EXAMINING SOCIAL VIGILANCE AND ASSOCIATED PHYSIOLOGICAL EFFECTS ACROSS TYPES OF SITUATIONAL STRESS

by

Riley M. O'Neill

A Thesis Submitted to the Faculty of the

DEPARTMENT OF PSYCHOLOGY

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF ARTS

In the Graduate College

THE UNIVERSITY OF ARIZONA

2022

THE UNIVERSITY OF ARIZONA
GRADUATE COLLEGE

As members of the Master's Committee, we certify that we have read the thesis prepared by: Riley M. O'Neill
titled:

and recommend that it be accepted as fulfilling the thesis requirement for the Master's Degree.

John Ruiz — Date: Apr 28, 2022

David Sbarra — Date: Apr 28, 2022

Heidi Hamann — Date: Apr 28, 2022

Final approval and acceptance of this thesis is contingent upon the candidate's submission of the final copies of the thesis to the Graduate College.

I hereby certify that I have read this thesis prepared under my direction and recommend that it be accepted as fulfilling the Master's requirement.

John Ruiz
Thesis Committee Chair
Department of Psychology

Date: Apr 28, 2022

Table of Contents

Abstract

Emerging work over the past four decades supports psychological stress as a critical determinant of cardiovascular disease (CVD). Previous research has supported social vigilance, or active watchful monitoring of one's social environment for interpersonal challenges or threats, as a candidate biobehavioral process linking stress exposure to adverse cardiovascular reactivity and recovery profiles. Review of findings in this area reveals the need for research examining whether experimentally controlled contextual vigilance cues evoke the hypothesized biobehavioral responses. The current study randomized 135 undergraduate young adults (49% male, 51% female; M_{age} = 19 years, SD_{age} = 4 years) to one of three videogames standardized as all first-person shooter scenarios, with the manipulation across games being type of situational stress (challenge, threat, neutral). Participants' dispositional social vigilance was measured via trait assessment, and participants' cardiovascular reactivity (CVR) was measured prior to, during, and after the experimental task. Analysis of covariance (ANCOVA) models revealed higher stress games evoked significantly more CVR than the neutral game, especially in the case of threat condition. Participants in the high threat condition also demonstrated the least overall recovery to baseline blood pressure. No effects of vigilance disposition were observed during gameplay and modest effects of higher vigilance were associated with better physiological recovery, in contrast to expectations. These findings contribute to understanding how higher threat social situations may connote CVD risk through pull for greater preparatory monitoring and the acute cardiovascular responses corresponding to that behavior.

Keywords: Stress, vigilance, cardiovascular reactivity

Overview

Since the onset of the COVID-19 global pandemic, high density social situations now present increased environmental threat in the form of possible transmission of the COVID-19 virus. The American Psychological Association (2020) recently published its findings that American adults are experiencing markedly high levels of stress during 2020, particularly around social situations and fear of contracting COVID-19. When in public, people are particularly attuned to social distance measures, whether others are wearing masks, and potential signs of illness in those around them.

This state of awareness and monitoring of illness risk in social situations is an example of vigilance. Vigilance refers to the active watchful monitoring of one's environment, intentional or automatic, for information signaling existing or potential threats to one's safety (Gump & Matthews, 1998; Smith et al., 2000; Himmelstein et al., 2014). Social vigilance, specifically, refers to this monitoring behavior when assessing social environments for potential or ongoing interpersonal challenges or threats (Smith et al., 2000; Ruiz, et al., 2017). Within Lazarus and Folkman's (1984) model of stress appraisal, social vigilance may appear as passive coping yet reflect active sensory information seeking to assess presence or change in threats and reappraisal of coping options to protect themselves.

This monitoring behavior may be adaptive in situations of immediate environmental threats, such as the case with infection risks. But on a daily basis, vigilance may become its own health risk through chronic activation of the stress response and implications for health. Research assessing social vigilance as a psychophysiological response has demonstrated individuals engaged in this excess watchful behavior exhibit increased blood pressure and vascular constriction (Gump & Matthews, 1998; Smith et al., 2000; Ruiz et al., 2017). Chronic activation of this biobehavioral pathway may be one critical moderator of stress-related disease risk including cardiovascular disease (CVD).

Social vigilance has many determinants, including socio-environmental factors, social roles, and individual differences, which have all been independently linked to CVD risk (Low, Thurston, & Matthews, 2010; Smith & Ruiz, 2002). Examining vigilance as a common, ecologically-valid, biobehavioral pathway to disease risk may help to understand how the CVD outcomes previously linked to psychosocial risk factors constitute ultimate endpoints of the vigilant behavior involved in those risk factors (Ruiz et al., 2017; Smith et al., 2000).

The present study aimed to use an experimental gameplay paradigm to examine social vigilance as a biobehavioral variable that may influence cardiovascular reactivity (CVR) across individuals and contexts.

Introduction

Ask a friend how they are doing and frequently you are soon commiserating about how "stressed out" you have both been lately. Challenges that tax one's emotional and physical resources seem inherent to the human experience. But, what does the strong emotional experience of *stress* actually entail? And beyond perception of stress, what physiological processes occur in an individual responding to psychosocial challenges and demands?

Stress encompasses an array of challenging life experiences that vary in timing, context, magnitude, and impact (Slavich, 2018). Stressors include acute, time-limited experiences, such as weaving through rush-hour traffic on the way to an important meeting. But, individuals must also cope in the face of extended, chronic adverse experiences, such as navigating daily life in a neighborhood with high-crime rates. Importantly, stressful experiences involve the perception that individuals are not able to fully control what is happening in their own lives (Pickering, 2001).

When faced with a challenging event or state with situational demands beyond the amount that an individual feels they are able to adapt and cope with, the body enacts differential stress responses according to the nature and timeframe of such stressors (Smyth et al., 2017). To deal with immediate, proximal threats, such as encountering a mountain lion mid-trail run or bumping into an ex-partner while at a bar, the primary stress response elapses within minutes or seconds of the stressor. It is an entirely electrical response that operates through the sympathetic nervous system, increasing heart rate and blood pressure to direct oxygen-rich blood to components of the body needed for the “fight-or-flight” response to overcome such situations.

However, any reader who has spent hours hustling to complete a last-minute term-paper knows not all stressful situations are resolved through quick conflict or escape. After a couple of minutes following the realization that a long-ignored due date is *tomorrow*, the body’s secondary stress response has enacted and can last hours or longer. To provide the necessary energy for dealing with longer stressors, this hormonal response operates through the hypothalamic–pituitary–adrenal (HPA) axis system to catalyze cortisol release. Cortisol is released non-specifically, throughout the entire body, breaking down anything it can find to generate energy (e.g. glucose) (Miller, Chen, & Cole, 2009). The student who finds a “second wind” around 2:30am for finishing that term-paper is experiencing the energic benefits of newly available glucose thanks to the cortisol released by the HPA axis.

The primary and secondary stress response systems mobilize energy needed to escape an immediate danger or overcome a short-term challenge, but the body calls upon a third system when coping with even longer-term stressors. The tertiary stress response takes the longest to mobilize (six to eight hours) and can then last indefinitely, with the goal of redirecting bodily resources towards meeting immediate needs moment to moment throughout the stressor. This immunological response operates by suppressing certain components of the immune system that are not needed for the immediate stressor, such as protection from viral infections, while also enhancing the functioning of other immune-responses, such as inflammatory responses (Miller, Chen, & Cole, 2009). Newlyweds who contract the flu on day one of their honeymoon are familiar with the weakened immune response from weeks of long-term stress leading up to the "happiest day of their lives."

This brief overview of physiological response systems illustrates how stress extends beyond a psychosocial phenomenon.

Conceptualizing Stress

It is important to understand the events and processes that comprise stress. Is stress confined to just the moment in which an adverse, challenging event takes place, or does it extend more broadly than the moment? There exists a large body of psychosocial stress research aimed at describing how individuals perceive stressful events and why stress responses are often carried out to detrimental extents. Throughout the 20th century, two major conceptual views prevailed: stress is defined as a response; or, stress constitutes an event.

Walter B. Cannon's famous "fight-or-flight" theory, posited in the 1920's, characterized stress response as a united activation of the sympathoadrenal system that automatically occurred when an organism's natural resting state was threatened by a stressor (Goldstein & Kopin, 2017). Once a stressor was overcome through direct, defensive action ("fight") or escape ("flight"), homeostasis was restored and the stress experience was resolved. Hans Selye (1946) subsequently included this "fight-or-flight" response concept in the first stage of his General

Adaptation Syndrome (GAS) theory, which characterized the stress response as three stages of physiological response uniformly carried out for all stressors. In the immediate response to a stressor, an initial "alarm phase" elapses, including high physiological activity (e.g. heart rate, cortisol secretion, etc.) comprising that fight-or-flight reaction. If the stressor continues to be present beyond this first phase, the body eventually has to limit how much it can contribute to a response and ultimately may stop being able to address the stressor entirely, and Selye (1946) posited this eventual exhaustion of physiological resources leads to disease.

To address the limitations related to the ideas of uniform, universal stress responses presented in these initial theories, Lazarus and Folkman (1984) presented their theory of stress appraisal and coping. Within this framework, the stress response begins with individuals' primary appraisal of whether a situation presents a threat or challenge to their safety and goals. If a situation is appraised as threatening or challenging, then individuals conduct a secondary appraisal of whether they possess sufficient resources to cope with the situation that has been appraised as stressful. The subsequent reactions and actions that will occur in response to coping with the stress are dependent on these appraisal processes (Folkman, 1984; Berjot & Gillet, 2011).

While Lazarus and Folkman's stress appraisal theory became a dominant framework in the field of stress research, more recent theoretical developments include Brosscot, Verkuil, & Thayer's (2018) Generalized Unsafety Theory of Stress (GUTS). GUTS maintains the "response" basis from earlier theories, but this theory posits the default stress response is consistently "on" and only inhibited in individuals who perceive themselves as being in safe contexts. Therefore chronic stress is really the result of the consistent perception of being unsafe (Brosscot, Verkuil, Thayer, 2018). Slavich's (2020) Social Safety Theory of stress presents a similar hypothesis that individuals' appraisal of potentially threatening social contexts is shaped by schemas they have developed regarding their own capacity to safely navigate social situations. Therefore, individuals' stressful experiences are influenced not only by the discrete stressors to which they are exposed, but also by the pre-set worldviews through which they perceive these experiences. The GUTS and Social Safety Theory frameworks were both developed to address the question of how psychosocial stress impacts individuals beyond the acute moment of an adverse event. Within both theories, vigilance for potential threats presents a behavioral indicator of an individual's perception of unsafety.

These models, among others, were developed as heuristics to explain how our stress response is activated and why our bodies would allow the accompanying physiological events to extend longer than necessary. Over the past decade, stress research has also begun evolving to recognize the stress experience beyond solely events or responses, but instead as processes (Verkuil et al., 2009; Brosschot et al., 2005; Slavich, 2018). After addressing stress's relevance to health, I will return to this conceptual shift to viewing stress at the process-level.

Stress, Allostatic Load, and Health

Stress is both beneficial and harmful for individuals. To handle certain threats to safety and challenges to goals, you need stress and its accompanying physiological responses. In life threatening situations, such as the aforementioned mountain lion scenario, the stress response is necessary for mobilizing the physiological resources required for possible escape. The stress response sounds an alarm signaling our goals are in jeopardy and motivates us to pay attention and do something (Smyth et al., 2013; Kiecolt-Glaser et al., 2020). However, modern daily stressors rarely include mountain lion-level threats to our safety. Coping with the daily frustrations of rush hour traffic or disagreements with a spouse over finances does not require the physiological resources mobilized by the stress response. In these situations, the stress response is unnecessarily activated and has an overall harmful effect on the body.

Following an adaptive stress response that mobilizes necessary resources for avoiding harm or performing as needed during short-term *acute stressors*, the body should return to pre-stress levels of physiological activation (e.g. heart rate, blood pressure) relatively quickly. Typical physiological functioning outside of the stress response characterizes the body's *homeostatic* set-point, or the tonic level of activation around which it aims to function regularly. *Allostasis* refers to the process of trying to maintain stability around that homeostatic state in the context of disruptions and challenges to the natural state, such as stressors (McEwen, 1998).

But, mobilization of the physiological processes required in stress responses is not free, and calling upon these resources can come at a cost over time. Stressful experiences and our perceived distress associated with such stressors do not always resolve quickly. The daily traffic and spousal disagreement referenced above exemplify how modern adversities and the physiological responses associated with such stressors often extend for longer periods, becoming *chronic stressors*. McEwen (1998) posited that when met with chronic environmental challenges to the body's internal balance, the effects of allostasis accumulate in *allostatic load*, or wear and tear on the body built up with repeated and chronic stress exposure. Continual and extended activations of the autonomic, cardiovascular, endocrine, and immunological mechanisms involved in stress response would then play a critical role in disease development over time (McEwen & Wingfield, 2003). While "wear and tear" presents an abstract description of physiological damage, a vast body of stress-health research includes robust epidemiological and experimental findings demonstrating stress's harmful impact on health and wellbeing.

Stress harms physical health in ways that include increasing one's risk for viral infections, HIV/AIDS, metabolic diseases, cardiovascular disease, and overall mortality (Smyth et al., 2013). Stress is particularly bad in the case of the leading cause of mortality in the U.S., cardiovascular disease (CVD) (Xu et al., 2020). CVD includes a number of conditions afflicting the vascular systems and heart organ, including coronary artery disease (CAD), coronary heart disease (CHD), hypertension, myocardial infarction, congestive heart failure, peripheral artery disease, and stroke (Benjamin et al., 2019). Epidemiological research has identified the robust clinical phenomenon that psychosocial stress presents one of the most significant sources of CVD risk. Cheng et al. (2014) examined temporal trends in the population attributable risk for CVD, finding that the five traditional risk factors (hypertension, diabetes, obesity, hypercholesterolemia, smoking) only accounted for 53% of population attributable risk for CVD. These findings indicate that research targeting solely traditional risk factors as intervention points for reducing CVD outcomes only addresses about half of CVD risk sources. The INTERheart study from Rosengreen et al. (2004), however, showed that when stress was included in heart disease risk assessment, the amount of variance explained in heart disease outcomes increased to 90%. These epidemiological findings indicated that psychosocial stress was comparable to hypertension and abdominal obesity as a predictor of individuals' risk of myocardial infarction events (also known as, "heart attacks") (Rosengreen et al., 2004).

Additional large-scale, epidemiological studies have supported this association between perceived stress exposure and various CVD risks. Ferrie et al. (2013) demonstrated that work-specific stress was associated with a marked increase in risk for developing CHD in a prospective study of over 4,000 adults. Through these large-scale associative studies, psychosocial stress has been established as an important predictor for CVD outcomes and mortality. But, mechanistic research has also begun establishing the pathways underlying these robust stress-health associations.

