

**Cardiovascular Reactivity to Interpersonal Stress:  
The Roles of Race and Chronic Stress**

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## Abstract

African Americans (AAs) have higher rates of hypertension than European Americans (EAs). Stress exposure and cardiovascular reactivity to stress may be significant contributors to these racial disparities, and AAs experience greater stress across the lifespan than EAs. The purpose of the present study was to examine cardiovascular stress reactivity in the lab and whether reactivity varies by race and chronic stress. We focused on heart rate variability (HRV) in which higher values are thought to be indicative of greater emotion regulation and adaptability to stress. We hypothesized that (1) AAs will report greater chronic stress in the past year, (2) AAs will show greater cardiovascular reactivity to stress, and (3) Chronic stress will account for race differences in cardiovascular reactivity. Fifty-seven AA and EA older adult participants ( $M = 56.7$ ; 47% AA) completed a baseline interview and a modified Trier Social Stress Test (mTSST). Interviews included questions about chronic stress exposure in the past year. The mTSST required participants to give a speech regarding a recent interpersonal stressor and had four components: pre-stress rest, speech preparation, speech, and post-stress rest. A multilevel model analysis revealed that EAs had higher HRV in post-stress rest compared to pre-stress rest, speech preparation, and speech. In contrast, AAs had lower HRV during speech compared to pre-stress rest. Chronic stress exposure was not associated with HRV. These findings indicate that EAs demonstrated higher recovery while AAs demonstrated reactivity and less recovery, which may have important implications for reducing racial disparities in cardiovascular health.

*Keywords:* racial disparities, chronic stress, cardiovascular reactivity, heart rate variability

## **Cardiovascular Reactivity to Interpersonal Stress: The Roles of Race and Chronic Stress**

Racial disparities in health among African Americans (AAs) and European Americans (EAs) remain prominent in the United States. AAs have higher death rates compared to EAs for eight of the top fifteen leading causes of death including hypertension (Kochanek et al., 2019). Stress exposure and cardiovascular reactivity to stress are believed to be significant contributors to these racial disparities. AAs experience greater stress across the lifespan than EAs which may be associated with increased cardiovascular reactivity to stress. However, AAs and racial disparities in stress exposure and reactivity remain understudied. In the current study, we ask: (1) Are there race differences in chronic stress exposure? (2) Are there race differences in cardiovascular reactivity to stress in the lab? and (3) Does chronic stress account for race differences in stress reactivity? Understanding the links between race, chronic stress, and cardiovascular reactivity to stress may be key to reducing racial disparities in negative health outcomes.

## **Cardiovascular Health in African Americans**

According to a 2014 report by the World Health Organization, cardiovascular disease (CVD) is the leading cause of death due to non-communicable deaths worldwide. It accounted for 46% of the deaths globally in 2010, in which one in three Americans had at least one form of CVD. Despite large empirical efforts to reduce CVD, racial disparities in CVD morbidity and mortality remain present in the United States (Carnethon et al., 2017). Overall, AAs have not shared the same declines in CVD mortality that EAs have benefitted from in recent years. Furthermore, hypertension, or abnormally high blood pressure, is one of the most influential, modifiable risk factors for poor cardiovascular health among AAs. Hypertension prevalence among AA women above age 20 is 44% and that of men are 42.4% in the United States, the

highest in the world (Carnethon et al., 2017). In quantifying mortality, Wong et al. (2002) suggest that hypertension accounts for 15%, the highest percentage among all health issues, of potential life years lost for AAs. As such, investigating the sources of CVDs such as hypertension may have important implications for mitigating disparities in cardiovascular health.

### **Stress and Cardiovascular Health**

Cardiovascular reactivity to stress has long been linked to CVD risk although its pathways are not completely understood. Stress is generally defined as “a process in which environmental demands tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease” (Cohen et al., 1997, p. 3). Similarly, chronic stress is the “persistent negative exposure or experience of threat or excessive demand” (Baum et al., 1993, p. 277). The general adaptation syndrome by Selye (1951) proposes that stress causes an activation in the sympathetic nervous system, stimulating the ‘fight-or-flight’ response in which stress hormones are released into the bloodstream. This leads to increased heart rate, blood pressure, sweat level, and respiration. Chronic stress and continuous reactivity can affect normal bodily functions and inhibit the body’s ability to heal, leading to additional health issues.

