Personality and Cardiovascular Stress Reactivity: A Comparison of the Cold Pressor and Mental Arithmetic Tasks

by

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Abstract

Stress reactivity, defined as an exaggerated response to stressors and impaired post-stressor recovery, significantly influences the connection between stress and health, particularly cardiovascular health. A wide body of literature has identified personality traits strongly associated with stress reactivity, namely trait neuroticism from the Five-Factor model of personality. Evidence suggests that individuals scoring highly in this personality construct are more likely to have exaggerated stress responses and experience more severe stress-related health problems. In the current study, participants (N = 700) completed an inventory of the Five-Factor model of personality along with medical questionnaires to assess eligibility for stress assessments. Eligible participants (N = 93) completed baseline electrocardiogram (ECG) and respiratory recordings. Participants then underwent continuous ECG/respiratory recording while completing two separate stress tasks: a mental stress task (MST) and a cold pressor task (CPT), each task followed by a second and third ECG/respiratory recording period. In light of the literature reviewed in chapters 1, 2, 3, and 4, I hypothesized that participants scoring more highly in trait neuroticism would exhibit decreased measures of vagally mediated heart rate variability (HRV) and increased measures of sympathetically mediated HRV during baseline and would exhibit increased stress responses to both stress tasks. Further, I hypothesized that the MST would induce greater stress responses than the CPT and that the task type would moderate the relationship between trait neuroticism and HRV measures of cardiovascular stress. Results revealed that there was no consistent relationship between baseline HRV and trait neuroticism, except for LF-HRV which was associated with greater neuroticism. Results also showed that there was a task difference such that LF-HRV was higher during the CPT compared to the MST.

There was no moderation effect of the task type on the relationship between neuroticism and HRV. Notably, stress appraisal was associated with increases These results are discussed in terms of both the transactional model of stress and coping and the neurovisceral integation model.

Artificial Intelligence (AI) Use Disclosure Statement

In the preparation of this thesis / dissertation, the following Artificial Intelligence (AI) tools were used: ChatGPT Model 5. These tools were used to perform troubleshooting on code generated in RStudio. The author acknowledges full responsibility for the intellectual content of this work and has ensured that all AI-assisted sections have been reviewed and revised for accuracy and appropriate academic style. All AI-generated content was reviewed and validated for relevance, appropriateness, and accuracy before incorporation into the final document to maintain scholarly integrity of this research.

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Table of Contents

List of Tables

List of Figures

List of Abbreviations

ANS	Autonomic Nervous System
BAI	Beck Anxiety Inventory
BDI-II	Beck Depression Inventory-II
CNS	Central Nervous System
CVT	Cardiac Vagal Tone
CVD(s)	Cardiovascular disease(s)
CPT	Cold Pressor Task
ECG	Electrocardiogram
FIR	Finite Impulse Response
HRV	Heart Rate Variability
HF-HRV	High-Frequency Heart Rate Variability
IBI	Interbeat Interval
LF-HRV	Low-Frequency Heart Rate Variability
LMM(s)	Linear Mixed-Effects Model(s)
IBI	Interbeat Interval
MST	Mental Stress Task
NIM	Neurovisceral Integration Model
NN50	Count of N-N intervals that differ more than 50ms
PNS	Parasympathetic Nervous System
pNN50	Percentage of N-N intervals that differ more than 50ms
PSD	Power Spectral Density
RMSSD	Root Mean Square of Successive Differences

ANOVA	Analysis of Variance
RSA	Respiratory Sinus Arrhythmia
SDNN	Standard Deviation of N-N intervals
SNS	Sympathetic Nervous System
SVS	Stereotype Vulnerability Scale
VIF	Variance Inflation Factor

“It’s the heart that really matters in the end.”

-Rob Thomas, *Little Wonders*

Chapter 1: Brief Overview and Introduction to the Current Research

Few topics in the health sciences have garnered more attention than the relationship between stress and health, especially cardiovascular health. Two physicians (M. Friedman & Rosenman, 1959) interested in predicting risk factors for heart disease proposed one of the earliest accounts of the relationship between psychological stress and cardiac dysfunction in the late 1950s. In their investigation, they proposed a typology of individuals separated into Types A, B, and C. The researchers identified these groupings from a sample of male participants with differing behavioral attitudes to their work. Briefly, Friedman and Rosenman observed that the group (Type A) most characterized by intense sustained drive for achievement, high stress competition, hostility, impatience, and anger exhibited a greater incidence of coronary artery disease and higher serum cholesterol level than the other groups (Type B and Type C) in their sample (M. Friedman & Rosenman, 1959). Type B consisted of age and sex matched controls without diagnosed cardiovascular health challenges and Type C consisted of age and sex matched controls that were currently unemployed. This research captured the scientific and public imagination and became a popular personality construct (M. Friedman & Ulmer, 1985), as well as an inspiration for scientists exploring personality determinants of health.

Although investigators performing additional meta-analytic work found no robust association between the Type A behavior pattern and coronary artery disease (Myrtek, 2001), a large body of future research built findings on the basic premise that personality traits might serve as risk factors for cardiovascular dysfunction. For example, several investigators have argued that the anger and hostility components of the Type A behavior pattern are relatively sensitive predictors of cardiovascular disease (CVD) based on meta-analytic evidence (Chida & Steptoe, 2009; Delunas, 1996; Myrtek, 2001). This project focuses on the interplay between

personality traits, stress, stress reactivity, and cardiovascular function, which I outline in chapter 4.

Researchers have continually explored the relationship between personality constructs and cardiovascular health outcomes, establishing a rich empirical literature spanning several decades (for a comprehensive review, see (Sahoo et al., 2018)). In tandem with discoveries about the relationship between cardiovascular dysfunction and personality, researchers also began to explore how stress influences cardiovascular health. The literature to date has consistently demonstrated that chronic stress can have deleterious effects on both physical and psychological health (Juster et al., 2010; Marin et al., 2011; McEwen, 2008, 2017; Schneiderman et al., 2005). More specifically, chronic stress has been linked to a wide range of adverse cardiovascular health outcomes, including atherosclerosis, cardiomyopathy, increased incidence of all categories of CVDs, and mortality due to adverse cardiovascular events (Lagraauw et al., 2015; Steptoe & Kivimäki, 2013; Yao et al., 2019). Among adults at risk for CVDs, stress can be a disease trigger, and it can play a determining role in CVD outcomes among individuals currently diagnosed with a CVD (Kivimäki & Steptoe, 2018). Stress is also a potent risk factor for adverse cardiovascular events independent of other confounding risk factors for CVD. In the largest cardiovascular case control study ever published (24,000 age and sex matched participants), researchers found that heightened psychosocial stress over the previous year doubled the risk of myocardial infarction, even when all other known CVD risk factors were controlled for (Rosengren et al., 2004). More recently, investigators have also demonstrated that psychosocial stress and stress conditions are independently associated with CVD in a way that depends not only on the degree and duration of the stressor(s), but also the individual appraisal of and

response to the stressor(s) (Dar et al., 2019). Such research illustrates the importance of inter-individual variability in predicting the health impacts of stress exposure for different individuals.

Another identified risk factor for cardiovascular dysfunction is stress reactivity, broadly defined as exaggerated reactivity to stressors and subsequent impaired post-stressor recovery (P. G. Williams et al., 2011). Researchers have pointed to meta-analytic evidence suggesting that individuals who exhibit greater cardiovascular responses to laboratory stressors are significantly more likely to have future instances of elevated blood pressure, hypertension, left ventricular mass, subclinical atherosclerosis, and clinical cardiac events (Chida & Steptoe, 2010). Consequently, a critical piece in unveiling our grasp of the relationship between personality, stress, and cardiac function must be the relationship between personality traits and cardiovascular stress reactivity.

A key source of motivation for this project is an empirical investigation carried out among a sample of Swedish men (Flaa et al., 2007). The investigators in this study selected men with blood pressure responses at the 1st, 25th, 75th, and 99th percentiles, respectively. These men were invited to participate in a laboratory study where they were exposed to a mental arithmetic stressor and a cold exposure stressor. Blood pressure readings were taken during stress exposure. The men were also assessed using the Karolinska Personality Scale (Ortet et al., 2002), a multi-dimensional personality questionnaire that assesses a wide range of personality and temperament traits, including negative emotionality, aggressive nonconformity, impulsive sensation seeking, and social withdrawal. They observed that cardiovascular reactivity (assessed by diastolic blood pressure) to the cold exposure stressor correlated with traits such as irritability, muscular tension, and somatic anxiety. Further, verbal aggression and detachment predicted blood pressure

responses to the mental stressor. Both findings converge on the notion that personality traits associated with hostility or aggression are linked to cardiac dysfunction (Chida & Steptoe, 2009).

A secondary source of motivation for this project comes from a review where investigators examined the empirical data linking personality traits and biological reactivity to stress (Soliemanifar et al., 2018). This paper examined over 38 different studies that assessed biological stress responses to laboratory-based stressors. In particular, the authors of this review demonstrated that effect sizes for stress responses tended to be larger for mentally demanding stress tasks compared to stress tasks that evoked physical or social stress. As it relates to the present research, Soliemanifar and colleagues (2018) demonstrate that personality traits, particularly trait neuroticism, strongly predict biological reactivity to stress and that different laboratory-based stress induction paradigms vary in their effectiveness at eliciting stress responses.

Lastly, a final source of motivation for the project came from an empirical study in which investigators explored the autonomic correlates of personality traits (Shepherd et al., 2015). Specifically, Shepherd and colleagues (2015) invited 106 healthy participants to the laboratory to complete a 10-minute electrocardiographic (ECG) recording. Participants also completed a Five-Factor model personality inventory (Costa & McCrae, 1992). They observed an inverse correlation between HRV and trait neuroticism. In addition, Shepherd and colleagues used structural analyses to combine personality traits into personality prototypes characterized by resilient functioning (low neuroticism, higher than average remaining big five scores), average functioning (moderate scores on all traits), and non-desirable functioning (high neuroticism and lower than average remaining big five scores). Shepherd and colleagues (2015) observed that the non-desirable functioning group had significantly reduced HRV compared to the average and

resilient groups. Lastly, they also observed significantly reduced HRV among participants that scored more highly on the State-Trait Anxiety Inventory. Shepherd and colleagues (2015) argued that these results suggest that researchers could refine metrics of cardiac function to assess different personality types. Although it is critical to understand how personality may modulate resting indices of HRV, I argue that it is equally critical to characterize how personality traits influence responses to diverse types of stressors.

Beyond the work of Shepherd and colleagues (2015), the relationship between trait neuroticism and psychophysiological responses to stress has been extensively examined (Ahmad et al., 2021; Brouwer et al., 2015; Evans et al., 2016; A. Hansen & Johnsen, 2013; Hughes et al., 2011; Koelsch et al., 2012; Norris et al., 2007; Ode et al., 2010; Reynaud et al., 2012; Roger & Jamieson, 1988; Schwebel & Suls, 1999). Trait neuroticism is a well-developed (Ormel et al., 2013), robust (Lahey, 2009), relatively stable (Bleidorn et al., 2022), and heritable (Power & Pluess, 2015) personality construct that assesses a dispositional tendency to experience negative affect (Widiger & Oltmanns, 2017), and it predicts exaggerated reactivity to a wide range of stressors (Bolger & Schilling, 1991; M. D. Robinson et al., 2010). Furthermore, investigators have demonstrated that previously observed associations between Five-Factor personality traits and consequential life outcomes are highly replicable (Soto, 2019), further underscoring the utility of the Five-Factor model for assessing significant individual differences. In chapter 2, I explore this literature in greater depth, and I argue that trait neuroticism represents the optimal personality measure to assess individual differences in stress reactivity.

The findings discussed in the preceding paragraphs serve as the primary motivation for the project, which is described in chapter 4. In chapter 2, I review existing theories of stress and describe the literature linking personality and stress reactivity to cardiovascular health outcomes.

In chapter 3, I review the basic physiology of the cardiovascular system and the role of the vagus nerve in promoting adaptive cardiovascular stress responses. I also discuss the primary methods for analyzing HRV and theories that account for its role in stress and health. Lastly, in chapter 4, I describe the current research project, present the findings, and discuss the implications. I conclude with a discussion of the limitations of this project and possible directions for future investigations.

Chapter 2: Stress, Personality, and Cardiovascular Health

In antiquity, the view that the human heart was the seat of the soul, or the primary foundation of mental life was commonplace. This notion was held by Greek philosophers such as Aristotle, ancient Confucian philosophers such as Xunzi, and it even found expression in the ancient Egyptian Book of the Dead (Brandt & Huppert, 2021). As empirical findings on the significance of the brain's role in mental function accumulated, researchers increasingly replaced this perspective with the idea that the brain plays the leading role in facilitating mental life. Of course, the brain's importance for mental life was hypothesized by many in the ancient world as well, first conjectured by Greek physicians and philosophers such as Hippocrates, Galen, and Pythagoras (Pandya, 2011). More recent research on the nature of the nervous system has further refined this understanding, showing that mental and emotional life arise from integrated communication between the central (CNS) and autonomic (ANS) nervous systems (CNS), including direct interaction between the brain and the heart (Collet et al., 2013). The Chinese character 心, spelled Xin in the English alphabet, is commonly translated as heart-mind (Shun, 2010). Although the word literally refers to the physical heart, it also denotes the seat of mental life, including both emotion and cognition, which are not viewed as separate in Chinese thought. The term, which has no direct equivalent in English, aptly captures the integration of cognition and emotion (Blair & Dennis, 2010). Consequently, the idea that heart health is closely linked to individual differences has a long and rich history both in medicine and beyond.

Our capacities for responding to the challenges of life are intimately tied to the integration of the CNS and ANS (Thayer et al., 2010). This integration enables variability in physiological rhythms that regulate bodily functions, creating the conditions needed to flexibly respond to an often unpredictable environment (Shaffer et al., 2014). Among these challenges is

the broad range of events that are loosely referred to as stressors, often described as those events that significantly affect our capacity to adapt. When these events occur chronically or overtax coping capacity, they can dysregulate and damage both the CNS (E. J. Kim & Kim, 2023) and especially the ANS, disrupting cardiovascular health (Thayer et al., 2010). HRV represents a physiological marker of the variability essential for responding to a continually changing environment (Thayer & Lane, 2009). Consequently, investigators have explored the relationship between psychological stress and HRV as a potential risk factor for cardiovascular dysfunction.

Of course, most stress research uses laboratory assessments given ethical constraints on inducing significant or chronic stress in a controlled way. Despite this limitation, laboratory-induced psychological stress reduces HRV and increases heart rate, blood pressure, cortisol, and other markers of autonomic reactivity (Delaney & Brodie, 2000). Further, resting HRV and HRV in response to psychological challenges in the lab have been demonstrated to robustly correlate with HRV responses to ecologically valid emotional stressors outside the laboratory (Dikecligil, 2010; Rajcani et al., 2016). Stress appraisal or perception seems to play a significant role. For example, Thayer and colleagues (2012) conducted a meta-analysis of neuroimaging and psychophysiological studies where they observed that the brain regions associated with threat appraisal are most strongly active in individuals with reduced resting HRV and reduced HRV in response to stressors. They concluded that reduced HRV may reflect top-down neural appraisal processes marked by a negativity bias to uncertainty (Thayer et al., 2012), further underscoring the notion that HRV may represent a critical nexus in the adaptive integration between the CNS and ANS.

Therefore, understanding the factors that predispose individuals to increased reactivity to stress is critical for identifying pathways to illness. It is also crucial to understand

how personality traits influence individual appraisals of acute stress. In turn, this can give us a deeper understanding of which kinds of tasks and experiences are more or less effective at inducing physiological reactivity on an individual basis. Given that personality traits can serve as predictors of stress levels, moderators of stress levels, or moderators of appraisal, coping capacity, and coping responses to stressors (Kern & Friedman, 2011), it is unsurprising that the relation between traits and health-related outcomes is extraordinarily complex. In this chapter, I discuss the nature of psychological stress and explore the complex and often inconsistent relationships between stress, physiological reactivity, personality, and cardiovascular health outcomes.

What is Stress?

Like many psychological constructs, the term stress suffers from an excess of definitions. This stems partly from conceptual ambiguity in how the term is used and its ubiquity in the psychological literature. Some researchers have argued that overuse of the term stress is a primary impediment to developing a conceptually clear definition of stress (S. M. Monroe, 2008; S. Monroe & Slavich, 2016, 2019). Often, researchers use the term stress to describe both external events that place metabolic demands on the organism but also to describe the response of the organism to those external events (Harkness & Hayden, 2020), which conceptually confuses stress exposure with stress response. Furthermore, individual differences heavily shape how organisms respond to stress, meaning that reactions to the same objectively defined stressors often vary widely (Ellis et al., 2011). Additionally, stress responses can be characterized at multiple levels of analysis, including subjective emotional states, psychophysiological reactions, and acute *vs.* chronic responses (Harkness & Hayden, 2020), highlighting the need for greater conceptual clarity in how the term is defined and applied. Before conceptually clarifying the

term stress, I will review its historical origins and the most influential psychological accounts of stress.

Endocrinologist Hans Selye introduced the concept of stress into modern psychology through his pioneering experiments with rats (Selye, 1936). As a young medical student, Selye observed that many of his patients had shared symptoms like lethargy, weakness, and depression even though their diagnoses varied widely. This caused Seyle to speculate that there may be a general bodily response to injury or illness, independent of the specific disease (Szabo et al., 2017). While stimulating female rats in an attempt to discover new ovarian hormones, Seyle observed an enlargement of the adrenal glands. Reasoning that these changes were not caused by the chemicals themselves, Selye injected rats subcutaneously with small doses of toxic formalin or subjected them to extreme cold under immobilization. At the end of his experiments, the unfortunate rats exhibited similar symptoms of lethargy and weakness to those of Selye's patients, along with enlarged adrenal glands, atrophied thymi, spleens, lymph nodes, and gastric ulcers (Selye, 1936). He then defined stress as a nonspecific bodily response to noxious stimuli. More specifically, Selye identified the adrenal cortex as the chemical seat of the stress response based on observed increases in steroid secretion in this cortex during and after his experiments. These chemicals were later named as glucocorticoids (Szabo et al., 2017).

Rather than the term stress, Selye (1936) referred to this response as a syndrome in his initial publication. He later formalized the term stress in modern psychological science through his comprehensive account of the General Adaptation Syndrome (GAS; Selye, 1950). Selye outlined three phases in the GAS: Alarm Reaction, Resistance, and Exhaustion. In brief, the alarm stage refers to the immediate physiological changes that occur in response to a stressor such as increases in heart rate or cortisol secretion. In the second stage, the body sustains

physiological arousal while initiating repair processes in response to the effects of stress hormones released during the alarm stage. Lastly, if the alarm stage persists (e.g., in persistent chronic stress), the exhaustion stage begins, which leads to mental, physical, and emotional depletion (Selye, 1950). Much later, and like many other researchers, Selye bemoaned the conceptual confusion around the term stress itself, infamously stating, "Everybody knows what stress is, and yet nobody knows what stress is" (Selye, 1973, p. 6). Nonetheless, in his last publication on the topic, Selye offered a broad definition of stress as "the nonspecific response of the body to any demand upon it" (Selye, 1976, p. 1). Although his definition left stress scholars with more questions than answers, his research laid the groundwork for future study of the biological mechanisms involved in our adaptations to the demands of life.

Since then, researchers have shown that stress processes are far more complex than Selye's original model suggested. Although often described as purely biological, stress involves a dynamic interplay between biological and psychological systems. To clarify this complex topic, I follow Pearlin in describing the stress process through three conceptual domains: the sources, mediators, and manifestations of stress (Pearlin et al., 1981). This approach characterizes stress as an interactive and transactional process shaped by an organism's states and traits (Lazarus & Launier, 1978). For example, researchers have frequently made the point that what counts as a stressor cannot be objectively determined because the manifestations of stress (e.g., increased serum cortisol) vary significantly between individuals, even upon exposure to similarly and sometimes objectively defined life events (Dohrenwend, 2006). Others have also argued that the manifestations of stress can be confounded by co-occurring mental and physical health symptoms (e.g., serum cortisol levels tend to be higher among individuals with

depression) (S. M. Monroe & Roberts, 1990). These issues underscore the need to examine stress exposure and stress response interdependent components of a unified process.

Furthermore, stress appraisal (Harkness & Hayden, 2020) plays a critical role in shaping the strength, intensity, and duration of the stress response, serving as a key mediator. Recognizing the importance of appraisal led to the development of the Transactional Model of Stress and Coping (Lazarus & Folkman, 1984), perhaps the dominant model for understanding the psychological components of stress since it was first proposed (Lazarus, 1966). This model views stress as a transaction between the organism and the environment (Lazarus & Launier, 1978), rather than merely a biological reaction to an external event. This perspective distinguishes between primary and secondary appraisal. During primary appraisal, the organism evaluates the significance of the event. In secondary appraisal, the organism evaluates its own coping resources and available options for managing the event. Along similar lines, other investigators have described stress in terms of a mismatch between environmental demands and perceived coping capacity (Cohen et al., 2016), conceptualizing stress as a relationship between an external demand and an inner capacity. Using this approach, personality traits can impact how appraisal and coping processes occur; for example, some evidence indicates that individuals characterized by elevated neuroticism tend to appraise their coping resources as inadequate to the demands of stressors, both negatively impacting emotional experience and task performance in lab-based stress paradigms (Schneider, 2004; Schneider et al., 2012).

Many theories of stress emphasize how stress is experienced, aligning with the second conceptual domain: manifestations of stress. For example, Biggs and colleagues argue that stress is most commonly conceptualized as an external stimulus, a response to a stimulus, an organism-environment interaction, or a transactional process between organism and environment

(Biggs et al., 2017). For example, a frequent practice in stress research involves classifying any event that is accompanied by changes in specific biological/behavioral measures as a stressor, regardless of any effect these changes have on long-term health or future coping capacity. Kagan has convincingly argued that this practice defines stress in an overly permissive way, claiming instead that stress should be limited to select events that pose a serious threat to an organism's well-being (Kagan, 2016). Kagan argues that lumping all psychological tension under the term "stress" dilutes the concept to the point of theoretical uselessness (Kagan, 2016). Given that a stressor cannot be objectively defined due to differences in stress appraisal, I argue that defining stress as Kagan does is overly narrow. Instead, I propose that the sources, mediators, and manifestations of stress comprise an integrated and evolving process, one that is shaped via interactions and transactions between the organism and the environment. Therefore, models that integrate these separate components of the stress process into a unifying explanation should be preferred.

Most stress research has focused more on manifestations than on sources and mediators of stress, particularly the physiological correlates of stress experience. An enormous and highly complex cascade of physiological processes is initiated when an event occurs that places an external metabolic demand on the organism. In brief, when the stress process occurs, the central nervous system (CNS) activates and stimulates the hypothalamic-pituitary-adrenal (HPA) axis, leading to a cascade of physiological changes facilitated by the ANS (Torpy & Chrousos, 1997). Selye's GAS was later superseded by models of stress based on the role of glucocorticoids produced in the adrenal cortex during stress experiences (E. J. Kim & Kim, 2023).

An exhaustive review of all the physiological correlates of stress experience is beyond the scope of this chapter; however, a brief description is necessary for understanding these processes at a broad level. In brief, sympathetic nervous system (SNS) activation begins within moments of stressor onset (Cacioppo et al., 1998). Shortly thereafter, parasympathetic withdrawal or activation can occur, either amplifying SNS activation to further increase the stress response or diminishing SNS activation to restore homeostatic balance (Porges, 2007). HPA activation facilitates increased secretion of glucocorticoid steroids, which help mobilize energy resources for adaptive action (Sapolsky et al., 2000). However, chronically elevated levels of these hormones can induce long-term structural (Radley & Morrison, 2005), functional (Golkar et al., 2014), and neurochemical (McEwen, 2017) changes to the brain, causing long-term changes that can impair adaptive function (Kemeny, 2003; E. J. Kim & Kim, 2023; O'Brien, 1997). For the purposes of this project, my primary focus will be on exploring the changes initiated in the ANS as a consequence of stress experience. For a more detailed account of the impacts of stress on the CNS, I recommend Kim & Kim's recent review paper (E. J. Kim & Kim, 2023). Biological theories of stress are appropriate for elucidating the physiological manifestations of stress, but they fall short of capturing the full scope of the stress process.

Returning to the transactional model of stress and coping, the notion that stress represents a transaction between organism and environment inspired further research exploring the means by which cognitive appraisals can trigger, amplify, and mediate the stress process. Investigators later developed the Cognitive Action Theory of Stress (Ursin & Eriksen, 2004), which views stress as a healthy alarm that triggers general neurophysiological activation when a discrepancy between a present state and a desired state occurs. Ursin and Eriksen argue that chronic stress reflects sustained activation in response to stressors when effective coping

mechanisms are absent (Ursin & Eriksen, 2010). Further, they argue that the conditions that define when a stressor occurs are the same across contexts for all organisms (Eriksen et al., 2005); namely, a stressor occurs when a mismatch between an expectation and an outcome occurs. Although this approach offers intriguing methods to explore cognitive mediators, its definition of stress has notable limitations, especially given that chronic stressors are subject to habituation processes that undermine a conceptual definition of stress as always representing an expectancy violation.

Continuing our review of the mediators of stress, the diathesis-stress model remains a widely used framework (Kendler, 2020) for explaining how dispositional vulnerabilities to stress increase susceptibility to a variety of disease processes, both physical (Banks & Kerns, 1996; Turk, 2002) and psychiatric (Burke & Elliott, 1999; S. M. Monroe & Simons, 1991; Walker & Diforio, 1997). In brief, these models propose that biological intermediaries modulate one's vulnerability to stress. As a result, individuals with greater vulnerability are more likely to exhibit heightened stress reactivity and develop illness following stress exposure (McKeever & Huff, 2003). However, some critics have argued that vulnerability genes may actually function as plasticity genes (Belsky et al., 2009), making individuals more biologically sensitive to both negative and positive environmental influences (Boyce & Ellis, 2005). Accordingly, these individuals may be more susceptible to both the harmful effects of stress and the beneficial effects of enriching environments (Belsky & Pluess, 2009). When applied to personality traits, a diathesis-stress model offers a compelling explanation for why personality traits are associated with health outcomes. However, there are multiple pathways for characterizing the personality-health relationship beyond stress reactivity, such as conferring increased risk of exposure to stressful events (Bolger & Zuckerman, 1995).

Research on adolescent stress responses led investigators to propose the psychophysiological reactivity model (Quas, 2011; Quas et al., 2006; Quas & Klemfuss, 2013). In brief, individuals who are more autonomically reactive to stress expend greater cognitive, emotional, and physiological resources to regulate that reactivity. This results in two major consequences: difficulty attending to important information (Kimhy et al., 2013; Park et al., 2012; Park, Van Bavel, et al., 2013; Park, Vasey, et al., 2013; Porges, 2014) and increased risk of illness due to immune system overload (K. A. Matthews & Woodall, 1988; McEwen & Seeman, 1999). In their approach, psychophysiological reactivity can represent a source of stress, a manifestation of stress, and a mediator of stress. As such, this model offers a comprehensive framework for understanding the full stress process and its links to personality and health outcomes.

While an exhaustive review of all psychological accounts of stress falls outside the scope of the current project, the key models discussed above illustrate the three conceptual domains of stress described earlier: sources of stress, manifestations of stress, and mediators of stress. This project is most strongly influenced by both the transactional model of stress and coping and the psychophysiological reactivity model, both of which offer frameworks that encompass all major components of the stress process. I adopt the view (Lazarus & Folkman, 1984) that stress is best conceptualized as an interactive and transactional process, mediated by traits, symptoms, biological intermediaries, and other influences. Importantly, this view is fully compatible with the psychophysiological reactivity model described above. As described earlier, traits represent a key mediator of stress related processes. Thus, the next section explores the complex relationship between personality and stress reactivity, which forms the core focus of the project outlined in chapter 4.

Personality and Stress Reactivity

Researchers broadly define stress reactivity as the tendency to respond with exaggerated arousal to a stressor, followed by impaired post-stressor recovery (P. G. Williams et al., 2011). However, such a broad definition, although conceptually simple, is vague and difficult to operationalize. For example, it does not clarify what constitutes exaggerated arousal or impaired recovery. Other researchers define the construct more concretely—as any change in a physiological parameter that deviates from a baseline value in response to acute stress, usually defined as an individual's resting physiological state (K. A. Matthews, 1986). Many early studies examining stress reactivity failed to collect and/or report recovery data or baseline resting data, leading to a relatively impoverished empirical understanding of the total stress reactivity process (Linden et al., 1997). Consequently, accurate assessment of physiological reactivity should include baseline assessments, stress-induced changes, and recovery assessments.

Studies show that stress reactivity is a stronger predictor of adverse health outcomes than subjective or objective reports of both the frequency and intensity of stress exposure (Kiecolt-Glaser et al., 2020). Some researchers have argued that physiological stress reactivity varies between individuals in a trait-like manner, suggestive of a stable predisposition (Kamarck & Lovallo, 2003), while others argue that significant variability in reactivity to lab-based stressors across time suggests that reactivity is more of a state than trait variable (Schwartz et al., 2003). For example, in a longitudinal diary study, investigators observed that stress reactivity varied significantly within individuals over time and that this variability was best predicted by perceived stress over the last month (Sliwinski et al., 2009). Whether stress reactivity is more appropriately conceptualized as a state or trait remains a topic of debate. However, the answer

ultimately has little bearing on its status as a predictor of future cardiac dysfunction, or the role personality traits may play in shaping it.

Two primary models attempt to explain stress reactivity: the psychophysiological reactivity model and the cognitive appraisal model (Lazarus & Folkman, 1984). The cognitive appraisal model argues that neural appraisals condition physiological reactivity, shaping top-down responses to perceived stress. This appraisal process prepares individuals to respond with exaggerated sensitivity to stressors (Lazarus & Folkman, 1987). However, the psychophysiological reactivity model argues that both top-down and bottom-up components of the CNS and ANS contribute to stress reactivity (Cacioppo et al., 1998). I argue that the psychophysiological reactivity model is more appropriate for understanding the role personality traits may play in shaping stress reactivity, particularly because personality traits impact all levels of psychological and biological functioning, across the CNS, ANS, and their integrated communication.

As described earlier, Boyce and Ellis (2005) have argued that stress reactivity is best characterized as increased biological sensitivity to context, pointing to evidence that individuals often exhibit increased reactivity to both positively and negatively valanced emotions and stimuli. Others conceptualize stress reactivity as a spectrum, with normative responses in the middle and pathological reactivity at both extremes (W. R. Lovallo, 2011). Both perspectives are supported by evidence showing that stress reactivity markers can be either elevated or suppressed in individuals with mental and physical health issues, as well as heightened levels of neuroticism (Bibbey et al., 2013; Oswald et al., 2006). However, such patterns are not always consistent from study to study (Coyle et al., 2020). Critically, biological markers of stress must

be understood contextually; heightened cortisol and blunted cortisol in response to stress can both represent unhealthy psychological/biological stress processes.

Researchers have long proposed that personality traits function as sources, moderators, and mediators of stress reactivity. Trait neuroticism is the personality factor most frequently hypothesized to predict stress reactivity. Hans Eysenck first formalized neuroticism as an independent dimension of personality (Eysenck, 1947). Since then, it has been a primary target in studies linking personality and stress, largely due to strong evidence that higher levels of trait neuroticism are associated with relatively worse mental (Malouff et al., 2005; Russo et al., 1997) and physical health outcomes (Mroczek et al., 2009; Shipley et al., 2007). Trait neuroticism is also a predictor of chronic illness and subjective perceptions of poorer health (Hudek-Knežević & Kardum, 2009), both common concomitants of the stress process. Longitudinal evidence suggests that increased trait neuroticism is causally linked to increased distress over time (Ormel & Wohlfarth, 1991), even when controlling for major life stressors. Consequently, investigators have overwhelmingly focused on trait neuroticism when examining the link between personality and stress reactivity. In a recent community sample study, investigators observed that neuroticism and extraversion predicted increased exposure to stressors. This greater exposure, in turn, moderated stress experience: more neurotic individuals exhibited greater daily stress reactivity (Kaurin et al., 2021). These results align with prior daily diary studies, which show that more neurotic individuals report more daily problems, react more intensely to stressors, experience greater mood spillover, and show stronger reactions to chronic stress (Suls et al., 1998; Suls & Martin, 2005). These approaches have provided compelling evidence that neuroticism is a robust personality candidate for predicting stress reactivity.

Many different mechanisms might link personality traits to increased stress experience. For example, personality traits might increase exposure to stressful events more than they increase reactivity to them, potentially confounding the relationship between personality and stress reactivity. However, in an influential study, researchers demonstrated that the majority of the variance between high and low neuroticism participants was explained by reactivity to stressors rather than mere exposure to stressors (Bolger & Schilling, 1991). Investigators have also observed a stronger relationship between daily stress and negative affect in more neurotic participants (Mroczek & Almeida, 2004), suggesting heightened emotional responses to stress. In a more recent longitudinal study of over 500 undergraduates, participants demonstrated low mean-level change and high rank-order stability in emotional stress reactivity. Additionally, intraindividual changes in stress reactivity were predicted by changes in neuroticism (Howland et al., 2017). While this study cannot confirm causality, the broader evidence suggests that stress reactivity is at least a core feature of trait neuroticism.

Furthermore, highly neurotic individuals often experience distress during primary and secondary stress appraisals. They are more prone to perceive events as stressful and more likely to doubt their ability to cope effectively (Gunthert et al., 1999). Stress appraisals are also an important source of stress reactivity, given that increased arousal in response to acute or chronic stress represents a global appraisal of a mismatch between an external event and perceived coping capacity. For example, in a large sample of 909 individuals, vagally mediated HRV during rest and psychological challenge was lower among those individuals with greater perceived stress (Sin et al., 2016). This was true regardless of the frequency of stressors, which showed no relationship to HRV. Brosschot et al. (2007) have also reported that daily time spent worrying is linked to reduced HRV both during the day and during sleep at night, indicating that

greater rumination on stress has physiological consequences for cardiovascular health even after the stress exposure has passed. This is in line with a body of research that convincingly demonstrates that more pessimistic appraisals mediate the relationship between trait neuroticism and impaired task performance in the context of laboratory stress tasks evoking a threat of punishment (Moeller & Robinson, 2010; M. D. Robinson et al., 2010; M. D. Robinson & Clore, 2007; O. J. Robinson et al., 2013). These findings suggest that trait neuroticism impacts all components of the stress process: the sources of stress, the mediators of stress, and the manifestations of stress.

Somewhat perplexingly, studies examining the relationship between non-cardiovascular stress reactivity and personality traits have yielded mixed results. For example, some researchers have observed no relationship between trait neuroticism and cortisol responses (Garcia-Banda et al., 2011; Habra et al., 2003; G. E. Miller et al., 1999; Xin et al., 2017). Others report the opposite: higher neuroticism is linked to blunted cortisol responses (Bibbey et al., 2013; Oswald et al., 2006). Resolving these discrepancies will require large, cross-cultural studies that use standardized stress induction protocols and compare multiple biological measures of stress reactivity directly.

Personality, Cardiovascular Stress Reactivity, and Cardiovascular Health

Cardiovascular stress reactivity is a narrower construct than stress reactivity, focused primarily on cardiovascular system changes in response to stress exposure. It has broadly been defined as any change in the cardiovascular system induced by acute or chronic stress exposure (Obrist et al., 1987), although most research relies on acute laboratory-based stressors to assess cardiovascular reactivity. The cardiovascular reactivity hypothesis posits a cascading chain of events through which stress-induced cardiovascular responses increase the risk of adverse health

outcomes. In brief, exaggerated autonomic responses to stress initiate and sustain a cascade of physiological processes that damage tissue, disrupt cardiovascular regulation, and ultimately contribute to disease (W. R. Lovallo & Gerin, 2003). Substantial evidence supports this theory, indicating that physiological reactivity to stress reliably predicts adverse cardiovascular outcomes (Kasagi et al., 1995; Liao et al., 1996; Menkes et al., 1989; Mona Bedi & Babbar, 2000; Schroeder et al., 2003). Individuals currently experiencing chronic psychosocial stress also present with reduced HRV, increased blood pressure, and reduced baroflex sensitivity; importantly, these autonomic markers of stress are correlated with subjective perceptions of stress, such that individuals who perceive greater daily stress have more pronounced autonomic reactivity (Lucini et al., 2005). Workers who perceive higher-than-usual occupational stress also show reduced HRV (Clays et al., 2011). Thus, I argue that the cardiovascular reactivity hypothesis is the most appropriate framework for understanding the link between stress reactivity and adverse cardiovascular health outcomes.

Indeed, investigators have long hypothesized that cardiovascular stress reactivity poses a critical risk factor for cardiovascular disease (Krantz & Manuck, 1984; Langewitz & Rüddel, 1989). Researchers have observed that comparatively elevated reactivity to lab-based stressors predicts future hypertension, increased diastolic blood pressure, and coronary artery disease (Treiber et al., 2003). However, some investigators have also observed reduced blood pressure responses to a stress task among participants more strongly characterized by negative affectivity (Childs et al., 2014). Additional studies have examined how subjective stress perceptions influence physiological stress reactivity. For example, using ecological momentary assessment and ambulatory ECG, Sloan and colleagues (1994) found that HRV declined as self-reported stress increased. Similarly, among physically fit adults, increased self-perceptions of emotional

stress in the last two weeks have predicted decreased HF-HRV, indicative of vagal withdrawal. These findings suggest that even short-term perceived stress can influence cardiovascular reactivity (Dishman et al., 2000). These findings show that stress reactivity affects multiple cardiovascular metrics, underscoring the importance of understanding which cardiac measures are most sensitive to stress.

Following the introduction of the Type A personality construct, researchers have actively investigated links between personality traits and cardiovascular health outcomes (Petticrew et al., 2012). This research eventually led to the identification of a new construct: Type D personality . In brief, this construct is characterized by two primary dimensions: the frequent experience of strong negative emotion, and the inhibition of emotional expression in social contexts (Denollet, 2005; Sher, 2005). Type D characteristics are primarily associated with mortality in coronary artery disease but have also been linked to increased mortality from both cardiac and non-cardiac causes (Denollet, 2000; Denollet et al., 1996). Type D personality characteristics have been linked to mental health symptoms, physical health complaints, declines in physical health status, and work-related stress (Mols & Denollet, 2010). Some researchers have argued that the use of avoidant coping strategies among Type D individuals may partly explain why this personality construct is associated with reductions in HRV and long-term cardiovascular health problems (Martin et al., 2011).

Furthermore, individuals with Type D traits also face elevated risk for coronary artery disease (Razzini et al., 2008). Similarly, O'Dell and colleagues (2011) found that Type D individuals face increased risk for all major adverse cardiovascular events. Moreover, patients with Type D traits often report larger declines in quality of life following cardiovascular surgery (Al-Ruzzeh et al., 2005). Pedersen and Denollet (2006) reviewed the literature on Type D and

cardiovascular outcomes, reporting a 2-5 fold increased risk of poor prognosis, impaired quality of life, and anxiety/depression symptoms, independent of disease severity and other risk factors. Extensions of this work have also demonstrated that the Type D personality construct is strongly heritable (Kupper et al., 2007; Li-Gao et al., 2021), replicable (Chapman et al., 2007), and predictive of some measures of cardiovascular reactivity to stressors (L. Williams et al., 2009).

Beyond the Type D personality construct, research consistently shows that three broad affective dispositions—depression, anxiety, and anger-hostility—increase cardiovascular risk (Suls, 2018). All three dispositions share considerable overlap, which has led some investigators to argue that a tendency towards general distress or negative affectivity (Watson & Clark, 1984) may be more useful as a predictor of future cardiovascular problems than any single affective disposition (Suls & Bunde, 2005). However, Suls (2018) found that anxiety and depression pose independent risks for cardiovascular outcomes. Some researchers have convincingly argued that the link between depression and cardiovascular problems involves bidirectional processes, such that aberrations in either cardiovascular function or mood increase the subsequent likelihood of developing the other problem (de Jonge & Roest, 2012). Diagnosed depression has been consistently linked to reduced heart rate variability (Carney et al., 1995, 2001; Carney & Freedland, 2009), as has clinical anxiety (Alvares et al., 2016; Carpeggiani et al., 2005; Chalmers et al., 2014; Miu et al., 2009; Watkins et al., 1998). Some have even suggested that increases in vagal tone can be used as a predictor of effective treatment response to antidepressants (Chambers & Allen, 2002).

The literature on anger-hostility is more mixed. Although studies have shown that anger-hostility predicts cardiovascular health outcomes (Booth-Kewley & Friedman, 1987; Brosschot & Thayer, 1998; Chida & Steptoe, 2009; Razzini et al., 2008; Sahoo et al., 2018), such

associations do not always replicate (Bleil et al., 2008). One explanation is that anger does not independently increase cardiovascular risk but does so indirectly through another factor, such as broader negative affectivity. This possibility highlights the significance of examining personality traits at a finer level of detail exploring how narrower facet traits may relate to cardiovascular outcomes.