Pathways from stress to adverse health

There are both indirect and direct pathways through which stress negatively impacts health. Indirect pathways include the increased likelihood of engaging in risky health behaviors and interpersonal behaviors that perpetuate stressful situations. Exposure to adverse psychosocial experiences disrupt our routines and subsequently impacts health choices in a variety of ways, such as increasing the likelihood that individuals opt for fast-food, smoking, and excessive alcohol consumption to cope with stressful experiences (Smyth et al., 2017; Anton & Miller, 2005; O'Connor et al., 2008; Clancy et al., 2016). Individuals endorsing higher levels of psychosocial stress have also been found less likely to engage in routine exercise or vegetable consumption (O'Connor et al., 2008; Kouvonen et al., 2007). While exposure to stress does not directly result in the physical toll these unhealthy choices take on the body, it significantly contributes to the likelihood that an individual engages in adverse health behaviors.

The direct pathways from stress to adverse health outcomes, which are more germane to the present discussion, include contributing to pathophysiological mechanisms, increasing risk for morbidity and mortality from disease conditions, and negatively altering how the body reacts to future stressors (Miller, Chen, & Cole, 2009; Krantz & McCeney, 2002; Pickering, 2001). A major form of CVD, coronary heart disease (CHD), is caused by coronary atherosclerosis, which is the buildup of plaque in coronary arteries. Stress responses have been shown to cause changes in blood flow, hormonal patterns, and immunological responses that could affect the development of atherosclerosis (Krantz & McCeney, 2002; Bo-Chen et al., 2019). In addition to research demonstrating linkages between physiological patterns in the stress response to mechanisms of atherosclerotic progression, prospective research has found higher atherosclerotic buildup as a function of exposure to challenging and distressing experiences in both primates and humans (Kaplan et al., 2009; Wang et al., 2007). The role of physiological responses to psychosocial stressors in the pathophysiological process of CHD development provides a robust example of a direct pathway from stress to disease outcomes.

Reacting to stress also presents a risk factor for acute cardiac events in individuals who have already begun developing various forms of CVD. When individuals already possess degrees of CVD progression, such as weakened arterial walls, elevated blood pressure overall, and atherosclerotic buildup, they are at a higher risk of suffering more intense, adverse impacts of the physiological changes accompanying the acute stress response. For example, Moller et al. (2005) demonstrated that individuals who endorsed experiences of heightened work-related stress were six times more likely to suffer myocardial infarction in the 24-hour period following acute, stressful events. Several studies have found similar results demonstrating increased risk of acute cardiac events and associated mortality in relation to natural disaster exposure (Dobson et al., 1991; Kabutoya & Kario, 2009; Bazoukis et al., 2018). The sheer force of the changes in blood pressure and immunological responses that accompany the stress responses described previously can prove to be too much for individuals with already weakened cardiovascular systems. Strike et al. (2005) demonstrated that changes in blood platelet activation and hemodynamic recovery accompanying individuals’ responses to stressful, challenging events may provide the pathophysiological explanation for the robustly established connection between stress exposure and acute cardiovascular events. Therefore, in addition to contributing to insidious onset of CVD, stress exposure also directly impacts risk for acute cardiac events.

The direct pathways linking stressful pathways to CVD have largely been explored in research addressing a "reactivity hypothesis," which posits larger and more frequent cardiovascular responses to stress will produce wear and tear on the body leading to disease. In this way, the extent to which people differ in how they react to stressors will also determine the extent to which their risk for developing diseases from stress differs (Krantz & McCeney, 2002; Lovallo, 2015). Many lines of research assessing individual difference in reactions to stress have demonstrated that stress exposure alone does not explain all associated disease risk, and that an individual's pattern of reactivity to stressful experiences also determines the subsequent risk for adverse disease outcomes. Magnitude of physiological reactions to during stressful events (e.g. changes in diastolic blood pressure [DBP] during cold pressor tasks), as well as length of time required for recovery to regular CV functioning following stress exposure (e.g. return to resting heart rate [RHR]) both predict adverse CV outcomes (Heponiemi et al., 2007; Verkuil et al., 2009; Kiecolt-Glaser et al., 2020). Related to the connection described above between the GUTS ans Social Safety theories of stress activation and vigilance behavior, activation of the stress response through anticipatory scanning and monitoring behavior would then increase one's overall propensity for physiological reactions of larger magnitudes and durations. Engaging in vigilance for threats may be an important behavioral determinant of cardiovascular reactivity and recovery in response to stress. Individual differences in experiences of and recovery from stress exposure determine variations in the amount of disease risk that is directly attributable to stress.

Shift in Stress Conceptualization

The robust associations between chronic exposure to psychosocial adversity and negative health outcomes, especially CVD, underscore the importance of understanding how individuals experience stress. To fully appreciate and effectively intervene on the adverse health outcomes resulting from stress exposure, our understanding of the stress concept must be refined to more accurately reflect the mechanisms that bring psychosocial challenges “under the skin.” Chronic stress is not experienced in terms of isolated events or individual episodes of responses. Progress in research examining the pathways from stress to health outcomes has involved the conceptual shift to viewing stress in terms of emotional, cognitive, and behavioral “processes” that enact the physiological impacts of stress even beyond the presence of stressors themselves.

To prepare the reader for the ensuing sections’ coverage of this shift in stress conceptualization, I will provide an example of a stressful situation for reference. When approaching the conceptualization of stress at the process-level, keep in mind this episode concerning our friend, Sally :

> *On Friday afternoon, Sally was working on the latest draft for a “revise-and-resubmit” request. Suddenly, Sally ’s advisor, Derek, stormed into the lab space, visibly upset. Brushing past her chair, Derek muttered, “Sally , see me in my office. Now!” Sally stood up, darting looks at her lab mates for any clues about what this meeting may concern. Everyone else appeared equally stunned. Sally hesitantly entered Derek’s office, where he chastised her for 10 minutes for failing to submit an IRB update. Although Sally wanted to speak up and remind Derek the IRB update had been entrusted to the lab’s post-doc, she held back hoping to finish the encounter quickly. Once Derek decided they were finished, Sally quietly left his office, hurriedly packed up her belongings, and headed home for the weekend. When Sally arrived to lab on the following Monday, she nervously peeked through the door for any signs of Derek. Although her thoughts mainly pertained to her tasks for the upcoming week, Sally still occasionally scanned the lab for indications of her fussy advisor and his current mood.*

Did Sally 's stressful experience begin upon Derek entering the lab and end once she headed home for the weekend? Within original stress conceptual frameworks, the "stress" in the presented scenario would have been defined as solely the angry encounter with Derek. However, developments in stress research have included a shift towards examining the ways in which the stress experience extends beyond the moment of a discrete adverse event. Specifically, worry and rumination are two cognitive processes that have been studied extensively for their role in activating stress responses beyond the presence of acute stressors.

Perseverative cognition as a stress process

Perseverative cognition (PC) refers to mentally creating or recreating a stressor by imagining symbolic representations of the event (Smyth et al., 2013). Two primary forms of this thought type are worry and rumination. The PC hypothesis from Brosschot et al. (2006) posits when people worry and ruminate in relation to stressful events, these mental acts of carrying a stressful event beyond the direct experiences of the episode, result in delayed cardiovascular recovery from the acute stressor (Verkuil et al., 2009).

While the experience of being berated by her advisor likely caused Sally 's heart rate to spike and her blood pressure to rise, how soon after leaving Derek's office was Sally able to regain her typical levels of physiological activation? Research into these PC thought processes demonstrates the length of Sally 's physiological recovery from this stressful event is linked to whether she engages in ruminating thoughts dwelling on the stressor afterwards. Worry, rumination, and stress-related thinking following adverse experiences have been linked to increased magnitude and duration of cortisol, blood pressure, and heart rate variability responses to stressors (O'Connor et al., 2012; Zoccola et al., 2011; Kiecolt-Glaser, 2020). Perseverative cognition extends an acute stressor into a chronic experience by causing continued appraisal and delaying eventual recovery from the initial stressors, while also generating additional stressors (Smyth et al., 2013; Verkuil et al., 2009). The body of work carried out regarding the PC hypothesis has powerfully demonstrated process-level aspects of the stress response that carry stress's effects beyond acute events and experiences.

Perseverative cognition has been studied as both a trait related to between-person differences and a state related to specific within-person changes following stressful events (Verkuil, 2009; Zoccola et al., 2011). Both lines of research have demonstrated that worrying and ruminating in relation to stressful events carries those stressful experiences out longer than had been conceptualized in earlier stress frameworks. The development of specific measures operationalizing PC behaviors, such as the Penn State Worry Questionnaire (PSWQ) and the Ruminative Response Scale (RRS), have been critical to facilitating research related to these domains (Meyer et al., 1990; Treynor et al., 2003). Through formal measurement of these PC behaviors, a growing body of research has addressed the extension of the stress response beyond acute experiences. Extending the stress response means lengthening the period of time when the associated 'wear and tear' is enacted on the body.

But regarding Sally 's episode with Derek, not every aspect of her experience has been covered in the stress-health research presented thus far. What about the scanning and watchful monitoring that Sally engaged in upon returning to the lab context where her threatening encounter with Derek occurred? If physiological arousal accompanies this vigilant behavior, then Sally 's stressful experience would be extended even further beyond the acute event and ruminating thoughts afterwards. While we have typically studied rumination over the last decade, we should now turn our attention to vigilance as a potentially critical component of the psychosocial stress process.

Vigilance: a potential behavioral variable in stress

Vigilance refers to watchful scanning and monitoring for contextual indicators of extant or potential threats to safety (Gump & Matthews, 1998; Smith et al., 2000). A more specific form of the behavior, social vigilance, refers to assessment of social environments for potential or ongoing interpersonal challenges or threats (Smith et al., 2000; Ruiz, et al., 2017). Social vigilance is a form of passive coping, where individuals deal with potential or imminent stressors by taking in sensory information from the environment for any updates on their personal safety.

Early research assessing the physiological responses occurring in individuals engaged in social vigilance indicates this behavior may represent an important pathway through which the stress response is extended beyond acute, discrete stress events. Gump and Matthews (1998) demonstrated vigilant anticipation of potential threats to one’s goals was accompanied by heightened CVR (e.g. BP, HR) during subsequent tasks. As covered earlier, research assessing the reactivity hypothesis has demonstrated that extended CVR is predictive of increased CVD risk. Therefore, a potential behavior contributing to longer periods of CVR following stressors is an important component of the stress response.

Smith et al. (2000) carried out a more direct analysis of whether individuals engaging in social vigilance to cope with potential threat exhibited different cardiovascular response profiles than participants not primed to engage in watchful social monitoring. Individuals in the socially vigilant condition demonstrated larger increases in blood pressure, but not accompanied by corresponding increases in HR (Smith et al., 2000). This profile of vascular resistance (e.g. BP increases unaccompanied by parallel HR increases) is indicative of pathophysiological mechanisms leading to some of the CVD outcomes described earlier (Ruiz et al., 2017; Kawai et al., 2013). While this watchful behavior may be adaptive in situations of immediate environmental threats, on a daily basis it may become its own health risk through chronic activation of the stress response and implications for health.

Furthermore, early research and theoretical foundations indicate individual and situational factors may moderate the extent to which one engages in vigilance behavior. Variations in key individual traits (e.g. neuroticism, anxiety, hostility) have been linked to differences in attentional bias towards potentially threatening information (Bishop et al., 2004; Koster et al., 2006; Lommen et al., 2010). Additionally, social roles (e.g. high vs. low social class; in- vs. out-group membership; professional occupation) are an established determinant of vigilant behavior in challenging or novel settings (Kraus et al., 2011; Dufford et al., 2019; Chen & Zhao, 2015; Damjanovic et al., 2014). Beyond individual-level determinants of vigilance, certain situational contexts elicit more awareness of threats than others. Contexts involving direct threats to one's goals, like walking home from work through a dangerous neighborhood, or challenges that may result in individual losses or gains, like approaching a supervisor whose judgement determines promotion or termination, demand more active awareness of environmental stimuli than benign situations. The extent to which a situation includes threats and/or challenges has been shown to determine levels of vigilant behavior (Bodala et al., 2016; Roelofs et al., 2007). The individual and situational factors summarized here represent modifiers of the potential pathophysiological pathway from social vigilance to CVD outcomes. Research assessing the physiological outcomes of social vigilance behavior should also address modifying factors that may amplify CVD risk associated with social vigilance by introducing an individual bias towards negative stimuli and heightened cardiovascular reactivity in socially threatening situations.

Thus, early work in the area of social vigilance suggests our friend Sally ’s physiological arousal related to her stressful encounter was not resolved upon leaving lab, nor even when she was able to eventually shut down any rumination related to the encounter. When Sally reentered the lab on Monday and scanned for potential signs of another interpersonal challenge related to her mentor, she was engaged in a behavior that likely elicited physiological arousal. Experiences of psychosocial stress extend beyond the discrete moment, influencing reflective thoughts and guiding anticipatory behavior; which can all enact physiological responses that accumulate in additional allostatic load.

Next directions in social vigilance work: measurement

Although this idea of social vigilance for threats has been mentioned in previous conceptualizations of stress processes (Brosschot et al., 2006; Cundiff & Smith, 2017; Brosschot et al., 2018), there have been no formal attempts to operationalize or measure this behavior. Given the early work demonstrating the CVR profile accompanying social vigilance, this behavior may play a critical role in understanding the pathway from chronic stress exposure to CVD outcomes. If social vigilance behavior is preventing individuals from recovering fully from stress and even contributing to some people engaging in anticipatory stress responses, then failing to measure this behavior means a major biobehavioral process is absent from our assessment of mechanisms leading to the development of CVD (Heponiemi et al., 2007).

An important next step for research in this area is the formal operationalization of social vigilance through a measure that can be administered in multiple contexts and settings, for both state and trait assessment of this variable. Ruiz et al. (2017) piloted an initial 10-item Social Vigilance Questionnaire (SVQ) survey measure assessing self-reported frequency of engagement in social vigilance behavior. However, the SVQ (Ruiz et al., 2017) remains to be validated by objective assessment of vigilant behavior, such as through within-individual comparison of SVQ responses against eye-tracking-based measurement of visual attention to threatening experimental stimuli.

Additionally, experimental assessments of social vigilance behavior should examine this biobehavioral variable across varying types of stressful contexts. As stated previously, predominant stress research has focused on variations in individuals' experiences of stress across appraisals of stressful contexts, such as challenge versus threat scenarios (Folkman, 1984; Berjot & Gillet, 2011). To expand upon foundational stress appraisal research and account for real-world variation in types of stressful contexts that individuals encounter, future work assessing social vigilance must examine for potential interactions between vigilance behavior and type of stress context on stress-related outcomes of interest.

The absences of these psychometric and experimental assessments of vigilance behavior across types of stressful contexts represent important gaps in the present understanding of social vigilance as a trait and state biobehavioral variable.