Consequently, chronic stress exposure and reactions to stress are known to cause changes to the cardiovascular system. For example, a systematic review of 175 studies found that greater CVD risk was predicted by augmented stress reactivity and slow recovery from stress (Chida & Steptoe, 2010). A cross-sectional study on 59,798 adults also reported that higher stress scores were associated with higher risk of hypertension and elevated cholesterol levels (Gawlik et al., 2019). However, findings on the association between stress and cardiovascular reactivity have been variable. Studies have shown no change in reactivity as well as heightened and blunted

stress reactivity. In a study with repetitive stressors, cardiovascular reactivity was found to attenuate over time (Al'Absi, 1997). Anderson et. al (1998) found no race differences in cardiovascular reactivity as measured by heart rate and blood pressure to a mental arithmetic task. Another study found that after controlling for socioeconomic status, cardiovascular reactivity to stress was not significantly different among AAs and non-Hispanic Whites (Gordon et al., 2017).

### **Stress and Heart Rate Variability**

The present study focuses on heart rate variability (HRV), which is the variation between consecutive heartbeats measured in milliseconds. It is mediated by the parasympathetic nervous system and considered to be an indicative measure of emotion regulation which is linked to psychological adaptability. A flexible autonomic nervous system reflects an individual's ability to adjust their physiological status to a rapidly changing environment (Appelhans & Luecken, 2006). HRV is an autonomic measure of the influences of the sympathetic and parasympathetic systems on heart rate, representing this flexibility. Segerstrom and Nes (2007) found that university students placed in an experimental group requiring high self-regulation effort to resist eating desirable foods were observed to have higher HRV compared to those in a group which required low self-regulation effort. A growing body of research has also demonstrated that low HRV is generally associated with increased reactivity to stress while high HRV is generally associated with recovery from stress (Balzarotti, 2017). One study on young adults found that individuals with higher resting HRV were more likely than those with lower resting HRV to self-report regular use of coping strategies in regulating negative emotional reactions to stress (Geisler, 2013). Among healthy adults, a decrease in HRV was also associated with the anticipation of a lab stress task and increase in task-induced cortisol (Pulopulos et al., 2018).

## **Race Differences in Chronic Stress**

AAs are exposed to more chronic stress during their lifetime in comparison to EAs in various areas of life including education, living situations, medical care, and work (Williams & Mohammed, 2009). One reason for higher stress exposure in AAs is racial discrimination, from both perceived discrimination and institutional racism. In a census survey, blacks compared to whites were more likely to report the following economic hardships: inability to meet essential expenses, inability to pay full rent or mortgage, inability to pay full utility bill, having utilities shut off, and being evicted from one's apartment (Bauman, 1998). Additionally, segregated housing conditions yield to increased neighborhood stress, such as violence and lack of health care access (Williams & Mohammed, 2009). Despite increased efforts and awareness of inclusion, racial discrimination is still prevalent in today's society (Blank et al., 2004). Due to this pervasiveness, AAs are continuously exposed to a unique form of chronic stress that stem from racial discrimination. Furthermore, although increases in socioeconomic status (SES) is linked to improved health and AAs are more likely to have lower SES, some research suggests that this relationship depends on race (Colen et al., 2018). Thus, AAs may be exposed to higher levels of chronic stress compared to EAs regardless of SES.

## **Cardiovascular Reactivity to Stress in the Lab**

Studies on chronic stress and cardiovascular reactivity have yielded varying results. One longitudinal study which measured chronic stress and stress reactivity approximately 3.3 years apart reported that adolescents with high chronic stress showed greater diastolic blood pressure reactivity to a series of stress tasks including a mirror image tracing and a mental arithmetic task (Low et al., 2009). Increasing stress scores from Time 1 to Time 2 were also associated with increased cardiovascular reactivity, demonstrating the effect of chronic stress. Jones et al. (2016)

found that in a study of 88 healthy adults, women with higher chronic stress had heightened heart rate reactivity while men with higher chronic stress had diminished heart rate reactivity compared to the baseline of a stress task.