Researchers have proposed the stress sensitization model as an explanatory link between affective disorders such as depression and anxiety and increased sensitivity to stress exposures (Stroud, 2018). This model argues that individuals become more sensitive to stressors over time, lowering the threshold of stress needed to trigger an affective disorder. After the first episode, less stress is required to increase the likelihood of another episode. This model builds on the earlier kindling hypothesis, which pointed to evidence that first episodes of affective disorders are often preceded by major increases in psychological stressors (Post, 1992). Understanding stress reactivity therefore requires accounting for the presence of affective disorders. Two systematic reviews indicate that individuals with depression (Hamilton & Alloy, 2016) or anxiety (Cheng et al., 2022) are more likely to exhibit reduced resting HRV.

A large body of literature has examined correlations between the Five-Factor personality traits and cardiovascular health, but findings remain inconsistent. Below, I summarize key empirical investigations linking stress reactivity to the Five-Factor traits before discussing possible reasons for these inconsistencies. A recent review (Sahoo et al., 2018) reports that hospitalizations and fatalities from CVD related causes occur significantly less among highly conscientious individuals (H. B. Lee et al., 2014) and that CVD-related hospitalizations are less frequent among individuals scoring high in trait openness to experience (Jokela et al., 2014).

The most comprehensive investigation to date is a meta-analysis examining stress reactivity and chronic psychosocial factors across 729 studies (Chida & Hamer, 2008). The authors found that hostility, aggression, and Type-A like behavior (competitive, ambitious, energetic) were associated with increased cardiovascular reactivity (heart rate or blood pressure), whereas anxiety, neuroticism, and negative affect were linked to decreased cardiovascular reactivity (Chida & Hamer, 2008). These findings highlight the value of examining facet traits within broader constructs such as neuroticism, which encompasses both negative affect and anger/hostility (J. A. Johnson, 2014). Puzzlingly, the results contradict previously referenced studies linking trait neuroticism to increased stress reactivity. These results underscore the importance of conceptual clarity in the terms researchers use; for example, stress reactivity is a much broader construct than cardiovascular stress reactivity, and both constructs can be measured with multiple methods.

Chida and Hamer (2008) also observed that general life stress, anxiety, neuroticism, and negative affect were linked to poorer cardiovascular recovery following stress induction. However, they found no clear connection between other psychosocial factors and baseline to stressor reactivity. These results emphasize the need to consider both the method of stress induction and the measure of stress reactivity used. For example, their study primarily examined heart rate and blood pressure as indices of cardiovascular stress reactivity, whereas a more recent literature review provides robust evidence that HRV reliably changes in response to stress and exhibits trait-like stability (H.-G. Kim et al., 2018).

Consequently, these findings have inspired investigators to explore associations between trait neuroticism, cardiovascular stress reactivity, and cardiovascular health. Outside the laboratory, researchers have established evidence that trait neuroticism predicts increased

reactivity to stress (as measured by increased heart rate, cortisol secretion, and subjective anxiety) among student teachers learning to lecture (Houtman & Bakker, 1991). In one small study sampling 36 individuals, researchers observed increased blood pressure and heart rate reactivity in participants scoring one standard deviation above the mean in trait neuroticism to multiple laboratory stressors (Schwebel & Suls, 1999). However, the researchers found no relationship between other measures of cardiovascular reactivity and trait neuroticism. Most empirical studies in this area tend to have larger sample sizes, so it is possible a relationship to other cardiovascular measures of reactivity was obscured due to the inability to detect smaller statistical effects.

However, even larger empirical investigations often yield a puzzling range of contradictory results. In a longitudinal study, investigators reported that trait neuroticism predicts increased pre-ejection period sensitivity to psychosocial lab stressors among adolescents (Evans et al., 2016). However, the remaining measures of stress reactivity analyzed in this sample of 327 Dutch youth showed no significant relationship with trait neuroticism. Another team observed that in a stress task involving the threat of an electric shock, sympathetically-mediated HRV increased only among participants with high trait neuroticism in the group exposed to the threat (A. Hansen & Johnsen, 2013). More recently, investigators (Mlynčeková et al., 2017) observed an association between neuroticism and increased sympathetically-mediated HRV during a baseline resting period. This association was not observed during the stress conditions; however, this finding was based on a much smaller sample size of 71. Some of these discrepancies are no doubt due to comparisons between adequately powered and inadequately powered studies, but such discrepancies cannot be exhaustively explained by differences in statistical power.

For example, among a sample of 1255 adults, higher levels of trait neuroticism predicted reduced vagally mediated heart rate at rest, and neuroticism predicted reductions in HRV during lab-based stress exposure (Čukić & Bates, 2015). Even after controlling for all other CVD risk factors, Čukić & Bates found trait neuroticism predicts reduced HRV, increased risk of cardiovascular disease, and increased risk of depression. However, in a more recent study, researchers observed no relationship between HRV and trait neuroticism, finding that only age and sex were significant predictors of baseline HRV among a sample of over 150,000 healthy adults and 14,000 children (Tegegne et al., 2018). Given the large sample sizes in both investigations, further research should examine facet traits cross-culturally to resolve these puzzling contradictions.

Furthermore, trait neuroticism has a strong theoretical overlap with the Type D personality construct, given that both measures assess a general propensity towards psychological distress (Denollet et al., 2010). Evidence suggests that the negative affectivity dimension of Type D correlates strongly with trait neuroticism at 0.74 and the social inhibition dimension correlates negatively with trait extraversion at -.61 (De Fruyt & Denollet, 2002). Unsurprisingly, a wealth of research has also explored the link between trait neuroticism and risk for cardiovascular mortality. Researchers have also observed that a one standard deviation increase in trait neuroticism predicts a 2.5 fold increased risk for death by cardiovascular disease among a sample of adults from the UK Biobank (Shipley et al., 2007). This study illustrates the significance of neuroticism as a broad index of health (Lahey, 2009). Investigators have also observed increased CVD-related mortality risk among women with high trait neuroticism. This effect was more pronounced among women lower in socioeconomic status, suggesting that chronic stress more profoundly impacts individuals with higher neuroticism (Hagger-Johnson et

al., 2012). However, not all large-scale studies have observed similar results; for example, a longitudinal study of approximately 45,000 Japanese civilians found no significant increase in the relative risk of developing ischemic heart disease when using the neuroticism scale of the Eysenck Personality Questionnaire (Nakaya et al., 2005). Critically, the outcome variable (ischemic heart disease) in the Nakaya investigation is narrower than all cardiovascular-related mortalities examined in the Shipley investigation, once again underscoring the principle that personality associations with cardiovascular dysfunction tend to be highly contingent on the methods of assessment used in defining personality constructs and cardiovascular outcomes.

Altogether, the literature clearly suggests that personality traits can serve as useful predictors of stress reactivity. Broadly, personality traits that tend towards a dimension of general distress are most frequently associated with stress reactivity, especially cardiovascular stress reactivity. Trait neuroticism has been the most commonly investigated trait linked to cardiovascular stress reactivity, although findings in this area remain highly mixed. I argue that these inconsistent results may be a consequence of comparisons between studies that use different metrics of physiological stress reactivity and different methods of stress induction, given that studies with larger sample sizes frequently yield different results . Therefore, investigations that compare several stress tasks, stress metrics, and personality metrics can fill a critical gap in the literature. Thus, investigators can perform more direct analyses of personality by stressor by physiological response comparisons. Such studies will enable researchers to develop a more precise characterization of the complex relationship between personality and physiological reactivity. In the next chapter, I offer a more detailed description of the ANS and its role in shaping cardiovascular stress responses, as well as an extended review of HRV metrics

and theories addressing the functional role of HRV as a measure of cardiovascular stress reactivity.

Chapter 3: Autonomic Function, Cardiac Vagal Tone (CVT), and HRV

The ANS is a critical nexus in the body's stress response. As metabolic demands change, the cardiovascular system also plays an essential role in the expression and regulation of ANS activity. French physiologist Claude Bernard was among the first scientists to argue that the brain and heart are closely interconnected via the mediating activity of the vagus nerve (Bernard, 1867), anticipating many key insights that would prove foundational for present day autonomic neuroscience. This interrelationship between brain and heart is necessary for the organism to adaptively shift its behavior in response to changing environmental demands (Liu et al., 2022). In this chapter, I review the physiology of the cardiovascular system, the role it plays in the ANS, and ways it can function adaptively and maladaptively. I also explore the use of HRV as a measure of cardiovascular stress and theories that establish a mechanistic link between HRV, stress, and health.

The Relationship between Vagal Tone and HRV

The ANS consists of two primary branches: the parasympathetic (PNS) and the sympathetic branch (SNS), a division first characterized in 1921 (Langley). Shortly before this description, Cannon broadened the notion of homeostasis to include the role of self-organizing systems in its development and maintenance (Cannon, 1915). The idea that such systems are fundamental to regulating internal physiological states is critical to research on HRV, much of which is based on the observation that increased HRV is linked to more stable homeostatic function (Goldberger, 1997). Importantly, variability in the heart rhythm is maintained by contributions from both the PNS and SNS.

Both branches of the ANS operate in a dynamic balance, competing for relative dominance (Yasuma & Hayano, 2004). The ANS is fundamental in regulating homeostatic

function (Porges, 1992) and facilitating the body's stress response (B. H. Friedman & Thayer, 1998). The vagus nerve, the longest cranial nerve, innervates the internal viscera and modulates cardiovascular activity. Originating in the medulla, it innervates bodily structures in the neck, thorax, and abdomen, and influences cardiac and digestive functions (Câmara & Griessenauer, 2015, p. 27). Additionally, the vagus nerve controls aspects of respiration (Bozler & Burch, 1951). Thus, cardiac and respiratory functions are closely linked to vagal activity.

Both the PNS and SNS originate in the brainstem (B. K. Johnson, 2018) and regulate a variety of organs throughout the body (McCorry, 2007). As a general principle, the PNS promotes growth and restoration while the SNS increases metabolic output to handle stressors (Porges, 1992). Both systems have reciprocal innervation that coordinates their responses to shifting environmental demands (Kollai & Koizumi, 1979). Their relationship is one of antagonistic processing: the PNS and SNS exert opposing effects on similar target organs to maintain a dynamic homeostatic balance (Gibbons, 2019). When assessing autonomic function, researchers should select metrics that reflect the activity of both branches, since behavior results from their dynamic interplay.

Cardiac vagal tone (CVT) refers to the parasympathetic contribution to regulating the heart rhythm (Katona & Jih, 1975). CVT has been associated with a wide variety of positive (Balzarotti et al., 2017; Souza et al., 2007) and negative (Beauchaine & Thayer, 2015; Dishman et al., 2000; Forkmann et al., 2016; Wekenborg et al., 2019) health outcomes. Researchers typically measure CVT indirectly through HRV metrics; however, it is misleading to treat HRV as a direct measure of CVT. Invasive measurements of vagal tone in rat models show only a weak correlation between vagal modulation of the heart rhythm and HRV (Marmerstein et al., 2021). This is partly because HRV reflects contributions from both parasympathetic and

sympathetic branches, as well as influences from the respiratory system, endocrine system, baroreceptors, and chemoreceptors (Shaffer & Ginsberg, 2017). Nonetheless, in human populations, HRV in synchrony with respiration is the only non-invasive metric that can indirectly measure parasympathetic contributions to the heart rhythm. Conversely, low-frequency components derived from spectral measures of HRV can also indirectly assess sympathetically mediated influences on the heart rhythm. Importantly, reductions in vagally mediated HRV predict increased risk of cardiovascular disease and mortality. For example, when researchers combined several indirect indices of vagal function, they observed that reductions in these metrics collectively predicted the risk of cardiovascular disease and mortality (Thayer & Lane, 2007). In this project, several HRV metrics were selected as indirect indices of vagally mediated and sympathetically mediated contributions to the heart rhythm with the understanding that the rhythm at any moment is comprised of multiple physiological sources.

Hering (1910) first clearly described the relationship between respiratory sinus arrhythmia (RSA) and vagal tone, noting that the vagus nerve modulates heart rate changes that occur in synchrony with the respiratory cycle. Later on, other investigators more accurately assessed parasympathetic control of the heart rhythm in dogs by controlling respiration rate (Katona & Jih, 1975). This work strengthened the case for using HRV as an index of parasympathetic regulation of the heart rhythm. Shortly thereafter, investigators demonstrated that different frequency levels in the power spectrum of HRV correspond to varying levels of sympathetic and parasympathetic activity (Akselrod et al., 1981).

I now offer a brief review of the physiology of the cardiovascular rhythm to better clarify how HRV is a useful index of cardiovascular stress reactivity. The cardiovascular system pumps blood throughout the body via two circuits: a pulmonary circuit and a systemic circuit (Levick,

2013). The pulmonary circuit primarily pumps blood through the lungs in order to oxygenate it, while the systemic circuit primarily delivers oxygen to peripheral tissues. Both circuits are essential for removing carbon dioxide from the bloodstream (Levick, 2013). Pacemaker cells, or rhythm generators, initiate depolarization and repolarization via the sinoatrial node, causing contractile cells to contract during depolarization and myocardial cells to relax during repolarization (Foëx & Higham, 2004). Multiple physiological sources regulate the heart rhythm, including the opposing branches of the ANS: the PNS and the SNS. Sympathetic nerve activity increases the excitability of the sino-atrial node (SA node), raising heart rate, while parasympathetically mediated nerve activity decreases SA node excitability, lowering heart rate (Joung & Chen, 2015).

However, it is important to note that vagal tone is modulated by both CNS and ANS mechanisms, including several distinct brainstem processes (Farmer et al., 2016) that contribute to regulating heart and respiratory rhythms. Alongside the cardiovascular system, the respiratory system facilitates gas exchange in the alveoli of the lungs, coordinated by CNS mechanisms. Oxygenated blood is then pumped by the cardiovascular system to the capillaries, allowing oxygen to diffuse into body tissues and carbon dioxide to enter the bloodstream. The blood returns to the heart, where it is pumped through the pulmonary system in preparation for gas exchange (Feldman & Ellenberger, 1988). Respiratory and cardiac activity compose a single integrated system, known as the cardiorespiratory system (Thayer et al., 2011). Therefore, it is critical to assess cardiac and respiratory activity concurrently, especially because they reciprocally influence one another to meet the body's energy demands (Hoffman, 2024). Although an exhaustive account of cardiorespiratory coupling is beyond the scope of this project, these mechanisms have been well described in detail elsewhere (Dick et al., 2014).

As described previously, CVT indirectly measures the vagus nerve's role in regulating cardiac rhythm. Over multiple decades, researchers have proposed various theories about the functional purpose of CVT. Stephen Porges argues that CVT is best understood as a physiological marker of stress vulnerability (Porges, 1992, 1995a), such that low CVT increases susceptibility to the effects of stress. In contrast, Friedman has suggested that CVT better reflects nervous system flexibility (B. H. Friedman, 2007), while others propose that it represents motivational and behavioral capacity to respond to stressors rather than vulnerability (Beauchaine, 2001). For example, individuals with low resting CVT, as measured by RSA, often show impaired post-stress recovery (Weber et al., 2010). However, the most accurate functional account of CVT still remains a subject of research and debate (Mastromatteo et al., 2024).

RSA is the variability of the heart rhythm in synchrony with respiration (Berntson et al., 1993) and is the most frequently used measure of cardiac vagal activity (Grossman, 2024). It is based on the observation that heart rhythms decrease during exhalation and increase during inhalation (Anrep et al., 1997a, 1997b). RSA is a common method for assessing HRV and indirectly estimating vagal tone (Berntson et al., 1993). Both HRV and RSA reflect the complex, dynamic integration of the CNS and ANS. Accordingly, researchers use numerous methods to measure them, which I examine in the next section.

Metrics of HRV

The idea that variability in the heart rhythm may be linked to health has been part of medicine since Ancient Greece and Ancient China (Ernst, 2017b); however, pulse rate variability could not be adequately measured until the development of an accurate pulse watch in 1707 (Floyer, 1707). Soon after, Rev. Stephen Hales observed that fluctuations in pulse rate varied at different points in the respiratory cycle (Hales, 1733). Over a century later, Carl Ludwig

discovered what would later be described as RSA (Billman, 2011). Using dogs as his model organism, Ludwig observed that pulse rate reliably increased during inspiration and decreased during expiration (Ludwig, 1847). Much later, Einthoven (1895) employed the galvanometer to produce continuous recordings of the heart rhythm (Billman, 2011). By the 1960s, portable recording devices made ECGs readily obtainable, augmenting the capacity to study the relationship between changes in the beat-to-beat interval of the cardiac rhythm and health (Billman, 2011).

As signal processing techniques improved, the capacity to detect subtle fluctuations and parameters of the beat-to-beat interval also improved. Initially, researchers calculated time-domain metrics, such as the mean or squared difference between successive beats. In the 1970s, multiple groups began applying power spectral analysis to the heart rhythm as well (Billman, 2011). More recently, investigators have successfully used nonlinear mathematical techniques originating from a revolution in physics known as chaos theory to analyze the heart rhythm (Goldberger, 1990; Goldberger & West, 1987).

A standard ECG signal consists of multiple interlocking components: a P wave, the QRS complex, and a T wave. The QRS comprises a Q wave, an R wave, and an S wave (Dupre et al., 2005). The ECG signal begins with the P wave, which corresponds to atrial depolarization due to an action potential initiated by the SA node (De Luna et al., 2006). This is followed by the QRS complex, representing the electrical impulse spreading through the ventricles, which is termed ventricular depolarization (Strauss & Selvester, 2009). Shortly thereafter, ventricular repolarization occurs, initiating the T wave (Bagliani et al., 2017). HRV is commonly measured by first calculating the intervals between successive R waves (R-R interval) and then applying

one of several measures to assess variability between these intervals (Shaffer & Ginsberg, 2017). I review the primary metrics for calculating HRV below.

Investigators use numerous indices to measure HRV, each with different physiological antecedents (Rajendra Acharya et al., 2006). Most studies employ three categories of HRV parameters: time-domain measures, frequency-domain measures, and non-linear indices (Laborde et al., 2017). Time-domain measures calculate variability in the interbeat interval (IBI), the time between sequential heartbeats, and are typically expressed in original units or as the natural logarithm of those units. Frequency-domain measures estimate the distribution of absolute or relative power into different frequency bands, each influenced by different physiological origins. In this context, power refers to the signal energy observed within a frequency band. Absolute power is expressed in milliseconds squared per hertz (ms^2/Hz) (Shaffer & Ginsberg, 2017); Relative power, by contrast, is calculated as a percentage of total HRV power, dividing the absolute power for a specific frequency band by the summed absolute power of low-frequency and high-frequency bands (Shaffer & Ginsberg, 2017). Non-linear indices quantify the unpredictability of the ECG time series and include measures such as approximate entropy, sample entropy, short-term signal fluctuations, and long-term signal fluctuations (Shaffer & Ginsberg, 2017). Given the abundance of existing HRV metrics, inconsistencies in the literature are unsurprising. Notably, intercorrelations between different HRV metrics tend to be exceptionally high during ECG recordings taken at rest but are less stable during stress tasks (J. J. Allen et al., 2007). These intercorrelations suggest shared patterns of cardiovascular activity that underscore the importance of including multiple HRV metrics in research studies.

A common time-domain method of assessing HRV is the root mean square of successive differences (RMSSD), which researchers widely regard as reflecting vagal contributions to the heart rhythm (Stein et al., 1994). To calculate RMSSD, the IBI for each heartbeat is determined, squared, averaged, and then square-root transformed (Shaffer & Ginsberg, 2017). This measure of HRV is thought to reflect the integrity of vagus nerve-mediated autonomic control of the heart (DeGiorgio et al., 2010). Numerous investigators have reported reliable decreases in RMSSD values after stress induction (for a meta-analysis, see Castaldo et al., 2015). These results support RMSSD's utility as an indirect index of vagal tone in studies of stress-related processes among healthy adults.

Two common frequency-domain measures are absolute low-frequency power (LF-HRV), and absolute high-frequency power (HF-HRV), each reflecting different aspects of physiological function. These frequency bands are generally defined as follows: 0.04-0.10 Hz (LF-HRV); 0.15-0.40 Hz (HF-HRV) (Shaffer & Ginsberg, 2017). HF-HRV is thought to reflect CVT, whereas LF-HRV reflects a mix of sympathetic activity, baroflex activity, and vagal activity (Laborde et al., 2017). Although LF-HRV is not a direct index of sympathetically mediated activity alone (Billman, 2013; Houle & Billman, 1999; Reyes del Paso et al., 2013), it remains a useful HRV metric for analyzing stress-related changes in cardiac activity because LF-HRV frequently increases in response to stress tasks (Soliemanifar et al., 2018). Importantly, researchers have argued that increased sympathetic activity is indicated by a relative shift towards the LF component of the cardiovascular rhythm, arguing that increases in LF-HRV serve as an indirect measure of stress-related changes to the cardiovascular rhythm (Malliani et al., 1991). Meta-analytic work also indicates that HF-HRV decreases after stress induction (Castaldo et al., 2015). In the project described in chapter 4, I utilized RMSSD, LF-HRV, and HF-HRV to

capture both frequency and time-domain aspects of HRV. These metrics were most commonly reported in research studies involving HRV.

Theories of HRV

HRV has been a subject of sustained interest among physicians, psychologists, and physiologists since the advent of reliable measurement techniques. Early experimental work in the 1980s and 1990s repeatedly demonstrated that HRV strongly predicts adverse outcomes among cardiac patients (Bigger Jr et al., 1993, 1995). Massive reductions in HRV reliably precede adverse cardiac outcomes, including arrhythmic death (Farrell et al., 1991; Kleiger et al., 1987; La Rovere et al., 2003; Villareal et al., 2002), increased cardiovascular morbidity, and first-episode cardiovascular events among individuals without known cardiovascular disease (Hillebrand et al., 2013). These findings have led some researchers to recommend incorporating HRV recordings as part of cardiovascular risk assessment protocols (Huikuri & Stein, 2013).

Several theories have attempted to explain HRV's role in disease, health, and stress reactivity. Polyvagal theory, first proposed in the 1990s (Porges, 1995a), conceptualizes vagus nerve function essential to facilitating ANS activity and behavior. Porges argues that the evolution of the mammalian ANS provided the neurophysiological substrates for the affective processes influencing social behavior. This perspective emphasizes the phylogenetic origins of nervous system components that regulate social and defensive behaviors (Porges, 1995b, 1998, 2001). According to this theory, the physiological state of the organism constrains the range of possible behaviors and psychological experiences. Adaptive social functioning is directly related to and partly caused by higher vagal tone during social encounters and reduced sympathetically mediated ANS activity. Polyvagal theory predicts that higher vagal tone should generally be associated with better social and behavioral functioning (Porges, 2007). Meta-analytic evidence

supports the assumption that baseline vagal tone predicts effective emotion regulation strategies (Balzarotti et al., 2017), and prosocial behavior among children (J. G. Miller et al., 2017). However, some evidence indicates that higher resting vagal tone is not linked to more adaptive social functioning in older women, contradicting polyvagal theory's predictions (Egizio et al., 2008). Additionally, some scholars suggest a quadratic relationship between vagal tone and adaptive functioning, whereby increases in vagal tone enhance adaptive functioning only up to a point, beyond which the association plateaus (Spangler et al., 2015).

Beyond polyvagal theory, Grossman and Taylor (2007) proposed a biological-behavioral model suggesting that HRV reflects the body's attempt to synchronize respiratory and cardiovascular processes during metabolic and behavioral changes. Other investigators have examined the role of breathing in influencing CVT (Lehrer & Gevirtz, 2014), citing evidence that slow-paced breathing tends to increase CVT and benefit both mental and physical health (McCraty & Childre, 2010). Lehrer and colleagues' resonance frequency model posits that delays in the baroreflex system's feedback loops create an individual's resonance frequency (Lehrer, 2013). When individuals breathe at this frequency, they align three primary oscillators: the baroreflex, heart rate, and blood pressure, resulting in more efficient gas exchange and oxygen saturation (Yasuma & Hayano, 2004). This principle underlies biofeedback training techniques designed to increase HRV (Lehrer, 2007).

As described previously, an appropriate dynamic balance between the rhythms of the CNS and ANS is essential for responding effectively to environmental demands and finding the optimal balance between behavioral, cognitive, and affective extremes. Some scholars propose that HRV reflects the capacity to regulate emotional responses to shifting environmental demands (Appelhans & Luecken, 2006). In line with this view, the neurovisceral integration

model (NIM) offers an increasingly common alternative framework for understanding the link between HRV, stress, and health. The NIM posits that a set of brain regions between the prefrontal cortex, the central autonomic network (Benarroch, 1993), and the vagus nerve operate in a dynamical system that shapes the functional capacity of the organism to flexibly respond to the shifting demands of the environment (Thayer & Lane, 2000). The brain regions regulating these functions include the anterior cingulate cortices, insular cortices, ventromedial prefrontal cortices, central nucleus of the amygdala, the paraventricular nuclei of the hypothalamus, the periaqueductal gray matter, and the ventrolateral and ventromedial medulla. HRV is viewed as an index of the degree of integration between the CNS and ANS. According to Thayer and colleagues (2009), individual differences in HRV provide a measure of variation in the functional capacity to adapt to environmental demand. From this perspective, HF-HRV fluctuations represent parasympathetic regulation of the heart rhythm, whereas fluctuations in LF-HRV reflect sympathetic regulation.

Numerous studies support the NIM, demonstrating clear relationships between cognitive deficits (Waldstein et al., 2005, 2008), affective deficits (B. H. Friedman & Thayer, 1998, 1998; Spyer, 1989), and individual differences in HRV and CVT. Thayer and colleagues have argued that CVT does not have a linear effect on adaptive functioning; rather, evidence suggests that moderate CVT is most strongly associated with well-being and adaptive function (Kogan et al., 2013; Miller et al., 2017; Spangler et al., 2015). In other words, the relationship between CVT and adaptive function appears quadratic, with individuals who have moderate levels of resting CVT exhibiting greater flexibility in responding to environmental demands than those with either exceptionally low or high CVT.

Broadly speaking, most researchers agree that reductions in heart rhythm variability represent a pathological aberration (Ernst, 2017a). Optimal homeostatic function fundamentally requires moment-to-moment variability in the functional rhythms that support autonomic activity (Shaffer et al., 2014). For the purposes of this investigation, I contend that the NIM offers the most comprehensive theoretical account of the relationship between HRV, stress reactivity, and health. The model explains a broad range of empirical findings on HRV and behavioral function by detailing how the CNS and ANS work together to facilitate adaptive responses. Consequently, the results of this investigation will be interpreted primarily through the lens of this framework.

In sum, the best available non-invasive measure of CVT in humans is RSA, although it remains an imperfect index due to the diverse physiological sources contributing to CVT (Stein, 2005). Autonomic reactivity via HRV and RSA can be measured easily, and generally demonstrates good reliability; however, its reliability tends to decrease during stress tasks (Bertsch et al., 2012; Kasprowicz et al., 1990; Salomon, 2005; Sloan et al., 1995). Regardless, a robust body of evidence consistently demonstrates that these physiological parameters are effective for assessing responses to acute stressors. Accordingly, they represent a valid choice for evaluating cardiovascular reactivity in response to acute laboratory-based stressors, which will serve as the primary methodology described in chapter 4.

Chapter 4: Personality and Cardiovascular Stress Reactivity

Cardiovascular disease represents one of the most significant public health challenges facing the United States. CDC data indicate that more than 944,000 Americans die each year due to cardiovascular disease and stroke, more than 1 in 3 deaths annually (Centers for Disease Control and Prevention, National center for Health Statistics, 2024). Recent estimates suggest that cardiovascular diseases cost the healthcare system over 254 billion USD annually, as well as costing 168 billion USD in job productivity losses (Tsao et al., 2023). The American Heart Association estimates that health care related costs of cardiovascular disease risk factors will triple from 2020 to 2050 and costs associated with cardiovascular disease will quadruple over the same time period (Kazi et al., 2024). Further, in representative samples, few Americans meet standard metrics of cardiovascular health, such as recommended physical activity levels, normal blood pressure, healthy blood glucose, optimal total cholesterol levels, and appropriate weight (Yang et al., 2012). Concerningly, only a small minority of the population have no cardiovascular disease risk factors and more than 70% of at-risk individuals present with multiple risk factors (Dahlöf, 2010). These alarming data highlight the pressing need for a continued research effort to understand, predict, and prevent cardiovascular disease risk factors.

A wealth of literature has examined a range of cardiovascular risk factors (for a summary, see Bays et al., 2021). However, one primary risk factor for cardiovascular disease is stress reactivity, broadly defined as both exaggerated reactivity to stressors and impaired post-stressor recovery (Lambert et al., 2010). Meta-analytic work suggests that greater cardiovascular responses to mental stress in laboratory settings are linked to subsequent cardiovascular risk status, including elevated blood pressure, hypertension, increased left ventricular mass, subclinical atherosclerosis, and clinical cardiac events (Chida & Steptoe, 2010). Importantly, as

an index of physiological stress reactivity, HRV robustly predicts sudden mortality (Dekker et al., 1997, 2000; Maheshwari et al., 2016) and other adverse cardiovascular outcomes (Buccelletti et al., 2009). This evidence indicates that individual differences in stress responsivity play a role in shaping both present and future cardiovascular health. Consequently, numerous scholars have examined individual factors that influence stress reactivity.

Personality and Physiological Stress Reactivity

Personality traits have long been proposed as a risk factor for cardiovascular disease risk (Arnetz & Fjellner, 1986; Booth-Kewley & Friedman, 1987; Denollet et al., 2010; Koelsch et al., 2012; G. E. Miller et al., 1999; Razzini et al., 2008; Sher, 2005; Steptoe & Molloy, 2007). However, the results of these investigations have been largely inconsistent across studies, likely due to inconsistencies in methods of personality assessment and in the specific cardiac outcomes examined.

Nonetheless, stress research has generally reflected the belief that stress responses are primarily shaped by enduring traits of the individual (P.G. Williams et al., 2011). A recent review of 38 studies supports this basic assumption (Soliemanifar et al., 2018). The responsiveness of physiological systems to stress is often correlated with personality traits that serve as markers of general distress (Denollet et al., 2010). Numerous studies have examined diverse personality models in this context, including the Type A and Type B personality typology (Billing & Steverson, 2013), the Type D personality type (Sher, 2005), the Five-Factor Model of Personality (Bibbey et al., 2013), and the Eysenck Personality Model (Marchant-Haycox & Wilson, 1992). The associations between personality traits and stress reactivity are generally robust; however, inconsistencies across studies suggest that stress responsivity may vary depending on both personality traits and the specific methods used for stress induction.

Some evidence also suggests that the novelty of a stress exposure can mask the impact of personality differences on stress responses, although aggregating stress responses over time seems to reverse this effect (Pruessner et al., 1997). These findings highlight the need for a clearer understanding of the relationship between personality traits and cardiovascular outcomes. Personality represents an important measure of individual difference in relationship to physiological outcomes. In particular, personality traits are relatively stable in adulthood (Bleidorn et al., 2022), are highly heritable compared to other measures of individual differences (Power & Pluess, 2015), and are generally predictive of long-term health outcomes (Strickhouser et al., 2017). In addition, family history of cardiovascular disease predicts stress reactivity to lab-based stressors (Wright et al., 2007), underscoring the intertwined relationship between cardiovascular health, stress reactivity, and heritable aspects of health. These data suggest that personality traits may play an important role in understanding individual differences in stress responsiveness and in shaping cardiovascular health outcomes.

Researchers have also examined the role of the five-factor personality model in relation to cardiac outcomes. Studies generally support the view that traits such as conscientiousness, agreeableness, and openness to experience are cardioprotective, conferring a reduced risk of adverse cardiovascular outcomes (Sahoo et al., 2018). In contrast, trait neuroticism has been linked to elevated cardiovascular disease risk. Data from a 21-year UK cohort study indicate a 12% increased risk of death from cardiovascular disease for every one standard deviation increase in trait neuroticism (Shipley et al., 2007). These findings have led scholars to describe trait neuroticism as a notable public health concern (Lahey, 2009). Importantly, investigators have examined the trait structure of type D personality in relation to the five-factor model, finding strong correlations between negative affectivity, social inhibition, and trait neuroticism

(De Fruyt & Denollet, 2002). These relationships between neuroticism, type D, and cardiovascular risk illustrate the importance of understanding the link between personality and stress reactivity. Specifically, trait neuroticism serves as a marker of general distress, influencing stress reactivity (Soliemanifar et al., 2018), and increasing the risk of adverse cardiac outcomes.

Substantial evidence supports the robustness of the five-factor model of personality (Costa & McCrae, 1992; McCrae & Costa, 1987; McCrae & John, 1992). Personality psychologists have argued that it represents the universal basic structure of all personality traits (McCrae & Costa, 1997). Moreover, extensive research indicates that the five-factor model reliably predicts stress responses (Penley & Tomaka, 2002). In particular, large sample studies consistently associate trait neuroticism with avoidance coping, hostility, and withdrawal in response to psychological stress (McCrae & Costa Jr, 1986). Accordingly, trait neuroticism constitutes a useful and fundamental marker to study the relationship between personality and cardiovascular stress reactivity.

Stress Reactivity, the CPT, and the MST

A substantial body of research has investigated relationships between personality traits and biological reactivity to stress, employing a wide range of stress responses, personality measures, and stress-induction paradigms (Soliemanifar et al., 2018). Common measures of stress reactivity include salivary cortisol levels, blood cortisol, HRV, and RSA (Soliemanifar et al., 2018). However, as discussed earlier, the relationship between Five-Factor personality traits and stress responses has been inconsistent across studies. This variability suggests that both the method of stress measurement and the type of stress induction significantly influence observed associations between personality and stress reactivity (Soliemanifar et al., 2018).

Two widely used methods for inducing stress are the CPT (W. Lovallo, 1975) and the MST (Jern et al., 1991). The MST typically involves the completion of math problems that either increase in difficulty/complexity or follow a systematic pattern. The CPT typically involves a cold water exposure in which participants insert a limb, typically a hand, into an apparatus that chills the water to a consistent temperature. Multiple versions of this task exist, and it is a well-documented paradigm for both pain tolerance and stress induction studies (Baeyer et al., 2005). The The MST induces significant increases in heart rate (Turner et al., 1987) and systolic blood pressure (Specchia et al., 1984). Evidence linking mental arithmetic to HRV is somewhat less consistent. For example, some investigators have reported decreases in resting state HRV in response to mental arithmetic in females, but not males (Sharpley et al., 2000). Such evidence is potentially indicative that gender might moderate or confound HRV responses- an interpretation that could be supported by evidence for gender differences in HRV (Huang et al., 2013; Jensen-Urstad et al., 1997; Pushpanathan et al., 2016). Importantly, such findings could also represent a stereotype threat, such that mental arithmetic is more stressful for females compared to males because of existing stereotypes around math performance (Spencer et al., 1999). Other scholars have observed significant increases in LF-HRV during mental arithmetic, indicating relatively greater sympathetic activation (Bernardi et al., 2000; Berntson et al., 1996; Berntson & Cacioppo, 2004; Singh et al., 2019). Most published studies have reported decreases in HF-HRV and RMSSD during mental arithmetic, accompanied by increases in LF-HRV (Borovkova et al., 2023; Sánchez-Hechavarría et al., 2019; Traina et al., 2011; Vurgun et al., 2023; Yu & Zhang, 2012).

The CPT typically enhances sympathetic outflow and induces peripheral vasoconstriction by activating the body's thermo- and nociceptive systems (Hilz et al., 2002). Consequently, the

CPT induces sympathetically mediated increases in LF-HRV and parasympathetic withdrawal, reflected in decreases in HF-HRV (Ghiasi et al., 2020; Mourot et al., 2009). One team observed significant increases in blood pressure, elevations in sympathetically mediated HRV, and reductions in parasympathetically mediated HRV during a six minute hand immersion in cold water (Wirch et al., 2006), demonstrating the CPT's effectiveness in eliciting cardiovascular reactivity. The CPT also appears sensitive to reactivity differences influenced by personality traits; for example, investigators examined the Type D personality construct, finding that both men and women characterized by Type D behavioral patterns had increased pre-ejection period (indicative of larger sympathetic activity) and increased RSA during the CPT, suggesting that individuals with high baseline stress levels respond aberrantly to stress tasks (Kupper et al., 2013). Some exceptions are also present as well; for example, a 2007 study in which participants completed attention tasks, speech tasks, and a cold pressor task found no significant differences in any frequency components from baseline to the CPT (Moses et al., 2007). Forte and colleagues (2022) have recently argued that HRV represents a marker for indexing the autonomic reactivity to pain stimuli on the basis of a systematic review of the literature, including a large sampling of studies using the CPT.

Several early studies directly compared cold exposure stress tasks against mental arithmetic stress tasks. Findings generally indicate that both tasks activate sympathetic activity and reduce parasympathetic activity, but to differing degrees. For example, researchers have reported increased diastolic blood pressure, heart rate, and pre-ejection period among a sample of male college students during the CPT compared to a verbal mental arithmetic task (M. T. Allen et al., 1987). In a subsequent study with a sample of fifty-one college students, the same investigators found significant RSA decreases during non-verbal mental arithmetic relative to the

CPT (M. T. Allen & Crowell, 1989), underscoring the influence of speech tasks on autonomic measures associated with respiration (Beda et al., 2007). Accordingly, the present project excludes speech-based tasks, although these are often incorporated into other well-validated stress paradigms (A. P. Allen et al., 2014; Seddon et al., 2020). In another investigation, researchers compared a MST with a speech task designed to mimic the MST without requiring participants to perform the calculations and found significantly greater cardiovascular reactivity to the MST (Brown et al., 1988).

Few studies have directly compared the CPT and MST while examining the role of personality in stress responses. In 2007, researchers investigated this question by assessing cardiovascular stress reactivity in 87 men selected from blood pressure screenings, specifically those at the 1st, 50th, and 99th percentiles (Flaa et al., 2007). Participants completed both tasks, and the researchers assessed fifteen personality traits from the Karolinska Scale of Personality. Stress reactivity was indexed using systolic and diastolic blood pressure, heart rate, and blood concentrations of epinephrine and norepinephrine.

Their regression analyses revealed distinct personality predictors for each stressor. For the CPT, irritability, muscular tension, detachment, psychasthenia, and somatic anxiety were significant explanatory variables. For the MST, verbal aggression and detachment emerged as significant predictors. These results suggest that not all facets of dispositional negativity relate equally to stress reactivity, underscoring the importance of using personality assessments with multiple domains or facets. Although informative, this work highlights the need to compare stress-induction methods within the framework of the Five-Factor model of personality. Factor analysis of the Karolinska Personality scale indicates strong overlap between negative emotionality and trait neuroticism (Ortet et al., 2002). The differential associations observed

across facets point to the value of higher-resolution trait assessments with broad scholarly acceptance, such as the International Pool and Inventory of Personality 120 (IPIP 120) (Johnson, 2014), which measures specific subcomponents of the Five-Factor traits that may uniquely predict stress reactivity.

As noted earlier, both experimental and daily-life stress reactivity are consistently predicted by the Five-Factor personality trait of neuroticism (Ode et al., 2010). A systematic review further indicates that stress inductions using the MST elicit larger changes in HF-HRV and LF-HRV (Soliemanifar et al., 2018) than other common paradigms, including the CPT. However, these conclusions are based on between-subjects comparisons across different studies. Supporting this approach, other investigators recently observed significantly greater cardiovascular stress responses to mental arithmetic than to Stroop conflict and speech stress tasks within the same participants (Brugnera et al., 2018).

One source of theoretical evidence that suggests the utility of the MST, particularly versions of the task that give error feedback, concerns error reactivity. Multiple studies have demonstrated a robust association between trait neuroticism and heightened reactivity to error (Moeller & Robinson, 2010; M. D. Robinson et al., 2010; M. D. Robinson & Clore, 2007). Individuals high in neuroticism not only exhibit greater stress responses to errors but also associated declines in cognitive performance and elevated stress levels in both laboratory and real-world contexts. From the perspective of the NIM, such anxiety-related traits reflect a failure of adaptive engagement with environmental demands (B.H. Friedman, 2007). Consequently, the MST offers a valuable tool for assessing stress reactivity in relation to neuroticism.

Present Study

Given the limited number of direct comparisons between stress tasks as a function of personality traits, the present study addresses a notable gap in the literature. Both the CPT and MST effectively induce cardiovascular stress responses, and these responses generally predict stress reactivity in daily life, although the robustness of these associations remains debated (Schwartz et al., 2003). Nonetheless, to my knowledge, no study has directly compared the CPT and MST while also assessing Five-Factor personality traits. This investigation is further distinguished as the first to examine stress reactivity both within each task and in direct comparison between tasks through the lens of the Five-Factor model. This approach aligns with recommendations from researchers who advocate for within-subjects designs that incorporate baseline as superior methods for investigating HRV and RSA (Quintana & Heathers, 2014).Informed by the literature reviewed above, I predict that HRV-based measures of stress reactivity will be associated with trait neuroticism during both relaxation periods and stress tasks. Researchers have recently compiled evidence that the most common pattern of HRV changes in response to stress is a decrease in HF-HRV along with a concomitant increase in LF-HRV, assumed to partly reflect a withdrawal of vagal tone and increase in sympathetic tone (Immanuel et al., 2023). The hypotheses are grouped into three categories.

H1. Baseline associations with neuroticism

H1a. Higher trait neuroticism will be associated with lower baseline HF-HRV.

H1b. Higher trait neuroticism will be associated with higher baseline LF-HRV.