Summary

The continual nature of psychosocial stressors and accompanying physiological responses carries significant implications for the onset of cardiovascular disease. The emerging evidence presented above suggests vigilance for social threats may represent an important behavioral pathway linking stress exposure to CVD outcomes. Social vigilance behavior can take place daily and be due to a range of threatening factors, from potential transmission of COVID-19 to interpersonal conflicts. Regardless of the source contributing to vigilant behavior, the cumulative physiological burden of that repeated activation may be a pathway to chronic disease. Evident gaps in the understanding of pathophysiological mechanisms linking psychosocial stress to cardiovascular disease outcomes provide justification for a formal experimental examination of social vigilance behavior as a determinant of cardiovascular reactivity.

Present Study

As part of a larger study examining social vigilance as a biobehavioral variable that may influence cardiovascular reactivity (CVR) across individuals and contexts, the present study used a videogame task to manipulate situational threat level, and measured subsequent cardiovascular stress responses. As stated above, magnitude of CVR (e.g. stressor-induced increases in HR and BP) is understood to influence CVD outcomes (Lovallo, 2015; Treiber et al., 2003). Upstream components of CVR, such as cardiac pre-ejection period (PEP) and respiratory sinus arrythmia (RSA), are also important variables to capture when examining contributions of sympathetic and parasympathetic stress responses, respectively, to CVD outcomes (Thayer et al., 2010; Kelsey, 2012; Masi et al., 2007). Given the early evidence suggesting social vigilance may influence CVR and CVR's known associations with CVD outcomes, the present study aimed to:

1. Assess whether nature of stress (challenging, threatening, or neutral) in a task condition evoked greater cardiovascular reactivity during and after the task.
2. Assess whether individual differences in trait-level social vigilance moderated cardiovascular response during experimental tasks.

Hypotheses

Hypothesis 1. We predicted higher stress games would evoke larger changes from baseline HR and BP.

Hypothesis 2. We predicted higher stress games would evoke greater decreases in positive affect and larger increases in negative affect, relative to baseline affect levels.

Hypothesis 3. We predicted individuals higher in trait-level social vigilance would display greater cardiovascular reactivity (HR and BP) during stressful gameplay, particularly in the threat condition.

Method

All data collection was conducted as in-person study in the year 2019, prior to the COVID-19 pandemic.

Participants

Participants were an undergraduate sample of 135 young adults (66 male, 69 female) ages 18 to 25 years (*M*= 18.81, *SD*= 3.51) recruited from the psychology research participant pool at a large university in the Southwestern U.S. Inclusion criteria were 1) fluent in English, 2) 18 years of age or older to consent, and 3) Undergraduate status. Exclusion criteria included 1) presence of strabismus (eye-misalignment), which interferes with eye-tracking calibration and gaze tracking (which was relevant to data collected within the larger study), and 2) baseline blood pressure values beyond the American Heart Association (2016) guidelines for pre-hypertension (systolic BP > 139mmHg or diastolic BP > 90mmg). The sample included 48.1% non-Hispanic (NH) Whites, 31.9% Hispanic/Latinos, 12.6% NH Asians, and 7.4% individuals identified as NH Other Race.

Measures

Three types of measures were collected: (1) psychosocial survey data, (2) vital biological information, and (3) stationary in-lab physiological data.

Demographics and vitals

Demographic data including age, race, ethnicity, and socio-economic status information were assessed through self-report. This initial demographics survey included a prior gameplay experience questionnaire assessing participants' self-reported familiarity with videogames like those used in the study protocol. These responses were coded to create a videogame exposure variable, which provided a composite measure of the frequency and duration of participants' self-reported videogame-play habits. Following the in-lab informed consent procedure described below, a trained experimenter collected participants' height and weight measurements. These variables were used as covariates in models testing the present study's aims.

Mood and affect

To assess participants' self-reported mood throughout the experimental session, an 18-item version of the Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988) was administered at four time-points: pre-baseline task, post-baseline task, post-gameplay, and post-recovery. Upon each administration, the survey stem instructed participants to report their current affect "in the present moment" by indicating the extent to which they were experiencing each item on a list of affective states through Likert-type ratings (0= 'Very slightly or not at all'; 5='Extremely').

Trait social vigilance

To assess participants' trait-level social vigilance behaviors, the Social Vigilance Questionnaire (SVQ; Ruiz et al., 2017) was administered during the recruitment survey and the initial psychosocial questionnaires portion of the experimental appointment. The SVQ is a 10-item, three-factor self-report measure which assesses the frequency at which an individual engages in stress-related vigilance or monitoring of the social environment. Respondents are presented with the question stem, 'In social situations …' and subsequent items to be rated on a 4-point Likert scale (0= 'almost never'; 4= 'almost always').

Cardiovascular reactivity

Using a GE Carescape V100 Blood Pressure monitor (2016), mean systolic (SBP) and diastolic (DBP) blood pressure readings were calculated at repeated timepoints outlined in the study procedure below. Mean arterial pressure (MAP) and heart rate (HR) were also derived from these episodic readings, which occurred at the timepoints outlined below. Participants were fitted with an automated blood pressure cuff attached to the upper portion of their non-dominant arm. Readings were taken every 60 s during the 10-min baseline period, and the last three readings were averaged to form baseline values. Readings were taken 10 times during the gameplay task, occurring every three minutes (180 s intervals) of gameplay. An average was also derived from the five readings during the 10-min recovery period, which were measured every two minutes (120 s intervals).

Impedance cardiography data were also collected via electrocardiogram (ECG), but these ECG-collected data were not examined within the scope of the present study. The ECG output from a MindWare Technologies 2000 D Impedance Cardiograph (2016) was used to obtain continuous measures of HR, high-frequency HR variability (hfHRV), pre-ejection period (PEP), and respiratory sinus arrythmia (RSA). Participants were fitted with seven standard ECG sensors on the upper torso in the tetra polar configuration (Thayer et al., 2010), in addition to two sensors on the non-dominant hand to assess skin conductance. Raw ECG and skin conductance were collected continuously throughout the baseline, experimental task, and recovery periods at 1, 000 Hz. HR was defined as the number of cardiac cycles per minute. HRV Analysis Software, Version 5.2 (MindWare Technologies, Gahanna, OH) was used to derive hfHRV (in ms^2/Hz) from the ECG data, which provides a measure of RSA reflecting level of parasympathetic activation (Cundiff et al., 2016; Billman, 2011). PEP was derived from the interval (milliseconds) between the Q-point on the ECG output and the B-point of the impedance signal (dZ/dt), which provides a measure of sympathetic activation (Cundiff et al., 2016; Mindware, 2016).

Procedures

This study was a 2 Sex (male vs. female) x 3 Gameplay stress condition (threat vs. challenge vs. neutral) factorial design. Figure one provides a timeline of the study procedures. Laboratory sessions were conducted in a room with a computer-monitor set-up for single participant appointments, with physiological recording equipment located in an adjoining room. Upon participant arrival, a trained experimenter facilitated a standard study introduction and initial informed consent process. Participants then completed the demographics, psychosocial (e.g. mood/affect, SV), and vital signs measurements. Participants were then fitted with the ECG sensors and BP arm-cuff as described above. All participants underwent an initial 10-min resting baseline period, 30-min experimental task period (stress manipulation), and a 10-min recovery period prior to debriefing.

Baseline period

The initial 10-min baseline period used a minimally involving task (i.e., “vanilla baseline”; Jennings et al., 1992), which required participants to indicate their preference between a pair of outdoor photographs (e.g. national parks) presented at a rate of one photo pair per minute. After baseline, participants completed a second administration of the PANAS as a state affect measure.

Gameplay stress manipulation

Participants were randomly assigned to the threat, challenge, or neutral conditions based on the 2 x 3 experimental design balanced for gender and video game type. Following the baseline period, participants were introduced to the vigilance gameplay task, which was intended to have participants engage in directed gameplay oriented towards an overall objective that varied by task condition. Participants' condition assignment for the vigilance task determined which game they would play for 30-min on a Microsoft Xbox One S (2016) gaming console. Participants in the threat gameplay condition played "Call of Duty Black Ops III: Zombies Mode" (Treyarch/Activision), which presented participants with a simulated hostile situation that they could not overcome, but instead could only play through until the game mode became too difficult. ECG was collected continuously during this task, as well as HR and BP samples occurring at 180 s intervals throughout gameplay. After the 30-min experimental gameplay portion, participants completed a third administration of the PANAS as a post-task state affect questionnaire.

Those assigned to the challenge gameplay condition followed a nearly identical procedure (i.e., introduction, 30 min gameplay with physiological recording, post-task state affect) but played a single player level of a videogame titled "Call of Duty: WWII" (SledgeHammer Games/Activision), which orients players in a simulated hostile situation which they have the means to overcome through normal gameplay.

Lastly, the described procedure (i.e., introduction, 30 min gameplay with physiological recording, post-task state affect) was adapted for participants in the neutral gameplay condition by requiring them to play a simple shooting range simulation game titled "Hunting Simulator" (Maximum Games), which presents players with a situation that is not hostile, but also has no definitive end that can be achieved through normal gameplay.

Recovery period

Following the gameplay stress manipulation, all participants then completed a 10-min recovery period, during which HR and BP samples were collected. Participants then reported affect through a final PANAS administration, prior to being debriefed and given partial course credit for their participation.

Overview of Analyses

Data Preparation

The central cardiovascular variables were derived from the oscillometric blood pressure readings (GE Carescape, 2016). Episodic data collected via the blood pressure monitor provided three measures of blood pressure (i.e., SBP, DBP, MAP) and one measure of heart rate frequency (i.e., HR). Affective changes throughout the experimental period were be measured through participants' responses to the four PANAS administrations.

Baseline values

As described above, within-person baseline values for each cardiovascular measure were derived from the average of the final three readings that occurred every minute throughout the vanilla baseline task. Specifically, baseline SBP, DBP, MAP, and HR were derived from the final three baseline readings. Baseline affect was measured from participants' responses to the PANAS items administered immediately after the baseline task.

Change scores

Consistent with prior recommendations (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), change scores (i.e., experimental task minus baseline, recovery period minus baseline) were calculated for physiological responses at each minute of the experimental task and recovery periods. For each physiological variable, the sum of all physiological change scores for a participant during an experimental phase (gameplay, recovery) was divided by the total number of change scores recorded for that individual to calculate within-person average change for each physiological variable (BP, HR) during the experimental task and during post-task recovery.

Change scores for affective responses were calculated following the experimental task (i.e., post-experimental task minus post-baseline) and recovery period (i.e., post-recovery period minus post-baseline).

Univariate Analyses

To account for known effects of baseline levels on subsequent physiological changes (Benjamin, 1967), baseline values were included as a covariate in mixed analyses of covariance (ANCOVAs). BMI was also be included as a covariate. Two-way ANCOVAs were conducted for all cardiovascular and affective change variables scores to assess average differences between groups during gameplay and during recovery. During the experimental task period, we computed two-way mixed ANCOVAs (i.e., Sex [male vs. female] x Gameplay stress condition [threat vs. challenge vs. neutral]) for each cardiovascular variable's change score. A two-way univariate ANCOVA (i.e., Sex x Gameplay stress condition) was performed on the affective change scores from post-baseline to post-experimental task. During the recovery period, we computed additional two-way mixed ANCOVAs (i.e., Sex x Gameplay threat condition) for each cardiovascular variable's change scores. Again, a two-way univariate ANCOVA (i.e., Sex x Gameplay threat condition) was performed on affective change from post-baseline to post-recovery period.

Significant interactions were further analyzed using mean comparisons (Wickens & Keppel, 2004).

Results

Overall, there were minimal differences in demographic characteristics across male and female participants (see table 1). Notably, male participants reported significantly higher levels of videogame exposure than female participants. To account for potential impacts of participants' differing videogaming habits on their performance on the experimental tasks, videogame exposure was included as a covariate in related analyses. In addition, a sex difference in age emerged with females being younger than males. There was also a sex difference in participants' racial/ethnic identities.

Overall, few differences in biological, physiological, and affective variables were observed by sex at baseline (see table 2). A difference was observed for SBP and MAP where males displayed higher average resting values compared to females. No other differences in resting biology, physiology, or affect were found.

Similarly, few differences in biological, physiological, and affective variables were observed by experimental condition at baseline (see table 3). BMI was greater among individuals assigned to the threat condition than for challenge or neutral conditions. No other differences at baseline in physiology or affect emerged across the experimental conditions.

Manipulation Checks

Mean values of physiological and affective variables during and following each experimental phase were compared to discern whether the task evoked expected effects. As shown in Table 4 and Figure 2, SBP, DBP, MAP, and HR all increased significantly from baseline to task and regressed during recovery. Post-hoc pairwise comparisons of estimated means with Bonferroni adjustment for multiple comparisons confirmed HR returned to baseline completely, whereas SBP, DBP, and MAP only showed partial recovery towards baseline. Overall, the experimental task evoked responses in the predicted directions on all physiological indices.

Similarly, within-person affect significantly differed following each experimental phase. As shown in Table 5 and Figure 3, positive affect significantly decreased from post-baseline to post-task and increased post-recovery, whereas negative affect significantly increased from post-baseline to post-task and regressed post-recovery.

Overall, the experimental task appeared to evoke the expected effects on the physiological and affective variables of interest.

Task Effects

Stress condition effects on BP and HR during experimental task

A series of analysis of covariance models (ANCOVAs) were used to test whether stress conditions, as operationalized by game type, evoked expected changes in BP and HR after controlling for relevant sociodemographic and matched mean baseline physiological variables. We predicted higher stress (challenge, threat) games would evoke larger changes from baseline in BP and HR throughout gameplay. As shown in Table 6, main effects of condition were observed for SBP, DBP, MAP, and HR, all F's $(1, 107) > 8.60$, p's $<.001$. Specifically, a priori contrasts illustrated threat differed from neutral for all BP and HR variables, and challenge differed from neutral for all BP (see Tables 7-10 and Figure 4). No other main effects or interactions were observed.

Stress condition effects on affect following experimental task

ANCOVAs were also conducted to assess for changes in positive and negative affect across stress conditions after controlling for baseline affect levels and videogame exposure. We predicted higher stress games would evoke greater decreases in positive affect and larger increases in negative affect, relative to baseline. As shown in Table 11, there was a main effect of sex on positive affect, as well as a significant interaction between sex and stress condition. Specifically, a priori contrasts illustrated females demonstrated greater decrease in positive affect relative to males, and the greatest decreases in positive affect across sexes and stress conditions were observed among females in the challenge condition (see Table 12 and Figures 5-6). No other main effects were observed.

Recovery Effects

Stress condition effects on BP and HR during recovery towards baseline

An additional series of ANCOVAs were conducted to assess whether stress conditions differentially impacted BP and HR recovery. Change scores (relative to baseline) for each variable were evaluated after controlling for relevant sociodemographic and matched mean baseline physiological variables. We predicted BP and HR recovery would be less complete among individuals assigned to higher stress games relative to neutral condition participants. No condition effects were observed for HR suggesting all conditions were equally recovered relative to baseline, F (1, 106) = .683, p = ns). However, condition effects were observed for SBP, DBP, and MAP (see Table 13), all F's (1, 106) > 4.50, p's <.05. Consistent with expectations, participants in the threat condition showed less recovery to baseline relative to participants in the neutral condition (see Figure 7). No other main effects or interactions were observed.

