0.034)
Race×Gender					0.019	(0.053)	0.011	(0.053)
Race×Age					0.062	(0.026)*	0.034	(0.027)
Educ.×Age							–0.025	(0.011)*
Inc.×Age							–0.014	(0.010)
Par. Ed.×Age							0.014	(0.011)
Child Pov.×Age							0.048	(0.036)

Note: $N = 1037$ for all models. ***$p < .001$. **$p < .01$. *$p < .05$. BMI = body mass index. SBP = systolic blood pressure. Med1 = blood pressure medication. Med2 = medication with sympathetic nervous system (SNS) inhibitory influence. Med3 = medication with SNS excitatory influence. Med4 = medication with parasympathetic nervous system (PNS) inhibitory influence. Med5 = medication with PNS excitatory influence.

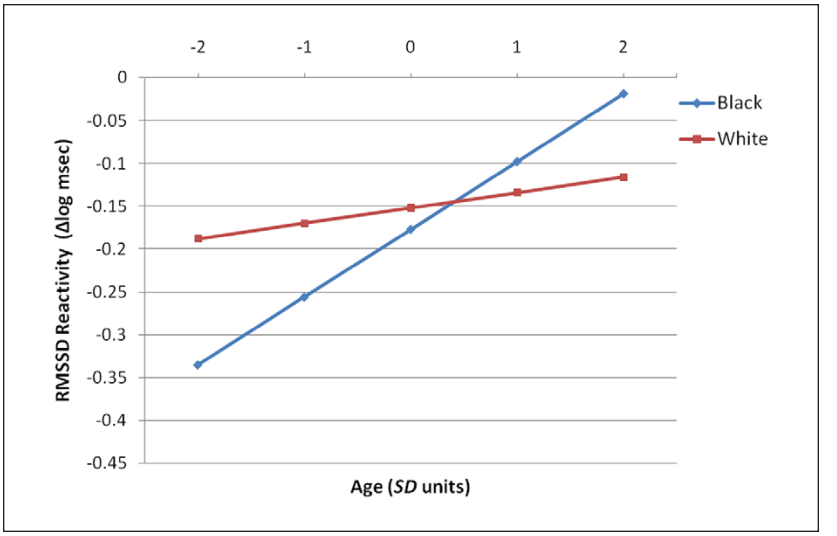

Figure 2. Fitted interaction plot showing race differences in age-trends of HRV reactivity.

Note: This plot is based on parameter estimates from Model 3, shown in Table 3. Larger negative numbers indicate greater vagal withdrawal in response to stress tasks (greater reactivity). The age range on the plot is from 31 to 77 (M = 54).

magnitude of the challenge-induced decline in RMSSD (herein referred to as RMSSD reactivity) was smaller for older participants than for younger participants (p = .017). For each 10-year increase in age, RMSSD reactivity was, on average, .08 *SD* units smaller. No difference in levels of RMSSD reactivity was found between Blacks and Whites (p=.313). Model 2 did not show direct effects of SES on RMSSD reactivity. Model 3 added a race by age interaction and showed that, as hypothesized, the age trend was significantly steeper for Blacks than for Whites (p = .019). These findings are shown in Figure 2. Model 3 also indicated that the race by gender interaction was not significant (p = .720). Model 4 added SES by age interactions. Findings from this model indicated that the age trend was significantly steeper for lower education groups (p = .020), and that after controlling for all four SES variables, the race difference in the age trend decreased by 45% and became nonsignificant (p = .208).

Discussion

It is increasingly recognized that reductions of group health disparities should be a central concern of governments at all levels (DHHS, 2011; Marmot,

Friel, Bell, Houweling, & Taylor, 2008; WHO, 2008). An understanding of the psychological and biological mechanisms behind group differences in health, and their links to context and behavior, is thus important for developing actionable plans to reduce group disparities (Williams et al., 2010). In this study, we considered race differences in age-trends in HRV and HRV reactivity—both of which are measures of autonomic nervous system functioning.