H1c. Higher trait neuroticism will be associated with lower baseline RMSSD.

H2. Task differences in stress reactivity

H2a. The MST will produce greater decreases in HF-HRV than the CPT.

H2b. The MST will produce greater increases in LF-HRV than the CPT.

H2c. The MST will produce greater decreases in RMSSD than the CPT.

H3. Task type as a moderator

H3a. The relationship between trait neuroticism and HF-HRV will be moderated by task type, with the MST producing greater HF-HRV reductions among individuals high in neuroticism.

H3b. The relationship between trait neuroticism and LF-HRV will be moderated by task type, with the MST producing greater LF-HRV increases among individuals high in neuroticism.

H3c. The relationship between trait neuroticism and RMSSD will be moderated by task type, with the MST producing greater RMSSD reductions among individuals high in neuroticism.

Method

Based on the literature reviewed above, I hypothesized that trait neuroticism would predict lower vagally mediated HRV and higher sympathetically mediated HRV at rest. I also predicted that stress tasks would induce reductions in vagally mediated HRV and increases in sympathetically mediated HRV among participants with higher trait neuroticism. Additionally, I predicted that the type of stress task would moderate this association, with a stronger effect observed during the MST compared to the CPT. Auburn University approved all study procedures (#23-671). Relevant materials are included in the appendices: the pre-recruitment survey (Appendix A), Subjective Stress Rating Form (Appendix B), Stereotype Vulnerability Scale (SVS; Appendix C), participant payment form (Appendix D) recruitment documents (Appendix E), informed consent document (Appendix F), the R code used for statistical analysis (Appendix G), and additional statistical output and figures inappropriate for inclusion in the main text (Appendix H).

Recruitment and Participants

Data collection occurred in two phases: (1) a pre-recruitment stage conducted via Auburn University Sona systems (https://www.sona-systems.com/), a cloud-based participant pool platform, and (2) a one-hour in-person experimental session located at the Auburn University Cognitive and Affective Neuroscience Laboratory (Thach Hall, Room 108C). Participants (N = 631) were pre-recruited from the Auburn University main campus and surrounding community, including undergraduate and graduate students as well as non-student adults (minimum age = 18). Undergraduate psychology students received one hour of research credit for completing the pre-recruitment process via Sona systems. The pre-recruitment battery included demographic and medical questionnaires, psychological assessments, and personality measures used to characterize participants and determine eligibility for the second phase. Pre-recruitment occurred through multiple channels: flyers posted around campus and the Auburn community, announcements on the Auburn University Cognitive and Affective Neuroscience Laboratory Facebook page, posts on local community social media pages, and in-class announcements in undergraduate psychology courses with instructor permission.

To qualify for the second phase, participants had to be between the ages of 18 and 50 years old, since HRV declines progressively with age (Choi et al., 2006; Garavaglia et al., 2021; Jensen-Urstad et al., 1997; Liao et al., 1995; Sloan et al., 2008; Stein et al., 1997; Tsuji et al., 1996; Zhang, 2007), decreasing gradually across decades (Umetani et al., 1998). Participants were also required to pass all attention checks embedded in the pre-recruitment measures. Exclusion criteria included:

- Current use of anti-hypertensive, antipsychotic, or antidepressant medications
- Current or past nicotine use (defined as use of one or more nicotine-based products per day over the course of a month)

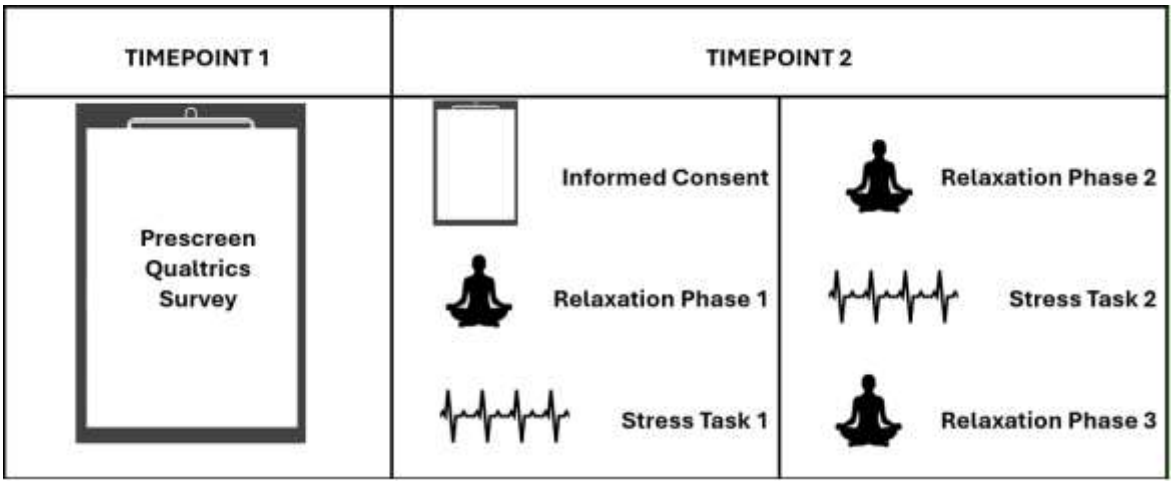
- Any medical conditions or medications known to impact cardiac function.

Study Design

The study employed a within-subjects repeated measures design, in which all participants completed both stress tasks in a counterbalanced order. During the pre-recruitment phase (Timepoint 1), participants completed measures to assess demographics, medical history, personality traits, and eligibility for continued participation at Timepoint 2. Eligible participants were invited to the Cognitive and Affective Neuroscience Laboratory, where they completed three relaxation phases and two stress phases while ECG and respiratory activity were continuously recorded (Figure 1).

Figure 1

Study Design Overview



Note. The above figure displays an overview of the study design

Screening Process and Inclusion Criteria

At the beginning of Timepoint 1, participants (N = 700) were presented with an information letter describing the pre-recruitment process and asked to provide informed consent. After consenting, participants completed a demographics questionnaire that collected the following: age, sex assigned at birth, gender identity, race, ethnicity (included due to

documented HRV differences by race/ethnicity; Arthur et al., 2004; Urbina et al., 1998; X. Wang et al., 2005), sexual orientation, past and present self-reported socioeconomic standing (SES), and class standing. Participants also self-reported their quantitative ACT score to provide an indirect index of mathematical ability. Participants reported any current or past physical health conditions (including cardiovascular or pulmonary disorders), current medications and dosages, history of mental health diagnoses, and current use of antidepressants, antipsychotics, antihypertensives, and oral contraceptives. Use of oral contraceptives was assessed since some evidence indicates that HRV parameters may be impacted by regular use (Kirschbaum et al., 1999). However, this was not chosen as an exclusion criterion because more recent research findings suggest there may be no significant impact of use on HRV measures (Blake et al., 2023; Teixeira et al., 2015; Wilczak et al., 2013) and excluding participants on this basis would significantly reduce the sample size of this study. Participants also reported any past or current smoking or vaping habits involving nicotine-based products since previous evidence demonstrates that both acute (Sjoberg & Saint, 2011) and chronic (Hayano et al., 1990) use can significantly modulate cardiac vagal control.

Measures

Depression

To assess depressive symptoms, participants completed the Beck Depression Inventory-II (BDI-II; Beck et al., 1996). The BDI-II (21 items; Appendix A) is a self-report assessment that measures depressive symptoms, asking participants to consider their thoughts/feelings over the last two weeks. Scores range from low depression to moderate depression to significant depression. Meta-analytic work has provided strong support for the reliability and validity of the BDI-II, indicating an average alpha of .85 and strong correlations with other depression

measures (Y.-P. Wang & Gorenstein, 2013), as well as an improved factor structure over the first version of the BDI (Dozois et al., 1998). Such results also extend to samples from multiple cultures as well (E.-H. Lee et al., 2017). Importantly, Item 9 of the BDI-II, which assesses suicidal thoughts and wishes, was excluded to avoid administering an item that would require safety planning procedures beyond the resources of the research team. As a result, only 20 BDI-II items were measured in this project.

Anxiety

Anxiety symptoms were assessed using the Beck Anxiety Inventory (BAI; Beck et al., 1988). The 21-item BAI (Appendix A) is a widely accepted self-report assessment of anxious symptoms, frequently used in research and clinical contexts. Participants were asked to consider how much they have been bothered by different anxious symptoms over the last month. Scores range from very low anxiety to moderate anxiety to cause for concern (Beck et al., 1988). Likewise, the BAI has substantial support for its reliability and validity, with an alpha of .94 and test-retest reliability of .67 (Fydrich et al., 1992). Similar results have been observed in multiple cultures (H.-K. Lee et al., 2016).

Personality

Personality traits were measured with the International Personality Inventory Pool 120 assessment (IPIP 120; Appendix A; J.A. Johnson, 2014), a multi-dimensional personality assessment that assesses participants on the Five-Factor model of personality. This self-report assessment (120 items) asks participants to rate their typical feelings, thoughts, and behaviors relative to other people their same age on a five-point Likert Scale, enabling researchers to assess personality traits from the Five-Factor model of personality (Costa & McCrae, 1992). Importantly, the IPIP-120 assesses both broad personality domains and facet-level traits,

enabling higher resolution personality analyses. In addition, this measure has strong support for its reliability and validity (Maples et al., 2014). Similar results have been observed across multiple cultures (Khan et al., 2019; Vedel et al., 2019).

Behavioral Activation and Behavioral Inhibition

Participants completed the Behavioral Activation and Behavioral Inhibition Scales (BIS/BAS; Carver & White, 1994; Appendix A). This scale consists of 20 self-report items that measure dispositional sensitivities to impending rewards and punishments using a four-point Likert scale. This scale operationalizes a theoretical model of two brain systems: a behavioral activation system and a behavioral inhibition system, which regulate dispositional sensitivity to opportunity and threat (Gray, 1982). These scales have demonstrated good reliability and validity among college students (Khaliq et al., 2023) and large community samples (Jorm et al., 1998). Researchers have discovered evidence that individual differences in these measures may be related to heart rate and RSA during mental stress (Knyazev et al., 2002).

Stereotype Vulnerability

Finally, given that gender stereotypes around mathematical ability can impair performance (H. J. Johnson et al., 2012) and reduce motivation (Fogliati & Bussey, 2013) among women, participants completed the Stereotype Vulnerability Scale (Appendix C), a brief 8-item self-report measure assessing vulnerability to gender-related mathematical stereotypes (Spencer et al., 1999).

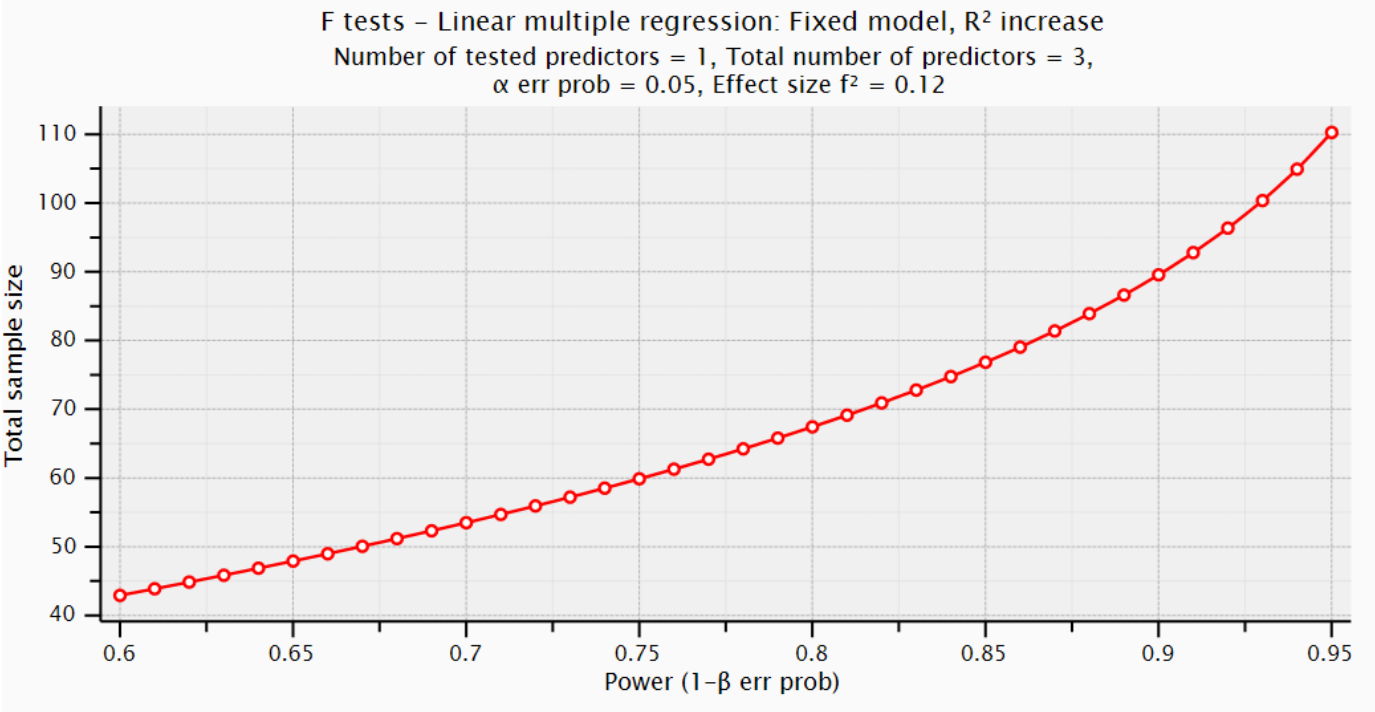
Power Analysis

An a priori power analysis was conducted in G*Power 3 (Faul et al., 2007) to determine the required sample size to detect a moderate effect ($f^2 = .12$; Quintana, 2017) with 80% power for multiple regression, in line with recommendations made based on prior literature (Quintana,

2017). Results indicated a minimum of 68 participants (Figure 2). To account for attrition and data loss, more than 68 participants from Timepoint 1 were scheduled for Timepoint 2 (N = 93). Participants who were invited to the second phase of the study received (a) one hour of research credit via Sona systems (for eligible undergraduate psychology students) and (b) $15 USD as compensation.

Figure 2

Power Analysis for Moderation Analyses



Note. This power analysis assumes an effect size of .12.

Stimuli and Procedure

Participants meeting the inclusion and exclusion criteria described above were invited to participate in the second timepoint of the study at the Cognitive and Affective Neuroscience Laboratory (Thach Hall 108B) between 1:00 and 6:00 PM to minimize circadian rhythm effects on cardiac function (van Eekelen et al., 2004). Prior to their study date, participants were emailed instructions to record their sleep and wake time given evidence that HRV values are sensitive to

the effects of sleep deprivation (Viola et al., 2002; Westphal et al., 2021; Zhong et al., 2005). They were also instructed to abstain from the following: caffeine 4 hours prior to their study time due to its effects on HRV (Rauh et al., 2006; Zimmermann-Viehoff et al., 2016); meals for 2 hours prior to their study time due to digestive impacts on HRV (Lu et al., 1999); alcohol consumption for 24 hours prior to their study time due to its impacts on HRV (Quintana et al., 2013); and intense physical training 24 hours prior to their study time due to its impacts on HRV (Stanley et al., 2013; Appendix D). All instructions aligned with the most current recommendations for conducting HRV research (Laborde et al., 2017).

Upon arrival at Thach Hall 108B, participants were presented an informed consent document. Study procedures were explained by me or one of two undergraduate research assistants (Aubrey Smith and Sophie Henegar) listed as key personnel on the project. All participant data collection performed during Timepoint 2 was completed by me, Aubrey Smith, or Sophie Henegar. During the informed consent process, participants were informed that they would undergo recordings of their respiratory and cardiac activity both at rest and during two stress tasks, one involving cold exposure and one involving mental math. After signing the consent form, participants were screened for COVID-19 symptoms and asked to confirm that they followed the preliminary instructions sent via email. Those participants who did not adhere to the instructions were re-scheduled. After confirming adherence, participants were then escorted next door to Thach Hall Room 108C, where their height and weight were measured, consistent with recommendations from researchers that have identified a significant impact of height/weight ratio on HRV measurements (Yi et al., 2013).

Participants were then seated in a chair and outfitted with a TSD201 BIOPAC respiration belt and three BIOPAC EL503 electrodes, located in the following locations: the left wrist, the

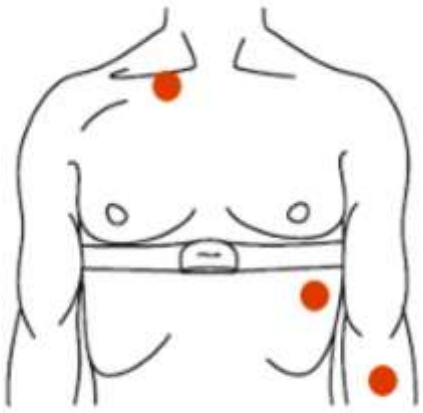
left fourth intercostal space on the ribcage, and the right collarbone (Figure 3). Participants were instructed to exhale completely, and the respiration belt was secured around the chest, just below the sternum. ECG and respiratory recordings were acquired using AcqKnowledge software and a BIOPAC MP150 system, connected with an MEC110C cable, one ECG100C amplifier module, and a RSP100C amplifier module for recording ECG and respiration, respectively (BIOPAC Systems, Inc., 2007). ECG recordings were sampled at 500 Hz, with a single bandpass filter between 0.5 Hz and 35 Hz. Respiration recordings were conducted with a single bandpass filter between 0.5 Hz and 1 Hz.

Participants with body hair on electrode placement sites were offered the opportunity to shave these areas with an electric razor prior to electrode application. Jewelry on electrode sites was removed. A small drop of BIOPAC Signa electrode gel was applied to each electrode cavity before placement. A brief test recording was conducted to ensure the ECG and respiratory signals were properly recorded.

ECG and respiratory recordings were collected during relaxation and stress phases. There were three relaxation phases (10 minutes each) and two stress phases (up to five minutes each). Stress phase order was counterbalanced so that half of the participants completed the CPT first and the other half completed the MST first. After each stress task, participants completed a brief stress rating on a Likert scale from 0 (“No Stress”) to 5 (“ Extreme Stress”; Appendix C). Upon completion of the final relaxation period, all physiological recording equipment was removed. Participants were thanked for their participation and compensated 15 USD after completing a participant payment form (Appendix D). Cash payments were stored in a lockbox housed in a locked drawer located in Thach Hall Room 108C.

Figure 3

ECG leads and Respiration Belt Placement



During the relaxation phase, participants sat in a soft chair, with eyes closed, maintaining an upright posture at 90-degree angle. Arms rested on their thighs with palms facing upward to minimize interoceptive effects of feeling their pulse at their wrists (Laborde et al., 2017). Participants were instructed to relax without engaging in any particular thoughts or activities, to remain seated and still, and to breathe naturally, as controlled breathing can alter cardiac responses in ways that may not reflect typical physiological functioning. (Larsen et al., 2010). The relaxation period lasted ten minutes; however, ECG and respiration recordings began five minutes after the start of the relaxation period to allow for physiological stabilization, consistent with prior recommendations (Laborde et al., 2017). Short-term tonic and phasic measurements of HRV have demonstrated strong reliability and validity across multiple investigations (Munoz et al., 2015; Sandercock et al., 2005). Given the wide range of HRV metrics available (J. J. Allen et al., 2007), multiple metrics were calculated.

After the initial relaxation period, participants began the first stress task. ECG and respiration signals were continuously recorded during both tasks. Following the first stress task, participants completed a second 10-minute relaxation period prior before beginning the second stress task. After the second stress task, participants completed a final 10-minute relaxation period. For both relaxation periods following stress tasks, ECG and respiration recordings began five minutes after the start of the relaxation phase. All participants invited to the second phase completed each stress task and relaxation period except a few participants who were excluded from further analysis ($N = 3$).

Half of the participants first completed the CPT, a widely used laboratory stressor (Birnie et al., 2012). While seated upright in the same chair used for relaxation, participants were instructed to immerse their right hand up to the wrist bone into a circulating cold-water bath, maintained at 5°C (±0.3°C). They were asked to keep their hand in the water for as long as possible, up to a maximum of five minutes, after which they were instructed to withdraw it, consistent with prior literature recommendations (Baeyer et al., 2005). ECG and respiratory recordings began as soon as participants verbally confirmed placement of their hand in the cold water and ended as soon as participants verbally confirmed removal of their hand from the water. Immersion duration times were recorded using the stopwatch feature on a cellular device.

The cold-water apparatus was a Neslab RTE-110 Refrigerated/Heating Circulating Bath Chiller (Thermo Fischer Scientific), which maintained constant circulation and water temperature. The chiller was positioned to the right of the participant's chair on a small stool designed to keep the water surface at hand level (Figure 4), allowing participants to insert their hand with minimal movement.

Figure 4

Cold Pressor Circulating Water Chiller

Note. The black handle is attached to a removable lid, revealing an open space for participants to insert their hand during the CPT.

Following the first stress task, participants completed a second relaxation phase to assess stress recovery before the next stress task. Instructions were identical to those in the initial relaxation period: participants were told to avoid focusing on any particular thoughts or activities, to remain as still as possible, and to keep their eyes closed. Each relaxation phase was the same length, and ECG and respiration recordings resumed five minutes after the start of the relaxation period.

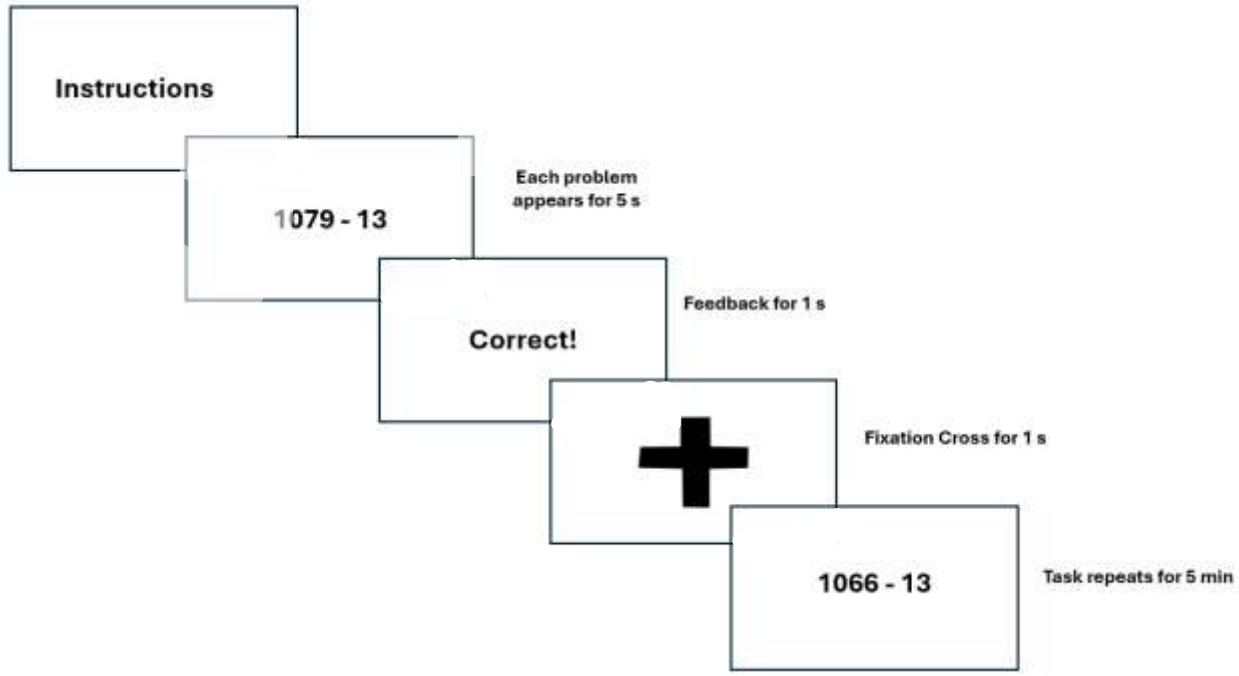
Participants then completed the MST, which consisted of a series of serial subtraction problems presented on a Windows Dell desktop computer. The task was programmed using PsychoPy version 2 (Peirce et al., 2019) and is a widely used laboratory stressor (Soliemanifar et al., 2018). In this version, participants began with the number 1079 and repeatedly subtracted 13 for the duration of the task, consistent with prior work examining short- and long-term stress reactivity in healthy participants (Hassellund et al., 2010).

Participants were instructed to work as quickly and accurately as possible. Before beginning, they viewed an instruction screen with bold black text on a white background, prompting them to press the space bar to start. Once initiated, subtraction problems were displayed in black bold text centered on a white background for five seconds each.

Responses were entered using the participant's right hand on a provided keyboard. Immediate feedback was displayed after each response: a one-second screen reading "Correct!" for correct answers, or "Incorrect!" for incorrect answers or missed responses. After feedback, a blank white screen with a black fixation cross appeared for one second before the next problem was displayed. Each new problem began with the solution to the previous one (e.g., 1079-13 = 1066; next problem: 1066–13). Each problem was displayed for a five-second duration, and problems were presented continuously for the full five-minute task duration. ECG and respiration recordings began at the onset of the MST and continued until immediately after participants solved the last problem. Any excess ECG/respiratory recording times beyond five minutes for any of the relaxation and task phases were trimmed to exactly five minutes prior to data analysis.

Figure 5

Protocol for the Mental Arithmetic Task



Physiological Data Analysis

Physiological data were processed using AcqKnowledge 5.0.2 (BIOPAC Systems, Inc., 2017). Respiratory signals were initially resampled from 500 Hz to 62.5 Hz to improve processing efficiency. After resampling, a finite impulse response (FIR) filter was applied with a lower bound of 0.05 Hz and upper bound of 1 Hz to reduce outliers and artifacts in the respiratory signal. The find cycle feature was then used to identify respiratory cycles needing manual correction. Although manual corrections were made, all participants still exhibited extended periods of weak respiratory signal or respiratory rates outside the recommended range of 9-24 cycles per minute for accurate vagal tone estimation (Laborde et al., 2017). Due to these persistent issues, respiratory data were excluded from further analysis, and all HRV metrics were derived solely from the ECG signal.

ECG data were processed in AcqKnowledge 5.0, then further analyzed using ARTiiFACT (Kaufmann et al., 2011), a MATLAB-based tool for automated artifact detection, correction, and HRV calculation. As described earlier, during data collection, timings for the start and stop times for ECG recordings were manually tracked using the stopwatch function on a cellular device. However, on several occasions, the ECG recording was mistakenly started slightly later than after the first five minutes of the relaxation phase or stopped slightly earlier than before the completion of the second five minutes of the relaxation phase by a few seconds, leading to inconsistent time lengths on the relaxation tasks. In those cases, participants were excluded from further analysis (n = 17). For participants where the ECG recording was slightly longer than the five-minute duration for the relaxation or MST tasks, the excess ECG signal was trimmed using the AcqKnowledge software to the task length of five minutes.

After data trimming, a linear-phase FIR band-pass filter (0.05 – 150 Hz) was applied to enhance signal quality using the AcqKnowledge software. Participants with poor quality ECG due to baseline drift or other motion-related artifacts after filtering (n = 3) were excluded following visual inspection. The remaining time series were converted to R-R intervals using the global threshold detection method provided by ARTiiFACT. Because artifacts in ECG can substantially inflate variance in HRV estimates (Berntson & Stowell, 1998), artifact corrections were employed prior to spectral analysis. Artifacts were identified within each time series employing a well-validated approach that detects artifacts using each individual participant's sampled IBI distribution to estimate what the real distribution of IBI values is for each participant (Berntson et al., 1990), implemented within the ARTiiFACT software. Detected artifacts were replaced via cubic (spline) interpolation, which estimates missing or corrupted N-N intervals by fitting a third-order polynomial to adjacent valid data and substituting the

interpolated values (Benchekroun et al., 2023). After reviewing the artifacts detected in the HRV signal, there were several participants (n = 6) with HRV signal lengths where half or more of the timepoints were detected as artifacts. These participants were excluded from further analysis, leaving a final analytic sample of 67.

Time- and frequency-domain HRV metrics were calculated in ARTiiFACT. Spectral measures included the absolute value, percent, and natural logarithm of LF-HRV and HF-HRV, Time-domain measures included mean and median heart rate, mean and median R-R intervals, the standard deviation of N-N intervals, which represents the standard deviation of the interval between successive N-waves (SDNN), the count of successive interval differences between heart beats greater than 50 ms (NN50), NN50 expressed as a percentage of all intervals (pNN50), and RMSSD. Power spectral density (PSD) estimates were computed within ARTiiFACT software using the Welch Periodogram Method to perform a Fast Fourier Transformation with a 256 second Hanning Window and a spline interpolation of 4 hz (Welch, 1967). Frequency bands were defined as follows: LF (0.04 Hz < 0.15 Hz), and HF (0.15 Hz < 0.4 Hz; Malik, 1996).

Statistical Analysis

All further analyses were conducted in R 4.5.1, an open-source statistical programming language (R Core Team, 2025). Data wrangling (e.g., re-structuring HRV metrics into long-format tables by task condition) was performed using the tidyr and dplyr packages (Wickham et al., 2023, 2024). The full analysis script is provided in Appendix G and was formatted and exported into Word using Rmarkdown (Allaire et al., 2024) and knitr (Xie, 2025) software packages. HRV metrics (HF-HRV, LF-HRV, and RMSSD) collected during the baseline phase, two relaxation phases, and both task conditions (MST and CPT) were natural log-transformed prior to analysis to reduce skewness and improve normality of distributions, consistent with prior

recommendations (Shaffer & Ginsberg, 2017). Descriptive statistics were computed for demographic variables, medical history, personality assessments, and HRV metrics using the dplyr package (Wickham et al., 2023) and the psych package (Revelle, 2025). Depending on the variable type, means, standard deviations, medians, ranges, and proportions were calculated. Correlation analyses were performed using Spearman's rho with the Hmisc package to address issues with non-normality (Harrell, 2025), applying listwise deletion for missing data. Correlation matrices were visualized with the corrplot package (Wei & Simko, 2024); variables were ordered using hierarchical clustering, significant associations were displayed with coefficients and circles, and non-significant correlations were left blank.

To evaluate H1, the relationship between baseline HRV and total neuroticism scores was assessed using three multiple linear regression models (ordinary least squares estimation). Shapiro-Wilk tests were conducted on each dependent variable to assess normality assumptions. Model assumptions were further evaluated by examining residuals plotted against fitted values to assess linearity and homoscedasticity (see Appendix H), and by conducting the Breusch–Pagan test for heteroscedasticity using the lmtest package (Zeileis & Hothorn, 2002). Variance inflation factors (VIF) were computed using the car package (Fox & Weisberg, 2019) to evaluate multicollinearity among predictors. Covariates included in the three models were age, gender, BDI-II scores, BAI scores, the researcher collecting the data (coded as Mackenzie, Aubrey, or Sophie) to assess experimenter effects, and self-reported sleep duration (hours) the night prior to the experiment. Gender was included as a covariate in all models because researchers have previously demonstrated that HRV varies between healthy men and women (Koskinen et al., 2009).

H2 initially involved a plan to perform three separate within-subjects repeated-measures Analysis of Variance (ANOVA) models to assess task-related differences in HRV metrics (HF-HRV, LF-HRV, and RMSSD) between the MST and CPT conditions. Prior to model fitting, the normality of dependent variables was assessed using Shapiro-Wilk tests conducted separately by task condition. Additionally, QQ plots were generated, and residual-*vs.*-fitted values were examined to assess distributional assumptions (Appendix H). Log-transformed RMSSD exhibited significant deviation from normality, so linear mixed-effects models (LMMs) were employed instead. Continuous covariates were not mean-centered because no interaction terms were included, ensuring that interpretation of main effects was not confounded.

For each outcome (HF-HRV for H2a, LF-HRV for H2b, and RMSSD for H2c), LMMs were fitted using the lme4 package (Bates et al., 2015), with normally distributed errors and an identity link. Fixed effects included task condition (MST vs CPT), trait neuroticism, SVS scores, math accuracy (percentage of math problems answered correctly on the MST), CPT duration, subjective stress ratings for both tasks, and experimenter identity. Initially, self-reported quantitative ACT scores were considered as a potential covariate to control for math ability. However, ACT data were missing for 22 of 67 participants (32.8%). Since including this variable with listwise deletion would reduce statistical power, ACT was not included as a covariate in the analysis. A random intercept for participant (ID) was included in all models to account for between-subject heterogeneity. Because each participant contributed only two observations (MST and CPT), random slopes for Task would have been unidentifiable (observations ≤ random-effects parameters), so the random-intercept-only structure was retained.

Models were estimated using restricted estimated maximum likelihood (REML). Significance testing was conducted with the Kenward-Roger approximation for denominator

degrees of freedom via the lmerTest package (Kuznetsova et al., 2017), which has been shown to provide robust control of Type 1 error rates in small samples (Luke, 2017). For RMSSD, which exhibited non-normality, a sensitivity analysis was conducted using a parametric bootstrap with 1999 simulations implemented in the afex package (Singmann et al., 2024). Multicollinearity was assessed using the performance package (Lüdecke et al., 2021). All VIF estimates were < 1.5, indicating no problematic collinearity.

H3 proposed three moderation analyses, each testing whether the task condition (MST *vs*. CPT) moderated the association between trait neuroticism and one of three HRV metrics (HF-HRV, LF-HRV, and RMSSD). The same covariates assessed in H2 were included as covariates in the moderation models: subjective stress ratings for both tasks, math accuracy, CPT duration, SVS scores, and experimenter identity. Task condition was first coded as a two-level factor (MST and CPT) with MST as the reference category. For Type III sums of squares tests, sum-to-zero contrasts were applied to the Task variable. Trait neuroticism was mean-centered prior to analysis to facilitate interpretation of the task main effect and to reduce potential multicollinearity.

Three ordinary least squares regression models were fitted, one for each HRV outcome. Each model included the main effects of mean-centered trait neuroticism and task condition, the neuroticism × task interaction term (the moderation test of interest), and the pre-specified covariates listed above. The interaction term tested whether the slope of the relationship between neuroticism and HRV differed between task conditions. Residual diagnostics were then performed to assess model assumptions. Model residuals were visually inspected using residuals-*vs.*-fitted plots and Q–Q plots (Appendix H). The Shapiro–Wilk test was used to test normality of residuals. Heteroscedasticity was evaluated with the Breusch–Pagan test. Multicollinearity

among predictors was examined using the car package (Fox & Weisberg, 2019), with all VIF estimates < 1.5, indicating no problematic collinearity. Any significant neuroticism × task interactions were probed graphically by plotting simple slopes for the MST and CPT separately using the ggplot2 package (Wickham, 2016).

Results

Participants (N = 67) included in the final analysis consisted of individuals with complete demographic, medical, and personality assessments, as well as complete ECG data for all five experimental phases (Baseline, the first stress task, Relaxation Phase 1, the second stress task, and Relaxation Phase 2). The mean age for participants was 21.3 years old (SD = 2.4, Range = 18 – 29 years). The sample was predominantly female (79%) and Caucasian (82%), as well as lower (20.9%) and upper middle class (46.3%).Approximately 89% of participants in the sample were right-handed. Roughly 25% of the participants in the sample endorsed a history of one or more psychiatric diagnoses. Mean participant scores on the BDI-II (M = 10.2, SD = 8.2) and BAI (M =11.7, SD = 12.1) indicated that the majority of participants experienced mild symptoms of depression and anxiety. Only 19% participants endorsed use of oral contraceptives, and all participants considered themselves to be generally healthy. Additionally, participants self-reported sleeping an average of 8.0 hours the night prior to the experiment (SD = 1.6). Descriptive statistics for demographic variables are listed in Table 1, while descriptive statistics for medical, psychological, and personality measures are displayed in Tables 2 and 3. Descriptive statistics for primary HRV metrics are displayed in Table 4, descriptive statistics for task performance data are listed in Table 5, and additional statistics for time- and frequency-domain HRV metrics not assessed in my hypotheses are documented in Tables 6 and 7, respectively.

Table 1

Demographic Characteristics of the Sample (N = 67)

Variable	*N*	%
Gender		
-Male	14	20.9
-Female	53	79.1
Race		
-White	55	82.1
-Black or African American	3	4.5
-Asian	3	4.5
-Native Hawaiian/Pacific Islander	1	1.5
-Multi-Racial	1	1.5
-Prefer Not to Answer	4	6
Ethnicity		
-Hispanic or Latine	7	10.4
-Not Hispanic or Latine	60	89.6
Socioeconomic Status		
-Working Class	12	17.9
-Lower Middle Class	14	20.9
-Upper Middle Class	31	46.3
-Upper Class	5	7.5
-Prefer not to Answer	5	7.5
Handedness		
-Right	60	89.6
-Left	7	10.4

Note. N = frequency count, % = frequency percent

Table 2

Medical and Psychological History of the Sample (N = 67)

Variable	*N*	%	*M*	*SD*	Range
Oral Contraceptive Use					
-Yes	13	19.4	—	—	—
-No	54	80.6	—	—	—
Count of Psychiatric Diagnoses					
0	50		—	—	—
1	8		—	—	—
2	9		—	—	—
Height (cm)	—		170.18	8.6	154.94–193.04
Weight (kg)	—		71	17	46.7–122.5
Sleep (Hours the Prior Night)	—		8.0	1.6	4–12

Note. Oral contraceptive use coded as Yes/No. Sleep reflects self-reported hours the previous night.

Table 3

Psychological Scales and Trait Measures

Variable	*M*	*SD*	Range
Stereotype Vulnerability Scale	22.0	8.0	8–42
Clinical Scales			
BDI-II Total	10.2	8.2	0–31
BAI Total	11.7	12.1	0–54
IPIP 120 Scales			
N1 Anxiety	12.8	3.8	5–20
N2 Anger	10.2	2.5	4–17
N3 Depression	9.9	3	4–18
N4 Self-Consciousness	12.6	2.5	8–18
N5 Impulsiveness	11.7	2.4	7–16
N6 Vulnerability	12.2	2.7	7–19
Neuroticism Total	69.4	10.8	47–97
BIS/BAS Scales			
BIS Punishment	21.9	3.7	11–28
BAS Reward	17.6	2.2	11–20
BAS Drive	11.1	2.3	6–16
BAS Fun Seeking	11.4	2.3	6–16

Note. BDI-II = Beck Depression Inventory II; BAI = Beck Anxiety Inventory; N1-N6 = Facet traits of the IPIP 120; BIS = Behavioral Inhibition System; BAS = Behavioral Activation System

Table 4

Primary HRV Metrics by Experimental Phase

Condition	Metric	*M*	*SD*	Median	Range
Baseline	HF-HRV (log)	6.12	1.29	6.04	3.69–9.17
	LF-HRV (log)	7.10	1.12	7.20	4.19–9.10
	RMSSD (log)	3.50	0.61	3.53	2.27–5.19
Relaxation 1	HF-HRV (log)	6.27	1.26	6.27	2.92–8.66
	LF-HRV (log)	7.10	1.03	7.18	4.36–9.44
	RMSSD (log)	3.66	0.51	3.60	2.42–4.73
Relaxation 2	HF-HRV (log)	6.05	1.22	6.09	3.32–8.89
	LF-HRV (log)	7.29	1.04	7.28	4.25–9.29
	RMSSD (log)	3.52	0.50	3.66	2.57–4.56
MST	HF-HRV (log)	6.11	0.98	6.26	3.97–9.34
	LF-HRV (log)	6.99	0.84	7.02	4.95–8.62
	RMSSD (log)	3.66	0.56	3.53	2.69–5.32
CPT	HF-HRV (log)	6.12	1.19	6.18	2.87–9.21
	LF-HRV (log)	7.48	1.04	7.46	5.23–11.0
	RMSSD (log)	3.71	0.62	3.69	2.62–6.10

Note. HF-HRV = High-frequency Heart Rate Variability; LF-HRV = Low-frequency Heart Rate Variability; RMSSD = Root Mean Square of Successive Differences; *M* = Mean; *SD* = Standard Deviation; Medians and Ranges are reported due to skew and outliers in HRV data;

Units are reported in ms^2

Table 5

Task Performance Data

Variable	*N*	*M*	*SD*	Range
Math Task				
-Correct Answers	67	18.5	8.3	0–33
-Incorrect Answers	67	15.5	8.3	1–34
-Percent Correct	67	0.5	0.2	0–0.9
Stress Task Ratings				
-MST	67	2.2	1.1	0–5
-CPT	67	2.9	1.3	0–5
Cold Exposure Duration (s)	67	108	103	16–300

Note. Math performance reflects number and percent of problems answered correctly/incorrectly. Percent is represented in decimal form. Stress task ratings were self-reported immediately after each task on a 0 to 5 Likert Scale. Cold exposure duration = number of seconds hand was submerged during CPT

Table 6

Time-domain HRV Metrics by Experimental Phase

Condition	Metric	*M*	*SD*	Median	Range
Baseline	Mean HR (bpm)	80.85	9.43	81.07	55.68–103.09
	SDNN (ms)	68.49	61.44	58.36	16.54–516.12
	NN50 (count)	56.60	53.81	43.00	0.00–242.00
	pNN50	15.17	15.47	10.30	0.00–79.61
	Mean RR (ms)	752.70	93.84	740.13	582.03–1077.24
	Median RR (ms)	750.33	94.90	740.00	578.00–1073.00
Relaxation 1	Mean HR (bpm)	78.73	9.38	78.93	53.78–102.60
	SDNN (ms)	63.43	25.56	60.37	21.41–139.64
	NN50 (count)	59.33	49.42	55.00	0.00–186.00
	pNN50	16.08	14.13	12.85	0.00–56.11
	Mean RR (ms)	773.07	94.78	760.16	584.79–1115.58
	Median RR (ms)	770.67	96.33	766.00	576.00–1118.00
Relaxation 2	Mean HR (bpm)	78.63	9.27	78.39	51.88–101.80
	SDNN (ms)	64.76	24.73	60.13	23.81–136.71
	NN50 (count)	58.93	50.07	47	0–187
	pNN50	15.70	13.97	12.8	0–50.16
	Mean RR (ms)	774.09	96.69	765.41	589.38–1156.57
	Median RR (ms)	773.18	98.87	767	592–1168
MST	Mean HR (bpm)	79.33	9.65	78.57	54.61–101.76
	SDNN (ms)	58.74	22.18	56.50	21.95–140.95
	NN50 (count)	58.91	48.73	49	1–227
	pNN50	15.88	14.89	12.36	0.21–83.15
	Mean RR (ms)	768.14	100.26	763.63	589.61–1098.79
	Median RR (ms)	768.57	101.61	758	590–1098
CPT	Mean HR (bpm)	81.53	11.19	80.75	56.57–106.23
	SDNN (ms)	91.41	177.18	56.19	19.16–1263.61
	NN50 (count)	25.96	37.22	11	0–187
	pNN50	17.36	15.80	12.90	0–72
	Mean RR (ms)	750.62	110.76	743.06	564.83–1060.70
	Median RR (ms)	747.67	103.68	744	579–1040

Note. Mean HR = mean heart rate; SDNN = standard deviation of normal-to-normal intervals; NN50 = number of adjacent NN intervals differing by more than 50 ms; pNN50 = percentage of NN50 relative to total NN intervals; RR = timer interval between successive R waves

Table 7

Frequency-domain HRV Metrics by Experimental Phase

Condition	Metric	*M*	*SD*	Median	Range
Baseline	LF/HF ratio (log)	1.05	0.98	1.00	-1.52–3.60
	HF-HRV %	18.92	13.53	16.03	2.43–69.19
	LF-HRV %	46.60	20.11	46.81	15.20–93.45
Relaxation 1	LF/HF ratio (log)	0.93	0.81	1.00	-0.66–2.77
	HF-HRV %	19.89	12.18	16.43	3.22–49.30
	LF-HRV %	43.85	15.51	40.48	20–79.60
Relaxation 2	LF/HF ratio (log)	1.14	0.90	1.29	-1.21–2.92
	HF-HRV %	16.98	12.59	11.93	2.68–66.84
	LF-HRV %	45.16	17.67	43.89	12.00–85.66
MST	LF/HF ratio (log)	0.88	0.61	0.88	-0.98–2.27
	HF-HRV %	19.76	10.15	17.62	6.51–66.76
	LF-HRV %	45.16	13.64	46.01	16.63–71.52
CPT	LF/HF ratio (log)	1.26	1.07	1.06	-0.52–4.93
	HF-HRV %	25.80	14.43	25.75	0.72–57.57
	LF-HRV %	72.76	15.04	72.97	32.61–99.28

Note. LF/HF ratio = ratio of low-to-high frequency power; % values represent normalized units of spectral power relative to total HRV power. Log-transformed values reported for LF/HF due to skew; Units are reported in $ms2$.

