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Autonomic Reactivity and Recovery in Healthy Black, White, and Hispanic Women With and Without a Family History of Cardiovascular Disease

by

Mardís Sara Karlsdóttir

A thesis submitted in partial fulfillment
of the requirements for the degree of
Master of Arts
Department of Psychology
College of Arts and Sciences
University of South Florida

Major Professor: Kristen Salomon, Ph.D.
Jonathan Rottenberg, Ph.D.
David Diamond, Ph.D.

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Dedication

To my two beautiful daughters, Erika Lind and Kapitóla Mist.

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Table of Contents

www.manaraa.com

www.manaraa.com

www.manaraa.com

List of Tables

www.manaraa.com

www.manaraa.com

List of Figures

www.manaraa.com

List of Abbreviations

BMI	Body mass index
CAN	Cardiac autonomic neuropathy
CHD	Coronary heart disease
CVD	Cardivoascular disease
DBP	Diastolic blood pressure
ECG	Electrocardiogram
FH+	Positive family history
FH-	Negative family history
HF	High frequency
HR	Heart rate
HRV	Heart rate variability
LF	Low frequency
MI	Miocardiocal infarction
PEP	Pre-ejection period
RSA	Respiratory sinus arrhythmia
SBP	Systolic blood pressure
SES	Socioeconomic status
VLF	Very low frequency

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Autonomic Reactivity and Recovery in Healthy Black, White, and Hispanic Women With and Without a Family History of Cardiovascular Disease

Mardís Karlsdóttir

Abstract

Exaggerated cardiovascular reactivity and impaired recovery to psychological stress is independently related to cardiovascular disease (CVD), and may play a causal role in its development. I examined autonomic reactivity and recovery in 136 black, white, and Hispanic women who were predisposed to CVD, as indicated by a positive family history (FH+). Pre-ejection period (PEP; sympathetic) and respiratory sinus arrhythmia (RSA; parasympathetic) were measured during public speaking, mental arithmetic, and cold pressor tasks. Overall, FH+ participants exhibited greater RSA reactivity, while black participants exhibited impaired RSA recovery. These findings suggest that a hereditary predisposition for CVD is related to altered autonomic reactivity *before* any differences in resting levels are observed. Further, black participants did not exhibit a RSA rebound after the stress tasks, a key component in buffering the damaging effects of exaggerated sympathetic reactivity. These findings demonstrate the importance of examining parasympathetic activity in addition to sympathetic reactivity measures.

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Background

Cardiovascular disease (CVD) is currently the leading cause of death among Americans (American Heart Association, 2007). While many risk factors for CVD have been identified, their role in the etiology of CVD is not fully understood. One such risk factor is heredity, and encompasses both a family history of CVD as well as ethnicity. It has been suggested that cardiovascular reactivity (i.e. changes in cardiovascular activity in response to an acute stressor) and recovery (i.e. the time it takes for cardiovascular reactivity to return to baseline following an acute stressor) may play a role in the development of CVD. The research on reactivity and recovery has primarily focused on sympathetic activation in predicting CVD. However, in recent years the protective effects of the parasympathetic nervous system have received more attention. The purpose of the present study is to investigate the effects of ethnicity and family history of CVD on parasympathetic reactivity in healthy women.

Cardiovascular Disease

Rates. The American Heart Association (2007) estimates that nearly one in three (i.e., 80 million) American adults have at least one form of CVD. Total CVD includes high blood pressure (hypertension), coronary heart disease, heart failure, and stroke. CVD accounts for 36.3% of all deaths in the United States, and has been the number one cause of death among Americans every year since 1919 (American Heart Association, 2007). Furthermore, CVD is the cause of 48% of all deaths in Europe (European Heart Network, 2008).

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Risk Factors. The American Heart Association (2007) has classified risk factors for cardiovascular disease into major risk factors and contributing risk factors. Major risk factors significantly increase the likelihood of developing CVD, while contributing factors are factors whose significance or prevalence have not been established as strongly as major risk factors. There are three major risk factors for CVD that cannot be changed: age, sex, and heredity. The heredity risk factor encompasses both a positive family history of heart disease and ethnicity. In addition, there are three major physical risk factors for cardiovascular disease that can be altered or controlled, including high cholesterol, high blood pressure, and Type II diabetes. The risk for cardiovascular disease increases as age increases. Males have higher rates of cardiovascular disease than women until women reach menopause, at which point there is a higher prevalence in women than men (American Heart Association, 2007).

Reactivity Hypothesis – The Link between Stress and CVD?

Physiological responses to stress. Although there is disagreement on how underlying physiological mechanisms may cause CVD, there is a consensus that pathological behaviors occur in certain elements within the autonomic cardiovascular control system. Autonomic control of the cardiovascular system may become dysregulated, leading to early cardiovascular pathology. This dysregulation is one hypothesized pathway explaining the relationship between psychosocial factors and CVD development. Before discussing further the literature regarding psychosocial risk factors and dysregulation of autonomic cardiovascular control, it is necessary to present an overview of the autonomic nervous and cardiovascular systems.

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The autonomic nervous system is comprised of the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system, or "fight or flight" system, is active during physical or psychological stress, increasing physiological arousal (e.g. blood pressure and heart rate) to facilitate coping and adaptation to the stressor. In contrast, the parasympathetic nervous system, or "rest and digest" system, is most active during safety and stability where it decreases physiological arousal (e.g. heart rate). It is adaptive for a person to have the ability to transition quickly between high and low arousal states (Appelhans & Luecken, 2006).

As early as the 1930's, researchers have pointed to a possible link between autonomic responsivity to stress and vulnerability to disease (Barnett, Hines, Schirger, & Gage, 1963; McEwen, 1998; Selye, 1937; Selye, 1938; Selye, 1965). For example, Hines and Brown (1932) first reported that a large increase in blood pressure during a cold pressor task, which consists of either placing a bag of ice on the forehead or submerging the forearm into ice water, was indicative of future hypertension (as cited in Lovallo, 2005). After a series of experiments demonstrated that a prolonged stressor could lead to disease, illness, and death, Selye developed his theory of the general adaption syndrome which consists of three stages in response to a stressor: alarm, resistance, and exhaustion (Selye, 1965). Alarm and resistance follow a stressor, and if the stressor is prolonged, exhaustion may set in. Although the body's physiological systems work adaptively to respond to a stressor, if these responses are prolonged they become maladaptive. After Selye's general adaptation syndrome introduced the concept of stress-induced sympathetic hyperactivity, interest in relating the syndrome to specific diseases grew

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quickly. Selye's theory has been linked to several cardiovascular diseases, including heart disease, atherosclerosis, hypertension, and diabetes (Hellstrom, 2007; McEwen, 1998).

Reactivity. One model that has directed much of the research on stress in general is the reactivity hypothesis (Krantz & Manuck, 1984). The original reactivity hypothesis proposed that an exaggerated blood pressure response to a psychological stressor is a risk marker of future CVD (Fredrikson & Matthews, 1990; Manuck, Kasprowicz, & Muldoon, 1990). The hypothesis was developed after several studies demonstrated that young participants with normal blood pressure, but at risk for future development of hypertension, had an exaggerated blood pressure response to a variety of laboratory challenges (Fredrikson & Matthews, 1990; Manuck, Kasprowicz, & Muldoon, 1990). The reactivity hypothesis drew more attention as increasing evidence showed a connection between the Type A behavior pattern, hostility and exaggerated reactivity to stress (Myrtek & Greenlee, 1984). Since it was originally proposed, the reactivity hypothesis has been expanded to include the reactivity of other cardiovascular measures, such as heart rate.

Reactivity typically refers to a change in cardiovascular functioning from baseline in response to an aversive or challenging psychological task that takes place in a laboratory (Kamarck & Lovallo, 2003). A number of physiological changes can be observed during an acute psychological stressor, such as elevated heart rate, systolic blood pressure (SBP), epinephrine and norepinephrine levels, and adrenocorticotropic hormone levels (McCann et al., 1995; Pike et al., 1997; Treiber et al., 1993). There are several tasks that are prominent in the cardiovascular reactivity and disease risk literature. These include mental arithmetic (Matthews, Woodall, & Allen, 1993; Sharpley et al.,

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2000), a cold pressor task (Kasagi, Akahoshi, & Shimaoka, 1995; Roy-Gagnon et al., 2008), mirror star tracing (Matthews, Salomon, Brady, & Allen, 2003; Salomon, 2005), and a public speech task (Salomon, Clift, Karlsdóttir, & Rottenberg, 2009). In general, cardiovascular reactivity is considered a trait characteristic, and is believed to indicate how an individual typically responds to daily stressors (Kamarck & Lovallo, 2003). Lovallo (2005) stated that individual differences in reactivity may be due to structural and functional variations in the cardiovascular and endocrine systems, signaling either susceptibility to disease or pathology. On the other hand, Salomon (2005) suggests that individual differences in reactivity reflects the interaction of biological, personality, and social factors within a person that leads to stable response tendencies.

According to the reactivity hypothesis, exaggerated responses to acute stress can result in pathophysiologic events, which can cause tissue damage that results in system dysregulation, which then leads to disease (Obrist, Light, James, & Strogatz, 1987). Previous studies have found that exaggerated cardiovascular reactivity to a laboratory stressor is an independent risk factor for CVD (Treiber et al., 2003). These include carotid artery disease (Barnett, Spence, Manuck, & Jennings, 1997), hypertension, coronary heart disease, increased left plaque rupture, thrombus formation, and the development of carotid atherosclerosis (Manuck, 1994; Treiber et al., 2003). One study found that SBP reactivity to a cold pressor task predicted hypertension 20-36 years later, even after adjusting for study entry age, body mass index (BMI), smoking, family history of hypertension, and pretest SBP (Menkes et al., 1989). Similarly, Everson and colleagues (1996) found that SBP reactivity to an exercise stress task predicted high blood pressure four years later, which was still significant after adjusting for relevant

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factors. Furthermore, exaggerated cardiovascular reactivity has predicted the development of hypertension up to 45 years later (see Treiber et al., 2003 for a review).

Despite the accumulated evidence, some studies have found that reactivity does not predict hypertension (Carroll et al., 2001). As a result of the inconsistencies in the literature, it has been recommended that reactivity not be considered a unitary construct, as blood pressure responses are not determined by one underlying mechanism (Kasprowicz, Manuck, Malkoff, & Krantz, 1990). In addition, the underlying mechanism's role in the etiology of CVD may differ (Manuck et al., 1990; Tomaka, Blascovich, Kelsey, & Leitten, 1993). For example, blood pressure can fluctuate as a result of cardiac change (e.g. heart rate, cardiac output), or vascular change (e.g. peripheral resistance). Some researchers have argued that vascular reactivity is maladaptive, while cardiac reactivity might indicate behavioral flexibility and effective coping (Dienstbier, 1989; Lovallo, 2005). However, findings in this area have been inconsistent. For example, one study found that cardiac reactivity as opposed to vascular reactivity predicted higher blood pressure in adolescents at a three-year follow up (Matthews et al., 2003). In addition, Manuck, Kaplan, and Clarkson (1983) found that heart rate reactivity was positively related to the development of atherosclerosis in cynomolgus monkeys.

Although there is a debate among researchers as to the clinical significance of cardiovascular reactivity (Linden, Gerin, & Davidson, 2003; Manuck, 1994), the question remains as to whether exaggerated reactivity plays a causal role in the development of CVD or if it is only an indication of future risk. In a recent review, Treiber and colleagues (2003) concluded that there was sufficient evidence for the predictive power

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of cardiovascular reactivity on the etiology of CVD and its outcomes (Treiber et al., 2003).

Recovery. While cardiovascular reactivity provides important insights into cardiovascular disease, the reactivity hypothesis does not paint a complete picture. A major criticism of the reactivity hypothesis is that it does not address the duration of stress, chronic stress, or prolonged activation after stress (Schwartz et al., 2003). The failure of the cardiovascular system to recover after a stressor has received increased attention recently and is emerging as another important risk factor for CVD (Schwartz et al., 2003). Christenfeld, Glynn, and Gerin (2000) proposed that the duration for recovery and the magnitude of reactivity might have equally important roles in the development cardiovascular disease. Researchers typically measure recovery in one of three ways: (1) the amount of time it takes the cardiovascular system to return to baseline; (2) the average elevation during a post-task period; or (3) calculating the difference between baseline levels and elevation at a fixed time after the stressor (Christenfeld et al., 2000). However, these measures have been found to have low reliability and are unable to capture the process of recovery as a whole. Christenfeld, Glynn, and Gerin (2000) suggest using curve-fitting approaches instead, where both speed and amount of recovery can be assessed.

In a sample of patients with chest pain, prolonged heart rate recovery in response to a treadmill exercise test was an independent predictor of vascular dysfunction, which contributes to the development of atherosclerosis (Huang et al., 2004). Impaired heart rate recovery also predicts coronary events (Pitsavos et al., 2004), hypertension, and even mortality (Cole, Foody, Blackstone, & Lauer, 2000). Further, impaired blood pressure

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recovery has been linked to acute myocardial infarction (Laukkanen et al., 2004; Laukkanen et al., 2006), carotid atherosclerosis and low socioeconomic status, a risk factor for CVD (Steptoe, Donald, O'Donnell, Marmot, & Deanfield, 2006). One longitudinal study using borderline hypertensives reported that everyone who developed hypertension five years later failed to reach baseline diastolic blood pressure (DBP) levels during a five-minute recovery period, compared to 21% of those who did not develop hypertension (Borghi, Costa, Boschi, Mussi, & Ambrosioni, 1986). Impaired SBP recovery in adolescents with a family history of CVD was predictive of higher SBP levels four years later (Treiber et al., 2001). In an adult sample, Singh and colleagues (1999) found that delayed SBP recovery to an exercise test predicted the onset of hypertension eight years later in men. Similarly, Stewart, Janicki, and Kamarck (2006) reported that SBP recovery predicted SBP and DBP at a three-year follow-up, even after traditional blood pressure predictors were accounted for. In this study, reactivity measures were not related to future blood pressure, suggesting that some predictive information is unique to cardiovascular recovery. There is also a relationship between delayed cardiovascular recovery and other variables known to play a role in cardiovascular disease, such as hostility (Neumann, Waldstein, Sollers, Thayer, & Sorkin, 2004), anxiety (see Brosschot, Gerin, & Thayer, 2006 for review), stressful life events, and physical fitness (see Hocking Schuler & O'Brien, 1997 for a review).

Heart Rate Variability

Just as inconsistencies in the reactivity hypothesis literature led researchers to consider the underlying hemodynamic influences on blood pressure reactivity, researchers are now expanding their view of reactivity to include contributions from the

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parasympathetic nervous system. Historically, studies using the reactivity hypothesis framework have focused solely on reactivity measures mediated by the sympathetic nervous system, and largely ignored the parasympathetic nervous system. The assumption was that there was a reciprocal relationship between the sympathetic and parasympathetic nervous systems, so that if activity in one increased, activity in the other would decrease. However, it is now accepted that the sympathetic and parasympathetic branches of the autonomic nervous system can interact in a variety of ways beyond just producing antagonistic effects (i.e. a coupled reciprocal mode of activity). These include coupled nonreciprocal modes where both systems increase or decrease, or uncoupled modes where activity in one system is uncorrelated to activity in the other system (Bernston, Cacioppo, & Quigley, 1991).

One way to assess parasympathetic activity is by measuring heart rate variability (HRV), or changes in heart rate on a beat-to-beat basis. The vagus nerve, located on the tenth cranial nerve, regulates the parasympathetic activity of the heart. In the cardiovascular literature, the term vagal is often used interchangeably with parasympathetic. The sympathetic and parasympathetic branches of the autonomic nervous system regulate heart rate by altering the activity of the sinoatrial node. The sinoatrial node consists of cells located in the right atrium of the heart that generate impulses that trigger cardiac contraction. When the sympathetic fibers are stimulated, they have an excitatory effect on the firing rate of the sinoatrial node, causing an increased heart rate and a decreased inter-beat interval (the time in milliseconds between sequential heart beats). The opposite occurs when the vagus nerve is stimulated, and heart rate decreases and inter-beat interval increases. Thus, heart rate can decrease through

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either sympathetic inhibition or parasympathetic activation (Appelhans & Luecken, 2006).

HRV is often apparent in the respiratory cycle, where inspiration blocks parasympathetic influence and heart rate accelerates, while exhalation restores parasympathetic influence and heart rate decelerates. This phenomenon is referred to as respiratory sinus arrhythmia (RSA), and is considered by most researchers to be a fairly direct measure of parasympathetic influence on the heart. High RSA, or high vagal tone, occurs when there is a large amount of variability coincident with the respiratory cycle. Likewise, low RSA, or poor vagal tone, describes little or no variability in heart rate during respiration. Cardiac vagal tone has been established as a reliable individual trait (Cacioppo, Uchino, & Berntson, 1994).

HRV is measured through continuous recording of heart rate using ECG, and then calculating inter-beat interval fluctuations (Task Force, 1996). Additionally, HRV occurs at different frequencies. These frequencies are due to the unique temporal characteristics of sympathetic and parasympathetic influences on the heart. Sympathetic influences on heart rate rely on norepinephrine neurotransmission, and take longer to affect changes in heart rate. On the other hand, parasympathetic influences on heart rate involve acetylcholine neurotransmission, which produces relatively quick changes in heart rate (Pumprla, Howorka, Groves, Chester, & Nolan, 2002). These frequency differences can be examined using power spectral analysis of HRV, which typically divides inter-beat interval variation into a spectrum of frequencies using the fast Fourier transform. The frequencies are categorized into very low frequency (VLF) power, low frequency (LF) power, and high frequency (HF) power. LF power may indicate mainly sympathetic or a

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combined sympathetic and parasympathetic influence, while HF power reflects parasympathetic influence caused by RSA. Since it is not certain whether LF includes parasympathetic activity, the ratio of LF to HF power is often reported, illustrating the balance between, or predominance of, the sympathetic and parasympathetic nervous systems (Task Force, 1996).

Evidence for Importance of HRV

Resting levels linked to disease. HRV is related to various other physiological processes such as arrhythmic events (Huikuri et al., 1993), cognitive impairment (Kim et al., 2006), subclinical inflammation (Sajadieha et al., 2004), and aging (Stratton et al., 2003). Furthermore, reduced HRV is linked to a number of diseases of the cardiovascular system, including hypertension (Huikuri et al., 1996), diabetes mellitus (Carnethon, Golden, Folsom, Haskell, & Liao, 2003; Lindmark, Wiklund, Bjerle, & Eriksson, 2003), and congestive heart failure (Boveda et al., 2001). Low HRV has also been linked to psychosocial factors related to the development of CVD, such as depression (O'Connor, Allen, & Kaszniak, 2005; Rottenberg et al., 2007), anxiety (Fuller, 1992), hostility (Demaree & Everhart, 2004), and neuroticism (Haug et al., 1994).

A large amount of evidence is available on the relationship between low HRV and higher mortality. Using a population-based sample, Dekker and colleagues (2000) examined HRV in relation to mortality. Results showed that in initially healthy subjects, those with the lowest RSA levels had the highest risk of coronary heart disease and death. Another population-based study using elderly participants reported that LF, HF, and total power HRV at baseline were related to all-cause mortality five years later (Tsuji et al., 1994). Additionally, Tsuji and colleagues (1996) reported that LF, HF, and total power

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HRV were associated with increased risk for cardiac events, even after adjusting for clinical risk factors. Similar findings have also been reported using clinical populations, such as congestive heart failure patients (Nolan et al., 1998) and in patients with recent myocardial infarction (La Rovere, Bigger, Marchus, Mortara, & Schwartz, 1998).