Few studies have investigated whether chronic stress accounts for race differences in cardiovascular reactivity, and its link is still unclear. Some studies have evaluated the effects of race on cardiovascular reactivity positing that elevated levels of chronic stress experienced by AAs due to minority status would affect cardiovascular outcomes. Lampert et al. (2005) took twenty-four-hour measurements of HRV in outpatients which revealed that AAs had the lowest HRV compared to Whites and Hispanics. In another lab study, AAs were shown to have marked changes in blood pressure and heart rate to a Cold Pressor Task compared to EAs (Gafane et al., 2016). However, these studies did not directly evaluate chronic stress. In a study in which AAs reported slightly more chronic stress than non-Hispanic whites, no significant race differences in blood pressure to a Trier Social Stress Test were found when differences in income, occupation, and education were controlled (Gordon et al., 2017). The study concluded that chronic stress may have contributed to a blunted cardiovascular reactivity. Overall, this area requires further investigation.

### **The Current Study**

Higher exposure to chronic stress by AA individuals may put them at higher risk for disparities in physical health, especially CVD. However, still little is known about the link between race, chronic stress, and cardiovascular reactivity to stress. The current study hypothesized that (1) AAs will have higher chronic stress in the past year, (2) AAs will show greater cardiovascular reactivity to stress, and (3) Chronic stress will account for race differences in cardiovascular reactivity. If the hypotheses are correct, it would suggest that chronic stress is

linked to changes in stress reactivity. Investigating the link between these concepts can help researchers understand the role of race and chronic stress in cardiovascular reactivity and cardiovascular health. Such knowledge could lead health institutions to create more group-specific interventions.

## **Method**

### **Participants**

Participants were from two studies approved by Institutional Review Board at the University of Michigan with similar design components including a baseline interview and a laboratory stress component (HUM00114002 and HUM00074983). A total of 30 participants were from a pilot study of respondents from a Midwestern suburban area who completed a baseline survey on a tablet and completed a laboratory stress study. A total of 32 participants were from a larger longitudinal sample in which respondents from a Midwestern city area completed a face-to-face baseline interview and a laboratory stress study. Five participants were dropped from analysis for incomplete data due to technical issues. Thus, the analytic sample for this study included 57 adults ages 34 to 74 ( $M = 58$ ; 47% AA; 37% male).

### **Procedure**

In the baseline, a survey questionnaire was administered to participants asking about their demographic information and chronic stress exposure in the past year. In the laboratory, participants completed two stress-inducing tasks in random order: Cold-Pressor Task (CPT) and a modified Trier Social Stress Test (mTSST). In the CPT, participants submerged their dominant hand in a tub of cold water for as long as they could, up to three minutes. In the mTSST, participants prepared for a speech about a recent stressful interpersonal event for three minutes and gave the speech in front of a large video camera for five minutes. Participants watched a



three-minute relaxing video before and after each task. Individuals wore a BodyGuardian Heart Monitor device which measured HRV. Cardiovascular data collected during the videos were used as pre-stress and post-stress baseline comparison points for the physiological measures. The current analysis included four tasks of the mTSST in the following order: pre-stress, speech prep, speech, and post-stress. All participants provided informed consent and received compensation for their participation.

### **Measures**

**Race.** Participants self-identified as African American (AA) or European American (EA). Race was coded as 0 = EA and 1 = AA.

**Chronic stress.** Participants answered a 10-item chronic stress questionnaire (Troxel et al., 2003). Example items included: *During the past year, have you experienced ongoing health problems?* and *Do you have ongoing problems due to the loss of a close friend or relative?* See Table 2 for a complete list of the 10 items. Participants responded to each item on a scale of 1 = *No*, 2 = *Yes, but not upsetting*, 3 = *Yes, somewhat upsetting*, and 4 = *Yes, very upsetting*. Each item was then dichotomously coded as 0 = no exposure and 1 = exposure to chronic stress so that each participant received a sum chronic stress score ranging from 0 to 10.