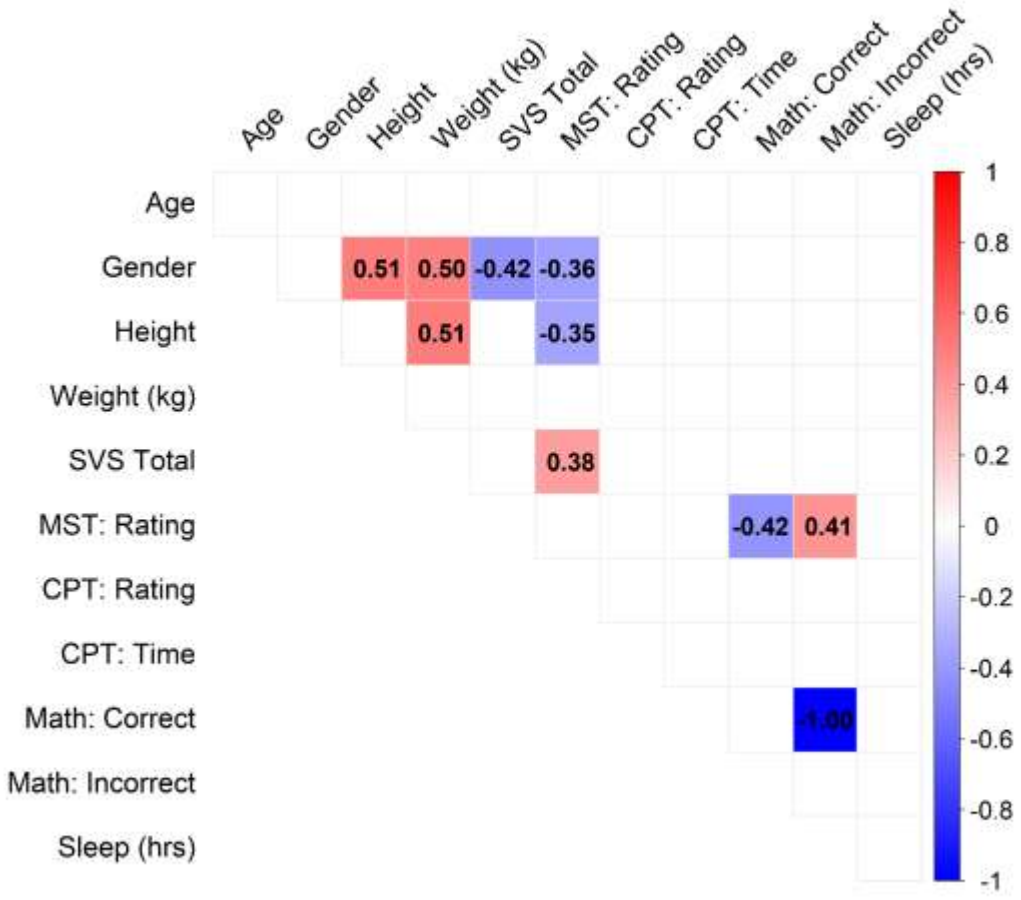
Correlation Analyses

Bivariate correlations between all key study variables were performed, including demographic/task results (Figure 6), psychological/personality assessments (Figure 7), and HRV metrics during each experimental phase (Figure 8). All correlations were computed using Spearman's rank-order method due to the non-normality of the data. To control for increased Type 1 error rates due to multiple comparisons, the Benjamini-Hochberg method was applied to adjust the false discovery rate (Benjamini & Hochberg, 1995). All reported *p*-values reflected this adjustment, with significant associations interpreted at the conventional $\alpha = .05$ level.

There were only a small number of meaningful correlations between most demographic variables, most of which were unsurprising. More importantly, a significant correlation between MST stress rating and math accuracy, $r_s(65) = -.42$, $p = .003$, was observed, indicating that increased stress ratings were linked to a decrease in the number of math problems answered correctly. Men tended to have lower SVS scores or reduced stereotype threat vulnerability, $r_s(65) = -.42$, $p = .003$. Individuals who rated the MST as more stressful also tended to be more vulnerable to the stereotype threat affect, $r_s(65) = .38$, $p = .01$. Additionally, men were more likely to rate the MST as less stressful than women , $r_s(65) = -.36$, $p = .016$. These particular findings may indicate that the stereotype threat affect could have impacted stress perception (no association between math accuracy and gender was observed); however, interpretive caution is warranted given that the sample was predominantly female.

Figure 6

Spearman Correlation Plot of Key Demographic and Behavioral Variables



Note. Benjamini-Hochberg method applied to address multiple comparisons. Only statistically significant correlations are filled in at $p < .05$.

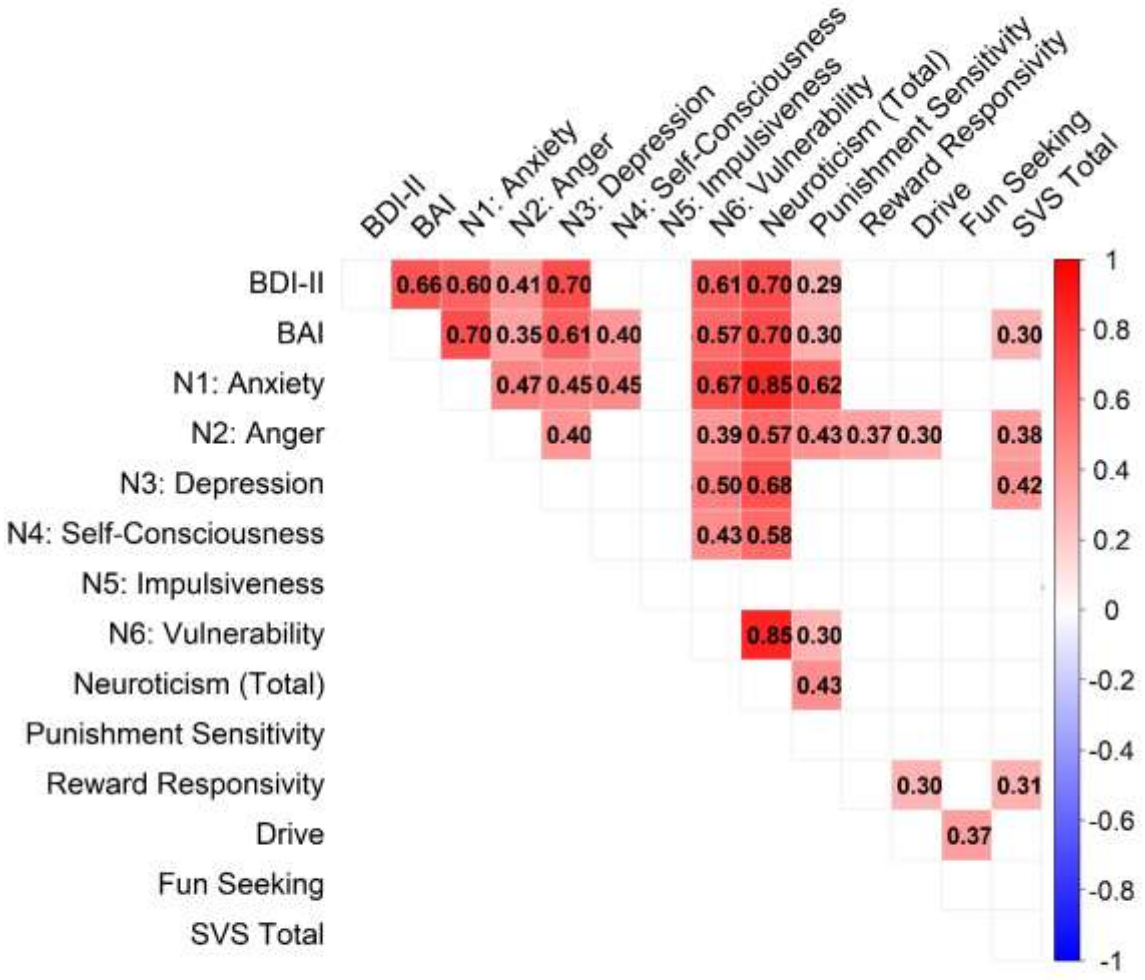
Psychological assessments, personality assessments, and state-related psychological surveys were assessed next. A wide range of significant and interesting correlations were observed. Unsurprisingly, the data revealed that more depressive symptoms related to more anxiety symptoms, $r_s(65) = .66$, $p < .001$, as well as higher scores on the depression facet of trait

neuroticism, $r_s(65) = .67$, $p < .001$. Likewise, the greater anxiety facet of neuroticism scores related to more anxiety symptoms, $r_s(65) = .70$, $p < .001$. Beyond this, greater depression symptoms related to higher scores on the majority of the other neuroticism facets: anxiety, $r_s(65) = .60$, $p < .001$, anger, $r_s(65) = .41$, $p = .002$, vulnerability, $r_s(65) = .61$, $p < .001$, and total neuroticism, $r_s(65) = .69$, $p < .001$. Stronger depression symptoms related to higher scores on the Punishment Sensitivity Scale of the BIS/BAS scales, $r_s(65) = .29$, $p = .04$. Similarly, more anxiety symptoms related to higher scores on neuroticism facets, including anger, $r_s(65) = .36$, $p = .007$, depression, $r_s(65) = .58$, $p < .001$, self-consciousness, $r_s(65) = .40$, $p = .003$, and vulnerability, $r_s(65) = .57$, $p < .001$, as well as total neuroticism, $r_s(65) = .70$, $p < .001$. Anxiety symptoms related to greater punishment sensitivity, $r_s(65) = .31$, $p = .03$.

The higher scores on the anger facet of neuroticism related to higher scores on drive scale of the BIS/BAS, $r_s(65) = .32$, $p = .02$ and the reward responsivity scale of the BIS/BAS, $r_s(65) = .35$, $p = .01$, possibly reflecting an underlying relationship between anger and broader approach-related motivations, goals, and states. reflecting statistical overlap between approach-oriented behaviors and impulsivity. In addition, stereotype vulnerability (SVS) was also assessed. Higher SVS scores related to more anxiety symptoms, $r_s(65) = .31$, $p = .03$, anger, $r_s(65) = .39$, $p = .004$, depression, $r_s(65) = .41$, $p = .002$, facets of neuroticism, and reward responsivity, $r_s(65) = .30$, $p = .03$. These results indicate that the more neurotic and reward-responsive individuals in this sample were more likely to experience vulnerability to stereotype threat effects. In summary, the measures in our sample associated with negative emotionality tend to be consistently correlated, in line with the broad patterns in the literature described earlier in this document.

Figure 7

Spearman Correlation Plot of Key Psychological and Trait Measurements



Note. Benjamini-Hochberg method applied to address multiple comparisons. Only statistically significant correlations are filled in at $p < .05$.

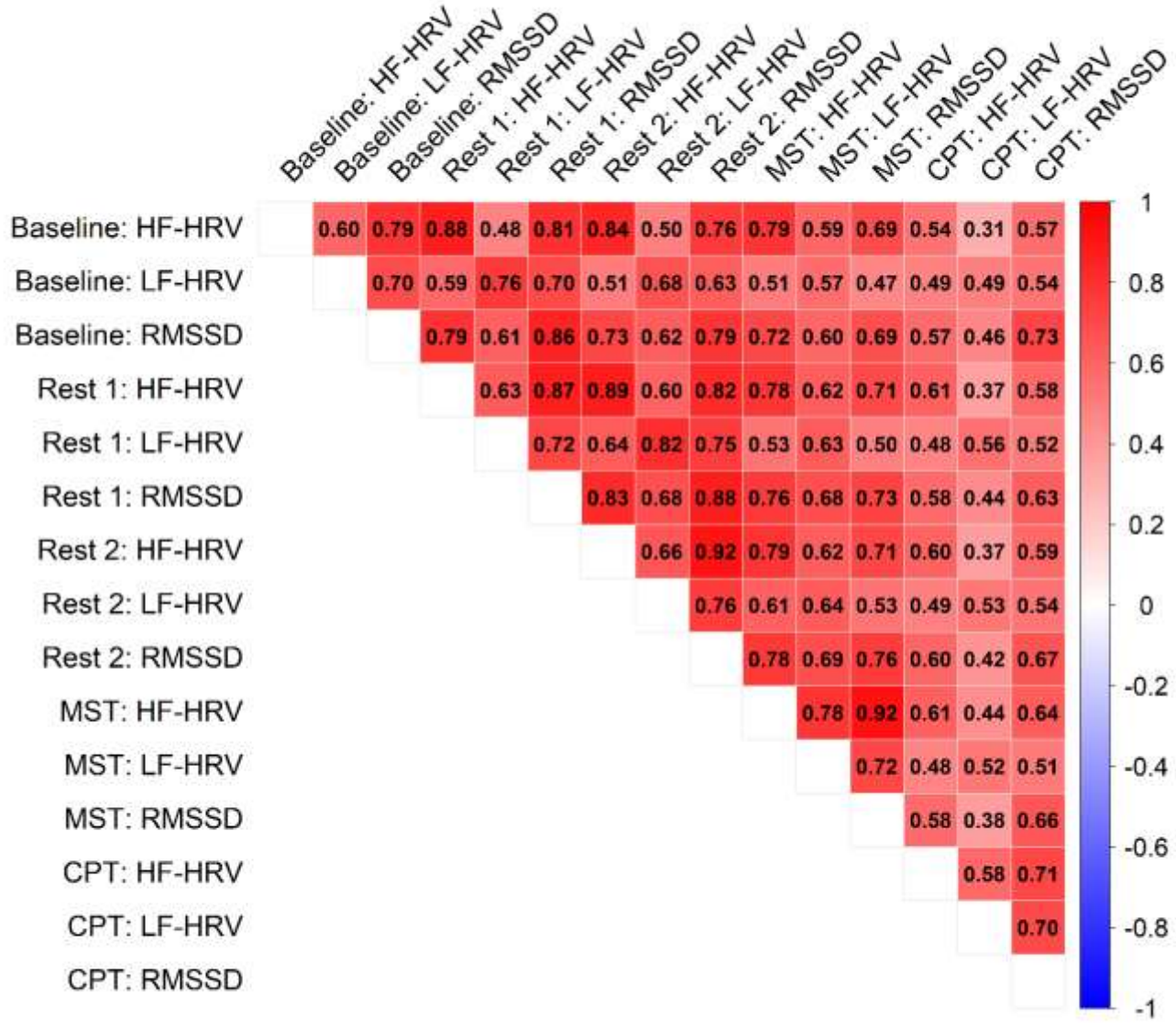
The three primary log-adjusted HRV metrics assessed in hypotheses were strongly correlated across all experimental phases. Not surprisingly, Higher HF-HRV metrics during the

baseline phase were related to higher scores on the first, $r_s(65) = .88$, $p < .001$, and second, $r_s(65) = .84$, $p < .001$, relaxation phases. Likewise, LF-HRV greater baseline phase scores related to higher scores at the first, $r_s(65) = .75$, $p < .001$ and second relaxation phases, $r_s(65) = .68$, $p < .001$. A similar pattern was also observed for RMSSD across the baseline phase and first, $r_s(65) = .86$, $p < .001$, and second relaxation phases, $r_s(65) = .78$, $p < .001$. These results highlight stability and consistency in HRV measures across time points. Similar results existed for relationships between HF-HRV collected during the MST and HF-HRV collected during the CPT phases, $r_s(65) = .61$, $p < .001$, MST LF-HRV and CPT LF-HRV, $r_s(65) = .46$, $p < .001$, and MST RMSSD and CPT RMSSD, $r_s(65) = .66$, $p < .001$.

Across experimental phases, there were also similar correlations. For example, baseline HF-HRV and HF-HRV at MST, $r_s(65) = .69$, $p < .001$, CPT, $r_s(65) = .54$, $p < .001$. Likewise, similar correlations existed between baseline LF-HRV and MST, $r_s(65) = .57$, $p < .001$ and CPT LF-HRV, $r_s(65) = .50$, $p < .001$, along with baseline RMSSD and MST RMSSD, $r_s(65) = .68$, $p < .001$, and CPT RMSSD, $r_s(65) = .73$, $p < .001$. However, these correlations appear to be consistently smaller than the correlations between identical HRV metrics across the baseline and relaxation periods. Overall, the findings illustrate strong interrelationships among HRV metrics, particularly within the same measure across time periods.

Figure 8

Spearman Correlation Plot of Key HRV Metrics



Note. Benjamini-Hochberg method applied to address multiple comparisons. Only statistically significant correlations are filled in at $p < .05$. All HRV Metrics listed here are log-adjusted.

H1: Linear Regression Analyses

To test H1, three linear regression models predicted baseline HRV metrics from trait neuroticism, controlling for age, gender, depressive symptoms, anxiety symptoms, sleep duration the previous night, and experimenter identity. Assumption checks indicated that residuals were normally distributed for all three models (all Shapiro-Wilk *p*s > .13), and no concerns with multicollinearity were observed (all VIFs < 2.6). Residual plots suggested no major deviations, although the Breusch-Pagan test indicated heteroscedasticity in the model predicting LF-HRV, $\chi^2(7) = 14.90$, $p = .037$. Nonetheless, the model was still conducted given the residual and QQ-plots and some evidence that the Breusch-Pagan test can be overly statistically sensitive (Alıca et al., 2025). The output for statistical assumption checks for all models included in Hypotheses 1-3 are displayed in Appendix H. Full regression results for each model are presented in Table 8.

H1a: HF-HRV

The overall model predicting HF-HRV was not statistically significant $F(7, 59) = 0.73$, $p = .65$, and only explained 8% of variance in HF-HRV, $R^2 = .08$, adj. $R^2 = –.03$. Neuroticism was unrelated to HF-HRV, $b = -0.002$, $t(59) = -0.08$, $p = .94$. Age, gender, depressive symptoms, anxiety symptoms, sleep duration and experimenter identity, were all non-significant (all *p*s > .14).

H1b: LF-HRV

The model predicting LF-HRV was not statistically significant, $F(7, 59) = 1.94$, $p = .08$, although it explained more variance than the HF-HRV model, $R^2 = .19$, adj. $R^2 = .09$. Neuroticism predicted lower LF-HRV, $b = –0.040$, $t(59) = –2.16$, $p = .035$, such that more neurotic participants exhibited lower baseline LF-HRV. No other predictors were significant (all *p*s > .12).

H1c: RMSSD

The third model predicting RMSSD from neuroticism was not significant, $F(7, 59) = 0.68$, $p = .69$, explaining only 7% of the variance in RMSSD, $R^2 = .07$, adj. $R^2 = –.04$. Neuroticism was not a significant predictor, $b = -0.005$, $t(59) = -0.47$, $p = .64$, and none of the covariates were significant. (all *p*s >. 29).

Overall, these results do not provide support for Hypothesis 1. HF-HRV and RMSSD were unrelated to neuroticism, even when covariates were considered. In contrast, LF-HRV was significantly lower among more neurotic participants, an effect contrary to what was hypothesized. However, this effect was weak and may not be robust in a larger sample.

Table 8

Regression of Neuroticism on Baseline HRV

Predictor	*b*	*SE*	*t*	*p*
HF-HRV Model 1	—	—	—	—
(Intercept)	6.80	1.72	3.95	<.001***
Neuroticism	-0.00	0.02	-0.08	.940
Age	-0.03	0.03	-0.93	.355
Gender	-0.07	0.37	-0.19	.849
Depression Symptoms	-0.02	0.03	-0.74	.461
Anxiety Symptoms	0.02	0.02	1.09	.279
Sleep Duration	-0.01	0.09	-0.13	.898
Researcher	0.13	0.19	0.69	.492
Model Summary	R^2 *model* = .08, adjusted R^2= .00, *p* = .645			
LF-HRV Model 2	—	—	—	—
(Intercept)	10.82	1.53	7.08	< .001
Neuroticism	-0.04	0.02	-2.16	.035*
Age	-0.02	0.03	-0.72	.477
Gender	0.09	0.33	0.27	.787
Depression Symptoms	-0.00	0.03	-0.36	.723
Anxiety Symptoms	0.02	0.02	1.57	.121
Sleep Duration	-0.11	0.09	-1.30	.199
Researcher	0.20	0.16	1.24	.221
Model Summary	R^2 *model* = .19, adjusted R^2 = .09, *p* = .08			
RMSSD Model 3	—	—	—	—
(Intercept)	4.09	0.89	4.59	< .001***
Neuroticism	-0.01	0.01	-0.47	.639
Age	-0.01	0.02	-0.47	.639
Gender	-0.13	0.19	-0.66	.510
Depression Symptoms	-0.02	0.02	-1.06	.296
Anxiety Symptoms	0.01	0.01	0.68	.500
Sleep Duration	0.00	0.05	0.02	.982
Researcher	0.04	0.10	0.45	.654
Model Summary	R^2 *model* = .07, adjusted R^2 = -.04, *p* = .69			

Note. *b* = unstandardized regression coefficient, SE=standard error, *t* = standardized regression coefficient/standard error. *p<.05, **p<.01, ***p<.001

H2: Linear Mixed-Effects Models

To assess H2a, H2b, and H2c, three LMMs were conducted to evaluate differences in HRV metrics collected between the two experimental phases: the MST and the CPT. HF-HRV and LF-HRV collected during both the CPT and MST met statistical assumptions for normality, including the Shapiro-Wilk test (Appendix H). No multicollinearity concerns were present among the predictors (all VIFs < 2.6). Residual plots suggested no major deviations. However, RMSSD collected during both the MST, $W = .95$, $p < .01$, and the CPT, $W =$, $p < .01$, exhibited significant non-normality. Due to statistical non-normality of log-adjusted RMSSD collected during the MST and CPT, a bootstrap sensitivity analysis was performed for H2c. Complete results for each model are displayed in Table 9.

H2a: HF-HRV

The model examining differences in HF-HRV between the two task phases explained little variance, $R^2 = .08$, adj. $R^2 = –.03$. There was no main effect of task, $F(1, 66) = 0.01$, $p = .907$. Higher stress ratings for the MST did predict lower HF-HRV, $b = -0.27$, $t = -2.29$, $p = .025$. Other covariates were not significant (all ps > .11).

H2b: LF-HRV

The model exploring task differences in LF-HRV explained more variance, $R^2 = .19$, adj. $R^2 = .09$. Task phase did significantly predict LF-HRV, $F(1, 66) = 10.25$, $p = .002$; LF-HRV was higher during the CPT compared to the MST, $b = 0.15$, $t(66) = 3.20$, $p = .002$. None of the other predictors, including neuroticism, stereotype vulnerability, stress ratings, math accuracy, CPT time, or experimenter identity reached statistical significance (all ps > .36).

H2c: RMSSD

The overall model explaining task differences in RMSSD explained little to no variance, $R^2 = .07$, adj. $R^2 = -.04$. Nonetheless, task phase significantly predicted RMSSD, $F(1, 66) = 4.75$, $p = .033$; RMSSD was higher during the CPT compared to the MST, $b = 0.15$, $t = 2.18$, $p = .033$. Since RMSSD violated normality assumptions, a parametric bootstrap sensitivity analysis (1999 simulations) was conducted to validate the results of the LMM. The results confirmed the task effect (PB $p = .028$) with a 95% confidence interval for the task coefficient [0.02, 0.28]. Other covariates were not significant (all ps >.13).

Together, these results indicate weak but partial support for H2, which is that there were significant differences in HRV between the CPT and MST. Across models, task phase influenced HRV; LF-HRV and RMSSD were both higher during the CPT compared to the MST. Although HF-HRV did not differ by task, higher MST stress ratings were associated with lower HF-HRV. RMSSD results were robust to non-normality in bootstrap sensitivity analyses.

Table 9

Linear Mixed-Effects Models of HRV Across Task Phases

Predictor				
HF-HRV log Model 1	*b*	SE	*t*	*p*
-(Intercept)	6.54	0.89	7.37	< .001***
-Task Phase	0.01	0.11	0.12	.907
-Neuroticism	-0.00	0.01	-0.33	.745
-Stereotype Vulnerability	0.01	0.01	0.52	.604
-MST Stress Rating	-0.27	0.12	-2.29	.025*
-CPT Stress Rating	-0.12	0.09	-1.31	.196
-CPT Time Duration	0.00	0.00	1.68	.098
-Math Accuracy	0.01	0.01	0.80	.426
-Researcher	0.18	0.13	1.44	.156
LF-HRV log Model 2				
-(Intercept)	8.21	0.79	10.40	< .001***
-Task Phase	0.40	0.12	3.20	.002**
-Neuroticism	-0.01	0.01	-1.16	.249
-Stereotype Vulnerability	-0.00	0.01	-0.26	.795
-MST Stress Rating	-0.17	0.10	-1.68	.099
-CPT Stress Rating	-0.05	0.08	-0.69	.493
-CPT Time Duration	0.00	0.00	0.78	.436
-Math Accuracy	-0.00	0.01	-0.29	.772
-Researcher	0.18	0.11	1.59	.117
RMSSD log Model 3				
-(Intercept)	4.15	0.46	8.95	< .001***
-Task Phase	0.15	0.07	2.18	.033*
-Neuroticism	-0.00	0.00	-1.01	.315
-Stereotype Vulnerability	-0.00	0.00	-0.22	.824
-MST Stress Rating	-0.08	0.06	-1.35	.183
-CPT Stress Rating	-0.05	0.05	-1.05	.299
-CPT Time Duration	0.00	0.00	0.17	.867
-Math Accuracy	0.00	0.00	0.504	.616
-Researcher	0.10	0.07	1.525	.133

Note. Stereotype Vulnerability refers to total score on the SVS scale. MST/CPT Stress rating refers to a rating of the stressfulness of the task, coded 1 to 5 Likert style. CPT time duration refers to the time length participants kept their hand in the cold water (in seconds). Math

accuracy refers to a numeric count of the math problems solved correctly. Researcher refers to the identity of the experimenter that collected data from a given participant.

Table 10

Bootstrapped Regression Results

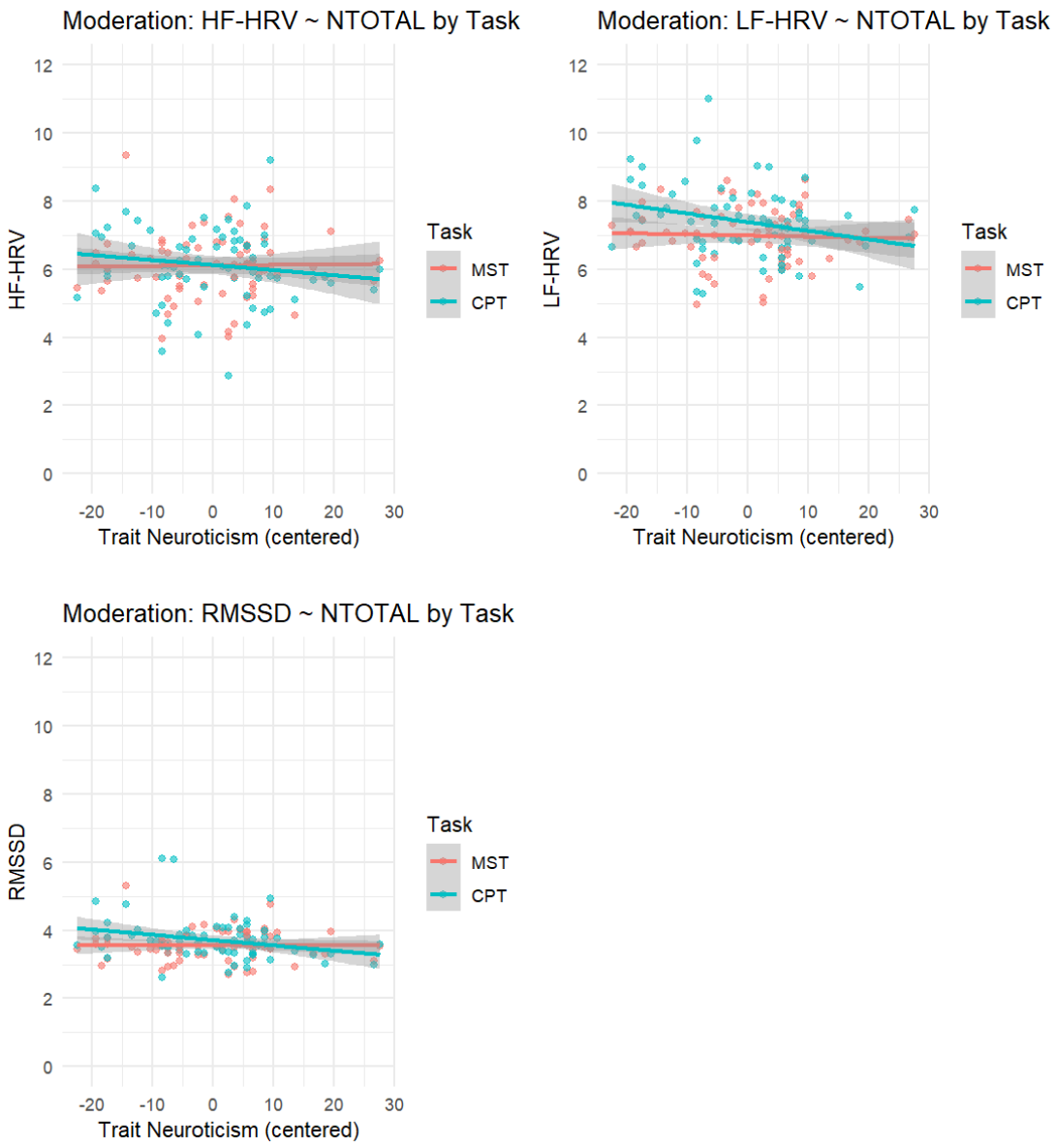
Bootstrap Model	*95% CI LL*	*95% CI UL*
-Task Phase	0.02	0.28
-Neuroticism	-0.02	0.00
-Stereotype Vulnerability	-0.02	0.01
-MST Stress Rating	-0.20	0.04
-CPT Stress Rating	-0.14	0.04
-CPT Time Duration	-0.00	0.00
-Math Accuracy	-0.01	0.02
-Researcher	-0.03	0.23

H3: Moderation Analyses

To assess H3a, H3b, and H3c, three moderation analyses were performed to test whether task condition (MST *vs.* CPT) moderated any statistical relationship between trait neuroticism and HRV metrics collected during the task phases. Trait neuroticism was mean-centered and SVS scores, MST accuracy, MST stress ratings, CPT stress ratings, CPT time, and experimenter identity were included as covariates. Results for all three models are summarized in Table 11. Although no interaction effects reached significance, plots were generated to visualize task differences in HRV (Figure 9).

Figure 9.

Moderation Plots of Neuroticism-HRV Association



Note. Fitted regression lines depict Neuroticism-HRV Association by task. Models include covariates (SVS, math accuracy, CPT time, MST/CPT stress ratings, and experimenter).

H3a: HF-HRV

The overall model was significant, $F(9, 124) = 3.36$, $p = .001$ $R^2 = .20$, adj. $R^2 = 0.14$. However, the neuroticism × task interaction was not statistically significant, $b = 0.01$, $t(124) =$

1.07, $p = .29$, indicating that the HF-HRV-neuroticism association did not differ by task. Two covariates did reach statistical significance. Higher MST stress ratings predicted lower HF-HRV, $b = –0.267$, $t(124) = –2.96$, $p = .004$, and longer CPT time predicted higher HF-HRV, $b = 0.002$, $t = 2.17$, $p = .032$. Assumption checks indicated approximately normal residuals and homoscedasticity, with no evidence of problematic multicollinearity (Appendix H).

H3b: LF-HRV

Although the model was statistically significant, $F(9, 124) = 2.68$, $p = .007$, $R^2 = .16$, adj. $R^2 = .10$, the neuroticism × task interaction was nonsignificant, $t(124) = 1.51$, $p = .13$. There was a significant task effect on LF-HRV, $t(124) = 6.37$, $p = .013$, indicating that LF-HRV was higher during the CPT compared to the MST. Higher MST stress ratings predicted lower LF-HRV, $b = –0.173$, $t\ (124) = –2.02$, $p = .046$. Other covariates were not significant (all ps ≥ .35). Statistical assumptions were largely acceptable; however, residual normality was marginal (Shapiro-Wilk $p = .049$). Tests for heteroscedasticity and multicollinearity indicated acceptable normality (Appendix H).

H3c: RMSSD

The third model was statistically significant, $F(9, 124) = 2.34$, $p = .018$, $R^2 = .15$, adj. $R^2 = .08$. However, the neuroticism × task interaction was not statistically significant, $t(124) = 1.85$, $p = .07$. No statistically significant task effect was observed, $F(1, 124) = 2.75$, $p = .100$, and none of the covariates in the third model reached statistical significance (all ps > .06). However, residuals were strongly non-normal (Shapiro-Wilk $p < .001$), so interpretative caution is warranted despite stable variance and low multicollinearity (Appendix H).

Across all three models, there was no evidence that the task phase moderated the relationship between trait neuroticism and HRV (all neuroticism × task ps < .13). Nonetheless,

task condition did impact LF-HRV, with LF-HRV increasing during the CPT compared to the MST. In addition, greater MST stress ratings consistently predicted reduced HRV (both HF and LF). Although no reliable neuroticism-HRV associations were observed, these findings suggest that differences in HRV may depend more on the type of stress task and differences in stress appraisal than trait-level personality factors.

Table 11

Task Condition as a Moderator of the Neuroticism-HRV Association

Predictor	β	SE	*t*	*p*	R^2	Adjusted R^2	*F*
HF-HRV log Model 1	—	—	—	—	—	—	—
Intercept	6.30	0.49	12.90	$p < .001$***	—	—	—
Neuroticism Centered	-0.00	0.01	-0.42	0.67	—	—	—
Task Condition	-0.01	0.08	-0.08	0.94	—	—	—
Neuroticism x Task	0.01	0.01	1.07	0.29	—	—	—
Stereotype Vulnerability	0.00	0.01	0.77	0.50	—	—	—
Math Accuracy	0.01	0.01	1.04	0.30	—	—	—
Cold Pressor Duration	0.00	0.00	2.27	0.03*	—	—	—
MST Stress Rating	-0.27	0.09	-2.97	0.00**	—	—	—
CPT Stress Rating	-0.12	0.07	-1.79	0.09	—	—	—
Researcher	0.28	0.09	1.96	0.07	—	—	—
Model Summary	—	—	—	$p < .001$***	0.20	0.14	3.36
LF-HRV log Model 2	—	—	—	—	—	—	—
Intercept	7.63	0.47	16.38	$p < .001$***	—	—	—
Neuroticism Centered	-0.01	0.01	-1.40	0.16	—	—	—
Task Condition	-0.20	0.08	-2.52	0.01*	—	—	—
Neuroticism x Task	0.01	0.01	1.51	0.13	—	—	—
Stereotype Vulnerability	0.00	0.01	-0.31	0.75	—	—	—
Math Accuracy	0.00	0.01	-0.35	0.73	—	—	—
Cold Pressor Duration	0.00	0.00	0.94	0.35	—	—	—
MST Stress Rating	-0.27	0.09	-2.02	0.05*	—	—	—
CPT Stress Rating	-0.05	0.07	-0.83	0.41	—	—	—
Researcher	0.28	0.09	1.91	0.06	—	—	—
Model Summary	—	—	—	0.007**	0.16	0.10	2.68
RMSSD log Model 3	—	—	—	—	—	—	—
Intercept	-0.01	0.00	14.29	$p < .001$***	—	—	—
Neuroticism Centered	-0.07	0.05	1.25	0.22	—	—	—
Task Condition	-0.08	0.05	-1.66	0.10	—	—	—
Neuroticism x Task	0.01	0.00	1.85	0.07	—	—	—

Stereotype Vulnerability	0.00	0.00	-0.27	0.78	—	—	—
Math Accuracy	0.00	0.00	0.62	0.54	—	—	—
CPT Time Duration	0.00	0.00	0.21	0.84	—	—	—
MST Stress Rating	-0.08	0.05	-1.66	0.10	—	—	—
CPT Stress Rating	-0.05	0.04	-1.39	0.20	—	—	—
Researcher	0.10	0.05	1.88	0.06	—	—	—
Model Summary	—	—	—	0.02*	0.15	0.08	2.34

Note. MST is the reference level for Task. All models include covariates (stereotype vulnerability, math accuracy, CPT time, MST/CPT stress ratings, researcher); coefficients for covariates omitted for brevity.

β = unstandardized coefficient; SE = standard error.

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

Discussion

Correlation Analyses

There were numerous statistically significant correlations. For example, both depression and anxiety symptoms were positively correlated with total neuroticism scores as well as several of the facet traits of neuroticism, which is consistent with meta-analyses of the relationships between these constructs (Kotov et al., 2010). Importantly, some data also links the association between neuroticism and depression/anxiety symptoms to perceived stress, suggesting that heightened perceived stress may mediate this association (Pereira-Morales et al., 2019). Additionally, increased trait neuroticism relates to more negative interpretations of life events, indirectly influencing the connection between neuroticism and depressive/anxiety disorders (Vinograd et al., 2020). Biased cognitive processing is a primary mechanism linking neuroticism and increased risk of mood disorders (Canli, 2008). Even highly neurotic individuals without any history of mood disorders exhibit negative biases in information processing (Chan et al., 2007).

There are also connections between neuroticism and physical health problems. Using a longitudinal diary approach, mindfulness (non-evaluative awareness of the present moment) relates to fewer physical health problems and that this relationship is mediated by stress appraisal (O'Loughlin et al., 2019). Individuals lower in trait mindfulness were more likely to perceive daily life events as stressful and suffer physical health problems related to them. This dovetails nicely with work using longitudinal experience sampling methods. Increases in affective reactivity to daily stressors drove increased trait neuroticism over time (Wrzus et al., 2021). Altogether, this pattern of evidence comports well with prior literature.