Overall, findings relating to HRV reactivity were largely consistent with our hypotheses. Analyses indicated that age-trends were steeper among African Americans than Whites for RMSSD reactivity, suggesting faster declines in flexibility of the ANS among African Americans (Figure 2). These findings are in line with theoretical perspectives suggesting that social stress influences the health of minority groups in a pattern that suggests accelerated declines in health with age (Clark et al., 1999; Geronimus, 2001; Meyer et al., 2008). These findings are also consistent with research indicating that vagal modulation of the heart declines with age and chronic stress exposure (El-Sheikh & Erath, 2011; Katz, 2007; Liao et al., 1995; Lucini, Fede, Parati, & Pagani, 2005; Sloan et al., 1994, 2008; Steptoe et al., 2005; Vrijkotte, Doornen, & Geus, 2000). Although the results indicated a steeper decline in HRV reactivity among African Americans, it should also be noted that, at younger ages, the magnitude of reactivity was greater among African Americans than Whites (Figure 2). This suggests that African Americans may have a more responsive ANS earlier in adulthood (Arthur et al., 2004). The reasons for these differences are not currently known, but seem worthy of attention in future research.

Analyses were also conducted to consider the influence of SES on age-trends in HRV reactivity, and the extent to which SES accounted for race differences. Results from these analyses suggested that age-trends in HRV reactivity were steeper for individuals with lower levels of education than their more educated counterparts. Furthermore, 45% of the race difference in age-trends for RMSSD reactivity was explained by SES. This finding is in line with studies showing that SES explains a portion of racial health disparities (Kawachi, Daniels, & Robinson, 2005; Williams, 1999), and extends this work by showing that SES is a mediator of race differences in age-trends of a specific, and neglected, biomarker of ANS functioning. Although the overall results relating to SES as a mediator were expected, the finding that education was the strongest mediator of the four SES variables considered was somewhat surprising given existing research suggesting that African Americans enjoy less health benefits from educational attainment than Whites (Shuey & Willson, 2008). In light of this research, we had expected that education may be a weaker mediator than other indicators of SES.[4] Such discrepancies in the literature—alongside the importance of distinguishing

causal mechanisms for group disparities—suggests a need for further research considering the role of specific SES indicators in racial health disparities across a range of samples and health outcomes.

Although race differences in the age trend dropped below statistical significance after controlling for SES, it is also important to note that the majority (55%) of the initial race effect (shown in Figure 2) remained. These findings are consistent with the notion that, while SES differences are important contributors to racial disparities, race often matters above and beyond the effects of social class (Farmer & Ferraro, 2005; Fuller-Rowell et al., 2012; Fuller-Rowell & Doan, 2010; Williams, 1999). This is not surprising given pervasive negative stereotypes towards African Americans (Dovidio, Kawakami, Smoak, & Gaertner, 2008; Steele, 2010) and the resulting higher levels of discrimination experienced (Fuller-Rowell et al., 2012; Kessler, Mickelson, & Williams, 1999), as well as structural factors which limit access to resources and social capital in neighborhoods with a higher proportion of Black residents (Laveist, 1993; Williams & Collins, 2001). While SES is likely to explain some of the effects of discrimination on health, studies reveal that discrimination has a direct effect above and beyond SES (Krieger, 2012; Pascoe & Smart Richman, 2009; Williams & Mohammed, 2008).

With respect to baseline levels of HRV, a range of studies have linked HRV at rest to mortality and cardiovascular outcomes (see Thayer & Lane, 2007 for a review). However, the analyses presented herein, consistent with mixed findings from prior research, showed no difference in age-trends between Blacks and Whites. Although preliminary, our results converge with findings from previous research (Sloan et al., 2008) to suggest that, unlike HRV reactivity, resting baseline levels of HRV may not be key to understanding racial disparities in health with aging.

Apart from the lack of support for race differences in age-trends of resting HRV, our analyses did show a main effect of race on on RMSSD. Specifically, consistent with the majority of previous research (Li et al., 2009; Liao et al., 1995; Wang et al., 2005), Blacks were found to have higher levels of HRV than Whites. Given that levels of cardiovascular disease are known to be higher among African Americans relative to Whites, and that higher levels of HRV are associated with better health outcomes, these findings add to the unresolved paradox in the literature (higher levels of HRV among African Americans alongside higher levels of cardiovascular disease). One promising direction for future research will be to explore psychosocial factors that may explain this paradox. For example, mental health, social competence, mindfulness, and emotion regulation have all been linked to HRV (Appelhans & Luecken, 2006; Blair & Peters, 2003;

Porges, 2007; Satyapriya, Nagendra, Nagarathna, & Padmalatha, 2009) and it is possible that these abilities may be differentially cultivated by members of a historically oppressed and currently stigmatized group. Specifically, research has shown that, as compared to Whites, African Americans have higher levels of psychological well-being (Keyes, 2009; Ryff, Keyes, & Hughes, 2003), higher levels of self-esteem and mastery (Williams et al., 2012), and lower rates of common mental disorders (Miranda, McGuire, Williams, & Wang, 2008).