Stress condition effects on affect following recovery towards baseline

ANCOVAs were also conducted to assess for condition effects on post-recovery changes in positive and negative affect. Change scores (relative to post-baseline) for each variable were evaluated after controlling for matched baseline affect and videogame exposure. We predicted recovery towards baseline levels of positive and negative affect would be less complete among individuals assigned to higher stress games relative to neutral condition participants. No main effects nor interactions were observed, all F's (1, 123) < 2.463, p's = ns, which indicated all conditions appeared equally recovered towards baseline affect.

Social Vigilance Effects

Social vigilance trait effects on BP and HR during experimental task

ANCOVAs were conducted to test whether trait-level social vigilance, a continuous measure assessed via self-report, was associated with changes in BP and HR after controlling for relevant sociodemographic and matched mean baseline physiological variables. We predicted individuals higher in trait-level social vigilance would display greater changes from baseline in BP and HR throughout gameplay, and the greatest changes would be among highly vigilant individuals assigned to higher stress conditions. No main effects of vigilance nor interactions between vigilance and condition were observed, all F's $(1, 106) < 2.60$, p's = ns, which indicated individuals reporting different levels of social vigilance did not display different changes in BP and HR during gameplay.

Social vigilance effects on affect following experimental task

ANCOVAs were also conducted to assess whether trait-level social vigilance was associated with patterns of changes in positive and negative affect after controlling for baseline affect levels and videogame exposure. No main effects nor interactions were observed, all F's $(1, 117) < 0.60$, p's = ns, which indicated individuals reporting different levels of social vigilance did not display different changes in affect following the experimental task. ***Social vigilance trait effects on BP and HR during recovery towards baseline***

An additional series of ANCOVAs were also used to assess whether trait-level social vigilance was associated with patterns of BP and HR recovery towards baseline after controlling for relevant sociodemographic and matched mean baseline physiological variables. We predicted recovery towards baseline BP and HR would be less complete among individuals who reported higher levels of social vigilance, and such recovery would be least complete among high vigilance individuals assigned to higher stress conditions. No main effects of vigilance were observed for DBP or HR, all F's (1, 119) < 2.01, p's = ns, which suggested individuals high and low in social vigilance were equally recovered towards baseline. However, vigilance effects were observed for SBP and MAP (see Table 14), all F's (1, 119) > 7.00, p's < .01. In contrast to expectations, higher total vigilance was associated with greater return to baseline for both SBP (B = -0.20, 95% CI = -0.34 to –0.07, p = .003) and MAP (B = -0.15, 95% CI = -0.26 to –0.04, p = .008) (see Figures 8-9).

Social vigilance effects on affect following recovery towards baseline

ANCOVAs were also conducted to assess whether trait-level social vigilance was associated with patterns of changes in positive and negative affect after controlling for baseline affect levels and videogame exposure. No main effects nor interactions were observed, all F's (1, 117) < 0.45, p's = ns, which indicated individuals reporting different levels of social vigilance appeared equally recovered towards baseline affect.

Discussion

The aims of this study were to test whether varying conditions of stress, including both challenge and threat, impacted acute cardiovascular reactivity and recovery. Using three videogame scenarios, we found that higher stress games produced significantly more CVR than a neutral game. In particular, the high threat game (Call of Duty World War Z) evoked significantly greater change across all physiological parameters. In addition, participants in the high threat condition also demonstrated the least overall recovery to baseline blood pressure (BP) following gameplay. In contrast to expectations, these effects were only modestly qualified by individual differences in social vigilance. No effects of vigilance disposition were observed during gameplay and modest effects of higher vigilance were associated with better physiological recovery, in contrast to expectations. Overall, there was a partial impact of condition on affect such that females assigned to the challenge condition demonstrated the greatest decrease in positive affect post-gameplay across sexes and conditions.

The current results contribute to our understanding of threat-related social vigilance and risk for CVD. Specifically, the current study found higher threat contexts requiring greater monitoring and attention evoked the largest changes in BP and heart rate (HR). This CVR pattern is broadly hypothesized to be a critical pathway linking psychosocial stress to greater disease risk (Krantz & McCeney, 2002; Lovallo, 2015). These results are consistent with previous findings that have indicated individuals assigned to contexts evoking watchful monitoring of potential threats display heightened BP and HR in response to experimental tasks compared to controls (Gump & Matthews, 1998; Smith et al., 2000; Ewart et al., 2004). Together, these findings help to inform our understanding of how higher threat social situations may connote CVD risk through their pull for greater preparatory monitoring and the acute cardiovascular responses corresponding to that behavior.

The current study also contributes to a methodological advancement in stress-CVR research. Previous research has predominantly examined CV responses to discrete events such as conflict, public speaking, or non-social tasks (Hilmert & Kvasnicka, 2010; Gerin et al., 1999). As noted, these studies are important but may represent the impact of only a rare endpoint to more common monitoring aspects of the stress experience in daily life. Specifically, the current study presented the first assessment of CVR in response to a sustained attentional demand task conceptualized as ongoing vigilance. Although previous studies have examined CVR during stress tasks following induction of vigilance states (Gump & Matthews, 1998; Smith et al., 2000), there has been limited examination of CV reactivity to and recovery from lab-based manipulation of active monitoring for environmental threats. Further, this study included novel assessment of individual differences in social vigilance within contexts evoking different degrees of situational monitoring, and examined for potential interactions. Although the anticipated effects of individual differences in vigilance were not observed, this inclusion of social vigilance disposition presented a methodological advancement.

In a comprehensive review of stress literature, Epel et al. (2018) proposed a unified transdisciplinary model of stress that emphasized the importance of conceptualizing stress processes across multiple levels, including unconscious cognitive processes involved in the psychological responses of acute stress. Within this unified model of “interactive” stress processes contributing to biological aging and early disease, psychological (e.g., unconscious vigilance, coping) and physiological responses (e.g., CVR) occur during the acute stress of daily life stressors that occur at frequencies determined by contextual factors (e.g., individual differences, environment) (Epel et al., 2018). The current examination of vigilance and associated CVR aligns with Epel et al.’s (2018) conceptualization of acute stress processes relevant to broader stress-disease pathways, in addition to supporting the GUTS and Social Safety Theory frameworks positing appraisal of environmental threats presents an unconscious cognitive process critically related to activation of physiological stress responses involved in pathogenesis (Brosscot et al., 2018; Slavich, 2020). Therefore, the current results linking environmental pull for preparatory monitoring to exaggerated CVR align with emerging contemporary theories of stress underscoring the importance of anticipatory cognitive appraisal, coping, and associated physiological stress responses occurring throughout daily life.

Environmental differences in contextual threat levels are not limited to the laboratory, and current results yield important understanding for broader work on vigilance as a stress concept. For example, socioeconomic status, household functioning, and presence of discriminatory prejudice are robust environmental determinants of physical and emotional wellbeing that have been shown to influence individuals' perceptions of situational threat (Chen et al., 2009; Repetti et al., 2002; Schmader et al., 2008). Extent of situational threat has been previously shown to determine vigilant behavior assessed via eye-tracking (Bodala et al., 2016) and task-relevant attentional bias scores (Roelofs et al., 2007), and the present results suggest individuals navigating daily environments requiring greater monitoring for situational threat regularly encounter contexts that evoke greater BP and HR responses.

Although the current examination of individual differences in social vigilance did not produce the expected effects of heightened CV reactivity and attenuated CV recovery, these findings may suggest vigilance is an especially relevant cognitive process to conceptualizing contextual determinants of stress-related health outcomes. Similar to previous findings wherein harassment-induction did not evoke expected CVR changes among individuals high in hostility and anger (Vella & Friedman, 2009; Jorgenson & Kolodziej, 2007), it may be the case that threatening contexts meet the 'situational expectations' of highly vigilant individuals and their superior post-task recovery in the current study may reflect intrinsic preparation for encountering situational stress. Future research in this area should include additional examinations of individual vigilance disposition to better tease apart the interactions between individual expectancies and situational stressors.

Importantly, vigilance is a basic behavior broadly relevant to examinations of cognitive processes involved in coping throughout adverse experiences, such as physical health outcomes of trauma, psychobiological effects of discrimination, and contemporary examinations of environmental uncertainty related to health threats. For example, individuals recovering from post-traumatic stress disorder (PTSD) have been shown to display heightened conscious and subconscious perception of environmental threat stimuli, and face heightened risk of CVD and reduced cardiac vagal control (Lanius et al., 2017; Thurston et al., 2020). Heightened vigilance for environmental threats among individuals recovering from traumatic experiences may present a biobehavioral variable influencing autonomic functioning and health outcomes. Additionally, previous work examining the impacts of stereotype threat has demonstrated individuals navigating contexts involving discrimination against their social identities demonstrate poorer task performance from reduced executive resources (Johns et al., 2010) and display heightened CVR responses to experimental stressors (Merritt et al., 2006). Theoretical models of stereotype threat effects have proposed vigilant monitoring for environmental threats may present a key cognitive process involved in the depletion of task-relevant psychological resources and engagement of stress arousal systems above and beyond non-threatened counterparts (Schmader et al., 2008). Further, emerging work has begun to examine mechanisms contributing to heightened stress, anxiety, and depression observed throughout the COVID-19 pandemic (Varma et al., 2021; Lakhan et al., 2020). Given the omnipresent environmental threat of viral contagion via airborne transmission and uncertainty around likelihood of health-related threats to safety, the present study supports vigilance as an importance cognitive process to consider when examining the psychological and physiological impacts of living through the COVID-19 pandemic. In sum, controlled laboratory examinations of vigilance across stressful situations and associated CV response patterns, like the current study, contribute basic scientific findings with implications across a variety of domains related to mental health, social identity, and environmental safety.

Limitations

Results and conclusions of these data should be interpreted in light of study limitations. Given this study utilized a convenience sample from a university subject pool, these results may not be generalizable beyond college-enrolled young adults. Future work in this area should examine these mechanisms and outcomes in samples representing additional ages, educational histories, and psychosocial backgrounds with threats and danger. The present study contributed a novel operationalization of challenging versus threatening contexts, however measurement of participants' subjective engagement with the challenging versus threatening conditions was limited. Participants assigned to the high stress conditions did not report whether they perceived themselves as possessing sufficient resources within the gameplay context to meet the task demands, therefore participants' appraisals of the different types of stress contexts were unclear. Additionally, the paradigm used in the current study, which required vigilant monitoring of targets in a gameplay environment, did not include explicit observation of other people. Therefore, the extent to which the experimental task evoked social vigilance is unclear, and future social vigilance research should build upon the present findings by assessing monitoring of other people. Further, the present experimental task did not exclusively evoke passive vigilance for environmental threats, as participants had the opportunity to respond through gameplay and therefore engage in a degree of active coping with ongoing stress. However, gameplay responses from participants in the threat and challenge conditions took place within environments dynamically pulling for vigilance, and thus allowed for assessment of CVR among individuals engaged in varying degrees of passive coping during stress. Relatedly, this was an in-laboratory, experimental manipulation and it is unclear whether this manipulation adequately captured differences of environmental threat and challenge present in real-world, daily experiences. Finally, the present study's assessment of physiological outcomes was limited to autonomic stress responses (i.e., heart rate, blood pressure), and it is unknown whether the acute stress processes of interest impacted other physiological systems involved in stress responses. Participants across the study conditions and trait levels of social vigilance may have experienced differential neuroendocrine or immunological responses, which were not captured in the present study.

Conclusions

The present study contributes to our understanding of associations between situational stress, vigilance, and acute cardiovascular reactivity and recovery. Results reveal that ongoing monitoring of environmental threats evokes heightened cardiovascular responses which may connote CVD risk to the degree that such behavioral patterns are chronically experienced. Moreover, the focus on vigilance as a unique component of the stress experience potentially improves the ecological validity of stress concept with implications for understanding how situational awareness to a broad range of social environments may connote risk.

Tables

Table 1

Demographic characteristics of overall sample and stratified by sex

Characteristic	Overall		Male		Female		X^2 (*df*)
	n	%	*n*	%	*n*	%	
Race/Ethnicity							10.90 (3)*
Hispanic	43	31.9	25	37.9	18	26.1	
Non-Hispanic (NH) Asian	17	12.6	13	19.7	4	5.8	
NH White	65	48.1	25	37.9	40	58.0	
NH Other	10	7.4	3	4.5	7	10.1	
Years in college							3.89 (4)
1	96	71.1	45	68.2	51	73.9	
2	21	15.6	9	13.6	12	17.4	
3+	18	13.3	12	18.2	6	8.7	
Videogame exposure							29.24 (2)***
Low	80	59.3	24	36.9	56	80.0	
Moderate	39	28.9	26	40.0	13	18.6	
High	16	11.9	15	23.1	1	1.4	

Note. N = 135 (n = 66 for Male; n = 69 for Female). Overall participants were on average 19.26 years old (*SD* = 2.65), and comparison using Student's unpaired t-test revealed a significant mean difference in age ($t[133] = 5.32$, $p < .05$) between males (M = 19.67, *SD* = 3.56) and females (M = 18.87, *SD* = 1.19). *df* = degrees of freedom; Videogame exposure = a composite measure of the frequency and duration of participants' self-reported videogame-play habits.

*$p<.05$; ***$p<.001$

Table 2.

Baseline levels of biological, physiological, and affect variables for overall sample and by sex

Variable	Overall		Male		Female		*t* (120)
	M	*SD*	*M*	*SD*	*M*	*SD*	
BMI	23.92	4.76	24.25	5.11	23.59	4.41	0.80
SBP	111.04	9.30	115.10	8.16	107.24	8.72	5.13***
DBP	67.34	7.03	67.50	6.90	67.19	7.20	0.24
MAP	83.71	6.79	85.44	6.36	82.10	6.83	2.79*
HR	77.04	12.07	76.62	12.58	77.43	11.66	-0.37
Positive Affect	20.72	4.61	21.22	4.75	20.28	4.48	1.14
Negative Affect	15.29	4.07	15.17	3.69	15.40	4.40	-0.33

Note. BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate. Group differences across sex were assessed using Student's unpaired *t*-test.

*p<.05; ***p<.001

Table 3.

Baseline levels of biological, physiological, and affect variables by stress condition

Variable	Challenge		Threat		Neutral		*F* (2, 119)
	M	*SD*	*M*	*SD*	*M*	*SD*	
BMI	23.92	4.76	24.25	5.15	23.61	4.38	3.88*
SBP	111.20	9.04	110.94	8.54	110.98	10.41	0.01
DBP	67.87	7.26	66.68	6.84	67.52	7.13	0.30
MAP	84.09	7.46	83.25	6.54	83.83	6.54	0.16
HR	77.29	12.16	78.11	11.60	75.74	12.61	0.41
Positive Affect	21.00	4.07	20.66	4.39	20.52	5.38	0.12
Negative Affect	15.61	4.24	15.14	4.21	15.14	3.82	0.18

Note. BMI = body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate. Group differences across the challenge ($n = 38$), threat ($n = 42$), and neutral ($n = 42$) conditions were assessed using one-way analysis of variance testing.