At the behavioral level, there were also correlations between stress appraisal and HRV metrics. Increased stress ratings for the MST related to reduced HF-HRV. A similar pattern of

results was observed for the CPT task: increased stress ratings were associated with increased LF-HRV. Although these tasks did not induce hypothesized changes, the link between stress appraisal and HRV metrics was consistent with prior literature. For example, increased stress appraisal was related to vagal withdrawal and increased sympathetic activation. For example, investigators have observed that participants with greater anxiety sensitivity experience decreased vagal activation in recovery after exposure to a CPT (Dodo & Hashimoto, 2017). Increased stress appraisal also related to less accuracy on the math task. Participants that rated the task as more stressful answered a higher number of questions incorrectly. This result is also well supported by prior literature linking increases in stress perception to decrements in task performance (Hancock et al., 2007; G. Matthews et al., 2002; Warm et al., 2008). For example, individuals with higher HF-HRV tend to perform better on working memory tasks in the context of stress (A. L. Hansen et al., 2003, 2009).

Not surprisingly, HRV metrics were intercorrelated with one another (e.g., Li et al., 2009). Additionally, the strength of these relationships was greater when comparing relaxation phases against each other as opposed to stress phases. This also replicates work showing that task-based HRV measures show less reliability than HRV measures collected during rest or relaxation (Goedhart et al., 2007).

Hypothesis 1

There was no relationship between baseline HF-HRV and trait neuroticism. None of the covariates (age, gender, depression symptoms, anxiety symptoms, sleep duration the previous night, and the identity of the experimenter performing data collection) related to HF-HRV. Although this result was not anticipated, it is not entirely unsurprising given the literature cited in chapter 2 that found significant inconsistencies between trait neuroticism and HRV metrics.

Although multiple studies found that neuroticism strongly predicts reduced resting HF-HRV, several also found no clear association between neuroticism and HRV. Given that this sample comprised largely healthy and young University students, it is possible that differences in HF-HRV between participants were not strong enough to demonstrate a clear relationship between resting HF-HRV and trait neuroticism.

In contrast, a statistically significant relationship between trait neuroticism and baseline LF-HRV did emerge. This relationship was in the opposite direction of the hypotheses, indicating that increased neuroticism related to lower LF-HRV. This pattern is not consistent with previous work. However, given the presence of some heteroscedasticity in log-adjusted LF-HRV, the linear relationship observed here should be interpreted with some caution. After application of bootstrapped estimates, the relationship disappeared. However, if this result is not a statistical artifact, it is still certainly possible given that LF-HRV alone does not directly measure sympathetic activity, particularly given other contributing sources of physiological activity. It is also surprising that none of the covariates related to HRV, especially given findings that that poor sleep consistently produces reductions in HRV (e.g., Zhong et al., 2005). Prior night sleep duration for participants averaged eight hours. There may have been too little sleep deprivation across the sample to affect HRV.

Trait neuroticism also was unrelated to baseline RMSSD. None of the predictors in the model reached statistical significance. Given that HF-HRV and RMSSD both reflect parasympathetic modulation, the consistency in these results is unsurprising. The literature on the relationship between neuroticism at rest and HRV is highly mixed. One possibility is that previous findings demonstrating a relationship between neuroticism and HRV actually demonstrate a relationship between positive affect and HF-HRV/RMSSD. This is consistent with

work linking positive emotionality to heightened resting HF-HRV (Cribbet et al., 2011; Frazier et al., 2004; Oveis et al., 2009; Silvia et al., 2014; Z. Wang et al., 2013). Perhaps some studies find a connection between neuroticism and reduced HRV because the primary connection is actually between positive affect and HRV.

Hypothesis 2

Inconsistent with the hypotheses, task differences did not emerge for HF-HRV collected during MST and CPT. This prediction assumed that the MST would be a more demanding stress task given the additional attentional resources needed to perform adequately. Previous work found exaggerated responsivity to error is linked with increased neuroticism, resulting in exaggerated reactivity to errors leading to continued impairments in cognitive performance (M. D. Robinson et al., 2010). However, there are several reasons why a task-based difference in HF-HRV did not emerge. It is possible that the stress tasks induced a relatively greater activation of sympathetic activity without a corresponding decrease in HF-HRV, such that HF-HRV may not have been strongly impacted by either task. Descriptive statistics support this pattern. HF-HRV collected during the first relaxation phase (M = 6.05, SD = 1.19), the MST (M = 6.11, SD = 0.98), and the CPT (*M* = 6.12, *SD* = 1.09) are all nearly identical. Importantly, this indicates that neither stress task caused a significant withdrawal of parasympathetic tone as measured by HF-HRV. There was a significant relationship between stress appraisal and HF-HRV, such that those individuals that found the MST more stressful exhibited reduced HF-HRV, in line with the empirical research discussed above. Individuals with higher resting HF-HRV tend to perform better on cognitive tasks and are less susceptible to stress-induced errors in performance. An important caveat to this pattern of results is that the measurement of CVT is significantly more

accurate when using properly collected respiratory data. These results should be interpreted with significant caution.

Contrary to hypotheses, LF-HRV collected during the CPT was greater than LF-HRV collected during the MST. The most plausible explanation is differences in the difficulty of the two stress tasks. The MST involved repetitive serial subtraction of the same number from a four-digit number. CPT involved exposure to a vat of water chilled to 5 °C. The mean duration for the CPT was less than two of the five allotted minutes, suggesting participants found the task difficult. Although the mean accuracy for the MST was low, it is plausible that the induced vasoconstriction and increased norephinephrine and ephephrine secretion induced by the CPT led to increased perceptions of stress compared to the stress induced by error feedback delivered during the MST. Previous work found healthy volunteers rate the MST and CPT as similarly unpleasant (Albus et al., 1990). In that study, MST induced greater increases in heart rate while the CPT induced greater increases in epinephrine and norephrinephrine concentrations.

Likewise, RMSSD collected during the CPT was greater than RMSSD collected during the MST, also contrary to hypotheses. This is the most surprising finding in this hypothesis, largely because HF-HRV and RMSSD are highly correlated variables and are both assumed to reflect parasympathetic activation. However, there were significant issues with the normality of RMSSD collected during the CPT, which may have compromised the results of the LMMs. Results were also confirmed after running a bootstrap model to account for the non-normality. Given the similar pattern of findings, a more plausible explanation is that the accuracy of the measurement for HF-HRV and RMSSD may have been more strongly impacted by the absence of respiratory data. Beyond the observation that these metrics are more accurately determined with respiratory data, respiration can also serve as a significant confounder of HRV data unless it

is controlled for. There is no direct method to determine which is the best explanation. However, I argue that the overall pattern of results most likely indicates that the CPT in this study was more effective for stress induction given the differences observed in appraisal ratings and the differences observed in LF-HRV between the two stress tasks. However, although increased LF-HRV has often been correlated with increased stress, a causal relationship between the two should not be assumed.

Hypothesis 3

Task type did not moderate the relationship between trait neuroticism and any of the three HRV metrics. Several statistically significant covariates did exist. For example, participants that rated the MST task as more stressful had relatively lower HF-HRV during the MST. Participants that kept their hand submerged in the cold water for a longer duration had relatively greater HF-HRV during the CPT. In comparing the two stress tasks, LF-HRV was higher during CPT compared to MST, contrary to hypotheses. This result was contrary to prior studies (Albus et al., 1990; M. T. Allen & Crowell, 1989) . This may be due to differences in task difficulty. The MST in this study was a relatively straightforward set of continuous serial subtraction problems among University students, many of whom may have found the task easy. Although the participants were not directly questioned about the ease of both tasks, an exploratory analysis was conducted to compare stress appraisals on both tasks. Due to non-normality of the stress ratings, an exploratory Wilcoxon signed-ranked test found differences between the task ratings, $V = 509.5$, $p = .002$. Stress ratings were greater for the CPT ($M = 2.94$, $SD = 1.29$) than the MST ($M = 2.27$, $SD = 1.11$). This difference could account for differences in HRV between the tasks. Additionally, the CPT task proved to be challenging for most participants since the time duration participants could withstand the water for was shorter than anticipated ($M = 108$s, $SD = 103$s). It

is unclear whether increasing the difficulty of the MST or increasing the ease of the CPT would eliminate the differences observed in HRV metrics or swapped the direction of the effect. Future studies should consider better controls for math ability in relationship to MST paradigms. For example, a study might compare physiological differences between contrasting stress tasks of different difficulty levels.

Exploratory Analyses

To further probe relationships between personality and HRV metrics, additional exploratory analyses assessed associations between facet level traits and HRV metrics. This follows previous work demonstrating that the vulnerability facet of trait neuroticism relates to increased autonomic arousal to stress and increased sensitization to a mental arithmetic stressor (O'Súilleabháin et al., 2019). Using Spearman's rho and Benjamini-Hoch corrections for multiple comparisons, only two significant correlations between neuroticism facets and HRV metrics were observed: baseline LF-HRV and the facet trait of depression, $r_s(65) = .31$, $p < .001$, as well as LF-HRV collected during the final relaxation phase and depression, $r_s(65) = .32$, $p < .001$. This finding runs contrary to prior literature discussed earlier in this document linking depressive symptoms to reduced HRV, including both HF-HRV and LF-HRV. One possibilty is that relatively greater sympathetic overactivation is more common among individuals expressing greater depression facet scores due to higher sensitivty to stress exposure. Given that the participants in this sample knew they were being exposed to stressors, it is possible that this activated greater sympathetic activity at baseline more strongly among the individuals more prone to a depressive disposition. Nontheless, it is possible that these may be spurious relationships given the inconsistency in the findings and the fact that the number of items measuring each facet trait is relatively small ($n = 4$).

Change scores for HRV metrics collected during the three relaxation phases were also computed to assess recovery effects, given that post-stress recovery indices are often more useful predictors for understanding the relationship between physiological manifestations of stress and adverse health outcomes (Haynes et al., 1991). Between the first and second relaxation phases, HF-HRV increased slightly, indicating a relative increase in parasympathetic activity over time. However, this trend did not continue into the third relaxation phase. Consistent with this observation, RMSSD followed a similar pattern, increasing slightly between the first and second relaxation phases and showing no change between the second and third relaxation phases.

No average change in LF-HRV existed between baseline relaxation and the second relaxation phase. However, there was a modest increase in LF-HRV between the second and third relaxation phases, although the variability within individuals was quite large. Taken together, these results indicate that post-stress recovery was greater after exposure to the first stress task than it was after exposure to the second stress task. Such a finding fits with the fact that LF-HRV increased more after the second stress exposure, indicating a possibly greater increase in sympathetic activity and an increase in parasympathetic withdrawal.

Implications

The results of this investigation contain several possible implications for understanding the relationship between personality and HRV. First, is that the relationship between personality and HRV is not well characterized by the correlation between singular traits and HRV metrics. Instead, much like previous work (e.g., Shepherd et al., 2015), multiple measures of distress-related personality constructs should be combined to create profiles of 'adaptive' and 'maladaptive' personality functioning. Although the null findings observed in this study could be due to the simplicity of the single measure of neuroticism assessed, it is also possible that these

findings could be related to low statistical power, inaccurate assessment of HRV metrics, or insufficiently controlled stress tasks impacting the fidelity of the HRV recordings.

Several findings observed in this study are, however, compatible with previous work. For example, the NIM and the transactional model of stress and coping represent two compatible frameworks to understand the relationship between personality as one mediator of the stress process and HRV metrics as manifestations of the stress process. Importantly, in the transactional model of stress and coping, appraisal is viewed as a primary mediator shaping manifestation of the stress response (Vollrath, 2001).

There is substantial literature demonstrating associations between trait neuroticism, stress appraisal, and stress response. For example, participants with higher trait neuroticism experience relatively greater physiological arousal (measured by heart rate, skin conductance, and respiration) in response to viewing emotional and negatively valanced film scenes (Brumbaugh et al., 2013). This suggests the relatively greater stress reactivity associated with trait neuroticism results in a chronically over-taxed nervous system. As another example, patients with clinical burnout present with significantly lower HF-HRV than age- and sex-matched controls (Lennartsson et al., 2016). On the social engagement front, longitudinal evidence supports the idea that increases in HF-HRV often accompany increases in social engagement over time (Kok & Fredrickson, 2010). These increases in social engagement and in positive mood were most strongly felt among participants with higher resting CVT at baseline. These findings are in line with work describing HRV as reflecting a capacity for self-regulation, psychological flexibility, and social engagement (Kemp & Quintana, 2013). This description also shares common ground with the NIM, which proposes a biological model describing these physiological and psychological mechanisms.

The NIM proposes that integrated communication between a set of brain regions described as the central autonomic network (Berntson et al., 2007) generates top-down appraisals of situations that then influence the autonomic component of the stress process (Thayer et al., 2009). This model proposes a process by which stress exposures cause functional deactivation of prefrontal cortical circuits, leading to a relative dominance of sympathetically mediated activity that leads to pathology when sustained over long periods of time (Thayer & Brosschot, 2005). This state is indicated by low parasympathetically mediated HRV. Importantly, this process is related to a functional decrease in the role of inhibitory circuits that can lead to maladaptive, perseverative, and rigid behavior which is not optimally fitted to the environmental contexts individuals face (Thayer & Friedman, 2002). More recent work has elucidated a more detailed hierarchical account of the top-down control that different levels of the nervous system exhibit over the ANS (Smith et al., 2017), consistent with predictive coding accounts that have gained recent popularity in the broader literature (Clark, 2013).

A wide range of empirical investigations support the general pattern summarized in the NIM. For example, one manifestation of psychologically maladaptive behavior influenced by a failure of inhibition is spontaneous avoidance in response to aversive stimuli. Investigators have observed that individuals with relatively greater resting CVT exhibit reduced spontaneous avoidance in response to disgust-eliciting film clips (Aldao et al., 2016). In a similar investigation, researchers also observed that individuals with lower resting HF-HRV were both more likely to experience a more negatively valenced emotional reaction to aversive pictures and to engage in avoidance behaviors related to the aversive stimuli in the study (Katahira et al., 2014). Likewise, research among adolescents demonstrates that higher vagal tone is associated with more adaptive emotion regulation strategies (Santucci et al., 2008). Conversely, post-

stressor recovery as measured by increases in HF-HRV during recovery after stress exposure is impaired among individuals that experience difficulties in emotion regulation (Berna et al., 2014). Affective instability in daily life is also associated with reduced resting HF-HRV, another manifestation of emotion regulation difficulties (Koval et al., 2013). Researchers have also observed that higher resting state HF-HRV is associated with a greater capacity to inhibit conditioned fear. These researchers argued that higher HRV may reflect the capacity of prefrontal cortical activity to inhibit subcortical fear responses (Wendt et al., 2015).

In this investigation, a clear connection between stress appraisal and difficulties on the stress task was observed. Individuals that rated the MST as more stressful made more errors and individuals that rated the CPT as more stressful endured the cold water for less time. Both trait and state rumination have been linked to stress appraisal and reduced HF-HRV recovery after exposure to stress (Key et al., 2008), which accords with the stress appraisal findings in this study. Researchers have also employed eye-tracking in a similar paradigm, observing that individuals with lower resting HF-HRV attend more to error feedback than the subsequent cognitive task, partly accounting for the increased error rates among those individuals (Azam et al., 2018). In another stress paradigm where participants were exposed to conditions of low cognitive load and high cognitive load, investigators observed a revealing pattern: individuals with higher resting HF-HRV had increased HF-HRV during the low load condition and no decrease in HF-HRV under the high load condition (Park et al., 2014). On the other hand, those participants with lower resting HF-HRV experienced reductions in HF-HRV under both the low and high load conditions. Individuals with higher resting HF-HRV more effectively engage top-down and bottom-up cognitive modulation of emotional stimuli while individuals with lower HF-HRV engage in more maladaptive and hyper-vigilant responses to emotional stimuli (Park &

Thayer, 2014). Nonetheless, it is still surprising that neuroticism was unrelated to HF-HRV in this sample, given that this trait is strongly linked to the maladaptive coping responses in both cross-sectional and longitudinal samples (Vollrath et al., 1995). These results should be interpreted with caution due to the inability to include respiration in measuring HF-HRV.

Both the NIM and the transactional model of stress and coping are useful explanatory accounts for this study's results. Although measurement issues do call some of these results into question, this study did replicate some existing patterns in the literature. Although there are many possible explanations for the null findings observed in this study, the findings that did replicate represent an important contribution. Most importantly, I argue that personality profiles comprised of overlapping traits and stress tasks of varying difficulty and complexity should be compared in future investigations with larger sample sizes.

Limitations

A key limitation for this project was the HRV methodology, specifically the inability to include respiration as a physiological outcome. HF-HRV is more accurate when calculated in synchrony with respiration (Draghici & Taylor, 2016). Some of the HF-HRV findings that were contrary to my predictions may result from unreliable calculations. Nonetheless, recent data indicate that both RMSSD and HF-HRV have good test-retest reliability over the course of a year using the intra-class correlation coefficient approach, in both healthy controls and individuals with a history of depressive symptoms (Seidman et al., 2024). The strong correlations observed between HRV metrics within individuals across the experimental phases offer additional support for the reliability of these metrics. A second limitation involves the cross-sectional nature of data collection. Future research should aim to continue exploring the relevant associations longitudinally. A third limitation is the lack of a diverse sample. Previous work

found ethnic differences in HRV both at rest and during task (X. Wang et al., 2005). It is plausible that the associations in this study may not be reliable in larger or more representative samples.

An additional limitation was the nature of the stress tasks. Reproducibility in physiological responses such as heart rate and blood pressure in response to the CPT is relatively low (Fasano et al., 1996). Additionally, the task duration for most participants performing this task was shorter than expected or desired. Most participants completing the CPT task found the task so difficult they could not keep their hand submerged in the water for more than two minutes, or participants found it easy enough to keep their hand in the water for full five minute period. Although recent research suggests reliability and validity of HRV estimates can be achieved with a 1-2 minute recording window (Burma et al., 2021), a significant number of participants were unable to keep their hand submerged for that period of time. Repeatability of HRV measures tends to exceed an intra-class correlation coefficient of 0.7 when the recording window is six minutes or greater and tend to exceed 0.5 when the recording window is two minutes or greater (Schroeder et al., 2004). The accuracy of HRV collected during the CPT is a critically important limitation to consider when interpreting these results. Consequently, it is possible that the null effects observed in this study may be a consequence of unreliable HRV calculation. Although the recording periods were five minutes for every other phase of the experiment, it is possible that the direct comparison to the CPT may have jeopardized the ability to reliably determine if HRV metrics properly vary between the tasks.

Conclusion

This area of research is ripe with unanswered questions. Given that the existing literature is mixed, the results of this study are unsurprising. However, these results do point to the

importance of variability, not only of the physiological rhythms that regulate our existence, but variability in the patterns observed in the literature linking personality traits and physiological stress responses. Future work aimed at characterizing the relationships that remain constant across studies certainly has its work cut out for it. As new techniques for analyzing the variability of physiological rhythms proliferate, there will be continued need to explore both the psychological correlates of these methods and develop further causal models elucidating the complex bi-directional relationship between traits and biological states. Such future work remains an ever open and evolving quest to untangle the variability at the heart of each and every organism's fundamental ability to adapt.

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Appendix A: Prescreen/Information Letter

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Welcome- Personality and Cardiovascular Stress Reactivity Study

You are invited to participate in a research study examining how personality traits influence cardiovascular stress responses to different kinds of tasks that evoke stress. This research study is being conducted by Mackenzie J. Leavitt, Doctoral Candidate at Auburn University, and Dr. Jennifer L. Robinson, Full Professor at Auburn University. You were selected as a possible participant because you expressed interest via email or SONA Systems.

What will be involved if you participate? If you decide to participate in Part 1 of this research study, you will be asked to complete online questionnaires. The questionnaires will relate to mental health, physical health, substance use, and personality traits. Completing these questionnaires should take between 15 and 30 minutes. Based on your responses to specific questions, you may be eligible to participate in Phase 2 of this research study, which will involve electrocardiogram/respiration recordings during stress tasks.

Are there risks or discomforts? The risks associated with participating in Phase 1 of this research study are that you experience emotional distress that could result from thinking about certain topics (e.g., mental health, your life experiences). If you find yourself experiencing distress, you may discontinue participation at any time. Should you decide to discontinue, you would receive research hours via SONA Systems that correspond to time spent completing the questionnaires. If you wish to speak with someone about your distress, a reference list of resources in the Auburn- Opelika area will be available following the questionnaires. Also, you can request a copy of the reference list by contacting the investigators listed on this letter. There are also risks associated with confidentiality breaches. To minimize this risk, only investigators have access to data obtained in connection with the research study that can be identified as belonging to you. If you decide to withdraw, you may withdraw any data that has been collected as long at it is identifiable. You will be assigned a participant number so that your name and other pieces of identifying information are not directly associated with data collected. All data, including your responses to these questionnaires, will be associated with that participant number. Following data collection completing, any/all links to identifiable information will be destroyed. The results of this study may be presented in a professional venue, such as a journal or conference. In such an event, group data will be presented.

Will you receive compensation? During Phase 1, you will be compensated for participation with one research hour via Sona Systems. Your instructors should assign specific values of course credit to these hours. Please check with your instructors for more information. If you are not enrolled in Auburn University, SONA participation will not be available to you. After completing Phase 2, you will be compensated $15 for the completion of both stress tasks,

as well as one additional hour of SONA credit, for a total of two hours of SONA credit for the completion of both parts.

Are there benefits to yourself or others? If you participate in Phase 1 of this research study, you can expect to receive no direct personal benefits.

Are there costs? If you decide to participate in this research study, you will not incur any costs. If you require medical attention, you will be responsible for all costs for medical attention/treatment. If you change your mind about participating, you can withdraw from the research study at any time. Your participation is completely voluntary. If you choose to withdraw, your data can be withdrawn as long as it is identifiable. Your decision about whether or not to participate will not jeopardize your relationship with Auburn University, or any associated/affiliated department, center, or office.

If you have questions about this research study, please ask them now. Alternatively, you can contact Mackenzie J. Leavitt, at mjl0062@auburn.edu, or Dr. Jennifer L. Robinson, jrobinson@auburn.edu, who are the research study investigators. A copy of this document will be given to you for your records at your request. If you have questions about your rights as a research participant, you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board by phone (334) 844-5966 or email at hsubjec@auburn.edu or IRBchair@auburn.edu.

HAVING READ THE INFORMATION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. PLEASE SELECT THE ORANGE ARROW KEY TO INDICATE YOUR AGREEMENT TO PARTICIPATE AND BEGIN THE SURVEY. You may print a copy of this information letter to keep for your records.

→

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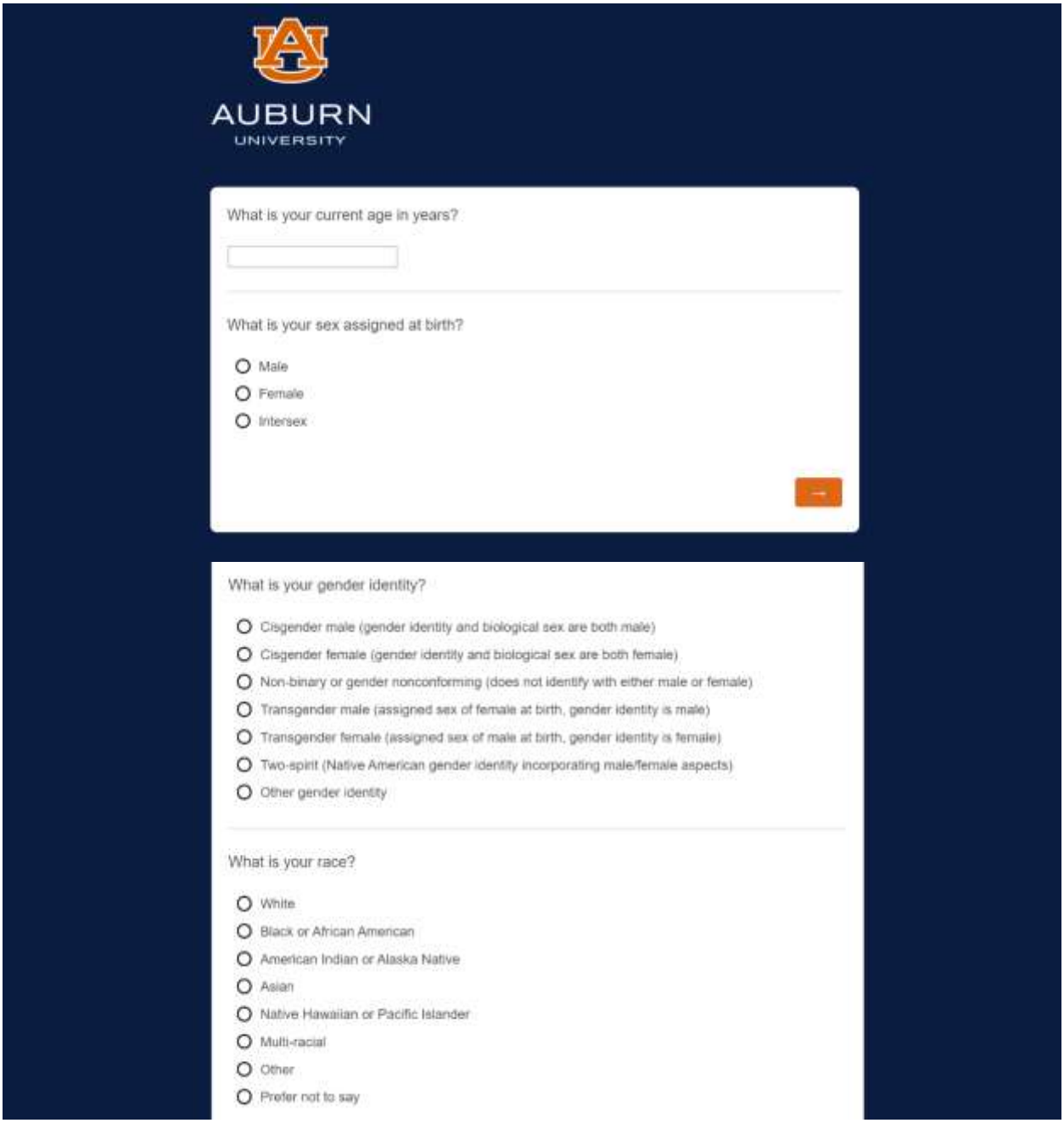
Please enter your Auburn University email in the text box below. If you do not have an Auburn University email, please enter your most frequently used email address.

→


AUBURN
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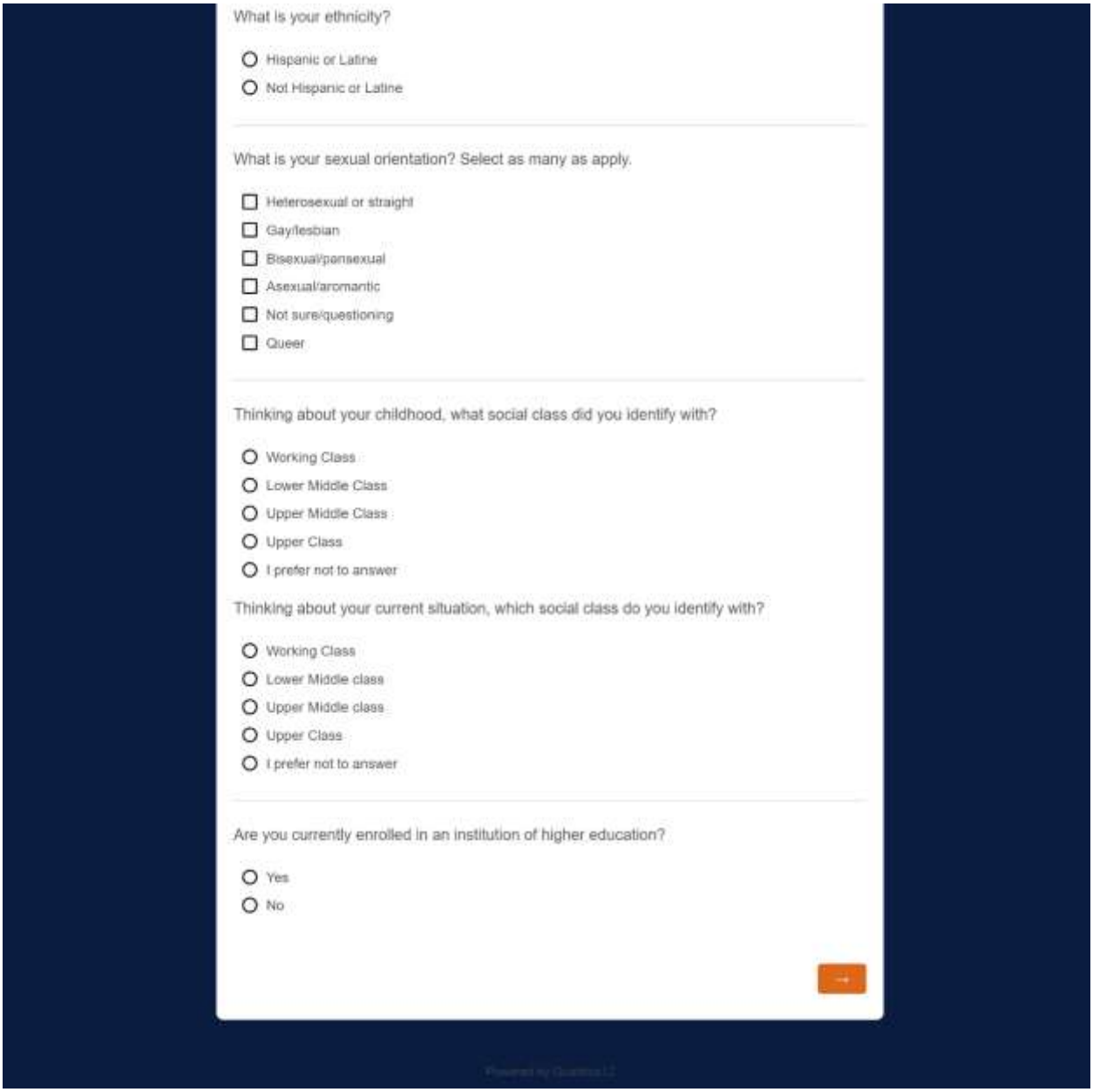
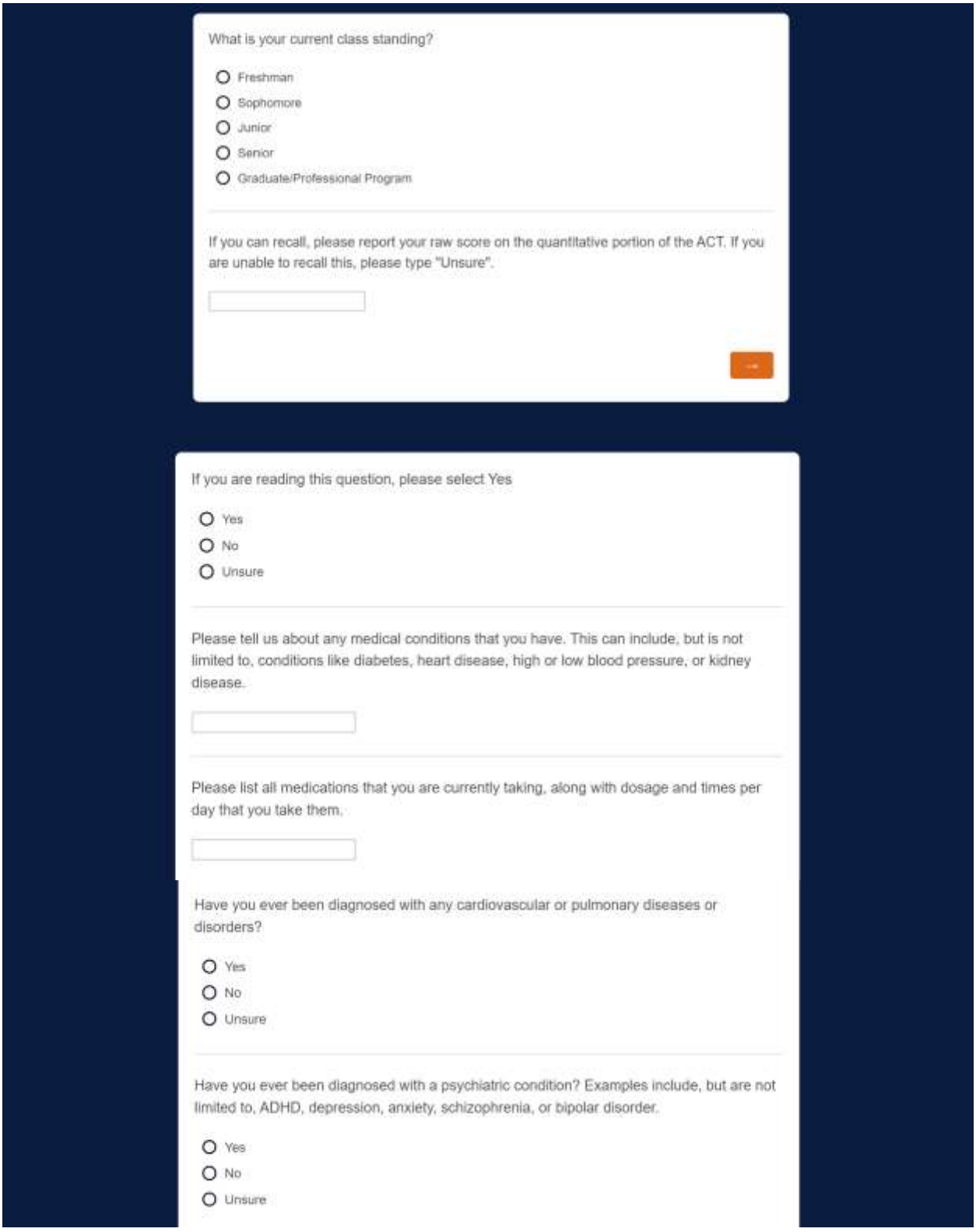
What is your current age in years?
What is your sex assigned at birth?
Male
Female
Intersex
What is your gender identity?
Cisgender male (gender identity and biological sex are both male)
Cisgender female (gender identity and biological sex are both female)
Non-binary or gender nonconforming (does not identify with either male or female)
Transgender male (assigned sex of female at birth, gender identity is male)
Transgender female (assigned sex of male at birth, gender identity is female)
Two-spirit (Native American gender identity incorporating male/female aspects)
Other gender identity
What is your race?
White
Black or African American
American Indian or Alaska Native
Asian
Native Hawaiian or Pacific Islander
Multi-racial
Other
Prefer not to say


What is your ethnicity?
Hispanic or Latine
Not Hispanic or Latine
What is your sexual orientation? Select as many as apply.
Heterosexual or straight
Gay/lesbian
Bisexual/pansexual
Asexual/aromantic
Not sure/questioning
Queer
Thinking about your childhood, what social class did you identify with?
Working Class
Lower Middle Class
Upper Middle Class
Upper Class
I prefer not to answer
Thinking about your current situation, which social class do you identify with?
Working Class
Lower Middle class
Upper Middle class
Upper Class
I prefer not to answer
Are you currently enrolled in an institution of higher education?
Yes
No


What is your current class standing?
Freshman
Sophomore
Junior
Senior
Graduate/Professional Program
If you can recall, please report your raw score on the quantitative portion of the ACT. If you are unable to recall this, please type "Unsure".
If you are reading this question, please select Yes
Yes
No
Unsure
Please tell us about any medical conditions that you have. This can include, but is not limited to, conditions like diabetes, heart disease, high or low blood pressure, or kidney disease.
Please list all medications that you are currently taking, along with dosage and times per day that you take them.
Have you ever been diagnosed with any cardiovascular or pulmonary diseases or disorders?
Yes
No
Unsure
Have you ever been diagnosed with a psychiatric condition? Examples include, but are not limited to, ADHD, depression, anxiety, schizophrenia, or bipolar disorder.
Yes
No
Unsure

Please list any/all mental health related diagnoses you have previously received in the text box provided below. If you are uncomfortable disclosing, please put "Do not wish to answer."

Are you currently taking any antidepressant medications?

- Yes
- No
- Unsure

Are you currently taking any antipsychotic medications?

- Yes
- No
- Unsure

Are you currently taking any antihypertensive medications (used to treat high blood pressure)?

- Yes
- No
- Unsure

Are you currently taking any oral contraceptives?

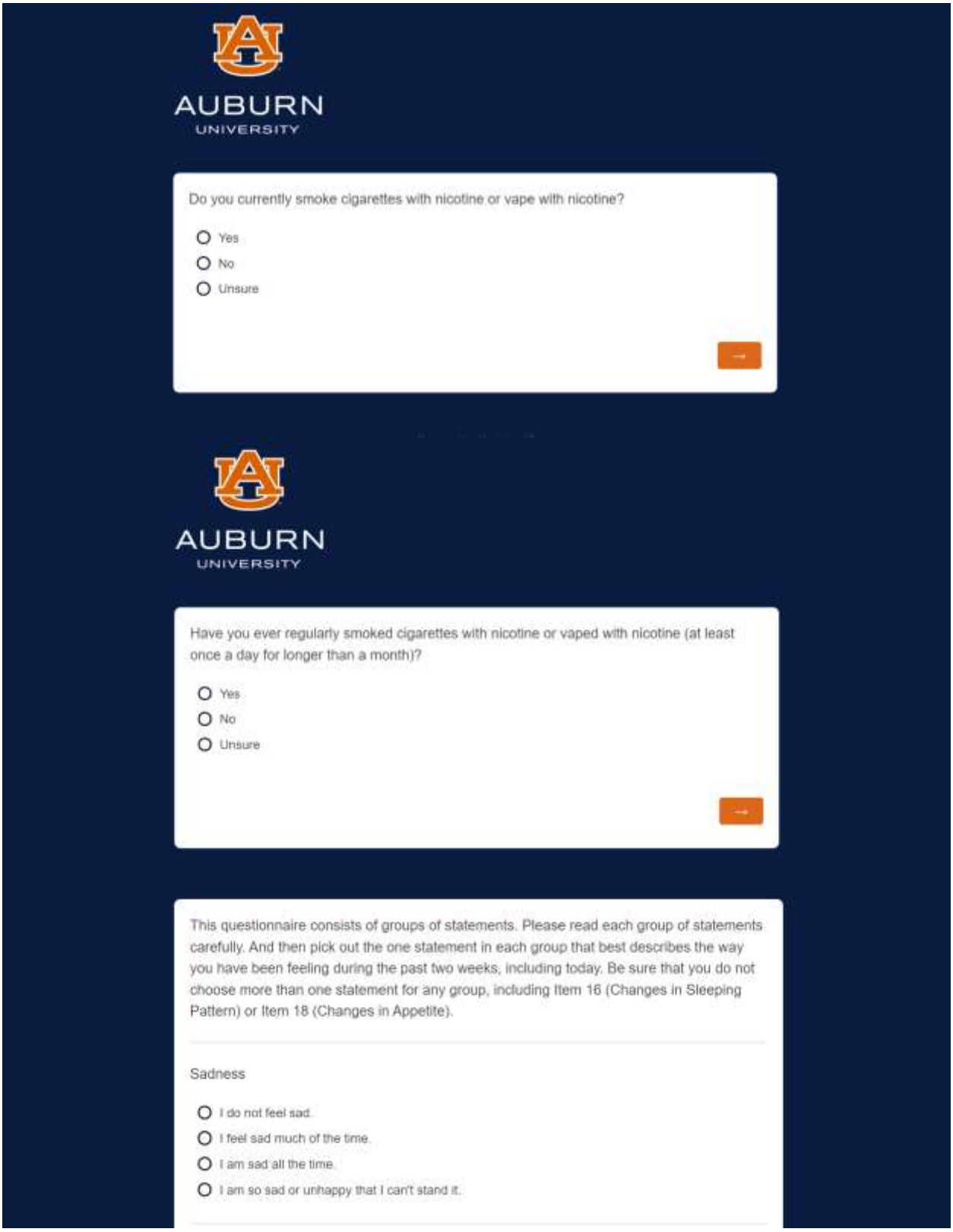
- Yes
- No
- Unsure

Do you consider yourself to be generally healthy?

- Yes
- No
- Unsure

If you are reading this, please select Unsure

- Yes
- No
- Unsure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Do you currently smoke cigarettes with nicotine or vape with nicotine?
Yes
No
Unsure
AUBURN
UNIVERSITY
Have you ever regularly smoked cigarettes with nicotine or vaped with nicotine (at least once a day for longer than a month)?
Yes
No
Unsure
This questionnaire consists of groups of statements. Please read each group of statements carefully. And then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).
Sadness
I do not feel sad.
I feel sad much of the time.
I am sad all the time.
I am so sad or unhappy that I can't stand it.

Pessimism

- I am not discouraged about my future.
- I feel more discouraged about my future than I used to.
- I do not expect things to work out for me.
- I feel my future is hopeless and will only get worse.

Past Failure

- I do not feel like a failure.
- I have failed more than I should have.
- As I look back, I see a lot of failures.
- I feel I am a total failure as a person.

Loss of Pleasure

- I get as much pleasure as I ever did from the things I enjoy.
- I don't enjoy things as much as I used to.
- I get very little pleasure from the things I used to enjoy.
- I can't get any pleasure from the things I used to enjoy.