Hayano and colleagues (1990) reported that RSA was reduced as the severity of atherosclerosis increased, and that this association remained significant after adjusting for age, sex, and previous myocardial infarction. Wennerblom and colleagues (2000) reported that in a sample of patients with uncomplicated coronary artery disease without previous myocardial infarction, total, LF and HF HRV were reduced, as compared to healthy controls. Using a patient sample, Huikuri and colleagues (1999) found that reduced total HRV predicted the progression of atherosclerosis, with those with the lowest total HRV had progressed coronary artery disease and those with the highest total HRV showing *regressed* coronary artery disease. Further, this association remained significant after adjusting for the severity of ischemic heart disease as well as common risk factors. This suggests that HRV is independently related to the progression of atherosclerosis, as opposed to being the result of severe ischemic heart disease.

Studies have indicated that reduced parasympathetic activity is apparent in hypertension. Singh and colleagues (1998) found that hypertensives displayed increased LF power and decreased RSA when compared to normotensives (i.e., those with normal blood pressure). Furthermore, normotensive males with high LF power at baseline were more likely to develop hypertension at a four-year follow up (Singh et al., 1998). Using a population-based sample, Liao et al. (1996) found that over a three year follow-up, there was an inverse relationship between RSA and incident hypertension.

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Additionally, patients have shown a decrease in RSA following a stroke, compared to controls (Dutsch, Burger, Dorfler, Schwab, & Hilz, 2007). After a right-sided stroke, LF/HF ratio is elevated compared to patients with a left-sided stroke and controls (Dutsch et al., 2007), suggesting an increase in sympathetic and a decrease in parasympathetic activity. Furthermore, a relationship is seen between HRV and the degree of neurological deficits and functional abilities in stroke patients. Specifically, individuals who were rated as functionally dependent had decreased LF and HF HRV (Arad, Abboud, Radai, & Adunsky, 2002).

Additionally, resting levels of HRV and RSA are related to diabetes, a disease that is closely related to CVD morbidity and mortality. HRV can be used to assess cardiac autonomic neuropathy (CAN), a condition that occurs when the autonomic nerve fibers that innervate the heart and blood vessels have been damaged (Maser, Mitchell, Vinik, & Freeman, 2003). CAN changes the regulation of blood pressure, heart rate, and HRV, and is related to sudden cardiac death and susceptibility to ventricular arrhythmias (Gerritsen et al., 2001). CAN has a high prevalence rate in Type I and Type II diabetes, and can be seen during the early stages of diabetes (Valensi, 2003). In diabetics, CAN is an independent risk factor for chronic kidney disease (Valensi, 2003), mortality (Gerritsen et al., 2001; Maser et al., 2003), and coronary heart disease (Liao, Carnethon, Evans, Cascio, & Heiss, 2002). Even in the absence of CAN, diabetics have increased heart rate and reduced total HRV (Pagani et al., 1988). Reduced RSA is seen even in the early stages of diabetes (Faulkner, Hathaway, Milstead, & Burghen, 2001). Additionally, Gottsäter, Ahlgren, Taimour, and Sundkvist (2006) found that HRV decreases with the

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duration of Type II diabetes in a population-based sample of adults, and that this decrease is more than what would be anticipated by age alone.

Impaired glucose regulation, an indicator of Type II diabetes (American Heart Association, 2007), is related to parasympathetic dysfunction in humans (Takayama, Sakura, Katsumori, Wasada, & Iwamoto, 2001). One study found that poor glycemic control measured in 1985 independently predicted lower absolute power values of all HRV indices ten years later (Makimattila et al., 2000). A negative correlation between insulin sensitivity and the ratio of LF/HF spectral power has also been reported (Lindmark et al., 2003). Autonomic imbalance is also seen in insulin-resistant individuals without diabetes (Flanagan et al., 1999). Low HRV may also predict future development of diabetes in both healthy subjects (Carnethon et al., 2003) and non-diabetic offspring of Type II diabetics (De Angelis et al., 2001). Lindmark, Wiklund, Bjerle, and Eriksson (2003) found that first-degree relatives of patients with Type II diabetes had lower HRV and RSA than healthy controls, even though there was no difference between groups on fasting blood glucose or glycosylated hemoglobin.

RSA reactivity hypothesis. There are three main assumptions of the reactivity hypothesis. First, reactivity should be a stable trait, and be consistent across stressors and situations. Second, reactivity should be a stable trait over time. The third assumption states that reactivity should be able to predict resting cardiovascular levels in the future (Manuck et al., 1990). In applying RSA to the reactivity hypothesis framework, these same assumptions must also hold true. There is evidence supporting the first assumption, with moderate to large correlations in RSA reactivity across different tasks (e.g., Cacioppo et al., 1994; Salomon, Matthews, & Allen, 2000; Salomon, 2005; Sloan et al.,

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1995; Sloan, Shapiro, Bagiella, & Gorman, 1995). Further, evidence suggests that RSA reactivity is temporally stable across months (Sloan, Shapiro, Bagiella, & Gorman, 1995) as well as years (Salomon, 2005). Lastly, there is evidence from a sample of children that RSA reactivity was predictive of resting levels of blood pressure and RSA at a three year follow-up (Matthews et al., 2003; Salomon, 2005). This suggests that RSA reactivity may also predict future hypertension and subsequent CVD. Gianaros and colleagues (2005) also reported that greater decreases in RSA to a stressor were related to coronary and aortic calcification, a subclinical marker of atherosclerosis. However, research is still young and more evidence needs to be collected to determine if RSA reactivity is related to disease.

RSA reactivity and biopsychosocial factors. The next step in fitting RSA into a reactivity hypothesis framework would require RSA reactivity to be linked to biopsychosocial factors that have already been shown to predict disease. Two candidates based on prior research would include a genetic susceptibility to CVD, i.e. a positive family history, as well as ethnicity. Both of these factors have been linked to an increased risk for CVD, and there is supporting evidence for increased sympathetically mediated cardiovascular reactivity related to both factors.

Family History

Family history and disease risk. A positive family history is typically defined as having one or more parents who have been diagnosed with, or prescribed medication for, a cardiovascular disorder such as hypertension or coronary heart disease. A negative family history is having both parents free of cardiovascular diseases. Positive family history is an established risk factor for future development of cardiovascular disease

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(American Heart Association, 2007). Several epidemiological studies have demonstrated an increase risk of hypertension if a family history exists (Harlan, Osborne, & Graybiel, 1962; Ness, Markovic, Bass, Harger, & Roberts, 2003; Tozawa et al., 2001), and the relationship becomes stronger as prevalence within family increases (Tozawa et al., 2001). To investigate the relationship between family history of cardiovascular disease (coronary heart disease, stroke, hypertension, and diabetes) and future onset, Williams and colleagues (2001) combined data from two large population-based studies for a total of over 130,000 families. Their results showed that although only 14% of families had a positive family history of coronary heart disease, 72% of all early coronary heart disease happened in these families. Furthermore, while only 11% of families reported a family history of stroke, 86% of early strokes occurred in these families. They concluded that most cardiovascular events with early onset are seen in people with a positive family history of cardiovascular disease.

Family history and reactivity. Although the mechanisms as to how a family history of CVD may lead to the future development of CVD is still unclear, a number of researchers have proposed that specific cardiovascular patterns or responses may be inherited and that these responses aid in the development of CVD (Fredrikson & Matthews, 1990; Manuck, Proietti, Rader, & Polefrone, 1985; Treiber et al., 1993). Subsequently, there have been many studies conducted on the relationship between a positive family history of hypertension and cardiovascular reactivity to an acute stressor in participants who are normotensive (see Pierce, Grim, & King, 2005 for a review). Early results indicated that healthy individuals with a positive family history exhibited greater reactivity compared to those with a negative family history (Fredrikson &

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Matthews, 1990; Manuck et al., 1985; Treiber et al., 1993). Treiber and colleagues (1993) reported that children with a positive family history had greater reactivity to a cold pressor and a video game task. In one study, those with a positive family history had increased reactivity to a role-playing task (Holroyd & Gorkin, 1983). Manuck and colleagues (1985) found similar results using a mental arithmetic stress task. A meta-analysis found that individuals with a positive family history of hypertension tend to have greater DBP responses to acute stressors (Fredrikson & Matthews, 1990).

However, several studies have failed to confirm the relationship between family history and cardiovascular reactivity (e.g., Anderson, Williams, Lane, Houseworth, & Muranaka, 1987; Polefrone & Manuck, 1988; Strickland, Myers, & Lahey, 1989). Polefrone and Manuck (1988) investigated the effect of family history in women and cardiovascular reactivity to a mental arithmetic and concept-formation task, but found no differences between family history groups. Similarly, Strickland, Myers, and Lahey (1989) examined parental history of hypertension in black and white females and reported no differences in reactivity to a mental arithmetic task. Anderson and colleagues (1987) showed contradicting results when they examined reactivity to a mental arithmetic stressor in young black females. They reported that subjects with a negative family history had *greater* reactivity. Muldoon and colleagues (1993) reviewed the past literature and concluded that a positive family history of hypertension on its own was not enough to predict future hypertension, but a positive family history combined with an elevated resting SBP was a significant predictor. Another study found that a positive family history of hypertension was not significantly related to cardiovascular reactivity in healthy individuals, but that a positive family history of CVD in general was related to

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slower blood pressure recovery and an increase in blood pressure reactivity (Wright, O'Donnell, Brydon, Wardle, & Steptoe, 2007). They also found that women with a positive family history exhibited higher heart rate and HRV reactivity measures than women with a negative family history (Wright et al., 2007).

Family history and recovery. The literature on cardiovascular recovery and family history is limited and inconsistent. For example, Wright and colleagues (2007) reported that subjects with a family history had delayed blood pressure recovery following a speech stressor and a Stroop task. On the other hand, using a sample of normotensive white males, Schneider et al. (2003) reported that those with a negative family history had delayed recovery following a mental arithmetic task. Thus, more research focusing on family history and recovery is warranted.

Family History and HRV/RSA Reactivity and Recovery. One study examined autonomic differences using adult men and women with either a positive or a negative family history of hypertension (Davrath, Goren, Pinhas, Toledo, & Akselrod, 2003). They reported that moving from a supine to standing position led to greater LF HRV reactivity in the positive family history group when compared to the negative family history group. Hatch and colleagues (1986) examined heart period reactivity to tilt, mental arithmetic, and cold pressor tasks in normotensive males with and without a family history of hypertension. They did not find family history differences in response to any of the tasks; however, their sample was limited to eight subjects per group. Miller (1994) also examined males with and without a family history of hypertension, who underwent several psychological stressors. Although all subjects had RSA reactivity to the cold pressor and handgrip tasks, they found no family history group differences to

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any task. Mezzacappa, Kelsey, Katkin, and Sloan (2001) reported that subjects with a positive family history of CVD did not differ from those with a negative family history in HRV reactivity to psychological stress tasks. However, they did find a significant difference in HRV recovery, with positive family history subjects showing less of a vagal rebound compared to negative family history subjects.

Limitations of Previous Family History Research. Previous literature on family history is inconsistent regarding cardiovascular reactivity, and extremely limited regarding cardiovascular recovery and RSA reactivity and recovery. Further, there are several factors that most family history studies have in common that may have resulted in a loss of power and validity. For example, although participants are often relatively young, most studies only assess parental history and do not include other relatives such as grandparents (e.g., al'Absi, Everson, & Lovallo, 1995; Anderson et al., 1987). This may result in an underestimation of familial risk of CVD when research is conducted on younger samples such as college students who also have younger parents, because symptoms of CVD may not have developed, and it is possible that many parents have yet to be diagnosed with a disease when it is in fact present. To resolve this, it has been suggested that information be collected on both sets of parents and grandparents (Silberberg, Fryer, Wlodarczyk, Robertson, & Dear, 1999). In the medical field, the "gold standard" for assessing family history is by including a three-generation pedigree interview (Wattendorf & Hadley, 2005), which includes the individual and their parents, as well as grandparents, siblings, aunts, and uncles. Several larger studies have incorporated this standard into their assessment, including the U.S. Surgeon General's Family History Initiative, the Utah Family Health Report, and the Heart of Diabetes

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study. Further, recording the age of disease onset is often overlooked, but early onset of CVD can be useful in identifying individuals with a high familial risk (Taraboanta, 2008).

Another limitation occurs when family history of CVD is assessed in the laboratory where participants are unable to confer with their relatives. One study reported that when black, white, and Hispanic college students were asked to report their family history of CVD and diabetes, they had a considerable lack of knowledge (Koutoubi & Huffman, 2002). Further, Yoon, Scheuner, and Khoury (2003) stated that take-home questionnaires where the individual is able to review family records or confer with relatives about their history were more accurate than questionnaires or surveys completed on the spot. Another limitation to most previous family history studies is that typically, only family history of hypertension is assessed, and not a broader range of cardiovascular diseases.

Ethnicity

Ethnicity and disease risk. Racial and ethnic disparities are evident in the prevalence of CVD. In particular, African Americans have the highest levels of total CVD, stroke, hypertension, and obesity. The highest levels of coronary heart disease are seen among white males and black females. Although Mexican Americans have the lowest total CVD rates of these three ethnic groups, they have higher total cholesterol, diabetes, and pre-diabetes rates than Caucasians (American Heart Association, 2007). Regarding total CVD, African Americans have the highest rates, with 44.6% of men and 49% of women over the age of 20 having some form of CVD. White Americans have the second highest rates, with 37.2% of men and 35.0% of women having some form of

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CVD. Hispanics have the lowest rates, with 31.6% of men and 34.4% of women having some form of CVD (Rosamond et al., 2008). Cardiovascular disease is the leading cause of death across all racial and ethnic groups (Rosamond et al., 2008).

Hypertension is an independent risk factor for the development of CVD (Rosamond et al., 2008). African Americans have the highest rates of hypertension, while hypertension rates for Hispanics and non-Hispanic Whites are similar (Rosamond et al., 2008). However, Hispanics have the lowest rate of control for hypertension (Ong, Cheung, Man, Lau, & Lam, 2007). The incidence of severe hypertension is four times higher among black men than white men (Lackland & Keil, 1996). Further, the death rates from hypertension are much higher in African Americans (~50 in Blacks, compared to 18.1 in total population) (Centers for Disease Control and Prevention, 2004). For first-time stroke, Blacks have almost twice the risk of Whites (NHLBI, 2006). Further, Mexican Americans also have a higher incidence of stroke compared to Whites (Morgenstern et al., 2004).

Racial and ethnic disparities are seen across most risk factors for cardiovascular disease as well. For example, being overweight (BMI >25) or obese (BMI >30) is a risk factor for developing CVD. While African American women are the most likely to be overweight compared to females of other ethnicities, African American men are the least likely to be overweight. Further, while there is little difference in the rates of obesity across ethnicity for men (all approximately 30%), there is a disparity seen in females. For African American females, ~50% are obese, compared to ~40% of Mexican American females and ~30% of White females (Rosamond et al., 2008).

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Racial and ethnic health disparities are also seen in the rates of diabetes. Data from the Center for Disease Control demonstrated that disparities in diabetes are particularly evident when comparing Hispanics in the United States and Puerto Rico to non-Hispanic Whites, with Hispanics being twice as likely to develop diabetes (9.8%, compared to 5.0%) (CDC, 2004). Robbins, Vaccarino, Zhang, and Kasl (2000) reported that both Hispanic and Black minority groups are at least twice as likely as Caucasians to develop Type II diabetes. Further, death rates from diabetes are twice as high in Blacks when compared to Whites (Rosamond et al., 2008).

Ethnicity and psychosocial risk factors for CVD. There are also social, environmental, and psychological risk factors that contribute to CVD risk. One of these is socioeconomic status (SES), which is typically assessed using highest education level attained and income, and is inversely related to CVD (e.g., Anderson & Armstead, 1995). One study that examined coronary heart disease in male civil servants in the United Kingdom over a ten-year period found that mortality was inversely related to SES across the entire gradient (Marmot & McDowall, 1986). Other studies have confirmed that people who are lower in SES are more likely to develop CVD and have more risk factors than people higher in SES (Cabrera et al., 2001; Kanjilal et al., 2006; Pickering, 1999). Among low-income populations, mortality from CVD is 24% higher than among the population as a whole (USDHHS, 1990). This puts certain minority groups at particular health risk, as black and Hispanic individuals have lower levels of accumulated wealth, less education, higher rates of unemployment, lower income, lower rates of private health insurance, and have to rely more on public health care programs than White Americans (Ashton et al., 2003; Krieger, Williams, & Moss, 1997). Further, Blacks and Hispanics

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often have less access to health care (USDHHS, 1990; Yee, et al., 1995). Minority individuals are also more likely to have misperceptions about the severity of an illness, have inadequate follow-up health care, have fewer blood pressure and cholesterol screening, and are less likely to withstand treatment costs (Winkleby, Kraemer, Ahn, & Varady, 1998).

Low SES and other environmental factors all contribute to a high level of daily, or chronic, stress. Results from the Malmö Preventative Project showed that chronic stress independently predicts CVD, particularly stroke, in men, even after adjusting for other known risk factors (Öhlin, Nilsson, Nilsson, & Berglund, 2004). This is particularly important to minorities' health, since ethnicity, in addition to being a hereditary risk factor, is also a psychosocial factor due to the added stress of discrimination and racism (Clark, Anderson, Clark, & Williams, 1999). This is particularly true for minorities in the United States, as economic, cultural, social, and historical environments vary greatly between racial groups.

Ethnicity and reactivity. Many studies have attempted to discover differential reactivity patterns between Blacks and Whites. As stated previously, exaggerated responses to stress may be a marker or a mediator for the development of CVD and Blacks are at an increased risk for CVD morbidity and mortality. Anderson and colleagues (1993) suggested that, on average, Black Americans encounter greater stress on a daily basis than Caucasian Americans, and this extra stress may lead to differential cardiovascular reactivity patterns. Further, racism, SES, social support, and various personality factors influence reactivity among Blacks (Anderson & Armstead, 1995). Racism and discrimination is often considered by researchers to be a chronic social

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stressor, which has been shown to be related to increased reactivity (Fang & Myers, 2001; Vrana, 2002).

Many of the early studies examining racial differences in reactivity were conducted by Anderson and his colleagues. One study reported that black men had greater cardiovascular reactivity than white men in response to a cold pressor task (Anderson, Lane, Muranaka, Williams, & Houseworth, 1988). Treiber et al. (1990) reported that black male children and young adults had greater blood pressure reactivity than Whites in response to a forehead cold pressor task. A study examining children's reactivity over a seven year time period found that black children had greater reactivity than white children (Murphy, Stoney, Alpert, & Walker, 1995). Jackson and colleagues (1999) evaluated racial differences in 272 black and white children with a positive family history of hypertension. Using aggregated reactivity scores from four different stress tasks, they found that black children had greater blood pressure reactivity, and less heart rate reactivity than white children.

However, reactivity studies examining racial differences have not yielded consistent results (see Anderson, McNeilly, & Myers, 1992 for a review). For example, Anderson et al. (1989b) reported that black females had exaggerated SBP responses to a cold pressor task than white females, but that there were no significant differences to a mental arithmetic task. One study conducted by Anderson et al. (1988) found an opposite effect using a mental arithmetic task, where black males had significantly less cardiovascular reactivity compared to white males. Other studies have reported no significant racial differences. For example, Anderson, Lane, Taguchi, and Williams (1989a) found no racial differences in black and white males in response to a mental

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arithmetic and forehead cold pressor task. Similar results were reported by Morell, Myers, Shapiro, Goldstein, and Armstrong (1988) in response to a mental arithmetic task, using a sample of black and white males.

One reason explaining the inconsistent literature may be that blood pressure and heart rate measures alone do not portray a complete picture. In other words, while Blacks and Whites may be similar in the magnitude of blood pressure reactivity to an acute stressor, there may be differences in the hemodynamic profile of the response (Treiber et al., 1990). There are many factors that can cause a change in blood pressure, including stroke volume, heart rate, and vascular resistance. A more accurate portrayal of racial differences can be seen by incorporating impedance cardiography techniques, so that the underlying hemodynamics of cardiovascular reactivity can be assessed. After researchers started including impedance cardiography, reactivity patterns between Blacks and Whites have become somewhat more consistent. More specifically, Blacks tend to show more vasoconstrictive reactivity (increase in vascular resistance), while Caucasians exhibit a myocardial response (decreased vascular resistance, increased cardiac output, stroke volume, and heart rate) (e.g., Light, Turner, Hinderliter, & Sherwood, 1993).