*p<.05

Table 4.

Within-person mean levels of physiological variables by experimental phase

Variable	Baseline		Experimental Task		Recovery		F (df_b, df_w)
	M	*SD*	*M*	*SD*	*M*	*SD*	
SBP	111.30	8.80	118.85	10.54	114.42	9.36	88.92 (1.72, 119)***
DBP	67.34	6.85	73.31	8.43	70.45	7.99	76.91 (1.86, 119)***
MAP	83.79	6.88	89.91	8.80	86.79	7.68	85.03 (1.70, 119)***
HR	76.65	11.75	78.93	11.23	76.77	11.18	10.89 (2, 119)***

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; df_b = degrees of freedom between subjects; df_w = degrees of freedom within subjects. Omnibus differences across experimental phases (e.g., baseline, experimental task, and recovery) were assessed using one-way analysis of variance testing.

[a]For variables where the assumption of homogeneity-of-variance-of-differences was violated (e.g., SBP, DBP, MAP), the reported degrees of freedom between subjects (df_b) have been adjusted according to the Greenhouse-Geisser correction.

[b]Following omnibus testing, post-hoc pairwise comparisons of estimated means for each variable were conducted with Bonferroni adjustment for multiple comparisons. Within physiological variables, all means significantly differed across experimental phases, with the exception of baseline and recovery heart rate.

***$p<.001$

Table 5.

Within-person levels of affective variables following each experimental phase

Variable	Post-Baseline		Post-Experimental Task		Post-Recovery		*F* (2, 121)
	M	*SD*	*M*	*SD*	*M*	*SD*	
Positive Affect	20.65	4.58	16.87	5.15	18.17	4.73	56.22***
Negative Affect	15.36	4.15	19.26	7.37	16.93	6.00	25.48***

Note. Omnibus differences in self-reported affect following each experimental phase (e.g., baseline, experimental task, and recovery) were assessed using one-way analysis of variance testing.

[a]Following omnibus testing, post-hoc pairwise comparisons of estimated means for each variable were conducted with Bonferroni adjustment for multiple comparisons. Within affective variables, all means significantly differed.

***$p<.001$

Table 6.

Analyses of Covariance in Average Change (Baseline to Gameplay) for Physiological Variables

Effect[a]	Δ SBP			Δ DBP			Δ MAP			Δ HR		
	F ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2
Stress condition	13.58***	2, 107	.202	16.15***	2, 107	.232	18.35***	2, 107	.255	8.69***	2, 107	.140
Sex	3.32	1, 107	.030	1.52	1, 107	.014	2.50	1, 107	.023	1.42	1, 107	.013
Stress condition x Sex	0.49	2, 107	.009	1.02	2, 107	.019	0.71	2, 107	.013	1.05	2, 107	.019

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; df_b = degrees of freedom between subjects; df_w = degrees of freedom within subjects.

[a]The p values for reported main effects were adjusted for age, race/ethnicity, body mass index (BMI), matched baseline physiological mean, and videogame exposure (a composite measure of the frequency and duration of participants' self-reported videogame-play habits) as covariates.

***p<.001

Table 7.

Comparisons of Average SBP Change (Baseline to Gameplay) across Stress Conditions

Stress Condition	*n*	*M*	95% CI for *M*		*SE*	Significance (*p*) of pairwise comparisons[a]	
			LL	*UL*		Threat	Challenge
Threat	41	11.16	8.98	13.33	1.10		
Challenge	38	8.14	5.91	10.37	1.13	.118	
Neutral	41	3.70	1.46	5.93	1.13	<.001	.011

Note. SBP = systolic blood pressure; CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

[a]Mean differences were compared across conditions with Bonferroni-adjustment for multiple comparisons.

Table 8.

Comparisons of Average DBP Change (Baseline to Gameplay) across Stress Conditions

Stress Condition	*n*	*M*	95% CI for *M*		*SE*	Significance (*p*) of pairwise comparisons[a]	
			LL	*UL*		Threat	Challenge
Threat	41	8.90	7.20	10.60	.86		
Challenge	38	7.51	5.77	9.26	.88	.658	
Neutral	41	2.77	1.03	4.52	.88	<.001	<.001

Note. DBP = diastolic blood pressure; CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

[a]Mean differences were compared across conditions with Bonferroni-adjustment for multiple comparisons.

Table 9.

Comparisons of Average MAP Change (Baseline to Gameplay) across Stress Conditions

Stress Condition	*n*	*M*	95% CI for *M*		*SE*	Significance (*p*) of pairwise comparisons[a]	
			LL	*UL*		Threat	Challenge
Threat	41	9.54	7.82	11.26	.87		
Challenge	38	7.20	5.44	8.97	.89	.127	
Neutral	41	2.75	0.99	4.52	.89	<.001	<.001

Note. MAP = mean arterial pressure; CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

[a]Mean differences were compared across conditions with Bonferroni-adjustment for multiple comparisons.

Table 10.

Comparisons of Average HR Change (Baseline to Gameplay) across Stress Conditions

Stress Condition	*n*	*M*	95% CI for *M*		*SE*	Significance (*p*) of pairwise comparisons[a]	
			LL	*UL*		Threat	Challenge
Threat	41	4.64	2.86	6.41	.89		
Challenge	38	2.64	0.82	4.46	.92	.282	
Neutral	41	-0.24	-2.07	1.59	.92	<.001	.058

Note. HR = heart rate; CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

[a] Mean differences were compared across conditions with Bonferroni-adjustment for multiple comparisons.

Table 11.

Analysis of Covariance in Positive Affect Change (Post-Baseline to Post-Gameplay)

Effect	*F* ratio	df_b, df_w	η_p^2	Overall Model Fit
Intercept	3.69	1, 116	.031	
Post-baseline PA	17.99***	1, 116	.134	
Videogame exposure	0.40	1, 116	.003	
Stress condition	2.35	2, 116	.039	
Sex	7.88**	1, 116	.064	
Stress condition x Sex	4.51*	2, 116	.072	
				R^2 = .259 Adjusted R^2 = .214

Note. PA = positive affect; df_b = degrees of freedom between subjects; df_w = degrees of freedom within subjects; Videogame exposure = a composite measure of the frequency and duration of participants' self-reported videogame-play habits.

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

Table 12.

Comparison of Positive Affect Change (Post-Baseline to Post-Gameplay) across Sex

Sex	*n*	*M*	95% CI for *M*		*SE*	Significance (*p*) of pairwise comparison[a]
			LL	*UL*		Female
Female	66	-4.80	-5.80	-3.79	0.51	
Male	58	-2.55	-3.63	-1.46	0.55	.006

Note. CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

[a]Mean differences were compared across sexes with Bonferroni-adjustment for multiple comparisons.

Table 13.

Analyses of Covariance in Average Change (Baseline to Recovery) for SBP, DBP, and MAP

Effect[a]	Δ SBP			Δ DBP			Δ MAP		
	F ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2
Stress condition	4.50*	2, 106	.078	3.76*	2, 106	.066	6.15**	2, 106	.104
Sex	0.05	1, 106	.000	0.04	1, 106	.000	0.33	1, 106	.003
Stress condition x Sex	1.57	2, 106	.029	0.44	2, 106	.008	1.12	2, 106	.021

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; df_b = degrees of freedom between subjects; df_w = degrees of freedom within subjects.

[a]The *p* values for reported main effects were adjusted for age, race/ethnicity, body mass index (BMI), matched baseline physiological mean, and videogame exposure (a composite measure of the frequency and duration of participants' self-reported videogame-play habits) as covariates.

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

Table 14.

Analyses of Covariance in Average Change (Baseline to Recovery) for SBP and MAP

Effect[a]	Δ SBP			Δ MAP		
	F ratio	df_b, df_w	η_p^2	*F* ratio	df_b, df_w	η_p^2
Stress condition	2.39	2, 119	.044	2.07	1, 119	.038
Social vigilance	9.20**	1, 119	.081	7.02**	1, 119	.063
Social vigilance x Stress condition	1.81	1, 119	.033	1.01	1, 119	.019

Note. SBP = systolic blood pressure; MAP = mean arterial pressure; df_b = degrees of freedom between subjects; df_w = degrees of freedom within subjects.

[a]The *p* values for reported main effects were adjusted for sex, age, race/ethnicity, body mass index (BMI), matched baseline physiological mean, and videogame exposure (a composite measure of the frequency and duration of participants' self-reported videogame-play habits) as covariates.

**p<.01

Figures

Figure 1.

*Schematic of the laboratory protocol (**manipulated variable is bolded**)*

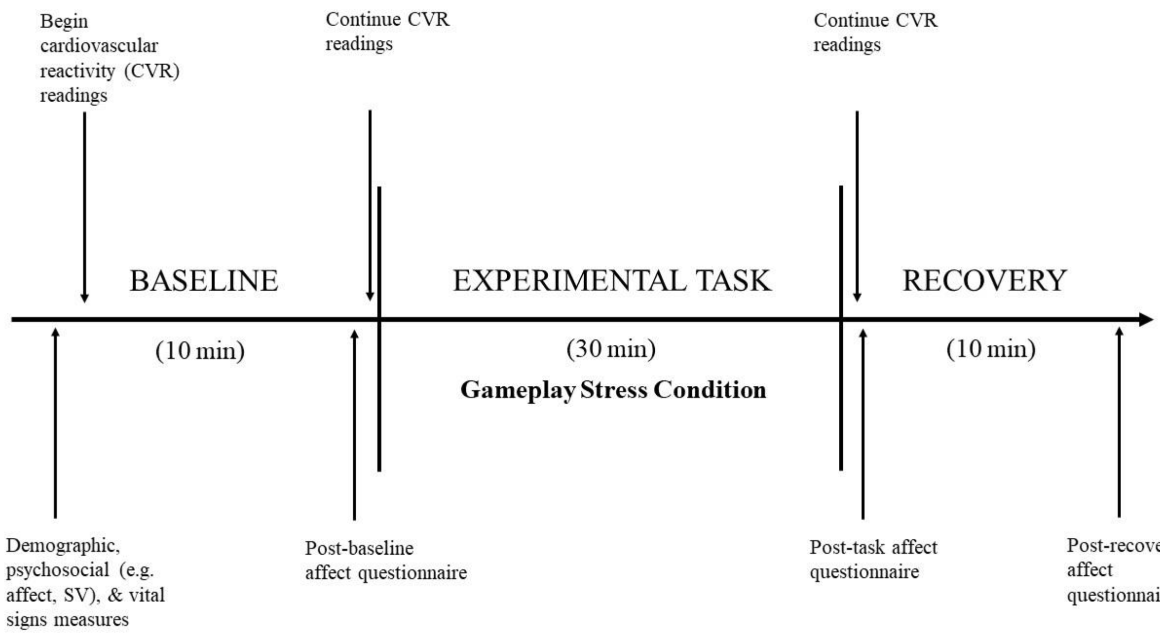
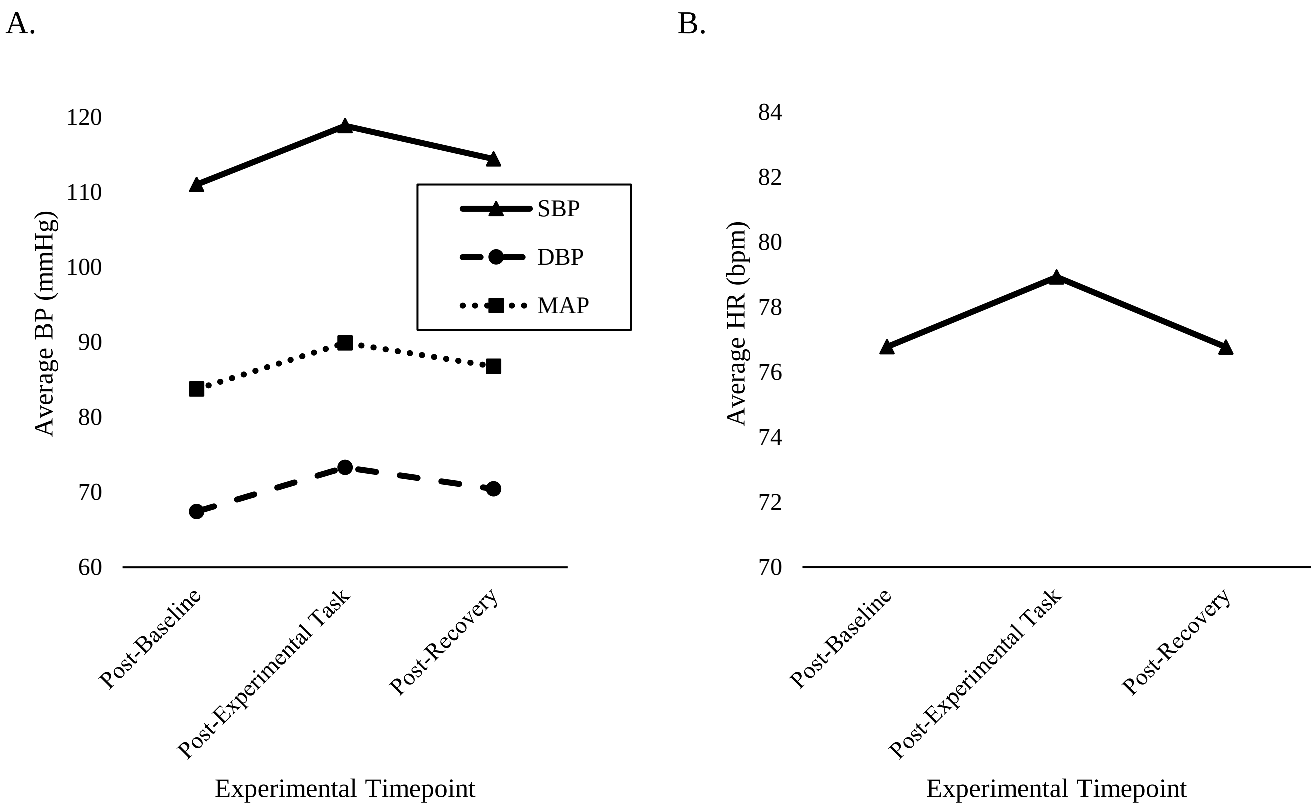


Figure 2.