**Heart rate variability.** Cardiovascular reactivity to stress was measured as mean changes in HRV. The BodyGuardian Heart monitor is a portable electrocardiogram (ECG) monitor that attaches onto the chest with an adhesive strip. The monitor continuously transmits data to a smartphone via a Bluetooth connection, which is then sent to a secure server. The BodyGuardian Heart monitor calculates heart rate as follows: (1) for each detected beat, it calculates the average R-R interval of the previous 8 beats including the currently detected beat and (2) for each average heart rate reported every 10 seconds, it averages each of the beat's

average R-R interval and converts it to beats per minute. The R-R variance reported every 10 seconds is the statistical variance of all of the beat's instantaneous heart rate with the R-R average used in the heart rate calculation. Changes in HRV demonstrate reactivity and recovery from stress. HRV was truncated at 164 at the 95th percentile range. Mean HRV was calculated for every task.

**Covariates.** Age, gender, and education level, and time spent on each task were included as covariates. Age and time were included as continuous variables. Gender was coded as 1 = *men* and 2 = *women*. Education was coded into five categories including 1 = *no high school degree*, 2 = *graduated high school*, 3 = *some college including Associates*, 4 = *graduated college*, and 5 = *more than college*.

### **Analysis Strategy**

The first model examining whether there were racial differences in chronic stress involved a linear regression in which race predicted chronic stress. Post hoc Tukey tests were used to compare pairwise HRV means by race and task. Next, multilevel models in SAS proc MIXED were used to assess whether race and task predicted HRV. Then, models tested mediation by assessing whether the interaction between race and task changes when chronic stress is added to the model as a mediator. Another model tested moderation of the interaction between race and task with chronic stress by assessing the three-way interaction between race, task, and chronic stress. The multilevel models included two levels: level 2 was the participant and level 1 was the tasks. Models predicted mean HRV for each task. Covariates for all models included age, gender, education, and time. Age and time were grand mean centered.

## **Results**

### **Descriptives**

Participants had an average HRV of 32.75 ( $SD = 31.49$ ) across all tasks. The mean score for chronic stress was 2.74 ( $SD = 1.55$ ) and the maximum score for all participants was 6. All participants had an education level of high school completion or higher. Table 1 includes a description of the sample by race.

Pearson correlations between study variables were calculated. Race, age, gender, education, time, chronic stress, and HRV were not correlated to each other. See Table 3 for a table of correlations.

### **Are There Race Differences in Chronic Stress Exposure?**

The first linear regression model assessed whether race predicted chronic stress as a main effect. Race was not associated with chronic stress (Table 4).

### **Are There Race Differences in Cardiovascular Reactivity to Stress in the Lab?**

Next, models assessed the effects of race and task on HRV to examine racial differences in cardiovascular reactivity. A significant main effect of task was found,  $F(3, 4974) = 14.72, p < .001$ . Race was not associated with HRV,  $F(1, 50.9) = 2.63, p = .111$ . There was a significant interaction between race and task,  $F(3, 4962) = 6.45, p < .001$ . See Table 5.

The least square means HRV were 33.92 (pre-stress), 30.37 (speech prep), 28.45 (speech), and 34.38 (post-stress) for AAs and 37.67 (pre-stress), 34.69 (speech prep), 38.80 (speech), and 43.05 (post-stress) for EAs. Tukey tests further showed that AAs demonstrated significantly lower HRV in speech compared to pre-stress ( $M = -5.46, p < .001$ ) and significantly higher HRV in post-stress compared to speech ( $M = 5.93, p < .001$ ). EAs showed significantly higher HRV in post-stress compared to pre-stress ( $M = 5.39, p < .01$ ), speech prep ( $M = 8.36, p < .001$ ), and speech ( $M = 4.25, p < .05$ ). Additionally, EAs also had higher HRV in speech compared to speech prep ( $M = 4.11, p < .05$ ). See Figure 1.

### **Does Chronic Stress Account for Race Differences in Stress Reactivity?**

Two models assessed the effects of race, task, and chronic stress on HRV to examine whether chronic stress accounted for race differences in stress reactivity. See Table 6.

One model tested whether chronic stress is a mediator of the interaction between race and task. The interaction between race and task was still significant,  $F(3, 4962) = 6.45, p < .001$ . The effect of chronic stress was not significant,  $F(1, 51.2) = 0.23, p = .631$ . Both findings show that chronic stress is not a mediator.

Another model tested the three-way interaction between race, task, and chronic stress to understand whether chronic stress moderated the effect of race on stress reactivity. There was no significant three-way interaction,  $F(3, 4964) = 2.56, p = .054$ .