Although racial group differences in reactivity have shown a generally consistent pattern of reactivity between black and white males, reports have been much more inconsistent with black and white females. For example, Anderson et al (1989b) found no differences between black and white females in vascular resistance in response to a mental arithmetic and forehead cold pressor task. Light et al. (1993) found that black men had greater vascular reactivity and white men had greater cardiac output and heart rate reactivity, but there was no consistent pattern found for reactivity in black or white

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females across tasks. Furthermore, in a beta-blockade study using propranolol, there were no racial differences observed between black and white females on any cardiovascular parameter (Light & Sherwood, 1989).

While there is a great deal of studies that have examined racial differences between Blacks and Whites in cardiovascular reactivity, there has been little research examining ethnic reactivity differences using Latinos. This is an important gap in the cardiovascular reactivity literature, as Hispanics/Latinos are the fastest growing population in the United States, and are now the largest minority group in the U.S. with 45.5 million people in 2007. Blacks are the second largest minority group with 40.7 million in 2007 (U.S. Census Bureau, 2008). Hispanics in the U.S. are subjected to many of the same daily stressors that are believed to contribute to chronic stress in African Americans, such as low SES and discrimination, as well as additional stressors such as language barriers, immigration and acculturation issues.

Ethnicity and recovery. Ethnic differences are also seen in some studies examining cardiovascular recovery from stress, although this literature is much more limited than the reactivity literature. For example, Treiber et al. (1993) reported that black children had impaired blood pressure recovery in response to an exercise and forehead cold pressor task. Jackson et al. (1999) reported that black males and females had more delayed blood pressure recovery than Whites after stress. Mills and Berry (1999), on the other hand, found that black men and women exhibited greater blood pressure recovery compared to Whites. Light et al (1993) found that Whites had less heart rate recovery than Blacks after a five-minute recovery period. Gillin et al (1996) reported that white men had less blood pressure recovery following a speech task than black men and

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women. Further, Whites had less total peripheral resistance recovery after a mirror-tracing task compared to Blacks, and white women had the least heart rate recovery compared to black women and men. However, due to the limited literature examining racial differences in cardiovascular recovery, consistent patterns have not been reported.

Ethnicity and resting levels of HRV/RSA. Previous literature on the differences in HRV between Blacks and Whites is not only limited, but inconsistent as well. For example, Zion et al. (2003) examined racial differences in autonomic function and reported that Blacks had significantly lower RSA and a higher LF/HF ratio (i.e., sympathovagal balance) when compared to non-Blacks. On the other hand, Liao et al. (1995) reported that Blacks had significantly lower LF HRV, higher RSA, and higher LF/HF ratio when compared to Whites, after adjusting for age and gender. Wang et al. (2005) examined black and white adolescents and reported that Blacks had a lower LF/HF ratio and higher RSA than Whites, after adjusting for age and heart period. Choi et al. (2006) reported that Blacks had lower LF and RSA, but that there were no differences in LF/HF ratio.

Ethnicity and HRV reactivity and recovery. Unfortunately, the literature on racial differences in HRV reactivity is even more limited. Urbina et al. (1998) investigated HRV reactivity using black and white adolescent males. Subjects underwent four stressors, including orthostatic tilt, isometric handgrip task, Valsalva maneuver, and a hand cold pressor task. They reported that Blacks had higher RSA values during all stressors, and that Whites had higher LF/HF ratio values across all stressors. This suggests that Blacks had higher parasympathetic activity and Whites had higher sympathetic activity.

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Current Study

The CVD epidemic is one that affects all Westernized countries. There are a number of factors that are related to the development of CVD, including social, biological, psychological, and environmental factors. While there are several known risk factors, the cause and etiology of CVD is not completely known. Further, there is even less known regarding explanations for the higher prevalence of CVD seen among minorities.

The reactivity hypothesis has been supported throughout the years as a link between psychological stress and the development of CVD. However, the reactivity hypothesis has largely ignored the parasympathetic contributions to cardiovascular reactivity. Resting levels of RSA, a measure of parasympathetic activity, have been linked to cardiovascular diseases, as well as diabetes. There are several known risk factors for CVD, including a positive family history of CVD and ethnicity. Cardiovascular reactivity has been researched for both of these factors, but is limited and inconsistent for RSA reactivity.

The present study attempts to expand the previous research by incorporating RSA reactivity into the traditional reactivity hypothesis model and linking it to known predictors of CVD, specifically positive family history and ethnicity. Additionally, this study will attempt to overcome methodological limitations of previous family history studies by assessing the history of CVD in both parents and grandparents, the age of onset of disease, and by using a wider definition of CVD that includes hypertension, high cholesterol, stroke, diabetes, myocardial infarction, and coronary heart disease. One more

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unique contribution will be the inclusion of Hispanics, which have been largely ignored in the previous reactivity literature.

Hypotheses:

1) Participants with a family history of cardiovascular disease will have greater reactivity to stress tasks than participants with a negative family history. This will be evident in greater decreases in RSA during tasks and greater increases in sympathetically mediated responses during tasks.
2) Participants with a family history of cardiovascular disease will have impaired recovery following stress tasks than participants with a negative family history.
3) Black and Hispanic participants will exhibit greater reactivity to stress tasks compared to European American participants.
4) Black and Hispanic participants will exhibit impaired recovery compared to European American participants.

I also explored interactions between family history and ethnicity on reactivity and recovery. However, I did not hypothesize effects specifically as research in this area is limited.

Method

Participants

A total of 136 undergraduate females from the University of South Florida participated in the study. Of these, 44 were white, 44 were black, 46 were Hispanic, one was Middle Eastern, and one was missing ethnicity data. The mean age was 20.8 years old. Participants were recruited from the psychology department's online participant pool, Sona Systems, and were compensated with course credit or extra credit in select

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psychology courses. Participants were excluded from participation if they reported a personal diagnosis of cardiovascular disease or arrhythmias, were currently taking any medication that might affect the cardiovascular system, reported having diabetes, or who reported being pregnant. Further, only participants who knew both of their biological parents and who reported having biological parents that were in the same racial/ethnic group as the other were included. Bi-racial or multi-racial participants were not included in the study.

Measures

Demographic questionnaire. Information regarding participants' age, household income, and ethnicity were collected online via Sona Systems and in the laboratory.

Family history questionnaire. Family history of cardiovascular disease was assessed online via Sona Systems. Participants were asked whether their parents or grandparents had ever been diagnosed with or prescribed medication for any of the following conditions: Coronary heart disease (such as myocardial infarction, coronary bypass graft surgery, or angioplasty), hypertension (high blood pressure), high cholesterol, stroke, or diabetes. If applicable, they were asked to further specify whether coronary heart disease occurred as early (before age 55) or late (after 55) onset, and whether diabetes was Type I or Type II. The questionnaire was completed without any time limitations, and the participants could exit and resume the questionnaire from where they left off. This made it possible for participants to confer with their parents or other relatives if they chose to do so. The family history questionnaire is presented in Appendix A.

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Health questionnaire. Information was collected regarding biological and behavioral factors that might affect the cardiovascular system. This included questions on previous history of heart disease, arrhythmias, and high blood pressure, as well as smoking, caffeine consumption, and exercise. Females were asked to name the first day of menstruation during their last cycle. The health questionnaire is presented in Appendix B.

Cardiovascular reactivity tasks

Cold pressor task. A re-usable ice pack was placed on participants' foreheads for three minutes. The temperature of the ice pack was kept between 0 °C to 3 °C. In the event that the task became unbearable, participants were told that they were allowed to remove the ice pack. All participants were able to continue the task for the full three minutes. This task typically elicits increases in RSA (e.g. Hughes & Stoney, 2000).

Mental arithmetic task. Participants were instructed to subtract the number seven from a four-digit number for three minutes. They were told to do this as quickly and accurately as possible, and to say each number aloud. If participants lost their place, another four-digit number was assigned to them. This task typically elicits large parasympathetic withdrawals (i.e. decreases in RSA) and sympathetic activation (i.e. decreases in PEP) (Berntson et al., 1994).

Speech task. Participants were asked to prepare and give a speech. Instructions were played over an audio speaker describing a scenario where the participant was pulled over by a police officer for an unfair traffic violation and is now in traffic court defending themselves. They were then given a study card with several points to make during their speech and three minutes to quietly prepare. After the preparation period, the

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experimenter informed them that they would be videotaped and that the quality of their speech would be evaluated. They were then given three minutes to deliver their speech. This task typically elicits large parasympathetic withdrawals (i.e. decreases in RSA) and sympathetic activation (i.e. decreases in PEP) (Berntson et al., 1994).

Physiological Recording Apparatus

A Biopac MP150 system with an ECG100 electrocardiogram (ECG) amplifier and two RSP100 respiration pneumogram amplifiers (Biopac Systems, Inc., Goleta CA) were used to amplify and transducer the ECG and respiration signals. The ECG was recorded using Cleartrace LT disposable Ag/AgCl electrodes (Conmed Andover Medical, Haverhill, MA), placed in a modified Lead II configuration on the participant's chest. Respiration was recorded using two TSD201 respiratory effort transducers amplified using two RSP100C respiration amplifiers (Biopac Systems, Inc., Goleta, CA). One transducer was placed around the abdomen and another was placed around the chest. Respiratory depth was calibrated against a fixed volume bag. Impedance cardiography (ZKG) and the ECG were used for the measurement of pre-ejection period (PEP; an indicator of sympathetic activity measured as the contractile force with which the heart pumps blood). A Biopac NICO100C (Biopac Systems, Inc., Goleta CA) obtained transthoracic impedance waveforms (Z_0, dZ/dt) using a tetrapolar lead configuration. Disposable aluminum/mylar band electrodes were applied to the neck and chest following published guidelines (Sherwood et al., 1990). Current electrodes supply a 4 mA, 100 kHz signal to the thoracic region. The signals were sampled at 1000 Hz per channel by a Dell Optiplex Pentium-4 PC with A/D board. Acquisition and storage of ECG, respiration, and impedance cardiography was accomplished using AcqKnowledge

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3.7.2 software (Biopac Systems, Inc.). Systolic (SBP) and diastolic (DBP) blood pressure was measured using an Accutorr Plus non-invasive blood pressure monitor (Datascope Corp., Mahwah, NJ) according to published guidelines (Shapiro et al., 1996).

Experimental Protocol

Before arriving at the laboratory, participants had completed the online questionnaire assessing their family history of cardiovascular disease. Upon arrival, participants were greeted by an experimenter and asked to carefully read the informed consent form. When participants finished reading the informed consent form, the experimenter answered any questions and asked the participant to provide consent by signing the form. Participants then completed the health questionnaire. Next, the experimenter attached two bands of disposable electrode Mylar tape to the participant's neck and two bands around their torso according to published guidelines (Sherwood et al., 1990). The experimenter then used an alcohol swab to clean and prepare the skin beneath the right collarbone and the left ribcage before adhering two silver-silver chloride electrodes to the skin in a modified lead II configuration.

Next, participants were led into a small room and seated in a comfortable chair. The experimenter attached leads to the Mylar bands and the ECG electrodes and attached a blood pressure cuff to the upper part of the participant's non-dominant arm. Two blood pressure readings were taken to make sure the equipment was working properly. The experimenter then left the room and instructed the participant to relax while they watched a neutral travel film about Alaska for a ten-minute baseline and acclimation period.

The three stressor tasks were then administered in counterbalanced order. The participant heard instructions through a computer speaker before completing a pre-task

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appraisal questionnaire. After completing each stressor task, the participant was instructed to sit quietly for a 10-minute recovery period before receiving a post-task appraisal questionnaire. Once all stressor tasks were completed, the Mylar tape and electrodes were removed and the participants' weight, height, waist, and hip measurements were recorded. The participant was thanked for their participation and debriefed.

Data Quantification and Reduction

During the baseline, impedance and ECG data was collected during the last five minutes, and three blood pressure recordings were taken at minutes 6, 8, and 10. Blood pressure readings were taken during minutes 1 and 3 during each of the stressor tasks, and on minutes 1, 3, 5, 7, and 9 of the recovery periods. Impedance and ECG data were collected continuously throughout each stressor and recovery period.

RSA was calculated using the MindWare HRV 2.51 Software module (MindWare Technologies, Ltd., Gahanna, OH). R-wave markers in the ECG signal was evaluated for artifacts by visual inspection and by the MAD/MED artifact detection algorithm implemented in the MindWare software (Berntson, Quigley, Jang, & Boysen, 1990). Suspected artifacts were corrected manually (<1% of all R-waves in past work needed correction). This approach accords with current guidelines for frequency domain methods to determine heart rate variability (Berntson et al., 1997; Task Force, 1996). To arrive at minute-by-minute estimates of heart rate and RSA during baseline and tasks, a 60-second time series of inter-beat intervals (IBIs: the time in milliseconds between sequential ECG R spikes) was created from an interpolation algorithm that had a 250-ms sample time. This 60-second IBI time series was (a) linearly-detrended, (b) mean-centered, and (c)

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tapered using a Hamming window. Spectral-power values were determined (in ms^2/Hz) with fast Fourier transformations, and the power values in the 0.15–0.50 Hz spectral bandwidth were integrated (ms^2). These spectral-power values were then natural-log transformed prior to statistical analyses because of distributional violations. The natural-logged spectral-power value in the 0.15–0.50 Hz bandwidth was the indicator of RSA for each experimental epoch. Primary measures of RSA reactivity and recovery were the arithmetic difference in these scores between task/recovery values and baseline values.

The MindWare software package was also used to calculate respiration rate from spectral analysis of the RSP100C respiration signals. These variables were used as control variables to analyze the contribution of respiratory parameters to group differences in RSA reactivity and recovery.

The impedance cardiography values (i.e., PEP) were obtained via ensemble averaging of the dZ/dt waveform for each minute of data collected using MindWare IMP 2.56 software (MindWare Technologies, Ltd., Gahanna, OH). The data were screened for artifact by visual inspection of the ensemble-averaged dZ/dt waveforms.

To calculate reactivity, the RSA and PEP values were first averaged for the three-minute cold, math, prep, and speech tasks. The arithmetic difference between each task segment and the baseline segment was then calculated in order to obtain a reactivity score. Thus, each participant had a RSA and PEP change score (i.e. reactivity score) for each task. To calculate recovery, participants' RSA and PEP values during recovery were compared to their respective baseline data. Once the average RSA and PEP values were calculated for each recovery period, the arithmetic difference from baseline was calculated to obtain a recovery change score following each task.

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Statistical Analyses

For Hypothesis 1, a series of one-way between subjects ANCOVAs were conducted with family history (FH+, FH-) as the between subjects factor and RSA reactivity as the dependent variable. Separate ANCOVAs were run for the math, cold, speech preparation, and speech tasks. Age, BMI, baseline RSA, respiration rate, and ethnicity were entered as covariates. PEP reactivity was analyzed in a similar fashion; however baseline and respiration rate were not included as covariates.

For Hypothesis 2, a series of one-way between subjects ANCOVAs were conducted with family history (FH+, FH-) as the between subjects factor and RSA recovery as the dependent variable. Separate ANCOVAs were run for the math, cold, and speech recovery periods. Age, BMI, reactivity, baseline RSA, respiration rate, and ethnicity were entered as covariates. PEP recovery was analyzed in a similar fashion; however, respiration rate was not used as a covariate.

For Hypothesis 3, a series of one-way between subjects ANCOVAs were conducted with ethnicity (White, Black, Hispanic) as the between subjects factor and RSA reactivity as the dependent variable. Separate ANCOVAs were run for the math, cold, speech preparation, and speech tasks. Age, BMI, baseline RSA and respiration rate were entered as covariates. PEP reactivity was analyzed in a similar fashion; however baseline and respiration rate were not included as covariates.

For Hypothesis 4, a series of one-way between subjects ANCOVAs were conducted with ethnicity (White, Black, Hispanic) as the between subjects factor and RSA recovery as the dependent variable. Separate ANCOVAs were run for the math, cold, and speech recovery periods. Age, BMI, reactivity, baseline RSA and respiration

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rate were entered as covariates. PEP recovery was analyzed in a similar fashion; however, respiration rate was not used as a covariate.

For Hypothesis 5, a series of two-way between subjects ANCOVAs were conducted with family history (FH+, FH-) and ethnicity (White, Black, Hispanic) as the between subjects factors, and RSA reactivity, RSA recovery, PEP reactivity, and PEP recovery as the dependent variables. Separate ANCOVAs were run for reactivity to the math, cold, speech preparation, and speech tasks, and recovery following the math, cold, and speech tasks. For all analyses, age and BMI were included as covariates, with baseline RSA and respiration rate added as additional covariates for RSA analyses. For recovery analyses, reactivity was covaried. When a significant interaction was observed, follow up analyses of the simple effects were conducted.

All omnibus analyses were run using a significance level of .05. For all analyses, the degrees of freedom were adjusted for measures that had incomplete data due to technical problems. Each dependent variable was examined for violations of normality.

Results

Descriptive statistics

Family history. Family history was categorized into the following categories 1) parental history of hypertension (n = 114), 2) parental history of any of the diseases (hypertension, high cholesterol, CVD, stroke, or diabetes) (n = 122), 3) either parental or grandparental history of CVD (MI, CHD; n = 80), 4) either parental or grandparental history of stroke (n = 76), 5) either parental or grandparental history of Type II diabetes (n = 61). These categories were chosen because I felt that they best represented a combination of classifications used in previous research (i.e. parental hypertension,

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family CVD), and classifications that are more novel (i.e. stroke, parents and grandparents with diabetes, broad definition of disease for parental history). Other categories, such as a broad definition of disease for parents and grandparents, were not feasible in the current study because the sample sizes were too small after participants with an "unknown" family history were dropped from the analysis.

For each category, participants were classified as positive family history if any of the responses were "yes". Participants were classified as negative family history if all of the responses were "no". For example, while assessing parental history of any of the diseases, if a participant answered "no" for hypertension, high cholesterol, CVD, and stroke, but "I don't know" for diabetes, they were classified as "unknown" status and consequently dropped from further analyses. This is a conservative method, as participants with an unknown family history are often classified as negative family history (e.g. Wright et al., 2007). A total of n = 22 participants were "unknowns" for parental history of hypertension, n = 14 "unknowns" for parental history of any of the diseases, n = 56 family history of CVD, n = 60 for family history of stroke, and n = 75 for family history of Type II diabetes. See Table 1 for the number of participants in each category as well as the racial/ethnic breakdown of each group.

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Table 1
Family history (FH) groups

FH Group	White	Hispanic	Black	Total
Parent HT				
Yes	20	23	22	66
No	18	14	15	48
Total	38	37	37	114
Unknown				22
Parent Any				
Yes	28	31	28	89
No	12	11	10	33
Total	40	42	38	122
Unknown				14
Family CVD				
Yes	26	17	16	61
No	8	6	5	19
Total	34	23	21	80
Unknown				56
Family Stroke				
Yes	13	12	19	45
No	14	9	7	31
Total	27	21	26	76
Unknown				60
Family Diabetes				
Yes	11	9	16	36
No	12	8	4	25
Total	23	17	20	61
Unknown				75

Demographic and baseline factors. A series of one-way ANOVA's were conducted assessing the differences between black, white, and Hispanic participants on demographic and physical characteristics. The results revealed that the groups did not differ on resting baseline PEP or RSA. There were significant effects of ethnicity on height, weight, and baseline SBP, DBP, and heart rate (HR). Post hoc tests further revealed that Hispanic participants were significantly shorter than white and black participants. Black participants were significantly heavier than white and Hispanic participants. However, there were no significant differences in BMI. Additionally, black

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participants had significantly higher resting baseline SBP, DBP, and HR compared to Hispanic participants, and slightly higher values when compared to white participants. Baseline and demographic characteristics for each racial/ethnic group are presented in Table 2.