Guilty Feelings

- I don't feel particularly guilty.
- I feel guilty over many things I have done or should have done.
- I feel quite guilty most of the time.
- I feel guilty all of the time.

Punishment Feelings

- I don't feel I am being punished.
- I feel I may be punished.
- I expect to be punished.
- I feel I am being punished.

Self-Dislike

- I feel the same about myself as ever.
- I have lost confidence in myself.
- I am disappointed in myself.
- I disklike myself.

Self-Criticalness

- I don't criticize or blame myself more than usual.
- I am more critical of myself than I used to be.
- I criticize myself for all of my faults.
- I blame myself for everything bad that happens.

Crying

- I don't cry anymore than I used to.
- I cry more than I used to.
- I cry over every little thing.
- I feel like crying, but I can't.

→

Agitation

- I am no more restless or wound up than usual.
- I feel more restless or wound up than usual.
- I am so restless or agitated, it's hard to stay still.
- I am so restless or agitated that I have to keep moving or doing something.

Loss of Interest

- I have not lost interest in other people or activities.
- I am less interested in other people or things than before.
- I have lost most of my interest in other people or things.
- It's hard to get interested in anything.

If you are reading this, please select Yes

- Yes
- No
- Unsure

Indecisiveness

- I make decisions about as well as ever.
- I find it more difficult to make decisions than usual.
- I have much greater difficulty in making decisions than I used to.
- I have trouble making any decisions.

Worthlessness

- I do not feel I am worthless.
- I don't consider myself as worthwhile and useful as I used to.
- I feel more worthless as compared to others.
- I feel utterly worthless.

Loss of Energy

- I have as much energy as ever.
- I have less energy than I used to have.
- I don't have enough energy to do very much.
- I don't have enough energy to do anything.

Changes in Sleeping Pattern

- I have not experienced any change in my sleeping.
- I sleep somewhat more than usual.
- I sleep somewhat less than usual.
- I sleep a lot more than usual.
- I sleep a lot less than usual.
- I sleep most of the day.
- I wake up 1-2 hours early and can't get back to sleep.

Irritability

- I am not more irritable than usual.
- I am more irritable than usual.
- I am much more irritable than usual.
- I am irritable all the time.

Changes in Appetite

- I have not experienced any change in my appetite.
- My appetite is somewhat less than usual.
- My appetite is somewhat greater than usual.
- My appetite is much less than before.
- My appetite is much greater than usual.
- I have no appetite at all.
- I crave food all the time.

Concentration Difficulty

- I can concentrate as well as ever.
- I can't concentrate as well as usual.
- It's hard to keep my mind on anything for very long.
- I find I can't concentrate on anything.

Tiredness or Fatigue

- I am no more tired or fatigued than usual.
- I get more tired or fatigued more easily than usual.
- I am too tired or fatigued to do a lot of the things I used to do.
- I am too tired or fatigued to do most of the things I used to do.

Loss of interest in sex.

- I have not noticed any recent change in my interest in sex.
- I am less interested in sex than I used to be.
- I am much less interested in sex now.
- I have lost interest in sex completely.

→

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past month, including today, by selecting the option that best describes how much this symptom bothered you during the last month.

	Not at all	Mildly, but it didn't bother me much	Moderately- it wasn't pleasant at times	Severely- it bothered me a lot
Numbness or tingling	○	○	○	○
Feeling hot	○	○	○	○
Wobbliness in legs	○	○	○	○
Unable to relax	○	○	○	○
Fear of worst happening	○	○	○	○
Dizzy or lightheaded	○	○	○	○
Heart pounding/racing	○	○	○	○
Unsteady	○	○	○	○
Terrified or afraid	○	○	○	○
Nervous	○	○	○	○

→

	Not at all	Mildly, but it didn't bother me much	Moderately- it wasn't pleasant at times.	Severely- it bothered me a lot
Feeling of choking	O	O	O	O
Hands trembling	O	O	O	O
Shaky/unsteady	O	O	O	O
Fear of losing control	O	O	O	O
Difficulty in breathing	O	O	O	O
Fear of dying	O	O	O	O
Scared	O	O	O	O
Indigestion	O	O	O	O
Faint/lightheaded	O	O	O	O
Face flushed	O	O	O	O
Hot/cold sweats	O	O	O	O

If you are reading this, please select No

- O Yes
- O No
- O Unsure

→

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The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Worry about things.	O	O	O	O	O
Make friends easily.	O	O	O	O	O
Have a vivid imagination.	O	O	O	O	O
Trust others.	O	O	O	O	O
Complete tasks successfully.	O	O	O	O	O
Get angry easily.	O	O	O	O	O
Love large parties.	O	O	O	O	O
Believe in the importance of art.	O	O	O	O	O
Use others for my own ends.	O	O	O	O	O
Like to tidy up.	O	O	O	O	O

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Often feel blue.	○	○	○	○	○
Take charge.	○	○	○	○	○
Experience my emotions intensely.	○	○	○	○	○
Love to help others.	○	○	○	○	○
Keep my promises.	○	○	○	○	○
Find it difficult to approach others.	○	○	○	○	○
Am always busy.	○	○	○	○	○
Prefer variety to routine.	○	○	○	○	○
Love a good fight.	○	○	○	○	○
Work hard.	○	○	○	○	○

If you are reading this, please select Very Inaccurate

- ○ Very Inaccurate
- ○ Moderately Inaccurate
- ○ Neither Accurate nor Inaccurate
- ○ Moderately Accurate
- ○ Very Accurate

→

Powered by Qualtrics

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Go on binges.	○	○	○	○	○
Love excitement.	○	○	○	○	○
Love to read challenging material.	○	○	○	○	○
Believe that I am better than others.	○	○	○	○	○
Am always prepared.	○	○	○	○	○
Panic easily.	○	○	○	○	○

Radiate joy.	○	○	○	○	○
Tend to vote for liberal (progressive) political candidates.	○	○	○	○	○
Sympathise with the homeless.	○	○	○	○	○
Jump into things without thinking.	○	○	○	○	○

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Fear for the worst.	○	○	○	○	○
Feel comfortable around people.	○	○	○	○	○
Enjoy wild flights of fantasy.	○	○	○	○	○
Believe that others have good intentions.	○	○	○	○	○
Excel in what I do.	○	○	○	○	○
Get irritated easily.	○	○	○	○	○
Talk to a lot of different people at parties.	○	○	○	○	○
See beauty in things that others might not notice.	○	○	○	○	○
Cheat to get ahead.	○	○	○	○	○
Often forget to put things back in their proper place.	○	○	○	○	○

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Dislike myself.	O	O	O	O	O
Try to lead others.	O	O	O	O	O
Feel others' emotions.	O	O	O	O	O
Am concerned about others.	O	O	O	O	O
Tell the truth.	O	O	O	O	O
Am afraid to draw attention to myself.	O	O	O	O	O
Am always on the go.	O	O	O	O	O
Prefer to stick with things that I know.	O	O	O	O	O
Yell at people.	O	O	O	O	O
Do more than what's expected of me.	O	O	O	O	O

If you are reading this, please select Very Accurate

- O Very Inaccurate
- O Moderately Inaccurate
- O Neither Accurate nor Inaccurate
- O Moderately Accurate
- O Very Accurate

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Rarely overindulge.	O	O	O	O	O
Seek adventure.	O	O	O	O	O
Avoid philosophical discussions.	O	O	O	O	O
Think highly of myself.	O	O	O	O	O
Carry out my plans.	O	O	O	O	O

Become overwhelmed by events.	○	○	○	○	○
Have a lot of fun.	○	○	○	○	○
Believe that there is no absolute right or wrong.	○	○	○	○	○
Feel sympathy for those who are worse off than myself.	○	○	○	○	○
Make rash decisions.	○	○	○	○	○

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Am often down in the dumps.	○	○	○	○	○
Take control of things.	○	○	○	○	○
Rarely notice my emotional reactions.	○	○	○	○	○
Am indifferent to the feelings of others.	○	○	○	○	○
Break rules.	○	○	○	○	○
Only feel comfortable with friends.	○	○	○	○	○
Do a lot in my spare time.	○	○	○	○	○
Dislike changes.	○	○	○	○	○
Insult people.	○	○	○	○	○
Do just enough work to get by.	○	○	○	○	○

If you are reading this, please select Neither Accurate nor Inaccurate

- ○ Very Inaccurate
- ○ Moderately Inaccurate
- ○ Neither Accurate nor Inaccurate
- ○ Moderately Accurate
- ○ Very Accurate

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Easily resist temptations.	○	○	○	○	○
Enjoy being reckless.	○	○	○	○	○
Have difficulty understanding abstract ideas.	○	○	○	○	○
Have a high opinion of myself.	○	○	○	○	○
Waste my time.	○	○	○	○	○
Feel that I'm unable to deal with things.	○	○	○	○	○
Love life.	○	○	○	○	○
Tend to vote for conservative political candidates.	○	○	○	○	○
Am not interested in other people's problems.	○	○	○	○	○
Rush into things.	○	○	○	○	○

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Get stressed out easily.	○	○	○	○	○
Keep others at a distance.	○	○	○	○	○
Like to get lost in thought.	○	○	○	○	○
Distrust people.	○	○	○	○	○
Know how to get things done.	○	○	○	○	○

Am not easily annoyed.	O	O	O	O	O
Avoid crowds.	O	O	O	O	O
Do not enjoy going to art museums.	O	O	O	O	O
Obstruct other's plans.	O	O	O	O	O
Leave my belongings around.	O	O	O	O	O

If you are reading this question, please select Moderately Accurate

- O Very Inaccurate
- O Moderately Inaccurate
- O Neither Accurate nor Inaccurate
- O Moderately Accurate
- O Very Accurate

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Feel comfortable with myself.	O	O	O	O	O
Wait for others to lead the way.	O	O	O	O	O
Don't understand people who get emotional.	O	O	O	O	O
Take no time for others.	O	O	O	O	O
Break my promises.	O	O	O	O	O

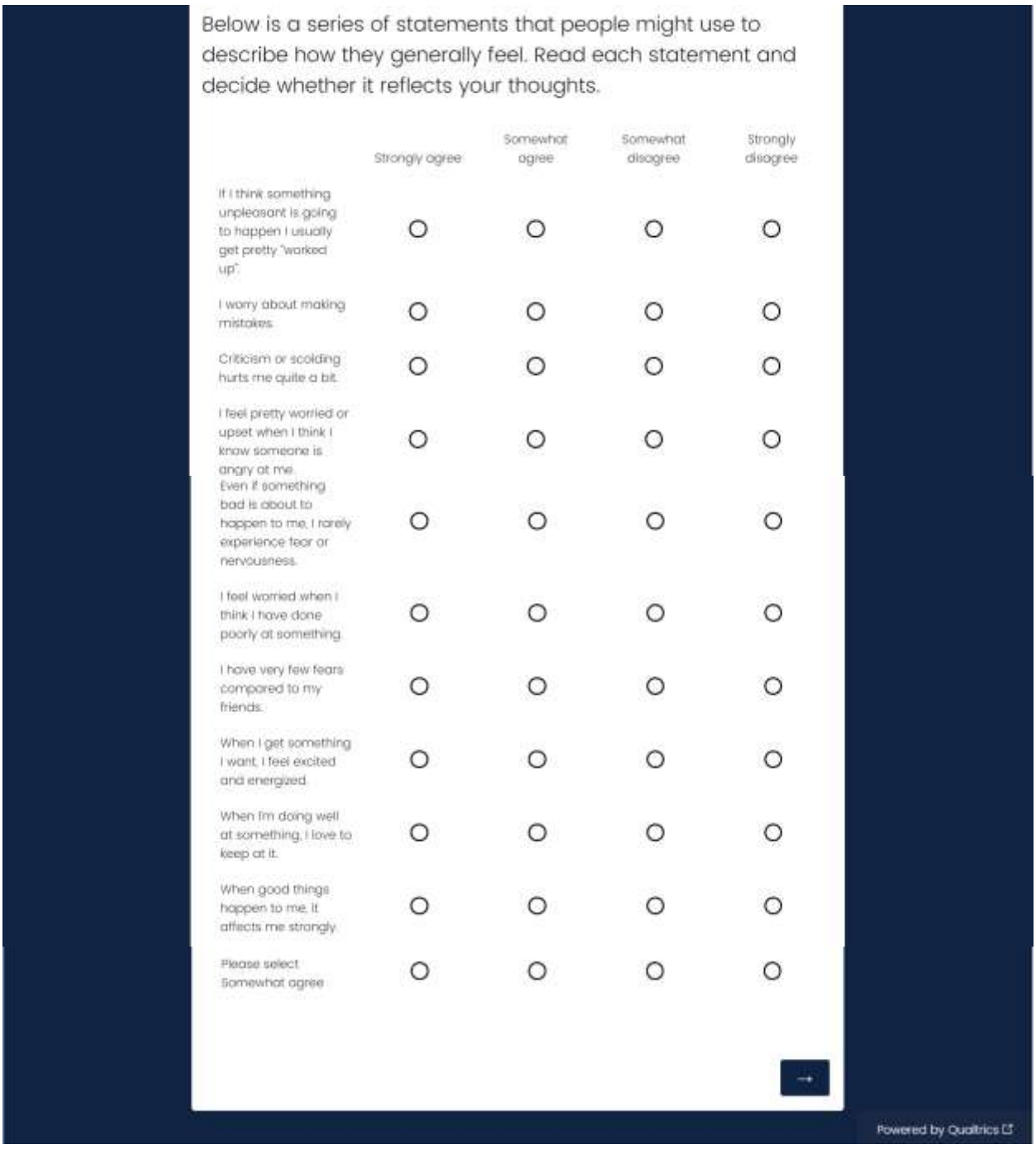
Take no time for others.	○	○	○	○	○
Break my promises.	○	○	○	○	○
Am not bothered by difficult social situations.	○	○	○	○	○
Like to take it easy.	○	○	○	○	○
Am attached to conventional ways.	○	○	○	○	○
Get back at others.	○	○	○	○	○
Put little time and effort into my work.	○	○	○	○	○

→

The following statements describe people's behaviors. Please select how accurately each statement describes you. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age.

	Very Inaccurate	Moderately Inaccurate	Neither Accurate nor Inaccurate	Moderately Accurate	Very Accurate
Am able to control my cravings.	○	○	○	○	○
Act wild and crazy.	○	○	○	○	○
Am not interested in theoretical discussions.	○	○	○	○	○
Boast about my virtues.	○	○	○	○	○
Have difficulty starting tasks.	○	○	○	○	○
Remain calm under pressure.	○	○	○	○	○
Look at the bright side of life.	○	○	○	○	○
Believe that we should be tough on crime.	○	○	○	○	○
Try not to think about the needy.	○	○	○	○	○
Act without thinking.	○	○	○	○	○

→


Below is a series of statements that people might use to describe how they generally feel. Read each statement and decide whether it reflects your thoughts.
Strongly agree
Somewhat agree
Somewhat disagree
Strongly disagree
If I think something unpleasant is going to happen I usually get pretty "worked up".
I worry about making mistakes.
Criticism or scolding hurts me quite a bit.
I feel pretty worried or upset when I think I know someone is angry at me.
Even if something bad is about to happen to me, I rarely experience fear or nervousness.
I feel worried when I think I have done poorly at something.
I have very few fears compared to my friends.
When I get something I want, I feel excited and energized.
When I'm doing well at something, I love to keep at it.
When good things happen to me, it affects me strongly.
Please select Somewhat agree
Powered by Qualtrics


Below is a series of statements that people might use to describe how they generally feel. Read each statement and decide whether it reflects your thoughts.
Strongly agree
Somewhat agree
Somewhat disagree
Strongly disagree
It would excite me to win a contest.
When I see an opportunity for something I like, I get excited right away.
When I want something, I usually go all-out to get it.
I go out of my way to get things I want.
If I see a chance to get something I want, I move on it right away.
When I go after something I use a "no holds barred" approach.
I will often do things for no other reason than that they might be fun.
I crave excitement and new sensations.
I'm always willing to try something new if I think it will be fun.
I often act on the spur of the moment.
Powered by Qualtrics

Appendix B: Subjective Stress Questionnaire

On the following scale from 0-5, please circle the number that corresponds to how stressful you found the task that you just performed.

No Stress	Some Stress	Mild Stress	Moderate Stress	Intense Stress	Extreme Stress
0	1	2	3	4	5

Appendix C: Stereotype Vulnerability Scale

The following questions are about how you feel about being a man or a woman, and about how these feelings may affect your perceptions of your math ability. For some people their gender is a major concern; for others it is less imponant. We would like you to consider your gender and respond to the following statements on the basis of how you feel about your gender. There are no right or wrong answers to any of these statements; we are interested in your honest reactions and opinions.

I. The experimenters expected me to do poorly on the test because of my gender.

1 2 3 4 5 6 7
strongly disagree strongly agree

2. The test may have been easier for people of my gender.

1 2 3 4 5 6 7
strongly disagree strongly agree

3. I doubt that others would **think** that I have less math ability because of my gender.

1 2 3 4 5 6 7
strongly disagree strongly agree

4. Some people feel I have less math ability because of my gender.

1 2 3 4 5 6 7
strongly disagree strongly agree

5. People of my gender rarely face unfair evaluations in math classes.

1 2 3 4 5 6 7
strongly disagree strongly agree

6. In math classes people of my gender often face biased evaluations from others.

1 2 3 4 5 6 7
strongly disagree strongly agree

7. My gender does not affect people's perception of my math ability.

1 2 3 4 5 6 7
strongly disagree strongly agree

8. In math classes I often feel that others look down on me because of my gender.

1 2 3 4 5 6 7
strongly disagree strongly agree

Appendix D: Participant Payment Form

PARTICIPANT PAYMENT FORM

Date completed:	
Participant name:	
Participant address:	
Amount:	
Auburn University, 90#	

Please check one if applies:

AU Student_____ AU Employee_____

Note: Receipt of funds from this study will be considered as income for tax and/or financial aid purposes. *Non-resident aliens do not have a de-minimus for reporting and withholding agents are required to withhold at the time of payment. The individual will receive a 1042s tax form to file with their tax return. Due to this withholding, the participant will not receive full payment due to taxes.*

Appendix E: Study Recruiting

Social Media Posts

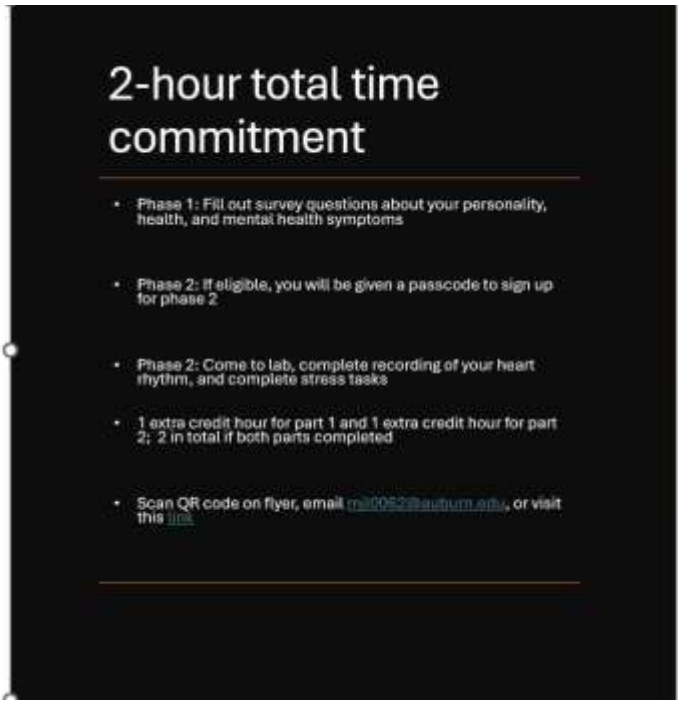
Version 1

We are currently recruiting for a research study examining the effects of personality traits on cardiovascular stress responses. If you are between the ages of 18 -50, you may be eligible to participate. The study will take approximately 2 hours of your time. To learn more, please visit this link, or email mjl0062@auburn.edu.

Version 2

Have you ever wondered how your personality traits might affect your response to stress? We're conducting a laboratory experiment to determine the effects that personality traits have on cardiovascular stress responses. To be eligible, you must be between the ages of 18-50, and you must answer a series of surveys and questionnaires. To learn more, please visit this link, or email mjl0062@auburn.edu.

PowerPoint Advertisement in Classes



Email Invitation to Part 2

Dear <insert name>,

Congratulations! You are eligible to participate in phase 2 of our research study entitled, "Personality and Cardiovascular Stress Reactivity Part 2".

I am <insert name>, and I am a <insert title> in the Department of Psychological Sciences at Auburn University.

You are receiving this email because you expressed interest in our study, and you completed phase 1.

Based on your responses in phase 1, you are eligible to participate in phase 2 of the study. Now that you are eligible to participate, your next step is to sign up for a time slot on SONA.

Here is a link to sign up for phase 2, which will involve an in-person lab visit to complete stress tasks while undergoing heart rhythm recordings:
You should have received a passcode at the end of the phase 1 study.
As a reminder, the passcode you will need to sign up phase 2 is the following: aucanlab. When you enter the passcode, you should be able to pick a timeslot to sign up for. You will receive a reminder email 48 hours and 24 hours prior to your scheduled timeslot. You will also receive one the morning of, should you choose to participate.

DEPARTMENT OF PSYCHOLOGICAL SCIENCES RESEARCH PARTICIPATION OPPORTUNITIES
DEPARTMENT OF PSYCHOLOGICAL SCIENCES RESEARCH PARTICIPATION OPPORTUNITIES
PSYCHOLOGICAL SCIENCES:
auburn.sona-systems.com

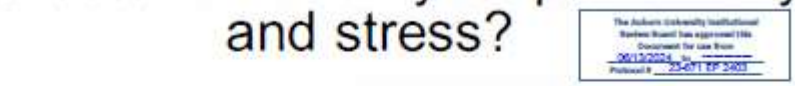
We look forward to working with you soon!
Confirmation of Part 2 Study Time Slot
Thank you for signing up for Part 2 of a research study entitled, "Personality and Cardiovascular Stress Reactivity."
As a reminder, your involvement will include a visit to the Cognitive and Affective Neuroscience Laboratory at Thach Hall on Auburn University Main Campus in Room 108. Thach Hall is located in the Haley Concourse, directly across from the Haley building. If you need additional instructions, please email me and those will be provided to you.
You signed up to participate on <insert date>, at <insert time>.
In order to participate in part 2, we will ask you to complete the following instructions:

- Record your bedtime and your wake-up time the night and morning before your scheduled time.
- Do not complete intense physical activity/training 24 hours before your sign-up time.
- Do not consume any alcohol 24 hours prior to your scheduled time
- Do not consume any caffeine 4 or less hours prior to your scheduled time.
- Do not consume any meals 2 hours before your scheduled time

You will also receive a reminder email with these instructions 48 hours prior to your scheduled study time and a reminder email the morning of your study date.
Please email me if you have any questions or concerns prior to your participation date.

Community Flyer

Interested in a study on personality and stress?



Researchers at Auburn University are interested in studying the heart's response to stress based on personality traits.

If you are between 18 and 50 years old, you may be eligible to participate!

- Complete online surveys to determine eligibility for lab visit (You will receive one hour SONA credit for completing surveys)
- If eligible for lab visit, complete stress tasks in lab while undergoing heart recordings (You will receive $15 for a one hour lab visit and 1 hour SONA credit)
- 2 hours of SONA credit for both parts

Email mjl0062@auburn.edu

OR

Scan the QR code to find out if you are eligible!

Auburn University, Department of Psychological Sciences

Personality Stress Study
mjl0062@auburn.edu

Appendix F: Informed Consent Document

DEPARTMENT OF PSYCHOLOGICAL SCIENCES

AUBURN UNIVERSITY
COLLEGE OF LIBERAL ARTS

NOTE: DO NOT SIGN THIS DOCUMENT UNLESS AN IRB APPROVAL STAMP WITH CURRENT DATES HAS BEEN APPLIED.

INFORMED CONSENT

for a Research Study, entitled

"Personality and Cardiovascular Stress Reactivity."

You are invited to participate in a research study to determine how the cardiovascular system of the body responds to stress. Specifically, we are interested in understanding how this stress response differs when different stress tasks are performed. We are also interested in understanding how personality differences may influence this stress response. The study is being conducted by Mackenzie J. Leavitt, Doctoral Candidate, under the direction of Jennifer L. Robinson, Professor of Psychological Sciences in the Auburn University Department of Psychological Sciences. You were selected as a possible participant based on your responses to the questionnaires you completed during Phase 1 and are between ages 18 to 50.

What will be involved if you participate? If you decide to participate in Phase 2 of this research study, you will be asked to undergo respiration recording while completing short tasks that involve some mental and physical stress.

We will first record height and weight using a scale and a tape measure. You will not be informed of your weight unless you ask. After collecting height and weight, we will ask some brief questions to ensure that you are able to participate in the study today. Specifically, you will be asked if you followed pre-study instructions to avoid alcohol and intense physical training 24 hours prior to the study time. You will also be asked if you have eaten or ingested caffeine in the last two hours. We will then ask you to report the time that you went to bed the night before and the time that you woke up this morning.

At the beginning of the study, you will be outfitted with a respiration belt, one that goes around either the abdomen or the chest. Prior to placing the respiration belt, you will be asked whether you feel that you breathe primarily through your chest or through your abdomen. Your response will determine the placement of the belt (e.g., if you say abdomen, that is where the respiration belt will be placed). This belt allows us to record your breathing rate and to assess measures of cardiac function (e.g., heart rhythms).

Participant's Initials _____

You will also have three electrodes place on your skin in the following locations: the upper right clavicle, the left abdomen between the ribs, and the left forearm. We will have you briefly clean these areas of skin with non-alcoholic baby wipes to increase the conductivity of the electrodes. For those of you with body hair, we will have an electric razor available to give you an opportunity to shave the portions of the skin where we will apply the electrodes. After applying the respiration belt and the electrodes, we will check in with you to ensure that you are comfortable with the equipment attached to you.

You will be asked to sit in a soft chair with your feet flat on the floor and your back as straight as you can comfortably hold it. We will ask you to rest your hands on your thighs, with your palms facing upward. You will be asked to close your eyes, breathe naturally, and allow yourself to relax. You will also be instructed not to do or think about anything in particular. We will then begin recording heart rate and respiration rate. This period will last approximately a total of 10 minutes.

After 10 minutes elapses, you will complete the first stress task. You will either complete a mental math task or a cold pressor task. For the mental math task, the chair will be seated facing a computer. We will move the chair forward slightly to give you room to reach the keyboard. You will see an instruction screen that will describe the mental math task on the computer. In brief, the task will involve subtraction problems where you must subtract a random sequence of two-digit numbers from a random sequence of four-digit numbers (e.g., 1079 – 13). You will be presented these problems one at a time. If you get a problem right, you will see the word correct on the screen. If you get a problem wrong, you will see the word incorrect on the screen. If you do not respond to a problem, you will also see the word incorrect on the screen. This task will continue for 5 minutes.

After completing the first stress task, you will again be instructed to close your eyes, rest your hands on your thighs palms up, breathe naturally, relax, and not do or think anything in particular. This period will last a total of 10 minutes.

After 10 minutes elapses, you will complete the second stress task. For some of you, this will be the mental math task and for others, this will be the cold pressor task. A cooler with a circulating bath of water chilled to 5 degrees Celsius will be positioned next to your chair, so you will be able to insert your wrist into the opening of the cooler. The cooler will be positioned next to your dominant hand, so you will merely have to stretch your hand forward to place it inside the cooler. You will be instructed to submerge your dominant hand into the water just above the wrist. You will be instructed to keep your hand in the water as long as you can possibly tolerate it. If your hand is still remaining in the cooler once 5 minutes have elapsed, we will ask you to remove your hand from the cooler. You will then be offered a hand towel to dry your skin off.

After completing the second stress task, you will again be instructed to close your eyes, rest your hands on your thighs palms up, breathe naturally, relax, and not do or think anything in particular. This period will last a total of 10 minutes.

Participant's Initials _____

Once the third relaxation period is complete, you will be thanked for your participation in the study, and you will be disconnected from the electrodes and the respiration belt will be removed. After this, you will be paid $15 in cash as compensation for participating in this study. You will also be granted one hour of SONA credit as well.

Your total time commitment will be approximately 50 minutes.

Are there any risks or discomforts? The risks or discomforts associated with participation in this research study are:

11. Breach of confidentiality: Participation may involve breach of confidentiality since identifiable confidential data will be retained during the study on a master code list. This breach of confidentiality could only be achieved if someone were to gain access to the master data sheet. The identifiable confidential data collected will the participant's contact information.
12. Emotional Discomfort: Answering survey and questionnaire items about mental health symptoms may cause psychological distress in some participants.
13. Physical discomfort: Engaging in the cold pressor task induces physical pain and discomfort among participants.
14. Mental stress: Engaging in the mental arithmetic task induces psychological stress in many participants.
15. Exposure to COVID-19: in-person laboratory contact with study personnel may carry a risk of conferring COVID-19 on the participant.

To minimize these risks, we will:

11. We will only use password protected computers, and secure servers for data storage. Only one master-list containing identifiable information will be stored. Only personnel mentioned on this protocol will have access to the password- protected master list.
12. You will be allowed to discontinue the study at any time or refuse to answer any questions. We will tell participants that should they experience psychological distress, they may withdraw from the protocol at any time.
13. You will be allowed to discontinue the study at any time or refuse to participate in stress- based tasks. Should you experience any physical discomfort that you cannot tolerate, you may withdraw from the protocol at any time.
14. You may discontinue the study at any time or refuse to participate in stress- based tasks. Should you experience any psychological stress that you cannot tolerate, you may withdraw from the protocol at any time.
15. Exposure to Covid-19: you will be asked if you have had any symptoms of COVID-19 in the last two weeks prior to beginning data collection. You will be excused from participation if you answer yes. In addition, you may also wear a mask at any point during the study protocol if you feel comfortable doing so.

Participant's Initials _____

Risks and Precautions for COVID-19

Due to the need for your physical presence at the research site, face to face interaction with the researcher or others, there is a risk that you may be exposed to COVID-19 and the possibility that you may contract the virus. For most individuals, COVID-19 only causes mild or moderate symptoms. For some, especially older adults, and those with existing health problems, it can cause more severe illness. Current information suggests that roughly 2% of individuals who are infected with COVID-19 may die as a result. You will need to review the information on COVID- 19 for Research Participants that is attached to this consent document. To minimize your risk of exposure, we will screen you for symptoms of COVID-19 prior to admitting you into the laboratory space. Anyone with symptoms of COVID-18 will be excused from participating in the experiment and escorted from the lab space. Upon arrival at Thach Hall 108B, we will take your temperature with a touchless forehead thermometer. If your temperature is 99.0 degrees or higher, we will inform you that you cannot complete the experiment today. At that time, we may reschedule the experiment for another date no less than 14 days away from the initial date. We will adhere to these procedures for all participants.

Are there any benefits to yourself or others? If you participate in this study, you can expect to receive no direct personal benefits. However, we hope that the results of this research study will provide better understanding of factors that influence stress reactivity. We hope that a clearer understanding of these factors may lead to more appropriate inventions for reducing excessive stress. We/I cannot promise you that you will receive any or all the benefits described.

Will you receive compensation for participating? To thank you for your time, you will be offered $15 for completing both stress tasks in the lab today. If you are a student at Auburn University, and if you volunteered through SONA systems, you will be compensation for participating with one credit hour. Your instructors should assign specific values of course credit to these hours. Please check with your instructors for more information.

Are there any costs? If you decide to participate in this research study, you will not incur any costs. If you require medical attention, you will be responsible for all costs for medical attention/treatment.

If you change your mind about participating, you can withdraw at any time during the study. Your participation is completely voluntary. If you choose to withdraw, your data can be withdrawn so long as it is identifiable. Your decision about whether to participate or to stop participating will not jeopardize your future relations with Auburn University or the Department of Psychological Sciences.

Your privacy will be protected. Any information obtained in connection with this research study will remain confidential. At the end of the research study, all links to identifiable information will be destroyed. Data obtained through your participation may be published in a professional journal or presented at a professional meeting.

Participant's Initials ___

If you have questions about this study, please ask them now. Alternatively, you can contact Mackenzie J. Leavitt at mjl0062@auburn.edu or Jennifer L. Robinson at jlr0029@auburn.edu. A copy of this document will be given to you to keep.

If you have questions about your rights as a research participant, you may contact the Auburn University Office of Research Compliance or the Institutional Review Board by phone (334)-844-5966 or e-mail at IRBadmin@auburn.edu or IRBChair@auburn.edu.

HAVING READ THE INFORMATION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATES YOUR WILLINGNESS TO PARTICIPATE.

Participant's signature	Date	Investigator obtaining consent	Date
Printed Name		Printed Name	

The Auburn University Institutional Review Board has approved this Document for use from 06/13/2024 to ----------
Protocol # 23-671 EP 2403