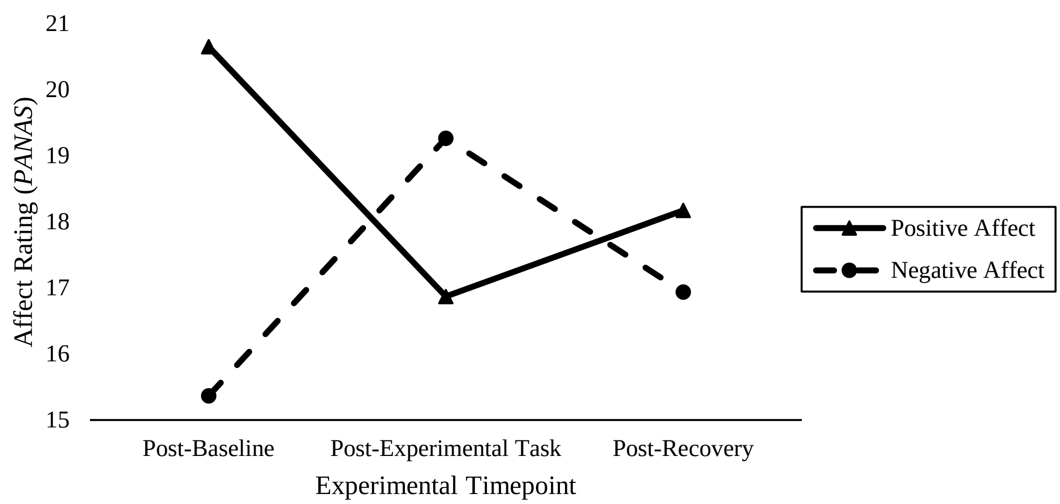
Effects of Experimental Phase on Physiological Variables



Note. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; HR = Heart Rate.

Figure 3.

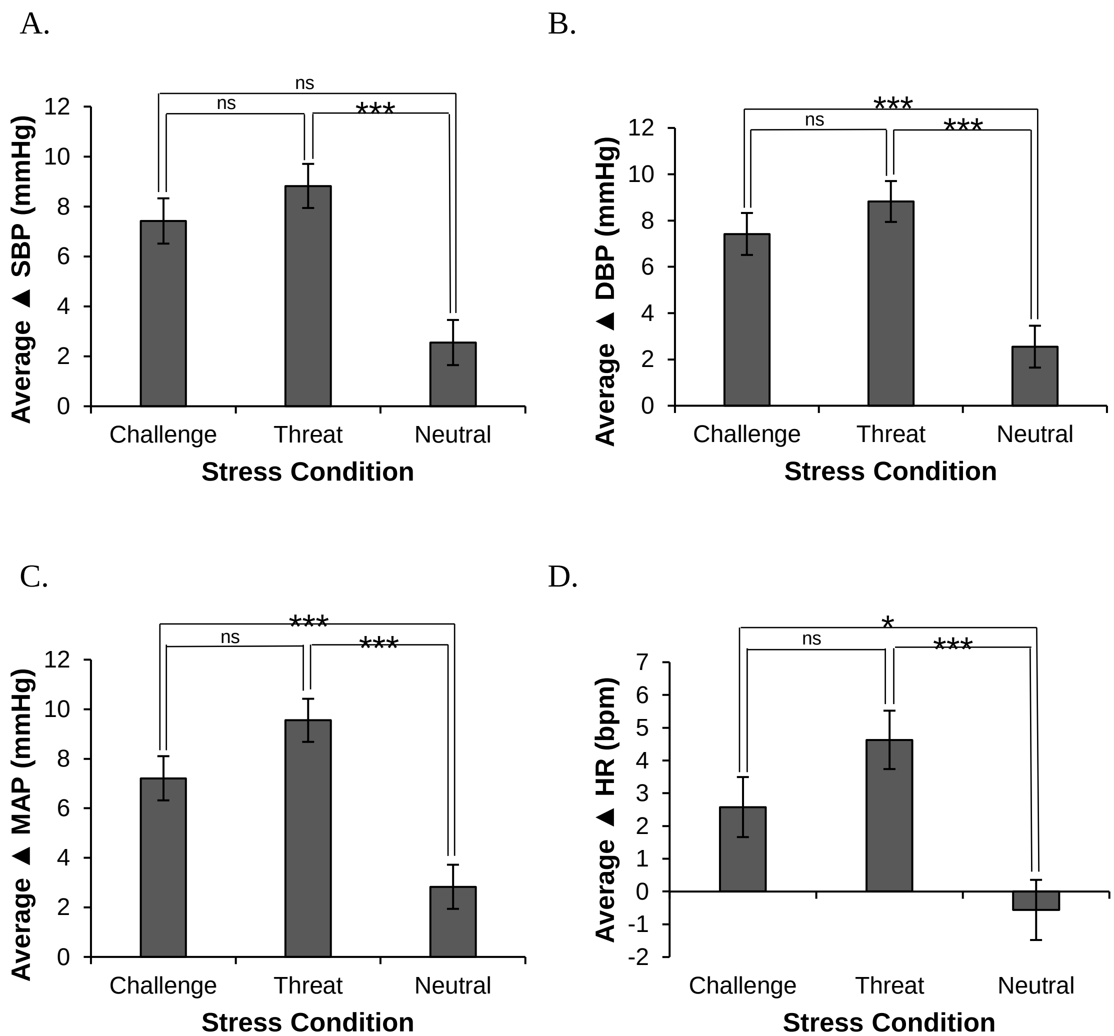
Effects of Experimental Phase on Affective Variables



Note. Self-reported ratings of positive and negative affect were measured via repeated administration of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) following the baseline, experimental task, and recovery periods.

Figure 4.

Average Change (Baseline to Gameplay) in Physiological Variables across Stress Conditions

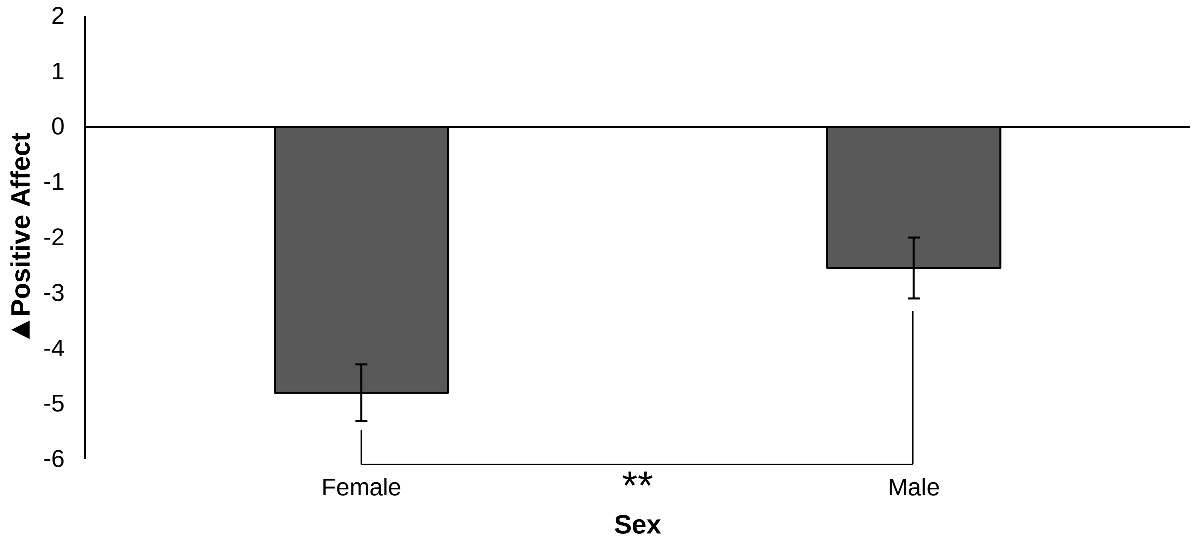


Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HR = heart rate; ns = non-significant difference. Error bars show standard errors. Brackets display the significance of pairwise comparisons of mean differences across stress conditions with Bonferroni-adjustment for multiple comparisons.

$^*p < .05$, $^{***}p < .001$

Figure 5.

Positive Affect Change (Baseline to Gameplay) across Sex

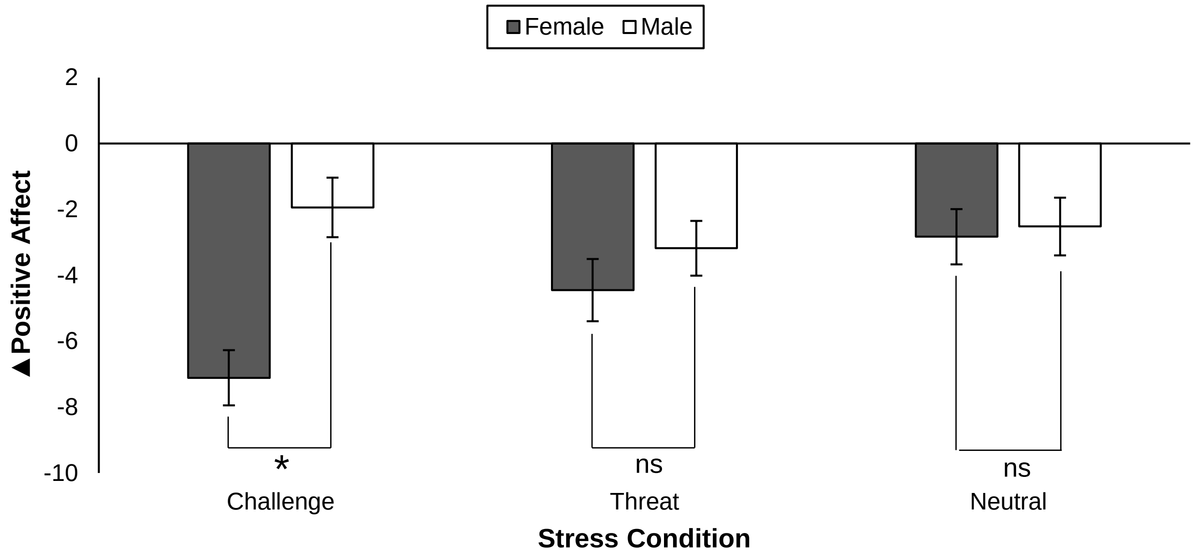


Note. Error bars show standard errors. Bracket displays the significance of the comparison of mean differences across sex with Bonferroni-adjustment for multiple comparisons.

**$p < .01$

Figure 6.

Positive Affect Change (Baseline to Gameplay) across Sex and Stress Conditions

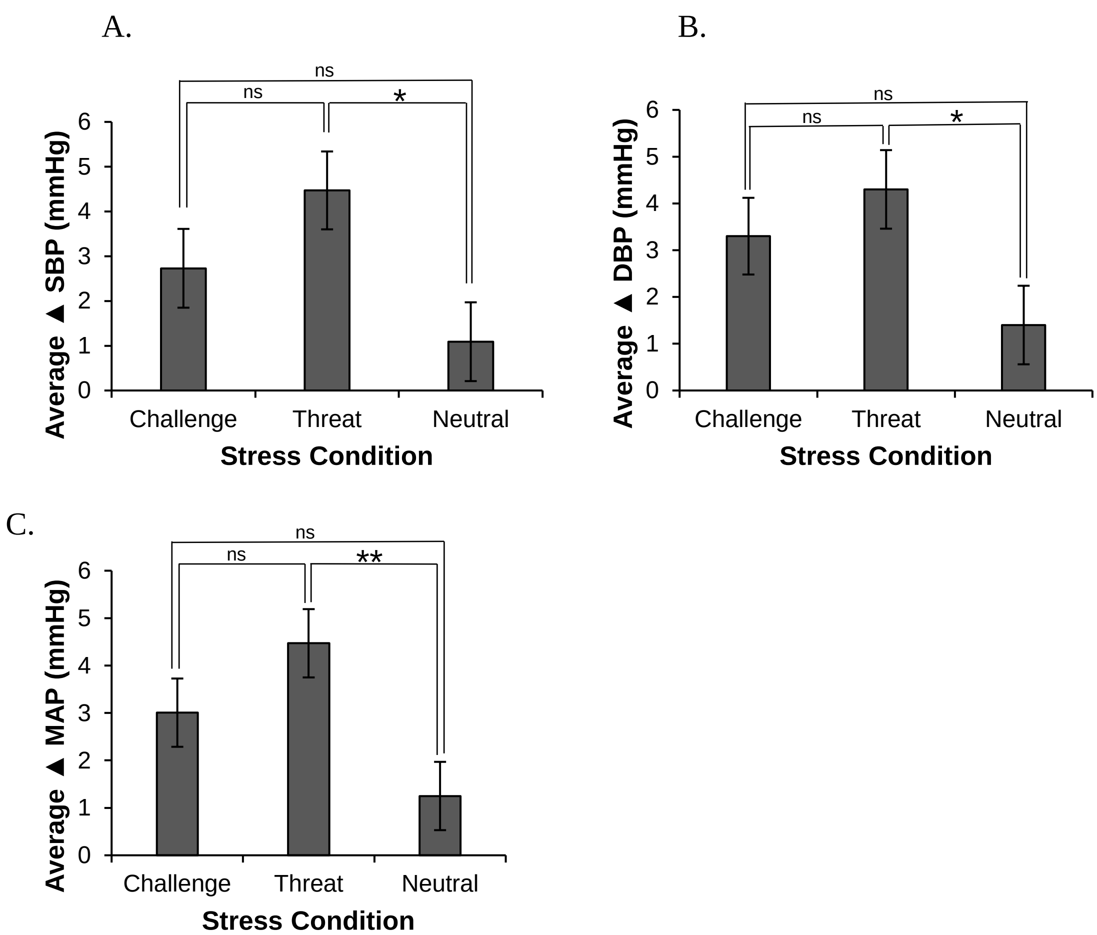


Note. Error bars show standard errors. Brackets display the significance of the comparison of mean differences across sex within stress condition with Bonferroni-adjustment for multiple comparisons.

*p < .05

Figure 7.