Table 2
Means and standard deviations for demographic and baseline variables by ethnicity

Variable	Total	White	Black	Hispanic
Demographic	n = 136	n = 44	n = 44	n = 46
Age (yrs)	20.8 (4.4)	21.0 (3.7)	20.5 (2.9)	20.7 (6.0)
Mother's age (yrs)	47.8 (6.7)	49.7 (5.7)	46.7 (5.9)	47.3 (8.1)
Father's age (yrs)	51.1 (6.7)	52.1 (6.3)	50.0 (6.8)	50.6 (6.3)
Height (in.)	64.6 (2.4)	65.0 (2.2)	65.1 (2.5)	63.7 (2.4)**
Weight (lbs)	142.3 (34.5)	136.9 (23.3)	153.5 (45.0)*	137.6 (30.5)
BMI	23.9 (5.1)	22.8 (4.0)	25.3 (6.5)	23.7 (4.3)
Baseline				
SBP (mmHg)	108.8 (10.2)	108.5 (9.2)	112.1 (12.1)*	106.2 (8.7)
DBP (mmHg)	65.5 (6.7)	65.0 (6.2)	67.7 (7.3)*	63.8 (6.2)
HR (bpm)	75.9 (8.6)	75.1 (9.8)	78.5 (8.4)*	74.2 (7.5)
PEP (ms)	120.9 (14.8)	123.0 (14.3)	118.1 (16.4)	121.8 (13.8)
RSA (ln ms^2)	6.4 (1.2)	6.3 (1.3)	6.4 (1.1)	6.6 (1.2)

*$p < .05$. ** $p < .01$

Baseline and demographic factors were examined for group differences in each family history category using a series of one-way ANOVA's. For parental history of any cardiovascular diseases, participants with a positive parental history had fathers who were significantly older than the fathers of participants with a negative family history. For family history of CVD, participants with a positive family history had significantly higher baseline DBP and PEP values than participants with a negative family history. Additionally, participants with a positive family history of diabetes were significantly older than participants with a negative family history of diabetes. Means, standard deviations, and significance testing information are presented in Tables 3 and 4.

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Table 3
Means and standard deviations for demographic variables by family history

	Parent HT		Parent Any		Family CVD		Family Stroke		Family Diabetes	
Variable	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
Age (yrs)	20.97 (4.07)	21.08 (5.45)	21.14 (4.99)	20.39 (3.12)	21.97 (5.98)	19.63 (1.86)	21.68 (6.36)	20.10 (2.02)	20.92* (3.06)	19.40* (1.85)
Mother's Age (yrs)	48.52 (6.86)	48.15 (6.62)	48.46 (7.30)	47.71 (4.37)	48.98 (7.65)	46.00 (6.02)	47.68 (8.13)	46.83 (5.28)	47.89 (5.05)	46.50 (5.60)
Father's Age (yrs)	52.16 (6.52)	50.02 (5.94)	51.98* (6.53)	49.34* (5.09)	51.33 (7.10)	50.50 (6.31)	50.83 (8.01)	49.29 (5.51)	51.11 (5.52)	48.87 (6.06)
Height (in.)	64.55 (2.36)	65.00 (2.50)	64.49 (2.52)	64.85 (2.33)	64.73 (2.46)	64.07 (2.30)	64.42 (2.15)	64.56 (2.55)	64.37 (2.85)	64.39 (2.37)
Weight (lbs)	143.4 (29.3)	145.2 (43.3)	142.0 (34.5)	143.1 (38.1)	141.5 (38.9)	142.8 (35.5)	144.5 (37.7)	147.7 (42.5)	149.4 (45.8)	137.8 (35.5)
BMI	24.15 (4.55)	23.96 (6.09)	23.87 (4.91)	23.83 (5.77)	23.58 (5.40)	24.40 (5.74)	24.38 (5.71)	24.70 (5.92)	25.07 (6.13)	23.28 (5.44)

*p<.05.

Table 4
Means and standard deviations for baseline variables by family history

	Parent HT		Parent Any		Family CVD		Family Stroke		Family Diabetes	
Variable	Yes	No	Yes	No	Yes	No	Yes	No	Yes	No
SBP (mmHg)	109.0 (9.3)	108.1 (11.8)	109.2 (10.6)	107.0 (9.5)	109.8 (11.1)	106.3 (9.5)	110.6 (11.5)	108.2 (11.4)	109.6 (13.0)	105.7 (8.5)
DBP (mmHg)	65.4 (6.4)	65.2 (7.0)	65.9 (7.0)	64.4 (5.4)	66.6 (7.3)	62.6 (6.1)	67.1 (6.1)	65.0 (8.0)	66.0 (8.4)	63.1 (6.0)
HR (bpm)	75.8 (7.9)	75.7 (9.8)	76.2 (8.3)	74.1 (9.7)	75.6 (8.9)	77.2 (8.8)	76.8 (8.5)	77.2 (9.1)	76.4 (9.1)	76.9 (8.9)
PEP (ms)	118.2 (13.4)	123.6 (16.1)	120.3 (14.6)	122.5 (14.7)	124.1* (15.5)	115.4* (14.1)	120.9 (12.7)	119.2 (16.8)	120.7 (13.7)	118.8 (16.7)
RSA	6.46 (1.16)	6.36 (1.25)	6.37 (1.22)	6.54 (1.12)	6.17 (1.23)	6.59 (0.83)	6.47 (1.23)	6.27 (0.90)	6.75 (0.94)	6.26 (1.07)

*p<.05 for yes/no comparison.

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Reactivity and Recovery Patterns

A series of paired t-tests were conducted in order to test for significant changes in PEP, RSA, and HR from baseline both during each of the stress tasks and throughout each of the recovery periods. See Table C1 in appendix for means and significance testing. As can be seen in Figures 1-3, the tasks were effective in evoking significant autonomic responses. During the speech preparation and speech delivery tasks, participants exhibited sympathetic activation and parasympathetic withdrawal, as seen by significant decreases in RSA and PEP, as well as significant increases in HR. During the math task, there was significant sympathetic excitation, as indicated by decreases in PEP and increases in HR; however, RSA did not differ from baseline. During the cold task, both parasympathetic and sympathetic activation occurred, reflected by significant RSA increases, and significant decreases in PEP and HR. It is important to note that *decreases* in PEP (i.e. heart contractility) indicate *greater* sympathetic activation.

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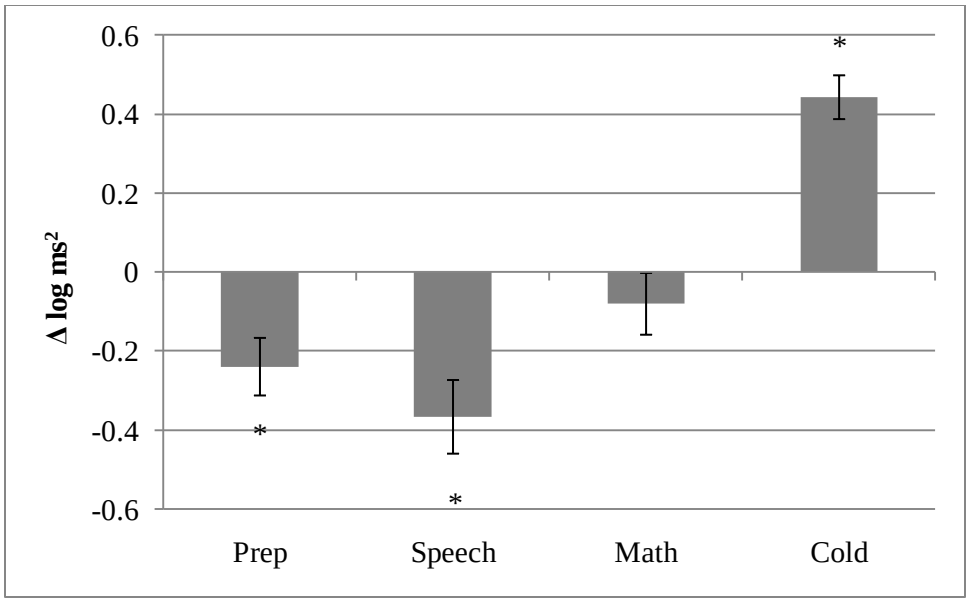

Figure 1. RSA Reactivity Scores by Task. *p<.05.

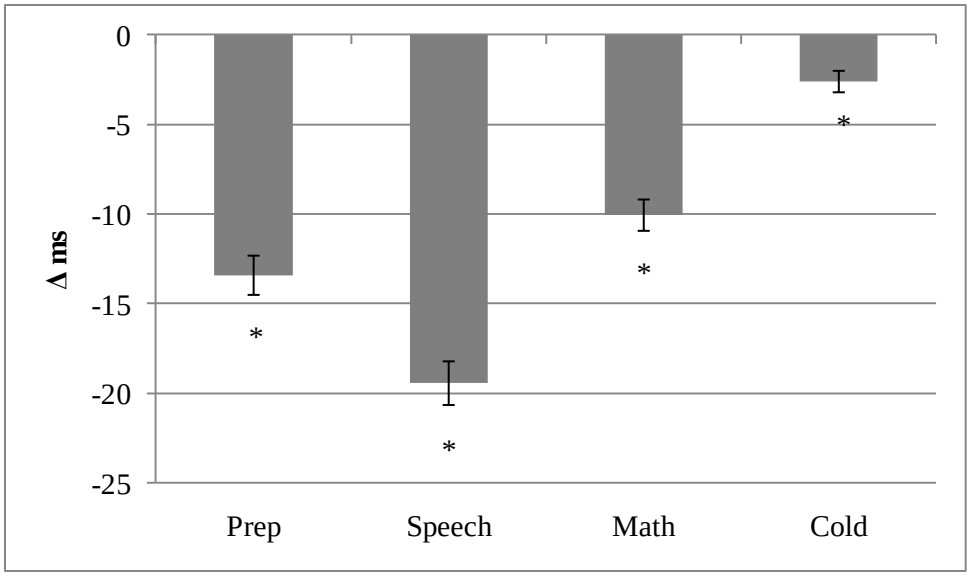

Figure 2. PEP Reactivity Scores by Task. *p<.05.

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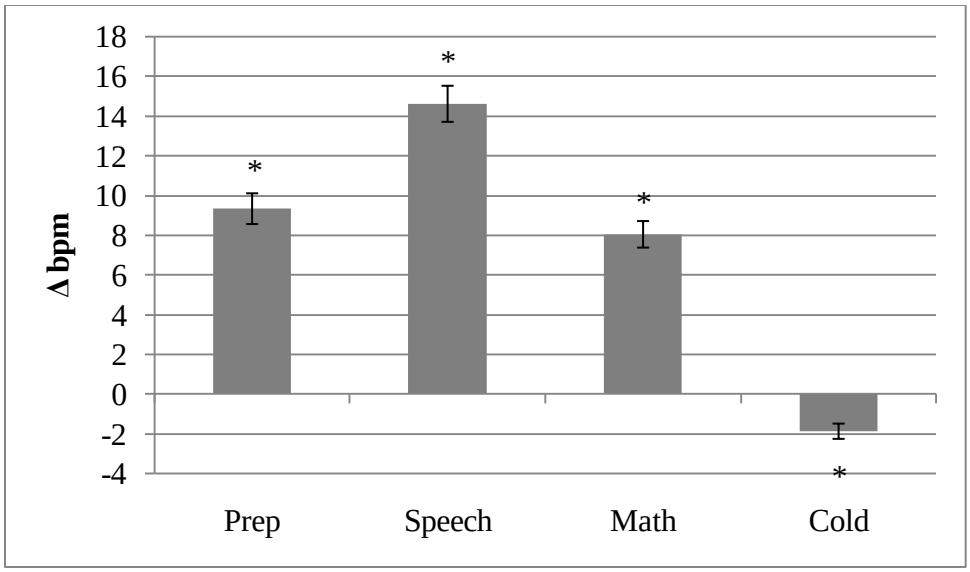

Figure 3. HR Reactivity Scores by Task. *p<.05.

There were also significant differences from baseline in RSA, PEP, and HR during the recovery periods, as seen in Figures 4-6. During the speech and math recovery periods participants exhibited parasympathetic and sympathetic activity greater than in their resting state, as RSA remained significantly elevated and PEP remained significantly decreased, while HR had returned to baseline. During the cold recovery period, a similar pattern was seen, with greater sympathetic and parasympathetic activity compared to baseline, as demonstrated by RSA that remained significantly elevated from baseline, both PEP, and HR that remained significantly decreased.

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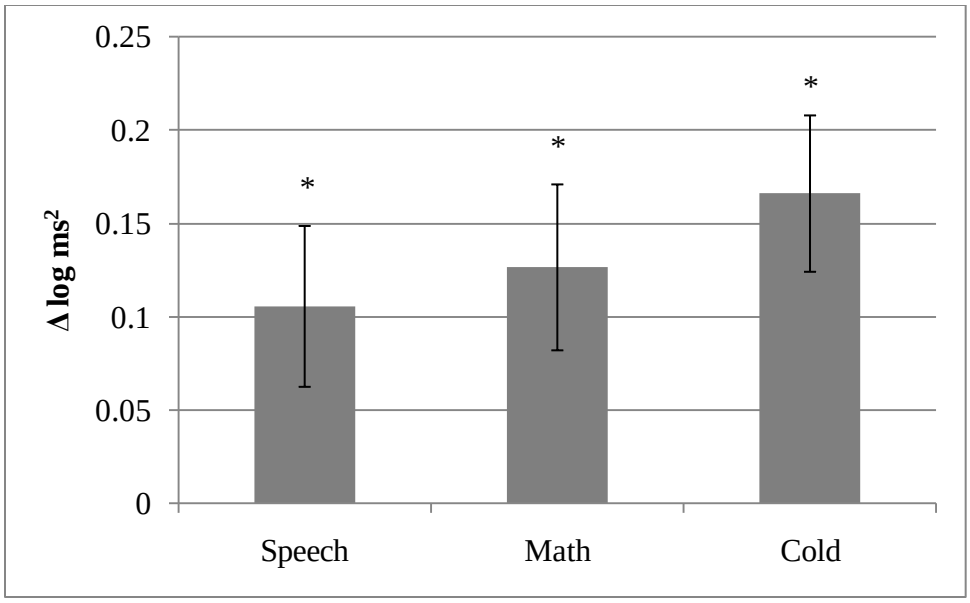

Figure 4. RSA Recovery Scores by Task. *p<.05.

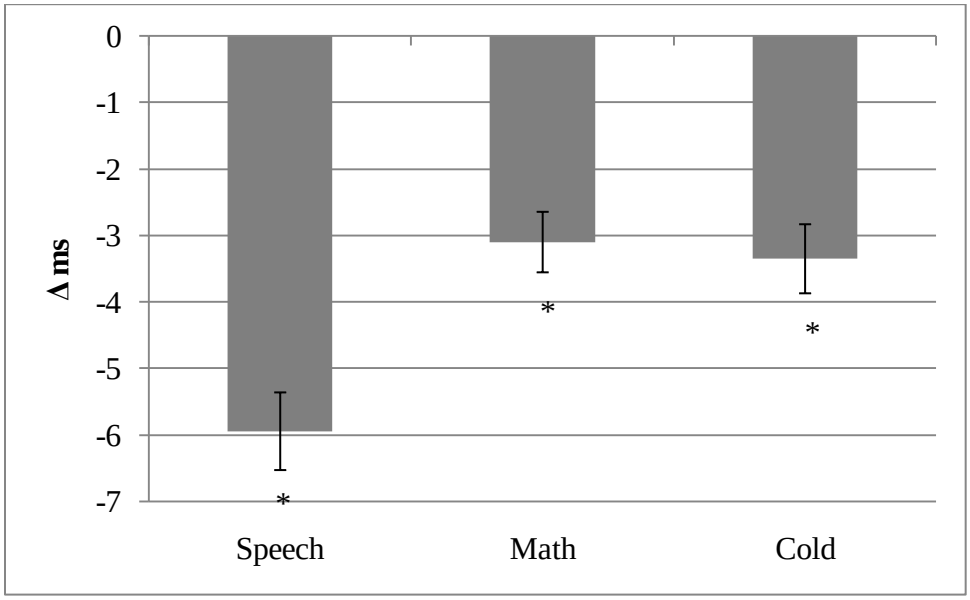

Figure 5. PEP Recovery Scores by Task. *p<.05.

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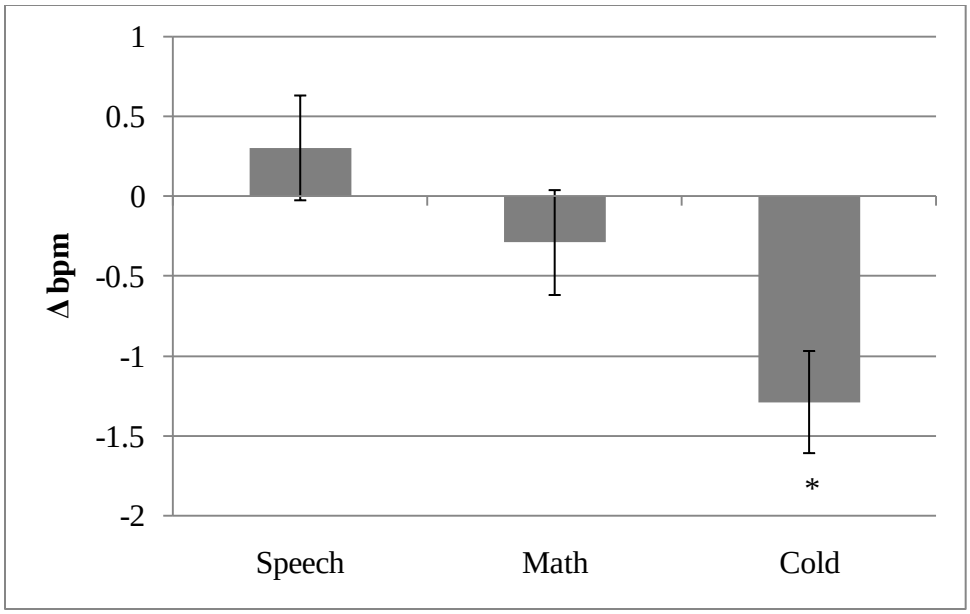

Figure 6. HR Recovery Scores by Task. *p<.05.

Hypothesis 1: Family History on RSA and PEP Reactivity

I hypothesized that participants with a family history of CVD would have greater RSA and PEP reactivity to stress tasks than participants with a negative family history. In order to determine the effect of a positive family history of CVD on cardiovascular reactivity to the stress tasks, one-way ANCOVAs with family history as the between subjects factor and each physiological reactivity score as the dependent variable were conducted. For all PEP analyses, age, BMI, and ethnicity were entered as covariates. For all RSA analyses, age, BMI, ethnicity, respiration, and baseline RSA values were entered as covariates.

Parental history of hypertension. There was no effect of parental hypertension on RSA reactivity for the math, prep, or speech tasks (p's > .10). For the cold task, the main effect of parental history of hypertension was significant, $F(1, 102) = 5.41$, $p = .022$. Participants with a positive family history had greater increases in RSA ($M = 0.540$, $SE = 0.072$) than participants with a negative family history ($M = 0.280$, $SE = 0.084$).

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There was no effect of parental hypertension on PEP reactivity for any of the tasks (*p*'s > .10).