### **Discussion**

The current study examined the link between race, chronic stress, and cardiovascular reactivity to stress. We hypothesized that (1) AAs would report greater chronic stress in the past year, (2) AAs would show greater cardiovascular reactivity to stress, and (3) Chronic stress would account for race differences in cardiovascular reactivity. Overall, findings showed that race is an important factor for cardiovascular reactivity to stress in the lab. However, differences in reactivity were not accounted for by chronic stress.

#### **Chronic Stress and Race**

Contrary to our first hypothesis, we found no race differences in chronic stress. The current sample had relatively low chronic stress overall with no significant differences in scores between races. AAs in comparison to EAs are theorized to have higher exposure to chronic stress throughout their lives due to factors such as racial discrimination and its effects on multiple life domains (Williams & Mohammed, 2009). The present findings do not support this theory.

However, it is possible that individuals under high levels of chronic stress are less likely to participate in research, leading to non-significant race effects. It is also possible that AAs are more resilient to stress and self-report less stress as a result.

### **Differences in Cardiovascular Reactivity by Race**

In support of our second hypothesis, AAs were more reactive to laboratory stress than EAs. Low HRV is associated with increased reactivity to stress while high HRV is associated with recovery from stress (Appelhans & Luecken, 2006; Balzarotti, 2017). EAs showed higher HRV in the post-stress task in comparison to all previous tasks. EAs also had higher HRV during the speech task compared to the speech prep task. They did not show any significant decreases in HRV. As higher HRV reflects better adjustment a changing environment, these findings indicate that EAs were able to recover from the stress. On the other hand, AAs showed a significant drop in HRV from pre-stress to the speech task, indicating an increased stress reaction. Although AAs did have higher post-stress HRV relative to the speech task, they did not show as much adjustment as EAs did. Post-stress HRV for AAs was not significantly higher than pre-stress HRV or the speech prep task. Overall, EAs demonstrated more recovery while AAs demonstrated reactivity and less recovery. These findings are similar to past studies which have found higher stress reactivity among AAs compared to EAs (Lampert et al., 2005; Gafane et al., 2016). Moreover, the present findings are noteworthy because heightened stress reactivity and slow recovery from stress is associated with poor longitudinal cardiovascular outcomes (Chida & Steptoe, 2010).

### **The Role of Chronic Stress in Racial Differences in Cardiovascular Reactivity**

Interestingly, chronic stress did not account for racial differences in cardiovascular reactivity. A mediation analysis revealed that the interaction between race and task did not

change when chronic stress was added as a predictor. Thus, chronic stress is likely not a mediator of this racial difference. Similarly, chronic stress is likely not a moderator because a significant three-way interaction was not found. These findings diverge from a study by Gordon et al. (2017) which concluded that observed differences in chronic stress may have contributed to differences in cardiovascular reactivity. However, additional research is needed as few studies have directly explored the role of chronic stress in the link between race and cardiovascular reactivity.

### **Limitations and Future Directions**

The present study should be interpreted in the context of its limitations. Future studies should test whether similar results can be observed in larger samples. Contrary to past studies, we found that chronic stress was not associated with race. The current sample may not have been representative of AA populations that have higher chronic stress scores. Chronic stress can be operationalized as continuous stress during any length of time. Although we operationalized chronic stress as exposure to stressful events in the past year, results could differ if chronic stress was measured across a longer timeframe. Furthermore, studies have shown that chronic stress can encompass a wide variety of stress across different life domains (Brown et al., 2020; Spruill, 2010). Decoupling and examining the effects of specific chronic stressors (occupational stress, socioeconomic status) may be useful in further analysis of the role of chronic stress.

As a whole, this study contributes to the literature on cardiovascular reactivity to stress by showing that reactivity varies by race but not chronic stress. Although there is still much to research, the current findings provide a foundation for future researchers to explore the sources of these race differences. There has been a long interest in minimizing racial disparities in cardiovascular health. Knowledge of race differences in cardiovascular reactivity to stress may

contribute to the development of more personalized interventions to reduce these racial disparities.