Average Change (Baseline to Recovery) in SBP, DBP, and MAP across Stress Conditions

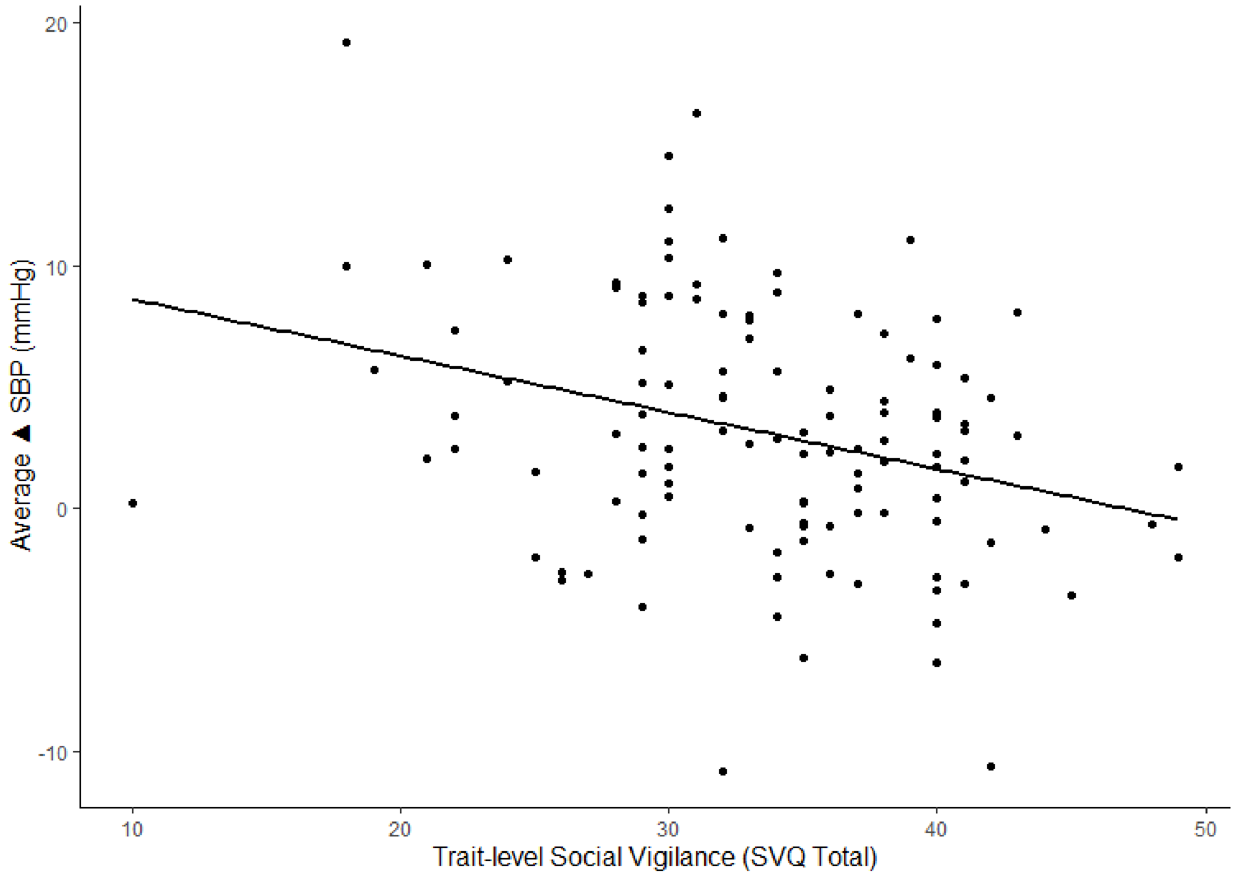


Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; ns = non-significant difference. Error bars show standard errors. Brackets display the significance of pairwise comparisons of mean differences across stress conditions with Bonferroni-adjustment for multiple comparisons.

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

Figure 8.

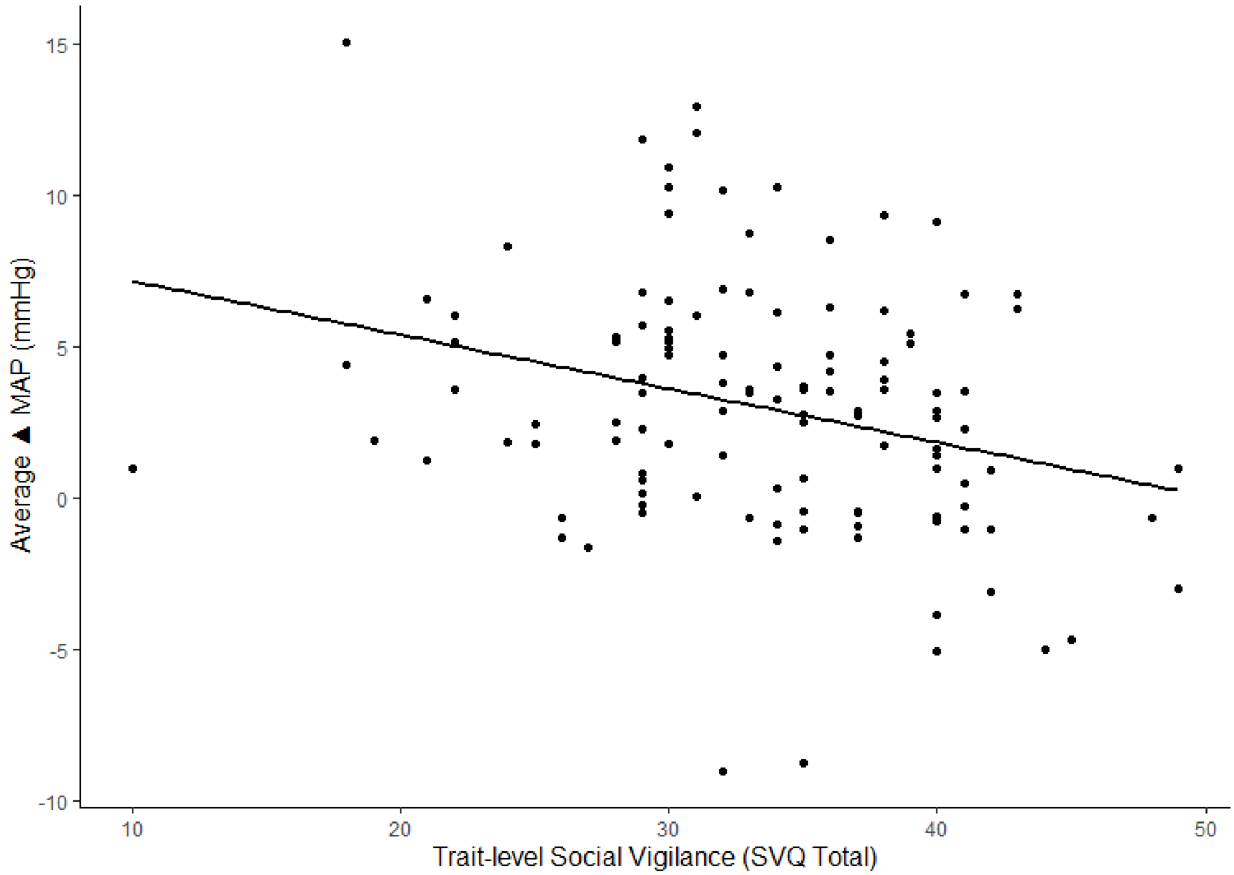
Main Effect of Trait-level Social Vigilance on Average Change (Baseline to Recovery) in SBP



Note. SBP = systolic blood pressure; SVQ = social vigilance questionnaire. Each dot represents an individual participant. Overall, higher levels of social vigilance trait were significantly associated with greater return towards baseline SBP following recovery (black line).

Figure 9.

Main Effect of Trait-level Social Vigilance on Average Change (Baseline to Recovery) in MAP



Note. MAP = mean arterial pressure; SVQ = social vigilance questionnaire. Each dot represents an individual participant. Overall, higher levels of social vigilance trait were significantly associated with greater return towards baseline MAP following recovery (black line).

References

Almeida, D. M. (2005). Resilience and vulnerability to daily stressors assessed via diary methods. *Current Directions in Psychological Science, 14*(2), 64-68.

Anton, S. D., & Miller, P. M. (2005). Do negative emotions predict alcohol consumption, saturated fat intake, and physical activity in older adults?. Behavior Modification, 29(4), 677-688. DOI: 10.1177/0145445503261164.

Bazoukis, G., Tse, G., Naka, K. N., Kalfakakou, V., Vlachos, K., Saplaouras, A., Letsas, K. P., Korantzopoulos, P., Thomopoulos, C., Michelongona, P., Bazoukis, X., Gonzalez-Salvado, V., Liu, T., Michalis, L.K., Baranchuk, A., Itoh, T., Efremidis, M., Tsioufis, C., & Stavrakis, S. (2018). Impact of major earthquakes on the incidence of acute coronary syndromes- A systematic review of the literature. Hellenic Journal of Cardiology, 59(5), 262-267.

Benjamin, E. J., et al. (2019). Heart Disease and Stroke Statistics—2019 Update: A Report from the American Heart Association. Circulation, 139, e56-e528.

Benjamin, L. (1967). Facts and artifacts in using analysis of covariance to "undo" the law of initial values. *Psychophysiology, 4,* 187-206.

Berjot, S., & Gillet, N. (2011). Stress and coping with discrimination and stigmatization. Frontiers in Psychology, 2(33), doi: 10.3389/fpsyg.2011.00033.

Berntson, G. G., Quigley, K. S., Jang, J. F., & Boysen, S. T. (1990). An approach to artifact identification: Application to heart period data. *Psychophysiology, 27*, 586–598. http://dx.doi.org/10.1111/j.1469-8986.1990.tb01982.x

Billman, G. E. (2011). Hear rate variability- A historical perspective. Frontiers in Physiology, 2(86). doi: 10.3389/fphys.2011.00086.

Bishop, S. J., Duncan ,J., & Lawrence, A. D. (2004). State anxiety modulation of the amygdala to unattended threat-related stimuli. Journal of Neuroscience, 24(46), 10364-10348. doi: 10.1523/JNEUROSCI.2550-04.2004.

Bo-chen, Y., Ling-bing, M., Meng-lei, H., Yuan-meng, Z., Gong, T., & Guo, Z. (2019). Chronic stress: a critical risk factor for atherosclerosis. Journal of International Medical Research, 47(4), 1429-1440. :
sagepub.com/journals-permissions
DOI: 10.1177/0300060519826820.

Bodala, I. P., Junhua, L., Thakor, N. V.,& Al-Nashash, H. (2016). EEG and Eye Tracking Demonstrate Vigilance Enhancement with Challenge Integration. Frontiers in Human Neuroscience, 10, 273. DOI=10.3389/fnhum.2016.00273.

Brosschot, J. F. (2010). Markers of chronic stress: Prolonged physiological activation and (un)conscious perservative cognition. Neuroscience and Biobehavioral Reivews, 25, 46-50. doi:10.1016/j.neubiorev.2010.01.004.

Brosschot, J. F., Pieper, S., Thayer, J. F. (2005). Psychoneuroendocrinology, 30, 1043-1049. doi:10.1016/j.psyneuen.2005.04.008.

Brosschot, J. F., Verkuil, B., & Thayer, J. F. (2018). Generalized Unsafety Theory of Stress: Unsafe environments and conditions, and the default stress response. International Journal of Environmental Research and Public Health, 15, doi:10.3390/ijerph15030464.

Brosschot, J.F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. Journal of Psychosomatic Research, 60, 113-124. doi:10.1016/j.jpsychores.2005.06.074.

Chen, Y., & Zhao, Y. (2015). Intergroup threat gates social attention in humans. Biology Letters, 11(2). https://doi.org/10.1098/rsbl.2014.1055.

Cheng, S., Clagget, B., Correia, A. W., Shah, A. M., Gupta, D. K., Skali, H., Ni, H., Rosamond, W. D., Heiss, G., Folsom, A. R., Coresh, J., & Solomon, S. D. (2014). Temporal trends in the population attributable risk for cardiovascular disease: The atherosclerosis risk in communities study. Circulation, 130(10), 820-828. https://doi.org/10.1161/CIRCULATIONAHA.113.008506.

Clancy, F., Prestwich, A., Caperon, L., & O'Connor, D. B. (2016). Perseverative cognition and Health Behaviors: A systemtic review and meta-analysis. Frontiers in Human Neuroscience, 10. doi:10.1016/j.neubiorev.2010.01.004.

Cundiff, J. M., & Smith, T. W. (2017). Social status, everyday interpersonal processes, and coronary heart disease: A social psychophysiological view. Social and Personality Psychology Compass, 11.https://doi.org/10.1111/spc3.12310

Damjanovic, L., Pinkham, A. E., Clarke, P., & Phillips, J. (2014). Enhanced threat detection in experienced riot police officers: Cognitive evidence from the face-in-the-crowd effect. Quarterly Journal of Experimental Psychology, 67(5), 1004-1018. https://doi.org/10.1080/17470218.2013.839724.

Dobson, A. J., Alexander, H. M., Malcolm, J., Steele, P. L., & Miles, T. A. (1991). Heart attacks and the Newcastle earthquake. The Medical Journal of Australia, 155(11-12), 757-761. https://doi.org/10.5694/j.1326-5377.1991.tb94029.x.

Dufford, A. J., Bianco, H., & Kim, P. (2019). Socioeconomic disadvantage, brain morphometry, and attentional bias to threat in middle childhood. Cognitive, Affective, Behavioral Neuroscience, 19, 309-326. https://doi.org/10.3758/s13415-018-00670-3.

Epel, E. S., Crosswell, A. D., Mayer, S. E., Praither, A. E., Slavich, G. M., Puterman, E., Berry Mendes, W. (2018). More than a feeling: A unified view of stress measurement for population science. *Frontiers in Neuroendocrinology, 49*, 146-169. https://doi.org/10.1016/j.yfrne.2018.03.001.

Ewart, K., Jorgenson, R. S., Schroder, K. E., Suchday, S., & Sherwood, A. (2004). Vigilance to a persisting personal threat: Unmasking cardiovascular consequences in adolescents with the Social Competence Interview. *Psychophysiology, 41*(5), 799-804. https://doi.org/10.1111/j.1469-8986.2004.00199.x.

Ferrie, J. E., Kivimaki, M., Shipley, M. J., Smith, G. D., & Virtanen, M. (2013). Job insecurity and incident coronary heat disease: The White Hall II prospective cohort study. Atherosclerosis, 227(1), 178-181. https://doi.org/10.1016/j.atherosclerosis.2012.12.027.

Folkman, S. (1984). Personal control and stress and coping processes: a theoretical analysis. Journal of Personality and Social Psychology, 46, 839-852.

GE. (2016). Carescape V100 Vitals Monitor. [Apparatus]. https://www.gehealthcare.com/products/patient-monitoring/patient-monitors/carescape-v100.

Gerin, W., Bovbjerg, D. H., Glynn, L., Davidson, K., Sanders, M., Sheffield, D., & Christenfield, N. (1999). Comment on “Negative emotions and acute cardiovascular responses to laboratory challenges.” *Annals of Behavioral Medicine, 21*(3), 223-224.

Gerin, W., Davidson, K., Christenfield, N. J. S., Goyal, T., & Schwartz, J. E. (2006). The role of angry rumination and distraction in blood pressure recovery from emotional arousal. Psychosomatic Medicine, 68(1), 64-72. doi: 10.1097/01.psy.0000195747.12404.aa.

Goldstein, D. S., & Kopin, I. J. (2009). Evolution of concepts of stress. The International Journal on the Biology of Stress, 10(2), 109-120. https://doi.org/10.1080/10253890701288935.

Gump, B.B., & Matthews, K. A. (1998). Vigilance and Cardiovascular Reactivity to Subsequent Stresors in Men: A preliminary study. Health Pscyhology, 17(1), 93-96.

Heponiemi, T., Elovainio, M., Pulkki, L., Puttonen, S., Raitakari, O., & Keltikangas-Järvinen, L. (2007). Cardiac autonomic reactivity and recovery in predicting carotid atherosclerosis: The cardiovascular risk in young finns study. Health Psychology, 26(1), 13–21. https://doi.org/10.1037/0278-6133.26.1.13

Hilmert, C., & Kvasnicka, L. (2010). Blood Pressure and Emotional Responses to Stress: Perspectives on Cardiovascular Reactivity. *Social and Personality Psychology Compass*, *4*(7), 470-483. https://doi.org/10.1111/j.1751-9004.2010.00275.x.