Parental history of any of the cardiovascular diseases. There was no effect of parental history of disease on RSA reactivity for the math, prep, or speech tasks (*p's* > .10). For the cold task, the main effect of parental history of disease was significant, $F(1, 110) = 6.72$, $p = .011$. Participants with a positive family history had greater increases in RSA ($M = 0.512$, $SE = 0.061$) than participants with a negative family history ($M = 0.210$, $SE = 0.099$). There was no effect of parental history of disease on PEP reactivity for any of the tasks (*p's* > .10).

Family history of CVD. There were no significant effects of family history of cardiovascular disease on RSA or PEP reactivity to any of the tasks (*p's* > .10).

Family history of stroke. There was no effect of family history of stroke on RSA or PEP reactivity to the cold, math or prep tasks (*p's* > .10). For the speech task, the main effect of family history of stroke on RSA reactivity was significant, $F(1, 64) = 9.50$, $p = .003$. Participants with a positive family history of stroke had greater decreases in RSA ($M = -0.677$, $SE = 0.129$) than participants with a negative family history ($M = -0.033$, $SE = 0.157$). Similarly, the main effect of family history of stroke on PEP reactivity was significant for the speech task, $F(1, 63) = 5.41$, $p = .023$. Participants with a positive family history had greater decreases in PEP ($M = -22.42$, $SE = 2.22$) than participants with a negative family history ($M = -14.15$, $SE = 2.68$).

Family history of Type II diabetes. There was no effect of family history of Type II diabetes on RSA reactivity to the cold task ($p > .10$). The effect of family history of Type II diabetes on RSA cold task reactivity showed trends for the math, prep, and

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speech tasks. For the math task, participants with a positive family history had greater decreases in RSA (M = -0.363, SE = 0.155) than participants with a negative family history (M = 0.127, SE = 0.192), $F(1, 52) = 3.43$, $p = .070$. For the prep task, participants with a positive family history had greater decreases in RSA (M = -0.442, SE = 0.131) than participants with a negative family history (M = -0.015, SE = 0.162), $F(1, 52) = 3.76$, $p = .058$. Similar trends were seen for the speech task, where participants with a positive family history had greater decreases in RSA (M = -0.603, SE = 0.159) than participants with a negative family history (M = -0.101, SE = 0.196), $F(1, 52) = 3.51$, $p = .067$.

There was no effect of family history of Type II diabetes on PEP reactivity for the math task ($p > .10$). The effect of family history of Type II diabetes on PEP reactivity showed trends for the prep, speech, and cold tasks. For the prep task, participants with a positive family history had greater decreases in PEP (M = -16.67, SE = 2.23) than participants with a negative family history (M = -10.21, SE = 2.61), $F(1, 51) = 3.24$, $p = .078$. For the speech task, participants with a positive family history had greater decreases in PEP (M = -23.58, SE = 2.66) than participants with a negative family history (M = -15.51, SE = 3.16), $F(1, 52) = 3.55$, $p = .065$. Following the same trend, during the cold task participants with a positive family history had greater decreases in PEP (M = -3.05, SE = 1.10) than participants with a negative family history (M = -0.02, SE = 1.29), $F(1, 51) = 2.93$, $p = .093$.

Hypothesis 2: Family History on RSA and PEP Recovery

I hypothesized that participants with a family history of cardiovascular disease would have impaired recovery following stress tasks than participants with a negative

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family history. In order to determine the effect of a positive family history of CVD on RSA and PEP recovery following the stress tasks, one-way ANCOVAs with family history as the between subjects factor and each physiological recovery score as the dependent variable were conducted. For all PEP analyses, PEP reactivity, age, BMI, and ethnicity were entered as covariates. For all RSA analyses, RSA reactivity, baseline RSA, age, BMI, ethnicity, respiration, and baseline RSA values were entered as covariates.

For a family history of stroke, there was a trend for group differences on PEP recovery following the cold task, $F(1, 62) = 3.29$, $p = .075$. Participants with a positive family history had smaller PEP decreases ($M = -2.40$, $SE = 0.64$) than participants with a negative family history ($M = -4.31$, $SE = 0.80$). There were no other significant effects of family history on PEP or RSA recovery for any of the tasks (*p's* > .10).

Hypothesis 3: Ethnicity on RSA and PEP Reactivity

I hypothesized that black and Hispanic participants would exhibit greater reactivity compared to European American participants. In order to determine the effect of ethnicity on RSA and PEP reactivity to the stress tasks, one-way ANCOVAs with ethnicity as the between subjects factor and each physiological reactivity score as the dependent variable were conducted. For all PEP analyses, age and BMI were entered as covariates. For all RSA analyses, age, BMI, respiration, and baseline RSA values were entered as covariates.

There was no effect of ethnicity on RSA or PEP reactivity for any of the tasks (*p's* > .10).

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Hypothesis 4: Ethnicity on RSA and PEP Recovery

I hypothesized that black and Hispanic participants would exhibit impaired recovery compared to European American participants. In order to determine the effect of ethnicity on RSA and PEP recovery following the stress tasks, one-way ANCOVAs with ethnicity as the between subjects factor and each physiological recovery score as the dependent variable were conducted. For all PEP analyses, PEP reactivity, age, and BMI were entered as covariates. For all RSA analyses, RSA reactivity, baseline RSA, age, BMI, respiration, and baseline RSA values were entered as covariates.

There were no significant effects of ethnicity on RSA recovery for the math or cold tasks (*p's* > .10). However, the effect of ethnicity on RSA recovery was significant following the speech task, $F(2, 120) = 3.45$, $p = .035$. A Tukey's HSD revealed that RSA changes were smaller in black participants compared to white ($p = .013$) and Hispanic participants ($p = .053$), see Figure 7. There were no significant effects of ethnicity on PEP recovery for any of the tasks (*p's* > .10).

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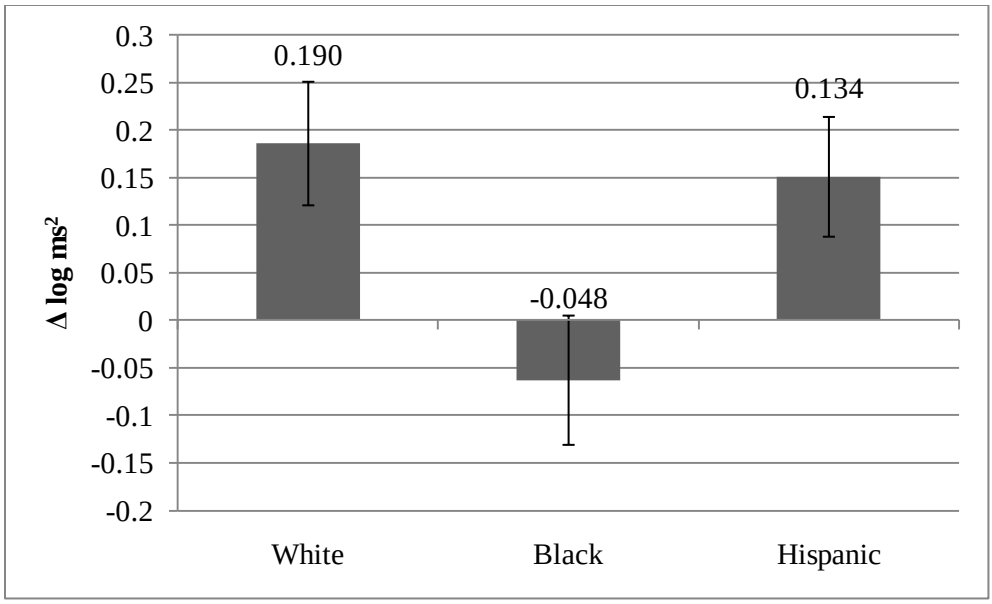

Figure 7. RSA Recovery after Speech Task as a Function of Ethnicity

Hypothesis 5a: Family History and Ethnicity on RSA and PEP Reactivity

I did not hypothesize specific effects, as research in this area is limited. In order to explore the interactions between family history and ethnicity, separate 2x3 ANCOVAs were run for each task with family history and ethnicity as the between subjects factors and each physiological reactivity score as the dependent variable. For all PEP analyses, age and BMI were entered as covariates. For all RSA analyses, age, BMI, respiration, and baseline RSA values were entered as covariates.

Parental history of hypertension. For the cold task, the main effect of parental history of hypertension on RSA reactivity was significant, $F(1, 99) = 6.59$, $p = .012$. Those with hypertensive parents had greater reactivity ($M = 0.539$, $SE = 0.071$) than those with normotensive parents ($M = 0.253$, $SE = 0.084$). There was a trend for the main effect of ethnicity, $F(2, 99) = 2.38$, $p = .098$. Black participants ($M = 0.219$, $SE = 0.099$) had lower RSA reactivity than white ($M = 0.487$, SE = 0.094, $p = .059$) and Hispanic ($M = 0.482$, $SE = 0.097$, $p = .061$) participants. The interaction between family history and

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ethnicity showed a trend, $F(2, 99) = 2.47$, $p = .09$. There was a significant simple effect of family history for black participants, $F(1, 99) = 4.75$, $p = .032$. Those with a positive family history had significantly greater reactivity than those with a negative family history. Similarly, there was a significant simple effect of family history for Hispanic participants, $F(1, 99) = 6.18$, $p = .015$. Those with a positive family history had significantly higher reactivity than those with a negative family history (see Figure 8). There were no significant effects of parental history of hypertension or ethnicity on PEP reactivity to any of the tasks, *p's* > .10.

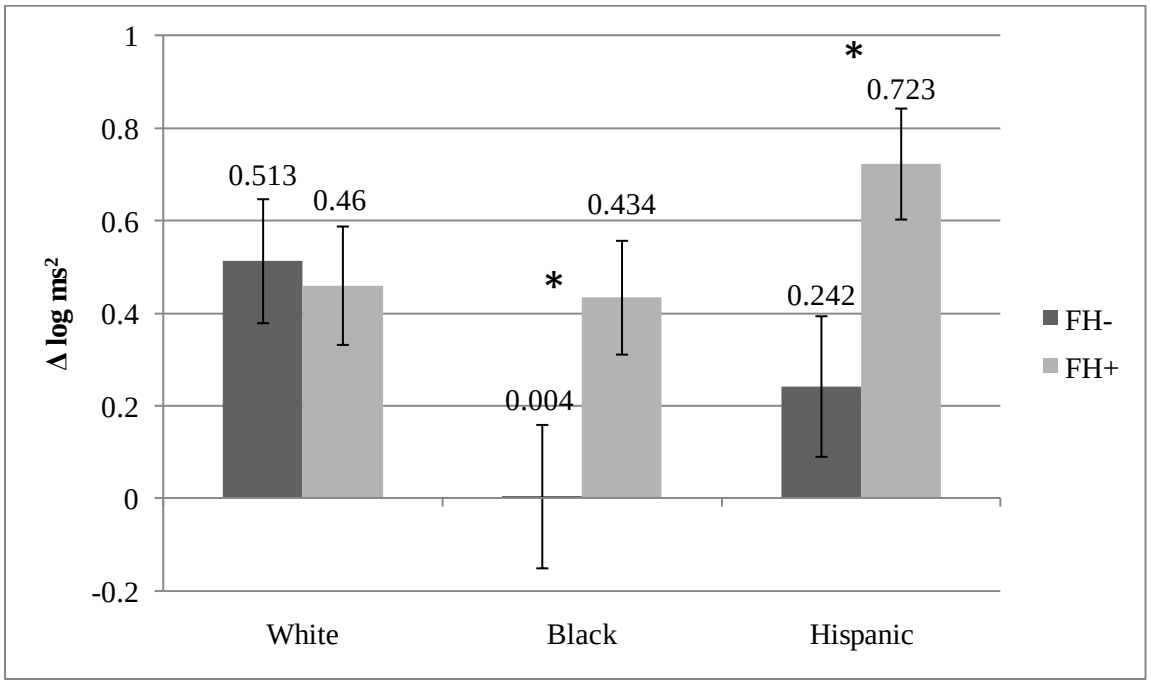

Figure 8. RSA Reactivity to the Cold Pressor Task as a Function of Parental History of Hypertension and Ethnicity, $p < .05$ for FH+/- comparison.

Parental history of any of the cardiovascular diseases. For the cold task, there was a significant main effect of family history, $F(1, 107) = 8.00$, $p = .006$. Participants with a positive parental history of any of the diseases had greater reactivity ($M = 0.507$, $SE = 0.060$) than those with disease-free parents ($M = 0.180$, $SE = 0.098$). There was also

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a significant main effect of ethnicity on RSA reactivity, $F(2, 107) = 3.64$, p = .029. Black participants had significantly lower reactivity ($M = 0.114$, $SE = 0.109$) than Hispanic ($M = .409$, $SE = 0.098$, $p = .046$) and white participants ($M = 0.506$, $SE = 0.098$, $p = .01$). The interaction showed a trend, $F(2, 107) = 2.60$, $p = .079$, see Figure 9. There were significant simple effects of family history for black participants, $F(1, 107) = 5.27$, $p = .024$. Black participants with a family history had significantly greater RSA reactivity than those without a family history. Similarly, there were significant simple effects of family history for Hispanic participants, $F(1, 107) = 7.07$, $p = .009$. Hispanic participants with a family history had significantly greater RSA reactivity than those without a family history. There were no significant effects of parental history of disease or ethnicity on PEP reactivity to any of the tasks, p's $> .10$.

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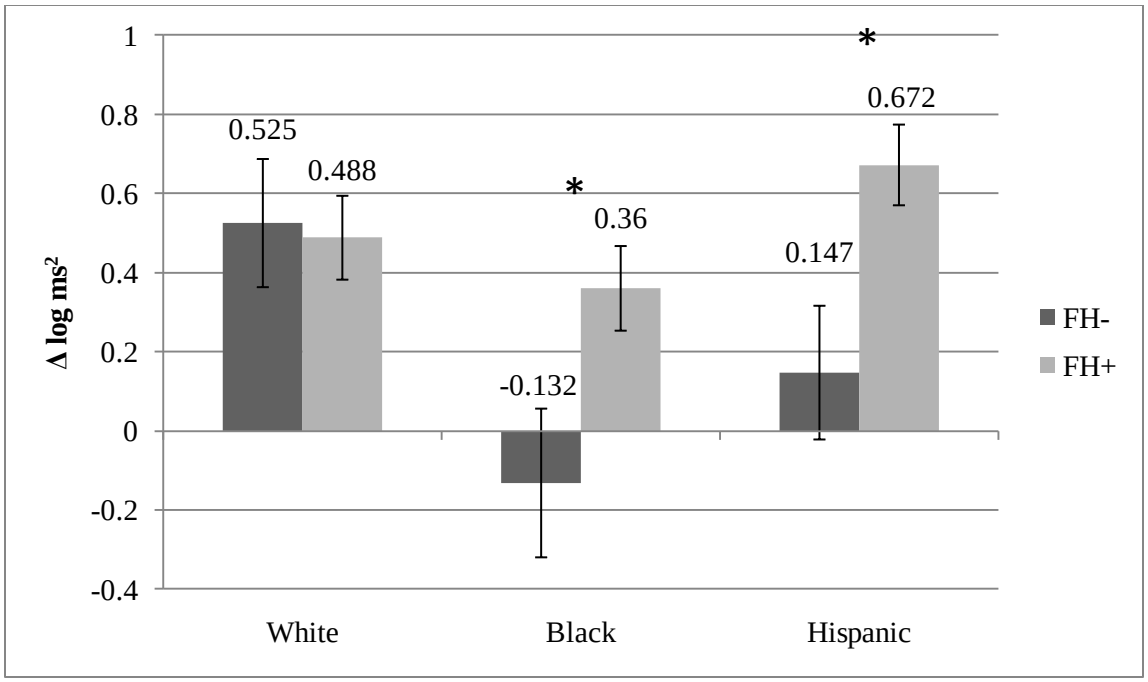

Figure 9. RSA Reactivity to the Cold Pressor Task as a Function of Parental History of Hypertension, High Cholesterol, CVD, or Diabetes and Ethnicity, $p < .05$ for FH+/- comparison.

Family history of CVD. There were no significant effects of family history of CVD or ethnicity on RSA or PEP reactivity to any of the tasks, *p's* > .10.

Family history of stroke. For the speech task, there was a significant main effect of family history of stroke on RSA reactivity, $F(1, 61) = 10.56$, $p = .002$. Participants with a family history of stroke had greater decreases in RSA ($M = -0.685$, $SE = 0.134$) than people without a family history ($M = 0.024$, $SE = 0.167$). Ethnicity was not a significant main effect. Further, there was a significant main effect of family history of stroke on PEP reactivity during the speech task, $F(1, 60) = 8.26$, $p = .006$. Participants with a family history of stroke had greater decreases in PEP ($M = -23.14$, $SE = 2.24$) than people without a family history ($M = -12.77$, $SE = 2.74$). There was also a significant main effect for ethnicity, $F(2, 60) = 3.14$, $p = .05$. Black participants had significantly smaller decreases in PEP ($M = -11.26$, $SE = 3.40$) than white participants ($M = -22.48$, *SE*

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= 2.82, p = .017) and slightly smaller decreases than Hispanic participants (M = -20.13, SE = 3.06, p = .062).

Family history of Type II diabetes. For the math, prep, and speech tasks, there was a trend for the main effect of family history of diabetes on RSA reactivity. For the math task, participants with a family history of Type II diabetes had greater decreases in RSA (M = -0.351, SE = 0.158) than people without a family history (M = 0.190, SE = 0.206), $F(1, 49) = 3.93$, p = .053. Similarly, participants with a family history of Type II diabetes had greater decreases in RSA (M = -0.451, SE = 0.133) than people without a family history (M = -0.006, SE = 0.172) for the prep task, $F(1, 49) = 3.91$, p = .054. Following the same trend, for the speech task participants with a family history of Type II diabetes had greater decreases in RSA (M = -0.601, SE = 0.164) than people without a family history (M = -0.100, SE = 0.215), $F(1, 49) = 3.17$, p = .081.

The main effect for family history of diabetes on PEP reactivity showed a trend for the prep task, $F(1, 48) = 3.60$, p = .064. Participants with a family history of diabetes had greater decreases in PEP (M = -16.60, SE = 2.28) than people without a family history (M = -9.62, SE = 2.78). Further, there was a significant main effect for family history of diabetes on PEP reactivity for the speech task, $F(1, 49) = 4.71$, p =.035. Participants with a family history of diabetes had greater decreases in PEP (M = -23.40, SE = 2.68) than people without a family history (M = -13.92, SE = 3.55).

Hypothesis 5b: Family History and Ethnicity on RSA and PEP Recovery

I did not hypothesize specific effects, as research in this area is limited. In order to explore the interactions between family history and ethnicity, separate 2x3 ANCOVAs were run for each task with family history and ethnicity as the between subjects factors

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and each physiological recovery score as the dependent variable. For all PEP analyses, PEP reactivity, age and BMI were entered as covariates. For all RSA analyses, RSA reactivity, age, BMI, respiration, and baseline RSA values were entered as covariates.

Parent history of hypertension. Following the speech task, there was a significant main effect of ethnicity on RSA recovery, $F(2, 95) = 3.60$, $p = .031$. A Tukey's HSD indicated that black participants ($M = -0.085$, $SE = 0.074$) had significantly lower RSA recovery values than white ($M = 0.182$, $SE = 0.068$, $p = .01$) and Hispanic ($M = 0.112$, $SE = 0.07$, $p = .06$) participants.

Parent history of any of the cardiovascular diseases. There was a significant effect of ethnicity on RSA recovery after the speech task, $F(2, 103) = 3.66$, $p = .029$. Tukey's HSD test further revealed that black participants ($M = -0.088$, $SE = 0.085$) had significantly lower RSA values than white participants ($M = 0.215$, $SE = 0.072$, $p = .008$) and slightly lower than Hispanic participants ($M = 0.118$, $SE = .073$, $p = .071$).