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## Tables

**Table 1**

*Sample Characteristics*

	Black ( <i>M, SD</i> )	White ( <i>M, SD</i> )
	<i>N</i> = 27	<i>N</i> = 30
Age (years)	57.2 (11.1)	56.2 (11.2)
Women (%)	77.8	56.7
Education	3.6 (0.9)	4 (0.9)
Chronic stress score	2.9 (1.9)	2.5 (1.6)

*Note.* Education was coded as 1 = *no high school degree*, 2 = *graduated high school*, 3 = *some college including Associates*, 4 = *graduated college*, and 5 = *more than college*. Chronic stress ranged from 0 to 10 with 6 as the maximum observed score.

**Table 2***Chronic Stress 10-item Questionnaire*

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Do you have ongoing health problems?

Does a close friend or relative have ongoing physical or emotional problems?

Does a family member have ongoing problems with alcohol or drug use?

Do you have ongoing difficulties at work?

Do you have ongoing financial strain?

Do you have ongoing housing problems?

Do you have ongoing problems in a close relationship?

Do you have ongoing legal problems?

Do you have ongoing problems due to the loss of a close friend or relative?

Do you provide help to at least one sick, limited, or frail family member or friend on a regular basis?

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*Note.* Participants responded on a scale of 1 = *No*, 2 = *Yes, but not upsetting*, 3 = *Yes, somewhat upsetting*, and 4 = *Yes, very upsetting*.

**Table 3***Pearson Correlations Among Study Variables*

	Race	Gender	Education	Age	Time	Chronic Stress	HRV
Race	–						
Gender	.224	–					
Education	-.224	-.109	–				
Age	.045	-.022	-.017	–			
Time	-.046	-.157	.123	.128	–		
Chronic Stress	.103	.123	-.234	-.012	.238	–	
HRV	-.254	.062	.003	-.139	-.210	-.106	–

*Note.* No correlations were significant at  $p < .05$ .

**Table 4***Results of Regression Model with Race Predicting Chronic Stress*

Variable	<i>B</i>	<i>SE</i>	<i>t</i>	<i>p</i>
(Intercept)	2.30	1.24	1.85	.070
Race	-0.18	0.46	-0.40	.694
Age	-.002	0.02	-0.10	.923
Gender	-0.37	0.47	-0.79	.434
Education				
Graduated high school	1.33	1.04	1.29	.205
Some college including Associates	0.87	0.55	1.57	.122
Graduated college	1.02	0.57	1.79	.079
More than college (ref)	–	–	–	–

*Note.* \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .



**Table 5***Results of Models Assessing Cardiovascular Reactivity by Race and Task*

Variable	Model 1		Model 2	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
(Intercept)	-73.44	125.84	-75.25	126.10
Race	-7.15	4.16	-8.67	4.37
Task				
Pre-stress	-3.14**	1.00	-5.39***	1.36
Speech prep	-6.26***	1.05	-8.36***	1.45
Speech	-5.09***	0.92	-4.25***	1.28
Post-stress (ref)	–	–	–	–
Race X Task				
Pre-stress			4.92*	1.97
Speech prep			4.34*	2.07
Speech			-1.68	1.84
Post-stress (ref)	–	–	–	–
Time	10.45	11.70	10.69	11.73
Age	-0.14	0.18	-0.14	0.18
Gender	2.88	4.31	2.86	4.32
Education				
Graduated high school	8.94	9.36	9.01	9.38
Some college including Associates (ref)	5.24	5.01	5.26	5.02
Graduated college	-5.66	5.13	-5.72	5.15
More than college	–	–	–	–

*Note.* \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

**Table 6***Results of Models Assessing Cardiovascular Reactivity by Race, Task, and Chronic Stress*

Variable	Model 1		Model 2	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
(Intercept)	-88.50	128.90	-75.09	128.91
Race	-8.61	4.37	-9.93	8.05
Task				
Pre-stress	-5.37***	1.36	-0.89	2.58
Speech prep	-8.34***	1.45	-11.09***	2.70
Speech	-4.25***	1.28	-6.38**	2.40
Post-stress (ref)	–	–	–	–
Chronic stress	-0.59	1.23	-0.59	1.96
Race X Task				
Pre-stress	4.92*	1.97	2.61	3.67
Speech prep	4.34*	2.07	7.39	3.85
Speech	-1.68	1.84	2.58	3.43
Post-stress (ref)	–	–	–	–
Chronic Stress X Task				
Pre-stress			-1.84*	0.90
Speech prep			1.14	0.94
Speech			0.89	0.83
Post-stress (ref)			–	–
Race X Stress			0.48	2.53
Race X Task X Chronic Stress				
Pre-stress			1.08	1.17
Speech prep			-1.24	1.23
Speech			-1.63	1.10
Post-stress (ref)				
Time	12.01	12.03	10.77	12.01
Age	-0.14	0.18	-0.13	0.18