Himmelstein, M. S., Young, D. M., Sanchez, D. T., & Jackson, J. J. (2015). Vigilance in the discrimination-stress model for Black Americans. Psychology & Health, 30(3), 253-267. http://dx.doi.org/10.1080/08870446.2014.966104.

Jennings, R. T., Kamarck, T. W., Everson-Rose, S.A.,Kaplan, G. A., Manuck, S. B., & Salonen, J T. (2004). Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged Finnish men. Circulation, 2198-2203. DOI: 10.1161/01.CIR.0000143840.77061.E9.

Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, P. (1992). Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. Psychophysiology, 29(6), 742-750. doi: 10.1111/j.1469-8986.1992.tb02052.x.

Johns, M., Inzlicht, M., & Schmader, T. (2008). Stereotype Threat and Executive Resource Depletion: Examining the Influence of Emotion Regulation. *Journal of Experimental Psychology: General, 137*(4), 691-705. https://doi.org/10.1037/a0013834.

Jorgenson, R. S., & Kolodziej, M. E. (2007). Suppressed anger, evaluative threat, and cardiovascular reactivity: A tripartite profile approach. *International Journal of Psychophysiology*, *66*(2), 102-108. https://doi.org/10.1016/j.ijpsycho.2007.03.015.

Kabutoya, T., & Kario, K. (2009). Earthquake and blood pressure. Hypertension Research, 32, 732-734. doi:10.1038/hr.2009.104

Kaplan, J. R., Chen, H., & Manuck, S. B. (2009). The relationship between social status and atherosclerosis in male and female monkeys as revealed by meta-analysis. American Journal of Primatology, 71, 732-741

Kawai, T., Ohishi, M., Ito, N., Onishi, M., Takeya, Y., Yamamoto, K., Kamide, K., & Rakugi, H. (2013). Alteration of vascular function is an important factor in the correlation between visit-to-visit blood presure variability and cardiovascular disease. Journal of Hypertension, 31(7), 1387-1395. doi: 10.1097/HJH.0b013e328360f796.

Kelsey, R. M. (2012). Beta-adrenergic cardiovascular reactivity and adaptation to stress: The cardiac pre-ejection period as an index of effort. In R. A. Wright & G. H. E. Gendolla (Eds.), How motivation affects cardiovascular response: Mechanisms and applications (p. 43–60). American Psychological Association. https://doi.org/10.1037/13090-002

Kiecolt-Glaser, J. K., Renna, M. E., Shrout, M. R., & Madison, A. E. (2020). Current Directions in Psychological Science 29(5), 491-498. DOI: 10.1177/0963721420949521

Koster, E. W., Crombez, G., Verschuere, B., & De Houwer, J. (2006). Attention to threat in anxiety-prone individuals: Mechanims underlying attentional bias. Cognitive Therapy and Research, 30, 635-643.

Kouveonen, A., Kivimaki, M., Vaananen, A., Heponiemi, T., Elovainio, M., Ala-Mursula, L., Virtanen, M., Pentti, J., Linna, A., & Vahtera, J. (2007). Job strain and adverse health behaviors: The Finnish public sector study. Journal of Occupational and Environmental Medicine, 49(1), 68-74. doi: 10.1097/JOM.0b013e31802db54a.

Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. Annual Review of Psychology, 53, 341-369.

Kraus, M. W., Horberg, E. J., Goetz, J. L., & Keltner, D. (2011). Social Class Rank, Threat Vigilance, and Hostile Reactivity. Personality and Social Psychology Bulletin, 37(10), 1376–1388. https://doi.org/10.1177/0146167211410987

Lanius, R. A., Rabellino, D., Boyd, J. E., Harricharan, S., Frewen, P. A., & McKinnon, M. C. (2017). The innate alarm system in PTSD: conscious and subconscious processing of threat. *Current Opinion in Psychology*, *14,* 109-115. https://doi.org/10.1016/j.copsyc.2016.11.006.

Llabre, M. M., Spitzer, S. B., Saab, P. G., Ironson, G. H., & Schneiderman, N. (1991). The reliability and specificity of delta versus residualized change as measures of cardiovascular reactivity to behavioral challenges. *Psychophysiology, 28*, 701–711. http://dx.doi.org/10.1111/j.1469-8986.1991.tb01017.x

Lommen, M. J. J., Englehard, I. M., & van den Hout, M. A. (2010). Neuroticism and avoidance of ambiguous stimuli: Better safe than sorry?. Personality and Individual Differences, 49(8), 1001-1006. https://doi.org/10.1016/j.paid.2010.08.012.

Lovallo, W. L. (2015). Can exaggerated stress reactivity and prolonged recovery predict negative healthoutcomes? The case of cardiovascular disease. Psychosomatic Medicine, 77, 212-214. DOI: 10.1097/PSY.0000000000000173.

Low, C. A., Thurston, R. C., & Matthews, K. A. (2010). Psychosocial factors in the development of heart disease in women: current research and future directions. Psychosomatic Medicine, 72(9), 842-854. doi: 10.1097/PSY.0b013e3181f6934f..

Masi, C. M., Hawkley, L. C., Rickett, E. M., & Cacioppo, J. T. (2007). Respiratory sinus arrhythmia and diseases of aging: Obesity, diabetes mellitus, and hypertension. Biological Psychology, 74(2), 212-223. https://doi.org/10.1016/j.biopsycho.2006.07.006.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. Annals of the New York Academy of Sciences, 840(1), 33-44. https://doi.org/10.1111/j.1749-6632.1998.tb09546.x.

McEwen, B. S., & Wingfield, J. (2003). The concept of allostasis in biology and medicine. Hormones and Behavior, 43, 2-15.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. Behaviour Research and Therapy, 28(6), 487-495. https://doi.org/10.1016/0005-7967(90)90135-6.

Merritt, M. M., Bennett, G. G., Williams, R. B., Edwards, C. L., & Sollers, J. J. (2006). Perceived racism and cardiovascular reactivity and recovery to personally relevant stress. *Health Psychology*, *25*(3), 364-369. https://doi.org/10.1037/0278-6133.25.3.364.

Microsoft. (2016). Xbox One S video game console. [Apparatus and software]. https://www.xbox.com/en-US/consoles/xbox-one-s.

Mindware Technologies. (2016). 2000 D Impedance cardiograph. [Apparatus and software]. https://mindwaretech.com/hrv-analysis-software/.

Moller, J., Theorell, T., de Faire, U., Ahlbom, A., & Hallqvist, J. (2005). Work related stressful life events and the risk of myocardial infarction. Case-control and case-crossover analyses within the Stockholm heart epidemiology programme (SHEEP). Journal of Epidmeiology & Community Health, 59(1), 23-30. doi: 10.1136/jech.2003.019349.

O'Connor, D. B., Conner, M., Jones, F., McMillan, B., & Ferguson, E. (2009). Exploring the benefits of consciousness: An investigation of the role of daily stressors and health behaviors. Annals of Behavioral Medicine, 37, 184-196. DOI 10.1007/s12160-009-9087-6.

O'Connor, D. B., Walker, S., Hendrickx, H., Talbot, D., & Schaefer, A. (2012). Stress-related thinking predicts the cortisol awakening response aqnd somatic symptoms in health adults. Psychoneuroendocrinology, 38, 438-446. http://dx.doi.org/10.1016/j.psyneuen.2012.07.004.

Ottaviani, C., Thayer, J.F., Verkuil, B., Lonigro, A., Medea, B., Couyoumdjian, A., & Brosschot, J.F. (2016). Physiological concomitants of perseverative cognition: A systematic review and meta-analysis. Psychological Bulletin, 142(3), 231-259. doi: 10.1037/bul0000036.

Pickering, T. G. (2001). Mental stress as a causal factor in the development of hypertension and cardiovascular disease. Current Hypertension Reports, 3, 249-254.

Repetti, R. L., Taylor, S. E., Seeman, T. E. (2002). Risky Families: Family Social Environments and the Mental and Physical Health of Offspring. *Psychological Bulletin, 128*(20, 330-366.

Roelofs, K., Bakvis, P., Hermans, E. J., van Pelt, J., & van Honk, J. (2007). The effects of social stress and cortisol responses on the preconscious selective attention to social threat. Biological Psychology, 75(1), 1-7. https://doi.org/10.1016/j.biopsycho.2006.09.002.

Rosengreen, A., Hawken, S., Ounpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., Blackett, K. N., Sitthi-amorn, C., Sato, H., & Yusuf, S. (2004) Association of psychosocial risk factors with risk of acute myocardial infarction in 11 119 cases and 13 648 controls from 52 countries (the INTERHEART study): case-control study. The Lancet, 364(9438), 954-962. DOI:https://doi.org/10.1016/S0140-6736(04)17019-0.

Ruiz, J. M., Taylor, D. J., Uchino, B. N., Smith, T. W., Allison, M., Ahn, C., Johnson, J. J., & Smyth, J. W. (2017). Evaluation the longitudinal risk of social vigilanceon atherosclerosis: study protocl for the North Texas Heart Study. BMJ Open, 7(8), http://dx.doi.org/10.1136/bmjopen-2017-017345.

Schamder, T., Johns, M., & Forbes, C. (2008). An Integrated Process Model of Stereotype Threat Effects on Performance. *Psychological Review, 115*(2), 336-356. https://doi.org/10.1037/0033-295X.115.2.336.

Selye, H. (1946). The genderal adaptation syndrome and the diseases of adaptation. The Journal of Clinical Endocrinology & Metabolism, 6(2), 117-230. https://doi.org/10.1210/jcem-6-2-117.

Slavich, G. M. (2018). Stressnology: The primitive (and problematic) study of life stress exposure and pressing need for better measurment. Brain, Behavior, and Immunity, 75, 3-5. https://doi.org/10.1016/j.bbi.2018.08.011.

Slavich, G. M. (2020). Social Safety Theory: A Biologically Based Evolutionary Perspective on Life Stress, Health, and Behavior. *Annual Review of Clinical Psychology*, *16*, 265-95. https://doi.org/10.1146/annurev-clinpsy-032816-045159.

Smith, T. W., & Ruiz, J. M. (2002). Psychosocial Influences on the Development and Course of Coronary Heart Disease: Current Status and Implications for Research and Practice. Journal of Counseling and Clinical Psychology, 70 (3), 548-568. DOI: 10.1037//0022-006X.70.3.548.

Smith, T. W., Ruiz, J. M., & Uchino, B. N. (2009). Vigilance, Active Coping, and Cardiovascular Reactivity During Social Interaction in Young Men. Health Psychology, 19(4), 382-392.

Smyth, J. M., Sliwinski, M. J., Zawadzki, M. J., Scott, S. C., Conroy, D. E., Lanza, S. T., Marucsson-Clavertz, D., Kim, J., Stawski, R. S., Stoney, C. M., Buxton, O. M., Sciamanna, C. N., Green, P. M., & Almeida, D. M. (2018). Everyday stress response targets in the science of behavior change. Behaviour Research and Therapy 101, 20-29. https://doi.org/10.1016/j.brat.2017.09.009

Smyth, J., Zawadzki, M., & Gerin, W. (2013). Stress and Disease: A Structural and Functional Analysis. Social and Personality Psychology Compass, 7(4), 217-227. 10.1111/spc3.12020.

Strike, P. C., Magid, K., Whitehead D. L., Battacharyya, M. R., & Steptoe, A. (2005). Pathophysiological processes underlying emotional triggering of acute cardiac events. Proceedings of the National Academy of Sciences of the United States in America, 103(11), 4322-4327. https://doi.org/10.1073/pnas.0507097103.

Teiber, F., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. Journal of Biobehavioral Medicine, 65(1), 46-62.

Thayer, J. F., Hansen, A. L., & Johnsen, B. J. (2010). The Non-invasive Assessment of Autonomic Influences on the Heart Using Impedance Cardiography and Heart Rate Variability. In A. Steptoe (Ed.), Handbook of behavioral medicine (pp. 723-740). Springer.

Thurston, R. C., Carson, M. Y., Koenen, K. C., Change, Y., Matthews, K. A., von Kanel, R., & Jennings, J. R. (2020). The Relationship of Trauma Exposure to Heart Rate Variability During Wake and Sleep in Midlife Women. *Psychophysiology, 57*(4). https://doi.org/10.1111/psyp.13514.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: a psychometric analysis. Cognitive Therapy and Research, 27, 247–259.

Turner, A. I., Smyth, N., Hall, S. J., Torres, S. J., Hussein, M., Jayasinghe, S. U., Ball, K., & Clow, A. J. (2020). Psychological stress reactivity and future health and disease outcomes: A systematic review of prospective evidence. Psychoneuroendocrinology, 114. https://doi.org/10.1016/j.psyneuen.2020.104599.

Vella, E. J., & Friedman, B. H. (2009). Hostility and anger in: Cardiovascular reactivity and recovery to mental arithmetic stress. *International Journal of Psychophysiology*, *72*(3), 253-259. https://doi.org/10.1016/j.ijpsycho.2009.01.003.

Verkuil, B., Brosschot, J. F., de Beurs, D. P., & Thayer, J. F. (2009). Effects of explicit and implicit perseverative cognition on cardiac recovery after cognitive stress. International Journal of Psychophysiology, 74, 220-228. doi:10.1016/j.ijpsycho.2009.09.003.

Verkuil, B., Brosschot, J. F., Meerman, E. E., & Thayer, J. F.. (2012). Effects of momentary assessed stressful events and worry episodes on somatic health complaints. Psychology and Health, 27(2), 141-158. http://dx.doi.org/10.1080/08870441003653470.

Verkuil, B., Brosschot, J. F., Tollenaar, M. S., Lane, R. D., & Thayer, J. F. (2016). Prolonged Non-metabolic Heart Rate Variability Reduction as a Physiological Marker of Psychological Stress in Daily Life. Annals of Behavioral Medicine, 50, 704-714. DOI 10.1007/s12160-016-9795-7.

Wickens, T. D., & Keppel, G. (2004). *Design and analysis: A researcher's handbook.* Prentice-Hall.

Wang, H. W., Leineweber, C., Kirkeeide, R., Svane, B., Schenck-Gustafsson, K., Theorell, T., & Orth-Gomer, K. (2007). Psychosocial stress and atherosclerosis: family and work stress accelerate progression of coronary disease in women. The Stockholm Female Coronary Angiography study. Journal of International Medicine, 261, 245-254. doi: 10.1111/j.1365-2796.2006.01759.x.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology, 54(6), 1063–1070.

Xu, J.Q., Murphy, S. L., & Arias, E. (2018). Mortality in the United States, 2018. NCHS Data Brief, no 355. Hyattsville, MD: National Center for Health Statistics. 2020.

Zoccola, P. M., Dickerson, S. S., & Yim, I. S. (2011). Trait and state perseverative cognition and the cortisol awakening response. Psychoneuroimmunology, 36, 592-595. doi:10.1016/j.psyneuen.2010.10.004.