Family history of CVD. Following the cold task, the main effect of ethnicity on RSA recovery showed a trend, $F(2, 65) = 2.58$, $p = .084$. Black participants had the highest RSA values ($M = 0.392$, $SE = 0.102$) followed by Whites ($M = 0.125$, $SE = 0.079$) and Hispanics ($M = 0.099$, $SE = 0.092$). Following the speech task, the main effect of ethnicity on RSA recovery showed a trend, $F(2, 64) = 2.57$, $p = .085$. White participants ($M = 0.274$, $SE = 0.09$) had higher RSA values than black participants ($M = -0.026$, $SE = 0.119$, $p = .051$) and Hispanic participants ($M = 0.037$, $SE = 0.106$, $p = .089$). There was a significant effect for the ethnicity by family history interaction, $F(2, 64) = 3.74$, $p = .029$. The simple effect of family history showed a trend for white participants, $F(1, 64) = 3.46$, $p = .068$. Those with a negative family history had higher RSA values

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than those with a positive family history. The simple effect of family history also showed a trend for Hispanic participants, $F(1, 64) = 3.80$, $p = .056$. However, the opposite pattern was seen, where those with a positive family history had higher RSA values than those with a negative family history, see Figure 10.

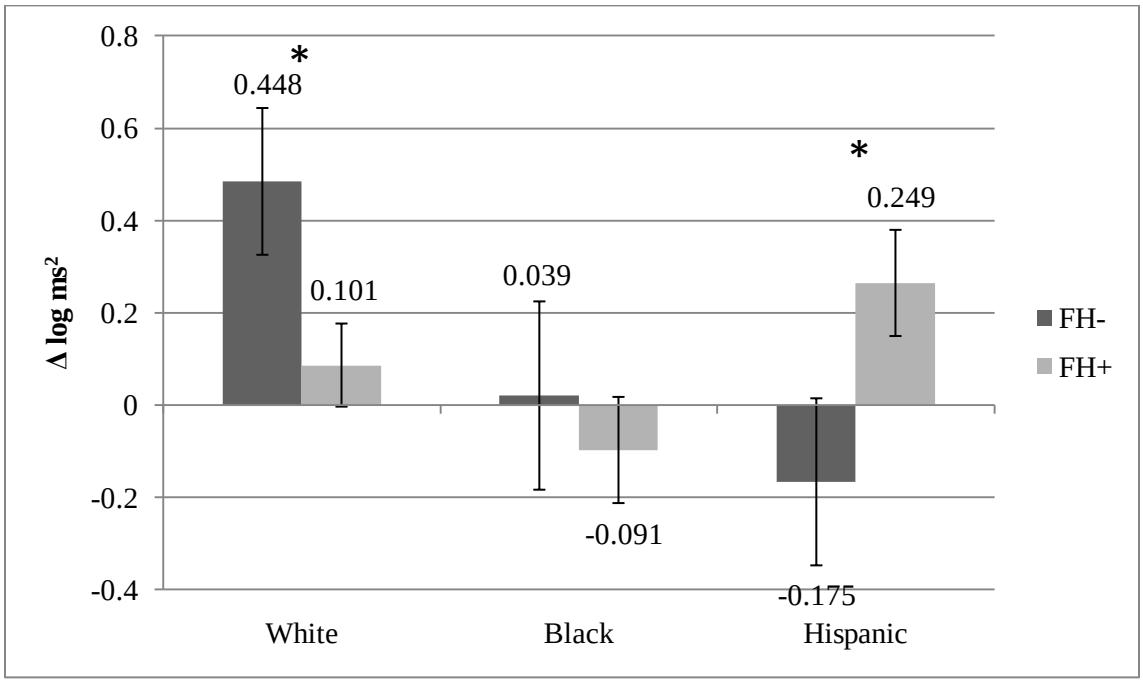

Figure 10. RSA Recovery after the Speech Task as a Function of Family History of CVD and Ethnicity, $p < .1$ for FH+/- comparison

Family history of stroke. Following the math task, there was a main effect of family history on RSA recovery, $F(1, 60) = 4.05$, $p = .049$. Participants with a positive family history had significantly higher RSA ($M = 0.189$, $SE = 0.065$) than participants with a negative family history ($M = -0.028$, $SE = 0.083$). There was a trend for the main effect of ethnicity, $F(2, 60) = 2.72$, $p = .074$. White participants had higher RSA ($M = 0.236$, $SE = 0.081$), than Hispanic ($M = 0.083$, $SE = 0.093$) and black participants ($M = -0.077$, $SE = 0.103$). The ethnicity by family history interaction on RSA recovery was significant, $F(2, 60) = 3.61$, $p = .033$. The simple effect of family history was significant

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for black participants, $F(1, 60) = 9.19$, $p = .004$. Black participants with a negative family history had greater decreases in RSA compared to participants with a positive family history.

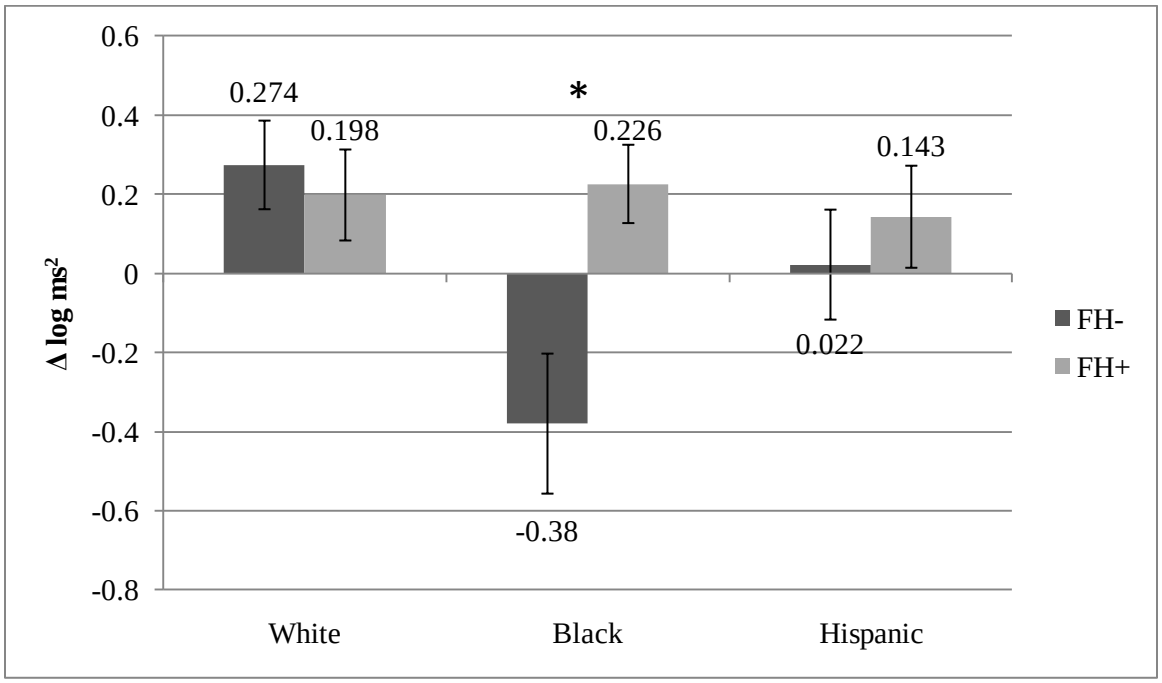

Figure 11. RSA Recovery after the Speech Task as a Function of Family History of Stroke and Ethnicity, $p < .05$ for FH+/- comparison

Following the speech task, the main effect of ethnicity on RSA recovery showed a trend, $F(2, 58) = 2.92$, $p = .062$. White participants ($M = 0.259$, $SE = 0.088$) had significantly higher RSA values than black participants ($M = -0.109$, $SE = 0.120$, $p = .02$), but were not statistically different from Hispanic participants ($M = 0.102$, $SE = 0.099$, $p = .239$). Additionally, there was a trend for the main effect of family history on PEP recovery following the cold task, $F(1, 59) = 3.32$, $p = .073$. Participants with a negative family history had greater decreases in PEP ($M = -4.44$, $SE = 0.85$) than participants with a positive family history ($M = -2.43$, $SE = 0.67$).

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Family history of Type II diabetes. Following the speech task, the main effect of ethnicity on RSA recovery showed a trend, $F(2, 47) = 3.07$, $p = .056$. Black participants ($M = -0.255$, $SE = 0.128$) had significantly lower RSA values than white participants ($M = 0.146$, $SE = 0.099$, $p = .017$), and slightly lower than Hispanic participants ($M = 0.017$, $SE = 0.110$, $p = .115$).

Discussion

Previous research has shown that exaggerated reactivity and delayed recovery is predictive of the future development of cardiovascular-related diseases. Further, people who have a heightened risk for the development of cardiovascular diseases have shown greater reactivity and impaired recovery to stressful tasks. The primary purpose of the present study was to examine the associations between ethnicity, a family history of cardiovascular and related diseases, and autonomic reactivity and recovery to three stress tasks.

In general, the results observed supported the hypothesis that a positive family history was related to greater reactivity; however, the hypothesis that a positive family history would lead to impaired recovery was only partially supported. The results indicated that ethnicity was related to reactivity and recovery, but in the opposite direction than was expected. The following discussion will evaluate the findings of the present study with regard to the specific research hypotheses, as well as the study's limitations, implications, and directions for future research.

Baseline and Demographic Factors

There were no significant differences in baseline measures of SBP, DBP, HR, PEP, or RSA between participants with a positive and negative parental history of

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hypertension, parental history of any cardiovascular or related diseases, family history of stroke, or family history of Type II diabetes. There were significant differences between resting levels of PEP between participants with a positive and negative family history of CVD, however the effect was opposite of what we would expect. That is, participants with a *negative* family history of CVD had higher sympathetic activation of the heart at rest. On the other hand, participants with a positive family history of CVD exhibited higher resting DBP levels. This may indicate a larger afterload (i.e. higher pressure), which would result in a slowing of PEP. Previous studies have found that participants with a positive parental history of hypertension exhibit a combination of increased sympathetic activity (e.g. SBP) and decreased parasympathetic activity (e.g. HF-HRV) at rest (Maver, Strucl, & Accetto, 2004). However, these differences may be explained through sample characteristics, as Maver, Strucl, and Accetto (2004) used a sample with a mean age of 28.6 years, while the current study had a mean age of 20.8 years.

Evaluation of Specific Aims

Family history and reactivity. One aim of the current study was to examine the affect of a positive family history of cardiovascular disease on autonomic reactivity to psychological stress. I hypothesized that participants with a positive family history would exhibit an exaggerated response to the stress tasks compared to participants with a negative family history. This was expected as an increase in parasympathetic activity during the cold task, and a decrease in parasympathetic activity combined with an increase in sympathetic activity during the math and speech tasks.

Parental history of hypertension/any of the diseases. The results were practically identical between these two categories of family history, so they will be presented

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together. As hypothesized, a positive parental history of hypertension or other cardiovascular diseases was related to greater parasympathetic reactivity. This was evidenced by greater RSA increases during the cold task among participants with a positive parental history. However, this effect was modified by ethnicity, as it was only observed in black and Hispanic participants. In this study, there was no effect of parental history of hypertension or other related diseases on autonomic reactivity for white participants. This finding contradicts previous studies conducted on Whites. For example, Miller (1994) examined RSA reactivity in participants with and without a parental history of hypertension using four stress tasks, including the cold pressor task. He found no family history differences in RSA across any of the tasks. Wright, O'Donnell, Brydon, Wardle, and Steptoe (2007) also reported no effects of family history of hypertension on DBP, HR, or HRV reactivity to a Stroop color word or speech task.

Additionally, there were no significant effects of parental history of disease on sympathetic reactivity to any of the tasks. This was surprising, as previous studies have generally reported greater reactivity in measures linked to sympathetic activation in offspring of hypertensives. For example, Frazer, Larkin, and Goodie (2002) reported that undergraduate males and females with a parental history of hypertension had greater SBP reactivity to mental arithmetic, mirror tracing, and role-playing tasks compared to participants with a negative family history. One study using a sample of 11-14 year old males found greater DBP reactivity to a cold pressor (Musante, Treiber, Strong & Levy, 1990). Another study found a significant effect of parental history of hypertension on HR and DBP reactivity to a mental arithmetic task (Manuck, Proietti, Rader, & Polefrone, 1985). A meta-analysis conducted by Fredrikson and Matthews (1990) reported that

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normotensive participants with hypertensive parents demonstrated greater SBP and HR responses to active- (e.g. speech, mental arithmetic) and passive-(e.g. cold pressor) coping stressors, as well as greater DBP responses to passive-coping stressors. However, they also reported that studies on offspring that were older (over 20 years) had more significant and more reliable increases in reactivity (Fredrikson & Matthews, 1990). The current sample had a mean age (M = 20.8) right around their cut-off for "older" vs. "younger" participants, which may explain some of the differences in our findings.

Family history of cardiovascular disease. Contrary to expectations, there was no significant effect of family history of cardiovascular disease on parasympathetic or sympathetic reactivity to any of the tasks. This was unexpected, as previous studies have shown a relationship between reactivity and family history of CVD. One study measured autonomic reactivity to a mental arithmetic and an anger induction task in participants with a family history of CVD (Nelson, Franks, Brose, Raven, Williamson, Shi, McGill, & Harrell, 2005). They found that participants with a positive family history had less parasympathetic activity during the anger induction task, but the effect was only significant in participants who were also high on trait hostility (Nelson et al., 2005). Wright, O'Donnell, Brydon, Wardle, and Steptoe (2007) found that participants with a positive family history of CVD had greater DBP reactivity to a Stroop task, and that women with a positive family history had greater parasympathetic withdrawals during Stroop and speech tasks. Nelson and colleagues (2005) proposed that parasympathetic variation is the primary factor that accounts for the differences seen between the positive and negative family history of CVD groups. However, Nelson et al. (2005) and Wright et

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al. (2007) failed to control for respiration when examining RSA reactivity, as recommended by Bernston et al. (1997).

Family history of stroke. A positive family history of stroke was related to significantly greater parasympathetic withdrawal coupled with greater sympathetic activation to the speech task, as demonstrated by greater RSA and PEP decreases in the positive family history group. To my knowledge, no other studies have been conducted examining autonomic reactivity to an acute stressor in participants with a family history of stroke. The results here clearly demonstrate exaggerated reactivity in both the sympathetic and parasympathetic nervous systems. It is interesting, as the degree of family history is strongly related to the future incidence of stroke (Williams et al., 2001), and the risk factors for stroke are very similar to other cardiovascular diseases, such as age, African-American ethnicity, and hypertension (Sturgeon, Folsom, Longstreth, Shahar, Rosamund, & Cushman, 2007).

Family history of Type II diabetes. A positive family history of Type II diabetes was related to exaggerated reactivity. This was evidenced by greater parasympathetic withdrawal to the math task, and greater parasympathetic withdrawal coupled with greater sympathetic activation to the prep and speech tasks. There was also a trend for greater sympathetic activity during the cold pressor. These would support my hypothesis; however, these effects were only of borderline significance. Altered autonomic control of the heart and exaggerated autonomic reactivity are commonly reported in offspring of Type II diabetics. One study reported that participants with a positive family history of Type II diabetes were more likely to have autonomic neuropathy (i.e., decreased parasympathetic control) compared to participants without a family history, even after

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adjusting for known confounding and risk factors such as age, SBP, DBP, BMI, cholesterol, glucose, and insulin (Foss, Vestbo, Froland, Gjessing, Mogensen, & Damsgaard, 2001). Greater sympathetic activation has also been reported among non-diabetic offspring of Type II diabetics, and sympathetic activity was correlated with plasma insulin and insulin resistance but only in participants with a positive family history of diabetes (Huggett, Hogarth, Mackintosh, & Mary, 2006). Further, another study reported that participants with a parental history of Type II diabetes combined with insulin resistance had blunted RSA reactivity to a cold pressor task, while offspring who had normal insulin sensitivity had increases in RSA during the task (Lindmark, Wiklund, Bjerle, & Eriksson, 2003).

Ethnicity and reactivity. A second aim of the current study was to examine the effect of ethnicity on sympathetic and parasympathetic reactivity to psychological stress. I hypothesized that participants who were black would exhibit an exaggerated response to the stress tasks compared to white and Hispanic participants. These would be exhibited as an increase in parasympathetic activity during the cold task, and a decrease in parasympathetic activity combined with an increase in sympathetic activity during the math and speech tasks.

The results were the opposite of what I hypothesized. As can be seen in the factorial ANCOVAs, after the variance attributed to family history was removed, black participants exhibited less reactivity compared to white and Hispanic participants. More specifically, during the cold task, black participants had less parasympathetic activation, as evidenced by smaller increases in RSA, than white or Hispanic participants. Additionally, black participants had less sympathetic activation during the speech task, as

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evidenced by smaller decreases in PEP. As described in the implications section, this indicates a blunted reactivity response that has been associated with chronic stress (McEwen & Seeman, 1999). It is important to point out that this research area is still relatively new, and the distinction between what causes exaggerated vs. blunted reactivity patterns has yet to be fully explained.

Family history and recovery. Another aim of the current study was to examine the affect of a positive family history of cardiovascular disease on autonomic recovery following an acute psychological stressor. I hypothesized that participants with a positive family history would exhibit an impaired recovery response following the stress tasks compared to participants with a negative family history. Due to the restorative effects of parasympathetic activity, I expected impaired RSA to be demonstrated by a failure to rebound to levels greater than baseline following the three stress tasks, and sympathetic activity above baseline following the math and speech tasks.

Parental history of hypertension/any of the diseases. There were no significant effects of parental history of disease on parasympathetic or sympathetic recovery following any of the tasks. This was surprising, as previous studies have reported impaired recovery, particularly in sympathetically controlled measures such as DBP and PEP (e.g., Schneider, Jacobs, Gevirtz, and O'Connor, 2003).

Family history of CVD. There was a significant effect of family history of CVD on parasympathetic recovery following the speech task; however, this effect was modified by ethnicity. More specifically, for white participants, a positive family history was related to rebound of RSA to levels greater than baseline while participants with a negative family history had RSA levels around baseline, thus failing to rebound. The

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opposite effect was seen in Hispanic participants, such that participants with a positive family history of CVD did not return to baseline levels while participants with a negative family history exhibited RSA rebound. Mezzacappa, Kelsey, Katkin, and Sloan (2001) reported that participants with a positive family history of CVD demonstrated significantly smaller vagal rebound after a math task than participants with a negative family history, but that there were no significant family history effects on parasympathetic withdrawal during the stressor. The findings in the current study are similar, however only for Hispanic participants. Another study found that participants with a family history of CVD had impaired SBP and DBP recovery, but there were no differences in rMSSD (i.e. RSA) (Wright, O'Donnell, Brydon, Wardle, & Steptoe, 2007), however the effects may be distorted since their family history classification included "unknown" family history as "negative" family history and was not as conservative as the current study.

Family history of stroke. There was a significant effect of family history of stroke on parasympathetic recovery following the math task, but only for black participants. In particular, black participants with a positive family history of stroke exhibited RSA rebound, while black participants with a negative family history of stroke had RSA levels that failed to return to baseline. Additionally, there was a trend for participants with a negative family history of stroke to have impaired sympathetic recovery, as indicated by greater decreases in PEP.

Family history of Type II diabetes. There was no effect of family history of Type II diabetes on sympathetic or parasympathetic recovery following any of the stress tasks. Thus, my hypothesis was not supported. Previous research on autonomic recovery in

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participants with a family history of Type II diabetes is limited. One study found an effect of family history on RSA reactivity, but the effect was moderated by insulin sensitivity, and there were no differences between groups on RSA recovery (Lindmark et al., 2003). The literature provides numerous examples of altered autonomic control in people with a family history of Type II diabetes (e.g. Carnethon et al., 2003). The small sample size in the family history group might have resulted in too little power to reveal an effect.