Gender	3.14	4.37	2.93	4.39
Education				
Graduated high school	9.74	9.52	9.67	9.51
Some college including Associates (ref)	5.76	5.13	5.73	5.16
Graduated college	-5.11	5.31	-5.21	5.39
More than college	–	–	–	–

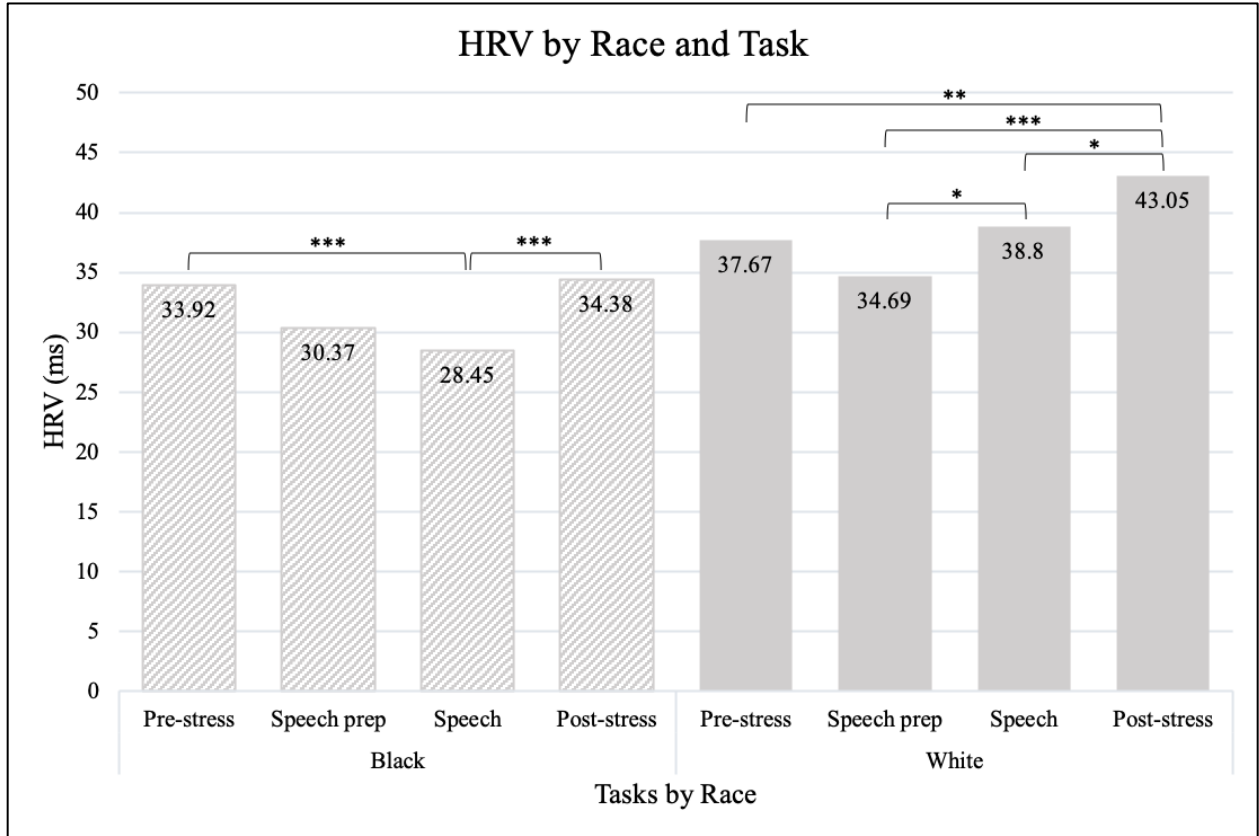
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*Note.* \* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$ .

### Figures

**Figure 1**

*Mean HRV per Task by Race After Controlling for Covariates*



*Note. \*p < .05, \*\*p < .01, \*\*\*p < .001.*