Appendix G: R Code for Statistical Analysis

```
# Script to Analyze my Dissertation Data
### PRELIMINARY SETUP OF ANALYSIS ###
# Clear the workspace
rm(list = ls())

# Install packages only if they are not already installed
packages <- c("readxl", "dplyr", "psych", "Hmisc", "corrplot",
              "lmtest", "tidyr", "lme4", "lmerTest",
              "afex", "performance", "ggplot2", "car", "knitr", "rmark
down")
installed_packages <- packages %in% rownames(installed.packages())

# Only install missing packages
if (any(installed_packages == FALSE)) {
  install.packages(packages[!installed_packages])
}

# Load all required packages
lapply(packages, library, character.only = TRUE)

# load data
DissData <- readxl::read_excel(file.choose())
View(DissData)

# Log Transform HRV Metrics
## NATURAL LOG (base e) – one line per variable
## R1-R3: HFABS, LFABS,VLFABS, LFHF RMSSD
DissData$R1HFLOG    <- log(DissData$R1HFABS)
DissData$R2HFLOG    <- log(DissData$R2HFABS)
DissData$R3HFLOG    <- log(DissData$R3HFABS)

DissData$R1LFLOG    <- log(DissData$R1LFABS)
DissData$R2LFLOG    <- log(DissData$R2LFABS)
DissData$R3LFLOG    <- log(DissData$R3LFABS)

DissData$R1RMSSDLOG <- log(DissData$R1RMSSD)
DissData$R2RMSSDLOG <- log(DissData$R2RMSSD)
DissData$R3RMSSDLOG <- log(DissData$R3RMSSD)

DissData$R1VLFLOG <- log(DissData$R1VLFABS)
DissData$R2VLFLOG <- log(DissData$R2VLFABS)
DissData$R3VLFLOG <- log(DissData$R3VLFABS)

DissData$R1LFHFLOG <- log(DissData$R1LFHF)
DissData$R2LFHFLOG <- log(DissData$R2LFHF)
```

```r
DissData$R3LFHFLOG <- log(DissData$R3LFHF)

## CPT / MST: HFABS, LFABS,VLFABS, LFHF RMSSD
DissData$CPTHFLOG   <- log(DissData$CPTHFABS)
DissData$MSTHFLOG   <- log(DissData$MSTHFABS)

DissData$CPTLFLOG   <- log(DissData$CPTLFABS)
DissData$MSTLFLOG   <- log(DissData$MSTLFABS)

DissData$CPTRMSSDLOG <- log(DissData$CPTRMSSD)
DissData$MSTRMSSDLOG <- log(DissData$MSTRMSSD)

DissData$CPTVLFLOG <- log(DissData$CPTVLFABS)
DissData$MSTVLFLOG <- log(DissData$CPTVLFABS)

DissData$CPTLFHFLOG <- log(DissData$CPTLFHF)
DissData$MSTLFHFLOG <- log(DissData$MSTLFHF)

### PRELIMINARY DESCRIPTIVE STATISTICS ###
# Demographic Analyses
# Age
age_summary <- DissData %>%
  summarise(
    mean_age = mean(AGE, na.rm = TRUE),
    sd_age = sd(AGE, na.rm = TRUE),
    min_age = min(AGE, na.rm = TRUE),
    max_age = max(AGE, na.rm = TRUE),
    median_age = median(AGE, na.rm = TRUE)
  )
print(age_summary)

# Gender
gender_summary <- DissData %>%
  count(GENDER) %>%
  mutate(percent = n / sum(n) * 100)
print(gender_summary)

# Race
race_summary <- DissData %>%
  count(RACE) %>%
  mutate(percent = n / sum(n) * 100)
print(race_summary)

# Ethnicity
ethnicity_summary <- DissData %>%
  count(ETHNICITY) %>%
```

```r
  mutate(percent = n / sum(n) * 100)
print(ethnicity_summary)

# Socioeconomic Status (if coded as categorical)
ses_summary <- DissData %>%
  count(SESAD) %>%
  mutate(percent = n / sum(n) * 100)
print(ses_summary)

# Dominant Hand
hand_summary <- DissData %>%
  count(HANDEDNESS) %>%
  mutate(percent = n / sum(n) * 100)
print(hand_summary)

# ACT Score Recall
ACT_summary <- DissData %>%
  summarise(
    n_total    = n(),
    n_reported = sum(!is.na(ACT)),
    n_missing  = sum(is.na(ACT)),
    percent_reported = (n_reported / n_total) * 100,
    percent_missing  = (n_missing / n_total) * 100
  )
print(ACT_summary)

# ACT Descriptive Stats
ACT_descriptives <- DissData %>%
  summarise(
    mean   = mean(ACT, na.rm = TRUE),
    sd     = sd(ACT, na.rm = TRUE),
    median = median(ACT, na.rm = TRUE),
    min    = min(ACT, na.rm = TRUE),
    max    = max(ACT, na.rm = TRUE)
  )
print(ACT_descriptives)

# Stereotype Vulnerability Total
Stereotype_descriptives <- DissData %>%
  summarise(
    mean   = mean(SVTOTAL, na.rm = TRUE),
    sd     = sd(SVTOTAL, na.rm = TRUE),
    median = median(SVTOTAL, na.rm = TRUE),
    min    = min(SVTOTAL, na.rm = TRUE),
    max    = max(SVTOTAL, na.rm = TRUE)
  )
```

```r
print(Stereotype_descriptives)

# Sleep (hours)
Sleep_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(SLEEP)),
    mean   = mean(SLEEP, na.rm = TRUE),
    sd     = sd(SLEEP, na.rm = TRUE),
    median = median(SLEEP, na.rm = TRUE),
    min    = min(SLEEP, na.rm = TRUE),
    max    = max(SLEEP, na.rm = TRUE)
  )
print(Sleep_summary)

# Contraceptive Use
contraceptive_summary <- DissData %>%
  count(CONMED) %>%
  mutate(percent = n / sum(n) * 100)
print(contraceptive_summary)

# Height
height_summary <- DissData %>%
  summarise(
    mean_height = mean(HEIGHT, na.rm = TRUE),
    sd_height = sd(HEIGHT, na.rm = TRUE),
    min_height = min(HEIGHT, na.rm = TRUE),
    max_height = max(HEIGHT, na.rm = TRUE),
    median_height = median(HEIGHT, na.rm = TRUE),
  )
print(height_summary)

# Convert WEIGHT from pounds to kilograms and store as a new variable
DissData$WEIGHT_KG <- DissData$WEIGHT * 0.45359237

# Quick descriptive check
summary(DissData$WEIGHT_KG)
sd(DissData$WEIGHT_KG, na.rm = TRUE)

# Personality and Psychological Descriptive Statistics
# Psych Diagnoses
psych_summary <- DissData %>%
  count(PSYCH) %>%
  mutate(percent = n / sum(n) * 100)
print(psych_summary)

# Count of Psych Diagnoses
```

```r
MENNUM_summary <- DissData %>%
  count(MENNUM) %>%
  mutate(percent = n / sum(n) * 100)
print(MENNUM_summary)

# Depression and Anxiety Symptoms Descriptive Statistics
symptom_summary <- DissData %>%
  summarise(
    mean_BDITOTAL = mean(BDITOTAL, na.rm = TRUE),
    sd_BDITOTAL = sd(BDITOTAL, na.rm = TRUE),
    min_BDITOTAL = min(BDITOTAL, na.rm = TRUE),
    max_BDITOTAL = max(BDITOTAL, na.rm = TRUE),
    median_BDITOTAL = median(BDITOTAL, na.rm = TRUE),
    mean_BAITOTAL = mean(BAITOTAL, na.rm = TRUE),
    sd_BAITOTAL = sd(BAITOTAL, na.rm = TRUE),
    min_BAITOTAL = min(BAITOTAL, na.rm = TRUE),
    max_BAITOTAL = max(BAITOTAL, na.rm = TRUE),
    median_BAITOTAL = median(BAITOTAL, na.rm = TRUE)
  )
print(symptom_summary)

personality_summary <- DissData %>%
  summarise(
    across(
      .cols = c(N1ANX, N2ANG, N3D, N4SC, N5IM, N6V, NTOTAL, BISPUN, BA
SREW, BASDRV, BASFUN),
      .fns  = list(
        mean   = ~mean(.x, na.rm = TRUE),
        median = ~median(.x, na.rm = TRUE),
        sd     = ~sd(.x, na.rm = TRUE),
        min    = ~min(.x, na.rm = TRUE),
        max    = ~max(.x, na.rm = TRUE)
      ),
      .names = "{.col}_{.fn}"
    )
  )
print(personality_summary)

psych_measures_summary <- describe(DissData %>% select(BDITOTAL, BAITO
TAL))
print(psych_measures_summary)

# Physiological Descriptive Statistics
# Time-Domain Descriptive Stats
# RMSSD
RMSSD_R1_summary <- DissData %>%
```

```r
  summarise(
    raw_mean    = mean(R1RMSSD, na.rm = TRUE),
    raw_sd      = sd(R1RMSSD, na.rm = TRUE),
    raw_median  = median(R1RMSSD, na.rm = TRUE),
    raw_min     = min(R1RMSSD, na.rm = TRUE),
    raw_max     = max(R1RMSSD, na.rm = TRUE),
    log_mean    = mean(R1RMSSDLOG, na.rm = TRUE),
    log_sd      = sd(R1RMSSDLOG, na.rm = TRUE),
    log_median  = median(R1RMSSDLOG, na.rm = TRUE),
    log_min     = min(R1RMSSDLOG, na.rm = TRUE),
    log_max     = max(R1RMSSDLOG, na.rm = TRUE)
  )
print(RMSSD_R1_summary)

# Mean HR
HR_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1MEANHR)),
    mean   = mean(R1MEANHR, na.rm = TRUE),
    sd     = sd(R1MEANHR, na.rm = TRUE),
    median = median(R1MEANHR, na.rm = TRUE),
    min    = min(R1MEANHR, na.rm = TRUE),
    max    = max(R1MEANHR, na.rm = TRUE)
  )
print(HR_R1_summary)

# SDNN
SDNN_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1SDNN)),
    mean   = mean(R1SDNN, na.rm = TRUE),
    sd     = sd(R1SDNN, na.rm = TRUE),
    median = median(R1SDNN, na.rm = TRUE),
    min    = min(R1SDNN, na.rm = TRUE),
    max    = max(R1SDNN, na.rm = TRUE)
  )
print(SDNN_R1_summary)

# NN50
NN50_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1NN50)),
    mean   = mean(R1NN50, na.rm = TRUE),
    sd     = sd(R1NN50, na.rm = TRUE),
    median = median(R1NN50, na.rm = TRUE),
    min    = min(R1NN50, na.rm = TRUE),
```

```r
    max    = max(R1NN50, na.rm = TRUE)
  )
print(NN50_R1_summary)

# pNN50
PNN50_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1pNN50)),
    mean   = mean(R1pNN50, na.rm = TRUE),
    sd     = sd(R1pNN50, na.rm = TRUE),
    median = median(R1pNN50, na.rm = TRUE),
    min    = min(R1pNN50, na.rm = TRUE),
    max    = max(R1pNN50, na.rm = TRUE)
  )
print(PNN50_R1_summary)

# Spectral Descriptive Stats
# HF
HF_R1_summary <- DissData %>%
  summarise(
    # Absolute power (raw)
    raw_mean    = mean(R1HFABS, na.rm = TRUE),
    raw_sd      = sd(R1HFABS, na.rm = TRUE),
    raw_median  = median(R1HFABS, na.rm = TRUE),
    raw_min     = min(R1HFABS, na.rm = TRUE),
    raw_max     = max(R1HFABS, na.rm = TRUE),

    # Log-transformed absolute power
    log_mean    = mean(R1HFLOG, na.rm = TRUE),
    log_sd      = sd(R1HFLOG, na.rm = TRUE),
    log_median  = median(R1HFLOG, na.rm = TRUE),
    log_min     = min(R1HFLOG, na.rm = TRUE),
    log_max     = max(R1HFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R1HFPERC, na.rm = TRUE),
    perc_sd     = sd(R1HFPERC, na.rm = TRUE),
    perc_median = median(R1HFPERC, na.rm = TRUE),
    perc_min    = min(R1HFPERC, na.rm = TRUE),
    perc_max    = max(R1HFPERC, na.rm = TRUE)
  )
print(HF_R1_summary)

# LF
LF_R1_summary <- DissData %>%
  summarise(
```

```
    # Absolute power
    raw_mean    = mean(R1LFABS, na.rm = TRUE),
    raw_sd      = sd(R1LFABS, na.rm = TRUE),
    raw_median  = median(R1LFABS, na.rm = TRUE),
    raw_min     = min(R1LFABS, na.rm = TRUE),
    raw_max     = max(R1LFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R1LFLOG, na.rm = TRUE),
    log_sd      = sd(R1LFLOG, na.rm = TRUE),
    log_median  = median(R1LFLOG, na.rm = TRUE),
    log_min     = min(R1LFLOG, na.rm = TRUE),
    log_max     = max(R1LFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R1LFPERC, na.rm = TRUE),
    perc_sd     = sd(R1LFPERC, na.rm = TRUE),
    perc_median = median(R1LFPERC, na.rm = TRUE),
    perc_min    = min(R1LFPERC, na.rm = TRUE),
    perc_max    = max(R1LFPERC, na.rm = TRUE)
  )
print(LF_R1_summary)

# LF/HF ratio
LFHF_R1_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(R1LFHF, na.rm = TRUE),
    raw_sd      = sd(R1LFHF, na.rm = TRUE),
    raw_median  = median(R1LFHF, na.rm = TRUE),
    raw_min     = min(R1LFHF, na.rm = TRUE),
    raw_max     = max(R1LFHF, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R1LFHFLOG, na.rm = TRUE),
    log_sd      = sd(R1LFHFLOG, na.rm = TRUE),
    log_median  = median(R1LFHFLOG, na.rm = TRUE),
    log_min     = min(R1LFHFLOG, na.rm = TRUE),
    log_max     = max(R1LFHFLOG, na.rm = TRUE),
  )
print(LFHF_R1_summary)

# First Relaxation Phase Physiological Descriptive Statistics
# Time-Domain Descriptive Stats
# RMSSD
RMSSD_R2_summary <- DissData %>%
```

```r
  summarise(
    raw_mean    = mean(R2RMSSD, na.rm = TRUE),
    raw_sd      = sd(R2RMSSD, na.rm = TRUE),
    raw_median  = median(R2RMSSD, na.rm = TRUE),
    raw_min     = min(R2RMSSD, na.rm = TRUE),
    raw_max     = max(R2RMSSD, na.rm = TRUE),
    log_mean    = mean(R2RMSSDLOG, na.rm = TRUE),
    log_sd      = sd(R2RMSSDLOG, na.rm = TRUE),
    log_median  = median(R2RMSSDLOG, na.rm = TRUE),
    log_min     = min(R2RMSSDLOG, na.rm = TRUE),
    log_max     = max(R2RMSSDLOG, na.rm = TRUE)
  )
print(RMSSD_R2_summary)

# Mean HR
HR_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2MEANHR)),
    mean   = mean(R2MEANHR, na.rm = TRUE),
    sd     = sd(R2MEANHR, na.rm = TRUE),
    median = median(R2MEANHR, na.rm = TRUE),
    min    = min(R2MEANHR, na.rm = TRUE),
    max    = max(R2MEANHR, na.rm = TRUE)
  )
print(HR_R2_summary)

# SDNN
SDNN_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2SDNN)),
    mean   = mean(R2SDNN, na.rm = TRUE),
    sd     = sd(R2SDNN, na.rm = TRUE),
    median = median(R2SDNN, na.rm = TRUE),
    min    = min(R2SDNN, na.rm = TRUE),
    max    = max(R2SDNN, na.rm = TRUE)
  )
print(SDNN_R2_summary)

# NN50
NN50_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2NN50)),
    mean   = mean(R2NN50, na.rm = TRUE),
    sd     = sd(R2NN50, na.rm = TRUE),
    median = median(R2NN50, na.rm = TRUE),
    min    = min(R2NN50, na.rm = TRUE),
```

```
    max    = max(R2NN50, na.rm = TRUE)
  )
print(NN50_R2_summary)

# pNN50
PNN50_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2pNN50)),
    mean   = mean(R2pNN50, na.rm = TRUE),
    sd     = sd(R2pNN50, na.rm = TRUE),
    median = median(R2pNN50, na.rm = TRUE),
    min    = min(R2pNN50, na.rm = TRUE),
    max    = max(R2pNN50, na.rm = TRUE)
  )
print(PNN50_R2_summary)

# Spectral Descriptive Stats
# HF
HF_R2_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(R2HFABS, na.rm = TRUE),
    raw_sd      = sd(R2HFABS, na.rm = TRUE),
    raw_median  = median(R2HFABS, na.rm = TRUE),
    raw_min     = min(R2HFABS, na.rm = TRUE),
    raw_max     = max(R2HFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R2HFLOG, na.rm = TRUE),
    log_sd      = sd(R2HFLOG, na.rm = TRUE),
    log_median  = median(R2HFLOG, na.rm = TRUE),
    log_min     = min(R2HFLOG, na.rm = TRUE),
    log_max     = max(R2HFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R2HFPERC, na.rm = TRUE),
    perc_sd     = sd(R2HFPERC, na.rm = TRUE),
    perc_median = median(R2HFPERC, na.rm = TRUE),
    perc_min    = min(R2HFPERC, na.rm = TRUE),
    perc_max    = max(R2HFPERC, na.rm = TRUE)
  )
print(HF_R2_summary)

# LF
LF_R2_summary <- DissData %>%
  summarise(
```

```
    # Absolute power
    raw_mean    = mean(R2LFABS, na.rm = TRUE),
    raw_sd      = sd(R2LFABS, na.rm = TRUE),
    raw_median  = median(R2LFABS, na.rm = TRUE),
    raw_min     = min(R2LFABS, na.rm = TRUE),
    raw_max     = max(R2LFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R2LFLOG, na.rm = TRUE),
    log_sd      = sd(R2LFLOG, na.rm = TRUE),
    log_median  = median(R2LFLOG, na.rm = TRUE),
    log_min     = min(R2LFLOG, na.rm = TRUE),
    log_max     = max(R2LFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R2LFPERC, na.rm = TRUE),
    perc_sd     = sd(R2LFPERC, na.rm = TRUE),
    perc_median = median(R2LFPERC, na.rm = TRUE),
    perc_min    = min(R2LFPERC, na.rm = TRUE),
    perc_max    = max(R2LFPERC, na.rm = TRUE)
  )
print(LF_R2_summary)

# LF/HF ratio
LFHF_R2_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(R2LFHF, na.rm = TRUE),
    raw_sd      = sd(R2LFHF, na.rm = TRUE),
    raw_median  = median(R2LFHF, na.rm = TRUE),
    raw_min     = min(R2LFHF, na.rm = TRUE),
    raw_max     = max(R2LFHF, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R2LFHFLOG, na.rm = TRUE),
    log_sd      = sd(R2LFHFLOG, na.rm = TRUE),
    log_median  = median(R2LFHFLOG, na.rm = TRUE),
    log_min     = min(R2LFHFLOG, na.rm = TRUE),
    log_max     = max(R2LFHFLOG, na.rm = TRUE)
  )
print(LFHF_R2_summary)

# Final Relaxation Phase Physiological Descriptive Statistics
# Time-Domain Descriptive Stats
# RMSSD
RMSSD_R3_summary <- DissData %>%
```

```r
  summarise(
    raw_mean    = mean(R3RMSSD, na.rm = TRUE),
    raw_sd      = sd(R3RMSSD, na.rm = TRUE),
    raw_median  = median(R3RMSSD, na.rm = TRUE),
    raw_min     = min(R3RMSSD, na.rm = TRUE),
    raw_max     = max(R3RMSSD, na.rm = TRUE),
    log_mean    = mean(R3RMSSDLOG, na.rm = TRUE),
    log_sd      = sd(R3RMSSDLOG, na.rm = TRUE),
    log_median  = median(R3RMSSDLOG, na.rm = TRUE),
    log_min     = min(R3RMSSDLOG, na.rm = TRUE),
    log_max     = max(R3RMSSDLOG, na.rm = TRUE)
  )
print(RMSSD_R3_summary)

# Mean HR
HR_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R3MEANHR)),
    mean   = mean(R3MEANHR, na.rm = TRUE),
    sd     = sd(R3MEANHR, na.rm = TRUE),
    median = median(R3MEANHR, na.rm = TRUE),
    min    = min(R3MEANHR, na.rm = TRUE),
    max    = max(R3MEANHR, na.rm = TRUE)
  )
print(HR_R3_summary)

# SDNN
SDNN_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2SDNN)),
    mean   = mean(R3SDNN, na.rm = TRUE),
    sd     = sd(R3SDNN, na.rm = TRUE),
    median = median(R3SDNN, na.rm = TRUE),
    min    = min(R3SDNN, na.rm = TRUE),
    max    = max(R3SDNN, na.rm = TRUE)
  )
print(SDNN_R3_summary)

# NN50
NN50_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R3NN50)),
    mean   = mean(R3NN50, na.rm = TRUE),
    sd     = sd(R3NN50, na.rm = TRUE),
    median = median(R3NN50, na.rm = TRUE),
    min    = min(R3NN50, na.rm = TRUE),
```

```r
    max    = max(R3NN50, na.rm = TRUE)
  )
print(NN50_R3_summary)

# pNN50
PNN50_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R3pNN50)),
    mean   = mean(R3pNN50, na.rm = TRUE),
    sd     = sd(R3pNN50, na.rm = TRUE),
    median = median(R3pNN50, na.rm = TRUE),
    min    = min(R3pNN50, na.rm = TRUE),
    max    = max(R3pNN50, na.rm = TRUE)
  )
print(PNN50_R3_summary)

# Spectral Descriptive Stats
# HF
HF_R3_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(R3HFABS, na.rm = TRUE),
    raw_sd      = sd(R3HFABS, na.rm = TRUE),
    raw_median  = median(R3HFABS, na.rm = TRUE),
    raw_min     = min(R3HFABS, na.rm = TRUE),
    raw_max     = max(R3HFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R3HFLOG, na.rm = TRUE),
    log_sd      = sd(R3HFLOG, na.rm = TRUE),
    log_median  = median(R3HFLOG, na.rm = TRUE),
    log_min     = min(R3HFLOG, na.rm = TRUE),
    log_max     = max(R3HFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R3HFPERC, na.rm = TRUE),
    perc_sd     = sd(R3HFPERC, na.rm = TRUE),
    perc_median = median(R3HFPERC, na.rm = TRUE),
    perc_min    = min(R3HFPERC, na.rm = TRUE),
    perc_max    = max(R3HFPERC, na.rm = TRUE)
  )
print(HF_R3_summary)

# LF
LF_R3_summary <- DissData %>%
  summarise(
```

```
    # Absolute power
    raw_mean    = mean(R3LFABS, na.rm = TRUE),
    raw_sd      = sd(R3LFABS, na.rm = TRUE),
    raw_median  = median(R3LFABS, na.rm = TRUE),
    raw_min     = min(R3LFABS, na.rm = TRUE),
    raw_max     = max(R3LFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R3LFLOG, na.rm = TRUE),
    log_sd      = sd(R3LFLOG, na.rm = TRUE),
    log_median  = median(R3LFLOG, na.rm = TRUE),
    log_min     = min(R3LFLOG, na.rm = TRUE),
    log_max     = max(R3LFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(R3LFPERC, na.rm = TRUE),
    perc_sd     = sd(R3LFPERC, na.rm = TRUE),
    perc_median = median(R3LFPERC, na.rm = TRUE),
    perc_min    = min(R3LFPERC, na.rm = TRUE),
    perc_max    = max(R3LFPERC, na.rm = TRUE)
  )
print(LF_R3_summary)

# LF/HF ratio (no normalized units here)
LFHF_R3_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(R3LFHF, na.rm = TRUE),
    raw_sd      = sd(R3LFHF, na.rm = TRUE),
    raw_median  = median(R3LFHF, na.rm = TRUE),
    raw_min     = min(R3LFHF, na.rm = TRUE),
    raw_max     = max(R3LFHF, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(R3LFHFLOG, na.rm = TRUE),
    log_sd      = sd(R3LFHFLOG, na.rm = TRUE),
    log_median  = median(R3LFHFLOG, na.rm = TRUE),
    log_min     = min(R3LFHFLOG, na.rm = TRUE),
    log_max     = max(R3LFHFLOG, na.rm = TRUE)
  )
print(LFHF_R3_summary)

# MST Descriptive Statistics
# Time-Domain Descriptive Stats
# RMSSD
RMSSD_MST_summary <- DissData %>%
```

```r
  summarise(
    raw_mean    = mean(MSTRMSSD, na.rm = TRUE),
    raw_sd      = sd(MSTRMSSD, na.rm = TRUE),
    raw_median  = median(MSTRMSSD, na.rm = TRUE),
    raw_min     = min(MSTRMSSD, na.rm = TRUE),
    raw_max     = max(MSTRMSSD, na.rm = TRUE),
    log_mean    = mean(MSTRMSSDLOG, na.rm = TRUE),
    log_sd      = sd(MSTRMSSDLOG, na.rm = TRUE),
    log_median  = median(MSTRMSSDLOG, na.rm = TRUE),
    log_min     = min(MSTRMSSDLOG, na.rm = TRUE),
    log_max     = max(MSTRMSSDLOG, na.rm = TRUE)
  )
print(RMSSD_MST_summary)

# Mean HR
HR_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTMEANHR)),
    mean   = mean(MSTMEANHR, na.rm = TRUE),
    sd     = sd(MSTMEANHR, na.rm = TRUE),
    median = median(MSTMEANHR, na.rm = TRUE),
    min    = min(MSTMEANHR, na.rm = TRUE),
    max    = max(MSTMEANHR, na.rm = TRUE)
  )
print(HR_MST_summary)

# SDNN
SDNN_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTSDNN)),
    mean   = mean(MSTSDNN, na.rm = TRUE),
    sd     = sd(MSTSDNN, na.rm = TRUE),
    median = median(MSTSDNN, na.rm = TRUE),
    min    = min(MSTSDNN, na.rm = TRUE),
    max    = max(MSTSDNN, na.rm = TRUE)
  )
print(SDNN_MST_summary)

# NN50
NN50_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTNN50)),
    mean   = mean(MSTNN50, na.rm = TRUE),
    sd     = sd(MSTNN50, na.rm = TRUE),
    median = median(MSTNN50, na.rm = TRUE),
    min    = min(MSTNN50, na.rm = TRUE),
```

```
    max    = max(MSTNN50, na.rm = TRUE)
  )
print(NN50_MST_summary)

# pNN50
PNN50_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTpNN50)),
    mean   = mean(MSTpNN50, na.rm = TRUE),
    sd     = sd(MSTpNN50, na.rm = TRUE),
    median = median(MSTpNN50, na.rm = TRUE),
    min    = min(MSTpNN50, na.rm = TRUE),
    max    = max(MSTpNN50, na.rm = TRUE)
  )
print(PNN50_MST_summary)

# Spectral Descriptive Stats
# HF
HF_MST_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(MSTHFABS, na.rm = TRUE),
    raw_sd      = sd(MSTHFABS, na.rm = TRUE),
    raw_median  = median(MSTHFABS, na.rm = TRUE),
    raw_min     = min(MSTHFABS, na.rm = TRUE),
    raw_max     = max(MSTHFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(MSTHFLOG, na.rm = TRUE),
    log_sd      = sd(MSTHFLOG, na.rm = TRUE),
    log_median  = median(MSTHFLOG, na.rm = TRUE),
    log_min     = min(MSTHFLOG, na.rm = TRUE),
    log_max     = max(MSTHFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(MSTHFPERC, na.rm = TRUE),
    perc_sd     = sd(MSTHFPERC, na.rm = TRUE),
    perc_median = median(MSTHFPERC, na.rm = TRUE),
    perc_min    = min(MSTHFPERC, na.rm = TRUE),
    perc_max    = max(MSTHFPERC, na.rm = TRUE)
  )
print(HF_MST_summary)

# LF
LF_MST_summary <- DissData %>%
  summarise(
```

```
    # Absolute power
    raw_mean    = mean(MSTLFABS, na.rm = TRUE),
    raw_sd      = sd(MSTLFABS, na.rm = TRUE),
    raw_median  = median(MSTLFABS, na.rm = TRUE),
    raw_min     = min(MSTLFABS, na.rm = TRUE),
    raw_max     = max(MSTLFABS, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(MSTLFLOG, na.rm = TRUE),
    log_sd      = sd(MSTLFLOG, na.rm = TRUE),
    log_median  = median(MSTLFLOG, na.rm = TRUE),
    log_min     = min(MSTLFLOG, na.rm = TRUE),
    log_max     = max(MSTLFLOG, na.rm = TRUE),

    # Percentage / normalized units
    perc_mean   = mean(MSTLFPERC, na.rm = TRUE),
    perc_sd     = sd(MSTLFPERC, na.rm = TRUE),
    perc_median = median(MSTLFPERC, na.rm = TRUE),
    perc_min    = min(MSTLFPERC, na.rm = TRUE),
    perc_max    = max(MSTLFPERC, na.rm = TRUE)
  )
print(LF_MST_summary)

# LF/HF ratio (no normalized units here)
LFHF_MST_summary <- DissData %>%
  summarise(
    # Absolute power
    raw_mean    = mean(MSTLFHF, na.rm = TRUE),
    raw_sd      = sd(MSTLFHF, na.rm = TRUE),
    raw_median  = median(MSTLFHF, na.rm = TRUE),
    raw_min     = min(MSTLFHF, na.rm = TRUE),
    raw_max     = max(MSTLFHF, na.rm = TRUE),

    # Log-transformed
    log_mean    = mean(MSTLFHFLOG, na.rm = TRUE),
    log_sd      = sd(MSTLFHFLOG, na.rm = TRUE),
    log_median  = median(MSTLFHFLOG, na.rm = TRUE),
    log_min     = min(MSTLFHFLOG, na.rm = TRUE),
    log_max     = max(MSTLFHFLOG, na.rm = TRUE)
  )
print(LFHF_MST_summary)

# CPT Descriptive Statistics
# Time-Domain Descriptive Stats
# RMSSD
RMSSD_CPT_summary <- DissData %>%
```

```r
  summarise(
    raw_mean    = mean(CPTRMSSD, na.rm = TRUE),
    raw_sd      = sd(CPTRMSSD, na.rm = TRUE),
    raw_median  = median(CPTRMSSD, na.rm = TRUE),
    raw_min     = min(CPTRMSSD, na.rm = TRUE),
    raw_max     = max(CPTRMSSD, na.rm = TRUE),
    log_mean    = mean(CPTRMSSDLOG, na.rm = TRUE),
    log_sd      = sd(CPTRMSSDLOG, na.rm = TRUE),
    log_median  = median(CPTRMSSDLOG, na.rm = TRUE),
    log_min     = min(CPTRMSSDLOG, na.rm = TRUE),
    log_max     = max(CPTRMSSDLOG, na.rm = TRUE)
  )
print(RMSSD_CPT_summary)

# Mean HR
HR_CPT_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(CPTMEANHR)),
    mean   = mean(CPTMEANHR, na.rm = TRUE),
    sd     = sd(CPTMEANHR, na.rm = TRUE),
    median = median(CPTMEANHR, na.rm = TRUE),
    min    = min(CPTMEANHR, na.rm = TRUE),
    max    = max(CPTMEANHR, na.rm = TRUE)
  )
print(HR_CPT_summary)

# SDNN
SDNN_CPT_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(CPTSDNN)),
    mean   = mean(CPTSDNN, na.rm = TRUE),
    sd     = sd(CPTSDNN, na.rm = TRUE),
    median = median(CPTSDNN, na.rm = TRUE),
    min    = min(CPTSDNN, na.rm = TRUE),
    max    = max(CPTSDNN, na.rm = TRUE)
  )
print(SDNN_CPT_summary)

# NN50
NN50_CPT_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(CPTNN50)),
    mean   = mean(CPTNN50, na.rm = TRUE),
    sd     = sd(CPTNN50, na.rm = TRUE),
    median = median(CPTNN50, na.rm = TRUE),
    min    = min(CPTNN50, na.rm = TRUE),
```

```
    max    = max(CPTNN50, na.rm = TRUE)
  )
print(NN50_CPT_summary)

# pNN50
PNN50_CPT_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(CPTpNN50)),
    mean   = mean(CPTpNN50, na.rm = TRUE),
    sd     = sd(CPTpNN50, na.rm = TRUE),
    median = median(CPTpNN50, na.rm = TRUE),
    min    = min(CPTpNN50, na.rm = TRUE),
    max    = max(CPTpNN50, na.rm = TRUE)
  )
print(PNN50_CPT_summary)

# Spectral Descriptive Stats
# HF – CPT
HF_CPT_summary <- DissData %>%
  summarise(
    raw_mean    = mean(CPTHFABS, na.rm = TRUE),
    raw_sd      = sd(CPTHFABS, na.rm = TRUE),
    raw_median  = median(CPTHFABS, na.rm = TRUE),
    raw_min     = min(CPTHFABS, na.rm = TRUE),
    raw_max     = max(CPTHFABS, na.rm = TRUE),

    log_mean    = mean(CPTHFLOG, na.rm = TRUE),
    log_sd      = sd(CPTHFLOG, na.rm = TRUE),
    log_median  = median(CPTHFLOG, na.rm = TRUE),
    log_min     = min(CPTHFLOG, na.rm = TRUE),
    log_max     = max(CPTHFLOG, na.rm = TRUE),

    perc_mean   = mean(CPTHFPERC, na.rm = TRUE),
    perc_sd     = sd(CPTHFPERC, na.rm = TRUE),
    perc_median = median(CPTHFPERC, na.rm = TRUE),
    perc_min    = min(CPTHFPERC, na.rm = TRUE),
    perc_max    = max(CPTHFPERC, na.rm = TRUE)
  )
print(HF_CPT_summary)

# LF – CPT
LF_CPT_summary <- DissData %>%
  summarise(
    raw_mean    = mean(CPTLFABS, na.rm = TRUE),
    raw_sd      = sd(CPTLFABS, na.rm = TRUE),
    raw_median  = median(CPTLFABS, na.rm = TRUE),
```

```
    raw_min     = min(CPTLFABS, na.rm = TRUE),
    raw_max     = max(CPTLFABS, na.rm = TRUE),

    log_mean    = mean(CPTLFLOG, na.rm = TRUE),
    log_sd      = sd(CPTLFLOG, na.rm = TRUE),
    log_median  = median(CPTLFLOG, na.rm = TRUE),
    log_min     = min(CPTLFLOG, na.rm = TRUE),
    log_max     = max(CPTLFLOG, na.rm = TRUE),

    perc_mean   = mean(CPTLFPERC, na.rm = TRUE),
    perc_sd     = sd(CPTLFPERC, na.rm = TRUE),
    perc_median = median(CPTLFPERC, na.rm = TRUE),
    perc_min    = min(CPTLFPERC, na.rm = TRUE),
    perc_max    = max(CPTLFPERC, na.rm = TRUE)
  )
print(LF_CPT_summary)

# LF/HF ratio — CPT (no percentage variant)
LFHF_CPT_summary <- DissData %>%
  summarise(
    raw_mean    = mean(CPTLFHF, na.rm = TRUE),
    raw_sd      = sd(CPTLFHF, na.rm = TRUE),
    raw_median  = median(CPTLFHF, na.rm = TRUE),
    raw_min     = min(CPTLFHF, na.rm = TRUE),
    raw_max     = max(CPTLFHF, na.rm = TRUE),

    log_mean    = mean(CPTLFHFLOG, na.rm = TRUE),
    log_sd      = sd(CPTLFHFLOG, na.rm = TRUE),
    log_median  = median(CPTLFHFLOG, na.rm = TRUE),
    log_min     = min(CPTLFHFLOG, na.rm = TRUE),
    log_max     = max(CPTLFHFLOG, na.rm = TRUE)
  )
print(LFHF_CPT_summary)

# Remaining time-domain descriptives
# Mean RR descriptives across phases
MeanRR_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1MEANRR)),
    mean   = mean(R1MEANRR, na.rm = TRUE),
    sd     = sd(R1MEANRR, na.rm = TRUE),
    median = median(R1MEANRR, na.rm = TRUE),
    min    = min(R1MEANRR, na.rm = TRUE),
    max    = max(R1MEANRR, na.rm = TRUE)
  )
print(MeanRR_R1_summary)
```

```r
MeanRR_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2MEANRR)),
    mean   = mean(R2MEANRR, na.rm = TRUE),
    sd     = sd(R2MEANRR, na.rm = TRUE),
    median = median(R2MEANRR, na.rm = TRUE),
    min    = min(R2MEANRR, na.rm = TRUE),
    max    = max(R2MEANRR, na.rm = TRUE)
  )
print(MeanRR_R2_summary)

MeanRR_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R3MEANRR)),
    mean   = mean(R3MEANRR, na.rm = TRUE),
    sd     = sd(R3MEANRR, na.rm = TRUE),
    median = median(R3MEANRR, na.rm = TRUE),
    min    = min(R3MEANRR, na.rm = TRUE),
    max    = max(R3MEANRR, na.rm = TRUE)
  )
print(MeanRR_R3_summary)

MeanRR_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTMEANRR)),
    mean   = mean(MSTMEANRR, na.rm = TRUE),
    sd     = sd(MSTMEANRR, na.rm = TRUE),
    median = median(MSTMEANRR, na.rm = TRUE),
    min    = min(MSTMEANRR, na.rm = TRUE),
    max    = max(MSTMEANRR, na.rm = TRUE)
  )
print(MeanRR_MST_summary)

MeanRR_CPT_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(CPTMEANRR)),
    mean   = mean(CPTMEANRR, na.rm = TRUE),
    sd     = sd(CPTMEANRR, na.rm = TRUE),
    median = median(CPTMEANRR, na.rm = TRUE),
    min    = min(CPTMEANRR, na.rm = TRUE),
    max    = max(CPTMEANRR, na.rm = TRUE)
  )
print(MeanRR_CPT_summary)

# Median RR descriptives across phases
```

```r
MedRR_R1_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R1MEDRR)),
    mean   = mean(R1MEDRR, na.rm = TRUE),
    sd     = sd(R1MEDRR, na.rm = TRUE),
    median = median(R1MEDRR, na.rm = TRUE),
    min    = min(R1MEDRR, na.rm = TRUE),
    max    = max(R1MEDRR, na.rm = TRUE)
  )
print(MedRR_R1_summary)

MedRR_R2_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R2MEDRR)),
    mean   = mean(R2MEDRR, na.rm = TRUE),
    sd     = sd(R2MEDRR, na.rm = TRUE),
    median = median(R2MEDRR, na.rm = TRUE),
    min    = min(R2MEDRR, na.rm = TRUE),
    max    = max(R2MEDRR, na.rm = TRUE)
  )
print(MedRR_R2_summary)

MedRR_R3_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(R3MEDRR)),
    mean   = mean(R3MEDRR, na.rm = TRUE),
    sd     = sd(R3MEDRR, na.rm = TRUE),
    median = median(R3MEDRR, na.rm = TRUE),
    min    = min(R3MEDRR, na.rm = TRUE),
    max    = max(R3MEDRR, na.rm = TRUE)
  )
print(MedRR_R3_summary)

MedRR_MST_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MSTMEDRR)),
    mean   = mean(MSTMEDRR, na.rm = TRUE),
    sd     = sd(MSTMEDRR, na.rm = TRUE),
    median = median(MSTMEDRR, na.rm = TRUE),
    min    = min(MSTMEDRR, na.rm = TRUE),
    max    = max(MSTMEDRR, na.rm = TRUE)
  )
print(MedRR_MST_summary)

MedRR_CPT_summary <- DissData %>%
  summarise(
```

```
    n      = sum(!is.na(CPTMEDRR)),
    mean   = mean(CPTMEDRR, na.rm = TRUE),
    sd     = sd(CPTMEDRR, na.rm = TRUE),
    median = median(CPTMEDRR, na.rm = TRUE),
    min    = min(CPTMEDRR, na.rm = TRUE),
    max    = max(CPTMEDRR, na.rm = TRUE)
  )
print(MedRR_CPT_summary)

#Behavioral Descriptive Statistics
# Stress Task Ratings
stress_ratings_summary <- DissData %>%
  summarise(
    mean_MSTRATING = mean(MSTRATING, na.rm = TRUE),
    sd_MSTRATING = sd(MSTRATING, na.rm = TRUE),
    min_MSTRATING = min(MSTRATING, na.rm = TRUE),
    max_MSTRATING = max(MSTRATING, na.rm = TRUE),
    median_MSTRATING = median(MSTRATING, na.rm = TRUE),
    mean_CPTRATING = mean(CPTRATING, na.rm = TRUE),
    sd_CPTRATING = sd(CPTRATING, na.rm = TRUE),
    min_CPTRATING = min(CPTRATING, na.rm = TRUE),
    max_CPTRATING = max(CPTRATING, na.rm = TRUE),
    median_CPTRATING = median(CPTRATING, na.rm = TRUE)
  )
print(stress_ratings_summary)

# Math Accuracy- Correct Answers
Math_Correct_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MathCorr)),
    mean   = mean(MathCorr, na.rm = TRUE),
    sd     = sd(MathCorr, na.rm = TRUE),
    median = median(MathCorr, na.rm = TRUE),
    min    = min(MathCorr, na.rm = TRUE),
    max    = max(MathCorr, na.rm = TRUE)
  )
print(Math_Correct_summary)

# Math Accuracy- Incorrect Answers
Math_Incorrect_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MathIncorr)),
    mean   = mean(MathIncorr, na.rm = TRUE),
    sd     = sd(MathIncorr, na.rm = TRUE),
    median = median(MathIncorr, na.rm = TRUE),
    min    = min(MathIncorr, na.rm = TRUE),
```

```r
    max    = max(MathIncorr, na.rm = TRUE)
  )
print(Math_Incorrect_summary)

#Math Accuracy- Percent Correct
Math_Percent_summary <- DissData %>%
  summarise(
    n      = sum(!is.na(MathAcc)),
    mean   = mean(MathAcc, na.rm = TRUE),
    sd     = sd(MathAcc, na.rm = TRUE),
    median = median(MathAcc, na.rm = TRUE),
    min    = min(MathAcc, na.rm = TRUE),
    max    = max(MathAcc, na.rm = TRUE)
  )
print(Math_Percent_summary)

# Cold Exposure Duration (CPTTIME)
cpttime_summary <- DissData %>%
  summarise(
    mean_CPTTIME = mean(CPTTIME, na.rm = TRUE),
    sd_CPTTIME = sd(CPTTIME, na.rm = TRUE),
    min_CPTTIME = min(CPTTIME, na.rm = TRUE),
    max_CPTTIME = max(CPTTIME, na.rm = TRUE),
    median_CPTTIME = median(CPTTIME, na.rm = TRUE)
  )
print(cpttime_summary)

# Correlation Plot for Demographic and Behavioral Measures
# 1) Select DEM variables & coerce numeric
DEM_selected_vars <- DissData %>%
  dplyr::select(
    AGE, GENDER, HEIGHT, WEIGHT_KG,
    SVTOTAL, MSTRATING, CPTRATING, CPTTIME,
    MathCorr, MathIncorr, SLEEP
  ) %>%
  mutate(across(everything(), ~ suppressWarnings(as.numeric(.))))

# 2) Spearman correlations (Hmisc handles ties)
res <- rcorr(as.matrix(DEM_selected_vars), type = "spearman")
R <- res$r            # Spearman rho
P <- res$P            # raw p-values
N <- res$n            # pairwise Ns

# --- IMPORTANT: do NOT convert NA p's to 1 before adjustment ---
# 3) Adjust p-values (Benjamini–Hochberg / FDR) only on valid upper-tr
i tests
```

```r
P_work <- P
diag(P_work) <- NA

upper <- upper.tri(P_work, diag = FALSE)
p_vec <- P_work[upper]              # may include NAs if some pairs cou
ldn't be computed
keep <- !is.na(p_vec)

padj_vec <- rep(NA_real_, length(p_vec))
padj_vec[keep] <- p.adjust(p_vec[keep], method = "BH")

# Rebuild a symmetric BH-adjusted p-value matrix
P_adj <- matrix(NA_real_, nrow = nrow(P_work), ncol = ncol(P_work),
                dimnames = dimnames(P_work))
P_adj[upper] <- padj_vec
P_adj[lower.tri(P_adj)] <- t(P_adj)[lower.tri(P_adj)]

# For plotting with corrplot, set diag to 1 so diagonal never shows as
significant
P_adj_plot <- P_adj
diag(P_adj_plot) <- 1

# 4) Friendly labels
label_map <- c(
  AGE        = "Age",
  GENDER     = "Gender",
  HEIGHT     = "Height",
  WEIGHT_KG  = "Weight (kg)",
  SVTOTAL    = "SVS Total",
  MSTRATING  = "MST: Rating",
  CPTRATING  = "CPT: Rating",
  CPTTIME    = "CPT: Time",
  MathCorr   = "Math: Correct",
  MathIncorr = "Math: Incorrect",
  SLEEP      = "Sleep (hrs)"
)

apply_labels <- function(M, map){
  keep <- intersect(names(map), colnames(M))
  if (length(keep)) {
    new <- unname(map[keep])
    colnames(M)[match(keep, colnames(M))] <- new
    rownames(M)[match(keep, rownames(M))] <- new
  }
  M
}
```

```r
R_lab      <- apply_labels(R,      label_map)
P_adj_plot <- apply_labels(P_adj_plot, label_map)
# 5) Save to high-resolution PNG
filepng("correlation_plot_demographic.png", width = 3600, height =
3600, res = 300)# 6) Plot with colored squares and BH-adjusted p's
masking non-sig cells
corrplot(
  R_lab,
  method      = "color",
  type        = "upper",
  order       = "original",
  diag        = TRUE,
  p.mat       = P_adj_plot,     # <-- BH-adjusted p-values
  sig.level   = 0.05,
  insig       = "blank",
  tl.pos      = "lt",
  tl.col      = "black",
  tl.srt      = 45,
  tl.cex      = 1.8,
  tl.offset   = 0.9,
  addCoef.col = "black",
  number.cex  = 1.6,
  number.font = 2,
  number.digits = 2,
  cl.cex      = 1.6,
  cl.ratio    = 0.15,
  cl.pos      = "r",  mar         = c(2, 2, 3, 3),
  col         = colorRampPalette(c("blue","white","red"))(200)
  addgrid.col = "gray90"
  )

dev.off()

# Correlation Plot for Psychological, Personality, and Trait Measures
# 1) Select variables & coerce numeric
PSY_selected_vars <- DissData %>%
  dplyr::select(
    BDITOTAL, BAITOTAL,
    N1ANX, N2ANG, N3D, N4SC, N5IM, N6V, NTOTAL,
    BISPUN, BASREW, BASDRV, BASFUN,
    SVTOTAL
  ) %>%
  mutate(across(everything(), ~ suppressWarnings(as.numeric(.))))

# 2) Spearman correlations (Hmisc handles ties/pairwise n)
res <- rcorr(as.matrix(PSY_selected_vars), type = "spearman")
```

```r
R <- res$r          # Spearman rho matrix
P <- res$P          # RAW p-value matrix
N <- res$n          # pairwise Ns (optional to keep)

# --- Do NOT set NA p-values to 1; adjust only real tests ---

# 3) Benjamini–Hochberg (FDR) on the unique, non-NA upper-triangle
tests
P_work <- P
diag(P_work) <- NA
upper <- upper.tri(P_work, diag = FALSE)

p_vec  <- P_work[upper]
keep   <- !is.na(p_vec)

padj_vec <- rep(NA_real_, length(p_vec))
padj_vec[keep] <- p.adjust(p_vec[keep], method = "BH")

# Rebuild a symmetric matrix of BH-adjusted p-values
P_adj <- matrix(NA_real_, nrow = nrow(P_work), ncol = ncol(P_work),
                dimnames = dimnames(P_work))
P_adj[upper] <- padj_vec
P_adj[lower.tri(P_adj)] <- t(P_adj)[lower.tri(P_adj)]
# For plotting with corrplot, set diagonal to 1 so diagonal never
shows as significant
P_adj_plot <- P_adj
diag(P_adj_plot) <- 1

# 4) Friendly labels
label_map <- c(
  BDITOTAL = "BDI-II",
  BAITOTAL = "BAI",
  N1ANX    = "N1: Anxiety",
  N2ANG    = "N2: Anger",
  N3D      = "N3: Depression",
  N4SC     = "N4: Self-Consciousness",
  N5IM     = "N5: Impulsiveness",
  N6V      = "N6: Vulnerability",
  NTOTAL   = "Neuroticism (Total)",
  BISPUN   = "Punishment Sensitivity",
  BASREW   = "Reward Responsivity",
  BASDRV   = "Drive",
  BASFUN   = "Fun Seeking",
  SVTOTAL  = "SVS Total"
)
```

```r
apply_labels <- function(M, map){
  keep <- intersect(names(map), colnames(M))
  if (length(keep)) {
    new <- unname(map[keep])
    colnames(M)[match(keep, colnames(M))] <- new
    rownames(M)[match(keep, rownames(M))] <- new
  }
  M
}

R_lab       <- apply_labels(R,          label_map)
P_adj_plotL <- apply_labels(P_adj_plot, label_map)

# 5) Save to high-resolution PNG file
png("correlation_plot_psych.png", width = 4200, height = 4200, res =
300)


# 6) Plot; BH-adjusted p's mask nonsignificant cells
corrplot(
  R_lab,
  method      = "color",
  type        = "upper",
  order       = "original",
  diag        = TRUE,
  p.mat       = P_adj_plotL,   # <-- BH-adjusted p-values supplied
here
  sig.level   = 0.05,
  insig       = "blank",
  tl.pos      = "lt",
  tl.col      = "black",
  tl.srt      = 45,
  tl.cex      = 2.0,
  tl.offset   = 0.9,
  addCoef.col = "black",
  number.cex  = 0.8,
  number.font = 2,
  number.digits = 2,
  cl.cex      = 1.8,
  cl.ratio    = 0.15,
  cl.pos      = "r",
  mar         = c(2, 2, 3, 3),
  col         = colorRampPalette(c("blue","white","red"))(200)
  addgrid.col = "gray90"
)
```

```
dev.off()

# HRV Correlation Plot
# 1) Select HRV variables & coerce numeric
hrv_selected_vars <- DissData %>%
  dplyr::select(
    R1HFLOG, R1LFLOG, R1RMSSDLOG,
    R2HFLOG, R2LFLOG, R2RMSSDLOG,
    R3HFLOG, R3LFLOG, R3RMSSDLOG,
    MSTHFLOG, MSTLFLOG, MSTRMSSDLOG,
    CPTHFLOG, CPTLFLOG, CPTRMSSDLOG
  ) %>%
  mutate(across(everything(), ~ suppressWarnings(as.numeric(.))))

# 2) Spearman correlations (Hmisc handles ties/pairwise Ns)
res <- rcorr(as.matrix(hrv_selected_vars), type = "spearman")
R <- res$r          # Spearman rho matrix
P <- res$P          # RAW p-value matrix
N <- res$n          # pairwise Ns (optional)

# --- Do NOT set NA p-values to 1; adjust only the real tests ---

# 3) Benjamini–Hochberg (FDR) on unique, non-NA upper-tri tests
P_work <- P
diag(P_work) <- NA
upper <- upper.tri(P_work, diag = FALSE)

p_vec  <- P_work[upper]
keep   <- !is.na(p_vec)

padj_vec <- rep(NA_real_, length(p_vec))
padj_vec[keep] <- p.adjust(p_vec[keep], method = "BH")

# Rebuild a symmetric BH-adjusted p matrix
P_adj <- matrix(NA_real_, nrow = nrow(P_work), ncol = ncol(P_work),
                dimnames = dimnames(P_work))
P_adj[upper] <- padj_vec
P_adj[lower.tri(P_adj)] <- t(P_adj)[lower.tri(P_adj)]

# For plotting with corrplot, set diagonal to 1 so diag never shows as
significant
P_adj_plot <- P_adj
diag(P_adj_plot) <- 1

# 4) Friendly HRV labels
label_map <- c(
```

```
  R1HFLOG     = "Baseline: HF-HRV",
  R1LFLOG     = "Baseline: LF-HRV",
  R1RMSSDLOG  = "Baseline: RMSSD",
  R2HFLOG     = "Rest 1: HF-HRV",
  R2LFLOG     = "Rest 1: LF-HRV",
  R2RMSSDLOG  = "Rest 1: RMSSD",
  R3HFLOG     = "Rest 2: HF-HRV",
  R3LFLOG     = "Rest 2: LF-HRV",
  R3RMSSDLOG  = "Rest 2: RMSSD",
  MSTHFLOG    = "MST: HF-HRV",
  MSTLFLOG    = "MST: LF-HRV",
  MSTRMSSDLOG = "MST: RMSSD",
  CPTHFLOG    = "CPT: HF-HRV",
  CPTLFLOG    = "CPT: LF-HRV",
  CPTRMSSDLOG = "CPT: RMSSD"
)

apply_labels <- function(M, map){
  keep <- intersect(names(map), colnames(M))
  if (length(keep)) {
    new <- unname(map[keep])
    colnames(M)[match(keep, colnames(M))] <- new
    rownames(M)[match(keep, rownames(M))] <- new
  }
  M
}
R_lab        <- apply_labels(R,           label_map)
P_adj_plot_L <- apply_labels(P_adj_plot,  label_map)

# 5) Save to high-resolution PNG file
png("correlation_plot_hrv.png", width = 4500, height = 4500, res =
300)

# 6) Plot with colored squares for better readability
corrplot(
  R_lab,
  method      = "color",
  type        = "upper",
  order       = "original",
  diag        = TRUE,
  p.mat       = P_adj_plot_L,   # <-- BH-adjusted p-values here
  sig.level   = 0.05,
  insig       = "blank",
  tl.pos      = "lt",
  tl.col      = "black",
```

```
  tl.srt      = 45,
  tl.cex      = 2.0,
  tl.offset   = 0.9,
  addCoef.col = "black",
  number.cex  = 1.5,
  number.font = 2,
  number.digits = 2,
  cl.cex      = 1.8,
  cl.ratio    = 0.15,
  cl.pos      = "r",
  mar         = c(2, 2, 3, 3),
  col         = colorRampPalette(c("blue","white","red"))(200)
)

dev.off()

### HYPOTHESIS 1 ANALYSES BEGIN HERE ###
# Hypothesis 1a: R1HFLOG
# Initial normality check for R1HFLOG
shapiro_h1a <- shapiro.test(DissData$R1HFLOG)
shapiro_h1a$p.value  # Print Shapiro-Wilk p-value for h1a

# Fit regression model for h1a
h1a <- lm(R1HFLOG ~ NTOTAL + AGE + GENDER + BDITOTAL + BAITOTAL +
                        SLEEP + RESEARCHER, data = DissData)

# Residuals vs. Fitted plot for homoscedasticity (h1a)
plot(fitted(h1a), residuals(h1a),
     main = "Residuals vs Fitted (h1a)", xlab = "Fitted Values", ylab
= "Residuals")
abline(h = 0, col = "red")

# Perform the Breusch-Pagan test
bptest(h1a)

# Variance Inflation Factor (VIF) for multicollinearity (h1a)
vif(h1a)

# Summary of the final model (h1a)
summary(h1a)

# Hypothesis 1b: R1LFLOG
# Initial normality check for R1LFLOG
shapiro_h1b <- shapiro.test(DissData$R1LFLOG)
shapiro_h1b$p.value  # Print Shapiro-Wilk p-value for h1b
```

```r
# Fit regression model for h1b
h1b <- lm(R1LFLOG ~ NTOTAL + AGE + GENDER + BDITOTAL + BAITOTAL +
SLEEP
                      + RESEARCHER, data = DissData)

# Residuals vs. Fitted plot for homoscedasticity (h1b)
plot(fitted(h1b), residuals(h1b),
     main = "Residuals vs Fitted (h1b)", xlab = "Fitted Values", ylab
= "Residuals")
abline(h = 0, col = "red")

#Breusch-Pagan Test
bptest(h1b)

# Variance Inflation Factor (VIF) for multicollinearity (h1b)
vif(h1b)

# Summary of the final model (h1b)
summary(h1b)

# Hypothesis 1c: R1RMSSDLOG
# Initial normality check for LOG_R1RMSSD
shapiro_h1c <- shapiro.test(DissData$R1RMSSDLOG)
shapiro_h1c$p.value  # Print Shapiro-Wilk p-value for h1c

# Fit regression model for h1c (LOG_R1RMSSD)
h1c <- lm(R1RMSSDLOG ~ NTOTAL + AGE + GENDER + BDITOTAL + BAITOTAL +
                        SLEEP + RESEARCHER, data = DissData)

# Residuals vs. Fitted plot for homoscedasticity (h1c)
plot(fitted(h1c), residuals(h1c),
     main = "Residuals vs Fitted (h1c)", xlab = "Fitted Values", ylab
= "Residuals")
abline(h = 0, col = "red")

#Breusch-Pagan Test
bptest(h1c)

# Variance Inflation Factor (VIF) for multi-collinearity (h1c)
vif(h1c)

# Summary of the final model (h1c)
summary(h1c)

### HYPOTHESIS 2 BEGINS HERE ###
#Transforming into long data
# Unified long table
```

```r
long_all <- DissData %>%
  dplyr::select(
    ID, RESEARCHER, NTOTAL, SVTOTAL, MSTRATING, CPTRATING, CPTTIME,
MathCorr,
    MSTHFLOG, CPTHFLOG, MSTLFLOG, CPTLFLOG, MSTRMSSDLOG, CPTRMSSDLOG
  ) %>%
  tidyr::pivot_longer(
    cols = c(MSTHFLOG, CPTHFLOG, MSTLFLOG, CPTLFLOG, MSTRMSSDLOG,
CPTRMSSDLOG),
    names_to   = c("Task", "Metric"),
    names_pattern = "^(MST|CPT)(HFLOG|LFLOG|RMSSDLOG)$",
    values_to  = "Value"
  ) %>%
  dplyr::mutate(Task = factor(Task, levels = c("MST","CPT"))) %>%
  tidyr::drop_na(Value)

# Then, for H2a/H2b/H2c you'd do:
H2a_dat <- long_all %>% dplyr::filter(Metric == "HFLOG")    %>%
dplyr::rename(HFLOG = Value)
H2b_dat <- long_all %>% dplyr::filter(Metric == "LFLOG")    %>%
dplyr::rename(LFLOG = Value)
H2c_dat <- long_all %>% dplyr::filter(Metric == "RMSSDLOG") %>%
dplyr::rename(RMSSDLOG = Value)

# ---- Step 2: Normality by task (Shapiro on outcome within Task) ----
normality_h2a <- H2a_dat %>%
  dplyr::group_by(Task) %>%
  dplyr::summarise(Shapiro_p = shapiro.test(HFLOG)$p.value, .groups =
"drop")
print(normality_h2a)

normality_h2b <- H2b_dat %>%
  dplyr::group_by(Task) %>%
  dplyr::summarise(Shapiro_p = shapiro.test(LFLOG)$p.value, .groups =
"drop")
print(normality_h2b)

normality_h2c <- H2c_dat %>%
  dplyr::group_by(Task) %>%
  dplyr::summarise(Shapiro_p = shapiro.test(RMSSDLOG)$p.value, .groups
= "drop")
print(normality_h2c)

## --------------------------
## H2a (HFLOG): MST vs CPT
## --------------------------
```

```r
H2a_model <- lmer(
  HFLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME +
MathCorr
  + RESEARCHER + (1 | ID),
  data = H2a_dat, REML = TRUE
)

# Kenward-Roger coefficient-level tests (t/df/p)
H2a_sum   <- summary(H2a_model, ddf = "Kenward-Roger")
# Kenward-Roger ANOVA-style tests (F/df/p)
H2a_anova <- anova(H2a_model,   ddf = "Kenward-Roger")

print(H2a_sum)
print(H2a_anova)

## ---------------------------
## H2b (LFLOG): MST vs CPT
## ---------------------------
H2b_model <- lmer(
  LFLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME +
MathCorr +
    RESEARCHER + (1 | ID),
  data = H2b_dat, REML = TRUE
)

H2b_sum   <- summary(H2b_model, ddf = "Kenward-Roger")
H2b_anova <- anova(H2b_model,   ddf = "Kenward-Roger")

print(H2b_sum)
print(H2b_anova)

## ---------------------------
## H2c (RMSSDLOG): MST vs CPT
## ---------------------------
H2c_model <- lmer(
  RMSSDLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME
+ MathCorr
  + RESEARCHER + (1 | ID),
  data = H2c_dat, REML = TRUE
)

H2c_sum   <- summary(H2c_model, ddf = "Kenward-Roger")
H2c_anova <- anova(H2c_model,   ddf = "Kenward-Roger")

print(H2c_sum)
print(H2c_anova)
```

```r
# Step 4: Residual diagnostics for H2a, H2b, H2c

par(mfrow = c(2, 3))  # 2 rows x 3 columns of plots

## H2a (HFLOG)
plot(fitted(H2a_model), resid(H2a_model),
     main = "H2a: Resid vs Fitted",
     xlab = "Fitted", ylab = "Residuals")
abline(h = 0, lty = 2, col = "red")

qqnorm(resid(H2a_model), main = "H2a: QQ plot")
qqline(resid(H2a_model), col = "red")

## H2b (LFLOG)
plot(fitted(H2b_model), resid(H2b_model),
     main = "H2b: Resid vs Fitted",
     xlab = "Fitted", ylab = "Residuals")
abline(h = 0, lty = 2, col = "red")

qqnorm(resid(H2b_model), main = "H2b: QQ plot")
qqline(resid(H2b_model), col = "red")

## H2c (RMSSDLOG)
plot(fitted(H2c_model), resid(H2c_model),
     main = "H2c: Resid vs Fitted",
     xlab = "Fitted", ylab = "Residuals")
abline(h = 0, lty = 2, col = "red")

qqnorm(resid(H2c_model), main = "H2c: QQ plot")
qqline(resid(H2c_model), col = "red")

## ---- Step 5 (final): random-intercept only, then re-run KR outputs
----

## H2a (HFLOG) — final random-effects: (1 | ID)
H2a_model <- lmer(
  HFLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME +
MathCorr
  + RESEARCHER + (1 | ID),
  data = H2a_dat, REML = TRUE
)
H2a_sum   <- summary(H2a_model, ddf = "Kenward-Roger")
H2a_anova <- anova(H2a_model,   ddf = "Kenward-Roger")
print(H2a_sum)
print(H2a_anova)
```

```r
## H2b (LFLOG) — final random-effects: (1 | ID)
H2b_model <- lmer(
  LFLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME +
MathCorr
  + RESEARCHER + (1 | ID),
  data = H2b_dat, REML = TRUE
)
H2b_sum   <- summary(H2b_model, ddf = "Kenward-Roger")
H2b_anova <- anova(H2b_model,   ddf = "Kenward-Roger")
print(H2b_sum)
print(H2b_anova)

## H2c (RMSSDLOG) — final random-effects: (1 | ID)
H2c_model <- lmer(
  RMSSDLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME
+ MathCorr
  + RESEARCHER + (1 | ID),
  data = H2c_dat, REML = TRUE
)
H2c_sum   <- summary(H2c_model, ddf = "Kenward-Roger")
H2c_anova <- anova(H2c_model,   ddf = "Kenward-Roger")
print(H2c_sum)
print(H2c_anova)

# Step 6: Multi-collinearity check for all three models

check_collinearity(H2a_model)
check_collinearity(H2b_model)
check_collinearity(H2c_model)

# Step 7: Parametric bootstrap sensitivity for H2c (RMSSDLOG)

set.seed(202)  # for reproducibility

# Kenward-Roger table for comparison (primary method; quick to
compute)
mx_H2c_KR <- mixed(
  RMSSDLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME
+ MathCorr
  + RESEARCHER + (1 | ID),
  data   = H2c_dat,
  method = "KR",
  REML   = TRUE
)
mx_H2c_KR   # Look at the Task row: "Pr(>F)" column
```

```r
# Parametric bootstrap (sensitivity). Increase nsim for final runs.
mx_H2c_PB <- mixed(
  RMSSDLOG ~ Task + NTOTAL + SVTOTAL + MSTRATING + CPTRATING + CPTTIME
+ MathCorr
  + RESEARCHER + (1 | ID),
  data    = H2c_dat,
  method  = "PB",
  REML    = TRUE,
  args_test = list(nsim = 1999)
)
mx_H2c_PB   # Look at the Task row: "Pr(>F)" (PB p-value)
pb_df <- as.data.frame(mx_H2c_PB$anova_table)
pb_df

# Parametric bootstrap CIs for all fixed effects (beta_) from the same
H2c_model
set.seed(202)
pb_ci <- confint(H2c_model, parm = "beta_", method = "boot",
                 type = "parametric", nsim = 1999)  # adjust sims as
desired
pb_ci

### HYPOTHESIS 3 BEGINS HERE ###
## ----------------------------
## H3 DATA PREP (single source)
## ----------------------------

# 1) Build the three analysis frames from the unified long table
H3a_dat <- long_all %>%
  dplyr::filter(Metric == "HFLOG") %>%
  dplyr::select(
    ID, Task,
    HFLOG = Value,
    NTOTAL, SVTOTAL, MathCorr, CPTTIME, MSTRATING, CPTRATING,
RESEARCHER
  )

H3b_dat <- long_all %>%
  dplyr::filter(Metric == "LFLOG") %>%
  dplyr::select(
    ID, Task,
    LFLOG = Value,
    NTOTAL, SVTOTAL, MathCorr, CPTTIME, MSTRATING, CPTRATING,
RESEARCHER
  )
```

```r
H3c_dat <- long_all %>%
  dplyr::filter(Metric == "RMSSDLOG") %>%
  dplyr::select(
    ID, Task,
    RMSSDLOG = Value,
    NTOTAL, SVTOTAL, MathCorr, CPTTIME, MSTRATING, CPTRATING,
RESEARCHER
  )

# 2) Ensure Task is a 2-level factor with MST as the baseline
H3a_dat$Task <- factor(H3a_dat$Task, levels = c("MST","CPT"))
H3b_dat$Task <- factor(H3b_dat$Task, levels = c("MST","CPT"))
H3c_dat$Task <- factor(H3c_dat$Task, levels = c("MST","CPT"))

# 3) For Type III tests later (car::Anova type = 3), use sum-to-zero
contrasts ONCE here
contrasts(H3a_dat$Task) <- contr.sum(2)
contrasts(H3b_dat$Task) <- contr.sum(2)
contrasts(H3c_dat$Task) <- contr.sum(2)

## ----------------------------
## H3 LINEARITY CHECKS (quick visuals)
## ----------------------------
plot_linearity <- function(dat, yvar, ylab) {
  ggplot(dat, aes(x = NTOTAL, y = .data[[yvar]], color = Task)) +
    geom_point(alpha = 0.6) +
    geom_smooth(method = "lm", se = TRUE) +
    labs(title = paste("Linearity check:", ylab, "~ NTOTAL by Task"),
         x = "Trait Neuroticism (NTOTAL)", y = ylab, color = "Task") +
    theme_minimal()
}

# (Optional to view)
plot_linearity(H3a_dat, "HFLOG",    "HF-HRV (log)")
plot_linearity(H3b_dat, "LFLOG",    "LF-HRV (log)")
plot_linearity(H3c_dat, "RMSSDLOG", "RMSSD (log)")

## ----------------------------
## H3 MODERATION MODELS (OLS)
## Outcome ~ NTOTAL * Task + covariates
## ----------------------------

# Center Neuroticism (for interpretability of Task main effect)
H3a_dat$NTOTAL_C <- scale(H3a_dat$NTOTAL, center = TRUE, scale =
FALSE)
H3b_dat$NTOTAL_C <- scale(H3b_dat$NTOTAL, center = TRUE, scale =
```

```
FALSE)
H3c_dat$NTOTAL_C <- scale(H3c_dat$NTOTAL, center = TRUE, scale =
FALSE)

# Formulas
form_H3a <- HFLOG    ~ NTOTAL_C * Task + SVTOTAL + MathCorr + CPTTIME
+ MSTRATING + CPTRATING + RESEARCHER
form_H3b <- LFLOG    ~ NTOTAL_C * Task + SVTOTAL + MathCorr + CPTTIME
+ MSTRATING + CPTRATING + RESEARCHER
form_H3c <- RMSSDLOG ~ NTOTAL_C * Task + SVTOTAL + MathCorr + CPTTIME
+ MSTRATING + CPTRATING + RESEARCHER

# Fit models
mH3a <- lm(form_H3a, data = H3a_dat)
mH3b <- lm(form_H3b, data = H3b_dat)
mH3c <- lm(form_H3c, data = H3c_dat)

# Coefficient-level output
summary(mH3a); summary(mH3b); summary(mH3c)

# Type III ANOVA tables (primary moderation test = NTOTAL_C:Task)
car::Anova(mH3a, type = 3)
car::Anova(mH3b, type = 3)
car::Anova(mH3c, type = 3)

## ----------------------------
## H3 ASSUMPTION CHECKS
## ----------------------------
check_assumptions <- function(model, label) {
  cat("\n---", label, "---\n")
  par(mfrow = c(1,2))
  plot(model, which = 1, main = paste(label, "Residuals vs Fitted"))
  plot(model, which = 2, main = paste(label, "QQ Plot"))
  par(mfrow = c(1,1))
  print(shapiro.test(residuals(model)))   # normality
  print(lmtest::bptest(model))            # heteroscedasticity
  print(car::vif(model, type = "predictor"))  # multicollinearity with
interaction
}

check_assumptions(mH3a, "H3a: HFLOG ~ NTOTAL_C * Task")
check_assumptions(mH3b, "H3b: LFLOG ~ NTOTAL_C * Task")
check_assumptions(mH3c, "H3c: RMSSDLOG ~ NTOTAL_C * Task")

## ----------------------------
## H3 MODERATION PLOTS (consistent Y-axis)
```

```r
## ----------------------------

# Single helper: pass desired y-limits once for consistent scaling
across plots
plot_mod <- function(dat, yvar, ylab, ylimits = c(0, 12)) {
  ggplot(dat, aes(x = NTOTAL_C, y = .data[[yvar]], color = Task)) +
    geom_point(alpha = 0.6) +
    geom_smooth(method = "lm", se = TRUE) +
    labs(title = paste("Moderation:", ylab, "~ NTOTAL by Task"),
         x = "Trait Neuroticism (centered)", y = ylab, color = "Task")
+
    scale_y_continuous(limits = ylimits,
                       breaks = seq(ylimits[1], ylimits[2], by = 2)) +
    theme_minimal()
}

# Create plots (shared y-axis 0–12; adjust y-limits if you prefer)
p_H3a <- plot_mod(H3a_dat, "HFLOG",    "HF-HRV", ylimits = c(0,12))
p_H3b <- plot_mod(H3b_dat, "LFLOG",    "LF-HRV", ylimits = c(0,12))
p_H3c <- plot_mod(H3c_dat, "RMSSDLOG", "RMSSD",  ylimits = c(0,12))

p_H3a; p_H3b; p_H3c

### Exploratory Analyses ###
# Normality testing for CPT and MST Stress Ratings
shapiro.test(DissData$CPTRATING - DissData$MSTRATING)

# Wilcoxon sign-ranked test comparing CPT and MST Stress Ratings
wilcox.test(DissData$MSTRATING, DissData$CPTRATING, paired = TRUE)

# Facet Level Personality Traits Exploratory Analyses

# HRV and Neuroticism Facets Correlation Plot
# 1) Select HRV variables & coerce numeric
hrv_pers_selected_vars <- DissData %>%
  dplyr::select(
    R1HFLOG, R1LFLOG, R1RMSSDLOG,
    R2HFLOG, R2LFLOG, R2RMSSDLOG,
    R3HFLOG, R3LFLOG, R3RMSSDLOG,
    NTOTAL, N1ANX, N2ANG, N3D,
    N4SC, N5IM, N6V

  ) %>%
  mutate(across(everything(), ~ suppressWarnings(as.numeric(.))))

# 2) Spearman correlations (Hmisc handles ties/pairwise Ns)
```