Ethnicity and recovery. The last aim of the current study was to examine the effect of ethnicity on sympathetic and parasympathetic recovery following the psychological stress tasks. I hypothesized that participants who were black would exhibit an impaired recovery response following the stress tasks compared to white and Hispanic participants. Due to the restorative effects of parasympathetic activity, this was expected to be observed as RSA close to or less than baseline following the three stress tasks (i.e., no vagal rebound), and sympathetic activity above baseline following the math and speech tasks.

In general, there was a significant effect of ethnicity on parasympathetic recovery following the speech task. More specifically, black participants exhibited decreased RSA recovery levels while white and Hispanic participants exhibited RSA levels that remained elevated. The hypothesis was therefore partially supported.

Previous Research and Implications

A genetic predisposition for cardiovascular disease, as indicated by a positive family history, appears to be linked to pathologic autonomic responses to acute stressors. Most studies have confirmed that the genetic component involved in the development of CVD is high. For example, the heritability of myocardial infarction has been shown to be

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anywhere from 25-60%, atherosclerosis ~50%, and Type II diabetes anywhere between 40-80% (Lusis, Mar, and Pajukanta, 2004). Additionally, one twin study on the genetic influence on resting and stress levels of SBP, DBP, HR, PEP, and RSA during a reaction time and mental arithmetic stressor reported the heritability of resting PEP to be 64 – 70%, PEP during stress to be 56 – 74%, resting RSA to be ~31%, and RSA during stress as 44 – 54% (De Geus, Kupper, Boomsma, & Snieder, 2007). Further, genetic influences on autonomic and cardiovascular reactivity have been reported to be between 79 – 82% (Lensvett-Mulders & Hettema, 2001; McCaffery, Bleil, Pogue-Geile, Ferrell, & Manuck, 2003).

Similarly, the current study found significant effects of family history of cardiovascular disease, hypertension, related cardiovascular diseases, stroke, and borderline effects for family history of Type II diabetes on exaggerated autonomic reactivity to both active- and passive-coping stressors. A meta-analysis found that DBP reactivity to stress is the most consistent reactivity measure in participants with a positive family history of hypertension (Fredrikson & Matthews, 1990). An interesting study reported that in black and white children matched with either their sibling or controls, only black children demonstrated a significant relationship between sibling DBP reactivity, indicated that black children have a stronger genetic influence on cardiovascular reactivity (Wilson, Holmes, Arheart, & Alpert, 1995). Given these previous studies, it is intriguing that the effect of parental history of hypertension was only seen in black and Hispanic, and not white, participants.

Genetic factors do not explain all of the variability in a person's stress sensitivity; it is more likely that an inherited hyper-reactive response combined with increased

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psychosocial stress and poor lifestyle choices can intensify the "wear-and-tear" of the body leading to allostatic load (McEwen, 1998). Allostatic load is the long-term effect of stress on the body that leads to chronic dysregulation of physiological systems, such as the autonomic nervous system, the hypothalamic-pituitary-axis, or the cardiovascular system. Allostatic load can occur through one of four situations: (1) frequent, repeated stressors, (2) the inability to adapt to a repeated stressor, (3) the inability to shut off the stress response resulting in impaired recovery, and (4) a blunted stress response that leads to overcompensation in another system (McEwen, 1998).

It has been proposed that race and ethnicity be approached by psychophysiologists not as a proxy for biological and genetic differences, but rather as a proxy for the outcome of varying chronic stressors in the environment, such as racism and discrimination (Anderson et al., 1993). Blacks and Hispanics share many of the same social (e.g. discrimination, racism), environmental (e.g. greater poverty, unemployment), and behavioral (e.g. obesity) stressors. Using physiological variables to measure allostatic load "constitutes a means to access the collective impact of the many environmental and behavioral factors that constitute differences in socioeconomic status" (McEwen & Seeman, 1999). The dysregulation in autonomic reactivity due to allostatic load is one mechanism that would explain why group differences in parental history of hypertension and related cardiovascular diseases are only apparent in black and Hispanic participants. The reactivity patterns seen in Blacks to the cold and speech tasks are examples of the fourth type of allostatic load patterns: an inadequate response of the autonomic nervous system. It is tempting to speculate as to whether other physiological systems, such as the

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inflammatory response, were concurrently over-activated, however this would have to be examined empirically.

Ethnic differences in recovery appear to be indicative of the third type of allostatic load, or the inability to shut off the stress response and restore homeostasis. Following the speech task, black participants had parasympathetic levels that were below baseline whereas white and Hispanic participants exhibited parasympathetic levels that rebounded, or were higher than baseline. A higher parasympathetic recovery score is considered protective and adaptive, in that high vagal control aids in buffering the damaging effects caused by an increased stress response, despite any residual sympathetic activity. For example, one study reported that participants who had PEP and RSA that were higher than baseline during recovery from a mental arithmetic had lower levels of carotid atherosclerosis at a two-year follow up (Heponiemi et al., 2007).

Limitations

Several limitations of this study warrant further discussion. One limitation is potential inaccuracy in classifying family history. Family history was assessed using an online questionnaire that was only a small part of a mass testing questionnaire packet that students were required to complete as a part of their registration on Sona Systems. While participants could hypothetically have discussed their answers with their parents or other relatives while they were completing the questionnaire, it is also possible that their responses are only best guesses as to their actual family history. Additionally, the family history assessment used in the current study is an imperfect measure of genetics.

Another limitation of the study is that ethnicity was not examined using subcategories that are more specific. For example, black participants identified

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themselves as African, African American, Caribbean American, or Jamaican American. Hispanic participants identified themselves as Puerto Rican, Cuban, Columbian, or Mexican American. Some research suggests that there are reactivity differences among ethnic subgroups. For example, Arthur, Katkin, and Mezzacappa (2004) reported different heart periods variability, PEP, and cardiac output reactivity between African Americans and Caribbean Americans during a cold pressor task. This may be an important factor to consider, as the prevalence of cardiovascular disease and related diseases differ between ethnic subgroups. Similarly, Hispanic Americans differ in the degree to which they may be identified as a minority because of differences in skin tone, accent, and name, and therefore can differ in the degree to which they experience the racism and discrimination that is associated with reactivity (Salomon & Jagusztyn, 2008).

All participants were college students; therefore, the different ethnic groups may be more similar to one another in terms of their socio-economic status than is seen in the population. Citing data obtained from the U.S. Census Bureau, Barr (2008) reported that Blacks in the United States are more likely than Whites to (a) never graduate from high school and (b) graduate from high school but not go on to college (Barr, 2008, p. 49). Differences in terms of socio-economic status, which is in part measured by educational attainment, are theorized to play important roles in cardiovascular hyper-reactivity and disease development (Pickering, 1999). This may affect the study's external validity and difficult to generalize the results to the general population.

The sample size may have been too small to detect true differences in the effect of ethnicity and family history of stroke and diabetes on reactivity and recovery. This can be attributed to the conservative criteria used in classifying the family history categories.

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Finally, a significance level of .05 was used for all a priori hypotheses and experiment-wise Type I error rates were not controlled for. As a result, I would caution against over-interpretation of the results until the findings have been successfully replicated.

Future Directions

The current study sheds some light on the combined effects of ethnicity and a hereditary predisposition for CVD on autonomic responses during and after a psychological stressor. Future studies should attempt to recruit participants from a community sample and take care that the sample is representative of the population in terms of SES and education. It would be ideal if future studies would assess family history through direct means, such as accessing medical records. Further examination of the effect of family history of diabetes is warranted, as the current study may have lacked enough participants in order to see a true effect. Type II diabetes is closely related to autonomic dysfunction, and it is hypothesized that prior to the onset of Type II diabetes, low vagal tone leads to a decrease in insulin secretion and an increase in the production of glucose, while high sympathetic activation leads to increases in circulating glucose (see Masi, 2007). Additionally, the current study only examined autonomic responses in females. Therefore, future studies should include a male sample so that gender effects could be examined. Finally, the current study demonstrates the importance of measuring both branches of the autonomic nervous system, and future studies should include measures of parasympathetic reactivity in addition to common sympathetic and vascular measures.

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Summary and Conclusions

Historically, research examining the relationship between cardiovascular reactivity and a genetic predisposition for the development of cardiovascular disease as indicated by a positive family history has focused on sympathetic hyper-reactivity. The current study found that family history groups generally differed not on sympathetic measures, but on parasympathetic responses. This supports the importance of exaggerated parasympathetic reactivity in the development of cardiovascular disease. Gianaros, Salomon, and colleagues (2005) reported similar findings, where greater parasympathetic withdrawal while preparing a speech was related to more extensive coronary and aortic calcification at a median of 282 days later. Interestingly, HR, SBP, and DBP were not significant predictors for calcification (Gianaros et al., 2005). However, opposite findings have also been reported. For example, participants from a large-scale epidemiological study that had greater HR increases, greater RSA decreases, and greater PEP decreases to a mental arithmetic task had *less* carotid atherosclerosis two years later (Heponiemi et al., 2007). Previous studies have found a relationship between increased sympathetic reactivity and risk for cardiovascular disease and hypertension in healthy (Treiber et al., 2003) and patient populations (e.g. Barnett, Spence, Manuck, & Jennings, 1997). There is also an established link between lower parasympathetic influences at a resting state and risk for future cardiovascular disease (Liao et al., 1996) as well as mortality from cardiovascular disease (La Rovere, Bigger, Marcus, Mortara, & Schwartz, 1998).

The results from the current study suggest that there are altered parasympathetic responses to a stressful task or event in healthy women who are at a higher risk for disease. Exaggerated parasympathetic responses to tasks at an early age may contribute to

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the lowered resting parasympathetic levels seen later in life. An important finding in the current study is the evidence of altered autonomic responses in healthy individuals with a genetic predisposition for cardiovascular and related diseases, and that these differences can be seen *before* any alterations in resting levels are observed. Family history of hypertension may only be related to exaggerated reactivity in people with an added environmental risk, such as being Black or Hispanic. The current study also points to the importance a family history of Type II diabetes and particularly a family history of stroke, both of which have been largely overlooked in previous research examining family history and cardiovascular reactivity.

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Appendices

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Appendix A: Family History Questionnaire

Please answer the following about your <u>biological mother</u>:

1. What is your **<u>mother's</u>** age? _____ or ☐ I don't know or ☐ Deceased
2. Has your **<u>mother</u>** ever been diagnosed with or treated for the following:
   a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
   b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
   c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know
   d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
   e. Stroke? ☐ Yes ☐ No ☐ I don't know
   f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
   g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
   h. Heart failure? ☐ Yes ☐ No ☐ I don't know
   i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know
   j. Depression? ☐ Yes ☐ No ☐ I don't know
3. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
   ☐ Yes ☐ No ☐ I don't know

Please answer the following about your <u>biological father</u>:

4. What is your **<u>father's</u>** age? _____ or ☐ I don't know or ☐ Deceased
5. Has your **<u>father</u>** ever been diagnosed with or treated for the following:
   a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
   b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
   c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know
   d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
   e. Stroke? ☐ Yes ☐ No ☐ I don't know
   f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
   g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
   h. Heart failure? ☐ Yes ☐ No ☐ I don't know
   i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know

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Appendix A (Continued)

j. Depression? ☐ Yes ☐ No ☐ I don't know

6. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
   ☐ Yes ☐ No ☐ I don't know

Please answer the following about your <u>biological paternal grandmother</u>:

7. What is your **<u>grandmother's</u>** age? _____ Years or ☐ I don't know or ☐ Deceased
8. Has your **<u>grandmother</u>** ever been diagnosed with or treated for the following:
   a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
   b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
   c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know
   d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
   e. Stroke? ☐ Yes ☐ No ☐ I don't know
   f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
   g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
   h. Heart failure? ☐ Yes ☐ No ☐ I don't know
   i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know
   j. Depression? ☐ Yes ☐ No ☐ I don't know
9. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
   ☐ Yes ☐ No ☐ I don't know

Please answer the following about your <u>biological paternal grandfather</u>:

10. What is your **<u>grandfather's</u>** age? _____ Years or ☐ I don't know or ☐ Deceased
11. Has your **<u>grandfather</u>** ever been diagnosed with or treated for the following:
    a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
    b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
    c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know

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Appendix A (Continued)

d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
e. Stroke? ☐ Yes ☐ No ☐ I don't know
f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
h. Heart failure? ☐ Yes ☐ No ☐ I don't know
i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know
j. Depression? ☐ Yes ☐ No ☐ I don't know

12. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
☐ Yes ☐ No ☐ I don't know

Please answer the following about your <u>biological maternal grandmother</u>:

13. What is your **<u>grandmother's</u>** age? _____ Years or ☐ I don't know or ☐ Deceased
14. Has your **<u>grandmother</u>** ever been diagnosed with or treated for the following:
a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know
d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
e. Stroke? ☐ Yes ☐ No ☐ I don't know
f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
h. Heart failure? ☐ Yes ☐ No ☐ I don't know
i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know
j. Depression? ☐ Yes ☐ No ☐ I don't know

15. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
☐ Yes ☐ No ☐ I don't know

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Appendix A (Continued)

Please answer the following about your <u>biological maternal grandfather</u>:

16. What is your **<u>grandfather's</u>** age? _____ Years or ☐ I don't know or ☐ Deceased
17. Has your **<u>grandfather</u>** ever been diagnosed with or treated for the following:
    a. Hypertension or high blood pressure? ☐ Yes ☐ No ☐ I don't know
    b. High cholesterol? ☐ Yes ☐ No ☐ I don't know
    c. Type 1 or insulin-dependent diabetes? ☐ Yes ☐ No ☐ I don't know
    d. Type 2, late-onset, or adult-onset diabetes? ☐ Yes ☐ No ☐ I don't know
    e. Stroke? ☐ Yes ☐ No ☐ I don't know
    f. Heart attack or myocardial infarction? ☐ Yes ☐ No ☐ I don't know
    g. Coronary heart disease? ☐ Yes ☐ No ☐ I don't know
    h. Heart failure? ☐ Yes ☐ No ☐ I don't know
    i. Any other cardiovascular diseases? ☐ Yes ☐ No ☐ I don't know
    j. Depression? ☐ Yes ☐ No ☐ I don't know
18. If you answered "yes" to question **e**, **f**, **g**, or **h**, did the event occur *before* age 55?
    ☐ Yes ☐ No ☐ I don't know

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Appendix B: Health Questionnaire

1. Age: ____

2. When was the first day of menstruation during your last cycle (mm/dd/yyyy)? _____

3. Have you ever been diagnosed with any of the following conditions:
   □ Heart disease □ Hypertension (high blood pressure) □ Stroke
   □ High cholesterol □ Arrhythmia (irregular heartbeat) □ Diabetes
   □ Heart valve problems

4. Please list all prescription and non-prescription medications that you are currently taking. Be sure to also include any medications you have taken in the last 48 hours, even if it is something you do not regularly take (such as aspirin or cold medicine).
   _____
   _____
   _____

5. When did you last eat? _____ am / pm (circle one)
   a. What did you eat? _____

6. Do you drink beverages containing caffeine? □Yes □No (check one)
   If yes, when did you last drink a caffeinated beverage?
   Time: _____ am/pm (circle one)
   How many caffeinated drinks have you had today? _____
   How many servings (8 oz.) of "energy drinks" (e.g., Redbull, Rockstar, etc.) do you consume in a typical day?
   Regular: _____ Diet: _____
   How many servings (8 oz.) of soda do you consume in a typical day?
   Regular: _____ Diet: _____

7. Do you smoke nicotine cigarettes? □Yes □ No (check one)
   If yes, when did you last smoke? Time: _____ am / pm (circle one)
   How many nicotine cigarettes have you smoked today? _____
   How many nicotine cigarettes do you normally smoke in a day? _____

8. Do any of the following describe your typical diet? ☐ Omnivore (Meat, etc.)
   ☐ Vegetarian ☐ Vegan ☐ Pescetarian ☐ Other: _____

9. When did you last exercise? Please consider any activity that elevated your heart rate for 30 or more minutes.
   Date: _____ Time: _____ Activity: _____

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Appendix C: T-Test Table for Mean Reactivity and Recovery Scores

Table C1
Mean RSA, PEP, and HR Reactivity and Recovery Scores

				95% CI				
		M	*SE*	*LL*	*UL*	*t*	*df*	*p*
Reactivity								
Prep								
	RSA	-0.240	0.073	-0.384	-0.095	-3.286	134	0.001
	PEP	-13.404	1.095	-15.571	-11.238	-12.240	129	<0.001
	HR	9.342	0.773	7.812	10.871	12.078	134	<0.001
Speech								
	RSA	-0.367	0.093	-0.551	-0.182	-3.932	134	<0.001
	PEP	-19.428	1.219	-21.840	-17.017	-15.942	128	<0.001
	HR	14.622	0.908	12.827	16.418	16.106	134	<0.001
Math								
	RSA	-0.080	0.078	-0.235	0.074	-1.028	133	0.306
	PEP	-10.049	0.874	-11.779	-8.319	-11.493	129	<0.001
	HR	8.050	0.667	6.730	9.370	12.062	133	<0.001
Cold								
	RSA	0.443	0.055	0.333	0.553	7.986	134	<0.001
	PEP	-2.590	0.599	-3.776	-1.404	-4.321	129	<0.001
	HR	-1.866	0.384	-2.625	-1.106	-4.857	134	<0.001
Recovery								
Speech								
	RSA	0.106	0.043	0.020	0.191	2.451	133	0.016
	PEP	-5.947	0.584	-7.103	-4.792	-10.184	129	<0.001
	HR	0.304	0.328	-0.346	0.953	0.925	133	0.357
Math								
	RSA	0.127	0.044	0.039	0.214	2.849	133	0.005
	PEP	-3.102	0.455	-4.002	-2.201	-6.815	129	<0.001
	HR	-0.289	0.328	-0.939	0.360	-0.881	133	0.380
Cold								
	RSA	0.166	0.042	0.083	0.249	3.964	134	<0.001
	PEP	-3.353	0.520	-4.381	-2.325	-6.453	130	<0.001
	HR	-1.288	0.320	-1.921	-0.656	-4.028	134	<0.001

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Appendix D: Extra Tables

Table D1
Hypothesis 1: Parent HT. Means, standard deviations, and significance testing

	FH +			FH -						
	M	*SE*	95% CI	*M*	*SE*	95% CI	*df*	*F*	*p*	η^2
RSA										
Math	-0.083	0.106	[-0.292, 0.127]	-0.118	0.123	[-0.361, 0.126]	1, 101	0.046	0.830	0
Cold	0.540	0.072	[0.397, 0.683]	0.280	0.084	[0.112, 0.447]	1, 102	5.411	0.022	0.050
Prep	-0.209	0.102	[-0.412, -0.006]	-0.304	0.119	[-0.540, -0.067]	1, 101	0.362	0.549	0.004
Speech	-0.324	0.120	[-0.563, -0.086]	-0.518	0.140	[-0.796, -0.241]	1, 101	1.100	0.297	0.011
PEP										
Math	-10.26	1.21	[-12.67, -7.86]	-8.04	1.40	[-10.82, -5.26]	1, 100	1.436	0.234	0.014
Cold	-3.01	0.87	[-4.73, -1.29]	-2.05	1.02	[-4.07, -0.03]	1, 100	0.516	0.474	0.005
Prep	-13.46	1.54	[-16.51, -10.42]	-12.70	1.81	[-16.29, -9.11]	1, 100	0.103	0.749	0.001
Speech	-18.24	1.81	[-21.84, -14.65]	-19.44	2.07	[-23.56, -15.33]	1, 99	0.190	0.664	0.002

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Appendix D (Continued)

Table D2
Hypothesis 1: Parent Any. Means, standard deviations, and significance testing