Significant race by gender interactions further revealed that race differences in baseline HRV were more pronounced for females than for males. Specifically, while Black females had substantially higher levels of HRV than White females, the difference between Black and White males was less pronounced (Figure 1). Although prior research has found higher levels of HRV among females than males (Ryan, Goldberger, Pincus, Mietus, & Lipsitz, 1994; Sloan et al., 2008), the reasons for this gender difference, and the reasons that race differences are particularly present among females, are open questions for future research.

Limitations and Future Directions

The current investigation is unique in its consideration of HRV reactivity as an important marker of autonomic regulation that may contribute to an understanding of racial disparities. Although this study has significant methodological strengths (e.g., large sample size and broad age range), some limitations are important to note. Firstly, since the data were cross-sectional and collected in a single wave of the MIDUS study, it is critical to build on this research with evidence from longitudinal, cohort sequential, or repeated cross-section surveys to rule out possible cohort or period influences on what we are interpreting as aging processes (Duncan, Duncan, & Strycker, 2006; Nesselroade & Baltes, 1979; Yang & Land, 2006). For example, to be sure that age-related declines in HRV reactivity are, in fact, greater in African American adults, as compared to Whites, it will be necessary to follow the same respondents over time. Given ongoing efforts to carry the MIDUS longitudinal study forward, these analyses will hopefully become possible with planned follow-ups of biological components of the study.

A further limitation relates to the sampling design. Although the Milwaukee sample allowed for the recruitment of a large number of African American respondents into the MIDUS study, the fact that Black respondents were largely from a city-specific sample rather than a national sample limits the generalizability of the findings. Replication with additional samples is therefore an important next step.

Conclusion

Consistent with the notion that currently stigmatized and historically oppressed groups are exposed to higher levels of social stress, the findings of this study suggest that HRV reactivity declines faster with age among African Americans than Whites. Future research will be necessary to determine the extent to which HRV reactivity is linked to prospective health outcomes and contextual stresses differentially experienced by historically oppressed and currently stigmatized groups.

Acknowledgements

We thank the staff of the Clinical Research Center at the University of Wisconsin -Madison, at the University of California -Los Angeles and at Georgetown University for their support in conducting this study. The content is solely the responsibility of the authors and does not necessarily represent the official views of the Eunice Kennedy Shriver National Institute of Child Health and Human Development or the National Institutes of Health. The authors also extend gratitude to Wendy Berry Mendes for insightful comments on earlier versions of this paper, and Barry Radler for conscientious dedication that made this project possible.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: Support for this research was provided by the Robert Wood Johnson Foundation Health & Society Scholars program. This research was also supported by a grant from the National Institute on Aging (P01-AG020166; Carol D. Ryff, Principal Investigator) to conduct a longitudinal follow-up of the MIDUS investigation. The original study was supported by the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife Development. Data collection was supported by the following grants: M01-RR023942 (Georgetown), M01-RR00865 (UCLA) from the General Clinical Research Centers Program, and 1UL1RR025011 (UW) from the Clinical and Translational Science Award (CTSA) program of the National Center for Research Resources, National Institutes of Health. The first author of this study was also supported, in part, by award number T32HD049302 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development.

Notes

1. Identical analyses were also conducted using two other measures of vagal modulation (high frequency HRV, and SDRR). Findings for these measures were

similar to those reported herein. Tables showing results for high frequency HVR and SDRR are available from the first author.

2. Please refer to note 1.
3. All education variables were considered as continuous variables on a 12-point scale in order to maintain the full distribution of original responses. Dichotomous and collapsed versions of the education variables were also explored but found to have less predictive power.
4. To address the possibility that the effect of education on age-trends in parasympathetic functioning might depend on race, we also considered the three-way interaction between race, education, and age, but did not find it to be significant.

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