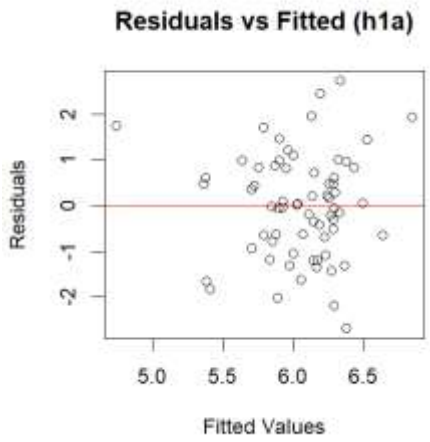
```r
res <- rcorr(as.matrix(hrv_pers_selected_vars), type = "spearman")
R <- res$r          # Spearman rho matrix
P <- res$P          # RAW p-value matrix
N <- res$n          # pairwise Ns (optional)

# --- Do NOT set NA p-values to 1; adjust only the real tests ---

# 3) Benjamini–Hochberg (FDR) on unique, non-NA upper-tri tests
P_work <- P
diag(P_work) <- NA
upper <- upper.tri(P_work, diag = FALSE)

p_vec  <- P_work[upper]
keep   <- !is.na(p_vec)

padj_vec <- rep(NA_real_, length(p_vec))
padj_vec[keep] <- p.adjust(p_vec[keep], method = "BH")

# Rebuild a symmetric BH-adjusted p matrix
P_adj <- matrix(NA_real_, nrow = nrow(P_work), ncol = ncol(P_work),
                dimnames = dimnames(P_work))
P_adj[upper] <- padj_vec
P_adj[lower.tri(P_adj)] <- t(P_adj)[lower.tri(P_adj)]

# For plotting with corrplot, set diagonal to 1 so diag never shows as
significant
P_adj_plot <- P_adj
diag(P_adj_plot) <- 1

# 4) Friendly HRV labels
label_map <- c(
  R1HFLOG     = "Baseline: HF-HRV",
  R1LFLOG     = "Baseline: LF-HRV",
  R1RMSSDLOG  = "Baseline: RMSSD",
  R2HFLOG     = "Rest 1: HF-HRV",
  R2LFLOG     = "Rest 1: LF-HRV",
  R2RMSSDLOG  = "Rest 1: RMSSD",
  R3HFLOG     = "Rest 2: HF-HRV",
  R3LFLOG     = "Rest 2: LF-HRV",
  R3RMSSDLOG  = "Rest 2: RMSSD",
  NTOTAL      = "Total Neuroticism",
  N1ANX       = "Anxiety",
  N2ANG       = "Anger",
  N3D         = "Depression",
  N4SC        = "Self-Consciousness",
  N5IM        = "Immoderation",
```

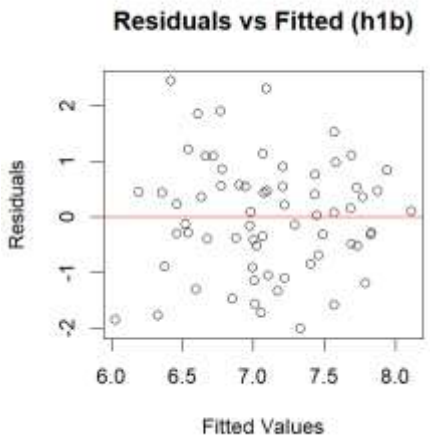
```
  N6V         = "Vulnerability"
)

apply_labels <- function(M, map){
  keep <- intersect(names(map), colnames(M))
  if (length(keep)) {
    new <- unname(map[keep])
    colnames(M)[match(keep, colnames(M))] <- new
    rownames(M)[match(keep, rownames(M))] <- new
  }
  M
}
R_lab        <- apply_labels(R,           label_map)
P_adj_plot_L <- apply_labels(P_adj_plot,  label_map)

# 5) (Optional) larger device in R GUI; RStudio pane usually auto-
scales
# grDevices::dev.new(width = 16, height = 14)

# 6) Plot: BH-adjusted p's mask nonsignificant cells
corrplot(
  R_lab,
  method      = "circle",
  type        = "upper",
  order       = "original",
  diag        = TRUE,
  p.mat       = P_adj_plot_L,   # <-- BH-adjusted p-values here
  sig.level   = 0.05,
  insig       = "blank",
  tl.pos      = "lt",
  tl.col      = "black",
  tl.srt      = 45,
  tl.cex      = 0.9,
  addCoef.col = "black",
  number.cex  = 0.8,
  cl.cex      = 0.9,
  cl.ratio    = 0.1,
  mar         = c(2, 2, 2, 2),
  col         = colorRampPalette(c("blue","white","red"))(200)
)

# Change Scores for HRV across Relaxation Phases
# Compute change scores

DissData <- DissData %>%
  mutate(
```

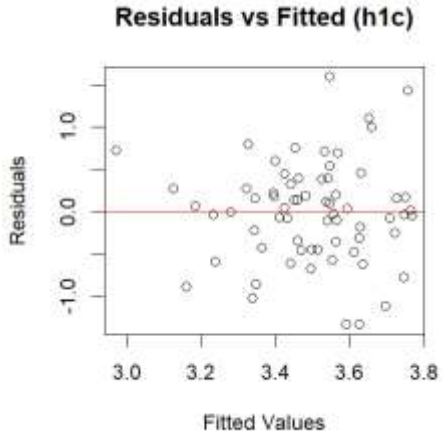
```
    HFLOG_Delta1 = R2HFLOG - R1HFLOG,
    HFLOG_Delta2 = R3HFLOG - R2HFLOG,
    HFLOG_TotalChange = R3HFLOG - R1HFLOG,

    LFLOG_Delta1 = R2LFLOG - R1LFLOG,
    LFLOG_Delta2 = R3LFLOG - R2LFLOG,
    LFLOG_TotalChange = R3LFLOG - R1LFLOG,

    RMSSDLOG_Delta1 = R2RMSSDLOG - R1RMSSDLOG,
    RMSSDLOG_Delta2 = R3RMSSDLOG - R2RMSSDLOG,
    RMSSDLOG_TotalChange = R3RMSSDLOG - R1RMSSDLOG
  )

#Inform individuals that the analysis has been completed
cat("Analysis completed successfully.\n")
```

Appendix H: Plots and Statistical Output for Assumption Checks

Hypothesis 1: Residual *vs.* Fitted Plots Linear Regressions







Statistical Assumptions

H1a:

Shapiro-Wilk: $W = 0.98$, $p = .49$
Breusch-Pagan: $BP = 3.53$, df = 7, $p = .83$

H1b:

Shapiro-Wilk: $W = 0.99$, $p = 0.72$
Breusch-Pagan: $BP = 14.90$, df = 7, $p = .04$

H1c:

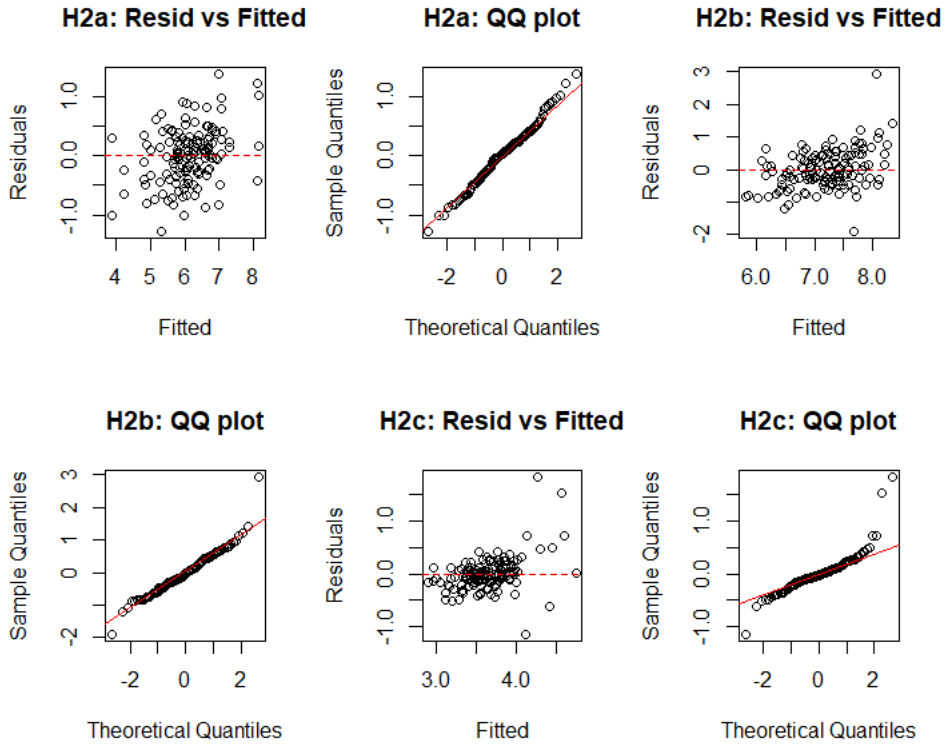
Shapiro-Wilk: $W = 0.97$, $p = .13$

Breusch-Pagan: $BP = 4.63$, df = 7, $p = .70$

H1a-H1C VIF Values:

- Neuroticism: 2.29
- Age: 1.31
- Gender: 1.03
- BDI-II Total: 2.60
- BAI Total: 2.09
- Sleep: 1.09
- Researcher: 1.23

Hypothesis 2:



H2a:

Shapiro-Wilk MST: $W = .98$, $p = .27$
Shapiro-Wilk CPT: $W = .98$, $p = .49$

H2b:

Shapiro-Wilk MST: $W = .98$, $p = .50$
Shapiro-Wilk CPT: $W = .97$, $p = .12$

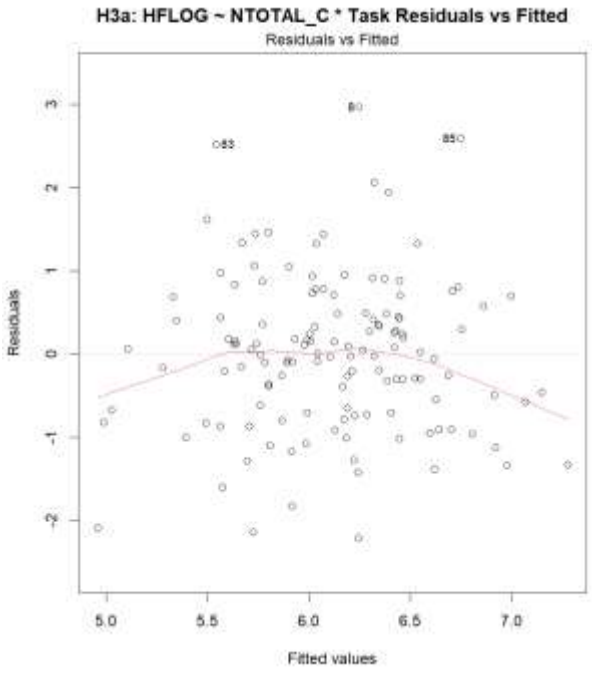
H2c:

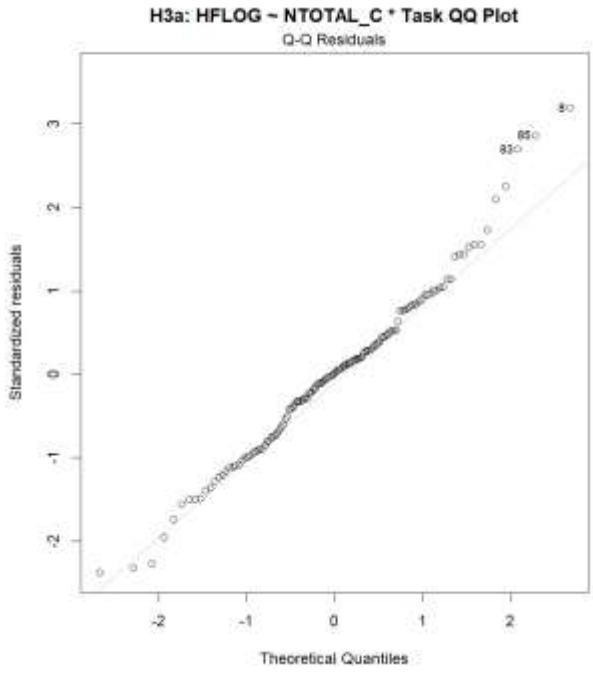
Shapiro-Wilk MST: $W = .95$, $p < .01$
Shapiro-Wilk CPT: $W = .86$, $p < .01$

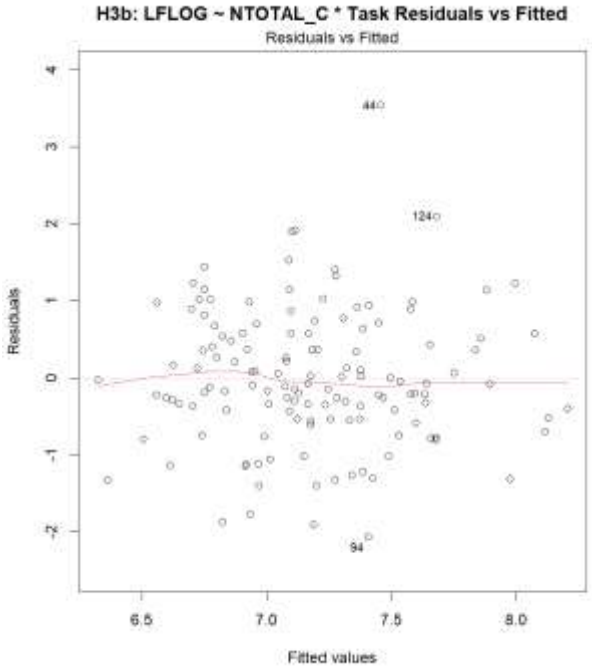
H2a-H2c VIF Values:

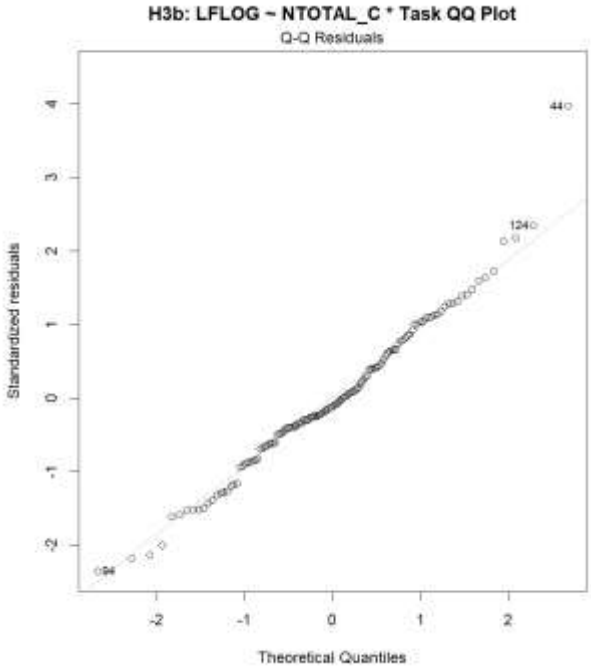
- Task: 1.00
- Neuroticism: 1.18
- Stereotype Vulnerability: 1.25
- MST Stress Rating: 1.44
- CPT Stress Rating: 1.13
- CPT Time: 1.12
- Math Accuracy: 1.25
- Researcher: 1.08

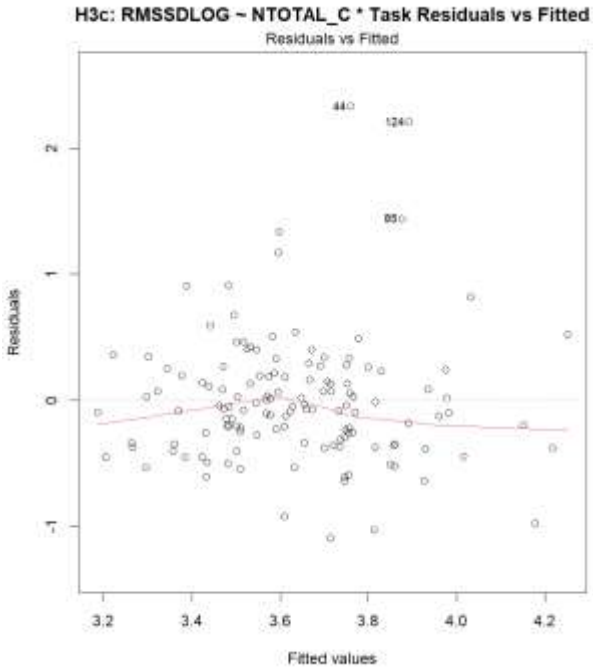
Hypothesis 3: Moderation Analyses

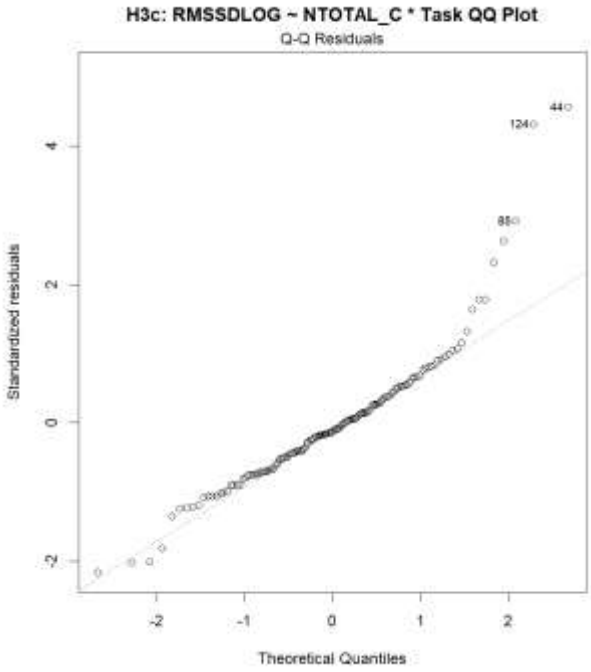












H3a:

Shapiro-Wilk: $W = 0.98$, $p = .13$
Breusch-Pagan: $BP = 4.34$, df = 9, $p = .89$

H3b:

Shapiro-Wilk: $W = 0.98$, $p = .05$

Breusch-Pagan: $BP = 10.32$, df = 9, $p = .32$

H3c:

Shapiro-Wilk: $W = 0.89$, $p < .01$
Breusch-Pagan: $BP = 4.95$, df = 9, $p = .84$

H3a-H3c VIF Values:

- Mean-Centered Neuroticism: 1.18
- Task: 1.18
- Stereotype Vulnerability: 1.25
- Math Accuracy: 1.25
- CPT Time: 1.12
- MST Stress Rating: 1.44
- CPT Stress Rating: 1.13
- Researcher: 1.08