	FH +			FH -						
	M	*SE*	95% CI	*M*	*SE*	95% CI	*df*	*F*	*p*	η^2
RSA										
Math	-0.059	0.092	[-0.241, 0.124]	-0.185	0.150	[-0.482, 0.113]	1, 109	0.505	0.479	0.005
Cold	0.512	0.061	[0.392, 0.632]	0.210	0.099	[0.013, 0.407]	1, 110	6.723	0.011	0.058
Prep	-0.192	0.087	[-0.364, -0.020]	-0.451	0.141	[-0.730, -0.172]	1, 109	2.434	0.122	0.022
Speech	-0.305	0.102	[-0.507, -0.103]	-0.608	0.166	[-0.936, -0.279]	1, 109	2.407	0.124	0.022
PEP										
Math	-10.62	1.15	[-12.90, -8.33]	-8.10	1.88	[-11.82, -4.39]	1, 108	1.304	0.256	0.012
Cold	-2.41	0.76	[-3.91, -0.91]	-2.99	1.26	[-5.49, -0.49]	1, 108	0.153	0.696	0.001
Prep	-13.04	1.36	[-15.74, -10.33]	-13.86	2.27	[-18.36, -9.36]	1, 108	0.096	0.757	0.001
Speech	-19.18	1.61	[-22.37, -16.00]	-19.47	2.60	[-24.63, -14.32]	1, 107	0.009	0.925	0

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Appendix D (Continued)

Table D3
Hypothesis 1: Family CVD. Means, standard deviations, and significance testing

	FH +			FH -						
	M	*SE*	95% CI	*M*	*SE*	95% CI	*df*	*F*	*p*	η^2
RSA										
Math	-0.115	0.119	[-0.353, 0.123]	0.081	0.211	[-0.340, 0.502]	1, 68	0.626	0.432	0.009
Cold	0.397	0.075	[0.246, 0.547]	0.650	0.133	[0.385, 0.915]	1, 69	2.695	0.105	0.038
Prep	-0.135	0.100	[-0.333, 0.064]	-0.320	0.176	[-0.671, 0.032]	1, 69	0.806	0.373	0.012
Speech	-0.327	0.118	[-0.561, -0.092]	-0.069	0.207	[-0.482, 0.344]	1, 69	1.147	0.288	0.016
PEP										
Math	-10.51	1.53	[-13.56, -7.47]	-10.28	2.58	[-15.43, -5.14]	1, 67	0.006	0.940	0
Cold	-2.12	0.93	[-3.97, -0.27]	-3.17	1.62	[-6.40, 0.07]	1, 67	0.310	0.579	0.005
Prep	-13.04	1.55	[-16.13, -9.94]	-13.79	2.71	[-19.20, -8.38]	1, 67	0.057	0.812	0.001
Speech	-19.86	1.85	[-23.55, -16.18]	-18.72	3.15	[-25.01, -12.44]	1, 68	0.096	0.758	0.001

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Appendix D (Continued)

Table D4
Hypothesis 1: Family Stroke. Means, standard deviations, and significance testing

	FH +			FH -						
	M	*SE*	95% CI	*M*	*SE*	95% CI	*df*	*F*	*p*	η^2
RSA										
Math	-0.201	0.139	[-0.479, 0.076]	-0.075	0.169	[-0.413, 0.262]	1, 64	0.313	0.578	0.005
Cold	0.327	0.094	[0.139, 0.514]	0.508	0.114	[0.280, 0.735]	1, 64	1.426	0.237	0.022
Prep	-0.301	0.117	[-0.535, -0.066]	-0.193	0.143	[-0.479, 0.092]	1, 64	0.320	0.573	0.005
Speech	-0.677	0.129	[-0.936, -0.418]	-0.033	0.157	[-0.347, 0.282]	1, 64	9.502	0.003	0.129
PEP										
Math	-11.43	1.86	[-15.13, -7.72]	-10.37	2.26	[-14.89, -5.84]	1, 64	0.126	0.724	0.002
Cold	-2.46	1.01	[-4.47, -0.45]	-0.18	1.25	[-2.68, 2.33]	1, 63	1.950	0.168	0.030
Prep	-14.44	1.72	[-17.88, -11.00]	-10.60	2.14	[-14.87, -6.32]	1, 63	1.882	0.175	0.029
Speech	-22.42	2.22	[-26.85, -17.98]	-14.15	2.68	[-19.50, -8.81]	1, 63	5.405	0.023	0.079

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Appendix D (Continued)

Table D5
Hypothesis 1: Family Diabetes. Means, standard deviations, and significance testing

	FH +			FH -						
	M	*SE*	95% CI	*M*	*SE*	95% CI	*df*	*F*	*p*	η^2
RSA										
Math	-0.363	0.155	[-0.673, -0.052]	0.127	0.192	[-0.259, 0.512]	1, 52	3.433	0.070	0.062
Cold	0.369	0.106	[0.156, 0.582]	0.510	0.131	[0.246, 0.774]	1, 52	0.618	0.436	0.012
Prep	-0.433	0.131	[-0.706, -0.181]	-0.015	0.162	[-0.340, 0.310]	1, 52	3.758	0.058	0.067
Speech	-0.603	0.159	[-0.921, -0.285]	-0.101	0.196	[-0.495, 0.293]	1, 52	3.511	0.067	0.067
PEP										
Math	-12.46	2.14	[-16.75, -8.16]	-8.07	2.54	[-13.16, -2.97]	1, 52	1.627	0.208	0.030
Cold	-3.05	1.10	[-5.27, -0.84]	-0.02	1.29	[-2.61, 2.57]	1, 51	2.934	0.093	0.054
Prep	-16.67	2.23	[-21.15, -12.19]	-10.21	2.61	[-15.46, -4.97]	1, 51	3.235	0.078	0.060
Speech	-23.58	2.66	[-28.92, -18.24]	-15.51	3.16	[-21.85, -9.17]	1, 52	3.547	0.065	0.064

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Appendix D (Continued)

Table D6
Hypothesis 5a: Race x Parent HT. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	-0.264	0.188	-0.038	0.200	-0.066	0.190	-0.366	0.227	0.057	0.179	0.019	0.225	2, 98	0.800	0.452	0.016
Cold	0.460	0.128	0.513	0.134	0.434	0.123	0.004	0.155	0.723	0.120	0.242	0.152	2, 99	2.473	0.090	0.048
Prep	-0.294	0.182	-0.203	0.193	-0.351	0.183	-0.369	0.220	0.002	0.173	-0.374	0.219	2, 98	0.797	0.453	0.016
Speech	-0.303	0.216	-0.468	0.229	-0.447	0.216	-0.574	0.261	-0.221	0.206	-0.531	0.258	2, 98	0.090	0.914	0.002
PEP																
Math	-8.72	2.22	-10.90	2.30	-10.63	2.11	-5.88	2.55	-11.19	2.00	-6.71	2.52	2, 97	1.480	0.233	0.030
Cold	-2.06	1.62	-1.28	1.67	-3.15	1.50	-2.81	1.85	-3.66	1.46	-2.24	1.90	2, 97	0.053	0.948	0.001
Prep	-8.95	2.83	-12.71	2.92	-14.58	2.62	-10.90	3.25	-16.16	2.55	-14.51	3.33	2, 97	0.871	0.422	0.018
Speech	-14.43	3.40	-21.54	3.40	-19.34	3.14	-15.34	3.78	-20.26	2.97	-20.90	3.74	2, 96	1.317	0.273	0.027

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Appendix D (Continued)

Table D7
Hypothesis 5a: Race x Parent Any. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	-0.227	0.162	-0.198	0.247	-0.058	0.170	-0.431	0.284	0.095	0.156	0.039	0.259	2, 106	0.440	0.645	0.008
Cold	0.488	0.106	0.525	0.162	0.360	0.107	-0.132	0.188	0.672	0.102	0.147	0.169	2, 107	2.600	0.079	0.046
Prep	-0.178	0.153	-0.426	0.234	-0.301	0.160	-0.455	0.270	-0.110	0.148	-0.475	0.247	2, 106	0.125	0.883	0.002
Speech	-0.378	0.180	-0.554	0.273	-0.422	0.187	-0.630	0.318	-0.134	0.174	-0.651	0.288	2, 106	0.311	0.733	0.006
PEP																
Math	-10.40	2.07	-10.57	3.18	-8.97	2.09	-7.23	3.54	-12.24	1.93	-6.35	3.21	2, 105	0.673	0.512	0.013
Cold	-1.74	1.37	-1.31	2.10	-2.27	1.36	-4.67	2.34	-3.12	1.28	-3.30	2.22	2, 105	0.317	0.729	0.006
Prep	-9.25	2.44	-14.48	3.75	-13.22	2.42	-11.77	4.18	-16.15	2.28	-15.05	3.96	2, 105	0.687	0.505	0.013
Speech	-17.97	2.94	-20.58	4.43	-17.35	2.92	-17.20	4.93	-21.76	2.69	-20.28	4.47	2, 104	0.157	0.855	0.003

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Appendix D (Continued)

Table D8
Hypothesis 5a: Race x Family CVD. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	-0.313	0.177	0.112	0.319	-0.184	0.242	-0.037	0.422	0.259	0.228	0.161	0.365	2, 65	0.445	0.643	0.014
Cold	0.349	0.114	0.870	0.204	0.322	0.149	0.309	0.262	0.540	0.150	0.653	0.234	2, 66	1.221	0.301	0.036
Prep	-0.200	0.152	-0.215	0.276	-0.315	0.201	-0.639	0.352	0.127	0.196	-0.160	0.313	2, 66	0.251	0.779	0.008
Speech	-0.472	0.177	0.302	0.313	-0.403	0.232	-0.563	0.411	-0.032	0.225	-0.118	0.363	2, 66	1.806	0.172	0.052
PEP																
Math	-11.17	2.29	-10.73	3.97	-8.12	3.14	-13.85	5.52	-11.52	2.84	-6.60	4.59	2, 64	0.884	0.418	0.027
Cold	-0.60	1.40	-4.49	2.41	-4.85	1.83	-3.16	3.14	-1.95	1.73	-1.23	3.05	2, 64	0.954	0.391	0.029
Prep	-9.92	2.33	-14.09	4.02	-17.49	3.05	-16.77	5.23	-13.72	2.88	-10.64	5.08	2, 64	0.526	0.594	0.016
Speech	-19.46	2.83	-18.51	4.89	-21.11	3.72	-23.60	6.37	-19.41	3.51	-14.84	5.66	2, 65	0.257	0.774	0.008

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Appendix D (Continued)

Table D9
Hypothesis 5a: Race x Family Stroke. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	-0.207	0.248	-0.386	0.239	-0.216	0.212	0.150	0.381	-0.204	0.280	0.299	0.294	2, 61	1.000	0.374	0.032
Cold	0.249	0.177	0.598	0.164	0.353	0.148	0.184	0.261	0.347	0.190	0.617	0.202	2, 61	1.009	0.370	0.032
Prep	-0.366	0.214	-0.292	0.207	-0.286	0.184	-0.159	0.330	-0.265	0.242	-0.043	0.255	2, 61	0.054	0.948	0.002
Speech	-0.635	0.236	-0.234	0.226	-0.657	0.202	0.129	0.361	-0.764	0.262	0.177	0.278	2, 61	0.652	0.525	0.021
PEP																
Math	-11.18	3.42	-14.96	3.24	-10.46	2.78	-3.80	4.99	-12.98	3.64	-8.46	3.89	2, 61	1.247	0.295	0.039
Cold	-1.16	1.91	0.17	1.81	-4.40	1.55	-2.24	2.78	-1.28	2.03	1.62	2.30	2, 60	0.079	0.925	0.003
Prep	-12.58	3.25	-12.44	3.08	-13.85	2.64	-7.87	4.73	-17.04	3.46	-10.17	3.91	2, 60	0.611	0.546	0.020
Speech	-24.38	4.18	-20.57	3.76	-16.97	3.24	-5.56	5.81	-28.08	4.24	-12.19	4.53	2, 60	1.121	0.333	0.036

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Appendix D (Continued)

Table D10
Hypothesis 5a: Race x Family Diabetes. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	-0.405	0.269	-0.138	0.277	-0.347	0.234	0.319	0.442	-0.303	0.297	0.389	0.310	2, 49	0.360	0.700	0.014
Cold	0.412	0.184	0.604	0.185	0.342	0.163	-0.107	0.297	0.286	0.200	0.763	0.209	2, 49	2.189	0.123	0.082
Prep	-0.328	0.225	-0.219	0.233	-0.391	0.198	-0.081	0.371	-0.635	0.251	0.282	0.263	2, 49	1.475	0.239	0.057
Speech	-0.446	0.284	-0.079	0.289	-0.708	0.247	-0.140	0.472	-0.649	0.311	-0.080	0.327	2, 49	0.078	0.925	0.003
PEP																
Math	-10.92	3.99	-12.46	3.55	-11.54	3.18	-3.33	5.91	-14.90	4.02	-4.54	4.23	2, 49	1.339	0.271	0.052
Cold	-1.29	2.04	0.92	1.83	-4.32	1.63	-2.40	3.03	-3.48	2.20	0.58	2.17	2, 48	0.139	0.871	0.006
Prep	-11.03	4.07	-11.72	3.64	-17.05	3.24	-7.03	6.03	-21.72	4.39	-10.12	4.33	2, 48	1.396	0.257	0.055
Speech	-18.45	4.91	-20.52	4.38	-22.28	3.92	-7.84	7.29	-29.47	4.95	-13.41	5.22	2, 49	2.177	0.124	0.082

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Appendix D (Continued)

Table D11
Hypothesis 5b: Race x Parent HT. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	0.097	0.102	0.250	0.107	0.018	0.101	0.051	0.123	0.123	0.096	0.130	0.121	2, 97	0.272	0.762	0.006
Cold	0.061	0.091	0.241	0.097	0.254	0.089	0.226	0.115	0.077	0.088	0.164	0.109	2, 98	0.569	0.568	0.011
Speech	0.065	0.092	0.299	0.100	-0.015	0.092	-0.154	0.116	0.169	0.088	0.055	0.110	2, 95	2.270	0.109	0.046
PEP																
Math	-1.45	1.01	-3.93	1.04	-3.11	0.96	-3.20	1.17	-3.60	0.91	-3.73	1.15	2, 96	0.854	0.429	0.017
Cold	-2.33	1.00	-3.27	1.04	-3.73	0.93	-4.22	1.15	-4.00	0.91	-4.21	1.18	2, 96	0.064	0.938	0.001
Speech	-5.36	1.18	-5.99	1.18	-6.20	1.08	-6.36	1.36	-7.28	1.02	-5.33	1.29	2, 94	0.686	0.506	0.014

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Appendix D (Continued)

Table D12
Hypothesis 5b: Race x Parent Any. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	0.158	0.085	0.231	0.130	0.085	0.089	-0.040	0.149	0.141	0.082	0.062	0.135	2, 105	0.420	0.658	0.008
Cold	0.122	0.076	0.229	0.116	0.244	0.078	0.285	0.139	0.087	0.075	0.217	0.123	2, 106	0.094	0.910	0.002
Speech	0.134	0.080	0.295	0.120	-0.034	0.082	-0.141	0.148	0.192	0.077	0.044	0.127	2, 103	1.308	0.275	0.025
PEP																
Math	-1.89	0.85	-4.67	1.31	-3.08	0.86	-3.85	1.46	-3.32	0.80	-4.08	1.33	2, 104	0.549	0.579	0.010
Cold	-2.15	0.81	-4.31	1.24	-3.91	0.80	-3.43	1.39	-3.92	0.75	-4.06	1.31	2, 104	0.835	0.437	0.016
Speech	-5.29	0.98	-6.65	1.48	-6.16	0.98	-7.15	1.75	-6.89	0.90	-5.70	1.49	2, 102	0.598	0.552	0.012

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Appendix D (Continued)

Table D13
Hypothesis 5b: Race x Family CVD. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	0.154	0.085	0.332	0.154	0.211	0.115	0.138	0.196	0.213	0.110	-0.118	0.173	2, 64	1.856	0.165	0.055
Cold	0.132	0.076	0.118	0.140	0.312	0.100	0.471	0.175	0.183	0.099	0.016	0.158	2, 65	0.717	0.492	0.022
Speech	0.101	0.090	0.448	0.159	-0.091	0.115	0.039	0.204	0.249	0.115	-0.175	0.181	2, 64	3.738	0.029	0.105
PEP																
Math	-2.09	0.92	-6.1	1.59	-3.52	1.26	-1.67	2.07	-3.65	1.14	-3.35	1.85	2, 63	2.211	0.118	0.066
Cold	-2.05	0.91	-3.94	1.56	-4.89	1.20	-2.11	2.02	-2.92	1.11	-4.16	1.96	2, 63	1.356	0.265	0.041
Speech	-5.06	1.02	-8.08	1.77	-6.36	1.35	-6.11	2.31	-7.26	1.27	-5.94	2.06	2, 64	1.077	0.347	0.033

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Appendix D (Continued)

Table D14
Hypothesis 5b: Race x Family Stroke. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	0.198	0.115	0.274	0.112	0.226	0.099	-0.380	0.177	0.143	0.129	0.022	0.139	2, 60	3.610	0.033	0.107
Cold	0.152	0.106	0.104	0.102	0.214	0.092	0.334	0.160	0.105	0.116	0.109	0.124	2, 60	0.258	0.774	0.009
Speech	0.191	0.133	0.326	0.120	0.108	0.107	-0.326	0.216	0.248	0.141	-0.043	0.152	2, 58	2.197	0.120	0.070
PEP																
Math	-2.95	1.36	-3.53	1.30	-3.38	1.10	-3.61	2.01	-2.98	1.45	-2.29	1.55	2, 60	0.104	0.902	0.003
Cold	-2.62	1.20	-3.83	1.15	-2.77	1.00	-6.65	1.75	-1.91	1.28	-2.85	1.47	2, 59	0.715	0.494	0.024
Speech	-6.32	1.43	-6.24	1.27	-5.37	1.10	-8.94	2.24	-5.26	1.48	-5.13	1.56	2, 58	0.879	0.421	0.029

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Appendix D (Continued)

Table D15
Hypothesis 5b: Race x Family Diabetes. Means, standard deviations, and significance testing

	White				Black				Hispanic							
	FH+		FH-		FH+		FH-		FH+		FH-					
	M	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*M*	*SE*	*df*	*F*	*p*	η^2
RSA																
Math	0.107	0.156	0.220	0.160	0.079	0.137	-0.176	0.257	0.058	0.172	-0.067	0.185	2, 48	0.598	0.554	0.024
Cold	0.185	0.120	0.050	0.125	0.069	0.107	0.414	0.200	-0.063	0.134	0.080	0.143	2, 48	1.592	0.214	0.062
Speech	0.060	0.140	0.232	0.149	-0.114	0.123	-0.396	0.229	0.108	0.155	-0.074	0.162	2, 47	1.208	0.308	0.049
PEP																
Math	-2.33	1.47	-3.97	1.31	-4.00	1.17	-4.79	2.21	-2.65	1.50	-3.30	1.59	2, 48	0.067	0.935	0.003
Cold	-4.38	1.35	-3.14	1.23	-4.21	1.10	-5.31	2.00	-5.04	1.46	-2.69	1.45	2, 47	0.651	0.526	0.027
Speech	-7.40	1.46	-6.07	1.30	-6.48	1.17	-10.11	2.22	-7.32	1.52	-6.09	1.57	2, 48	1.459	0.242	0.057

www.manaraa.com

About the Author

Mardís Sara Karlsdóttir is currently pursuing her doctorate degree in Psychology. Prior to moving to Tampa to attend the University of South Florida, Mardís graduated Magna Cum Laude with a Bachelor of Arts in Psychology from Shawnee State University in Portsmouth, Ohio, where she received an award as the Outstanding Psychology Student of the year in 2006. She has presented her research at conferences for the American Psychological Association and the Society for Psychophysiological Research, and has co-authored a paper published in *Health Psychology*. Mardís's research interests include health psychology, stress and cardiovascular disease, positive psychology, and psychophysiology. She enjoys traveling, visiting family and friends in her native country of Iceland, and spending time with her husband and two daughters.

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